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Abstract N°: 6

Dual Targeted IL-23 and IL-31 Blockade Enables Continuation of Immune Checkpoint Inhibitor Therapy in Immune-Related Psoriasis Flare and Refractory Pruritus

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Introduction

Dermatologic immune-mediated toxicities may constitute a critical barrier to potentially life-prolonging oncologic therapy. Immune-driven pruritus and psoriasis flares during platinum-based cytotoxic chemotherapy and immune checkpoint inhibitor therapy are particularly challenging, especially when intractable pruritus is present and broad systemic immunosuppression is undesirable. IL-23 acts as a key cytokine in the pathogenesis of psoriasis, driving Th17 cell differentiation and the production of pro-inflammatory mediators such as IL-17 and IL-22. IL-31 is a key mediator of neuroimmune itch signaling and represents a targeted therapeutic approach distinct from conventional immunosuppressive strategies.

Case Presentation

We report a 48-year-old male patient with metastatic adenocarcinoma of the gastroesophageal junction (AEG type II according to Siewert, UICC stage IVA) who developed a marked exacerbation of pre-existing psoriasis as well as severe pruritus, extending beyond the psoriatic lesions. The flare was characterized by extensive erythematosquamous plaques and severe, therapy-refractory pruritus with significant sleep disturbance and functional impairment. Due to the intensity of symptoms and reduced overall condition, immune checkpoint inhibitor therapy had to be temporarily paused.Topical anti-inflammatory therapy and supportive measures failed to achieve sufficient control. Given the pruritus-dominant clinical phenotype and the need to avoid systemic immunosuppression in the oncologic setting, treatment with the IL-23 inhibitor risankizumab was initiated for the palmoplantar psoriasis flare, followed by the IL-31 receptor antagonist nemolizumab for pruritus symptoms.Following initiation of IL-23 and IL-31 receptor blockade, a rapid and clinically meaningful reduction in pruritus was observed, accompanied by improvement of cutaneous inflammation and overall quality of life. The antipruritic response enabled resumption and continuation of immune checkpoint inhibitor-based oncologic therapy without further dermatologic interruption. Treatment was well tolerated, with no relevant adverse events observed. The patient is currently in oncologic remission with sustained dermatologic stability.

Conclusion

This case illustrates how targeted neuroimmune modulation via IL-23 and IL-31 receptor inhibition can overcome psoriasis and pruritus-driven barriers to essential oncologic therapy. In this patient, selective antipruritic treatment enabled continuation of immune checkpoint inhibitor therapy without the need for broad systemic immunosuppression. To our knowledge, this represents the first published report describing the use of risankizumab and nemolizumab for the management of psoriasis and refractory pruritus in an oncologic setting. Importantly, pruritus is a recognized adverse effect of several cytotoxic agents, including oxaliplatin, a key component of widely used regimens such as FLO cytotoxic chemotherapy. Dermatologists therefore play a critical interdisciplinary role in identifying and managing therapy-limiting cutaneous adverse events in oncologic patients. Effective dermatologic management may therefore allow continuation of potentially life-prolonging cancer therapy while maintaining patient quality of life. Further studies are needed to better define the role of IL-23 and IL-31 blockade in complex oncologic settings with inflammatory dermatoses and severe pruritus.This case further highlights how detailed dermatologic understanding of neuroimmune and inflammatory pathways can benefit

immuno-oncologic therapeutic regimens beyond their regulatory approval. This may enable rational and targeted off-label interventions in complex interdisciplinary clinical settings.

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Safety of dupilumab in pregnancy: final results of the dupilumab prospective pregnancy registry

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Introduction
As many as 10-12% of women of reproductive age are estimated to have asthma or atopic dermatitis, many of whom may require treatment for moderate to severe disease. However, there are limited published data on the safety of dupilumab in pregnancy. To address this gap, a pregnancy registry (NCT04173442) was conducted to study the safety of dupilumab in pregnant women who were being treated for asthma or atopic dermatitis or both.

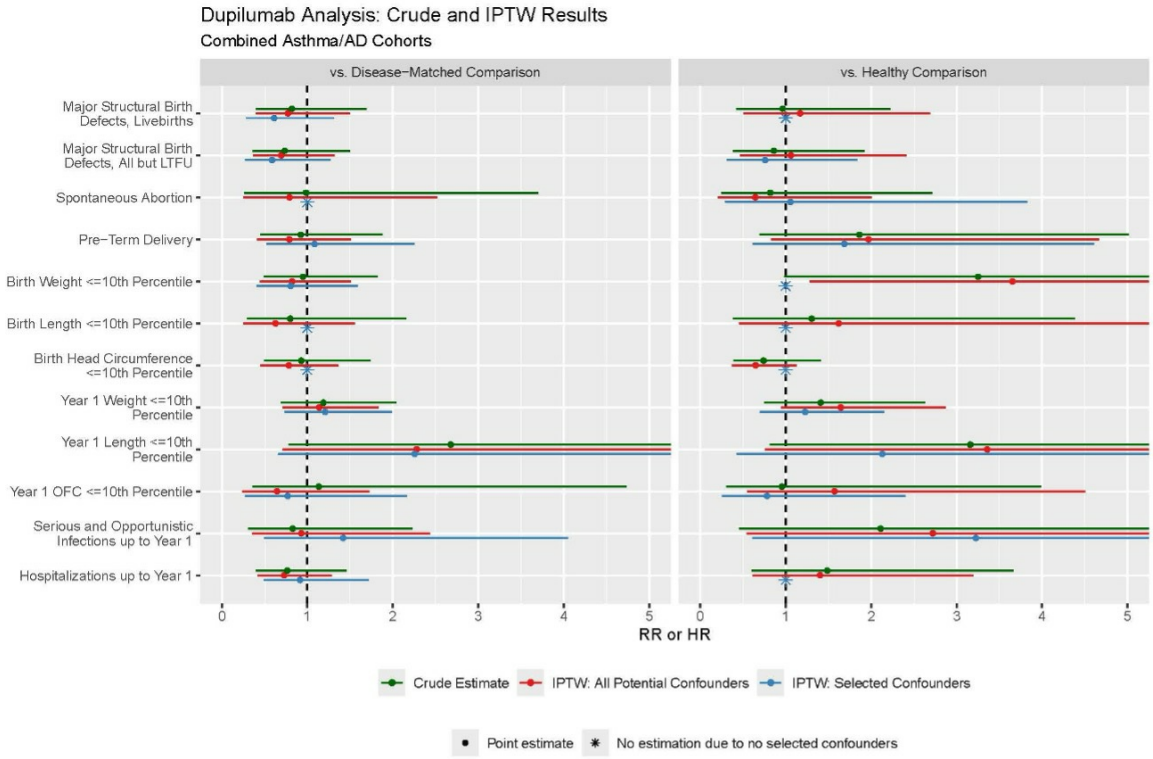
Materials and Methods
A prospective study was conducted by the Organization of Teratology Information Services (OTIS) Research Center between 2018 and 2025. Pregnant women in the U.S. or Canada were enrolled in a dupilumab-exposed group, a disease-matched group that was not exposed to dupilumab, or a non-diseased unexposed group. Outcomes included major birth defects, spontaneous abortion, preterm delivery, pre- and postnatal infant growth, and serious or opportunistic infections or hospitalizations in the child's first year of life. Data were collected from multiple telephone interviews on maternal demographics, pregnancy and medical history, and any exposures to prescription and over-the-counter medications, vaccines, and recreational substances throughout pregnancy. Medical records from the pregnant woman and her infant's health care providers as well as the delivery hospital were obtained and abstracted. Classification of major birth defects was performed by an expert in teratology. Inverse probability of treatment weighted (IPTW) risk or hazard ratios (RRs or HRs) and their 95% confidence intervals (CIs) were calculated including all potential confounders and secondarily using only selected confounders. Primary comparisons were made between the dupilumab-exposed group and the disease-matched group for each outcome, and secondary comparisons were made to the non-diseased group.

Results
A total of 539 pregnant participants enrolled in the study. Of these, 238 were in the dupilumab-exposed group (95 with atopic dermatitis alone; 143 with asthma with or without atopic dermatitis), 175 were in the disease-matched group (33 with atopic dermatitis alone; 142 with asthma with or without atopic dermatitis) and 126 were in the non-diseased group. A total of 14.1% were lost to follow-up. In group comparisons including all potential confounders, the weighted RR for major structural birth defects among liveborn infants was 0.77 (95% CI 0.40, 1.50) in dupilumab-exposed vs disease-matched, and 1.17 (95% CI 0.51, 2.69) vs non-diseased. In the atopic dermatitis alone subset, the weighted RR in dupilumab-exposed vs disease-matched was 0.90 (95% CI 0.10, 8,04),

and 0.66 (95% CI 0.22, 2.01) vs non-diseased. Moreover, there was no evidence of a pattern of birth defects in the dupilumab-exposed group. For spontaneous abortion, the weighted HR was 0.79 (95% CI 0.25, 2.52) for dupilumab-exposed vs disease-matched, and 0.64 (95% CI 0.21, 2.01) vs non-diseased. In the atopic dermatitis alone subset, the HR was not estimated for the primary comparison due to no events in the disease-matched group, and was 0.60 (95% CI 0.16, 2.24) in dupilumab-exposed vs non-diseased. For preterm delivery in the overall cohort, the weighted HR was 0.79 (95% CI 0.41, 1.51) for dupilumab-exposed vs disease-matched, and 1.97 (95% CI 0.83, 4.67) vs non-diseased. In the atopic dermatitis alone subset, the HR was not estimated for the primary comparison due to no events in the disease-matched group, and was 2.28 (95% CI 0.82, 6.35) in dupilumab exposed vs non-diseased. As shown in the figure, there were no significant differences between groups for infant growth, serious or opportunistic infections or hospitalizations with the exception of infants ≤10th centile on birth weight in the dupilumab-exposed vs non-diseased groups.

Conclusion

The findings of this registry were consistent with the limited previous literature and did not suggest an increased risk with dupilumab use in pregnancy for the outcomes studied.



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Bullous pemphigoid induced by antiPD1 therapy second reported case in Algeria

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Introduction

Immunecheckpoint inhibitors, particularly anti-PD-1 antibodies like pembrolizumab,have revolutionized the management of metastatic cancers but are frequentlyassociated with immune-related adverse events. Among these, induced bullouspemphigoid is a rare yet severe cutaneous toxicity, often characterized by adelayed onset of several months following the initiation of therapy. We reportthe clinical case of a pembrolizumab-induced bullous pemphigoid in a patienttreated for metastatic renal cell carcinoma. This case highlights thesignificant therapeutic challenges encountered when managing severe autoimmuneskin toxicities in patients with highly restrictive comorbidities,necessitating a delicate balance between effective immunosuppression and theavoidance of life-threatening metabolic complications.

Case Presentation

A 67-year-old woman presented with a 10-day history of intensely pruritic vesiculobullous eruption. Her history included a solitary kidney (post-nephrectomy for clear cell renal carcinoma), treated cerebral metastasis, and insulin-dependent type 2 diabetes. She was receiving axitinib and pembrolizumab (5 cycles since August 2025).The eruption began with pruritic erythematous papules on the inner thighs, rapidly generalizing and evolving into tense, clear bullae (0.5–3 cm). A 10-day course of oral corticosteroids was ineffective. On admission, she was afebrile with preserved general status. Examination showed a diffuse, symmetrical vesiculobullous eruption on an erythematous base, mainly on the trunk and extensor limbs, sparing face and mucosa; Nikolsky sign was negative. Pruritus was severe and persistent.Laboratory tests revealed eosinophilia (1220/mm^3), lymphopenia (1340/mm^3), and elevated CRP (59.5 mg/L). Histology showed subepidermal blistering. Direct immunofluorescence demonstrated linear IgG, IgA, and C3 deposits along the dermoepidermal junction. ELISA was positive for anti-BP180 (7.5) and anti-BP230 (3).Treatment started with high-potency topical corticosteroids, followed by systemic prednisone (0.5 mg/kg/day) due to partial response. Dose escalation was limited by severe hyperglycemia (fasting glucose 2.1 g/L). Methotrexate (12.5 mg/week) was introduced as a steroid-sparing agent. The patient was discharged with partial disease control (<10 new blisters/day) and a prednisone taper.

Conclusion

Pembrolizumab is highly imputable in the bullous pemphigoid, supported by a compatible temporal relationship and confirmatory immunopathological findings. The underlying mechanism is presumed to involve immune checkpoint inhibition–induced loss of peripheral tolerance, promoting autoreactive T-cell activation with subsequent cross-reactivity against shared epitopes between tumor antigens and components of the dermoepidermal junction, particularly BP180 and BP230. management was constrained by the patient’s comorbid conditions. Although systemic corticosteroids remain the first-line treatment for grade 3 immune-related cutaneous adverse events, their use was limited by poorly controlled insulin-dependent diabetes mellitus.methotrexate was introduced as a steroid-sparing IS agent, achieving satisfactory control of disease activity while minimizing metabolic complications. This case underscores the critical importance of an

individualized, multidisciplinary approach—integrating dermatological, oncological, and endocrinological expertise—in the management of complex immune-related adverse events associated with immune checkpoint inhibitors.

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Abstract N°: 85

Vitiligo improved by ustekinumab in a patient with psoriasis

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Introduction

Psoriasis is an immune-mediated condition characterized by the development of well-defined plaques covered with a silver scale. While medications like cyclosporine and methotrexate can cause significant side effects, biologic therapies now offer a safer alternative. Ustekinumab, an interleukin (IL) 12/23 inhibitor, has been approved by the US Food and Drug Administration for managing moderate to severe chronic plaque psoriasis.Vitiligo is an acquired autoimmune disorder that leads to the loss of skin pigmentation, resulting in distinct depigmented patches. Treatment options for this condition remain limited, often relying on topical or systemic corticosteroids. Despite the available treatments, vitiligo remains a therapeutic challenge. Our aim was to treat a patient’s psoriasis with ustekinumab, and interestingly, it also led to an improvement in her vitiligo.



Case Presentation

We report the case of a 23 year old female, with a medical history of cured pulmonary tuberculosis five years ago. She had no personal history of other associated autoimmune disorders and presented to our clinic with a history of severe psoriasis since the age of 15. In the past, the patient had been treated with topical steroids and phototherapy for her psoriasis and vitiligo with only minimal improvement. During the course of her disease, vitiligo developed, showing a stark contrast under Wood's lamp examination. All tests were normal, including autoimmune and metabolic investigations.Clinical examination revealed erythematous-squamous plaques on the elbows, trunk, abdomen, and lower limbs, associated with multiple depigmented spots, surrounding the psoriatic plaques and on other areas of the body, suggesting an association between vitiligo and psoriasis.We wanted to treat the patient for both psoriasis and vitiligo to achieve effective and safe results. Because of a lack of established protocols in the published literature for associated conditions.Therefore, we administered the biologic

treatment Ustekinumab. No other adjuvant medications were used with ustekinumab. The patient responded well without any side effects.The results were impressive; by week 8, the patient showed a major reduction in psoriatic lesions and achieved complete clearance with a body surface area of 0% by week 16. Surprisingly, the vitiligo also improved significantly.



Conclusion

In recent years, treatment options for plaque psoriasis have increased dramatically because of advances in understanding its immunopathogenesis, different molecular targets, including tumor necrosis factor–alpha, ILs-12 and -23, IL-17, and others, have paved the way for targeted biologic therapies. Although the development of vitiligo is complex and likely results from a combination of genetic predisposition and environmental influences, it is clear that proinflammatory cytokines contribute to the disease process. This suggests that the same treatments

that have been highly effective for psoriasis may also be beneficial for vitiligo. Genomic association studies have identified IL-12 and IL-23 as key players in the pathway involved in psoriasis, making them prime targets for drug development. This research led to the discovery of ustekinumab, an IL-12/23 inhibitor that has demonstrated high effectiveness in treating plaque psoriasis. IL-23 plays a crucial role in the growth and survival of TH17 cells, an important T-cell subset involved in various autoimmune diseases beyond psoriasis, including Crohn's disease, vitiligo, and alopecia areata.It should be noted that the complex pathophysiology of these conditions means that ustekinumab may not have such robust response in all patients with vitiligo. However, the success seen in this patient with concomitant psoriasis and vitiligo should prompt consideration of ustekinumab in those for whom vitiligo have proven refractory to other available treatments. Additional prospective studies will be required to determine the long-term safety and efficacy of ustekinumab in these populations.



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Retrospective Evaluation of High-Dose Omalizumab Use in the Treatment of Refractory Chronic Idiopathic Urticaria: A Real-Life Observational Study

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Introduction

Urticaria is a chronic, inflammatory, recurrent disease that can affect individuals of all age groups. It is classified into two subtypes: acute and chronic. The chronic form affects approximately 1% of the general population. While identifiable triggering factors can be determined in some patients, others are classified as idiopathic. In our study, we aimed to retrospectively evaluate the use of high-dose omalizumab in patients diagnosed with chronic idiopathic urticaria and to contribute our clinical experience to the existing literature.

Materials and Methods

This study was conceived as a prospective study and accomplished under the ethical regulations of the Declaration of Helsinki. It was authorized by the University of Health Sciences Izmir Tepecik Training and Research Hospital Ethical Review Committee (08.12.2025/11-28). Our study was conceived as a retrospective, real-life observational study. Data collection was conducted over the period from January 2020 to October 2025. We recorded age, gender, education, occupation, income, height, weight, body mass index, cigarette smoking, alcohol consumption, allergy, comorbidity, psychiatric disorder because of urticaria, duration of urticaria, angioedema, information about omalizumab treatment, urticaria activity scores 7 (UAS7). Statistical analyses were conducted by the Statistical Package for Social Sciences (SPSS v.24.0, IBM) program. Descriptive data were given as means±standard deviations (SDs) and ranges [minimum–maximum]. Changes in angioedema frequency and UAS7 scores across pre-omalizumab, fixed-dose, and high-dose omalizumab treatment periods were analyzed using the non-parametric Friedman test due to non-normal distribution of the data. The p values were regarded as statistically significant when less than 0.05.

Results

Among 532 patients diagnosed with chronic idiopathic urticaria who had received or were currently receiving omalizumab therapy, we identified 7 patients who were treated with high-dose omalizumab. Six of the patients were female and one was male. Mean patient age was 47.6 ± 13.5 [33–70]. Two patients were housewifes and the average monthly income of the employees was 492 euros. One patient had cardiac arrhythmia and dyspepsia and another patient had hypothyroidism. None of the patients consumed alcohol. The patients had a mean disease duration of 78.8 ± 44.3 [12-120] months. A history of angioedema was present in all patients. Fixed-dose omalizumab therapy was started in the 30.3 ± 37.4rd month of disease for the patients [6–108]. Patients demonstrated a statistically significant decrease in angioedema frequency and UAS7 scores across pre-omalizumab, fixed-dose, and high-dose omalizumab treatment periods ($p = 0.004$, $p = 0.006$, respectively).

Conclusions

Being female and having additional allergic manifestations, excluding food allergy, increase the likelihood of

requiring high-dose omalizumab in the treatment of chronic idiopathic urticaria. Administration of high-dose omalizumab resulted in a statistically significant reduction in angioedema attack frequency and UAS7 scores. Among 532 patients receiving fixed-dose omalizumab, only 7 patients (1.3%) required escalation to high-dose therapy, supporting the high efficacy of fixed-dose omalizumab in the majority of patients.

Tables:

Table 1. Information on the duration of omalizumab treatment of our cases

Case	Duration of urticaria (month)	Duration of omalizumab 300mg every four weeks (month)	Duration of omalizumab 300mg every three weeks (month)	Duration of omalizumab 300mg every two weeks (month)
1	84	33	-	3
2	36	27	3	-
3	120	101	5	2
4	120	4	2	6
5	120	78	6	12
6	60	48	2	2
7	12	2	3	1

Table 2. Information about angioedema frequencies and UAS7

Pre-omalizumab angioedema frequency (mean ± std) (attack frequency/3 month)	22.3 ± 33.7	**UAS7 pre-omalizumab (mean ± std)**	38.3 ± 6.9
Angioedema frequency on fixed-dose omalizumab (mean ± std) (attack frequency/3 month)	18.3 ± 33.4	**UAS7 on fixed-dose omalizumab 3. month (mean ± std)**	15.7 ± 12.2
Angioedema frequency on high-dose omalizumab (mean ± std) (attack frequency/3 month)	13.3 ± 33.8	**UAS7 on high-dose omalizumab 3. month (mean ± std)**	9.8 ± 16.9
P	**0.004***	***P***	**0.006***

UAS7: urticaria activity score 7; *$p \leq 0.05$

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Abstract N°: 134

Systemic Janus Kinase Inhibitors at the Atopic Dermatitis–Psoriasis Interface: A Systematic Review of Overlap and Paradoxical Dermatoses

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Introduction
Psoriasis and atopic dermatitis are usually positioned at opposing immune poles, yet stable overlap and bidirectional treatment-associated switching challenge this binary model. Oral Janus kinase (JAK) inhibition may be suited to these interface states because JAK-STAT signalling integrates multiple cytokine pathways relevant to both phenotypes. We systematically characterised clinical outcomes and the clinicopathologic and molecular organisation of the psoriasis-eczema interface.

Materials and Methods
PubMed, Embase, Scopus and CENTRAL were searched from inception to 18 August 2026. Original human reports describing oral JAK inhibitor treatment for stable psoriasis-eczema overlap, treatment-associated psoriasiform switching or treatment-associated eczematous/atopic dermatitis-like switching were included. Overlapping reports were linked to avoid double counting. Patient-level responses were harmonised as complete/near-complete, substantial/partial or poor/no response. Trigger directionality, simultaneous control of both cutaneous poles, culprit treatment management, durability, lesion-linked histopathology, molecular findings and safety were synthesised separately. The review was prospectively registered in PROSPERO (CRD420251207725).

Results
Sixty-one publications were included; 54 contributed patient-level data for 167 unique JAK-treated patients: 103 with stable overlap, 31 with psoriasiform switching and 33 with eczematous switching. Complete or near-complete response was reported in 113/166 evaluable patients (68.1%), but fell to 79/166 (47.6%) under a stringent endpoint definition.
Trigger direction was strongly organised. All 23 IL-4/IL-13-associated switches were psoriasis-directed, whereas 24/26 IL-17-associated and 7/7 broad IL-23-pathway-associated switches were eczema-directed. This clinical polarity was mirrored histologically: approximately 88% of biopsied stable-overlap patients showed mixed psoriasiform-spongiotic architecture, while psoriasiform switches were predominantly psoriasis-polarised (77-79%) and eczematous switches predominantly spongiotic (67-71%). Limited molecular observations were directionally concordant with these patterns.
Simultaneous control or improvement of both cutaneous poles was documented in 38/59 applicable switch cases and in 38/42 cases with both outcomes separately ascertainable. However, the implicated therapy was withdrawn in 59/60 assessable switches, limiting attribution of improvement specifically to JAK inhibition. Upadacitinib accounted for 122/167 definitive regimens, and safety reporting was insufficient in 29/54 patient-level publications.

Conclusions
The psoriasis-eczema interface shows structured clinicopathologic organisation rather than a simple binary. Oral

JAK-containing strategies were repeatedly associated with improvement across stable and switched states, but response depth was definition-sensitive and, in treatment-associated switching, the JAK-specific contribution was usually inseparable from culprit withdrawal. Prospective phenotype-defined studies should measure both cutaneous poles separately and integrate lesion-linked histopathology with longitudinal clinical and molecular assessment.

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Abstract N°: 145

Efficacy and Safety of Brepocitinib in Cutaneous Sarcoidosis: Results from the Phase II Portion of the Randomized, Double-Blind, Placebo-Controlled BEACON Trial

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Introduction

Cutaneous sarcoidosis (CS) is a highly morbid, granulomatous skin disease with no approved therapies. Current treatment relies on off-label antimalarials, conventional disease-modifying antirheumatic drugs (DMARDs), and other immunomodulatory therapies, often in combination with systemic glucocorticoids; however, complete and durable disease control remains uncommon. Several key cytokines implicated in CS, including IFNγ, IL-6, and IL-12, signal through tyrosine kinase 2 (TYK2) and/or Janus kinase 1 (JAK1), providing a mechanistic rationale for selective TYK2/JAK1 inhibition.

Materials and Methods

BEACON (NCT06978725) is an ongoing, global, multicenter, randomized, double-blind, placebo-controlled Phase 2/3 trial. This prespecified interim analysis evaluated the 16-week, dose-ranging, Phase 2 portion of the study. Adults with biopsy-proven active CS (Cutaneous Sarcoidosis Activity and Morphology Instrument-Activity [CSAMI-A] ≥ 10; range 0–165) were randomized 3:2:2 to once-daily oral brepocitinib 45 mg, brepocitinib 15 mg, or placebo. Stable background antimalarial, tetracycline, and/or DMARD therapy was permitted. Participants receiving oral corticosteroids at baseline underwent a protocol-mandated taper, with complete discontinuation required by Week 8. The primary efficacy endpoint was change from baseline in CSAMI-A at Week 16. Additional endpoints included achievement of Cutaneous Sarcoidosis Activity – Investigator Global Assessment (CSA-IGA) "Clear" or "Almost Clear" with ≥ 2-category improvement, CSAMI-A response (≥ 50% improvement), ≥ 10-point CSAMI-A reduction, achievement of CSAMI-A < 5, patient-reported outcomes (PROs), and safety.

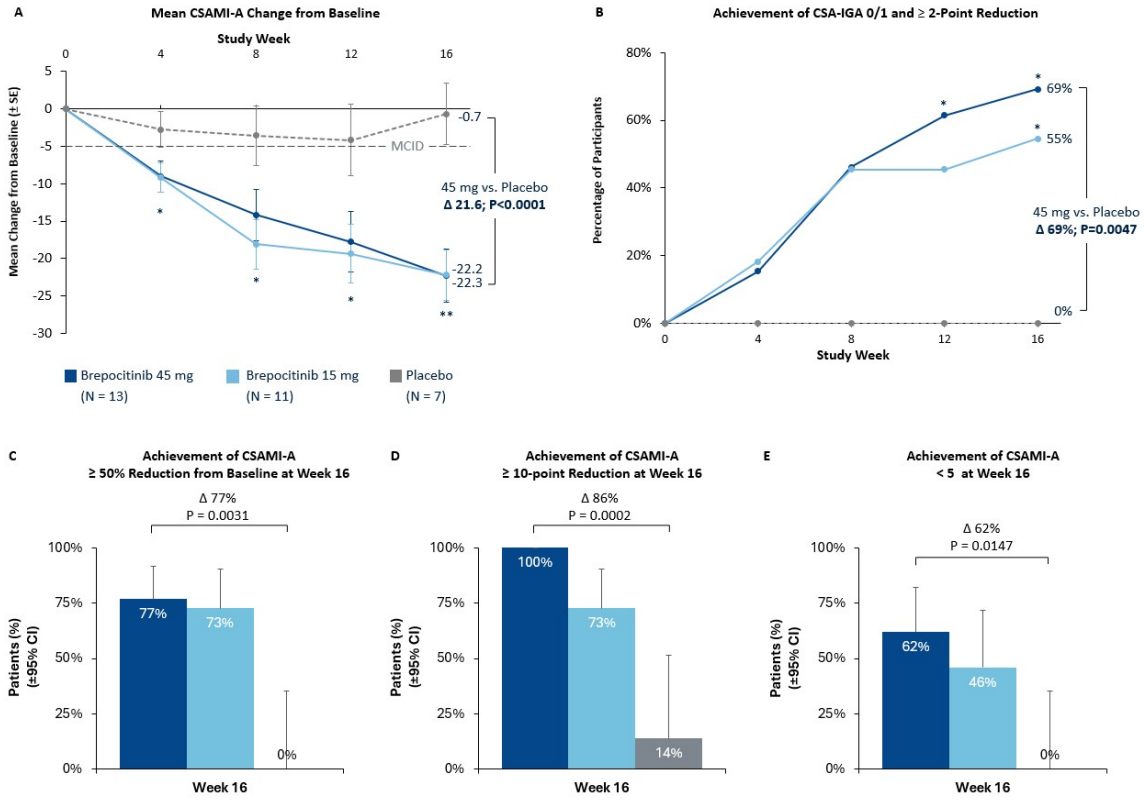

Figure 1. Cutaneous Sarcoidosis Disease Activity and Remission-Oriented Outcomes with Brepocitinib. (A) Mean change from baseline in CSAMI-A and (B) achievement of CSA-IGA 0/1 with a ≥ 2-category improvement over time. Proportion of participants achieving (C) ≥ 50% reduction in CSAMI-A (D) ≥ 10-point reduction in CSAMI-A and (E) CSAMI-A < 5 at Week 16. *P < 0.05; **P < 0.0001.
Abbreviations: CSAMI-A, Cutaneous Sarcoidosis Activity and Morphology Instrument-Activity; MCID, Minimal Clinically Important Difference; SE, standard error; CSA-IGA, Cutaneous Sarcoidosis Activity Investigator Global Assessment

Results

Thirty-one participants (58% female; mean age 53.3 years) were randomized to brepocitinib 45 mg (n=13), brepocitinib 15 mg (n=11), or placebo (n=7). Despite prior and/or concomitant use of off-label systemic therapies, participants had longstanding and highly active skin disease at baseline (mean [SD] disease duration, 8.1 [7.5] years; CSAMI-A, 33.6 [17.6]; CSA-IGA, 3.0 [0.6]). Overall, 52% were receiving at least one CS-directed systemic therapy at baseline. Both brepocitinib doses produced rapid improvements in CS disease activity, with treatment effects evident by Week 4 and increasing through Week 16 (Figure 1A). At Week 16, mean CSAMI-A reduction was 22.3 points with brepocitinib 45 mg versus 0.7 with placebo ($p < 0.0001$) (Figure 1A). Across responder-based and remission-oriented endpoints, efficacy consistently favored brepocitinib 45 mg, supporting a dose-dependent treatment effect. Compared with placebo, brepocitinib 45 mg resulted in higher rates of CSA-IGA "Clear" or "Almost Clear" (69% vs 0%; p=0.0047), CSAMI-A response (77% vs 0%; p=0.0031), ≥ 10-point CSAMI-A reduction (100% vs 14%; p=0.0002), and CSAMI-A < 5 (62% vs 0%; p=0.0147) (Figure 1B-E). Clinical improvements were accompanied by significant benefits in skin-related quality of life (Figure 2A-C). Brepocitinib was well tolerated; no deaths or serious adverse events occurred, and there were no malignancies, major adverse cardiovascular events, venous thromboembolic events, or viral reactivations (Table 1).

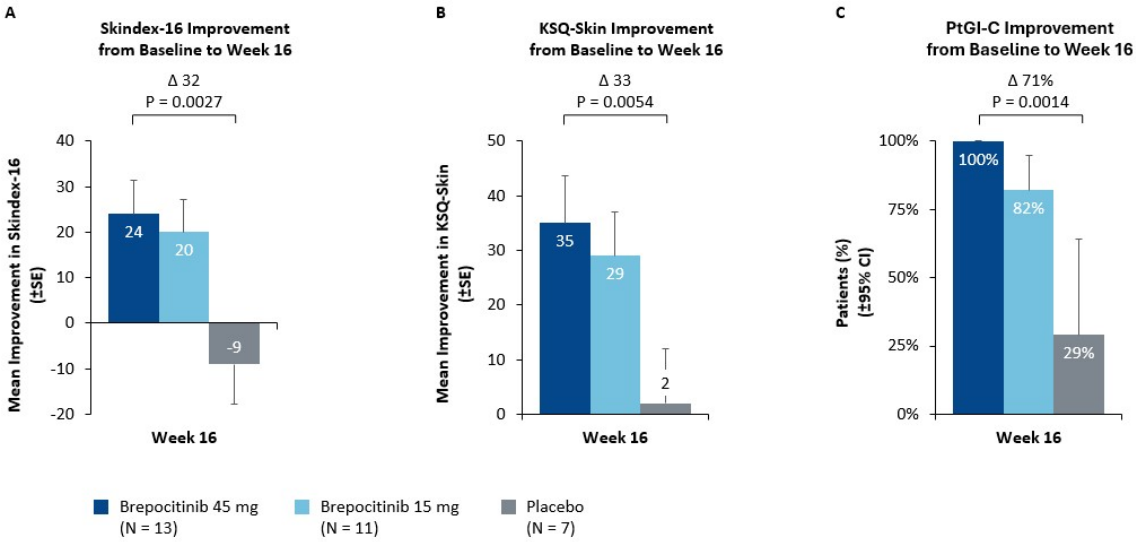

Figure 2. Improvements in Skin-Related Quality of Life and Patient Perception of Disease Activity with Brepocitinib Mean improvement from baseline to Week 16 in Skindex-16 (A) and KSQ Skin (B). Proportion of participants reporting improvement on Patient Global Impression of Change at Week 16 (C). Abbreviations: KSQ, King's Sarcoidosis Questionnaire; PtGI-C, Patient Global Impression of Change; SE, standard error; CI, confidence interval

Conclusion

In the Phase 2, dose-ranging portion of the BEACON trial, brepocitinib 45 mg produced rapid, dose-dependent, and clinically meaningful improvements in CS disease activity, with corresponding improvements in skin-related quality of life and an acceptable safety profile. As the first positive, randomized, placebo-controlled trial to demonstrate efficacy of a targeted therapy in CS, these findings support selective TYK2/JAK1 inhibition as a promising therapeutic strategy and provide a strong rationale for evaluation of brepocitinib 45 mg in the ongoing Phase 3 portion of the BEACON trial.

References: [1] Noe MH, et al. JAMA Dermatol. 2020;156(1):98-99.

Table 1. Summary of Safety Events During the 16-Week BEACON Trial

	Brepocitinib, 45 mg (N=13)	Brepocitinib, 15 mg (N=11)	Placebo (N=7)
	number of patients (percent)		
Adverse events (AEs)	12 (92%)	9 (82%)	6 (86%)
Death	0	0	0
Serious adverse events (SAEs)	0	0	0
AEs leading to treatment discontinuation	1 (8%)	1 (9%)	1 (14%)
AEs leading to study discontinuation	0	0	0
Adverse events of special interest (AESI)[1]			
Creatine kinase (CK) elevation[2]	1 (8%)	0	0
Anemia[3]	1 (8%)	0	0
Viral reactivation	0	0	0
Malignancy	0	0	0
Major adverse cardiovascular event	0	0	0
Venous thromboembolism	0	0	0
Liver enzyme elevation	0	0	0

[1]Both AESIs were moderate in severity.
[2]CK elevation was asymptomatic, did not result in study drug discontinuation, and improved with continued treatment.
[3]Anemia represented worsening of baseline anemia and improved following study drug discontinuation.

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Abstract N°: 146

Golidocitinib Plus Brentuximab Vedotin for Brentuximab Vedotin-Refractory/Relapsed Advanced Folliculotropic Mycosis Fungoides and Sézary Syndrome: A Retrospective Cohort Study

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Introduction
Advanced folliculotropic mycosis fungoides (FMF) and Sézary syndrome (SS) represent aggressive subtypes of cutaneous T-cell lymphoma (CTCL) with limited therapeutic options after brentuximab vedotin (BV) failure. Aberrant JAK/STAT signaling contributes to CTCL pathogenesis, and golidocitinib, a selective JAK1 inhibitor, has shown activity in relapsed/refractory T-cell lymphomas. This study aimed to evaluate the efficacy and safety of golidocitinib plus brentuximab vedotin (Go-BV) in BV-refractory/relapsed advanced FMF and SS.

Materials and Methods
We conducted a retrospective cohort study of 10 patients with histologically confirmed FMF (n=7) or SS (n=3), all with stage ≥IIB disease and prior BV failure. BV refractoriness/relapse was defined as progressive disease after ≥2 cycles, failure to achieve response after ≥3 cycles, or relapse within 12 months after discontinuation. Patients received golidocitinib (150 mg daily, adjustable to every other day) in combination with BV (1.8 mg/kg every 3 weeks). Primary endpoint was objective response rate (ORR). Secondary endpoints included ORR at 4 months (ORR4), progression-free survival (PFS), duration of response (DoR), time to response (TTR), pruritus numeric rating scale (NRS), and treatment-related adverse events (TRAEs).

Results
At a median follow-up of 36.7 weeks, the ORR was 70% (7/10), including 1 complete response and 6 partial responses. ORR4 was 40%. Response rates were higher in SS compared with FMF (100% [3/3] vs 57.1% [4/7]). Organ-specific responses were observed in skin (70%), lymph nodes (40%), and blood (100% in SS patients). Median TTR was 6.6 weeks, median DoR was 15.6 weeks, and median PFS was 20.6 weeks. Patients with low skin CD30 expression (≤5%) demonstrated higher ORR than those with higher expression (100% vs 40%，P=0.058) and significantly longer PFS (38.6 vs 10.1 weeks; HR=9.97, P=0.013). Pruritus improved markedly, with median NRS decreasing from 8 to 0.5 (P=0.006).A total of 15 treatment-emergent adverse events occurred in 8 patients. The most common were cytomegalovirus infection (n=4, including 1 grade ≥3 event), peripheral neuropathy (n=3), leukopenia, anemia, thrombocytopenia, and hypertriglyceridemia. No treatment-related deaths occurred.

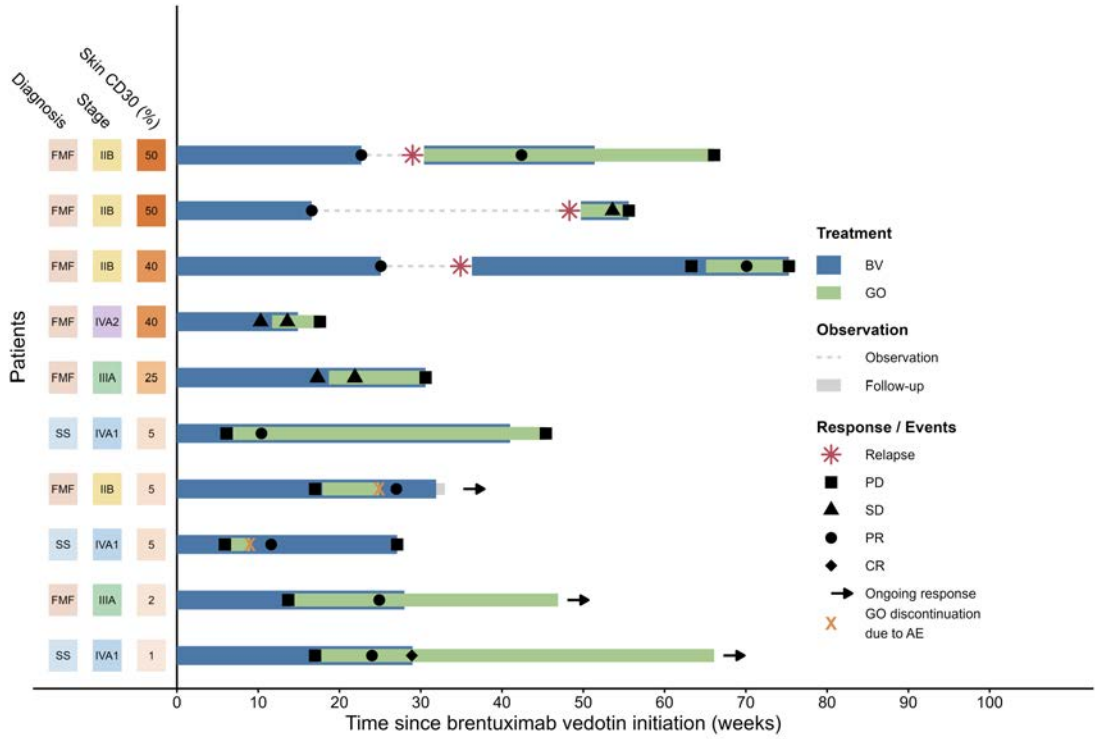

Fig 1. Responses to golidocitinib plus brentuximab vedotin and response duration since brentuximab vedotin initiation. FMF, folliculotropic mycosis fungoides; SS, Sézary syndrome; BV, brentuximab vedotin; Go, golidocitinib; PD, progressive disease; SD, stable disease; PR, partial response; CR, complete response; AE, adverse event.

Conclusion

The combination of golidocitinib and BV demonstrated encouraging clinical activity with manageable toxicity in patients with BV-refractory/relapsed advanced FMF and SS, achieving a high ORR and pruritus improvement. Responses appeared more pronounced in patients with low CD30 expression, suggesting a potential role of JAK1 inhibition in overcoming BV resistance. These findings support further prospective studies to validate Go-BV as a therapeutic strategy in advanced CTCL.

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Abstract N°: 156

Novel Immuno-Keratolytic Therapy For Recalcitrant Verruca Plana Using BCG, Needling, And Tretinoin

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Introduction
Verruca plana, caused primarily by human papillomavirus (HPV) types 3 and 10, remains a therapeutic challenge owing to its chronicity, cosmetic implications, and resistance to conventional modalities such as cryotherapy, cauterization, and keratolytics. Recurrence and post-inflammatory dyschromia are frequent, particularly in darker skin types. With growing evidence supporting immune-based interventions, this study introduces a novel triple synergistic regimen combining topical Bacillus Calmette–Guérin (BCG), Falkner's needling, and topical tretinoin to achieve rapid, scar-free clearance in recalcitrant verruca plana.

Materials and Methods
Objectives
To evaluate the clinical efficacy, immune-modulating response, and tolerability of a combined protocol involving topical BCG, Falkner's needling, and topical tretinoin in patients with resistant verruca plana.

Methods
Fifteen patients (aged 17–42 years) with biopsy-confirmed verruca plana unresponsive to standard therapies (cryotherapy, salicylic acid, or imiquimod) were included. Under aseptic precautions, Falkner's needling was performed to fragment lesions and facilitate intradermal exposure of viral antigens. Immediately post-needling, reconstituted topical BCG (derived from an intradermal vial) was applied over lesions and occluded for six hours. From the following day, patients initiated nightly topical tretinoin 0.05% for six to eight weeks to promote epidermal turnover and enhance antigen presentation. The procedure was repeated every three weeks for up to three cycles. Clinical response, dermoscopic evolution, and side effects were recorded at each visit, with follow-up for three months post-treatment to assess recurrence.

Results
Thirteen of fifteen patients (86.6%) achieved complete clearance within 8–10 weeks, while the remaining two demonstrated >75% improvement. Lesions showed progressive flattening, surface normalization, and pigment restoration. Notably, distant untreated lesions regressed concurrently, signifying systemic immune activation. Adverse effects were limited to transient erythema, mild scaling, and burning, resolving spontaneously without pigmentary sequelae. No recurrence or scarring was observed during follow-up. The therapeutic synergy of needling-induced antigen exposure, BCG-driven immune activation, and tretinoin-mediated keratolysis facilitated robust viral clearance and epidermal remodeling.

Conclusions
This triple synergistic regimen represents a groundbreaking immuno-keratolytic approach in the management of recalcitrant verruca plana. It combines simplicity, safety, and cost-effectiveness with exceptional clinical outcomes and minimal downtime. The protocol's ability to trigger both local and systemic antiviral immunity while preserving cosmetic integrity positions it as a promising alternative to conventional destructive modalities. Larger controlled studies are warranted to further substantiate its efficacy and define optimal treatment parameters.

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Abstract N°: 163

Development and Characterization of IBI3013, an IL15 Targeting Monoclonal Antibody with Half-life Extension

Jinyang Li*[1], Mengqiang Di[1], Chenjuan Zhu[1], Meng Ni[1], Bin Li[1], Huijun Sha[1], Hao Wang[1], Jinling Xu[1], Enhong Zhong[1], Yao Xiong[1], Li Li[1], Huizhong Xiong[1]

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Introduction

Vitiligo and alopecia areata both remain global unmet medical needs. No biologics have been approved or successfully demonstrated efficacy in late-stage clinical trials. Thus, it is urgent to explore new therapeutic targets and develop therapies targeting specific disease-driving mechanisms to achieve favorable efficacy, safety profile, and dosing frequency. Pre-clinical and clinical studies have demonstrated the potential important role of IL15 signaling in the modulation of auto-reactive, cytotoxic, tissue-resident T cells as well as the initiation and progression of vitiligo and alopecia areata. Indeed, therapies targeting IL15 signaling have been tested in clinical trials for these indications. Here, we developed IBI3013, a monoclonal antibody antagonizing IL15 signaling, aiming to address remaining unmet medical needs.

Materials and Methods

IL15 reporter assay, IL15 induced immune cell expansion assay, graft-versus-host disease (GvHD) mouse model, skin inflammation mouse model, pharmacokinetics (PK) analysis using human FcRn-knockin mice and monkey, and various developability analysis strategies have been used to characterize IBI3013.

Results

IBI3013 targets a unique epitope of IL15, enabling specific blockade of IL15 - Rγ interaction without interfering IL15 - IL2Rβ interaction. Evaluated by several in vitro assays including reporter assay, immune cell expansion assay, and IFNγ production assay, IBI3013 has been demonstrated to elicit potent and robust blockade of IL15 signaling induced by both IL15 and IL15/IL15Rα complex, with superior efficacy to all identified benchmark IL15 mAbs. Moreover, IBI3013 shows potent and durable efficacy in in vivo mouse models, such as IL15 stimulated GvHD and skin inflammation. Notably, a single dose of IBI3013 deeply can suppress skin inflammation for up to three weeks in vivo. Importantly, IBI3013 exhibits favorable physical-chemical properties, developability, viscosity at high concentration, and immunogenicity features, de-risking further development. In both human FcRn-knockin mice and monkeys, IBI3013 exhibits significantly improved PK profile, achieved by a validated Fc engineering, YTE (M252Y/S254T/T256E), potentially allowing low dosing frequency in human.

Conclusions

IBI3013 is an IL15 targeting monoclonal antibody with half-life extension achieved by clinically validated Fc-engineering strategy. IBI3013 targets a unique epitope of IL15 cytokine which enables the potent and robust blockade of IL15 signaling, a potential key and shared disease-driving mechanism. IBI3013 exhibits favorable drug developability and PK features, and thus demonstrates potential for subcutaneous administration at low frequency. We aim to develop IBI3013 to explore the clinical efficacy of IL15 blockade and delivery potential benefits to patients with vitiligo, alopecia areata, and other T cell driving autoimmune diseases.

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Abstract N°: 253

Late granulomatous reaction to dermal filler triggered by PD-1 blockade

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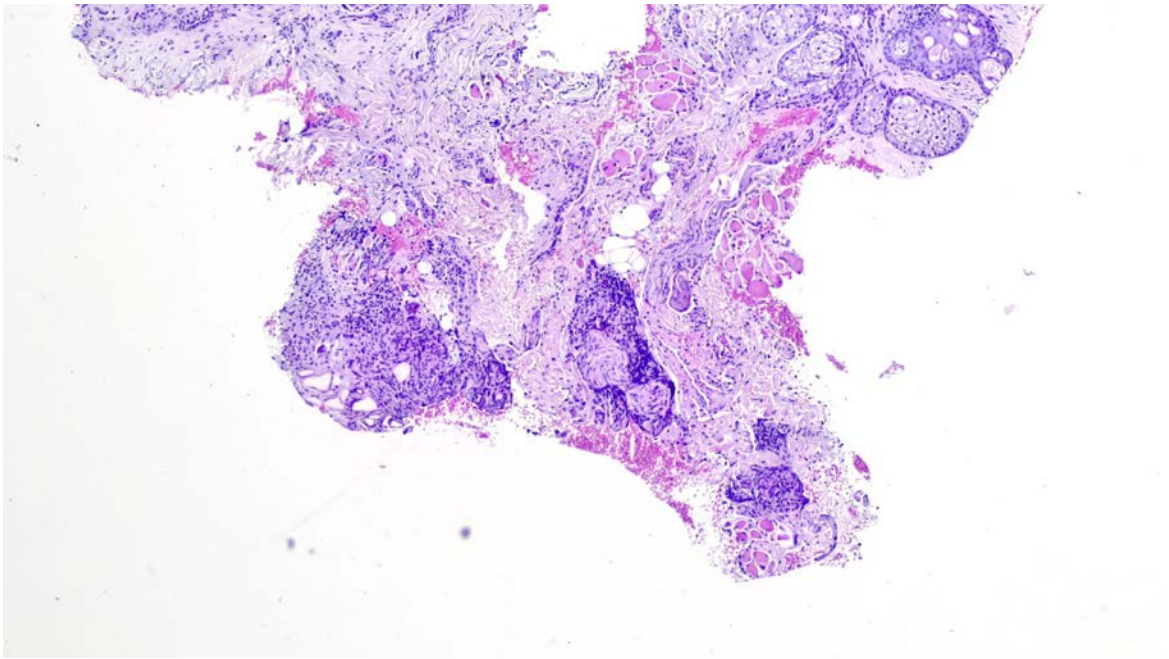
Introduction
Immunotherapy targeting the PD-1 pathway has transformed the management of advanced melanoma but is associated with diverse immune-related adverse effects. Late inflammatory responses involving previously injected dermal fillers represent a rare manifestation and may be mistaken for disease progression.

Case Presentation
A 79-year-old woman (ECOG 0) with metastatic melanoma (BRAF wild-type) initiated pembrolizumab in December 2025 following staging that revealed hypermetabolic right axillary lymphadenopathy and abdominopelvic nodules, which were subsequently confirmed as nodal metastases. Her medical history included hypertension, type 2 diabetes mellitus, dyslipidaemia, and Wolff–Parkinson–White syndrome. Two weeks after starting pembrolizumab, she developed firm perioral and labial subcutaneous nodules with associated oedema. When questioned, the patient acknowledged having received a filler injection 30 years prior. Histopathological examination revealed a non-caseating granulomatous reaction with multinucleated giant cells surrounding foreign material consistent with a methacrylate-based filler (hydroxyethyl methacrylate and ethyl methacrylate). These findings supported the diagnosis of a foreign-body granulomatous reaction triggered by pembrolizumab.In addition to the localized filler reaction, our patient later developed systemic manifestations including arthralgias, dry cough, asthenia, and periarticular subcutaneous plaques, accompanied by elevated serum angiotensin-converting enzyme levels and radiographic pulmonary changes, raising concern for a sarcoidosis-like syndrome. She was then treated with low-dose oral prednisolone (5 mg daily), resulting in marked clinical improvement. Importantly, immunotherapy was continued without flaring of the clinical picture.



Histopathological examination showing non-caseating granulomas with multinucleated giant cells

surrounding foreign material consistent with methacrylate-based filler (H&E stain).

Conclusion

Granulomatous and sarcoidosis-like reactions are recognised immune-related adverse events of PD-1 inhibition, typically occurring weeks to months after treatment initiation.Permanent dermal fillers may act as immunological targets, leading to delayed inflammatory responses even decades after implantation. Only four similar cases have been reported in the literature, following CTLA-4 or PD-1 inhibition in melanoma or lung cancer, suggesting that immune checkpoint blockade may reactivate a previously subclinical chronic foreign-body inflammatory response to long-standing dermal filler. Management strategies in reported cases have varied, ranging from surgical excision to expectant management. Unlike prior reports, our patient also developed systemic manifestations suggestive of a sarcoidosis-like response, currently under investigation. Sarcoidosis-like reactions may be managed conservatively but sometimes require hydroxychloroquine or systemic corticosteroids. This case highlights several clinically relevant issues. First, inflammatory reactions at prior filler sites may clinically and radiologically mimic tumour progression, posing a diagnostic challenge in oncologic patients receiving immunotherapy. Second, permanent fillers carry a lifelong risk of delayed immune-mediated complications. Third, the occurrence of this clinical presentation is likely to increase, as both filler injections and immunotherapy have expanded their use and knowledge of this granulomatous reaction pattern may prevent unnecessary treatment discontinuation. Careful clinical history, histopathological confirmation, and multidisciplinary assessment are essential for accurate diagnosis and management.

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Abstract N°: 271

Subcellular antigen targeting to the endoplasmic reticulum as a rational design strategy for dermatologic DNA immunotherapy

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Introduction
Optimizing intracellular antigen trafficking represents a potential strategy to enhance major histocompatibility complex class I loading in DNA-based immunotherapy. In dermatology, rational vector engineering may improve immune activation against virus-associated disease while preserving physiologic costimulatory signaling.

Materials and Methods
Four plasmid constructs were generated encoding E7 alone, E7 with CD40 ligand, CD40 ligand alone, or empty vector. Endoplasmic reticulum targeting was achieved using signal peptide and retention sequences. Expression and subcellular localization were assessed in epithelial cell lines using immunoblotting and confocal microscopy. Endoplasmic reticulum stress marker expression was analyzed as an indirect indicator of intracellular processing. Functional immune activity was evaluated in macrophage co-cultures with or without lipopolysaccharide stimulation through interleukin-12 quantification and activation-associated morphological changes.

Results
Construct integrity and protein expression were confirmed. Endoplasmic reticulum-targeted E7 increased expression of stress-associated markers compared to controls. CD40 ligand maintained membrane localization despite bicistronic expression. Functionally, co-stimulatory conditions resulted in increased interleukin-12 secretion, with enhanced activation profiles compared to controls.

Conclusions
Subcellular antigen targeting to the endoplasmic reticulum can coexist with preserved membrane costimulatory signaling within a single DNA platform. This design supports rational engineering of dermatologic immunotherapy vectors and provides a translational framework for virus-associated cutaneous disease.

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Abstract N°: 283

One Stone, Two Birds: Successful Treatment of Generalized Granuloma Annulare and Urticarial Vasculitis with Dupilumab

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Introduction

Granuloma annulare (GA) is a granulomatous inflammatory dermatosis that may present in localized or generalized forms. Localized GA often responds favorably to topical or intralesional corticosteroids, whereas generalized GA is typically refractory and difficult to manage. The Th1 axis has classically been implicated in GA pathogenesis; however, recent evidence suggests involvement of the Th2 pathway. Therapeutic management of refractory and generalized lesions remains challenging. We report a complex case of generalized GA with coexisting urticarial vasculitis successfully treated with dupilumab, highlighting its potential as a novel therapeutic option.

Case Presentation

A 65-year-old woman presented with a 7-year history of persistent non-blanchable wheals with central red macules lasting longer than 24 hours, involving the arms, abdomen, back, and legs. Her medical history included goiter and cardiac arrhythmia, treated with levothyroxine, bisoprolol, propafenone, and aspirin. An initial skin biopsy was consisted with urticarial vasculitis (UV). Laboratory investigations, including antinuclear antibodies (ANA) and complement levels (C3 and C4), were within normal limits. No systemic involvement or underlying malignancy was identified. She had received systemic corticosteroids, methotrexate, hydroxicholoroquin and azathioprine; each for at least 3 months at different time points. Despite these treatments, her lesions exhibited a waxing and waning course throughout the 6-year period. In 2023, she developed new lesions that differed from the initial presentation, consisting of erythematous, annular plaques with central atrophy predominantly affecting the extremities, especially the hands, forearms, and thighs, with a few lesions on the trunk. A subsequent biopsy obtained from annular plaques revealed granulomatous inflammation with degenerated collagen bundles, confirming granuloma annulare (GA). Given the presence of more than 10 plaques, the disease was classified as generalized GA based on clinicopathological correlation. Paraneoplastic causes were excluded through comprehensive laboratory and radiological evaluations. Multiple treatments, including intralesional and systemic corticosteroids, narrowband UVB phototherapy with acitretin, omalizumab, and cyclosporine, were ineffective. A new biopsy was taken in 2025 due to resisntance of lesions, demonstrated histopathological features of UV, confirming dual pathology of generalized GA and UV. We aimed to treat patietns both conditions UV with Dupilumab, which was initiated with a 600 mg loading dose followed by 300 mg biweekly. After 3 months, a marked regression of lesions was observed without adverse events.

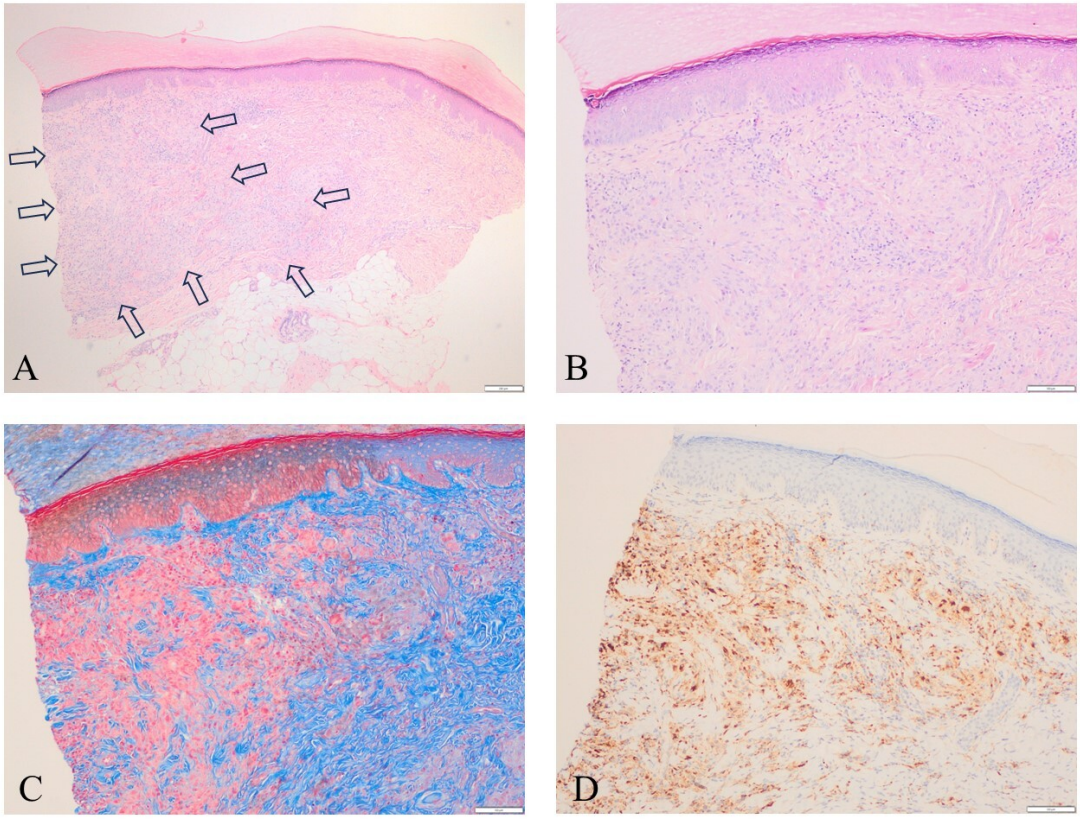

Figure 1: Histological sections of the skin tissue. Dermis showing a granulomatous reaction with collagen degeneration (arrows) (H&E, ×40). (A); Higher magnification of panel A, demonstrating collagen fibers and histiocytes with abundant eosinophilic cytoplasm (H&E, ×100) (B); Collagen degeneration highlighted by eosinophilic staining (Masson's trichrome, ×100). (C); CD68 positivity in histiocytes forming the granulomatous reaction (immunohistochemistry, ×100). (D)

Conclusion

Dupilumab demonstrated efficacy and safety in the management of generalized GA and urticarial vasculitis. To our knowledge, this is the first reported case of dupilumab successfully used in both conditions.

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Abstract N°: 287

Comparison of efficacy and safety of baricitinib versus methotrexate therapy in the treatment of severe alopecia areata

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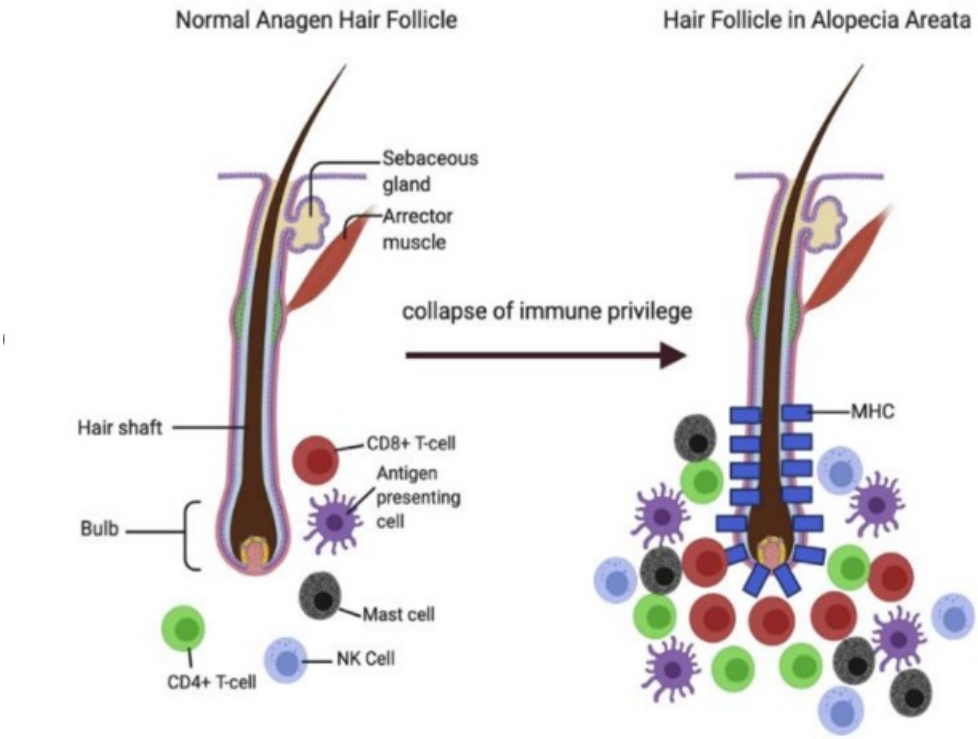
Introduction

Alopecia areata is a chronic autoimmune disorder characterized by non-scarring hair loss, typically presenting as well-demarcated patches. Its pathogenesis involves a complex interplay of genetic predisposition, environmental triggers, and immune dysregulation, with cytotoxic CD8+ NKG2D+ T cells playing a central role through cytokine-mediated follicular damage, particularly via interferon-γ and interleukin-15 pathways. Despite multiple therapeutic options, management of moderate-to-severe or treatment-resistant cases remains challenging, with conventional agents such as methotrexate often limited by variable efficacy and adverse effects. Recently, Janus kinase (JAK) inhibitors have emerged as a targeted therapeutic approach by interrupting key inflammatory signalling pathways. Baricitinib, a selective JAK1/JAK2 inhibitor, has demonstrated promising efficacy and was approved by the FDA in 2022 for severe alopecia areata. However, comparative data between baricitinib and conventional systemic therapies like methotrexate remain limited. This study aims to evaluate and compare the efficacy and safety profiles of these two agents in patients with alopecia areata.



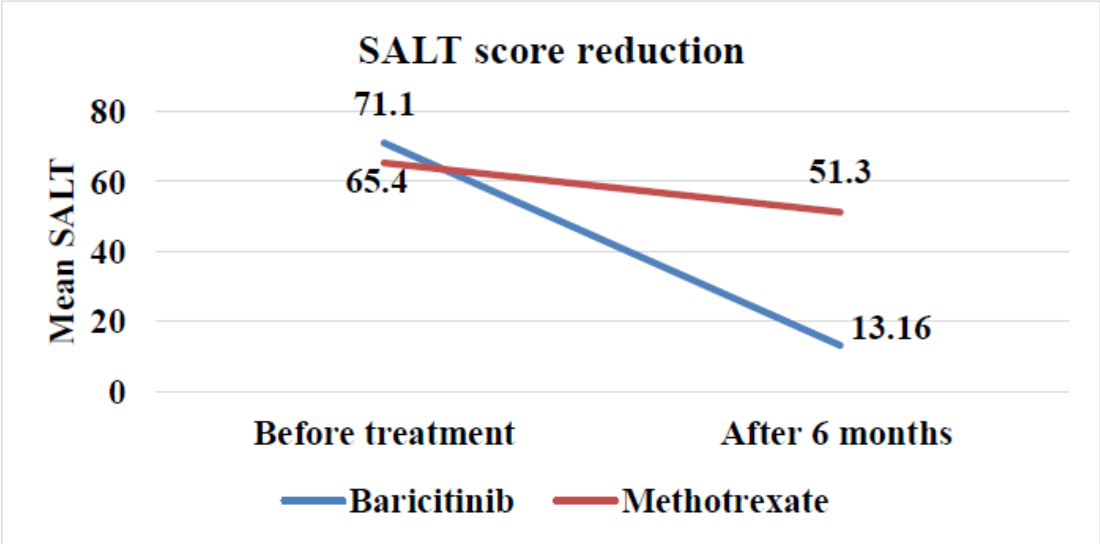
Pathogenesis of Alopecia areata

Materials and Methods

This randomized controlled trial was conducted in the department of Dermatology and Venereology of Bangladesh Medical University from the period of January 2023 till September 2024. After evaluating eligibility criteria, 84 patients of alopecia areata with more than 50% scalp involvement, patients with alopecia totalis and alopecia universalis were divided into two groups. Patients of group A (42) were treated with oral baricitinib 4mg once daily and group B (42) were treated with oral methotrexate 15mg once weekly with oral folic acid 5mg the following day for 6 months. Patients were assessed at baseline, 1st month, 3rd month and 6th month using Severity of Alopecia Tool (SALT) score and investigations to monitor adverse effects. After stopping treatment, patients were followed up for further 1 month to assess relapse of the condition

Results

The most common type of alopecia areata was patchy alopecia areata in both of the groups. Before treatment the SALT score was 71.1±19.62 in group A and 65.4±18.6 in group B. After 1 month, 3 month and 6 month the SALT score was 55.3±19.6, 37.9±23.3 and13.2±22.6 respectively in group A, whereas it was 64.6±19.3, 58.8±24.9 and 51.3±31.1 respectively in group B, which was statistically significant ($p<0.001$). Regarding percent change in SALT score, at month 6, SALT75 i.e excellent response was seen in 19% patients of group A compared to 9.5% in group B; and SALT90 i.e. complete response was achieved in 57.1% patients in group A compared to none in group B. Regarding safety profile, the most frequent adverse effects observed in baricitinib group were upper respiratory tract infection (14.3%), hyperlipidemia (11.9%) and acne vulgaris (7.1%) and in methotrexate group were, nausea (28.6%), fatigue (11.9%), elevated liver transaminase (11.9 %) and respiratory tract infection (4.8%).



Absolute change of SALT score from baseline in two groups

Conclusions

In this study, after completion of 6 months of treatment, the SALT score was lower in baricitinib group compared to methotrexate group and the percent change in SALT score in baricitinib group was higher compared to percent change in SALT score in methotrexate group, which was statistically significant. Besides a significant number of patients achieved complete regrowth of hair with baricitinib. Considering all the facts, it can be concluded that, baricitinib is a more effective (with negligible adverse effects) than methotrexate in the treatment of severe alopecia areata.

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Abstract N°: 309

SPOTLIGHT-204: Primary Results of Intralesional BO-112 in Resectable Basal Cell Carcinoma

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Introduction

Basal cell carcinoma (BCC) is the most common human malignancy with raising global incidence. Mohs micrographic and conventional surgery achieve high cure rates, however, can result in significant scarring and functional compromise, particularly on the head and neck (H&N). Effective non-surgical options remain an unmet clinical need. BO-112 is a dsRNA immunotherapy designed to activate an innate and adaptive immune response and immunogenic tumour-cell death.

Materials and Methods

SPOTLIGHT-204 is a multi-centre, phase 2b, single-arm, two-cohort clinical trial evaluating efficacy, safety and tolerability of intralesional BO-112 injection in 50 patients (40 high-risk [HR] and 10 low-risk [LR], as defined by National Comprehensive Cancer Network [NCCN] criteria) with resectable, histologically confirmed BCC lesions.

Patients received three once-weekly intralesional injections followed by complete surgical excision at week 24. Visual and pathological responses were evaluated by a Central Review Committee. Visual efficacy was Grade: 1) complete (complete lesion disappearance or scar); 2) partial (≥30% reduction in lesion size); 3) no change / smaller than partial; or 4) lesion growth (≥20% increase in lesion size). Pathological response, at the time of surgical excision, was Grade: 1) complete (no residual viable tumour (RVT); 2) major (≤10% RVT); 3) partial (>10% ≤50% RVT); or 4) no response (>50% RVT). We report results in a 46-patient modified intention to treat population with 54 evaluable lesions.

Results

The median age was 75 years (range 34–90), 48% (n=22) were female. Lesions were located on the face (61%,

n=33); scalp (11%, n=6); extremities (15%, n=8); and trunk (13%, n=7). Lesion median longest diameter was 12 mm (range 6–34 mm). Mean BO-112 cumulative exposure was 1.25 mg. The composite primary endpoint was defined as both visual and pathological response of Grade 3 or better in all treated lesions at patient level. This was achieved by 61% (n=28) of patients, with high visual and histological concordance. Response rates were 58% (n=21) in HR patients; and 70% (n=7) in LR patients. Complete and major (Grades 1 and 2) pathological responses were observed in: 56% (n=20) of HR patients; and 50% (n=5) of LR patients. A complete visual and complete pathological response (both Grade 1), was achieved by 56% (n=18) of HR-H&N patients. Of 100 treatment related adverse events (TRAEs (at event level)): 91% (n=91) were Grade 1 (mild); 9% (n=9) were Grade 2 (moderate); none were ≥Grade 3 (severe); none were serious. The most frequent TRAEs were: pyrexia (12%, n=12); injection site pain (20%, n=20); injection site swelling (8%, n=8); and headache (6%, n=6). These events are consistent with BO-112s mechanism of action. No AEs were serious, led to dose reduction, or discontinuation.

Conclusion

Intralesional BO-112 achieved high rates of composite visual and pathological response in both LR and HR patients, with a manageable and predominantly low-grade safety profile. Of note, in HR patients with H&N lesions, high percentages of complete visual and pathological responses were achieved. These results support further development of BO-112 as a non-surgical option for treating BCC.

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Abstract N°: 332

Real-world effectiveness, safety and clinical impact of dose escalation of tildrakizumab in moderate-to-severe psoriasis

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Introduction

Tildrakizumab, a selective IL-23p19 inhibitor, has demonstrated efficacy and a favorable safety profile in randomized clinical trials and real-world practice for moderate-to-severe plaque psoriasis. However, evidence evaluating treatment optimization strategies in routine clinical settings, including dose escalation and predictors of therapeutic response in heterogeneous patient populations, remains limited. Objective: To evaluate the effectiveness, safety, and clinical impact of tildrakizumab dose escalation in a real-world cohort.

Materials and Methods

We retrospectively analyzed 146 patients treated with tildrakizumab (100 mg or 200 mg). Patients were stratified into three groups: fixed-dose 100 mg (n=83), fixed-dose 200 mg (n=26), and dose escalation from 100 mg to 200 mg due to insufficient response (n=37). Escalation occurred at a median of 9 months. Baseline characteristics, comorbidities, and prior systemic therapies were recorded. Effectiveness was assessed by changes in PASI, BSA, and DLQI from baseline to last follow-up. Responder rates, predictors of response, safety parameters, and treatment persistence were also evaluated. Subgroup analyses examined body weight, obesity, and prior therapies. Non-parametric tests were used.

Results

A total of 146 patients were included (mean age 49.0 ± 16.1 years; 38.4% female; baseline PASI 13.4 ± 7.4). Mean baseline body weight differed between treatment groups: 82.9 ± 20.5 kg in the 100 mg group, 114.6 ± 16.6 kg in the 200 mg group, and 107.1 ± 26.1 kg in the dose-escalation group. Significant improvements in disease severity were observed across all cohorts. In the 100 mg group, PASI decreased from 13.0 ± 7.2 at baseline to 1.66 ± 2.29 at the last visit ($p < 0.0001$), BSA from 18.4 ± 15.1 to 1.9 ± 3.5 ($p < 0.0001$), and DLQI from 11.9 ± 7.8 to 2.8 ± 4.4 ($p < 0.0001$), over a median follow-up of 14 months. Similar improvements were observed in the 200 mg cohort, where PASI decreased from 15.4 ± 8.5 to 3.9 ± 4.7 ($p < 0.0001$) over a median follow-up of 9 months. Dose escalation from 100 mg to 200 mg, performed at a median of 9 months, resulted in additional significant improvements in PASI (baseline: 12.7 ± 6.9; at escalation: 5.2 ± 3.4; last visit: 3.8 ± 3.8), BSA (baseline: 19.3 ± 12.8; at escalation: 7.2 ± 7.2; last visit: 4.8 ± 7.7), and DLQI (baseline: 12.8 ± 7.8; at escalation: 7.6 ± 6.6; last visit: 4.9 ± 5.7) (all $p < 0.01$). Furthermore, 42.3% of patients who initiated treatment with 200 mg achieved PASI ≤ 2, while a comparable proportion of patients in the dose-escalation group reached this outcome after switching to 200 mg. Treatment persistence remained high, particularly in the escalation cohort, supporting sustained effectiveness.

Regression analysis identified prior biologic exposure ($\beta = -22.5$, $p < 0.001$) and methotrexate use ($\beta = -19.5$, $p = 0.001$) as negative predictors of response, while higher baseline PASI predicted greater improvement ($\beta = +0.75$, $p = 0.05$). Baseline body weight was not associated with PASI or BSA response. For DLQI, higher weight was linked to smaller improvement in adjusted analysis ($\beta = 0.046$; $p = 0.038$), but not in subgroup analyses. Dose

escalation did not affect liver enzymes, inflammatory markers, hematological parameters, or body weight.

Conclusions

Tildrakizumab demonstrated sustained effectiveness and a favorable safety profile in a real-world cohort of patients with moderate-to-severe plaque psoriasis. Dose escalation from 100 mg to 200 mg provided additional clinical benefit in patients with suboptimal response, without compromising safety. Higher baseline disease severity was associated with greater relative improvement, whereas prior exposure to biologic therapies and methotrexate negatively impacted treatment response. These findings highlight the importance of individualized treatment approaches and support the use of dose escalation to enhance clinical outcomes in selected patients.

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Abstract N°: 336

A retrospective cohort study on psoriasis developing in adult patients with atopic dermatitis treated with dupilumab

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Introduction

Atopic dermatitis (AD) and psoriasis are chronic inflammatory skin diseases, driven predominantly by Th2 and Th1/Th17 immune pathways, respectively. Dupilumab, an IL-4/IL-13 receptor antagonist, significantly improves outcomes in moderate-to-severe AD. However, the development of psoriasis following dupilumab therapy has been reported, likely reflecting immune shifts toward Th1/Th17-mediated inflammation after IL-4/IL-13 blockade. Factors predisposing patients to psoriasis during dupilumab treatment remain poorly characterized. The objective of this study was to assess the association between the development of psoriasis in patients with AD treated with dupilumab and a family history of psoriasis.

Materials and Methods

We conducted a retrospective cohort study of adult (≥18 years) patients with AD who received at least one dupilumab dose between 2018-2025 at a specialized AD clinic in a tertiary medical center. Data was extracted from electronic health records, with prospective questionnaires to collect missing data. Exclusion criteria included active psoriasis in the decade prior to dupilumab initiation, baseline psoriasiform rash preventing definitive diagnosis, less than one follow-up visit post-dupilumab, or missing data on family history of psoriasis. The primary outcome was the development of psoriasis as assessed by a clinical examination of an experienced dermatologist. The primary endpoint was the risk of developing psoriasis associated with a first-degree family history of psoriasis. Additional analyses evaluated the association with other Th1/Th17-mediated diseases—psoriatic arthritis (PsA), hidradenitis suppurativa (HS), and inflammatory bowel disease (IBD). The study was conducted in accordance with the Declaration of Helsinki and received approval from the Institutional Review Board (IRB).

Results

A total of 158 patients met inclusion criteria, of whom 17 had first-degree relatives with psoriasis. Ten patients developed classic seborrheic dermatitis after dupilumab and were not classified as having psoriasis. Baseline characteristics were generally similar between exposed (family history of psoriasis) and unexposed groups: mean age 50±17 vs 46±18 years, female sex 41% vs 44%, and BMI 24.7±5.5 vs 26.4±4.8. Indicators of atopy were similar too, including elevated IgE levels (>150 IU/ml) (82% vs 69%) and personal or family history of atopy (65% and 70% vs 65% and 67%, respectively). Smoking prevalence was higher in the exposed group (40%,6/15) vs the unexposed group (27%,34/125). Psoriasis developed in 35.3% of the exposed group vs 2.8% in the unexposed group (Fisher's exact test, $p<0.001$), corresponding to an unadjusted RR of 12.4 (95% CI 3.9-39.9). In a multivariable Firth penalized logistic regression model including smoking (n=140), exposure remained strongly associated with the outcome (OR 17.2, 95% CI 4.2–70.3, $p<0.001$), while smoking was not. In additional analyses, personal and/or family history of Th1/Th17-mediated diseases (psoriasis, HS, IBD, PsA) were also associated with an increased risk of psoriasis following dupilumab therapy. Sensitivity analyses excluding patients with seborrheic dermatitis, or alternatively classifying them as psoriasis cases, yielded similar results, indicating that the findings are robust to potential misclassification.

Conclusions

The risk of developing psoriasis following dupilumab treatment for atopic dermatitis is significantly higher in patients with a first-degree family history of psoriasis, or a personal or familial history of Th1/Th17-mediated diseases. These findings suggest an underlying genetic predisposition contributes to this adverse event following IL-4/IL-13 blockade. Assessing personal and family history of psoriasis and other Th1/Th17-mediated diseases may inform treatment decisions prior to initiating dupilumab therapy.

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Abstract N°: 355

Efficacy and Safety of Immune Checkpoint Inhibitors for Advanced Malignant Melanoma: A Meta-Analysis on Monotherapy Vs Combination Therapy

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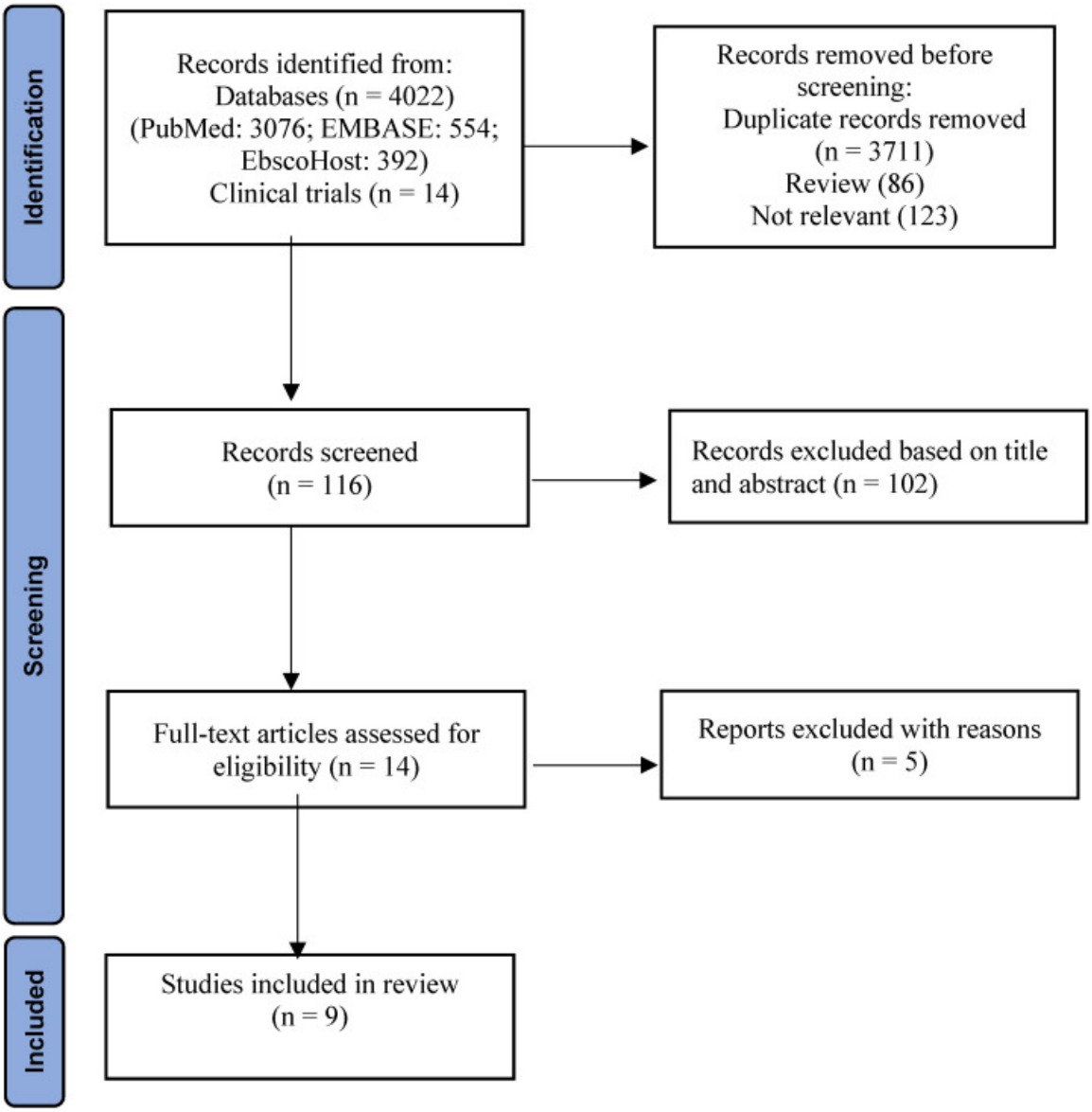
Introduction

Nivolumab and pembrolizumab of PD-1 inhibitors, and ipilimumab of CTLA-4 inhibitors are approved by FDA in 2014 as cancer immunotherapeutic agents and nivolumab and ipilimumab are the most widely used ICIs in the treatment of advanced malignant melanoma (MM), either alone or in combination. The systematic review (SR) on ICIs comparing monotherapy and combination therapy on advanced solid cancers which was published in 2021 included only three randomized control studies (RCTs) for advanced MM. The meta-analysis (MA) on efficacy and safety between monotherapy and combination ICIs in advanced MM was published in 2017. To fill this literature gap after the updated literature search, we aimed to perform this MA to update the efficacy and safety of ICIs comparing monotherapy and combination therapy; and to provide up-to-date comprehensive evidence for clinical decision making to choose different treatment options available in the treatment of advanced MM.

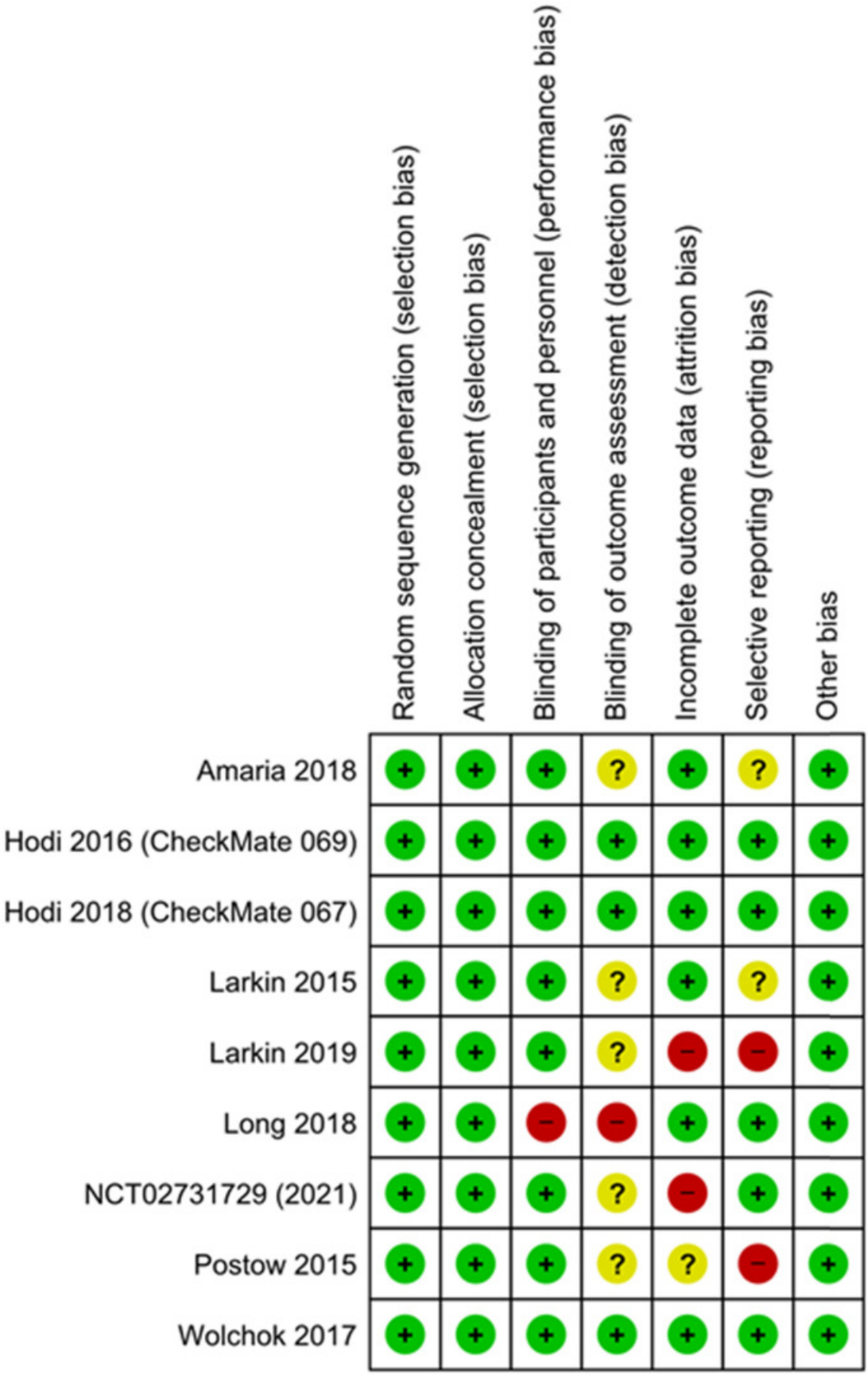
Materials and Methods

Studies in the selected articles were to meet five criteria of PICOS format: (1) Participants: individuals with histopathologically diagnosed advanced MM (Stage III and IV) of any age and sex; (2) Intervention: combined ICIs including nivolumab or pembrolizumab and ipilimumab simultaneously regardless of the dose; (3) Comparison: nivolumab or pembrolizumab or ipilimumab alone; (4) Outcomes: primary outcomes were overall survival (OS) which is defined as the time from randomization to death from any cause and treatment-related adverse effects (AEs). The secondary outcomes included progression free survival (PFS) which is defined as the time from randomization to cancer progression or death from any cause and objective response rate (ORR). (5) Studies: parallel RCTs reporting on the efficacy and or safety of ICIs; nivolumab, pembrolizumab and ipilimumab. Review articles, case reports, editorials, and studies that used other combinations were excluded for this MA.



Results

A total of nine RCTs were included in this MA. Regarding the efficacy, combined nivolumab and ipilimumab therapy showed statistically significant prolonged OS and PFS with HR 0.65, 95% CI [0.53, 0.79], p <0.0001 and HR 0.48, 95% CI [0.38, 0.60], p<0.0001 respectively. Combination therapy with nivolumab and ipilimumab also showed statistically significant longer ORR than monotherapy; with RR 2.15, 95% CI [1.63, 2.84], p <0.00001. In terms of safety, the incidence of all AEs which include any AEs, high-grade, haematological, gastrointestinal, dermatological, pulmonary, liver and endocrine AEs were significantly lower with monotherapy (either nivolumab or ipilimumab) of ICI compared to combination ICI therapy with a p-value <0.00001 to 0.03.

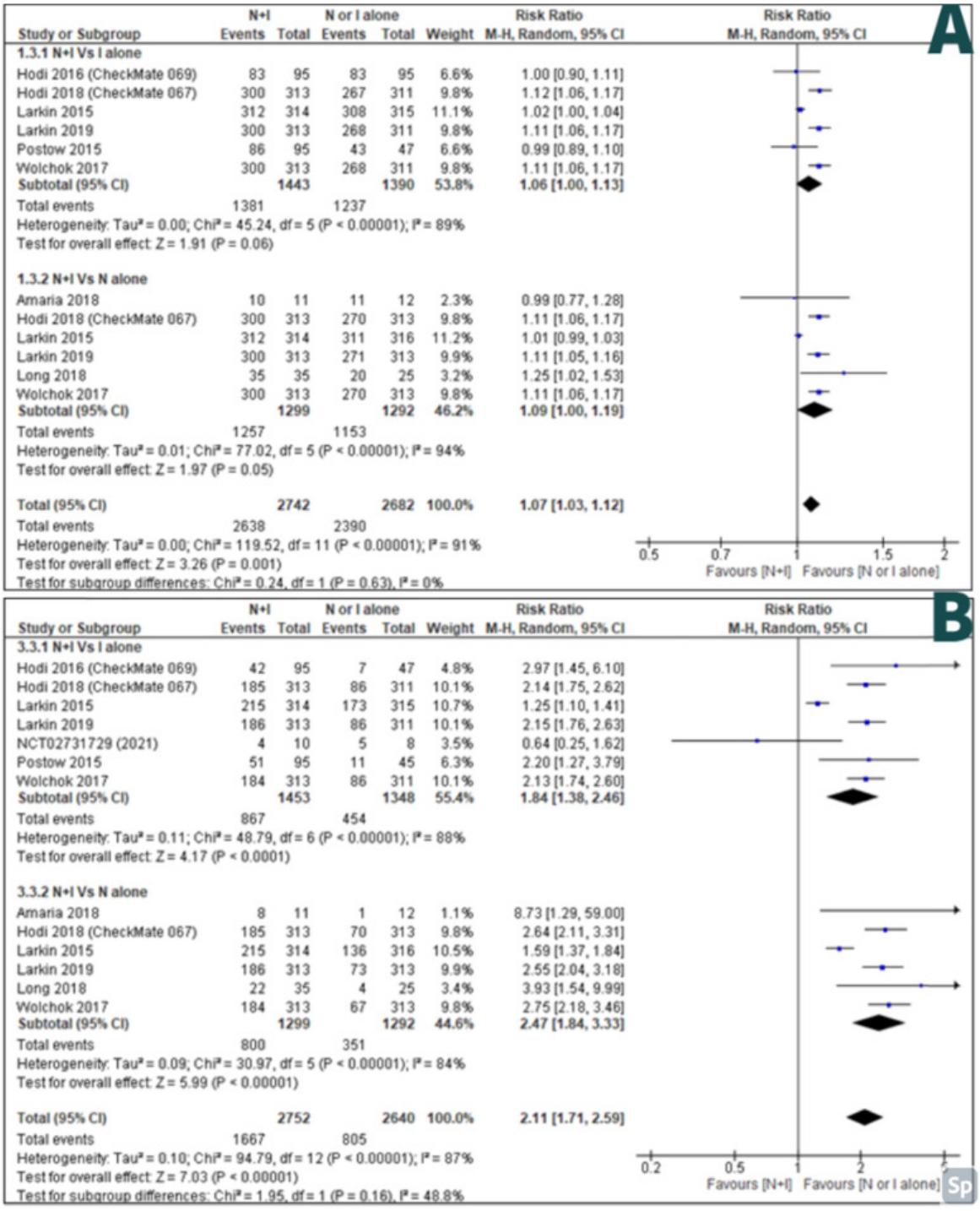


Conclusions

In conclusion, this MA showed that the efficacy of the combined ICIs was more favourable than single ICI in terms of OS, PFS and ORR. There was a significant difference in the combination Vs monotherapy in terms of OS, PFS and ORR. Although combination ICIs therapy showed better efficacy than monotherapy, monotherapy (either nivolumab or ipilimumab) was safer than combination therapy as it tended to decrease the incidence of most of the treatment-related AEs. Taking the patient's safety as our main priority, the most appropriate clinical practice

would be prescribing monotherapy over combination therapy in the treatment of advanced MM. Starting with monotherapy using a single ICI followed by adding another ICI can be considered to minimize the AEs and improve efficacy in the treatment of advanced MM.

Efficacy of the combined nivolumab and ipilimumab was better than a single ICI, especially in the treatment of advanced MM. Although combination therapy showed better efficacy than monotherapy, monotherapy (either nivolumab or ipilimumab) was safer than combination therapy as it tended to decrease the incidence of most of the treatment-related AEs.



EA DV CONGRESS
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Abstract N°: 375

Bempikibart, a novel IL-7Rα antibody, produces hair regrowth that persists off treatment in severe and very severe alopecia areata: efficacy and biomarker results from the Phase 2a SIGNAL-AA Part B trial

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Introduction
Alopecia areata (AA) is a T-cell-mediated disease of hair loss. Approved oral JAK inhibitors require continuous administration, with relapse commonly observed after discontinuation. Bempikibart is an antibody against IL-7Rα that potently blocks IL-7 and TSLP signaling, targeting the pathogenic T- cells and type 2 inflammation. SIGNAL-AA Part B evaluated a pharmacokinetically optimized regimen of bempikibart to further characterize safety, efficacy and remittive potential among patients with severe AA.

Materials and Methods
In this ongoing open-label Phase 2a study, 33 patients with severe (SALT score 50-94) or very severe (SALT score 95-100) AA and current-episode duration ≤4 years received bempikibart 200 mg subcutaneously extended through 36 weeks (4 weekly loading doses followed by Q2W doses), followed by 16 weeks off treatment. Baseline mean SALT score was 75.6 (24% SALT score ≥ 95) with a mean current episode duration of 2.07 years. 73% of patients were female; 36% were oral JAKi-experienced. At a 2 September 2026 cutoff, Week 36 analysis datasets were complete, and 10 patients had completed Week 52; missing SALT score values were imputed by LOCF. The pre-specified mITT population (n=25) excluded 8 patients per pre-specified SAP rules. Off-treatment durability was assessed in 12 mITT responder patients with a ≥10 point SALT score improvement by Week 36 who entered follow-up.

Results
Pharmacokinetics: The loading regimen produced a 3-fold higher mean trough of 28.8 µg/mL at Day 22 and reached steady state ~10 weeks earlier than dosing without loading (Week 3 vs Weeks 12-14).

Pharmacodynamics: Peripheral CD3+ T cells showed a median 44% reduction from baseline (n=21), with on-mechanism reductions in four T-effector cytokines assessed at baseline and Weeks 24 and 36 (biomarker-evaluable, n=25). Patients in the upper baseline tier (n=10) showed median reductions up to 46% for IFNγ, 46% for IL-17, 41% for IL-6 and 28% for TNFα. CXCL10, a downstream marker of IFNγ signaling, decreased by a median 32% at Week 36 in responders (biomarker evaluable, ≥$SALT_{30}$, n=8) versus 3% in non-responders (n=12). Th2 biomarkers downstream of TSLP signaling, TARC, IgE (baseline >200 IU/mL) and eosinophils, showed median reductions up to 39%, 29% and 52%. Twelve biopsies were immunostained and post-treatment biopsies showed reduced CD3+/CD4+/CD8+ infiltrates around hair follicles with increased Treg/CD8+ ratios concordant with increased hair growth.

Efficacy: At Week 36, SALT≤20 (≥80% scalp hair coverage) was achieved by 40.0% (10/25) of mITT and 30.3% (10/33) of ITT patients; SALT≤10 by 8.0% and 6.1%; ≥$SALT_{30}$ by 44.0% and 33.3%; ≥ $SALT_{50}$ by 44.0% and 33.3%.

Mean SALT score improvement from baseline was 35.3% (mITT). SALT≤20 responses occurred in severe and very severe disease and in IgE-high and IgE-low patients.

Off-Treatment Durability: Twelve of 25 mITT patients with at least a 10-point SALT score improvement by Week 36 entered follow-up; all 12 (100%) maintained response and 6 (50%) deepened at 8 weeks post (Table 1), one reaching SALT 0 and two additional patients SALT≤10 during off treatment period. Maintenance among Week 36 responders who entered off-treatment period was 10/10 (100%) for ≥$SALT_{30}$. Additionally, six non-responders entered follow-up and one converted to ≥$SALT_{30}$ off treatment.

Safety: All drug-related adverse events were mild or moderate. Injection-site reactions occurred with 4% of administrations, all mild. Asymptomatic reduction in absolute lymphocyte count flattened at Weeks 24-28 with no relationship to infection events.

Table 1. Off-Treatment Durability in Week 36 Responders Who Entered Follow-Up Period (n=12 of 25 mITT)

	Final on-treatment visit (Week 36)	Through 8 weeks post final dose (LOCF, n=12)	Through 16 weeks post final dose (Completers, n=7)
n	12	12	7
Mean Absolute SALT Score	22.5	22.9	23.9
Maintained Response[1], n (%)	—	12 (100%)	7 (100%)
Deepening of Response[2], n (%)	—	6 (50%)	3 (43%)

[1]Maintained Response defined as <20-point SALT score worsening as defined in King et al. JAMA Dermatol. 2024. Deepening of Response defined as any improvement in SALT score. Note one patient who was a non-responder converted to a $SALT_{30}$ by 8 weeks post final dose. LOCF, last observation carried forward. Completers—results only from the specified visit.

Conclusion

An optimized bempikibart regimen led to hair regrowth in severe and very severe AA, including JAK-experienced patients, with concordant biomarker and histologic evidence of dual IL-7/TSLP blockade and a favorable safety profile. Regrowth was maintained in all patients and deepened among a subset after the final dose. This combination of efficacy, safety and durability supports advancement of bempikibart to registrational studies.

EADV CONGRESS
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Abstract N°: 385

Dupilumab-induced paradoxical psoriasiform and nummular eruptions in atopic dermatitis: two case reports

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Introduction
Targeted biologics such as dupilumab have significantly improved the management of moderate-to-severe atopic dermatitis. However, increasing evidence suggests that IL-4/IL-13 blockade may induce paradoxical cutaneous reactions, including psoriasiform and less commonly nummular dermatitis–like manifestations, likely driven by immune pathway shifts. These reactions remain diagnostically challenging and may mimic disease exacerbation or de novo psoriasis.

Case Presentation
We report two patients with longstanding atopic dermatitis who developed distinct paradoxical eruptions after initiation of dupilumab therapy. The first patient presented with well-demarcated erythematous scaly plaques consistent with a psoriasiform pattern. The second patient developed multiple coin-shaped inflammatory plaques with nummular morphology. Neither patient had a prior history of psoriasis. Dermoscopic and histopathological findings supported a paradoxical inflammatory phenotype. Due to the severity and persistence of lesions, dupilumab was discontinued in both cases, followed by therapeutic modification and gradual clinical improvement.

Conclusion
These cases demonstrate that dupilumab-associated paradoxical reactions may present with heterogeneous morphologies, including psoriasiform and nummular patterns. This phenomenon highlights the complex interplay between immune pathways, whereby Th2 inhibition may promote a relative Th17 polarization. Awareness of biologic-induced phenotype switching is essential in dermatological practice as the use of targeted therapies expands. Early recognition and appropriate therapeutic adjustment are crucial for optimal patient management.

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Abstract N°: 437

Sonidegib for the treatment of multiple and locally advanced basal cell carcinomas in a case of severe Gorlin syndrome: a 3-year follow-up

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Introduction

Gorlin syndrome (also known basal cell nevus syndrome) is an autosomal dominant genodermatosis characterized by the development of multiple basal cell carcinomas (BCC) from a young age, and a heterogenic phenotype of other syndrome-defining features. It is caused by inactivating mutations in the hedgehog-related patched 1 (PTCH1) tumor suppressor gene and, less frequently, in the SUFU, PTCH2, SMO and GLI genes. De novo mutations may be responsible for up to 20% of cases.Treatment of BCCs in patients with Gorlin syndrome can be extremely difficult due to the large number of tumors and the possible presence of locally advanced lesions. In recent years, the hedgehog pathway inhibitors (HPI) vismodegib and sonidegib have been positioned as a systemic treatment for these patients.

Case Presentation

A 66-year-old woman presented with a history of multiple BCCs since puberty, some of them treated with surgery, cryotherapy and electrocoagulation. Her brother and sister presented a similar history. At clinical examination, dozens of superficial, nodular and ulcerated BCCs of different sizes were observed on the scalp, face, trunk, and extremities. Some BCCs were classified as locally advanced and caused very discomfort to the patient, due to pain and bleeding. The patient also showed palmar pits and reported a history of multiple odontogenic keratocysts, ovarian fibroma and skeletal anomalies. A diagnosis of Gorlin syndrome was formulated and, considering the number and characteristics of BCCs, a treatment with sonidegib 200 mg/die was started. After 6 months of treatment, the clinical response was excellent, with complete disappearance of several BCCs and considerable size reduction of others. Adverse events were mild: grade 2 cramps and grade 1 alopecia and weight loss, along with mild creatinine kinase elevations. At month 6, treatment was discontinued for one month and then restarted with alternate-day dosing. One year later, an almost complete disappearance of BCCs was observed and the dosing regimen was well-tolerated. A single, large BCC persisted on the trunk and was surgically removed. Three years after baseline, the patient is still under treatment with good disease control.

Conclusion

Our case confirms the efficacy and safety of sonidegib in a patient affected by severe Gorlin syndrome. These subjects often require numerous surgical procedures for treating BCCs throughout their lives, leading to physical and psychological exhaustion. An individualized long-term management and a multimodal approach integrating medical and procedural treatments is recommended. In this view, sonidegib represents a convenient therapeutic alternative, with good tolerability profile and dosing flexibility.

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Abstract N°: 450

Severe Neuro-Ophthalmic Varicella-Zoster Virus Infection During Upadacitinib-Associated JAK-1 Inhibition

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Introduction

Upadacitinib, a selective Janus kinase-1 (JAK-1) inhibitor licensed for rheumatoid arthritis, atopic dermatitis and psoriatic arthritis, exerts immunomodulatory effects through inhibition of interferon-mediated antiviral signalling pathways1. Across the JAK inhibitor class, herpes zoster incidence rates approximate 3.2 events per 100 patient-years, exceeding background population rates1,2. Current guidance recommends recombinant zoster vaccination prior to initiating immunosuppressive therapy3. Nevertheless, severe varicella-zoster virus (VZV) reactivation may manifest with ophthalmic or neuroinvasive complications4. We report severe neuro-ophthalmic VZV infection during upadacitinib therapy, underscoring the need for vaccination optimisation and early recognition of JAK-1 inhibition in herpes zoster.

Case Presentation

An 81-year-old woman with rheumatoid arthritis previously treated with methotrexate and hydroxychloroquine was escalated to upadacitinib (15 mg/day) in February 2024. Her history included prior herpes zoster and varicella-associated pneumonia.

She presented with a five-day history of right periorbital swelling, erythema and purulent discharge. Herpes zoster was diagnosed, and oral acyclovir initiated. She re-presented the following day with worsening neuropathic pain and a vesicular eruption in the right ophthalmic (V1) distribution. She was admitted and commenced on intravenous acyclovir. Ophthalmologic assessment confirmed herpes zoster ophthalmicus (HZO). She was discharged with topical chloramphenicol and ganciclovir.

Two days later, she developed acute confusion and worsening ocular pain. In the context of active HZO and altered mental status, viral encephalitis was suspected, and intravenous acyclovir recommenced. Cutaneous swabs yielded Staphylococcus aureus whilst cerebrospinal fluid polymerase-chain-reaction detected VZV DNA, confirming VZV encephalitis.

Dermatology review on day eight identified a well-demarcated erosive eruption to the right V1 distribution with yellow crusting; Hutchinson sign was negative. Medication reconciliation identified continued upadacitinib therapy throughout recurrent presentations despite progressive cutaneous and neurological disease. In view of the established association between JAK-1 inhibition and VZV reactivation, immediate cessation was advised.

A three-week course of intravenous acyclovir was completed with flucloxacillin administered for secondary bacterial superinfection. The eight-week inpatient course was complicated by persistent delirium and functional decline. Extensive V1 inflammation progressed to cicatricial fibrosis resulting in ectropion, lagophthalmos and exposure keratopathy requiring oculoplastic surgery. After eight weeks, cognitive function improved but the patient remained deconditioned. She was discharged to an intermediate care facility for rehabilitation, with ongoing oculoplastic follow-up.

Conclusion

The UK Health Security Agency guidance recommends the non-live recombinant zoster vaccine for adults aged ≥50 years and for individuals who will receive immunosuppressive therapy3. The vaccine is administered as 2 doses given 2 months apart (minimum interval 8 weeks) and should ideally be completed prior to initiation of immunomodulatory treatment or at least 14 days before starting therapy where treatment cannot be delayed3. Previous herpes zoster infection does not preclude vaccination once the acute episode has resolved. In this case, vaccination had been declined prior to escalation to JAK-1 inhibition, representing a potentially modifiable risk factor.

In essence, this case highlights the importance of structured vaccination review prior to initiating targeted immunomodulatory therapy. Early medication reconciliation and recognition of JAK-1 inhibition are also essential at first presentation with herpes zoster to enable timely risk stratification.

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Abstract N°: 454

Efficacy of Risankizumab in Prevention of Radiographic Progression in Patients With PsA at Low and High Risk of Joint Erosive Disease: 5-Year Data From the KEEPsAKE 1 Phase 3 Trial

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Introduction

Chronic joint inflammation in patients with PsA can lead to structural damage such as joint erosion (JE) and joint space narrowing (JSN), which are visible by radiography. This damage can have profound impacts on physical function, with high levels of damage being associated with greater disability and poor disease outcomes [1]. However, real-world estimates from a recently published study in newly diagnosed patients found that the radiographic baseline median modified Total Sharp Score (mTSS, which ranges on a scale of 0-528) was 0, indicating many patients with PsA do not present with radiographic lesions in routine clinical practice [2]. A recent 3-year observational study suggests that many patients with PsA have a slow rate of radiographic progression [2], highlighting the need for even longer-term evaluation. Risankizumab (RZB) is an approved IL-23 inhibitor for active PsA and has demonstrated over 5 years of consistent safety and sustained efficacy in treating the signs and symptoms of PsA. No statistical difference versus placebo in the mean change from baseline in mTSS at week 24 was previously observed with RZB [3]. The objective of this analysis was to evaluate long-term efficacy of RZB in inhibiting radiographic progression in high- and low-erosive risk populations of biologic-naïve patients with PsA over 5 years.

Materials and Methods

Data were derived from the KEEPsAKE 1 phase 3 trial (NCT03675308) which was placebo (PBO) controlled to 24 weeks. Trial eligibility criteria required patients to have either CRP≥0.3 mg/dL or JE≥1. In this post hoc analysis of biologic-naïve patients (N=679), we defined high- (CRP≥0.3 mg/dL and JE≥1, N=261) and low- (mTSS=0, [JE=0, JSN=0], N=329) structural damage risk groups per baseline characteristics. For patients initially randomized to PBO or RZB 150 mg, we report change from baseline in mTSS at weeks 24 (PBO and RZB) and 244 (continuous RZB only) as LS means derived from MMRM analysis. Nominal P values are reported for the difference between PBO and RZB at week 24. For each visit over approximately 5 years (244 weeks), we also report the proportion of patients in the RZB arm with no radiographic progression, defined as mTSS change from baseline ≤0, and the proportion of patients achieving change from baseline in JE score (JES) and JSN score ≤0, each reported as observed.

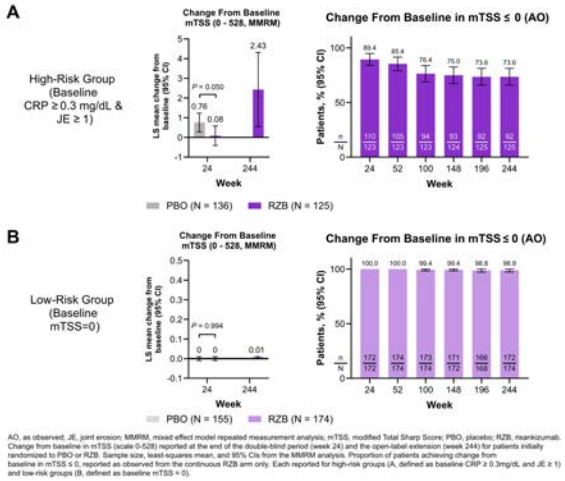
Results

For patients randomized to PBO and RZB, the mean baseline mTSS among the high-risk subgroup was 30.6 (N=136) and 29.8 (N=125), respectively, and for the low-risk group was 0 (N=155) and 0 (N=174), respectively. At week 24, patients in the high-risk group receiving RZB demonstrated lower progression versus the PBO group (change from baseline mTSS PBO: 0.76, RZB: 0.08, nominal P=.05, Figure 1A). Of the patients in the high-risk RZB subgroup, 73.6% displayed no radiographic progression through 5 years (Figure 1A), with the majority displaying no change from baseline in JES (76%) or JSN (80%) by week 244 (Figure 2A, B). The mean change from baseline in mTSS in the low-risk group receiving RZB and PBO remained minimal by weeks 24 (PBO: 0, RZB: 0) and 244 for the RZB group (0.01, Figure 1B). Of these low-risk patients, 98.9% displayed no radiographic progression by 244 weeks (Figure 1B), with over 99% showing no change in either JES nor JSN scores by week 244 (Figure 2A, B).

Conclusions

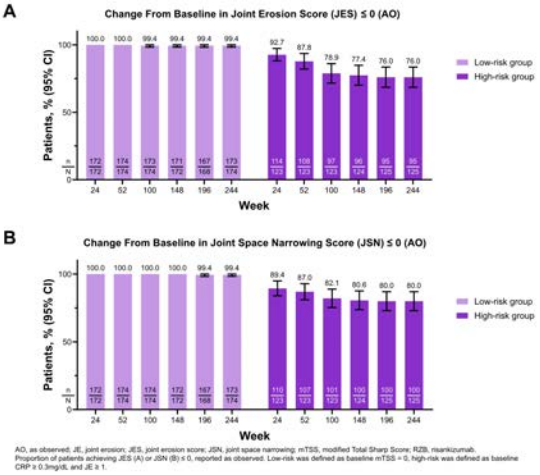
In this post hoc analysis, treatment with RZB is associated with a reduction in radiographic damage at week 24 as compared to PBO in patients with PsA at high risk of joint damage, and most patients in both high- and low-risk groups showed no radiographic progression over 5 years. In addition to previously published data demonstrating sustained efficacy in the peripheral arthritic signs of PsA, these data further suggest that patients receiving RZB show low rates of radiographic progression across a spectrum of erosive disease risk, and that most patients with PsA do not generally progress radiographically.

Figure 1: Change From Baseline in mTSS and Proportion of Patients Achieving Change From Baseline mTSS ≤ 0 in High and Low Erosive Risk Groups



AO, as observed; JE, joint erosion; MMRM, mixed effect model repeated measurement analysis; mTSS, modified Total Sharp Score; PBO, placebo; RZB, risankizumab. Change from baseline in mTSS (scale 0-528) reported at the end of the double-blind period (week 24) and the open-label extension (week 244) for patients initially randomized to PBO or RZB. Sample size, least-squares mean, and 95% CIs from the MMRM analysis. Proportion of patients achieving change from baseline in mTSS ≤ 0, reported as observed from the continuous RZB arm only. Each reported for high-risk groups (A, defined as baseline CRP ≥ 0.3mg/dL and JE ≥ 1) and low-risk groups (B, defined as baseline mTSS = 0).

Figure 2: Proportion of Patients Treated With RZB in High and Low Erosive Risk Groups Achieving Change From Baseline in JES and JSN ≤ 0



AO, as observed; JE, joint erosion; JES, joint erosion score; JSN, joint space narrowing; mTSS, modified Total Sharp Score; RZB, risankizumab. Proportion of patients achieving JES (A) or JSN (B) ≤ 0, reported as observed. Low-risk was defined as baseline mTSS = 0; high-risk was defined as baseline CRP ≥ 0.3mg/dL and JE ≥ 1.

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Abstract N°: 532

Rapid improvement in signs and symptoms with topical delgocitinib cream 20 mg/g in mixed atopic and irritant chronic hand eczema: a case report

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Introduction

Chronic hand eczema (CHE) is a debilitating inflammatory skin disease often associated with pain, pruritus, fissuring, and functional impairment[1-3]. CHE has different etiological and clinical forms[2]. Mixed forms frequently exist, with atopic hand eczema and irritant contact dermatitis coexisting in approximately one quarter of cases[4]. These subtypes involve both Th1 and Th2 immune pathways[5]. Delgocitinib is a topical pan-Janus kinase (JAK) inhibitor that targets multiple inflammatory pathways involved in eczema pathogenesis[6]

Case Presentation

Objective:

To highlight the rapid onset of clinical improvement within the first 6 days of treatment with delgocitinib cream 20 mg/g in a patient with severe, treatment-resistant chronic hand eczema.

Case description:

A 35-year-old female patient presented with a mixed form of chronic hand eczema combining atopic dermatitis and irritant contact dermatitis, complicated by secondary bacterial infection. Clinical examination revealed erythema and fissuring involving both palms and wrists, with a baseline Hand Eczema Severity Index (HECSI) score of 64 and a Physician Global Assessment (PGA) score of 4, indicating severe disease. Histopathological findings were consistent with atopic dermatitis, and patch testing was negative. The condition had a marked impact on quality of life and daily functioning, with a Dermatology Life Quality Index (DLQI) score of 14. At baseline, work productivity and activity were substantially impaired, as assessed using the Work Productivity and Activity Impairment (WPAI) questionnaire, with overall work productivity loss of approximately 60%, driven by both absenteeism and reduced job performance, as well as activity impairment of approximately 50%. Previous treatments, including potent topical corticosteroids (4 weeks), systemic corticosteroids (1 week), a topical phosphodiesterase-4 inhibitor (4 weeks), and oral antihistamines (2 weeks), failed to achieve adequate disease control. Treatment with delgocitinib cream 20 mg/g was initiated twice daily.

Results:

A rapid improvement in signs and symptoms was observed within two days of treatment initiation. By day two, erythema and fissuring were markedly reduced, accompanied by substantial relief of pruritus and pain. Continued improvement was noted at days four and six, with progressive normalization of skin appearance and recovery of hand function. Severity assessment scores demonstrated consistent improvement from baseline through day six

Tool	Baseline	Day 2	Day 4	Day 6
HECSI	64 (Severe)	31 (Moderate)	18 (Moderate)	8 (Mild)
PGA	4 (Severe)	3 (Moderate)	2 (Mild)	1 (Almost clear)
NRS Itch	9	5	2	1
NRS Pain	7	4	2	1
DLQI	14	8	6	3

HECSI, Hand Eczema Severity Index; PGA, Physician Global Assessment; NRS, Numeric Rating Scale; DLQI, Dermatology Life Quality Index

Severity assessment tools for 6 days the initial treatment plan

Conclusion

Discussion:

This case illustrates the rapid onset of action of delgocitinib cream 20 mg/g in a mixed form of chronic hand eczema involving both atopic and irritant components. The early improvement observed within two days supports the involvement of JAK mediated signaling pathways in both Th1 and Th2 driven inflammation[5]. Rapid symptom control resulted in meaningful functional recovery and improvement in quality of life.

Conclusion:

Delgocitinib cream 20 mg/g demonstrated a rapid and clinically meaningful response in a severe case of chronic hand eczema unresponsive to conventional therapies. The early onset of action within the first week supports its potential role as an effective topical treatment option for difficult to treat chronic hand eczema.

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Characterization of the IHS4 Response to Lutikizumab Treatment in Biologic-Naïve Patients With Moderate-to-Severe Hidradenitis Suppurativa: A Single-Arm, Open-Label Phase 2 Study

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Introduction

Lutikizumab, an interleukin-1α/1β antagonist, improved International Hidradenitis Suppurativa Severity Score System (IHS4) in patients with moderate-to-severe hidradenitis suppurativa (HS) who failed anti-TNF therapy in a phase 2b study. Here, we dynamically characterized IHS4 responses to lutikizumab in biologic-naïve adults with moderate-to-severe HS.

Materials and Methods

In this multicenter, phase 2, open-label study, 47 biologic-naïve adults with moderate-to-severe HS received lutikizumab 300 mg every week (EW) for 16 weeks. Post-hoc efficacy assessments included improvement from baseline in IHS4, percent change from baseline in IHS4 among patients with IHS4 score ≥4 at baseline, percent change from baseline in abscess and inflammatory nodule (AN) count (≥3 AN at baseline), and percent change from baseline in draining tunnels (≥3 draining tunnels at baseline). Mixed model for repeated measures was used to handle missing data for continuous endpoints.

Results

Overall, 47 patients (70.2% female; mean [SD] age 34.4 [9.1] years; mean [SD] IHS4 24.4 (26.26) were enrolled across 20 sites. At Week 16, patients receiving lutikizumab showed improvement from baseline in IHS4 (-19.3) and improvement in percent change from baseline in IHS4 (-71.3%). The proportion of patients with severe HS (IHS4 ≥11) decreased from 74.5% at baseline to 22.5% at Week 16. At Week 16, patients receiving lutikizumab showed improvement in percent change from baseline in AN count (-70.8%) and draining tunnels (-84.8%).

Conclusions

In biologic-naïve adults with moderate-to-severe HS, lutikizumab 300 mg EW improved IHS4, as well as AN and draining tunnel counts over 16 weeks. These results support further investigation of lutikizumab in HS.

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Abstract N°: 578

Simultaneous Achievement of Clinical and Patient-Reported Outcomes in Response to Lutikizumab Treatment in Adults with Moderate-to-Severe Hidradenitis Suppurativa Who Have Failed Anti-TNF Therapy

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Introduction
Hidradenitis suppurativa (HS) significantly affects patients' clinical condition and quality of life. Examining the impact of HS treatments on both clinical outcomes and patient-reported outcomes simultaneously is therefore important. This analysis evaluated simultaneous achievement of clinical and patient-reported outcomes for patients treated with lutikizumab versus placebo.

Materials and Methods
Patients with moderate-to-severe HS who failed or demonstrated intolerance to anti-TNF therapy were randomized (1:1:1:1) to lutikizumab 300mg every week (Luti300EW), lutikizumab 300mg every other week (Luti300EOW), Luti100EOW, or placebo in a phase 2b study (NCT05139602). Post-hoc outcomes at Week 16 included simultaneous achievement of Dermatology Life Quality Index Minimally Clinically Important Difference (DLQI-MCID) and ≥50% improvement in HS Clinical Response (HiSCR50), DLQI-MCID+HiSCR75, ≥30% reduction and at least 1-unit reduction from baseline in worst skin pain (Numeric Rating Scale [NRS] 30) among patients with baseline NRS ≥3 and HiSCR50, and NRS30+HiSCR75. Missing data were handled using non-responder imputation.

Results
A total of 153 patients (61.4% female; mean [SD] age 40.5 [12.4] years; 70.6% Hurley Stage 3) were randomized across 54 sites. At baseline, mean (SD) DLQI was 16.5 (7.5) and mean (SD) NRS30 was 6.8 (1.8).At Week 16, DLQI-MCID+HiSCR50 was achieved by 35.1%, 33.3%, and 13.5% of patients receiving Luti300EW, Luti300EOW, and placebo, respectively. Similarly, greater proportions of patients on Luti300EW (29.7%) and Luti300EOW (27.3%) achieved DLQI-MCID+HiSCR75 at Week 16 versus placebo (5.4%).At Week 16, NRS30+HiSCR50 was achieved by 26.1%, 31.0%, and 6.5% of patients receiving Luti300EW, Luti300EOW, and placebo, respectively. Similarly, greater proportions of patients on Luti300EW (17.4%) and Luti300EOW (24.1%) achieved NRS30+HiSCR75 at Week 16 versus placebo (0%).Luti100EOW results were similar to placebo.

Conclusions
In a difficult-to-treat HS patient population that failed anti-TNF therapy, a higher proportion of patients treated with Luti300EW and Luti300EOW achieved simultaneous and meaningful improvements in both clinical and

patient-reported outcomes compared to placebo.

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Abstract N°: 608

Efficacy of Secukinumab in Patients with Psoriasis Affecting Difficult to Treat Areas: A Post Hoc Analysis of 4 Phase 3 Secukinumab Trials

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Introduction
Difficult-to-treat manifestations of psoriasis (PsO) include scalp PsO (sPsO) and palmoplantar PsO (ppPsO), each with their own unique challenges for achieving adequate disease control. This analysis assessed the efficacy of secukinumab in patients with difficult-to-treat PsO.

Materials and Methods
This analysis included pooled populations of patients with moderate to severe sPsO and ppPsO from the STATURE/GAIN (NCT01412944/ NCT02474069) and GESTURE/2PRECISE (NCT01806597/NCT02008890) phase 3 secukinumab trials, respectively. Efficacy outcomes for sPsO were assessed at Week 24, including Psoriasis Scalp Severity Index (PSSI), Investigator's Global Assessment (IGA), and scalp-specific IGA (ssIGA). Efficacy outcomes for ppPsO were assessed at Week 16, including Palmoplantar Psoriasis Area and Severity Index (ppPASI) and Palmoplantar IGA (ppIGA).

Results
This analysis included patients with sPsO receiving secukinumab 300 mg (n=575) and patients with ppPsO receiving secukinumab 300 mg (n=260), secukinumab 150 mg (n=134), or placebo (n=138). The proportion of patients with sPsO achieving 75%, 90%, and 100% improvements in PSSI (PSSI75/90/100) at Week 24 was 64.7%/58.8%/47.1%. IGA ≤1 and ssIGA ≤1 were achieved by 56.9% and 62.7% of patients with sPsO, respectively. Among patients with ppPsO, rates of ppPASI75/90/100 at Week 16 were greater in those receiving secukinumab 150/300 mg vs placebo (23.6%/31.8% vs 8.9%; 12.2%/19.6% vs 3.4%; 6.1%/12.2% vs 1.4%, respectively). Rates of ppIGA ≤1 at Week 16 were also greater in patients receiving secukinumab 150/300 mg vs placebo (22.1%/52.7% vs. 1.5%).

Conclusions
This post hoc analysis demonstrates the clinical efficacy of secukinumab in patients with difficult to treat manifestations of PsO affecting the scalp and palmoplantar regions.

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Abstract N°: 616

Clinical and Immunologic Overlap Between Psoriasis and Atopic Dermatitis: Two Illustrative Cases

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Introduction
Psoriasis and atopic dermatitis (AD) have traditionally been regarded as distinct immune-mediated dermatoses driven by Th17/Th1 and Th2 pathways, respectively. However, accumulating evidence suggests a dynamic inflammatory spectrum with overlapping cytokine profiles and clinical features. Targeted biologic therapies have further highlighted this interplay, with paradoxical psoriasiform or eczematous reactions emerging during pathway-specific inhibition.

Case Presentation
We report two illustrative cases demonstrating bidirectional phenotypic shifts between psoriasis and AD under targeted therapies. Clinical, histopathological, and laboratory findings were evaluated, and treatment responses were documented during follow-up.Case 1 involved a 45-year-old woman presenting with widespread pruritic erythematous plaques showing combined psoriasiform and spongiotic features, along with elevated total IgE levels. Initial therapies, including topical corticosteroids and cyclosporine, were ineffective or discontinued due to adverse effects. Treatment with a Th2 pathway inhibitor resulted in paradoxical worsening of psoriasiform lesions. Subsequent initiation of a JAK1 inhibitor led to rapid clinical improvement and sustained remission.Case 2 described a 44-year-old man with severe plaque psoriasis (PASI 32) who achieved near-complete clearance under IL-23 inhibition. After one year of therapy, he developed flexural eczematous plaques confirmed histologically as spongiotic dermatitis. The lesions were controlled with topical corticosteroids without discontinuation of biologic therapy.

Conclusion
These cases highlight the immunologic plasticity within the psoriasis–AD spectrum and demonstrate how targeted therapies may shift dominant inflammatory pathways, resulting in paradoxical reactions. Recognition of such phenotype switching is essential for accurate diagnosis and optimal therapeutic management. Clinicians should be aware of these potential transitions and consider alternative pathway-targeted treatments when paradoxical responses occur.

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Comparing Cancer Risks Between IL-17 and IL-23 Inhibitors in Psoriasis: A Multicenter Retrospective Cohort Study by Drug Class and Individual Agents

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Introduction

The safety of biologics for psoriasis in cancer development has been widely explored. The potential protective effect of individual biologic drugs remains to be investigated. We herein aim to compare the cancer risk in patients with psoriasis and psoriatic arthritis starting interleukin-17 inhibitor (IL-17i) and IL-23i biologics and identify the at-risk and at-benefit patient groups.

Materials and Methods

Using the TriNetX Global Collaborative Network dataset collected on February 6, 2026, this retrospective target-trial emulation study compared patients with psoriasis who initiated biologic agents targeting IL-17 or IL-23 pathways. After 1:1 propensity score (PS) matching for demographic, socioeconomic, and comorbidity factors, as well as previous medication exposure, cancer incidence was evaluated using Kaplan-Meier and Cox regression models. Secondary and sensitivity analyses for specific biologic agents were performed, stratified by demographic factors and comorbidities.

Results

Among patients with psoriasis or psoriatic arthritis, those who started IL-17i (N=11856) had a lower cancer risk than those who used IL-23i (N=11856) (with hazard ratio [HR] 0.80, 95% confidence interval [CI] 0.68–0.93, p=0.0043) over five years of follow-up, particularly among males and individuals without major comorbidities. The reduced risks were most notable for melanoma and non-melanoma skin cancer. (HR 0.76, 95% CI 0.59-0.98, p = 0.04). Sensitivity analyses limited to long-term exposures and monotherapies supported these findings. All individual agents from the IL-17i and IL-23i classes were associated with a lower cancer risk compared with TNFi. However, these individual IL-17 inhibitors showed lower, though not statistically significant, cancer hazard ratios than those of IL-23 inhibitors.

Conclusions

IL-17i was associated with a lower risk of cancer in patients with psoriasis than IL-23i, especially among males and those without major comorbidities.

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Abstract N°: 669

Intralesional Biologics in Dermatology: A Systematic Review

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Introduction
Intralesional administration of biologic and targeted agents has emerged as a potential therapeutic option for localized and treatment-refractory dermatologic diseases. Although biologics are widely used systemically in dermatology, evidence regarding intralesional use remains limited and dispersed across different article types, mostly consisting of case-based reports and small observational publications. We aimed to systematically review the available literature on intralesional biologics in dermatology and summarize the current evidence regarding indications, treatment regimens, clinical outcomes, recurrence, and safety.

Materials and Methods
A systematic literature search was performed in PubMed and Scopus for studies published up to February 2026. In total, 1,170 records were identified, of which 906 remained after duplicate removal. After exclusion of articles published before 2000 and screening of titles, abstracts, and full texts, 48 eligible English-language articles were included in the final review. Extracted data included study type, indication, patient number, intralesional agent, treatment regimen, time to response, concomitant therapies, follow-up, recurrence, treatment response, and adverse effects. Studies were further classified as randomized/prospective studies, retrospective studies or case series, and case reports.

Results
The 48 included articles were heterogeneous in both design and indication. Most were case reports, followed by retrospective studies or case series, while only a limited number were randomized, prospective, or interventional studies. Higher-level evidence was available for selected indications, including metastatic melanoma, oral potentially malignant disorders, psoriasis, oral lichen planus, Kaposi sarcoma, and other localized lesions treated with intralesional immunomodulatory approaches. Retrospective studies and case series most commonly evaluated rituximab, particularly in lymphoproliferative and orbital inflammatory disorders, and generally reported favorable local responses. Case reports comprised the largest body of evidence and included a broad range of conditions such as prurigo nodularis, palmoplantar pustulosis, pemphigus vulgaris, pemphigus foliaceus, cutaneous sarcoidosis, granuloma faciale, lichen sclerosus, keloids and hypertrophic scars, mycosis fungoides, primary cutaneous anaplastic large-cell lymphoma, conjunctival extranodal marginal zone lymphoma, mucosal melanoma, and balanitis xerotica obliterans. Rituximab was the most frequently reported intralesional biologic, while other agents included adalimumab, dupilumab, brentuximab vedotin, spesolimab, secukinumab,

guselkumab, bevacizumab, nivolumab, talimogene laherparepvec, and interleukin-2-based therapies. Reported outcomes were often favorable, with partial or complete local responses in many studies. Adverse effects were usually mild and localized, although occasional clinically significant events were also described.

Conclusions

Intralesional biologics appear to be a promising treatment option for selected localized and refractory dermatologic diseases. However, the currently available evidence is limited by marked heterogeneity and the predominance of low-level evidence, especially case reports. Further prospective and controlled studies are needed to better define efficacy, safety, optimal dosing strategies, and the most appropriate indications for intralesional biologic therapy in dermatology.

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Two birds, one stone: Concurrent control of chronic plaque psoriasis and pouchitis with tildrakizumab therapy

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Introduction

Psoriasis and ulcerative colitis (UC) share convergent immunopathogenic pathways, including IL-23/Th17 axis dysregulation, impaired T-regulatory function and microbial dysbiosis. Pouchitis, occurring in 32% of UC patients, refers to ileal pouch reservoir inflammation following proctocolectomy and is primarily driven by microbiota dysfunction. We report a case of sustained, concurrent remission of chronic plaque psoriasis and pouchitis with tildrakizumab therapy.

Case Presentation

A 66-year-old male with a 20-year history of psoriasis and chronic, antibiotic-refractory pouchitis following restorative proctocolectomy for UC, presented with recurrent psoriatic flares affecting the face, elbows, back, and lower extremities. His Psoriasis Area and Severity Index (PASI) score at presentation was 8. Prior therapies, including ultraviolet phototherapy, topical calcipotriol/betamethasone dipropionate gel, cyclosporine and adalimumab, provided suboptimal disease control. Concurrently, his pouchitis was causing recurrent diarrhoea and cramping, necessitating triple antibiotic cycling (amoxicillin, cephalexin and ciprofloxacin) and colonoscopic surveillance.

To address both cutaneous and gastrointestinal pathology, 100mg subcutaneous tildrakizumab injections every 12 weeks were initiated on compassionate grounds. Complete remission of psoriasis was achieved within nine months, paralleled by quiescent pouchitis confirmed on colonoscopy. Over five years of continued therapy, he maintained durable control of both conditions, with only very occasional, minor psoriasis flares and no adverse events.

Conclusion

Chronic antibiotic-refractory pouchitis may represent an autoimmune phenotype, particularly in patients with comorbid systemic autoimmune-mediated disorders such as psoriasis. Tildrakizumab, an IL-23p19 monoclonal antibody, likely downregulated mucosal inflammation via suppression of pathogenic Th17 responses, restoration of immune regulatory balance, and modulation of microbiota-driven immune activation. Clinical efficacy of ustekinumab, another IL-12/23 inhibitor, in autoimmune pouchitis further supports the mechanistic plausibility of selective IL-23 blockade in managing these coexisting autoimmune inflammatory conditions. This case highlights the potential utility of tildrakizumab in patients with overlapping systemic autoimmune-mediated disorders, underscoring the need for further mechanistic and clinical evaluation.

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Abstract N°: 677

Linear bullous lichen planus induced by docetaxel and successfully treated with dupilumab

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Introduction

Bullous lichen planus (BLP) is a rare, immune-mediated variant of lichen planus (LP) characterised by tense, pruritic vesicles and bullae on pre-existing plaques. BLP is thought to arise from CD4+ T-cell activation and CD8+ T-cell–mediated keratinocyte apoptosis, distinct from the autoantibody-driven mechanism of blistering in lichen planus pemphigoides (LPP). Treatment of BLP is challenging, as few cases have been reported in the literature, and no standard treatment or management guidelines exist. BLP may be distinguished from LPP both clinically and through diagnostic tests (Table 1). We report a case of a 57-year-old man with linear BLP affecting the bilateral lower limbs, triggered by docetaxel chemotherapy for metastatic prostate cancer, successfully treated with dupilumab.

Feature	Disease	
	BLP	**LPP**
Pathogenesis	Cell-mediated (CD4+ T cell activation and CD8+ T cell-driven keratinocyte apoptosis), resulting in secondary subepidermal blistering.	Autoantibody production against BP180 and/or BP230.
Clinical Features	Vesiculobullous lesions secondarily arise on or close to existing LP plaques, often in a localised distribution.	Papulosquamous and vesiculobullous lesions classically arise on unaffected skin or occasionally on existing LP lesions, often in a generalised distribution.
Histopathology	Classical LP features: hyperkeratosis, saw-tooth acanthosis, lymphocytic infiltrate in the DEJ, apoptotic or dyskeratotic keratinocytes (Civatte or colloid bodies) and interface dermatitis.	Subepidermal blistering and eosinophilic infiltrate with or without histological evidence of LP.
Immunofluorescence	Usually negative or shows nonspecific IgM deposition at the DEJ.	IgG and C3 deposition at the DEJ.
Serology	Seronegative.	Circulating BP180 and/or BP230 antibodies.

Table 1: Distinguishing between BLP and LPP1. Abbreviations: BLP; bullous lichen planus, LPP; lichen planus pemphigoides, LP; lichen planus, DEJ; dermal-epidermal junction.

Case Presentation

A 57-year-old man of Greek heritage presented in May 2020 with an 18-month history of a blistering rash over the bilateral lower limbs, manifesting as tense, pruritic, oedematous bullae that frequently ruptured causing painful erosions and crusting. This was suspected to be secondary to docetaxel chemotherapy, which the patient was receiving for metastatic prostate cancer, as there was a temporal relationship between the initiation of docetaxel and the onset of the patient's cutaneous symptoms. Notably, there was further progression of the rash with ongoing docetaxel therapy, which was administered over six cycles from November 2018 to March 2019 due to prioritisation of his cancer management. During this time he was treated with topical betamethasone ointment, psoralen and ultraviolet A (PUVA) therapy, and hexachlorophene antibacterial wash, although this led to minimal disease improvement.

Diagnosis of linear BLP was confirmed by LP features on histopathology including parakeratosis, epidermal atrophy, Civatte bodies in the basal epidermis, mild interface inflammation with scarring, subepidermal vesicles and lymphocytic infiltrate in the papillary dermis (Figure 1). Direct immunofluorescence was negative for IgG, IgA, IgM, albumin, fibrinogen C3 and C1q, and serology was negative for BP180, BP230, DSG1, DSG3, envoplakin, and collagen VII autoantibodies on three separate occasions, excluding LPP. Conventional interventions, including UVB therapy, prednisone, acitretin, isotretinoin and rituximab, provided minimal benefit, and prolonged corticosteroid use led to steroid-induced type 2 diabetes, osteoporosis, and Cushing syndrome. Given the refractory disease course and steroid-induced complications, 300mg subcutaneous dupilumab was initiated fornightly alongside prednisone tapering.

By four months of dupilumab therapy, his Bullous Pemphigoid Disease Area Index (BPDAI) activity score decreased from 20/120 to 6/120, and pruritus improved from 23/30 to 16/30. Following 28 months of dupilumab,

his BPDAI activity score was 1/120 alongside residual post-inflammatory hyperpigmentation.

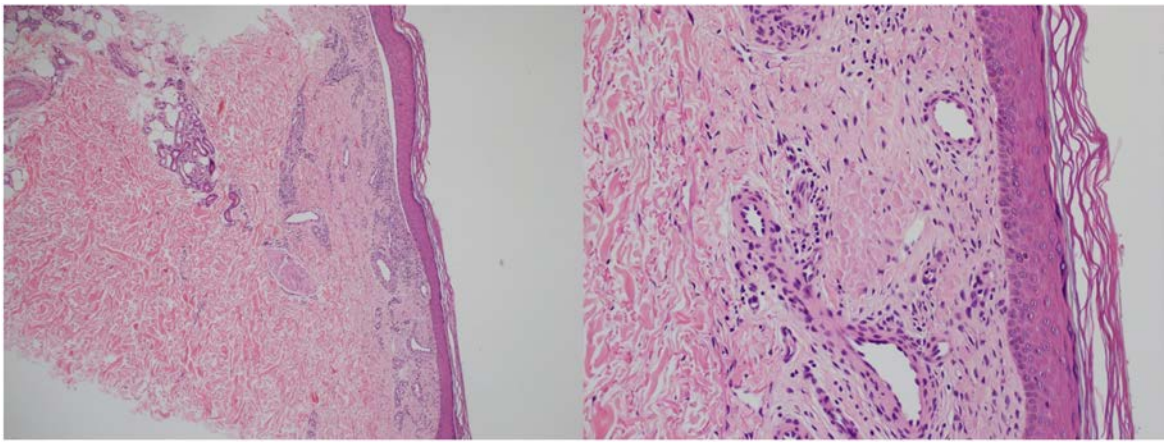

Figure 1: H&E stained section demonstrating parakeratosis, epidermal atrophy, Civatte bodies in the basal epidermis, mild interface inflammation with scarring, subepidermal vesicles and lymphocytic infiltrate in the papillary dermis.

Conclusion

Dupilumab, an IL-4 and IL-13 inhibitor, has demonstrated efficacy in BP and LP. The favourable response in this case suggests a potential therapeutic role in BLP, likely via downregulation of Th2-mediated pathways, reducing $CD4^+$ T-cell-driven inflammation central to BLP pathogenesis. This report highlights dupilumab as a promising option for refractory BLP and underscores the need for further research into its mechanism and broader applicability in this rare condition.

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Abstract N°: 683

UNFOLDING THE EMERGING APPLICATIONS OF JAK INHIBITORS IN DERMATOLOGY – A SYSTEMATIC REVIEW

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Introduction

Janus kinase (JAK) inhibitors are established treatments for select dermatologic conditions; however, their off-label use has expanded across refractory skin diseases.

Materials and Methods

We conducted a PRISMA-compliant systematic review (PROSPERO CRD420251252167) of MEDLINE, Embase, and Web of Science from inception to October 1, 2025. Eligible studies reported off-label JAK inhibitor use in dermatologic disorders beyond approved or late-phase trial indications.

Results

Of 9,182 records screened, 277 studies met inclusion criteria, comprising 210 case reports, 56 case series, and 11 retrospective studies involving 764 patients. Due to clinical heterogeneity, findings were narratively synthesised using a structured disease-family framework. Off-label use spanned diverse inflammatory and autoimmune phenotypes, most frequently lichenoid, neutrophilic, and granulomatous dermatoses. Overall, 725 of 764 (94.88%) of patients were reported to achieve clinical benefit, including complete or near-complete response in 209 patients (27.35%) and significant or partial improvement in 516 patients (67.53%) while 35 patients (4.58%) showed no clinical change, while a small proportion; 4 patients (0.52%) experienced disease worsening. A total of 154 adverse events were documented, most commonly with tofacitinib; most were mild to moderate, although 6 were serious and 25 led to treatment discontinuation.

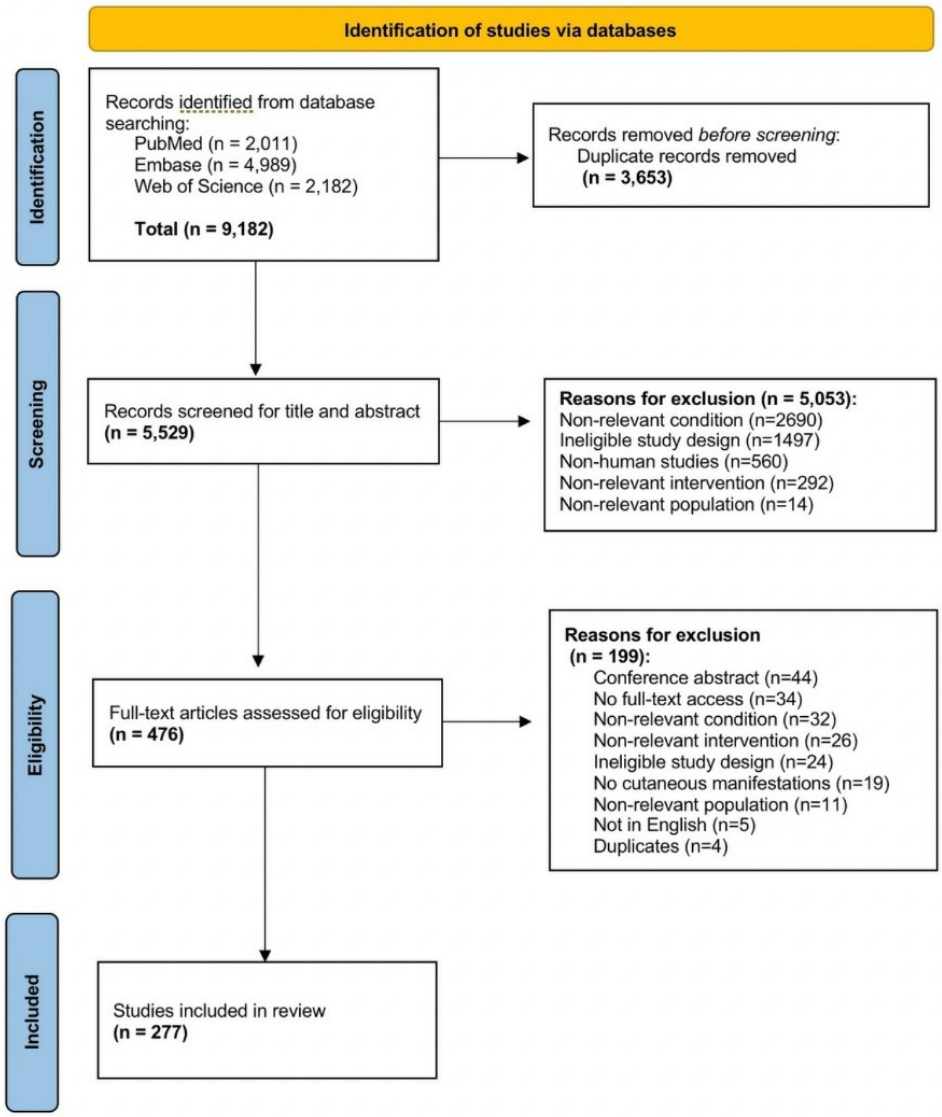

Figure 1: PRISMA flow diagram of study selection: Flow diagram illustrating the study selection process in accordance with the PRISMA guidelines. The diagram presents the number of records identified through database searching, records after duplicate removal, records screened, full-text articles assessed for eligibility, and studies included in the final qualitative (narrative) synthesis, with reasons for exclusion at each stage.

Conclusions

Despite encouraging signals across multiple refractory dermatologic conditions, the predominance of uncontrolled case-based evidence, heterogeneous treatment regimens, variable outcome reporting, and frequent concomitant immunosuppressive therapy substantially limits attribution of observed efficacy or safety specifically to JAK inhibitors as monotherapy. Current evidence therefore remains hypothesis-generating rather than practice-defining. Prospective disease-specific studies employing standardised outcome measures, clearly defined treatment positioning, and long-term safety monitoring are

required to better define the therapeutic role, optimal patient selection, and safety profile of JAK inhibitors across emerging off-label dermatologic indications.

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Abstract N°: 694

Deucravacitinib-Induced Remission of Refractory Discoid Lupus Erythematosus and Plaque Psoriasis: A Clinical Case Supported by Reflectance Confocal Microscopy

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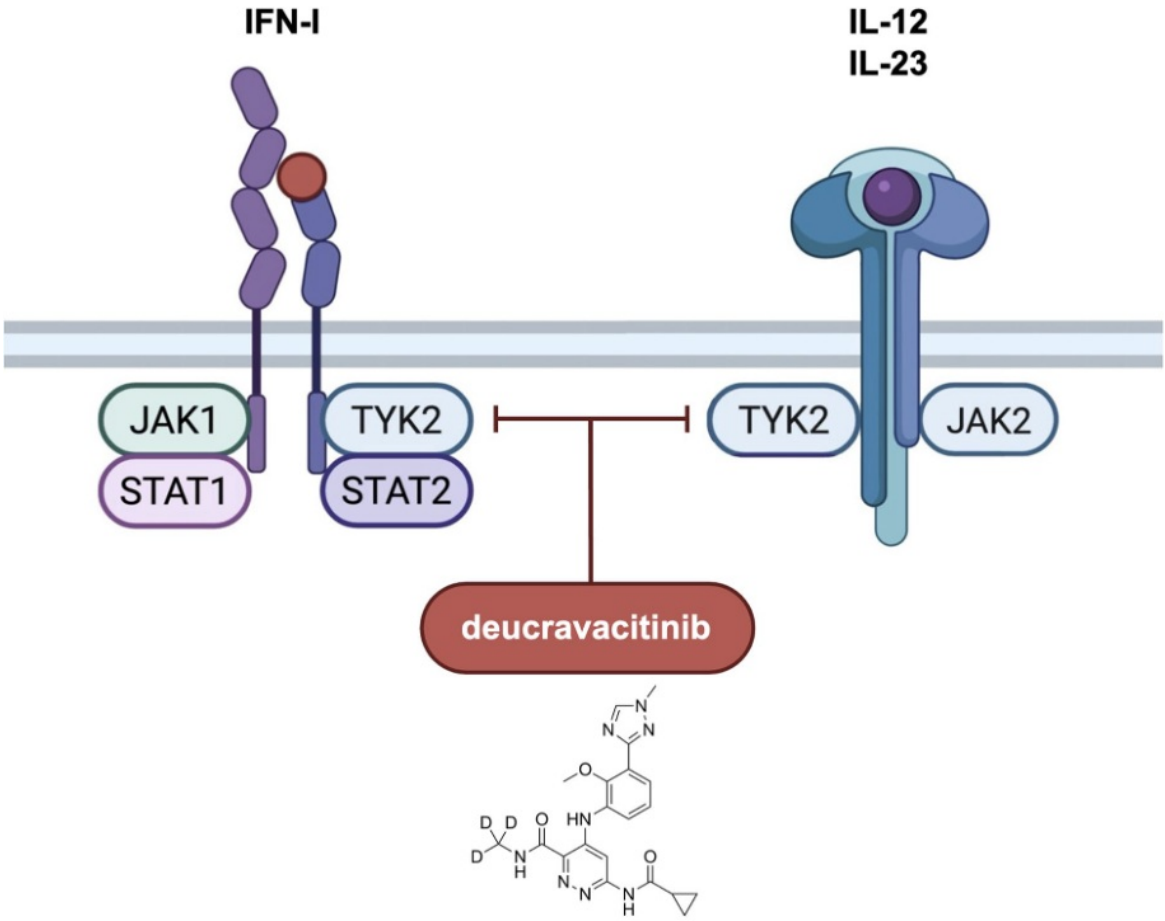
Introduction

Discoid lupus erythematosus (DLE) is a chronic form of cutaneous lupus erythematosus characterized by persistent inflammatory plaques that may evolve into scarring, dyspigmentation, and permanent cosmetic damage, with a substantial impact on quality of life. Despite this burden, no systemic therapies are specifically approved for cutaneous lupus erythematosus, and treatment is usually extrapolated from systemic lupus erythematosus. Antimalarials remain the therapeutic cornerstone, whereas corticosteroids, immunosuppressants, retinoids, and other systemic agents show variable and often unpredictable responses in refractory disease. Type I interferon signaling is a central driver of disease pathogenesis, making tyrosine kinase 2 (TYK2), a key mediator of the JAK–STAT pathway, an attractive therapeutic target. Deucravacitinib, an oral selective allosteric TYK2 inhibitor approved for moderate-to-severe plaque psoriasis, has recently emerged as a promising option in lupus because it modulates type I interferon, IL-12, and IL-23 signaling. Here, we describe a clinical case of refractory DLE associated with plaque psoriasis successfully treated with deucravacitinib, with supportive reflectance confocal microscopy findings.



Deucravacitinib inhibits TYK2, a key mediator of the JAK–STAT pathway shared by interferon-driven lupus and IL-12/IL-23–driven psoriasis, providing a common therapeutic mechanism for both diseases.

Case Presentation

A 29-year-old woman with biopsy-proven DLE presented with persistent facial plaques involving the nasal dorsum and left eyebrow, refractory to previous treatment with prednisone and hydroxychloroquine. Rheumatologic work-up excluded systemic lupus erythematosus and inflammatory arthropathy, and the autoimmune serologic panel, including antinuclear antibodies, anti-dsDNA, and extractable nuclear antigens, was negative. At dermatologic reassessment, reflectance confocal microscopy of the nasal lesion showed irregular epidermal architecture, leukocyte exocytosis, dilated follicular openings with keratotic plugs, dermal fibrosis, and pigment incontinence, consistent with active disease. During follow-up, the patient developed symmetrically distributed erythematous scaly plaques on the trunk and extremities; histopathological examination confirmed moderate-to-severe plaque psoriasis, with a Psoriasis Area and Severity Index (PASI) of 16. Ixekizumab was started according to the approved schedule and initially improved psoriasis, but DLE lesions remained active and psoriatic disease later relapsed. Treatment was therefore switched to deucravacitinib 6 mg once daily. After 9 weeks, psoriasis showed near-complete clearance (PASI 0.2), accompanied by early regression of DLE lesions. By week 27, complete clinical remission of psoriasis (PASI 0) and full resolution of active DLE lesions were achieved. Residual findings were limited to mild post-inflammatory atrophy and hypopigmentation. Follow-up reflectance confocal microscopy documented restoration of the regular honeycomb epidermal pattern, disappearance of follicular keratotic plugs, inflammatory cells, pigment incontinence, and peri-adnexal fibrosis, confirming remission. Treatment was well tolerated overall; the only adverse event reported was a transient episode of stomatitis.



Clinical, dermoscopic, and reflectance confocal microscopy evaluation of the nasal discoid lupus erythematosus lesion before and after treatment with deucravacitinib. Baseline images show an active erythematous plaque with follicular keratotic plugging and confocal features of inflammation and epidermal disorganization. Follow-up images at week 27 demonstrate complete resolution of active disease, with disappearance of keratotic plugs and inflammatory findings, and restoration of a regular epidermal architecture.

Conclusion

This case supports deucravacitinib as a potentially effective targeted treatment for refractory DLE, particularly when associated with concomitant plaque psoriasis, allowing simultaneous control of two immune-mediated skin diseases with a single oral agent and reducing the need for multiple systemic therapies. It also highlights the value of reflectance confocal microscopy as a noninvasive tool for diagnosis and therapeutic monitoring in cosmetically sensitive sites. Larger prospective studies and ongoing clinical trials are needed to better define the long-term efficacy, safety, and positioning of TYK2 inhibition in cutaneous lupus erythematosus.

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Abstract N°: 726

Preliminary Results for an Open-label Phase 2a Trial (SIGNAL-AA Part B) of Bempikibart, a Novel IL-7Rα Antibody in Patients with Severe or Very Severe Alopecia Areata (AA)

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Introduction

Bempikibart, an IL-7Rα antibody that potently blocks IL-7 and TSLP signaling, is a potentially novel therapeutic for autoimmune and inflammatory diseases including alopecia areata (AA). Bempikibart demonstrated proof-of-concept in Part A of SIGNAL-AA with both improvement in SALT score versus placebo and indication of remittive effects (King et al., AAD 2025).

Materials and Methods

Based on the POC established in Part A, a Part B study with an optimized dosing schedule was undertaken. This ongoing Part B study enrolled 33 patients with severe (SALT score 50-94) or very severe (SALT score 95-100) AA with duration of current episode ≤ 4 years and includes a 36-week treatment regimen of 200mg subcutaneous bempikibart (including a loading regimen of 4 weekly doses followed by 16 every other week doses) and 16-week post-treatment follow-up. Females comprise 73% of overall patients. The mean SALT score at baseline is 75.7, 24% of patients have very severe AA, and the mean duration of the current episode of severe or very severe AA is 2.2 years. Prior exposure to oral JAK inhibitors (36% of patients enrolled) was permitted. Part B data will reach the 36-week primary endpoint in all patients mid-2026.

Results

Preliminary pharmacokinetics demonstrate the loading regimen provided a 3-fold higher mean trough concentration of 28.76 ug/ml at Day 22 and achieved steady state concentrations approximately 10 weeks earlier as compared to dosing without a loading regimen (week 3 vs week 12-14). Among the 33 patients enrolled, 36.4%, 30.3%, and 27.3% of patients to date have achieved a $SALT_{30}$, $SALT_{50}$, and SALT score ≤20, respectively. Thirteen of 33 patients remain on therapy and have not yet reached the 36-week endpoint. SALT score ≤20 responses have been observed in both severe and very severe patients and both IgE high (≥200 IU/mL) and low (<200 IU/mL) patients.

The SIGNAL-AA bempikibart safety profile remains favorable with all drug-related AEs being mild or moderate to date. Injection site reactions (ISRs) remain low at 6% of all bempikibart administrations in AA with all ISRs being mild to date.

Conclusions

Preliminary results of the bempikibart SIGNAL-AA Part B study support advancement into registrational development, reinforce bempikibart's potential as a first-in-class biologic, and further highlight the potential for

targeted biologics for the treatment of severe and very severe AA.

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Abstract N°: 735

EFFICACY OF INTRALESIONAL ACYCLOVIR VERSUS CRYOTHERAPY FOR TREATMENT OF PLANTAR WARTS

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Introduction
Warts, benign proliferation of skin and mucosa caused by human papilloma virus (HPV), are treated by different modalities among which cryotherapy remains the most common. The objective of this study was to assess comparative efficacy of Intralesional acyclovir versus cryotherapy for plantar warts.

Materials and Methods
This quasi-experimental study was conducted on eighty-four patients of plantar warts (1–10 in number) enrolled after obtaining ethical approval and written informed consent. Patients were divided into group A and B. Group A patients received intralesional Acyclovir in a dose of 0.1ml (70 mg/ml) at the base of each wart while Group B patients had cryotherapy for two freeze and thaw cycles with each cycle of 10 seconds duration. Lesion size and clinical photographs were recorded at baseline and at follow-up visits scheduled every 2 weeks (maximum 5 sessions) and followed for further 4 months. Response was considered as complete (100% resolution), partial (resolution 50-99%) or no response (resolution <50%).

Results
A total of 84 patients with 42 (79% Males and 21% females) in group A and 42 (55% males and 45% females) in group B, having mean age 27.79±4.36 and 26.74±6.03 years, mean wart size 0.43±0.09 and 0.49±0.09 cm and mean number of warts 1.61±1.06 and 1.67±1.12 in each group respectively. 83% achieved complete response, 10% lost to follow-up and 7% refused treatment in Group A whereas 48% achieved complete and partial responses respectively and 4% had no response in Group B ($p<0.001$).

Conclusions
Acyclovir is an effective treatment modality of cutaneous warts with better results in males presenting with single plantar wart.

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Abstract N°: 771

Exploring the relationship between lesion severity and itch improvement in patients with prurigo nodularis treated with dupilumab

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Introduction
Prurigo nodularis (PN) is a chronic inflammatory disease that profoundly impacts quality of life. The complex pathophysiology of PN involves immunologic and neuropathic imbalance that drive fibrotic nodule formation. A fundamental unanswered question in PN is whether the mechanisms underlying lesion and itch severity are connected, i.e.: is higher baseline lesion severity associated with greater itch and does higher baseline lesion severity predict greater itch improvement with dupilumab? Here, we explore the relationship between lesion severity and itch intensity and improvement in patients treated with dupilumab.

Materials and Methods
LIBERTY-PN PRIME (NCT04183335) and PRIME2 (NCT04202679) were randomized, double-blind, 24-week, phase 3 trials of dupilumab in adult patients with PN. Patients were randomized to dupilumab 300 mg every 2 weeks or matched placebo. In this pooled post-hoc analysis, we assessed baseline (BL) lesion severity (Investigator's Global Assessment of PN Stage [IGA PN-S] 3 [moderate] vs 4 [severe]) and itch response (Worst Itch Numeric Rating Scale [WI-NRS] ≥4-point improvement) in patients treated with dupilumab. Analysis was based on observed data. All p-values are nominal and no adjustment was made for multiple testing.

Results
The relationship between baseline lesion severity and itch was investigated in patients with PN. Dupilumab treatment demonstrated robust and consistent itch improvement versus placebo regardless of baseline lesion severity. At Week 24, patients with a moderate lesion score (BL IGA PN-S 3; dupilumab N=103, placebo N=102) achieved ≥4-point WI-NRS improvement of 62% vs 23% ($p<0.0001$), while those with a severe lesion score (BL IGA PN-S 4; dupilumab N=50, placebo N=54) showed nearly identical response rates of 66% vs 26% ($p=0.0046$). In patients achieving ≥4-point WI-NRS improvement at Week 24, treatment-emergent adverse events (TEAEs) were similar between the dupilumab (61 [62.9%]) placebo (22 [59.5%]) groups. Overall safety was generally consistent with the known safety profile of dupilumab.

Conclusions
Dupilumab provides substantial and equivalent itch relief in patients with both moderate and severe lesion scores, supporting its use for pruritus management across the spectrum of PN lesion severity. These findings indicate that IL-4/IL-13 may be a primary driver of pruritus in PN, independent of visible lesion burden

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Abstract N°: 779

INFLIXIMAB-INDUCED PSORIASIFORM DERMATITIS WITH OOZY PSORIATIC SCALP LESION IN AN IBD PATIENT: A DIFFICULT-TO-TREAT CASE SCENARIO

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Introduction

Infliximab, an anti-TNF agent, is used to treat various inflammatory conditions such as inflammatory bowel disease, rheumatoid arthritis, psoriasis, and psoriatic arthritis. Paradoxically, anti-TNF-alpha agents may induce psoriatic eruptions or worsen preexisting psoriatic skin lesions. This case report describes a paradoxical reaction to infliximab and the challenges encountered in managing the condition.

Case Presentation

A 30-year-old male presented with erythematous skin lesions all over his body and oozy scalp lesions that had persisted for 4–5 months. The patient is a known case of IBD (Inflammatory Bowel Disease) and was receiving treatment accordingly. He was administered infliximab intravenously on a monthly basis, along with Azathioprine 25 mg. A biopsy of the skin and scalp lesions was performed, and the findings were suggestive of psoriasiform dermatitis. In consultation with his gastroenterologist, infliximab was discontinued after completion of its course. The patient was subsequently treated with oral steroids (as the lesions appeared more atopic-eczematous than typical of psoriasis), topical steroids, and oral antibiotics for the scalp lesions. Although the skin lesions initially improved with oral steroids, a switch to steroid-sparing agents such as methotrexate (MTX) led to exacerbation. Similarly, while the scalp lesions improved with ciprofloxacin, they did not subside when the treatment was changed to a long-term anti-inflammatory agent like doxycycline. The patient is currently being treated with oral cyclosporine and remains under evaluation.



Erythematous oozy scalp lesions with matting of hair over the occipital region of the scalp

Conclusion

There is currently no standardized treatment protocol for infliximab-induced psoriasiform dermatitis or associated scalp lesions. This case highlights the paradoxical reaction linked with infliximab and underscores the complexities involved in managing such conditions, emphasizing the need for future treatment guidelines.

EADV CONGRESS
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Abstract N°: 828

From Refractory Disease to Sustained Clinical Remission: Rituximab in Severe Pemphigus Vulgaris

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Introduction

Pemphigus vulgaris is a chronic autoimmune blistering disease that can be severe and difficult to control with conventional immunosuppressive therapies. In advanced cases, it may become life threatening due to extensive skin involvement, fluid loss, electrolyte imbalance, and secondary infections. Before the introduction of rituximab, systemic corticosteroids were the mainstay of treatment, although associated with significant morbidity.

Case Presentation

A 46 year old male with clinically and histopathologically confirmed pemphigus vulgaris was admitted after 20 days of extra institutional treatment with severe disease activity, extensive mucocutaneous involvement, deterioration of general condition, electrolyte imbalance, and sepsis. Initial management was conducted in collaboration with Internal Medicine and included fluid and electrolyte replacement as well as targeted antibiotic therapy. The patient had previously received high dose corticosteroids (up to 150 mg per day of prednisone) and mycophenolate mofetil, without adequate disease control. Steroid related adverse effects were observed, including myopathy, infections, and weight loss. Due to persistent disease activity, rituximab was initiated (1 g intravenously on days 0 and 14). Progressive clinical improvement was observed, with complete resolution of lesions. Corticosteroids were gradually tapered and eventually discontinued, maintaining low dose mycophenolate mofetil (1 g per day) as maintenance therapy. After clinical stabilization, rehabilitation therapy was initiated, with significant improvement in functional status and quality of life. At 12 months, the patient remained in sustained clinical remission, without new lesions and only residual post inflammatory hyperpigmentation. A maintenance cycle of rituximab was scheduled at month 12.

Conclusion

Rituximab was effective and safe in severe refractory pemphigus vulgaris with systemic complications, achieving sustained clinical remission, corticosteroid discontinuation, and improved functionality and quality of life. This case highlights the importance of a multidisciplinary and comprehensive approach, combining targeted therapy with supportive and rehabilitative care to optimize patient outcomes.

EADV CONGRESS
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Abstract N°: 830

Fluorescence Molecular Imaging of Ustekinumab-800CW in Patients with Psoriasis: Insights Into Local Drug Distribution

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Introduction

Biologic agents significantly improved psoriasis treatment, yet a subset of patients show primary non-response or experience secondary loss of response. Because treatment evaluation typically occurs after 12-16 weeks, ineffective therapy may lead to clinical deterioration. Moreover, biologics impose a considerable financial burden on healthcare systems and may cause adverse effects. Fluorescence molecular imaging has shown value in inflammatory bowel disease, by visualizing in vivo distribution of labeled biologics such as vedolizumab. These findings suggest similar opportunities within dermatology. This study investigates the tissue distribution of ustekinumab conjugated to IRDye800CW in patients with psoriasis, aiming to identify target cell populations and gain insight into treatment response. A deeper understanding of these processes may ultimately support future strategies for patient stratification and personalized biologic therapy.

Materials and Methods

In arm one, fifteen patients with active psoriasis will receive an intravenous sub-therapeutic dose of ustekinumab-800CW (15 or 25 mg). After 2-4 days, they will undergo a study procedure consisting of fluorescence signal visualisation using a fluorescence camera, followed by quantitative spectroscopy measurements of lesional skin and non-lesional skin. Subsequently, skin biopsies will be collected from both areas. Arm two includes ten patients who have received at least three months of standard-of-care ustekinumab treatment, followed by an additional intravenous dose of 25 mg ustekinumab-800CW, after which they will undergo the imaging and biopsy procedure. Additionally, three control patients will undergo the full procedure without tracer administration. Finally, ex vivo analysis will be performed on all biopsies, including fluorescence microscopy and spatial transcriptomics.

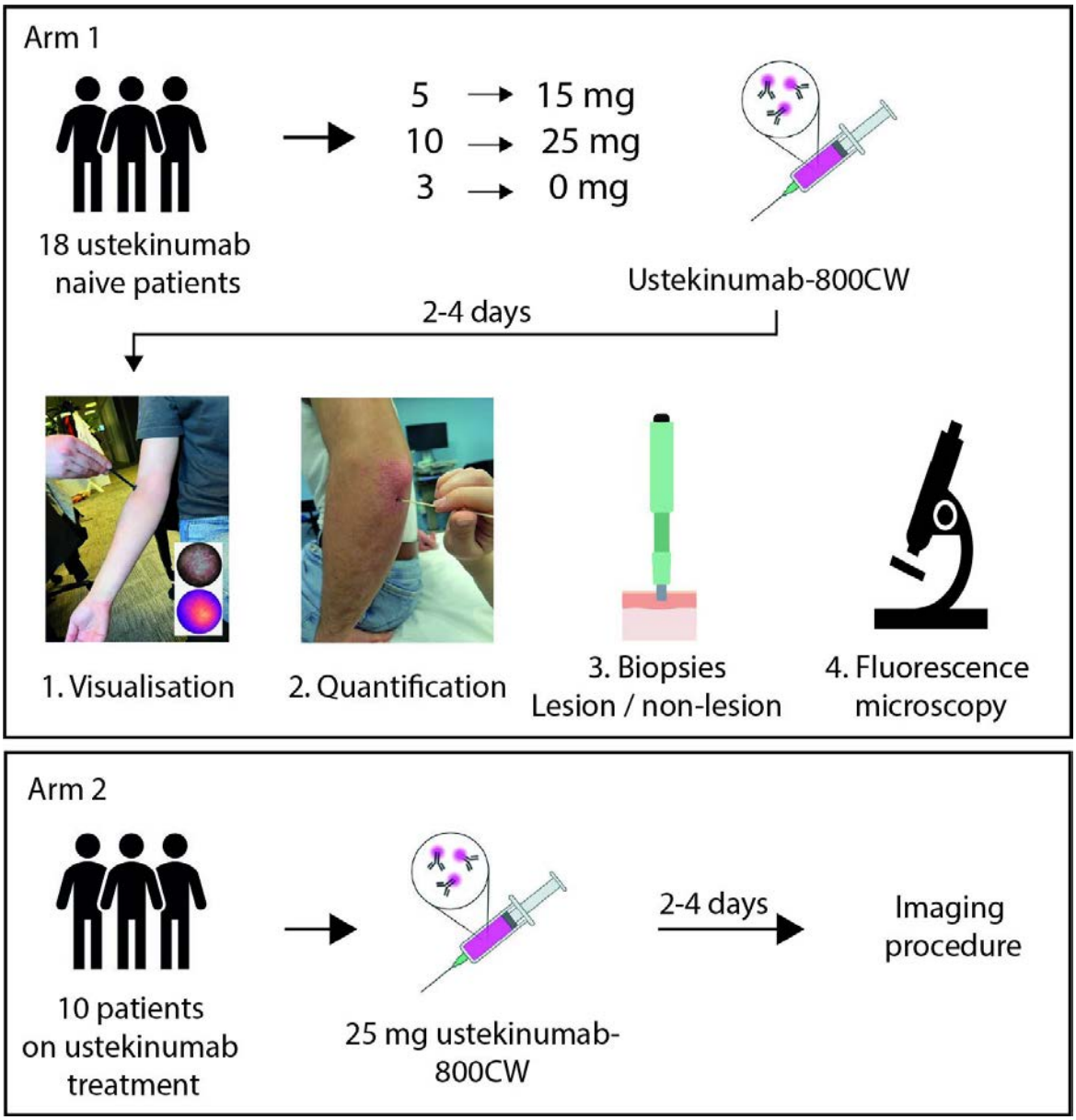

Figure 1 - study design.

Results

Six patients have been included in arm one, two in arm two and one as a control patient. No adverse events were observed. Preliminary analysis of fluorescence imaging data revealed strong light reflectance from the camera's light source on the skin, complicating image interpretation. However, spectroscopy data showed a clear increase in fluorescence intensity in lesional skin (median [IQR]: 0.018 mm^{-1} [0.014–0.022]) compared with non-lesional skin (0.009 mm^{-1} [0.009–0.010]). Fluorescence microscopy confirmed tracer presence in skin biopsies.

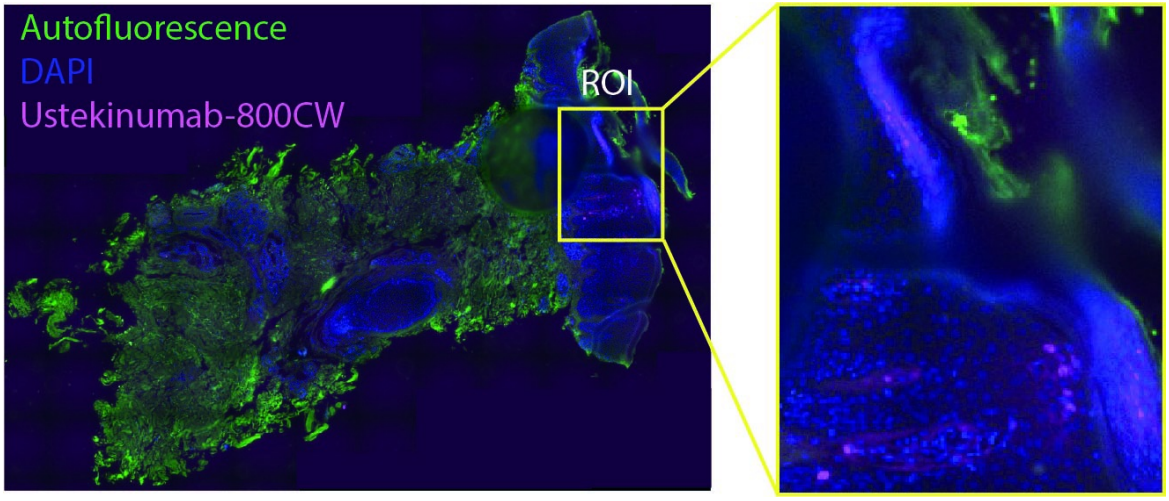

Figure 2 - Fluorescence microscopy of an active psoriatic lesion following administration of 15 mg ustekinumab-800CW. The biopsy section is counterstained with DAPI (blue) to visualize cell nuclei. The green channel represents tissue autofluorescence, while the magenta signal corresponds to ustekinumab-800CW, confirming tracer localization within the lesional skin.

Conclusions

These preliminary findings from our pilot study demonstrate the feasibility of detecting ustekinumab-800CW in vivo in patients with psoriasis. Both spectroscopy and fluorescence microscopy successfully identified tracer presence in lesional and non-lesional skin. Completion of the full cohort is essential to validate these early observations and to further characterize drug distribution patterns. In the future, this imaging-based approach may support prediction of treatment response and might enable stratification of patients for personalized biologic therapy.

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Abstract N°: 858

Characteristics Influencing the Prevention of Psoriatic Arthritis With Secukinumab in Patients With Moderate to Severe Plaque Psoriasis: 5-Year Real-World Results From the SERENA Study

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Introduction
Secukinumab, an IL-17A inhibitor, has shown a ≈67% reduction in annual psoriatic arthritis (PsA) incidence among patients with psoriasis (PsO) with no history of PsA in the SERENA real-world study versus a historic non-biologic treatment cohort.[1] This post hoc analysis of the 5-year SERENA study aimed to identify characteristics influencing PsA prevention.

Materials and Methods
SERENA was a long-term, prospective, observational study, conducted at 438 sites in 19 countries, to collect data in a real-world setting on the safety and effectiveness of secukinumab in adult patients with moderate to severe plaque PsO. This post-hoc analysis assessed the association between the following variables and PsA development: age, sex, weight, body mass index (BMI), race, duration of secukinumab before enrolment, PsO disease duration, prior biologic exposure, smoking status, baseline Psoriasis Area and Severity Index (PASI), nail involvement, baseline Physician Global Assessment (PGA) 0/1, and baseline body surface area (BSA). Adjusted hazard ratios with 95% confidence intervals from Cox regression analysis were used to quantify associations between these factors and PsA development.

Results
Among 1334 PsO patients without PsA (mean age 47.4 years; 70% male), secukinumab yielded 4672 patient-years of follow-up. After 5 years, 96.9% (1292/1334) of patients remained free from PsA, with an exposure-adjusted incidence rate of 0.90 per 100 patient-years. Sex (male vs female: HR, 0.52, P=0.0353) and PsO disease duration (<15 vs ≥15 years: HR, 0.48, P=0.0546) were strong influencing factors on PsA prevention; age, weight, BMI, race, prior biologic exposure, smoking status, baseline PASI, nail involvement, PGA, and BSA were not. Skin clearance was sustained, with 67.3% and 44.0% achieving PASI 90 and PASI 100 at 5 years, respectively.

Conclusions

Conclusions: Five years of secukinumab treatment resulted in a low PsA incidence rate (0.90 per 100 patient-years), with 96.9% of patients remaining PsA free. Sex and PsO duration were significant factors; other variables showed no significant effect. Secukinumab provided durable skin control and may play a role in PsA risk reduction.

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Abstract N°: 859

Early secukinumab treatment within the window of opportunity: a systematic review of its potential as a disease modifying strategy in the management of psoriatic disease

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Introduction

Early intervention with biologic therapies in psoriasis and psoriatic arthritis may provide significant benefits, including improved disease control, modification of disease pathology, and the potential to delay or prevent progression to psoriatic arthritis. This systematic review evaluated published evidence on the impact of early versus delayed initiation of secukinumab treatment on clinical outcomes, quality of life, and molecular disease signatures in patients with psoriasis and psoriatic arthritis. It also assessed the effect of early intervention with secukinumab on disease activity, progression to psoriatic arthritis, and related comorbidities in psoriatic arthritis.

Materials and Methods

A systematic literature search was performed according to a pre-defined search strategy. Eligible publications were English-language studies or meta-analyses of secukinumab in psoriasis or psoriatic arthritis that investigated early intervention or stratified outcomes by disease duration. Of 293 records identified, 14 met the inclusion criteria, representing over 5,000 patients.

Results

Most clinical trials and observational cohorts suggest that patients with psoriatic arthritis who initiated secukinumab earlier in their disease course achieved higher rates of disease remission and treatment retention, and lower disease activity and severity, than those with longer-standing disease. Some analyses, however, reported no clear differences by disease duration, especially at later timepoints. In psoriasis, new-onset disease responded more rapidly than long-standing disease, and shorter disease duration was associated with greater time to relapse after secukinumab withdrawal. Mechanistic studies revealed distinct DNA methylation and transcriptome responses in early psoriasis, suggesting that early treatment may modify pathogenic pathways. Longitudinal data further suggested that early biologic intervention may reduce the risk of progression from psoriasis to psoriatic arthritis.

Conclusions

Current evidence suggests that, in psoriatic disease, early intervention with secukinumab may improve disease

control and potentially modify disease trajectory; however, larger, prospective trials are required to confirm these findings.

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Abstract N°: 872

Prospective study on the effectiveness and safety of baricitinib in alopecia areata under real-world conditions, including the analysis of specific subpopulations

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Introduction
Baricitinib is a selective Janus kinase (JAK) 1 and 2 inhibitor that has been approved for the treatment of adult patients with alopecia areata (AA). However, there is limited evidence regarding the role of this treatment in populations excluded from clinical trials and from the drug's label. The aim of this study was to evaluate the effectiveness and safety of baricitinib in the treatment of AA under real-world clinical practice conditions, as well as to describe its use in special populations.

Materials and Methods
Prospective, observational, single-center study in patients with AA treated with baricitinib. Demographic, clinical, and safety variables were collected for up to 12 months. Overall effectiveness and safety were analyzed, as well as in subgroups: patients under 18 years of age and patients with a current episode duration longer than 8 years.

Results
A total of 56 patients with severe AA were included, with a mean age of 36.8 years; 66% were women, 32.1% had alopecia universalis, and 14.2% were patients under 18 years old. The mean duration of the current episode was 10.13 years, and 44.6% had a duration longer than 8 years. The baseline SALT score was 68.3, reaching a mean SALT of 21.94 at 12 months ($p<0.05$).Patients under 18 years of age more frequently had a family history of AA (25% vs 4.1%; $p=0.04$) and started baricitinib at a lower mean dose (3 mg vs 3.91 mg; $p<0.01$). Although both groups had a similar baseline SALT ($p=0.70$), pediatric patients achieved greater reductions in SALT at 3, 6, 9, and 12 months ($p<0.01$).Patients with a current episode duration longer than 8 years showed a poorer response to treatment at all follow-up points ($p<0.01$). Regarding safety, the only difference was a higher incidence of acne in patients under 18 years old (62.5% vs 8.3%; $p<0.01$).

Conclusions
Baricitinib appears to be an effective and safe alternative in pediatric patients with AA, with a faster response and at lower doses than in adults, but with a higher frequency of mild adverse effects. A prolonged duration of the episode could be considered a predictor of poorer response, although not a limitation for initiating treatment.

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Abstract N°: 896

APPLYCATION OF RITUXIMAB IN DERMATOLOGY: CHALLENGES AND CAUTIONS

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Introduction

Rituximab is a monoclonal anti-CD20 antibody on B-cells that is primarily used to treat certain cancers and some autoimmune diseases. Rituximab had been approved by FDA for some conditions including Non-Hodgkin's Lymphoma, Chronic Lymphocytic Leukemia, Rheumatoid Arthritis, Granulomatosis with Polyangiitis (GPA) (Wegener's Granulomatosis), Microscopic Polyangiitis (MPA) and Pemphigus Vulgaris. In this study, we focused on two groups of dermatologic conditions which Rituximab had been used on label and off-label, including autoimmune bullous and connective diseases.

Materials and Methods

This was a series report of more than 40 pemphigus patients and several cases of bullous pemphigoid and systemic sclerosis treated with rituximab. Clinical characteristics, treatment response and adverse events had been described in detail

Results

Overall, rituximab showed efficacy in pemphigus with nearly 80% of patients who achieved CR-on within 6 months and CR-off within 10 months. The prevalence of relapses after disease control was 34.21%. In systemic sclerosis, because limitations of case numbers so that the improvement was quite low and need more time of following up. The most common adverse event related to rituximab was infection, however there were some cases suffering from serious pneumonia that need hospitalization. Infusion reactions related to rituximab were mild and transient.

Conclusions

Rituximab has been shown efficacy expanded indications in dermatology due to direct action on B cells. Therefore, it had been used as the first line treatment of pemphigus and could be a potential modality for other skin diseases like bullous pemphigoid and systemic sclerosis. Moreover, treatment with rituximab could help to reduce long-term corticosteroid treament and maintain response. However, side effects related to rituximab treatment need to be cautioned by doing routine screening.

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Abstract N°: 976

Nanobody-Based Therapeutics in Inflammatory Skin Diseases: Emerging Clinical Targets and Future Directions

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Introduction
Nanobodies are an emerging class of single-domain antibody fragments with high specificity, small molecular size, and flexible multispecific design. These properties have positioned them as a promising next-generation biologic platform. Their potential role in inflammatory skin disease is gaining increasing attention in dermatology. In this study, we investigated the potential role of emerging nanobody-based therapeutics relevant to inflammatory skin diseases and assessed their clinical significance in dermatology practice.

Materials and Methods
A literature search was performed using PubMed/MEDLINE, Scopus and ClinicalTrials.gov databases. The following keywords were used individually and in combination: “nanobody,” “VHH,” “single-domain antibody,” “psoriasis,” “hidradenitis suppurativa,” “atopic dermatitis,” “systemic lupus erythematosus,” and “inflammatory skin diseases.”

Results
Current nanobody development in dermatology is centered on inflammatory pathways involving IL-17A/F, IL-13, thymic stromal lymphopoietin, TNF-α, OX40L, and IL-6R. Sonelokimab has demonstrated promising efficacy in psoriasis and hidradenitis suppurativa, supporting IL-17A/F blockade as a relevant nanobody-based strategy in skin inflammation. Lunsekimig and brivekimig illustrate the potential of multispecific nanobody constructs targeting complementary inflammatory pathways, particularly in atopic dermatitis and hidradenitis suppurativa. Beyond cytokine inhibition, nanobody technology offers theoretical advantages in tissue penetration and modular engineering. Nevertheless, available evidence in dermatology remains limited, and most agents are still in early clinical development.

Table 1. Nanobodies in several inflammatory disorders

Nanobody	Targeted antigens	Structure	Indication	ClinicalTrials.gov ID
Sonelokimab	IL-17A/F	Trivalent-bispecific	Psoriasis	NCT03384745
			HS	NCT05322473
			Psoriatic arthritis	NCT06641076
Lunsekimig	IL-13/TSLP	Pentavalent-bispecific	Atopic dermatitis	NCT06790121
			Asthma	NCT06102005
Brivekimig	TNF- α/OX40L	Pentavalent-bispecific	HS	NCT07170917
Vobarilizumab	IL-6R	Bivalent-bispecific	SLE	NCT02437890
			RA	NCT02518620
Ozoralizumab	TNF- α	Trivalent bispecific	RA	NCT04077567
Caplacizumab	vWF	Bivalent nanobody	TTP	NCT02553317

Table 1. Nanobodies in several inflammatory disorders

Conclusions

Nanobody-based therapeutics represent a rapidly evolving area in inflammatory dermatology, with early signals of potential benefit in psoriasis, hidradenitis suppurativa, and atopic dermatitis. Their small size, high specificity, and capacity for multispecific engineering support their promise as next-generation targeted biologic platforms. Although current evidence remains preliminary, further translational and clinical studies are necessary to better define their efficacy, safety, and future role in the treatment of inflammatory skin diseases.

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Abstract N°: 981

Geographic Variation in the Adoption of Biologic Therapies Among Dermatologists in the United States, 2018–2023

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Introduction

Biologic therapies have advanced the management of many dermatologic diseases through targeted modulation of specific inflammatory pathways.[1] A recent analysis reported increasing usage of biologics among dermatologists, but regional differences in adoption remain poorly understood.[2] As their use expands, evaluating regional patterns may provide insight into access and variations in clinical practice.

Materials and Methods

A cross-sectional analysis of 2018-2023 Medicare Part D data was completed to identify dermatologists who prescribed biologic therapies to Medicare beneficiaries. Each year, the proportion of biologic-prescribing dermatologists in each state was calculated by dividing number of biologic-prescribing dermatologists by total number of dermatologists in that state. States were then grouped by US census regions and regional proportions were calculated using the same approach. Empirical Bayes adjustment was applied to stabilize state-level prescribing proportions.

Results

From 2018 to 2023, the proportion of dermatologists prescribing biologics increased across all US regions. In 2018, prescribing rates ranged from 21% in the Northeast (550/2675) and West (613/2890), to 26% (1096/4231) in the South and 28% (624/2251) in the Midwest. By 2023, these proportions had increased to 44% (1185/2675) in the Northeast, 47% (1268/2890) in the West, 49% (2091/4231) in the South, and 53% (1192/2251) in the Midwest. Relative increases from 2018 to 2023 were 110% in the Northeast, 124% in the West, 88% in the South, and 89% in the Midwest.

Despite regional increases, variation in state-level adoption rates remained. In 2023, the adjusted proportion of dermatologists prescribing biologics ranged from 38% in Maryland and New York (lowest) to 62% in Indiana (highest), with several other states above 50%, including Wisconsin (59%) and Massachusetts (59%). Most states were between 45% and 55%, while several states remained below 40%, including New Jersey (39%), and Florida (39%). Overall, geographic variation in biologic prescription rates persist [Figure 1].

Tables

Table 1. Proportion of dermatologists prescribing biologics by U.S. region, 2018–2023.

Region	2018 (%)	2019 (%)	2020 (%)	2021 (%)	2022 (%)	2023 (%)	% Change (2018-2023)
Northeast	21	26	30	32	39	44	110
Midwest	28	34	38	42	50	53	89
South	26	32	36	40	46	49	88
West	21	28	32	36	41	47	124

Table 1 Legend: Adjusted proportion of dermatologists prescribing biologic therapies by U.S. region from 2018 to 2023.

Figures

Figure 1. Proportion of dermatologists prescribing biologic therapies by state in (A) 2018 and (B) 2023.

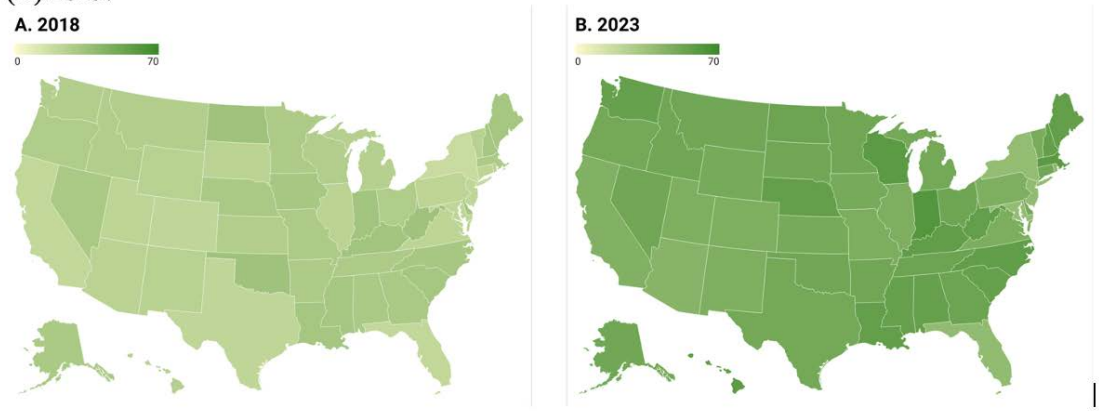

Figure 1 Legend. Adjusted proportion of dermatologists prescribing biologic therapies by state in (A) 2018 and (B) 2023.

Conclusions

The proportion of dermatologists prescribing biologics has risen across all geographic regions of the US. Prior literature has reported that early adoption of newer biologic therapies is more common among higher-volume, more experienced dermatologists and those practicing in urban settings.[3,4] Our data showed that, in contrast to rate of early adoption of biologics, prescribing behavior of dermatologists has become relatively consistent across geographic settings, and workforce size alone does not explain variation in utilization.

Differences in training, comfort with newer therapies, and frequency with which dermatologists are exposed to biologics are also factors that may contribute to variations in prescribing patterns. There are also system-level barriers to consider. Access to dermatologic care varies by region, influenced by proximity to specialty centers and dermatologist density.[5] In addition, insurance coverage further shapes treatment availability and utilization. These findings suggest that there is geographic variation in integration of biologic therapies into dermatologic practice. As the use of these therapies grows, understanding the factors leading to these regional differences may inform efforts to improve access to advanced dermatologic care.

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Abstract N°: 1003

The adoptability of an AI-supported dynamic treatment decision aid for adults with psoriasis

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Introduction
With a growing number of treatments available for psoriasis, shared decision-making (SDM) is increasingly important. Static clinical decision aids (DAs) offer evidence-based information to support SDM discussions, whilst dynamic DAs also offer personalised management options. Our primary aim was to determine the extent that UK dermatology clinicians support an artificial intelligence (AI)-supported dynamic biologics treatment DA for adults with psoriasis. Our secondary aim was to determine the structure of existing DAs in dermatology.

Materials and Methods
An anonymised electronic survey was circulated via the British Association of Dermatologists (BAD) and British Dermatological Nursing Group newsletters, the Scottish Dermatology Society, Irish Association of Dermatologists and the BAD National Trainee Committee Representatives. A scoping review of EMBASE and MEDLINE were undertaken in accordance with PRISMA guidance. From a total of 122 abstracts, 25 duplicates were removed. 97 abstracts were screened against inclusion and exclusion criteria and 42 articles were included for full text screening. 30 articles were included in the final analysis.

Results
186 complete responses were received from 175 responders between April and December 2025. In 11 cases of duplicate responses the later response was included in the analysis. 74.9% (131/175) expressed interest in trialing an AI-supported DA; 73.3% (96/131) and 72.5% (95/131) felt confident in discussing and prescribing biologics respectively. Of the 25.1% (44/175) who would not trial the DA, 40.9% (18/44) provided free text comments. The main themes were limited clinic time, the limitations of AI, and a lack of necessity. Our scoping review revealed that the existing literature mainly describes static rather than dynamic DAs; 76.6% (23/30) and 23.3% (7/30), respectively.

Conclusions
Regardless of pre-existing confidence levels, within the UK dermatology community, there is an appetite for a dynamic AI-supported DA for biologics treatments in adults with psoriasis, updated in real-time thereby ensuring long-term relevance.

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Abstract N°: 1005

Postoperative surgical site infection rate after Mohs surgery in patients on biologic therapy or JAK inhibitors: a retrospective analysis

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Introduction

The use of biologic therapy and Janus kinase inhibitors (JAKi) is becoming more common, but knowledge of their impact on surgical site infection (SSI) rates in Mohs surgery (MMS) is limited. This study aims to examine whether use of biologic therapy or JAKi affects rate of SSI in MMS.

	Total, N (%) N = 6737	Control, N (%) N = 3834	Biologics, N (%) N = 441	Immuno-suppressive, N (%) N = 2812	JAKi, N (%) N = 72
Age, mean (SD), years	68.6 (13.3)	68.4 (14.2)	68.5 (11.6)	68.6 (12)	67.2 (10.8)
Gender					
– Female	2796 (41.5)	1585 (41.34)	234 (53.06)	1155 (41.07)	22 (30.56)
– Male	3939 (58.47)	2249 (58.66)	207 (46.94)	1655 (58.85)	50 (69.44)
– Undefined	2 (0.03)	0 (0)	0 (0)	2 (0.07)	0 (0)
Current tobacco use	475 (7.05)	269 (7.02)	25 (5.67)	204 (7.25)	3 (4.17)
DM	1295 (19.22)	445 (11.61)	132 (29.93)	842 (29.94)	32 (44.44)
HIV	49 (0.73)	18 (0.47)	5 (1.13)	31 (1.1)	3 (4.17)
SOTR	948 (14.07)	24 (0.63)	127 (28.8)	924 (32.86)	52 (72.22)
Lymphoma	452 (6.71)	125 (3.26)	113 (25.62)	326 (11.59)	9 (12.5)
Tumor type					
– BCC	3236 (48.03)	2037 (53.13)	166 (37.64)	1147 (40.79)	18 (25)
– SCC	2711 (40.24)	1183 (30.86)	248 (56.24)	1497 (53.24)	53 (73.61)
– LM/MMIS/MM	652 (9.68)	520 (13.56)	25 (5.67)	125 (4.45)	1 (1.39)
– DFSP	23 (0.34)	20 (0.52)	0 (0)	3 (0.11)	0 (0)
– AFX	11 (0.16)	8 (0.21)	0 (0)	3 (0.11)	0 (0)
– Other	110 (1.63)	69 (1.8)	3 (0.68)	40 (1.42)	0 (0)
Number of stages, mean (SD)	1.2 (0.5)	1.2 (0.5)	1.1 (0.4)	1.2 (0.5)	1.2 (0.4)
Use of flap or graft	2267 (33.65)	1448 (37.77)	119 (26.98)	791 (28.13)	16 (22.22)
Secondary intention healing	271 (4.02)	151 (3.94)	20 (4.54)	119 (4.23)	5 (6.94)

AFX, atypical fibroxanthoma; BCC, basal cell carcinoma; DFSP, dermatofibrosarcoma protuberans; DM, diabetes mellitus; JAKi, Janus kinase inhibitor; LM, lentigo maligna; MM, malignant melanoma; MMis, melanoma in situ; SCC, squamous cell carcinoma; SD, standard deviation; SOTR, solid organ transplant recipient.

Table 1. Baseline characteristics of cohorts.

Materials and Methods

This retrospective study investigates SSI incidence in patients on biologic therapy vs non-biologic therapy treated

with MMS between January 1, 2019 and September 1, 2024 at a tertiary academic center. Patient and tumor characteristics, along with Mohs case details, were collected.

	N	SSI, N (%)	OR (95% CI)	OR p-value
Control (no drugs)	3834	77 (2.01)	-	-
Biologics total	441	16 (3.63)	1.84 (1.03, 3.09)	**0.03**
– Biologics only	86	4 (4.65)	2.38 (0.71, 5.9)	0.098
– Biologics + Immunosuppressive	339	11 (3.24)	1.64 (0.82, 2.98)	0.133
– Biologics + JAKi	2	0 (0)	0 (NA, 8.3e+37)	0.986
Current tobacco use				
– Control (no drugs)	269	4 (1.49)	-	-
– Biologics total	25	0 (0)	0 (NA, 1.51e+108)	>0.99
DM				
– Control (no drugs)	445	8 (1.8)	-	-
– Biologics total	132	6 (4.55)	2.6 (0.84, 7.62)	0.082
HIV				
– Control (no drugs)	18	0 (0)	-	-
– Biologics total	5	0 (0)	-	-
SOTR				
– Control (no drugs)	24	0 (0)	-	-
– Biologics total	127	8 (6.3)	21133849.79 (0, NA)	>0.99
Lymphoma				
– Control (no drugs)	125	7 (5.6)	-	-
– Biologics total	113	4 (3.54)	0.62 (0.16, 2.11)	0.453
Linear				
– Control (no drugs)	1981	28 (1.41)	-	-
– Biologics total	282	8 (2.84)	2.04 (0.86, 4.31)	0.08
Flap				
– Control (no drugs)	1321	33 (2.5)	-	-
– Biologics total	113	5 (4.42)	1.81 (0.61, 4.33)	0.227
Graft				
– Control (no drugs)	127	6 (4.72)	-	-
– Biologics total	6	0 (0)	0 (NA, 1.09e+83)	>0.99
Secondary intention healing				
– Control (no drugs)	151	6 (3.97)	-	-
– Biologics total	20	1 (5)	1.27 (0.07, 8.02)	0.828
1 stage				
– Control (no drugs)	3160	62 (1.96)	-	-
– Biologics total	381	15 (3.94)	2.05 (1.11, 3.54)	**0.014**
2 stages				
– Control (no drugs)	584	14 (2.4)	-	-
– Biologics total	54	1 (1.85)	0.77 (0.04, 3.94)	0.801
3 or more stages				
– Control (no drugs)	90	1 (1.11)	-	-
– Biologics total	6	0 (0)	0 (NA, Inf)	>0.99

SSI rate compared to control based on drug status, patient comorbidities, and Mohs case characteristics.

Results

6737 patients were included in this study. SSI rate in patients on biologic therapy was 3.63% vs 2.01% in the control group, with an odds ratio (OR) of 1.84 (95% CI = 1.03-3.09, p=0.03). Additional analysis of patients on biologics only, without concomitant immunosuppressive or JAKi therapy, showed an even higher SSI rate of 4.65% and OR of 2.38 (95% CI = 0.71-5.9, p=0.098), Patients on JAKi had a 5.56% SSI rate and OR of 2.87 (95% CI = 0.86-7.16, p=0.046).

	N	SSI, N (%)	OR (95% CI)	OR p-value
Control (no drugs)	3834	77 (2.01)		
Biologics total	441	16 (3.63)	1.84 (1.03, 3.09)	**0.03**
– Biologics only	86	4 (4.65)	2.38 (0.71, 5.9)	0.098
– Biologics + Immunosuppressive	339	11 (3.24)	1.64 (0.82, 2.98)	0.133
– Biologics + JAKi	2	0 (0)	0 (NA, 8.3e+37)	0.986
– Biologics + Immunosuppressive + JAKi	14	1 (7.14)	3.75 (0.21, 19.2)	0.205
– JAKi total	72	4 (5.56)	2.87 (0.86, 7.16)	**0.046**
– JAKi only	3	0 (0)	0 (NA, 1.73e+24)	0.983

Table 3. SSI rate compared to control based on drug status

Conclusions

These findings suggest that biologic therapy and JAKi use may be significant risk factors for SSI following MMS. As biologic therapy is becoming more prevalent, dermatologic surgeons should take this information into consideration when evaluating and counseling patients for MMS preoperatively.

	N	SSI, N (%)	OR (95% CI)	OR p-value
Control (no drugs)	3834	77 (2.01)		
Biologics total	441	16 (3.63)	1.84 (1.03, 3.09)	**0.03**
– Biologics only	86	4 (4.65)	2.38 (0.71, 5.9)	0.098
– Biologics + Immunosuppressive	339	11 (3.24)	1.64 (0.82, 2.98)	0.133
– Biologics + JAKi	2	0 (0)	0 (NA, 8.3e+37)	0.986
– Biologics + Immunosuppressive + JAKi	14	1 (7.14)	3.75 (0.21, 19.2)	0.205
– JAKi total	72	4 (5.56)	2.87 (0.86, 7.16)	**0.046**
– JAKi only	3	0 (0)	0 (NA, 1.73e+24)	0.983

Table 4. SSI rate in each drug group based on patient characteristics and comorbidities, tumor characteristics, and Mohs case details.

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Abstract N°: 1009

Real-world treatment patterns and effectiveness of abrocitinib in atopic dermatitis: Interim results on Abrocitinib Chinese rEgistry on AD (AHEAD)

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Introduction
Atopic dermatitis (AD) is a chronic inflammatory skin disease that significantly impairs quality of life. Although the Janus kinase (JAK) inhibitor abrocitinib has shown efficacy in moderate-to-severe AD in clinical trials, real-world evidence in Chinese patients is limited. The ongoing prospective, multicenter Abrocitinib Chinese Registry on AD (AHEAD) across 42 sites in China. After previous interim analyses focused mainly on demographics and both short and long-term treatment patterns of abrocitinib in Chinese AD patients, a third interim analysis of the AHEAD data was performed to detail the treatment patterns and effectiveness of abrocitinib during extended follow-up.

Materials and Methods
Adults (≥18 years) who were abrocitinib-naïve or had discontinued abrocitinib for >30 days were eligible. The index date was the first abrocitinib prescription during the study. Data collected at routine visits (weeks 2, 12, 24, 52, and 64) included demographics, treatment patterns, and AD outcomes, such as flare-ups, Investigator's Global Assessment (IGA), Eczema Area and Severity Index (EASI), Peak Pruritus Numerical Rating Scale (PP-NRS), and other patient- or physician-reported measures. Subgroups of interest included prior biologic use, initiation dose, dose titration pattern, and early response of EASI-90 and PP-NRS.

Results
From 18 Oct 2023 to 10 Jan 2026, a total of 1275 AD patients were enrolled with a mean (SD) follow-up of 260.3 (186.7) days. The mean (SD) age of all participants was 43.2 (16.3), and 48.0% were female. In all 1275 patients with records of medication history, 232 (18.2%) had received at least one form of systemic treatment, among whom, 107 (46.1%) received traditional treatments, 90 (38.8%) received biologics, and 64 (27.6%) received small-molecule drugs. 1117 (88.4%) patients started with 100 mg daily abrocitinib, while 147 (11.6%) took 200 mg daily. 695 (54.5%) patients underwent abrocitinib dose adjustment. Among them, 45.3% decreased the dose, 17.1% increased the dose, and 67.6% discontinued abrocitinib treatment. For patients who increased the dose from 100 mg to 200 mg daily (n=65), 36.9% were female, with a mean age of 42.4 years (SD: 15.9), and 21.5% had received prior systemic treatment. The median time for patients increased daily dose from 100 mg to 200 mg was 30.5 days. As of the cutoff date, 1178 (92.4%) of patients had good adherence (proportion of days covered [PDC] ≥ 0.8) to abrocitinib. IGA success rates rose from 15.4% (week 2) to 56.0% (week 64). EASI-75 responses increased from 24.7% to 78.0%, and EASI-90 from 11.0% to 61.3%. PP-NRS 4 rose from 57.4% to 74.3% while PP-NRS 0/1 increased from 28.1% to 47.7% over the same period. Changes in IGA, EASI, PP-NRS, and other AD clinical outcomes from baseline during the follow-up were presented in Table 1. For subgroups, EASI-90 response increased from 11.0% to 62.8%, and PP-NRS 4 increased from 58.1% to 76.4% for patients with no prior systematic treatment, and from 10.8% to 56.4% and 54.5% to 66.3%, respectively, with prior systematic treatment (week 2 to week 64). Among patients achieving EASI-90 and PP-NRS 0/1 response within 12 weeks, 63.0% retained PP-NRS 0/1 at week 64, and 71.3% retained EASI-90. The mean EASI change from baseline increased from -8.5 to -16.2, and mean PP-NRS change from baseline increased from -4.1 to -5.4 with 200 mg initiation, and from -6.5 to -10.5 and -3.6 to -4.7, respectively, with 100 mg initiation (week 2 to week 64).

		Baseline	Week 2	Week 4	Week 12	Week 24	Week 52	Week 64
	N	1249	963	924	801	683	437	420
IGA	Mean (SD)	3.0 (0.6)	2.2 (0.9)	1.8 (0.9)	1.6 (0.9)	1.4 (0.9)	1.3 (1.0)	1.2 (1.0)
	Change from baseline, Mean (SD)	-	-0.8 (0.9)	-1.2 (0.9)	-1.5 (1.0)	-1.7 (1.0)	-1.7 (1.1)	-1.9 (1.1)
	N	1248	963	925	801	683	437	421
EASI	Mean (SD)	13.5 (12.0)	7.0 (7.6)	4.8 (5.9)	3.2 (4.5)	2.3 (4.4)	2.1 (3.4)	1.8 (3.9)
	Change from baseline, Mean (SD)	-	-6.7 (9.3)	-8.8 (10.0)	-10.2 (10.6)	-11.0 (10.6)	-10.6 (10.1)	-11.3 (10.8)
	N	1248	968	933	805	683	450	428
PP-NRS	Mean (SD)	6.7 (2.4)	3.1 (2.4)	2.7 (2.2)	2.6 (2.3)	2.5 (2.2)	2.4 (2.1)	2.1 (2.1)
	Change from baseline, Mean (SD)	-	-3.6 (3.0)	-4.1 (2.9)	-4.1 (3.1)	-4.3 (3.0)	-4.2 (3.0)	-4.7 (3.0)
	N	1246	961	921	798	679	436	417
SCORAD	Mean (SD)	50.3 (16.5)	29.3 (15.6)	23.1 (13.9)	19.0 (14.1)	16.4 (13.5)	15.4 (12.9)	13.5 (13.3)
	Change from baseline, Mean (SD)	-	-21.3 (15.7)	-27.1 (16.7)	-31.1 (17.7)	-33.9 (18.6)	-33.3 (19.0)	-36.6 (19.8)
	N	1248	967	930	805	684	450	428
PtGA	Mean (SD)	3.3 (0.5)	2.6 (0.8)	2.3 (0.9)	2.2 (0.9)	2.1 (1.0)	2.0 (1.0)	1.8 (1.0)
	Change from baseline, Mean (SD)	-	-0.7 (0.9)	-1.0 (1.0)	-1.2 (1.0)	-1.2 (1.1)	-1.3 (1.1)	-1.5 (1.2)
	N	1248	966	931	805	684	450	427
POEM	Mean (SD)	15.9 (7.0)	7.9 (6.2)	6.1 (5.5)	5.9 (5.8)	5.8 (5.4)	5.9 (5.7)	4.9 (5.6)
	Change from baseline, Mean (SD)	-	-8.3 (7.0)	-10.1 (7.4)	-10.2 (8.0)	-10.6 (7.9)	-10.0 (8.0)	-11.0 (8.1)
	N	1249	967	931	805	684	450	427
ADCT	Mean (SD)	13.8 (5.4)	6.4 (4.7)	5.1 (4.2)	5.0 (4.4)	4.7 (4.3)	4.6 (4.0)	3.8 (4.0)
	Change from baseline, Mean (SD)	-	-7.6 (5.7)	-8.9 (5.9)	-9.1 (6.5)	-9.4 (6.4)	-9.3 (6.4)	-10.3 (6.3)
	N	1248	966	931	805	684	450	427
DLQI	Mean (SD)	11.7 (7.4)	6.5 (5.9)	5.2 (5.5)	4.7 (5.3)	4.5 (5.4)	4.2 (4.7)	3.4 (4.6)
	Change from baseline, Mean (SD)	-	-5.6 (6.3)	-6.9 (6.7)	-7.7 (7.3)	-8.2 (7.3)	-8.3 (7.4)	-9.1 (7.4)

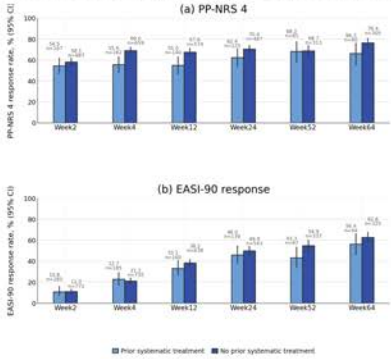
IGA: Investigator's Global Assessment; EASI: Eczema Area and Severity Index; PP-NRS: Peak Pruritus Numerical Rating Scale; SCORAD: Scoring Atopic Dermatitis; PtGA: Patient Global Assessment; POEM: Patient-Oriented Eczema Measure; ADCT: Atopic Dermatitis Control Tool; DLQI: Dermatology Life Quality Index

Table 1. AD clinical outcomes in patients receiving Abrocitinib at baseline and during follow-up

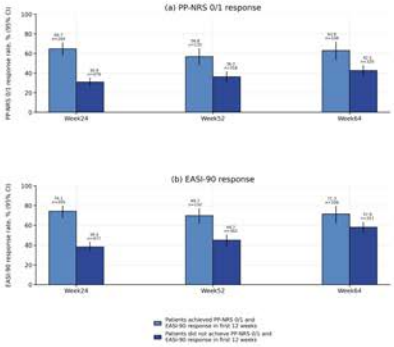
Conclusions

This interim analysis demonstrated that abrocitinib consistently improved AD clinical outcomes during 64 weeks of follow-up. Patients had no prior systematic treatment, receiving starting dose of 200 mg daily, and patients achieved high response in the first 12 weeks showed more improvement in EASI and PP-NRS by 64 weeks of follow-up (Figure 1).

I. Changes in AD clinical outcomes by patients' prior systematic treatment status



II. Changes in AD clinical outcomes by patients' achievement status of PP-NRS 0/1 and EASI-90 response in the first 12 weeks



III. Changes in AD clinical outcomes by treatment starting dose

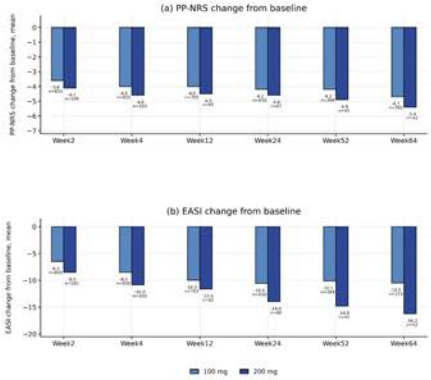


Figure 1. Changes in AD clinical outcomes during follow-up by subgroups of interest. Abbreviations: EASI, Eczema Area and Severity Index; PP-NRS, Peak Pruritus Numerical Rating Scale

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Cost Effectiveness Analysis of Biological Drugs Targeting IL-17 Pathway in Plaque Psoriasis From the Perspective of the Brazilian Private Healthcare System

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Introduction

Plaque psoriasis is a chronic, immune-mediated systemic disease with a 1.31% prevalence in Brazil, causing physical and psychosocial burdens comparable to cancer or heart disease.Targeting the TNF/IL-23/IL-17 axis allows for high efficacy and complete lesion clearance (PASI 100), yet long-term therapy is frequently interrupted by efficacy loss, adverse events, or financial barriers. The IL-17 pathway is considered a therapeutic target that delivers rapid clinical responses and complete clearance in patients with psoriasis. In the Brazilian market, there are currently four molecules available for managing the disease: bimekizumab, brodalumab, ixekizumab, and secukinumab. This study evaluates the cost-effectiveness of IL-17 pathway inhibitors within the Brazilian supplementary health system over a one-year period aiming to support sustainable access to high-efficacy therapies. Objectives: Evaluate the cost-effectiveness of all anti-IL17 immunobiologics available for treating moderate to severe plaque psoriasis in adults within Brazil's Supplemental Health System (ANS Rol) and compare the costs associated with achieving PASI 90 and PASI 100 responses across IL-17 inhibitors:, bimekizumab, brodalumab, ixekizumab and secukinumab.

Materials and Methods

The therapeutic agents included in this comparative study were biological medications targeting the interleukin-17 (IL-17) pathway, available for the treatment of moderate-to-severe plaque psoriasis in adults within the Brazilian supplementary healthcare system. A cost-per-responder analysis was performed using an economic health calculator. Efficacy, defined as clear (PASI 100) or almost clear skin (PASI 90), was derived from network meta-analysis data. Drug prices were extracted from CMED (Brazilian Drug Market Regulation Chamber) PF 18% (cutoff: August 2025[1]Table 1) and dosages were based on Brazilian labels. The analysis excluded costs for administration, monitoring and adverse events and dose adjustments. These results reflect the first year of treatment.

TABLE 1: PRICE PER PACKAGE AND PER UNIT

MOLECULE	MODE OF ACTION	PACK PRICE (REAIS – R$)	UNITS PER PACK	PRICE PER UNIT (REAIS – R$)
BIMEKIZUMAB	ANTI IL-17A and F	R$ 5.978,51	1	R$ 5.978,51
BRODALUMAB	ANTI IL-17RA	R$ 6.670,83	2	R$ 3.335,42
IXEKIZUMAB	ANTI IL-17A	R$ 7.100,78	1	R$ 7.100,78
SECUKINUMAB	ANTI IL-17A	R$ 9.061,67	1	R$ 9.061,67

Table 1: Price per Package and Per Unit

Results

Brodalumab showed the lowest cost per responder among anti-IL-17 biologics for PASI 90 and PASI 100 outcomes at the end of one year of treatment. For PASI 90, costs (in BR$) were R$114,575 for brodalumab, R$135,533 for bimekizumab, R$167,657 for ixekizumab, and R$209,537 for secukinumab (Figure 2). For PASI 100, the estimated costs (in BR$) were R$160,528 for brodalumab, R$187,807 for bimekizumab, R$252,538 for ixekizumab, and R$335,867 for secukinumab. The Brazilian Psoriasis Consensus establishes PASI 90 as the therapeutic goal, in line with other global guidelines and aligned with the treat-to-target approach.



Chart 1 & 2: Cost Per Responder in Reais (R$) for First Year of Treatment for PASI 90 and PASI 100, respectively

Conclusions

Immunobiologic therapies are effective and safe for the management of moderate-to-severe psoriasis, with IL-17 inhibitors representing an important treatment class. In this analysis, brodalumab was associated with the most favorable cost-effectiveness profile among IL-17 inhibitors. Within the context of the Brazilian Supplementary Health System, where resources are limited, economic evaluations of high-cost treatments can support managerial decision-making, particularly in the management of moderate-to-severe plaque psoriasis, for which multiple therapeutic options are available.

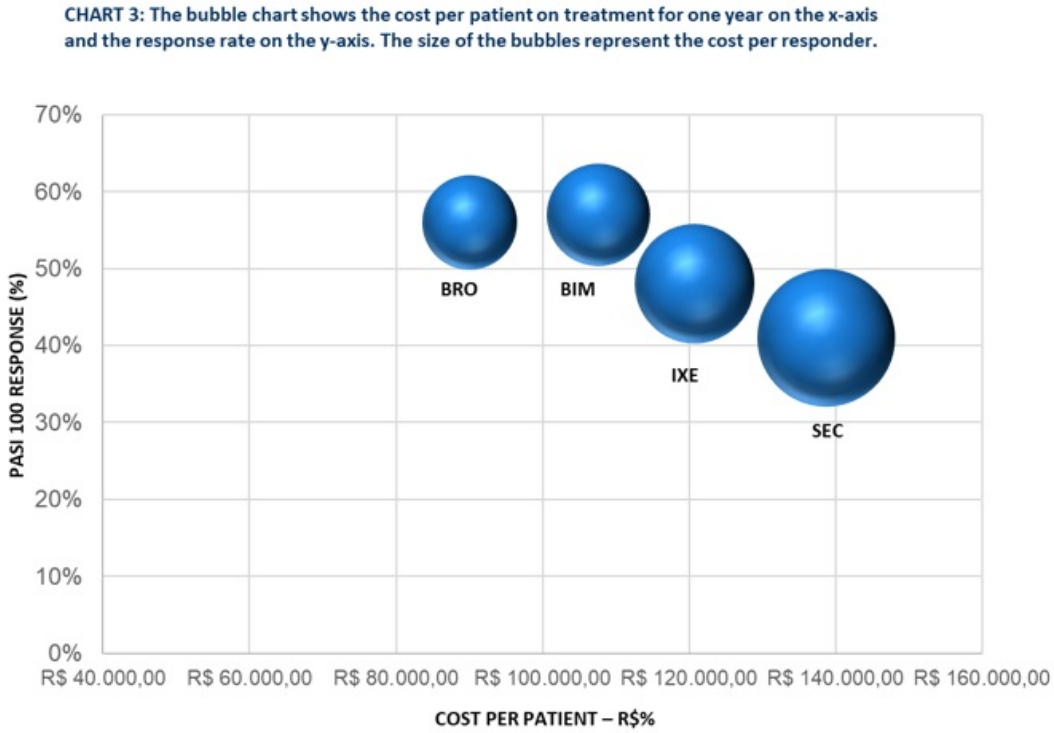

Chart 3: The bubble chart shows the cost per patient on treatment for one year on the x-axis and the response rate on the y-axis. The size of the bubbles represent the cost per responder.

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Epithelial-Derived IL-17C and IL-17E as Psoriatic Inflammation Amplifiers and Targets

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Introduction

Psoriasis (PsO) is a chronic immune-mediated disease driven by complex cytokine networks. The interleukin-17 (IL-17) family plays a central role in keratinocyte hyperproliferation and inflammation. While IL-17A, IL-17A/F, and IL-17F are well-established drivers, emerging evidence highlights important roles for IL-17C and IL-17E.

Materials and Methods

This is a narrative review of experimental, translational, and clinical studies addressing the roles of IL-17C and IL-17E in psoriasis. Literature searches were conducted in Medline via PubMed and SciELO databases. Articles published up to July 2025 were considered. Search terms included combinations of psoriasis, IL-17C, IL-17E (IL-25), keratinocytes, Th17, and inflammation. Studies were selected based on relevance to cytokine biology, signaling pathways, epidermal effects, and therapeutic implications. Evidence from in vitro studies, animal models, ex vivo human skin, and clinical observations was narratively synthesized.

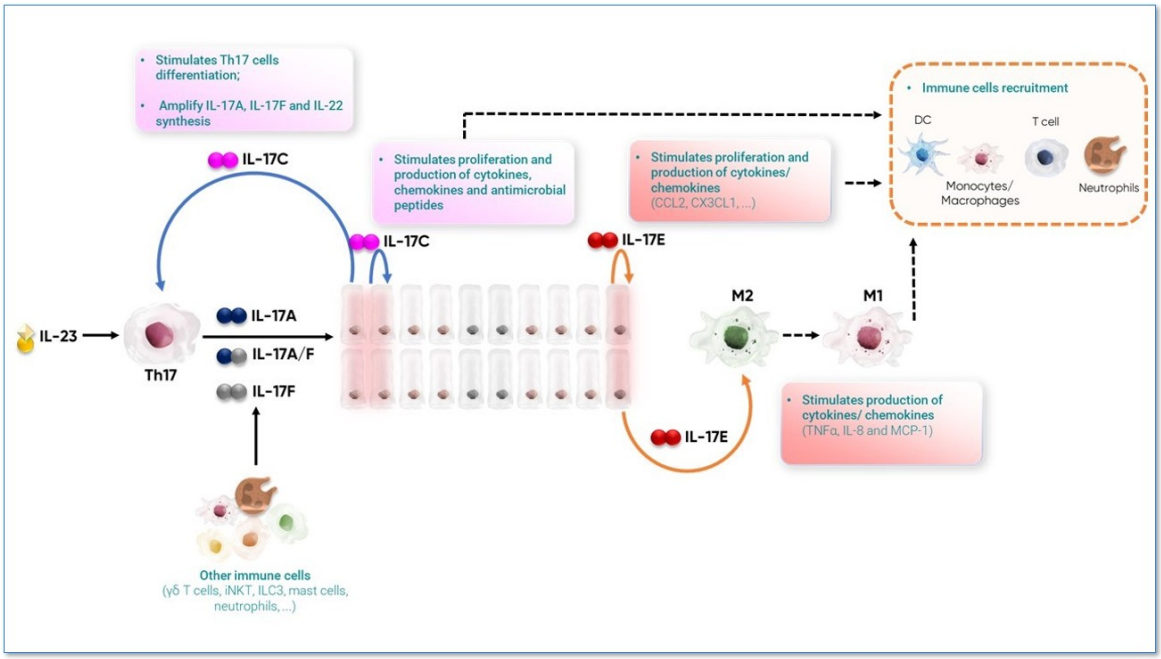

Figure 1. Role of IL-17C and IL-17E in psoriasis. Both cytokines drive keratinocyte proliferation and inflammatory mediator release. IL-17C enhances Th17 and antimicrobial pathways, whereas IL-17E promotes macrophage-mediated inflammation.

Results

IL-17C is predominantly produced by keratinocytes and is highly expressed in psoriatic lesions. It signals through the IL-17RA/IL-17RE receptor complex via Act1-dependent pathways (Fig. 2). IL-17C promotes keratinocyte proliferation, antimicrobial peptide production, and pro-inflammatory cytokine release. A reciprocal amplification loop exists between IL-17C and Th17-derived IL-17A . IL-17C is the most abundantly expressed IL-17 family cytokine in psoriatic plaques and Overexpression in keratinocytes induce psoriasiform skin inflammation in animal models (Fig. 1).IL-17C acts synergistically with tumor necrosis factor-alpha (TNF-α), amplifying epithelial and endothelial inflammation, and its neutralization reduces inflammatory mediators in ex vivo human psoriatic skin. IL-17E is structurally distinct from IL-17A and was traditionally linked to type 2 immune responses and keratinocytes are both a source and a target of IL-17E in the skin. IL-17E signals through the IL-17RA/IL-17RB receptor complex, activating Act1, TRAF, NF-κB, MAPK, and JAK/STAT pathways (Fig. 2). IL-17E promotes keratinocyte proliferation and epidermal thickening via mammalian target of rapamycin (mTOR) activation. Serum IL-17E levels are elevated in patients with PsO and are further increased in erythrodermic psoriasis, where they correlate with disease activity. In contrast, studies evaluating IL-17E mRNA and protein expression in psoriatic lesions report heterogeneous findings, with most demonstrating increased expression and some reporting no significant difference. IL-17E contributes to immune cell recruitment, particularly neutrophils and macrophages. Keratinocyte-derived IL-17E activates dermal macrophages, enhancing pro-inflammatory cytokine production (Fig. 1).IL-17E-induced inflammation occurs independently of IL-17A signaling (Fig. 2)

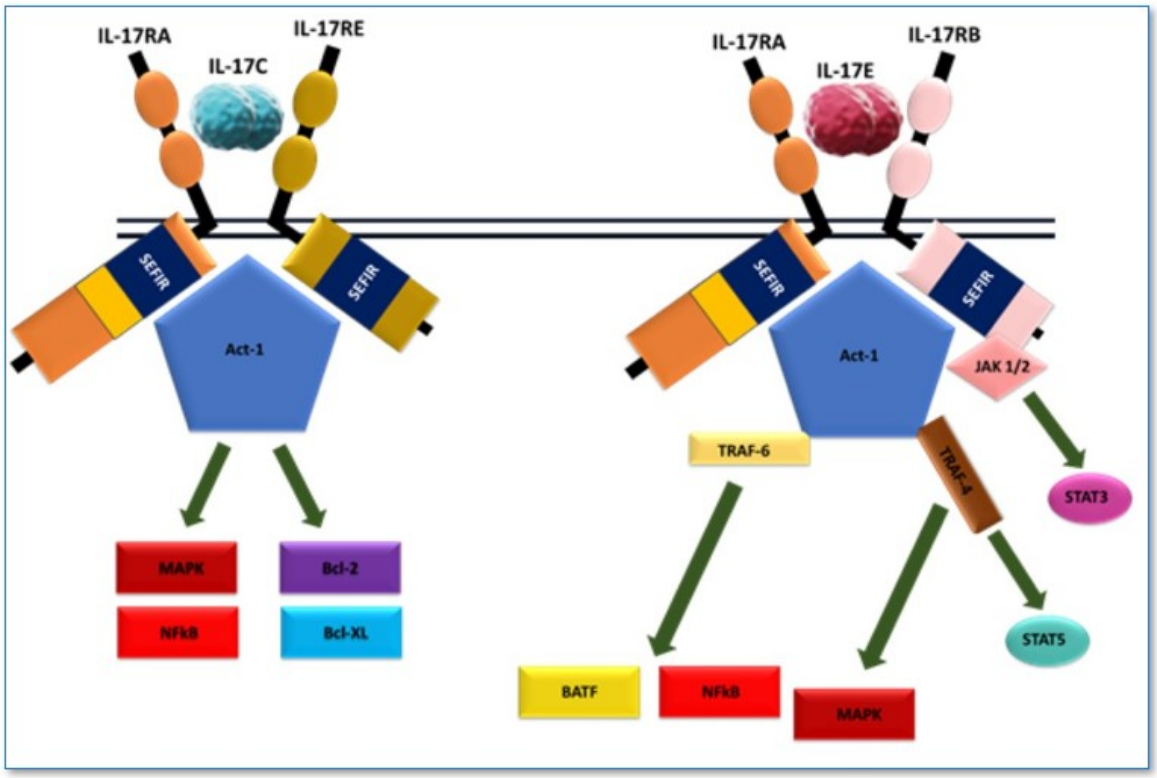

Figure 2. Signaling pathways of IL-17C and IL-17E (IL-25). IL-17C signals through the IL-17RA/RE–Act1 axis, activating NF-κB and MAPK pathways, whereas IL-17E engages Act1–TRAF-dependent signaling, including NF-κB, MAPK, BATF, and JAK/STAT pathways.

Conclusions

IL-17C and IL-17E expand the pathogenic framework of psoriasis beyond Th17 lymphocytes, highlighting additional cytokine circuits in disease maintenance. Both cytokines exert autocrine and paracrine effects, acting on keratinocytes and modulating immune cell responses within the skin.These findings emphasize keratinocytes as active immune effector cells rather than passive inflammatory targets. IL-17C reinforces epithelial inflammation and amplifies Th17 responses through a reciprocal feedback loop, in which Th17-derived IL-17A induces keratinocyte IL-17C, and IL-17C promotes Th17 differentiation and IL-17A production. IL-17E links epidermal dysfunction with innate immune activation, particularly macrophage and neutrophil recruitment and proinflammatory cytokine release. Beyond IL-17A, psoriasis pathogenesis involves additional IL-17 family

members, including the keratinocyte-derived cytokines IL-17C and IL-17E, which signal through distinct but overlapping pathways. Targeting IL-17C or IL-17E, directly or indirectly, offers a rational strategy to expand therapeutic options beyond IL-17A/F and IL-23.

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When “BRAF wild-type” is not the end of the story: metastatic melanoma with a non-V600 BRAF mutation detected by next-generation sequencing

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Introduction

The management of metastatic melanoma has undergone a paradigm shift with the introduction of immune checkpoint inhibitors and BRAF/MEK-targeted therapies. Despite these advances, a significant proportion of patients experience either primary resistance or severe toxicity, making therapeutic sequencing particularly challenging. Moreover, while most treatment algorithms focus on BRAF V600 mutations, non-V600 BRAF alterations remain underrecognized and are often missed by standard testing. In this setting, next-generation sequencing (NGS) can uncover actionable mutations and open additional therapeutic avenues, with potential impact on clinical outcomes.

Case Presentation

A 59-year-old woman with an ECOG performance status of 0 was initially diagnosed with stage IIB dorsal melanoma (pT4aN0M0) and remained disease-free on regular follow-up for approximately six years. She subsequently developed a subcutaneous metastasis in the left gluteal region, which was completely excised and histopathologically confirmed as melanoma, with BRAF wild-type status. In the setting of completely resected stage IV disease, adjuvant nivolumab was initiated.

During treatment, she developed severe immune-related adverse events, including an extensive grade 3 psoriasiform eruption and grade 4 immune-mediated colitis, requiring systemic corticosteroids and discontinuation of nivolumab. At the same time, PET imaging documented locoregional and distant progression, with bilateral pulmonary and cutaneous metastases. Given the combination of primary resistance and substantial toxicity, nivolumab was permanently discontinued and next-generation sequencing (NGS) was requested.

Three months later, restaging revealed rapidly progressive nodal, pulmonary, hepatic, and bone metastases. Owing to the aggressive disease course, off-label ipilimumab 1 mg/kg plus pembrolizumab 200 mg was initiated after multidisciplinary discussion, despite the recognized risk of colitis reactivation. However, shortly after the second cycle, the patient developed recurrent immune-mediated colitis requiring high-dose corticosteroids, leading to treatment discontinuation. Her clinical condition deteriorated to ECOG 2, and repeat PET confirmed further disease progression.

NGS subsequently identified a Tier II-C BRAF c.1794_1796dupTAC (p.Thr599dup) variant, a non-V600 alteration. On the basis of this finding, targeted therapy with dabrafenib plus trametinib was started. While still tapering corticosteroids, the patient developed pyrexia, myalgias, arthralgias, and severe rhabdomyolysis with renal failure, requiring hospitalization. As toxicity recurred despite temporary interruption and dose reduction, treatment was switched within class to encorafenib plus binimetinib.

Approximately three months after the treatment switch, the patient was asymptomatic and PET showed no metabolically active disease. Three years after recurrence, and despite having at one point been considered for best supportive care alone, she remains in complete response on full-dose encorafenib plus binimetinib.

Conclusion

This case highlights the complexity of therapeutic sequencing in metastatic melanoma and underscores the value of NGS in clarifying the true molecular profile of tumors initially classified as BRAF wild-type. It also illustrates how identification of an actionable non-V600 BRAF mutation may be decisive for treatment selection, and how an intra-class switch between BRAF/MEK inhibitors may allow sustained efficacy despite major toxicity with the initial regimen.

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Abstract N°: 1155

Bimekizumab time to meaningful improvements in clinical and patient-reported outcomes in patients with psoriatic arthritis: Results from a post hoc analysis of two pooled phase 3 studies

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Introduction

Psoriatic arthritis (PsA) is a chronic, inflammatory disease that manifests across multiple domains including joints, skin, nails, tendons, bones, enthesitis and dactylitis, which substantially impacts patient quality of life.[1,2] Bimekizumab (BKZ), a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated sustained and consistent efficacy and safety up to 3 years in patients with active PsA who were biological disease-modifying antirheumatic drug (bDMARD)-naïve or had prior inadequate response or intolerance to tumour necrosis factor inhibitors (TNFi-IR).[3,4]

Here, the median time to clinically meaningful improvement in efficacy responses, including composite endpoints (American College of Rheumatology score [ACR], minimal disease activity [MDA]) and patient-reported outcomes (PROs) in BKZ-treated PsA patients, was assessed using pooled data from two phase 3 studies.

Materials and Methods

BE OPTIMAL (NCT03895203; bDMARD-naïve) and BE COMPLETE (NCT03896581; TNFi-IR) assessed the efficacy and safety of subcutaneous BKZ 160mg every 4 weeks (wks; Q4W) in patients with PsA and placebo (PBO)-controlled to Wk16, then switched to BKZ (PBO/BKZ). Completers entered BE VITAL (NCT04009499; open-label extension); all patients received BKZ. BE OPTIMAL included a subcutaneous adalimumab 40 mg Q2W reference arm; data not reported here.

In this post hoc analysis, data were pooled and reported by treatment group at randomisation or as BKZ Total

(PBO/BKZ and BKZ groups combined) to Year 3 (pooled Week 160/156 BE OPTIMAL/BE COMPLETE data) as observed case. Kaplan-Meier estimated median time to initial achievement/probability of achieving minimal clinically important improvements (MCII) or higher improvements in clinical outcomes and PROs by Year 3. Time to response was calculated from the time of first study treatment for PBO/BKZ and BKZ groups, and from time of first BKZ treatment for BKZ Total. Only patients who did not meet the target minimal clinically important threshold for an outcome at baseline were included, or from time of first BKZ treatment in the BKZ Total group. Time to MCII outcomes and higher clinical outcomes were reported, list of outcomes can be found in **Table.**

Table. Median time to the achievement of minimal clinically important or meaningful improvements in joint, skin, composite and patient-reported outcomes

	Pooled BE OPTIMAL and BE COMPLETE[a] (bDMARD-naïve and TNFi-IR)					
	PBO/BKZ 160 mg Q4W n=414		BKZ 160 mg Q4W n=698		BKZ 160 mg Q4W Total[b] n=1,112	
	Median time to response weeks (95% CI)[c]	Included patients n	Median time to response weeks (95% CI)[c]	Included patients n	Median time to response weeks (95% CI)[d]	Included patients n
Minimal Clinically Important Improvement						
ACR20	20.1 (20.1, 20.3)	410	8.0 (4.9, 8.1)	698	8.1 (NE, NE)	931
SJC≤1[e]	20.3 (20.0, 24.0)	413	8.4 (8.1, 11.6)	698	11.9 (8.4, 12.1)	927
PASI75[f]	24.1 (20.3, 24.3)	228	4.9 (4.3, 8.0)	393	8.0 (5.3, 8.1)	575
Pain30	16.6 (12.4, 20.1)	410	7.9 (4.3, 8.1)	697	8.1 (NE, NE)	890
HAQ-DI MCID[g]	20.1 (16.1, 24.0)	327	8.1 (8.1, 11.9)	549	12.1 (11.7, 12.1)	711
FACIT-Fatigue MCID[h]	16.9 (16.1, 24.1)	375	12.6 (12.0, 16.0)	631	16.1 (16.0, 16.3)	808
PsAID-12 MCID[i]	52.1 (40.6, 52.9)	295	24.0 (16.6, 24.6)	492	25.0 (24.1, 36.1)	743
High Treatment Targets						
ACR50[j]	29.6 (28.1, 36.0)	410	16.1 (14.0, 16.3)	698	16.1 (16.0, 19.9)	1,032
SJC=0[e]	27.7 (24.1, 28.1)	413	12.1 (12.1, 12.4)	698	12.3 (12.1, 12.6)	980
PASI100[f]	29.3 (28.3, 36.0)	228	15.7 (12.1, 16.1)	393	15.9 (12.4, 16.1)	598
MDA	36.4 (36.0, 44.1)	405	16.1 (16.0, 19.9)	682	20.1 (19.9, 24.0)	1,005
VLDA	136.3 (104.0, 156.3)	413	111.1 (88.1, 147.4)	698	113.1 (96.1, 136.3)	1,079
Pain50[k]	28.0 (24.0, 28.1)	410	8.9 (8.3, 12.1)	697	12.1 (12.1, 16.0)	952
Pain70[k]	40.3 (32.4, 52.0)	410	21.0 (16.6, 28.1)	697	28.1 (24.1, 32.3)	1,016
PsAID-12 LDA/REM[l]	40.1 (39.7, 41.7)	352	16.3 (16.1, 16.9)	589	24.1 (20.3, 24.1)	852

Randomised set from BE OPTIMAL and BE COMPLETE (including data from BE VITAL). Time to response presented as median weeks to response. Patients achieving target threshold prior to or at baseline were not included. [a] Data were pooled for all PBO/BKZ, BKZ-randomised and BKZ Total group patients who were bDMARD-naïve or TNFi-IR; [b] BKZ Total group includes BKZ-randomised patients and PBO patients who switched to BKZ at Week 16. Patients achieving threshold while receiving placebo are not included in the Total BKZ time to event estimation; [c] Time to response = Date of response – Date of first placebo/BKZ treatment + 1; [d] Time to response is calculated from time of first BKZ treatment regardless of treatment group; [e] SJC≤1 and SJC=0 were assessed in 66 joints; [f] In patients with psoriasis affecting ≥3% BSA at baseline; [g] HAQ-DI MCID defined as decrease from baseline ≥0.35; [h] FACIT-Fatigue MCID defined as score increase from baseline ≥4 in patients scoring ≤48 at baseline; [i] Defined as PsAID-12 decrease of ≥3; PsAID-12 total scores range from 0–10; higher scores indicate worse status; [j] ACR50 at Week 16 was the primary endpoint of BE OPTIMAL and BE COMPLETE;3,4 [k] Pain VAS ≥50% and ≥70% improvement represents substantial improvements in patient-reported pain;5 [l] Defined as PsAID-12 total score ≤1.95. Efficacy in MCII

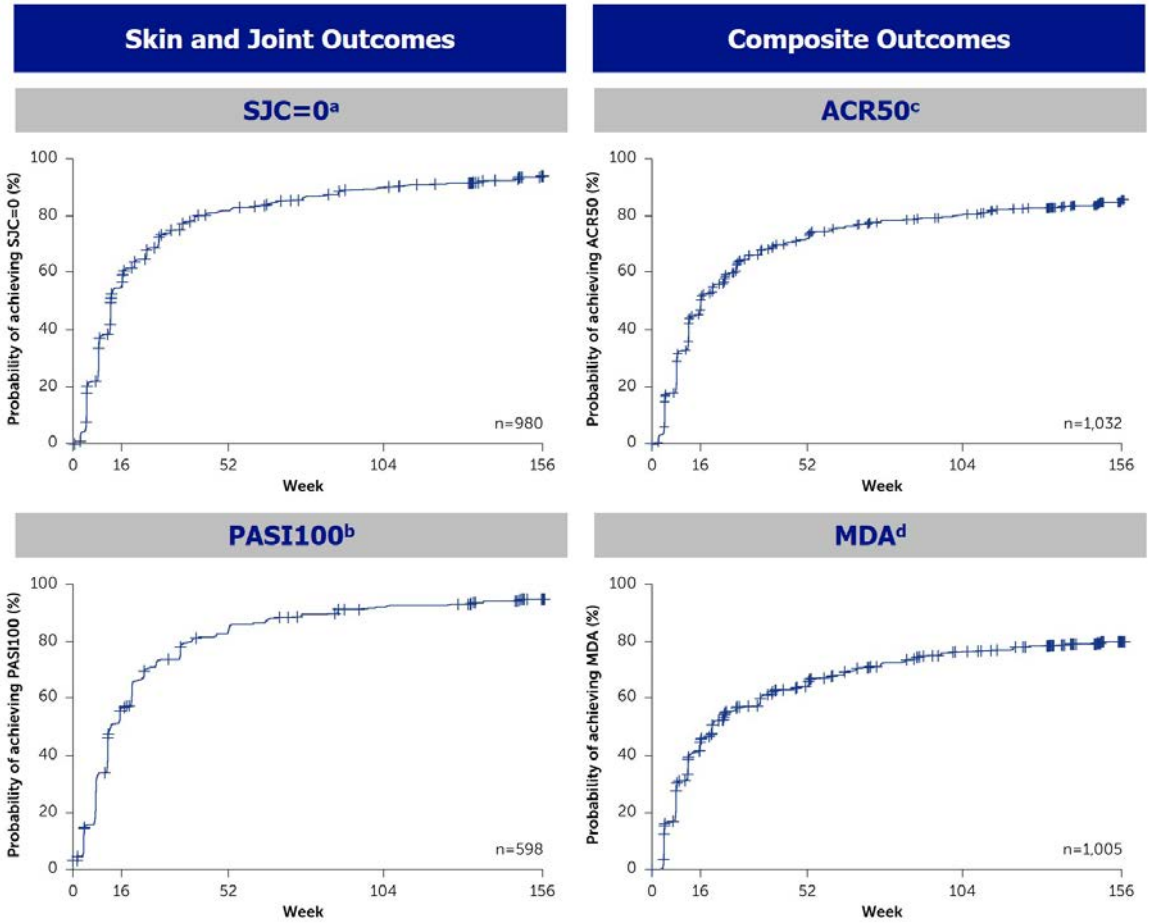
outcomes reported include median time to ACR20, SJC≤1 and PASI75.6 Efficacy in higher clinical outcomes were reported, including median time to achieve ACR50/SJC resolution (SJC=0)/PASI100/MDA/very low disease activity (VLDA). PROs MCIIs reported include median time to achieve Pain visual analogue scale (VAS)/30% improvement (Pain30)/Health Assessment Questionnaire-Disability Index (HAQ-DI)/minimal clinically important difference (MCID, ≥0.35-point decrease)/Functional Assessment of Chronic Illness Therapy (FACIT)-Fatigue MCID (≥4-point increase in patients scoring ≤48 at baseline)/PsA Impact of Disease 12-item (PsAID-12) total score MCID (≥3-point decrease). Higher meaningful PROs reported include Pain VAS 50%/70% improvement (Pain50/70) and PsAID-12 low disease activity/remission (LDA/REM, score ≤1.95). ACR20/50: ≥20%/50% improvement from baseline in American College of Rheumatology response criteria; bDMARD: biological disease-modifying antirheumatic drug; BKZ: bimekizumab; BSA: body surface area; CI: confidence interval; FACIT: Functional Assessment of Chronic Illness Therapy; HAQ-DI: Health Assessment Questionnaire-Disability Index; LDA/REM: low disease activity/remission; MCID: minimal clinically important difference; MCII: minimal clinically important improvement; MDA: minimal disease activity; NE: not estimable; Pain30/50/70: ≥30/50/70% improvement on the Pain VAS scale; PASI75/100: ≥75%/100% improvement from baseline in Psoriasis Area and Severity Index; PBO: placebo; PsAID-12: Psoriatic Arthritis Impact of Disease-12; Q4W: every 4 weeks; SJC: swollen joint count; TNFi-IR: inadequate response or intolerance to tumour necrosis factor inhibitors; VAS: visual analogue scale; VLDA: very low disease activity.

Results

1,112 BKZ Total patients were included (PBO/BKZ, n=414; BKZ 160mg Q4W, n=698). BKZ group demonstrated rapid achievement of clinically meaningful improvements across outcomes relative to PBO/BKZ group (**Table**). Median times to achieve higher treatment thresholds of ACR50/SJC=0/PASI100/MDA in BKZ Total group from first BKZ dose were similar to median time to achievement of most PROs. MDA was achieved at 16.1wks (16.0, 19.9) in BKZ group vs 20.1wks (19.9, 24.0) in BKZ Total, suggesting benefit of earlier treatment.

Probability of achieving ACR50/SJC=0/PASI100/MDA response at Wk16: 4.9–23.4% in the PBO/BKZ group and 46.3–57.3% in BKZ group. By Year 1, probability for ACR50/SJC=0/PASI100/MDA: 56.5-80.3% in PBO/BKZ group and 66.6–82.9% in BKZ group. The BKZ Total group demonstrated sustained, increasing probabilities of response to Year 3 (**Figure**).

Figure. Kaplan-Meier time curves showing the probability of achieving ACR50, SJC=0, PASI100 and MDA response to Year 3 in BKZ Total group



Randomised set from BE OPTIMAL and BE COMPLETE (including data from BE VITAL). Data from BE OPTIMAL and BE COMPLETE were pooled to Year 3 for all PBO-randomised patients and BKZ-randomized patients, reported as BKZ Total, who were bDMARD-naïve or TNFi-IR. All patients were censored (+) at last visit if response was not achieved. Time to achievement = date of first achievement of threshold – date of first administration of BKZ + 1, calculated from time of first BKZ treatment regardless of treatment group (Week 0 for BKZ-randomised patients and Week 16 for PBO-randomised patients). Patients achieving target threshold prior to or at first administration of BKZ were not included. [a] SJC=0 was assessed in 66 joints; [b] In patients with psoriasis affecting ≥3% BSA at baseline; [c] ACR50 at Week 16 was the primary endpoint of BE OPTIMAL and BE COMPLETE;3,4 [d] In patients who did not achieve MDA prior or at baseline. ACR50: ≥50% improvement from baseline in American College of Rheumatology response criteria; BSA: body surface area; BKZ: bimekizumab; bDMARD: biological disease-modifying antirheumatic drug; MDA: minimal disease activity; PASI100: 100% improvement from baseline in Psoriasis Area and Severity Index; PBO: placebo; Q4W: every 4 weeks; SJC: swollen joint count; TNFi-IR: inadequate response or intolerance to tumour necrosis factor inhibitors.

Conclusions

Bimekizumab treatment was associated with rapid, clinically meaningful improvements across joint, skin and PRO measures; many responses were achieved within 8–16wks and sustained through 3 years in patients with active PsA who were bDMARD-naïve or TNFi-IR, though interpretation should consider the post hoc design and potential confounding factors. Notably, bimekizumab-treated patients reported meeting improvements for pain (Pain30) and physical function (HAQ-DI MCID) within approximately 8–12wks, highlighting the potential benefit of BKZ for prompt patient well-being enhancement.

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Abstract N°: 1160

Successful Treatment of Refractory Hyaluronic Acid Filler-Induced Granulomas with Baricitinib

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Introduction

Hyaluronic acid (HA) fillers are the preferred choice for non-surgical facial rejuvenation due to their biocompatibility and biodegradability. However, as the global volume of filler procedures rises, so does the incidence of late-onset complications, ranging from 0.01% to 14% depending on the material and technique. Among these, delayed-onset nodules, particularly granulomatous reactions, represent one of the most severe and disfiguring immune-mediated adverse events.

Clinically, filler-induced granulomas typically present weeks or even years after the procedure as firm, erythematous, or skin-colored nodules at or near injection sites. Histopathologically, they are characterized by dense macrophage and lymphocytic infiltration, along with multinucleated giant cells attempting to sequester the foreign material. The etiology is multifactorial, involving low-molecular-weight HA fragments, infectious triggers, or host immune responses that drive a chronic pro-inflammatory state.

Although hyaluronidase can degrade HA and corticosteroids suppress acute inflammation, many patients become treatment-refractory, requiring long-term high-dose systemic steroids—risking skin atrophy, telangiectasia, and metabolic side effects—or surgical excision with potential permanent scarring.

Recent research has identified the JAK-STAT pathway as a central regulator of granulomatous inflammation. This report highlights the successful use of baricitinib, a selective JAK1/2 inhibitor, in a patient with recurrent, recalcitrant facial and cervical HA filler-induced granulomas.

Case Presentation

A 56-year-old female presented with a 2-year history of recurrent erythematous papules and nodules on the face and neck, occasionally pruritic. The lesions first appeared in March 2024, three months after facial HA filler injections. Topical corticosteroids and tacrolimus failed to achieve resolution, and an acute flare occurred following a subsequent HA injection in August 2025, characterized by firm, erythematous papules concentrated in the periorbital and perioral regions, with scattered lesions on the neck and increased pruritus.

Laboratory workup, including infectious disease screening, tuberculosis evaluation, blood count, coagulation, liver and renal function tests, and electrolytes, was unremarkable. A facial biopsy performed at an outside institution revealed epidermal acanthosis, dense lymphohistiocytic infiltrates in the superficial and mid-dermis, granuloma formation, multinucleated giant cells, and focal necrosis, establishing a diagnosis of granulomatous reaction.

Prior to our evaluation, the patient had been refractory to multiple therapies. A one-month course of topical tacrolimus and mometasone furoate with oral compound glycyrrhizin and cetirizine failed to control symptoms. Subsequent treatment with oral minocycline and Tripterygium wilfordii glycosides for two weeks was ineffective. A six-week systemic regimen of prednisone (25 mg/day) and isotretinoin (10 mg/day) also yielded no improvement.

Given the recalcitrant nature of the lesions and the associated psychosocial burden, oral baricitinib (2 mg/day) was initiated in December 2025.

At 4 weeks, a marked reduction in the number, size, and induration of nodules was observed, with only sparse erythematous papules remaining in the perioral and periorbital regions, and pruritus had completely resolved. By week 6, most facial and cervical lesions had resolved, leaving only minimal post-inflammatory hyperpigmentation. No adverse events occurred, and follow-up laboratory tests remained within normal limits.

Conclusion

Baricitinib may represent a promising and targeted therapeutic option for refractory HA filler-induced granulomas. This case highlights the potential role of JAK inhibitors in managing complex, filler-associated granulomatous reactions.

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Dupilumab Improves Itch, Quality of Life, and Patient-Reported Disease Severity in Patients With Moderate-to-Severe Prurigo Nodularis

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Introduction
Prurigo nodularis (PN, or nodular prurigo), the most common subtype of chronic prurigo, is a chronic inflammatory skin disease characterized by intensely pruritic nodular lesions that profoundly impact patients' daily functioning. To comprehensively assess disease burden and treatment response, improvements in clinical signs should be monitored along with patient-reported outcomes (PROs). This post hoc analysis aims to evaluate dupilumab's impact on itch, patient-reported quality of life (QoL) and disease severity perception in patients with moderate-to-severe PN.

Materials and Methods
In the PRIME/PRIME2 trials (NCT04183335/NCT04202679) 152 adult patients with PN whose disease was inadequately controlled with topical prescription therapies, or for whom those therapies were inadvisable, received dupilumab 300 mg every 2 weeks or matched placebo dose for 24 weeks. The proportion of patients who achieved a 4-point improvement in Worst Itch Numeric Rating Scale (WI-NRS) score, ≥9-point improvement in Dermatology Life Quality Index (DLQI) score, or a Patient Global Impression of Severity (PGIS) score of 1 or 2 was assessed at Weeks 4, 8, 12, and 24.

Results
At baseline, patients reported WI-NRS mean (standard deviation, SD) scores of 8.6 (0.9) and 8.4 (1.1), and DLQI mean (SD) scores of 18.0 (6.7) and 17.0 (7.2), in the dupilumab and placebo group, respectively, and a PGIS mean (SD) score of 3.7 (0.5) in both groups. A significantly greater proportion of dupilumab vs placebo patients achieved meaningful improvements as early as 4 weeks in: itch (17.8% vs 5.8%), QoL (41.3% vs 23.0%), and disease severity perception (20.7% vs 10.1%) (all $P \leq 0.01$). By Week 24, significantly greater proportions achieved: 4-point WI-NRS score improvement (65.1% vs 31.4%), ≥9-point DLQI score improvement (71.1% vs 35.2%), and PGIS score 1–2 (69.1% vs 37.8%) (all $P \leq 0.0001$). Overall, safety was generally consistent with the known dupilumab safety profile.

Conclusions

Dupilumab demonstrated rapid and sustained improvements over time in both the clinical and PRO endpoints, with meaningful benefits evident as early as Week 4 and achieved by almost two-thirds of patients by Week 24.

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Abstract N°: 1168

From Historical Observations to Modern Melanoma Therapy: The Evolution of Oncolytic Virotherapy

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Introduction

Spontaneous regression of advanced malignancy following acute infection has been described for over a century, including in patients with cutaneous and metastatic melanoma. These early observations formed the conceptual basis of oncolytic virotherapy, in which viruses are used to selectively target tumour cells and stimulate anti-tumour immunity. Talimogene laherparepvec (T-VEC), a modified herpes simplex virus type 1, is now the first approved oncolytic virus for the treatment of unresectable melanoma. This study aims to review the historical origins of oncolytic virotherapy and examine how these observations informed the development of contemporary melanoma treatment.

Materials and Methods

A narrative review was undertaken using nineteenth- and twentieth-century medical literature describing tumour regression associated with infection or vaccination, alongside contemporary literature on oncolytic viral therapies in melanoma. Historical reports were analysed in relation to current understanding of tumour immunology, viral oncolysis and the clinical role of T-VEC.

Results

Early clinicians documented tumour regression following infections such as erysipelas, influenza and smallpox vaccination. Subsequent experimental work demonstrated that viruses could induce selective tumour necrosis and stimulate anti-tumour immune responses. Advances in molecular virology enabled the development of engineered oncolytic viruses, including vaccinia-based constructs and herpes simplex virus-derived therapies. This ultimately led to the introduction of T-VEC, which combines direct tumour lysis with granulocyte-macrophage colony-stimulating factor-mediated immune activation. T-VEC is now established as a treatment option for patients with injectable cutaneous, subcutaneous and nodal melanoma metastases, particularly in locoregional disease.

Conclusions

Understanding the historical origins of oncolytic virotherapy provides important insight into current melanoma management and emerging therapeutic strategies. Re-examining early reports of infection-associated tumour regression highlights the longstanding relationship between dermatological observation and immunotherapy, and demonstrates how historical clinical observations can inform modern translational medicine.

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Abstract N°: 1192

Real-World Study of IL-36R vs. IL-17A Inhibitors in Generalized Pustular Psoriasis

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Introduction
Generalized pustular psoriasis (GPP) is a rare and potentially life-threatening inflammatory dermatosis. For decades, its clinical management has largely been driven by biologic therapies initially developed for psoriasis vulgaris (PsV), a paradigm particularly prevalent in patients with a history of antecedent or concomitant PsV. Among these interventions, IL-17A inhibitors emerged as the earliest and most extensively utilized agents, exerting a profound influence on GPP therapeutic strategies. Although the efficacy of IL-17A inhibitors has been documented across sparse small-scale reports, comparative data between these established therapies and the newly developed IL-36R antagonists remain scarce and poorly elucidated. This paucity of evidence is largely attributable to the limited clinical availability of IL-36R antagonists following their initial approval in late 2022, their prohibitive costs, and the inherent rarity of GPP. Furthermore, studies integrating IL36RN genomic dosage effects with longitudinal therapeutic outcomes remain scarcely documented.

Materials and Methods
We conducted a real-world study (RWS) retrospectively reviewing the clinical data of nine GPP patients, comprising five cases treated with IL-36R inhibitor (spesolimab) and four treated with IL-17A inhibitor (xeligekimab). All enrolled patients had documented comprehensive IL36RN genomic profiling and a minimum follow-up duration of 24 weeks. Therapeutic efficacy was evaluated via longitudinal assessment of the GPP Area and Severity Index (GPPASI) and GPP Physician Global Assessment (GPPGA) scores (including pustule and erythema sub-scores) over a minimum follow-up of 24-weeks. Intergroup comparisons were performed to delineate relative therapeutic performance across these clinical indices.

Results
All five patients in the IL-36R inhibitor group carried bi-allelic IL36RN mutations. In contrast, the IL-17A inhibitor group (N=4) exhibited greater genotypic diversity, comprising one patient with a bi-allelic IL36RN mutation, two with mono-allelic mutations, and one who was wild-type. During the follow-up period, no statistically significant differences were observed between the two groups regarding clinical scores or therapeutic response rates at any time point ($P>0.05$). Notably, IL-36R inhibitor group remained free of acute flares within the initial 24-week follow-up period. Subsequent acute relapses occurred after this 24-week window, with a median recurrence time of 280 days (range: 187-302 days), a cycle potentially associated with the disruption of re-established immune homeostasis. In contrast, no relapses were observed in the IL-17A inhibitor group to date, which may be associated with the standardized bi-weekly (Q2W) injection schedule throughout the study period. Analysis of improvement slopes revealed that IL-36R inhibition achieved a steeper decline in GPPASI, GPPGA total scores, and pustule sub-scores. In contrast, IL-17A inhibition demonstrated a superior rate of improvement specifically in erythema sub-scores reduction.

Conclusions
Both targeted IL-36R antagonism and standardized IL-17A inhibition are capable of maintaining a relapse-free

interval exceeding 24 weeks in GPP. Although IL-36R antagonism is characterized by a superior onset of action regarding pustular clearance and prolonged remission periods, IL-17A inhibition provides a unique clinical benefit by expediting the resolution of erythema and maintaining a more stable, relapse-free profile over time. These divergent clinical responses underscore the dual-axis inflammatory drive inherent to GPP pathogenesis. Moreover, this study provides empirical evidence linking the declining incidence of GPP to the widespread adoption of biologics for PsV, indicating that rigorous management of the underlying psoriatic background can effectively mitigate the progression of pustular phenotypes.

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Abstract N°: 1193

Tofacitinib for diffuse systemic sclerosis confined to flexural areas: a case report

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Introduction

Systemic sclerosis (SSc) is a chronic autoimmune connective tissue disease characterized by vasculopathy and progressive fibrosis of the skin and internal organs. Diffuse cutaneous SSc (dcSSc) usually presents with rapid, widespread skin thickening of the extremities and trunk and is frequently associated with cardiopulmonary or gastrointestinal involvement. In contrast, skin sclerosis restricted to flexural or intertriginous areas is uncommon and may delay recognition of dcSSc, especially when systemic manifestations and disease-specific autoantibodies are absent.

Therapeutic options for dcSSc remain limited, and responses to conventional immunosuppressants and topical agents are often unsatisfactory. Experimental data implicate the Janus kinase (JAK)–signal transducer and activator of transcription (STAT) pathway in the pathogenesis of SSc, providing a rationale for the off-label use of JAK inhibitors as potential antifibrotic treatments. Here, we report a patient with dcSSc presenting with flexural-predominant skin sclerosis and no evident internal organ involvement who showed notable clinical improvement after tofacitinib therapy.

Case Presentation

A 61-year-old woman presented with a 2-year history of slowly progressive skin tightness and induration confined to flexural and intertriginous areas. The eruption began in the inframammary folds and gradually extended to the axillae and lateral neck. She denied Raynaud’s phenomenon, digital ulcers, dysphagia, gastroesophageal reflux, dyspnea, cough, or other systemic symptoms. There was no history of infection, trauma, radiation, or use of drugs associated with scleroderma-like changes, and her personal and family history were negative for autoimmune diseases.

Examination revealed symmetrical, non-pitting induration and sclerosis of the skin in the inframammary folds, bilateral axillae and lateral neck, with a sensation of tightness and mild limitation of shoulder and neck movement. The face, hands, forearms, remaining trunk and lower extremities were spared, without sclerodactyly, fingertip pitting scars, telangiectasia or digital ulcers.

Routine blood tests and liver and renal function were normal, and inflammatory markers were not elevated. Autoimmune serology was unremarkable, including negative antinuclear antibodies, anti-topoisomerase I (Scl-70), anti-centromere and anti-RNA polymerase III antibodies. There was no clinical evidence of cardiopulmonary, renal or gastrointestinal involvement. A skin biopsy from an inframammary lesion showed dilated, congested small vessels in the superficial dermis with perivascular lymphocytic infiltrates, supporting a diagnosis of systemic sclerosis in the appropriate clinical context. The patient had previously been treated with topical heparinoid ointment and madecassoside cream, with minimal benefit.

Tofacitinib 5 mg twice daily was initiated together with emollients. After 3 months, the patient showed marked softening of the indurated skin in the axillae, lateral neck and inframammary folds, with clear improvement in the

range of motion of the shoulders and neck. No new organ involvement or significant adverse events were observed during this period.



Clinical photographs of flexural skin lesions before and after tofacitinib treatment. Upper panel: baseline lesions before treatment, showing marked skin thickening and induration. Lower panel: lesions after 3 months of tofacitinib (5 mg twice daily), showing reduced induration and improved skin pliability.

Conclusion

This case illustrates an atypical form of dcSSc with skin sclerosis limited to flexural and intertriginous sites and no detectable internal organ involvement or disease-specific autoantibodies. The observed improvement in skin sclerosis and mobility after a short course of tofacitinib, in a patient refractory to topical therapy, suggests that JAK inhibition may be a useful therapeutic option in selected dcSSc patients. Larger, controlled studies are needed to confirm the efficacy and safety of tofacitinib and other JAK inhibitors in systemic sclerosis.

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30 SEP - 3 OCT 2026

Abstract N°: 1198

Efficacy and Safety of Guselkumab in Participants With Active Psoriatic Arthritis and Inadequate Response/Intolerance to One Prior Tumor Necrosis Factor Inhibitor Through 1 Year of the SOLSTICE Study

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Introduction

Guselkumab (GUS) is a fully human, dual-acting[1] monoclonal antibody inhibiting the interleukin (IL)-23p19 subunit. In SOLSTICE, a phase 3b, multicenter, randomized, placebo (PBO)-controlled study, GUS 100 mg every 4 weeks (Q4W) and every 8 weeks (Q8W) significantly improved psoriatic arthritis (PsA) signs and symptoms through Week 24 in participants with inadequate response (IR; inadequate efficacy or intolerance) to one prior tumor necrosis factor inhibitor (TNFi).[2] Here we summarize results through Week 52.

Materials and Methods

TNFi-IR participants with active PsA (≥3 swollen/≥3 tender joints; C-reactive protein [CRP] ≥0.3 mg/dL) after one prior TNFi were randomized to GUS 100 mg Q4W or Week 0, Week 4, then Q8W, or PBO to GUS 100 mg Q4W at Week 24. American College of Rheumatology (ACR)20/50/70, Investigator's Global Assessment response (IGA 0/1; score 0 or 1 plus ≥2-grade improvement), ≥90% improvement in Psoriasis Area and Severity Index (PASI 90), and minimal disease activity (MDA) were assessed through Week 52.

Results

Of 451 analyzed participants, 88.0% completed treatment through Week 52. Baseline characteristics were consistent with moderate to severe PsA. Response rates increased from Week 24 to Week 52 in GUS-randomized participants (Q4W/Q8W, respectively) for ACR20 (70.0/64.9), ACR50 (46.9/43.7), ACR70 (26.7/27.8), IGA 0/1 (72.8/66.3), PASI 90 (73.0/66.3), and MDA (36.7/31.8) (**Figure**). Rates for PBO participants who initiated GUS Q4W at Week 24 approached those of GUS-randomized participants at Week 52. Time-adjusted frequencies of adverse and serious adverse events were 184.3/100 patient-years and 5.8/100 patient-years, respectively.

Conclusions

Response rates were maintained or increased through Week 52 regardless of treatment regimen in GUS-randomized TNFi-IR participants with active PsA. No new safety signals were identified.

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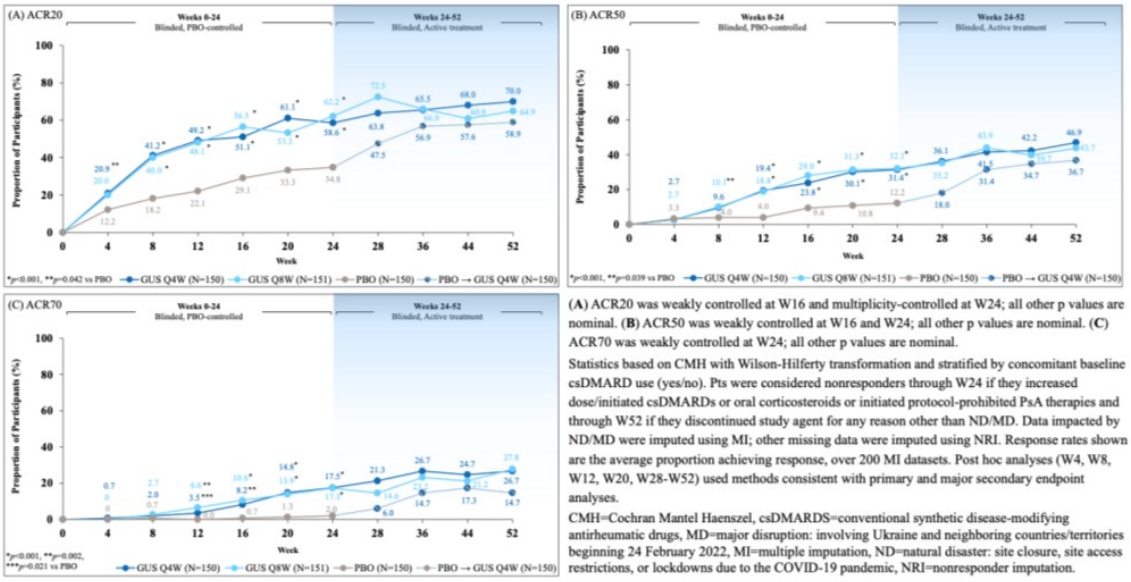
***Figure. (A)** ACR20, **(B)** ACR50, and **(C)** ACR70 response rates increased from Week 24 to Week 52 in GUS-randomized participants*



(A) ACR20 was weakly controlled at W16 and multiplicity-controlled at W24; all other p values are nominal. **(B)** ACR50 was weakly controlled at W16 and W24; all other p values are nominal. **(C)** ACR70 was weakly controlled at W24; all other p values are nominal.

Statistics based on CMH with Wilson-Hilferty transformation and stratified by concomitant baseline csDMARD use (yes/no). Pts were considered nonresponders through W24 if they increased dose/initiated csDMARDs or oral corticosteroids or initiated protocol-prohibited PsA therapies and through W52 if they discontinued study agent for any reason other than ND/MD. Data impacted by ND/MD were imputed using MI; other missing data were imputed using NRI. Response rates shown are the average proportion achieving response, over 200 MI datasets. Post hoc analyses (W4, W8, W12, W20, W28-W52) used methods consistent with primary and major secondary endpoint analyses.

CMH=Cochran Mantel Haenszel, csDMARDS=conventional synthetic disease-modifying antirheumatic drugs, MD=major disruption: involving Ukraine and neighboring countries/territories beginning 24 February 2022, MI=multiple imputation, ND=natural disaster: site closure, site access restrictions, or lockdowns due to the COVID-19 pandemic, NRI=nonresponder imputation.

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Abstract N°: 1210

SPECTREM: Guselkumab Effects on Uncontrolled Psoriatic Arthritis in Participants With Moderate, Low Body Surface Area, High-Impact Site Psoriasis Through Week 48

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Introduction
In patients with psoriasis, high-impact sites (the scalp and intertriginous areas, especially the intergluteal/perianal regions) are risk factors for psoriatic arthritis (PsA). The phase 3b SPECTREM study evaluated the efficacy of guselkumab in participants with low body surface area (BSA; 2–15%), moderate (Investigator's Global Assessment [IGA]=3) plaque psoriasis involving ≥1 high-impact sites (scalp, face, intertriginous areas, or genitals). This post-hoc analysis reports the 12-item Psoriatic Arthritis Impact of Disease questionnaire (PsAID-12) results through Week 48 for participants with uncontrolled PsA at baseline.

Materials and Methods
Participants were randomized 2:1 to receive guselkumab 100 mg (N=225; at Week 0, Week 4, then every 8 weeks [q8w]) or placebo (N=113; at Week 0, Week 4, and Week 12, with crossover to guselkumab [placebo to guselkumab]) at Week 16, Week 20, then q8w. PsA was identified based on history of rheumatologist-diagnosed PsA or the Psoriasis Epidemiology Screening Tool (PEST) score ≥3 at screening. Participants meeting either of these criteria were considered to have uncontrolled PsA at baseline if they had a PsAID-12 score of >4.0 (the PsAID patient-acceptable symptom state [PASS] is defined as a score of ≤4.0). PsAID-12 scores range from 0–10, and the minimal clinically important improvement is defined as a reduction of at least 3.0 points.

Results
Among 338 randomized participants, 15.4% (n=52) had a history of rheumatologist-diagnosed PsA (n=14) or PEST ≥3 at screening (n=38 with previously undiagnosed PsA) and a PsAID-12 score >4.0 at baseline. Prior psoriasis treatments included topicals (100%), phototherapy (17.3%), methotrexate (13.5%), and advanced orals (3.8%). The mean baseline data reflected moderate skin disease (Psoriasis Area and Severity Index=9.1; BSA=7.4%; and IGA=3) and a moderate impact of PsA on health (mean PsAID-12 scores of 6.6 [guselkumab group] and 6.1 [placebo group]). From baseline to Week 16, the least-squares mean change in PsAID-12 score was −2.9 in the guselkumab group and −1.3 in the placebo group (nominal p=0.083); the mean PsAID-12 score was below the PASS at Week 16 in the guselkumab group (3.7). Mean PsAID-12 scores continued to improve and were 2.7 in the guselkumab group and 1.0 in the placebo→guselkumab group at Week 48, with mean changes from baseline of −3.9 and −5.2, respectively.

Conclusions

At baseline, approximately 15% of SPECTREM participants had uncontrolled PsA based on having a PsAID-12 score above the PASS threshold of 4.0, indicating a need for improved routine PsA screening. Specifically, patients with psoriasis having low BSA and PsA risk factors should be considered for PEST screening to enable early detection of PsA and the initiation of appropriate treatment to achieve improved outcomes. In these participants with uncontrolled and often undiagnosed PsA at baseline, the mean PsAID-12 scores decreased below the PASS after 3 doses of guselkumab, indicating this group achieved clinically meaningful improvements in the physical and psychological impact of joint disease. Improvements continued and were maintained through 48 weeks of treatment.

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Abstract N°: 1217

Pregnancy Outcomes in Maternal Exposure to Guselkumab: Review of Cases Reported to the Company Global Safety Database

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Introduction

The Global Consensus Consortium suggests that women with inflammatory bowel disease can continue treatment with interleukin (IL)-23 inhibitors during pregnancy.[1] Guselkumab (GUS), a dual-acting IL-23p19 subunit inhibitor, potently blocks IL-23 signaling and binds to CD64, a receptor on IL-23-producing immune cells.[2] GUS is approved to treat moderate-to-severe psoriasis (PsO), active psoriatic arthritis (PsA), and moderately to severely active ulcerative colitis (UC) and Crohn's disease (CD).[3] We report pregnancy outcomes in women exposed to GUS during pregnancy.

Materials and Methods

Pregnancy data reported to the Company Global Safety Database through 12 July 2025 were analyzed for maternal GUS exposure cases by therapeutic indication, trimester of GUS exposure, and pregnancy outcomes.

Results

A total of 1126 pregnancy events (twins [2]; triplets [1]; women with 2 pregnancies [4]) from 1118 maternal GUS exposure cases were reported (outcomes: known [N=400], ongoing pregnancies [N=369], unknown/not reported [N=357]). This analysis focuses on the 396 cases with known pregnancy outcomes (400 events) that were reported prospectively (N=120) or retrospectively (N=280). The mean maternal age was 32 years. The GUS therapeutic indications for women exposed during pregnancy were: psoriatic disease (73.0% [289/396]), other/not reported (19.9% [79/396]), CD (3.8% [15/396]), UC (2.5% [10/396]), and healthy subjects (0.8% [3/396]). Maternal GUS exposure occurred before conception (≤3 months prior to conception; 10.8% [43/400]), during the first trimester (35.0% [140/400]), after the first trimester only (0.5% [2/400]), throughout pregnancy (4.5% [18/400]), or not reported (49.2% [197/400]). Pregnancy outcomes included live birth without congenital anomaly (65.0% [260/400]), live birth with congenital anomaly (1.5% [6/400]), spontaneous abortion (21.0% [84/400]), elective termination without fetal defects or unknown (8.5% [34/400]), elective termination with fetal defects (0.25% [1/400]), ectopic pregnancy (2.5% [10/400]), stillbirth without fetal defects (1.0% [4/400]), and unspecified abortion with fetal defects (0.25% [1/400]).

Rates of live births, spontaneous abortions, elective terminations, and congenital anomalies in women exposed to GUS are comparable with rates reported in the general population (**Figure**).[4,5]

Conclusions

These findings suggest no apparent impact of GUS on pregnancy outcomes; however, they should be interpreted cautiously given data limitations. Further studies are warranted to confirm these observations and to better characterize the safety profile of GUS exposure during pregnancy.

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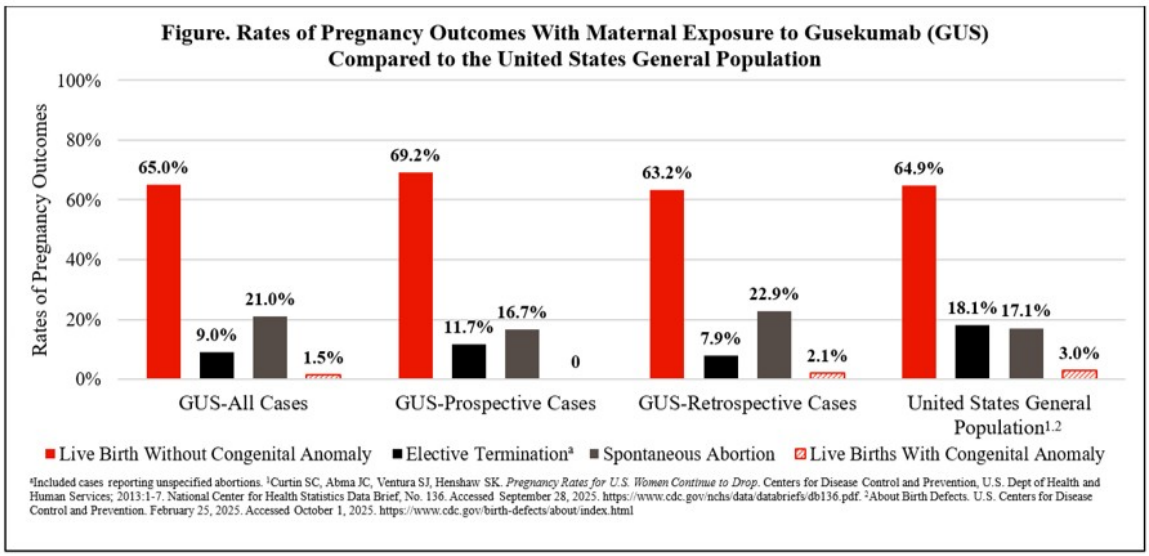

EA DV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 1242

When PASI 0 becomes an emergency: disseminated tuberculosis under anti-TNF therapy

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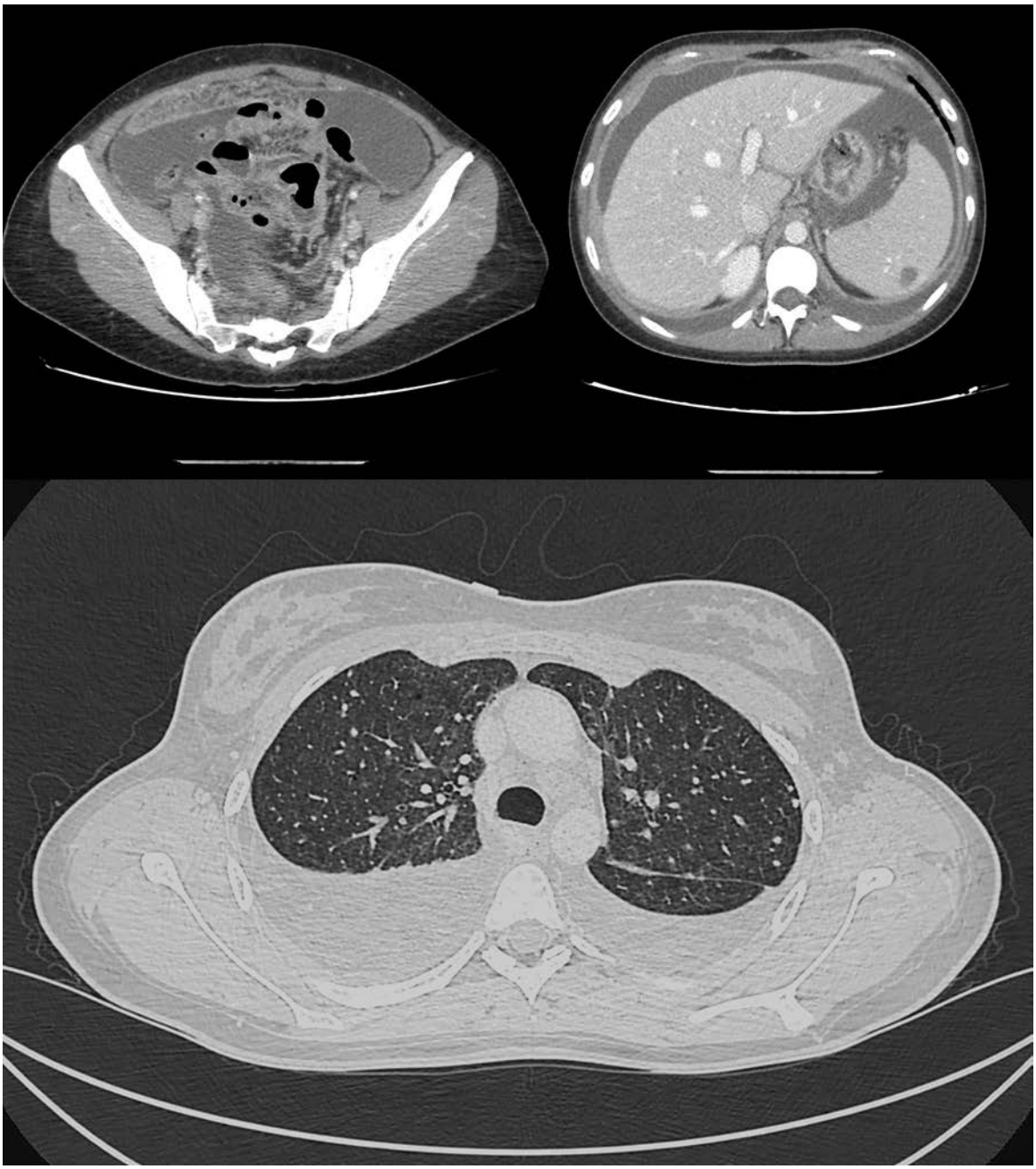
Introduction

Tumor necrosis factor-α inhibitors (anti-TNF) have transformed the management of psoriasis, but they are associated with an increased risk of tuberculosis (TB), with atypical and disseminated presentations being more frequent. Despite systematic baseline screening, cases continue to occur, highlighting the limitations of current screening strategies.

Case Presentation

A 25-year-old patient with moderate psoriasis showed inadequate response to phototherapy and methotrexate, and was subsequently treated with adalimumab, achieving complete skin clearance (PASI 0). Eight months later, the patient presented to the emergency department with severe abdominal pain, distension and fever. Physical examination revealed diffuse abdominal guarding, and laboratory tests showed elevated inflammatory markers. Computed tomography demonstrated abundant ascites, nodular peritoneal thickening, bilateral pleural effusion, moderate pericardial effusion, splenic hypodense lesions, and multiple bilateral pulmonary micronodules, raising suspicion of a possible peritoneal carcinomatosis without an identifiable primary tumor (image 1). Tumor markers were negative. Finally, peritoneal biopsy revealed necrotizing granulomatous inflammation, and microbiological culture confirmed *Mycobacterium tuberculosis* infection.

Adalimumab was immediately discontinued, and anti-tuberculosis therapy was initiated, with progressive clinical improvement. After 3 months of sustained clinical stability, a biologic agent targeting a different pathway (interleukin-23 inhibitor) was introduced without complications (1).



CT scan showed abundant ascites, nodular peritoneal thickening, bilateral pleural effusion, splenic hypodense lesions, moderate pericardial effusion and multiple bilateral pulmonary micronodules

Conclusion

This case illustrates a severe disseminated TB presentation mimicking malignancy in a patient receiving anti-TNF therapy despite prior negative screening. Current evidence suggests that TB may still occur in approximately 0.3–1.7% of patients with negative baseline screening, due to reactivation (most cases) or new infection (2). Screening tools have inherent limitations: chest radiography may be normal in up to 20% of active TB cases (3), and interferon-γ release assays may yield false-negative results, particularly in immunosuppressed individuals, during the window period, or in extrapulmonary disease (4).

Routine annual rescreening is not cost-effective in low-incidence settings and may increase false positives (5). Therefore, clinical vigilance remains essential. A structured and repeated risk-oriented clinical history remains essential to identify patients at risk and should be systematically integrated into follow-up. Negative baseline screening should not provide false reassurance. Dermatologists should maintain a high index of suspicion, recognizing that negative screening does not exclude tuberculosis risk in patients receiving biologic therapy.

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Abstract N°: 1248

SAFETY, PHARMACOKINETICS, AND PHARMACODYNAMICS OF ATTO-1310, A NOVEL ANTI-IL31 ATTOBODY-FC FUSION PROTEIN, IN HEALTHY ADULTS

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Introduction

Interleukin-31 (IL-31) is a potent pruritogenic cytokine that stimulates sensory neurons to trigger itching. This signaling initiates a debilitating itch-scratch cycle, where mechanical skin damage further compromises the skin barrier and promotes a self-perpetuating inflammatory response. In patients with atopic dermatitis or prurigo nodularis, treatment with an IL-31 receptor (IL-31RA) antagonist demonstrated itch reduction and improvement in skin lesions. ATTO-1310 is a novel ATTOBODY-Fc fusion protein aiming to achieve improved efficacy by directly targeting IL-31. This first-in-human Phase 1 study sought to assess the safety, pharmacokinetics, immunogenicity, and pharmacodynamics of ATTO-1310 in healthy volunteers, patients with atopic dermatitis, and patients with chronic pruritus.

Materials and Methods

This study consisted of 4 parts. Here we present Parts 1 and 2, which included healthy adults aged 18 and older (Parts 3 and 4 presented separately). Part 1 was a single ascending-dose design with 5 cohorts: 0.5 mg/kg IV (intravenous), 1.5 mg/kg IV, 4.5 mg/kg IV, 4.5 mg/kg SC (subcutaneous), and 9.0 mg/kg IV. Part 2 was a multiple ascending-dose design with 2 cohorts: 1.5 mg/kg SC and 4.5 mg/kg SC administered on Day 1 and Day 29. Each cohort consisted of 8 subjects randomized 3:1 (active: placebo). Assessments included frequency and severity of treatment emergent adverse events (TEAEs), clinical laboratory testing, serum concentration of ATTO-1310, and incidence of anti-drug antibodies (ADAs). Pharmacodynamic activity was assessed using percent free IL-31, expressed as the ratio of free / total serum IL-31.

Results

Fifty-six subjects were enrolled: 40 subjects in Part 1 (mean age, 36.6 years; 62.5% female) and 16 subjects in Part 2 (mean age, 41.5 years; 50.0% female). No serious TEAEs were observed. One severe TEAE (syncope, Grade 3) occurred in a subject who received placebo. All other TEAEs were mild or moderate in severity. Treatment-related TEAEs reported in at least 2 subjects were headache and nausea (Grade 1 only). Treatment was discontinued for 1 subject with pyrexia (Grade 1). No injection site reactions were observed. There were no clinically meaningful laboratory trends. ATTO-1310 exhibited linear pharmacokinetics with approximate dose-proportional increases in systemic exposure across tested dose levels and good bioavailability with SC administration. ADAs were detected in 4 of 42 subjects treated with ATTO-1310. Two of 4 subjects had treatment-induced ADAs (2 had pre-existing ADAs at baseline). ADA positive samples were all low titer (<200) and non-neutralizing. Total serum IL-31,

reflecting drug-bound and free IL-31, increased after ATTO-1310 treatment in all groups consistent with target engagement. Percent free IL-31 decreased by 95% within the first 24 hours after a single dose of ATTO-1310 for all doses tested. Greater than 95% reduction in mean percent free IL-31 was retained for up to 12 weeks at all doses.

Conclusions

ATTO-1310 was well tolerated in healthy adults with no serious or severe treatment-related TEAEs and a low incidence of ADAs. Single and multiple SC administration of ATTO-1310 in healthy adults resulted in rapid and sustained reduction of percent free IL-31 extending up to 12 weeks.

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Abstract N°: 1283

Imiquimod-Induced Lupus-Like Reaction: A Diagnostic Challenge and Pathophysiological Insight

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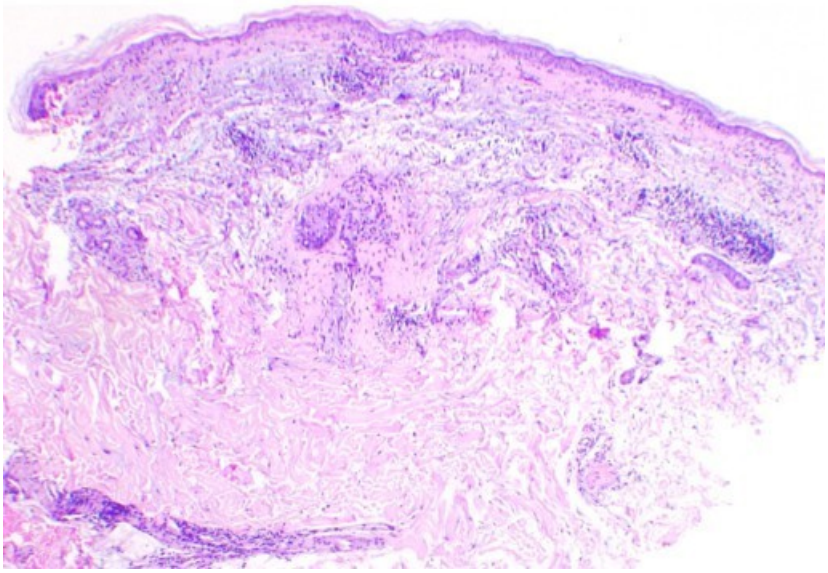
Introduction

Imiquimod is a potent immune response modifier and a toll-like receptor 7 and 8 (TLR7/8) agonist, widely used for actinic keratosis. While it primarily enhances local innate immunity, it can exceptionally trigger systemic or distal autoimmune phenomena in genetically predisposed individuals. This report highlights the importance of recognizing imiquimod-induced lupus-like reactions to prevent unnecessary aggressive systemic therapy and to understand the delicate balance of TLR stimulation.

Case Presentation

An 88-year-old female with a history of Waldenström macroglobulinemia presented with an intense erythematous and crusty reaction on the forehead 15 days after initiating 5% imiquimod cream. Despite drug cessation, she developed systemic symptoms; arthralgia and myalgia— ollowed by the emergence of distal, annular, erythematous scaly plaques with central atrophy in photo-exposed areas (chest and upper extremities). Laboratory findings revealed leucopenia (3.2 \times 10^9/L), significant hypocomplementemia (C3 42 mg/dL, C4 6 mg/dL), and positive ANA (speckled pattern, 1:320), while anti-Ro, anti-La, and anti-DNA antibodies remained negative. Histopathology of a distal lesion confirmed an interface dermatitis with vacuolar basal degeneration, necrotic keratinocytes, and mucin deposits. Direct immunofluorescence and CD123 staining highlighted a focal plasmacytoid dendritic cell (pDC) distribution, consistent with a lupus-like process. The patient showed remarkable clinical and laboratory improvement after 4 weeks of strict photoprotection and topical 0.1% tacrolimus, avoiding the need for systemic immunosuppressants.



Conclusion

This case underscores that imiquimod-induced lupus-like reactions, although rare, must be considered when atypical lesions appear in photo-exposed areas following treatment. The activation of the TLR7/8 pathway by imiquimod likely mirrors the endogenous activation seen in systemic lupus erythematosus, providing a unique "in vivo" model of drug-induced autoimmunity. Early clinical-pathological correlation is paramount; immediate drug withdrawal and the use of topical calcineurin inhibitors are effective and safe, even in patients with complex hematological comorbidities. Long-term monitoring is essential as autoantibodies may persist as sentinels for potential relapses.

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30 SEP - 3 OCT 2026

Abstract N°: 1301

Effectiveness and Safety of Baricitinib and Ritlecitinib in Severe Alopecia Areata: A Real-World Monocentric Study

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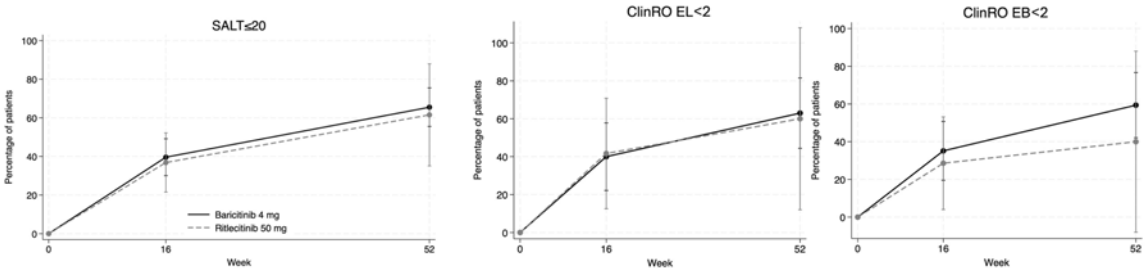
Introduction
Alopecia areata (AA) is a chronic autoimmune disease characterized by non-scarring hair loss that may involve the scalp, eyebrows, eyelashes, and other hair-bearing areas, often with a substantial psychosocial burden. In recent years, Janus kinase (JAK) inhibition has become a targeted therapeutic strategy for severe disease. Baricitinib and ritlecitinib are currently approved for severe AA, but direct comparative evidence from routine clinical practice remains limited. We therefore performed a real-world study to compare the effectiveness and safety of baricitinib 4 mg and ritlecitinib 50 mg in patients with severe AA treated in a tertiary dermatology center.

Materials and Methods
This was a monocentric retrospective observational study conducted at the Dermatology Unit of IRCCS Humanitas Research Hospital, Rozzano, Italy. We included 160 adolescent and adult patients with severe AA, defined by a baseline Severity of Alopecia Tool (SALT) score ≥50, treated with baricitinib 4 mg once daily (n=110) or ritlecitinib 50 mg once daily (n=50) in routine clinical practice. Clinical assessments were performed at baseline, week 16, and week 52; an additional week 104 evaluation was available for baricitinib-treated patients who remained on treatment. The primary endpoint was the proportion of patients achieving SALT ≤20 at week 52. Secondary endpoints included SALT ≤20 at week 16 and week 104, improvement in eyebrow and eyelash involvement using ClinRO Eyebrow (EB) and ClinRO Eyelash (EL) scores, and treatment safety. Baseline demographic, clinical, and comorbidity data were collected from medical records. Comparisons between groups were performed using chi-square and Student's t-tests, with p≤0.05 considered statistically significant.

Results
Overall, both groups had a high baseline disease burden, with mean SALT scores of 78.0 in the baricitinib group and 72.7 in the ritlecitinib group. Patients receiving baricitinib were older than those receiving ritlecitinib (44.25 vs 37.40 years; p=0.005), reflecting different age eligibility criteria. Effectiveness improved progressively over time in both cohorts. At week 16, SALT ≤20 was achieved by 39.6% of baricitinib-treated patients and 36.8% of ritlecitinib-treated patients (p=0.766). At week 52, response rates increased to 65.5% and 61.5%, respectively (p=0.779), showing no significant difference between treatments. At week 104, 70.0% of evaluable baricitinib-treated patients achieved SALT ≤20. Eyelash responses were also comparable, with ClinRO EL <2 observed in 40.0% versus 41.7% of patients at week 16 and in 63.0% versus 60.0% at week 52. Similarly, ClinRO EB <2 was achieved by 35.1% versus 28.6% of patients at week 16 and by 59.4% versus 40.0% at week 52, without statistically significant between-group differences. Treatment discontinuation due to adverse events was infrequent and similar in the two groups (3.6% vs 3.1%; p=0.867). No major cardiovascular, thromboembolic, or malignant events were observed. Secondary inefficacy was reported more frequently with baricitinib than with

ritlecitinib (17.1% vs 4.7%; p=0.017), a finding likely influenced by the earlier availability of baricitinib and the longer treatment exposure in that cohort.



From left to right, proportion of patients achieving SALT score ≤20, ClinRO EL score <2, and ClinRO EB score <2 over time in the baricitinib 4 mg and ritlecitinib 50 mg treatment groups up to week 52. Error bars indicate 95% confidence intervals.

Conclusions

In this real-world monocentric cohort of patients with severe AA, baricitinib 4 mg and ritlecitinib 50 mg showed comparable effectiveness through week 52, with progressive improvement in scalp, eyebrow, and eyelash outcomes and favorable safety profiles. The higher rate of secondary inefficacy observed with baricitinib should be interpreted cautiously in light of longer exposure time. Overall, these findings support the use of both agents as effective targeted therapies for severe AA in routine clinical practice and provide one of the first direct real-world comparisons between baricitinib and ritlecitinib.

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Abstract N°: 1329

Bimekizumab for Refractory Darier Disease: Targeting the IL-17 Axis in a Challenging Genodermatosis

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Introduction

Darier disease (DD) is a rare autosomal dominant genodermatosis caused by mutations in the ATP2A2 gene, leading to impaired keratinocyte adhesion and abnormal epidermal differentiation. Management of DD remains challenging, particularly in refractory cases. Emerging evidence suggests a role of inflammatory pathways, including the IL-17 axis, in disease pathogenesis. We report a case of DD successfully treated with bimekizumab combined with low-dose acitretin.

Case Presentation

A 20-year-old female presented with widespread hyperkeratotic papules and plaques involving the face, neck, trunk and flexural areas. Dermatological examination revealed yellowish hyperkeratotic papules on the hairline, medial eyebrows, nasolabial folds and chin; erythematous, scaly hyperkeratotic papules on the neck, anterior ches, and flexural surfaces of the extremities; and skin-colored papules on the forearms and dorsal hands. Nail examination showed longitudinal ridging and distal V-shaped notching. Skin biopsy confirmed an acantholytic dyskeratotic dermatosis consistent with DD.

The disease had been present since adolescence and was refractory to multiple treatments, including long-term cyclosporine (intermittently over 4 years), systemic isotretinoin (8 months), and acitretin (25 mg/day for 4 months), all without clinical improvement and with progressive worsening of symptoms. At presentation, the patient had persistent, treatment-resistant plaques despite ongoing treatment with acitretin at a dose of 0.5 mg/kg/day (25 mg daily for 4 months). Given the lack of response to high-dose acitretin, bimekizumab was initiated at a dose of 320 mg at weeks 0, 4, 8, 12, and 16, followed by every 8 weeks, in combination with low-dose acitretin (10 mg/day).

After six months of treatment, marked clinical improvement was observed, with significant regression of papular lesions on the head and neck, accompanied by a marked improvement in symptoms and daily functioning, and partial improvement on the extremities. Baseline malodour associated with skin lesions resolved completely during follow-up. Nail involvement remained unchanged. No significant adverse events were reported, and no disease flare occurred during follow-up.

Conclusion

This case highlights the potential efficacy of bimekizumab in refractory DD, supporting the role of IL-17A/F inhibition in its pathogenesis. The addition of low-dose acitretin may have contributed to clinical improvement while minimizing retinoid-related adverse effects. In the present case, acitretin was planned to be discontinued due to lack of efficacy and concerns related to its use in a woman of childbearing age, prompting the search for alternative therapeutic options. Bimekizumab may represent a promising therapeutic option in refractory DD, particularly in patients unresponsive to conventional systemic therapies; however, further studies are needed to establish its long-term efficacy and safety.

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Atypical Appendicitis associated with Selective JAK1 Inhibition in Atopic Dermatitis

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Introduction

Abrocitinib is a selective Janus kinase-1 (JAK1) inhibitor approved for the treatment of moderate-to-severe atopic dermatitis. It provides rapid and sustained improvement in pruritus and skin inflammation through modulation of multiple type 2 cytokine pathways. While its efficacy is well established, JAK inhibitors are associated with potential safety concerns, particularly an increased risk of infection and, less commonly, gastrointestinal complications. The immunomodulatory effects of JAK1 inhibition may also alter the clinical presentation of inflammatory or infectious conditions, potentially delaying diagnosis.

Case Presentation

A 59-year-old gentleman with long-standing, severe atopic dermatitis was commenced on abrocitinib, achieving near-complete disease clearance and a marked improvement in quality of life. After eight months of therapy, he presented to the emergency department with a three-week history of intermittent vague right iliac fossa discomfort, diarrhoea, reduced appetite, and unintentional weight loss. Notably, he remained systemically well, afebrile, and had no significant elevation in inflammatory markers. The initial clinical assessment was therefore relatively reassuring but did not explain the persistence of his symptoms. In view of this, cross-sectional imaging of the abdomen and pelvis was performed. This revealed chronic appendicitis complicated by fistula formation and a peri-appendiceal collection. Following surgical review, a conservative management approach was recommended, consisting of oral antibiotics with a plan for interval appendicectomy at a later stage.

Conclusion

This case highlights a rare and previously undescribed presentation of chronic, fistulating appendicitis in a patient receiving abrocitinib. It is plausible that JAK1 inhibition attenuated peritoneal inflammatory signalling, thereby blunting both systemic and local inflammatory responses. Consequently, significant intra-abdominal pathology manifested with indolent, non-specific symptoms rather than the classical features of an acute abdomen. These findings contribute to the evolving safety profile of JAK inhibitors and underscore the importance of maintaining a high index of suspicion when assessing patients on these therapies. Clinicians should be alert to subtle or atypical presentations of serious pathology, even in the absence of overt inflammatory markers.

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Phase 1 Clinical Trial of ORKA-002, a Novel Half-Life Extended IL-17 A/F Monoclonal Antibody with Potential for Twice Yearly Dosing in Psoriasis and Psoriatic Arthritis, and Quarterly Dosing in Hidradenitis Suppurativa

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Introduction
ORKA-002 is a novel monoclonal antibody targeting IL-17A and IL-17F that is similar to bimekizumab in potency and epitope binding and has an Fc region that has been engineered to significantly extend serum half-life. The pharmacokinetics (PK) of ORKA-002 suggest that ORKA-002's extended half-life creates the potential for twice yearly dosing in psoriasis and psoriatic arthritis, and quarterly dosing in hidradenitis suppurativa (HS). Here, data from the first 24 weeks of the first-in-human (FIH) Phase 1 study of ORKA-002 in healthy volunteers are presented.

Materials and Methods
This Phase 1, double-blinded, placebo-controlled, randomized FIH study evaluated safety, tolerability, PK, and pharmacodynamics (PD) of single ascending doses of ORKA-002 in 24 healthy adult volunteers. Participants were randomized 6:2 to receive a single subcutaneous dose of ORKA-002 or placebo across three ascending dose-level cohorts: 160 mg, 320 mg, and 640 mg. Participants were admitted to a clinical research unit, where they remained until day 4, and then returned to the clinic for follow-up visits over one year for safety and PK assessments (NCT06944379).

Results
Eight participants were dosed in each of the three cohorts, 6 with ORKA-002 and 2 with placebo. Baseline characteristics were typical of a healthy volunteer population. The half-life of ORKA-002 was approximately 75-80 days (Figure 1). Individual PK profiles showed no indication of anti-drug antibodies. In an *ex vivo* IL-17 stimulation assay, serum from subjects dosed with ORKA-002 durably inhibited IL-17 signaling for 24 weeks (study duration to date). The study remains blinded; however, no serious or severe adverse events (AEs) were reported and no discontinuations occurred. AEs reported in >2 participants were contusion, headache, skin abrasion, and upper respiratory tract infection. All of these events were mild. No dose-dependent trends in AEs were observed.

Conclusions

PK and PD results from this Phase 1 trial of ORKA-002, including a half-life of 75-80 days, support the potential for twice-yearly dosing in psoriasis and psoriatic arthritis, and quarterly dosing in HS while maintaining trough antibody concentrations above bimekizumab. ORKA-002 was well-tolerated across all dose levels, with a favorable safety profile consistent with the IL-17 inhibitor class. These attributes are being further explored in a Phase 2 study, ORCA-SURGE, which is evaluating efficacy and safety of ORKA-002 in adults with moderate-to-severe psoriasis.

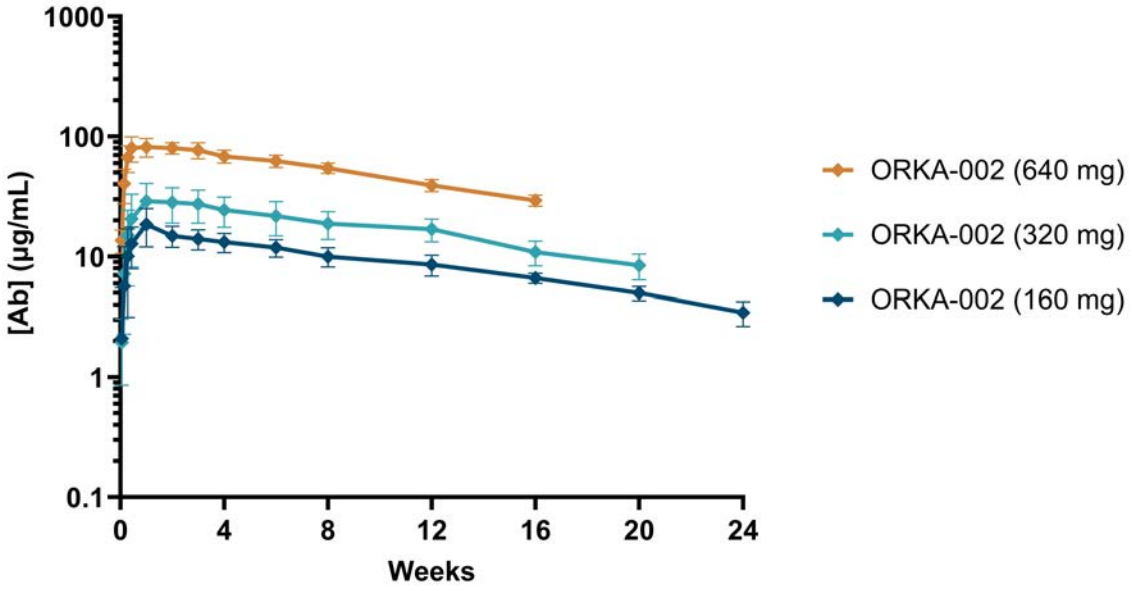

Figure 1: Pharmacokinetic profile of a single subcutaneous dose of ORKA-002

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Abstract N°: 1373

Efficacy and Safety of QX005N in Adults with Prurigo Nodularis: Results from a multicenter, randomized, double-blinded, placebo-controlled phase 3 trial

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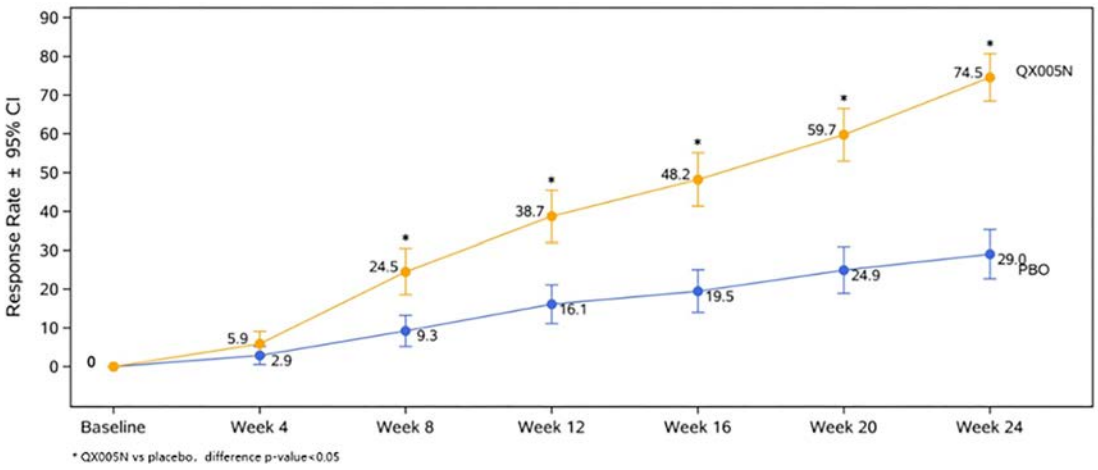
Introduction
Prurigo nodularis (PN) is a chronic, recurrent disease characterized by intense pruritus and hyperkeratotic nodules. QX005N is a novel, humanized anti-IL-4Rα IgG4 monoclonal antibody. A phase 3 trial was conducted and the results from 24 weeks treatment were reported.

Materials and Methods
Adults with PN who had an inadequate response or intolerance to topical corticosteroids (TCS), a weekly average Worst Itch Numeric Rating Scale (WI-NRS) score ≥7, and an Investigator’s Global Assessment for PN-Stage (IGA PN-S) ≥3 were randomized 1:1 to QX005N 450 mg every 2 weeks (Q2W) or placebo for 24 weeks, followed by a

28-week extension period where patients in both groups received QX005N 450 mg Q2W through Week 52, and a final 8-week follow-up. The primary endpoint was the proportion of participants achieving a ≥4-point reduction from baseline in the weekly average WI-NRS at Week 24. The key secondary endpoint was the proportion of participants with an IGA PN-S score of 0 or 1 at Week 24. Other secondary endpoints included the proportion of patients achieving an Investigator's Global Assessment for PN-Activity (IGA PN-A) of 0 or 1, Dermatology Life Quality Index (DLQI) and Hospital Anxiety and Depression Scale (HADS). The safety of QX005N was also evaluated.

Results

A total of 206 participants received QX005N and 206 received placebo. Baseline demographics and disease characteristics were well balanced in two groups. At week 24, a significantly more patients in QX005N group achieved an itch improvement than in placebo group(74.5% vs.29.0%; difference, 45.67%; 95% confidence interval [CI], 37.00% to 54.35%). Additionally, more patients in QX005N group achieved IGA PN-S of 0 or 1 (34.7% vs. 11.1%; difference, 23.77%; 95% CI, 15.76% to 31.77%). Significant benefits were also observed across other secondary end points, including IGA PN-A of 0 or 1 at week 24 (37.9% vs. 10.2%), and improvements in DLQI (-10.07 vs. -6.81) and HADS (-4.57 vs. -2.04) from baseline at week 24 (P<0.0001 for all comparisons). The treatment emergent adverse events(TEAEs) were comparable between two groups (80.1% vs. 77.2%). Serious adverse events (SAEs) were reported in 4.9% of QX005N group and 5.3% of placebo group. The most frequently reported events were upper respiratory tract infection (QX005N vs. placebo□18.4% vs. 18.0%), hyperuricemia (9.2% vs. 6.8%), and red blood cell sedimentation rate increased (7.3% vs. 4.9%). Overall, QX005N demonstrated a favorable safety profile. The safety results were consistent with those from previous studies of QX005N, and no new safety signals were identified.



Proportion of patients achieving a WI-NRS improvement of ≥ 4 points from baseline across 24 weeks

Conclusions

QX005N 450 mg Q2W for 24 weeks demonstrated a high efficacy and a good safety profile in adult patients with PN.

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Abstract N°: 1392

Three-year Treatment Persistence and Durable Response to Dupilumab in Palmoplantar Pustulosis: Real-world Longitudinal Follow-up from a Single Centre

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Introduction
Palmoplantar pustulosis (PPP) remains a therapeutic challenge because of its chronic relapsing course and the limited durability of many available treatments. Although early clinical benefit of dupilumab has been observed in PPP, evidence regarding longer-term treatment retention and maintenance of response remains scarce. We therefore evaluated the long-term clinical course of dupilumab-treated PPP in an extended real-world follow-up of a previously characterised cohort.

Materials and Methods
This longitudinal observational study included 10 patients with histologically confirmed PPP who had previously entered a prospective dupilumab study at our institution. Ongoing follow-up was performed at a tertiary dermatology centre between April 2021 and April 2025. Dupilumab was started at 300 mg every 4 weeks, and most patients remained on this schedule throughout follow-up. One patient later adopted an 8-week interval because of financial constraints after disease stabilisation. No concomitant systemic therapy or corticosteroid treatment was given during dupilumab exposure. The main outcomes were maintenance of PPPASI75 and treatment persistence over time, the latter assessed by Kaplan-Meier analysis.

Results
One of the 10 patients discontinued dupilumab within 3 months because of lack of clinical improvement. Among the remaining 9 early responders, 7 continued treatment and preserved PPPASI75 for at least 3 years, corresponding to 77.8% of responders. Mean treatment duration was 40 months, and the longest observed duration of disease control was 48 months. When analysed across the full original cohort, the proportions maintaining PPPASI75 at years 1, 2 and 3 were 90%, 80% and 80%, respectively. Two patients stopped treatment after the initial response period: one because of waning efficacy at 16 months, and one for economic reasons after the end of the 1-year research phase. The latter remained stable for a prolonged period off treatment and regained PPPASI75 within 1 month after dupilumab was reintroduced following relapse. Safety findings were limited, with only one mild injection-site reaction recorded during follow-up.

Conclusions
In this extended real-world cohort, dupilumab showed sustained treatment persistence and prolonged disease control in a substantial proportion of patients with PPP. The observation that clinical benefit could be re-established after treatment interruption in one patient further supports the biological activity of this approach in selected cases. These findings add long-term practice-based evidence for dupilumab in PPP, although confirmation in larger controlled cohorts is still required.

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Abstract N°: 1400

Lebrikizumab as an effective therapeutic alternative in a frail patient with refractory prurigo nodularis

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Introduction

Prurigo nodularis (PN) is a chronic, highly burdensome neuroinflammatory disease characterized by severe pruritus and hyperkeratotic nodules, driven by complex neuroimmune dysregulation with a central role for type 2 cytokines, particularly interleukin (IL)-13. Although new targeted therapies have emerged, management of refractory PN remains challenging, especially in elderly patients with significant comorbidity, in whom therapeutic options are often limited. We report the clinical response to selective IL-13 inhibition with lebrikizumab in a frail patient with severe, treatment-refractory PN

Case Presentation

A 70-year-old woman with biopsy-confirmed PN was evaluated. The disease was refractory to multiple therapies, including high-potency topical corticosteroids, methotrexate (17.5 mg/week), and intensified dupilumab. Baseline severity was assessed using the Investigator's Global Assessment (IGA) and numerical rating scales (NRS) for pruritus and sleep disturbance. Given the patient's significant cardiovascular comorbidity (hypertension, prior myocardial ischemia, and hypercholesterolemia), Janus kinase inhibitors were considered unsuitable. Lebrikizumab was initiated at standard dosing (500 mg at weeks 0 and 2, followed by 250 mg every 2 weeks). Clinical outcomes were assessed at week 12.

At baseline, disease severity was high (IGA 4; pruritus NRS 10; sleep NRS 10), with marked impairment in quality of life. At week 12, the patient achieved a rapid and near-complete clinical response, with resolution of nodular lesions and only residual post-inflammatory hyperpigmentation. Patient-reported outcomes improved substantially (IGA 0; pruritus NRS 0; sleep NRS 0). Treatment was well tolerated, with no relevant adverse events.

Conclusion

Selective IL-13 blockade with lebrikizumab led to rapid, complete disease control in a frail, elderly patient with PN refractory to both conventional systemic therapy and dupilumab. Importantly, it demonstrated an excellent safety profile in a context of high cardiovascular comorbidity, where alternative systemic options were limited. This case highlights IL-13 as a critical therapeutic target and supports lebrikizumab as a promising and potentially practice-changing option in difficult-to-treat PN. Further studies are warranted to confirm these findings in broader populations.

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Abstract N°: 1416

Persistence, Effectiveness, Safety, and Patient Reported Impact of Guselkumab and IL-17 Inhibitors in Psoriatic Arthritis: Full Population Results of the PsABIOnd Global Observational Study Over 12 Months

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Introduction

Guselkumab (IL-23 inhibitor [i]; GUS) and IL-17i showed early and durable efficacy in randomized controlled trials (RCTs) in psoriatic arthritis (PsA), improving clinical-and-patient-reported outcomes (PROs). However, real-world longer-term data are limited. Interim analysis of the PsABIOnd study showed similar 6-month (M) treatment (tx) persistence and effectiveness with GUS and IL-17i. Here we assessed 12M tx persistence, effectiveness, safety, and PROs of GUS and IL-17i.

Materials and Methods

PsABIOnd (NCT05049798) is a global observational study in participants (pts)with PsA starting GUS or IL-17i as 1st to 4th line of biologic therapy. Primary outcome is tx persistence. Ptswith a baseline (BL) and ≥1 follow-up assessment up to the 12M visit (±3M) were analyzed according to their initial tx. Tx persistence was assessed via Kaplan-Meier. Propensity score (PS) analysis evaluated hazard ratio (HR) of stopping/switching GUS vs IL-17i prior to the 12M visit, adjusting for BL variable imbalances. Other assessments included achievement of low disease activity (LDA)/remission (REM) by cDAPSA (≤13; among pts with BL cDAPSA >13) and DAPSA (≤14; among pts with BL DAPSA>14), psoriasis (PsO) body surface area (BSA)<3% and Dermatology Life Quality Index (DLQI) 0/1 (among pts with BSA≥3% at BL), minimal disease activity (MDA; among MDA non-achievers at BL), mean changes from BL in Leeds Enthesitis Index (LEI) and number of digits affected by dactylitis and nails affected by PsO (in pts affected at BL). PROs were assessed using PsAID-12 (0–10) questionnaire. Adverse events (AEs)

were assessed up to 12M.

Results

Of 1,141 pts, 562 and 579 received GUS or IL-17i, respectively, as their initial tx. BL characteristics of the GUS/IL-17i cohorts were generally well balanced (mean age 53.1/53.4 y; mean BMI 30.0/29.4 kg/m^2; 63.5%/62.7% pts had received ≥1 targeted therapy); however, a higher proportion of GUS pts were initiating their 4th targeted tx line (15.3% vs 7.9% with IL-17i). Tx persistence up to 12M was high in both cohorts, with 427/562 (76.0%) GUS and 446/579 (77.0%) IL-17i pts remaining on their initial tx line (PS-adjusted HR GUS vs IL-17i stop/switch [95% CI] 0.89 [0.73–1.08]; **Figure**). Main reasons for initial tx discontinuation were primary failure (GUS/IL-17i: 11.7%/9.8%) and AEs (7.3%/9.2%).

Improvements in joint (GUS/IL-17i; cDAPSA LDA/REM: 45.0%/43.3%; DAPSA LDA/REM: 44.0%/43.9%), skin (BSA<3%: 77.7%/80.4%; DLQI 0/1: 53.4%/54.7%), and overall disease activity (MDA: 28.6%/29.0%) at 12M were similar with GUS and IL-17i. Mean changes from BL to 12M in LEI (-1.3/-1.5) and number of digits affected by dactylitis (-1.9/-2.1) and nails affected by PsO (-5.4/-4.9) were comparable. Rate of PsAID-12 minimal clinically important improvement (≥1.4) achievement (53.3%/49.6%) and mean improvement in PsAID-12 total (-1.7/-1.8) and item scores were similar with GUS and IL-17i.

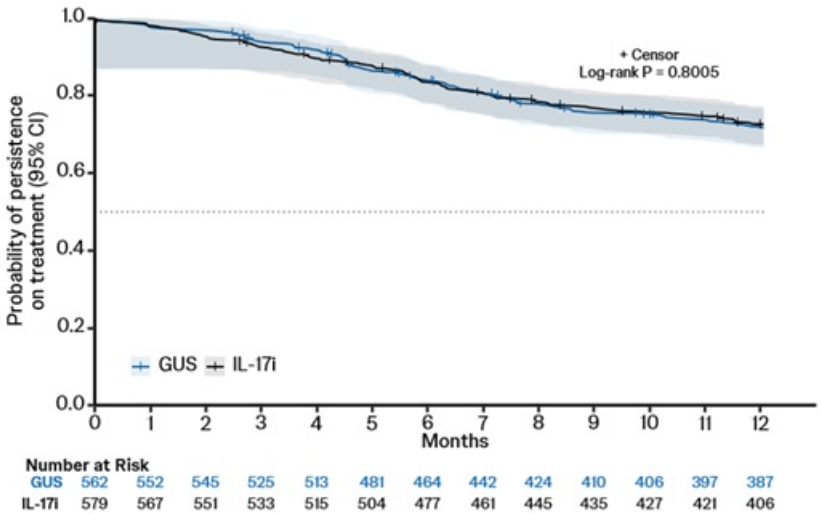
Rates of ≥1 AE and ≥1 serious AE (SAE) were similar between cohorts: for GUS, these were 53.3% (exposure-adjusted incidence rate [95% CI]: 116.6 [108.1, 125.5]/100 patient years [PYs]) and 6.8% (9.4 [7.1, 12.2]/100PYs), respectively; for IL-17i, 57.4% (156.0 [146.6, 165.9]/100PYs) and 6.9% (8.4 [6.3, 10.9]/100PYs). The most frequently observed AEs were COVID-19 infection (7.5% of GUS pts [7.4 (5.4, 9.9)/100PYs]; 5.6% [5.6 (4.0, 7.8)/100PYs] with IL-17i) and nasopharyngitis (5.3% of GUS pts [5.8 (4.0, 8.0)/100PYs]; 5.5% of IL-17i pts [6.9 (5.0, 9.2)/100PYs]). Gastrointestinal disorder-related AE occurred in 6.8% of GUS pts (7.8 [5.7, 10.3]/100PYs) and 11.2% of IL-17i pts (15.5 [12.7, 18.8]/100PYs). No cases of active tuberculosis occurred.

Conclusions

At 12M, pts with PsA treated with GUS or IL-17i had similar tx persistence, effectiveness across key PsA domains, and improvements in PROs with both therapies. Safety profiles were comparable, with no new safety signals. Both mechanisms of action appear to be effective for long-term PsA management in routine clinical practice, supporting data from RCTs.

Figure: Persistence on treatment over 12 months in PsA participants treated with GUS and IL-17i

Number at risk shows participants remaining under observation and switch/stop event-free at each time point; patients were right censored at their last observation or at study termination and were removed from these counts after their censoring time.



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Abstract N°: 1418

Bimekizumab treatment in routine clinical care for psoriatic arthritis: Interim observational evidence of patient-reported outcome improvements from the European, multicentre SPEAK study

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Introduction

Bimekizumab (BKZ), a monoclonal IgG1 antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated sustained improvements in patient-reported outcomes (PROs) up to 3 years in psoriatic arthritis (PsA) clinical trials.[1–3] SPEAK is the first prospective, observational, real-world study evaluating BKZ effectiveness on PROs in patients with active PsA or axial spondyloarthritis (axSpA) treated with BKZ in Europe.

Here, planned interim data from the ongoing SPEAK study are reported from patients with PsA, including baseline characteristics and BKZ effectiveness on PROs to 24 weeks (wks), stratified by prior treatment history. Safety data are also reported.

Materials and Methods

SPEAK is a 52-wk, multicentre, multi-country study of adult patients with PsA commencing BKZ treatment in routine clinical practice. Enrolment began in September 2023; this planned interim analysis reports data to April 2,

2025.

Baseline characteristics (**Table**) and change from baseline (CfB) in PROs up to 24 wks are reported, stratified by prior biological/targeted synthetic disease-modifying antirheumatic drug (b/tsDMARD) exposure. PROs reported as observed case: PsA Impact of Disease 12-Item Questionnaire (PsAID-12) total score (0–10), Patient Global Assessment of Disease Activity score (PGADA; 0–100), Short Form 36-item Health Survey (SF-36) Physical Component Summary (PCS).[4–6] Adverse events (AEs) were collected and reported in line with pharmacovigilance reporting guidelines.

Table. Baseline characteristics of patients with PsA in the SPEAK study

Characteristic, mean (SD) [n] unless otherwise stated	Prior b/tsDMARD exposure			Total N=376
	b/tsDMARD-naïve n=80	1 b/tsDMARD n=89	≥2 b/tsDMARDs n=207	
Age, years	51.6 (12.1)	50.8 (11.8)	53.2 (10.6)	52.3 (11.2)
Sex, male, n (%)	39 (48.4)	43 (48.3)	74 (35.7)	156 (41.5)
BMI, kg/m^2	29.3 (6.5) [79]	30.9 (6.4) [88]	29.5 (6.2) [205]	29.8 (6.3) [372]
Time since diagnosis, years	4.7 (6.0)	7.2 (8.0)	11.6 (9.2) [206]	9.1 (8.8) [375]
History of IBD, n (%)	0 (0)	1 (1.1)	4 (1.9)	5 (1.3)
History of uveitis, n (%)	1 (1.3)	1 (1.1)	4 (1.9)	6 (1.6)
Tender joint count	10.5 (8.3) [49]	10.2 (10.1) [64]	13.1 (12.3) [146]	11.9 (11.2) [259]
Swollen joint count	5.0 (4.9) [49]	6.7 (8.0) [64]	5.4 (6.6) [146]	5.7 (6.7) [259]
CRP, mg/L, median (min–max)	3.6 (0.0–46.0) [66]	3.3 (0.0–49.8) [67]	3.6 (0.0–112.0) [165]	3.6 (0.0–112.0) [298]
HAQ-DI score	1.02 (0.64) [60]	1.16 (0.75) [74]	1.37 (0.72) [162]	1.25 (0.73) [296]
PsAID-12 total score	5.0 (1.9) [67]	5.3 (2.2) [77]	5.5 (2.1) [178]	5.4 (2.1) [322]
PGADA score	57.0 (19.3) [63]	57.6 (23.3) [74]	61.1 (21.3) [170]	59.4 (21.4) [307]
SF-36 PCS score[a]	37.7 (8.1) [61]	36.4 (9.4) [71]	34.3 (9.3) [157]	35.6 (9.2) [289]
SF-36 PCS <45, n (%)	49 (61.3)[80][b]	57 (64.0)[c]	132 (63.8)[d]	238 (63.3)[e]
SF-36 MCS score[a]	44.3 (12.1) [61]	43.0 (13.2) [71]	40.9 (12.0) [157]	42.1 (12.4) [289]
SF-36 MCS <45, n (%)	32 (40.0)[b]	40 (44.9)[c]	96 (46.4)[d]	168 (44.7)[e]

Safety analysis set (includes all patients enrolled into the study). Where the number of patients with available data for a specific baseline characteristic differed from the overall population size, the number of patients with available data has been given in square brackets, [n]. [a] SF-36 PCS and MCS are norm-based T-scores with mean (SD) of 50 (10) in the US general population, where higher scores reflect better HRQoL. When an individual respondent's score falls outside the T-score range of 45 to 55, it should be considered outside the average range for the US general population. When considering group level data, a score below 47 should be considered indicative of impaired functioning;6 [b] 19 patients (23.8%) had missing scores at baseline; [c] 18 patients (20.2%) had missing scores at baseline; [d] 50 patients (24.2%) had missing scores at baseline; [e] 87 (23.1%) of patients had missing scores at baseline. BMI: body mass index; b/tsDMARD: biological/targeted synthetic disease-modifying antirheumatic drug; CRP: C-reactive protein; HAQ-DI: Health Assessment Questionnaire-Disability Index; IBD: Inflammatory Bowel Disease; PGADA: Patient Global Assessment of Disease Activity; PsA: psoriatic arthritis; PsAID-12: PsA Impact of Disease 12-item questionnaire; SD: standard deviation; SF-36 PCS/MCS: Short Form 36-Item Health Survey Physical Component Summary/Mental Component Summary; US: United States.

Results

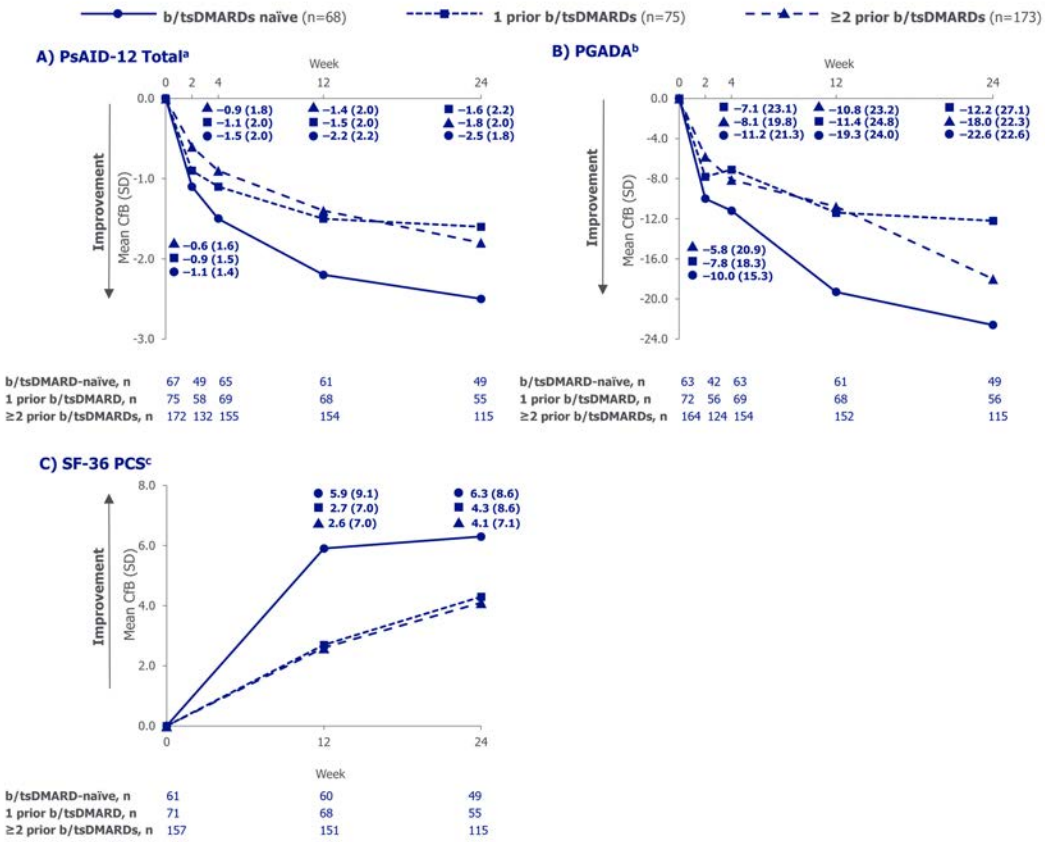
Of 376 patients with available baseline data, 80 (21.3%) were b/tsDMARD-naïve, 89 (23.7%) had 1 prior b/tsDMARD, and 207 (55.1%) had received ≥2 prior b/tsDMARDs, 10.6% (22/207) of whom had received ≥7 prior b/tsDMARDs.

Patients previously treated with ≥2 b/tsDMARDs had longer disease duration at baseline, and a greater proportion were female (**Table**). Baseline PsAID-12, PGADA, SF-36 and HAQ-DI scores were progressively worse with increasing number of prior treatment lines, suggesting greater patient-reported impairment at baseline for patients starting BKZ as a later line therapy.

Improvements in PsAID-12 and PGADA were observed as early as 2 wks after BKZ initiation regardless of treatment line and continued to improve to Wk 24 (**Figure 1A–B**). SF-36 PCS scores also improved to Wk 24 (**Figure 1C**). For PsAID-12, PGADA and SF-36 PCS, improvements across outcomes were numerically greater in b/tsDMARD-naïve patients than those with prior exposure.

AEs were reported in 61.4% of patients; drug-related AEs in 34.8% of patients. One patient (0.3%) reported uveitis, deemed not drug-related. *Candida* infections reported by 43 patients (11.4%); 46 cases in total with 31 oral candidiasis cases. Serious infections reported by 15 patients (4.0%). One patient (0.3%) reported a major adverse cardiovascular event (myocardial infarction), deemed not drug-related. No cases of venous thromboembolism, serious hypersensitivity, anaphylaxis reactions, inflammatory bowel disease or suicidal ideation/behaviour reported.

Figure 1. Change from baseline in (A) PsAID-12, (B) PGADA and (C) SF-36 PCS scores in patients with PsA stratified by prior treatment history, to Week 24 (OC)



Full analysis set. Values were rounded to 1 decimal place. Dates are approximate depending on how close to baseline the first dose was received. [a] PsAID-12 total score ranges from 0–10 (higher scores indicate worse status);4 [b] PGADA in this study was assessed using a 0–100 VAS (higher scores indicate higher disease activity);5 [c] The SF-36 PCS is a norm-based T-scores with mean (SD) of 50 (10) in the general US population, where higher scores reflects better HRQoL.6 b/tsDMARD: biological/targeted synthetic disease-modifying antirheumatic drug; CfB: change from baseline; HRQoL: health-related quality of life; OC: observed case; PGADA: Patient Global Assessment of Disease Activity; PsA: psoriatic arthritis; PsAID-12: PsA Impact of Disease 12-item questionnaire; SD: standard deviation; SF-36 PCS: Short Form 36-Item Health Survey Physical Component Summary; US: United States; VAS: visual analogue scale.

Conclusions

Patients with PsA showed improvements in PROs, including HRQoL, as early as Wk 2 after BKZ treatment initiation, which showed steady improvement through Wk 24. Improvements were seen across all prior treatment lines, with

numerically greater changes observed in b/tsDMARD-naïve patients, a group characterised by better baseline HRQoL and shorter disease duration. These preliminary findings suggest that BKZ treatment as part of routine clinical practice may lead to improved PROs with no additional safety concerns, providing early evidence that the risk–benefit profile established in clinical trials may extend to real-world use.

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Abstract N°: 1421

SPECTREM: High-Impact Site Skin Clearance and Itch Improvement Through Week 48 With Guselkumab in Participants With Low Body Surface Area, Moderate Psoriasis

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Introduction
Psoriasis affecting high-impact sites (eg, scalp, face, intertriginous, or genitals) is often associated with pruritus, pain, and poor quality of life. However, these patients generally have only a low overall proportion of body surface area (BSA) affected, which has historically limited their access to advanced therapies. SPECTREM is a phase 3b, multicenter, randomized, double-blind, placebo-controlled study that evaluated guselkumab efficacy and safety in participants with low BSA (2–15%), moderate (Investigator's Global Assessment [IGA]=3) plaque psoriasis involving ≥1 high-impact site. Significant improvements in overall and site-specific skin clearance and patient-reported outcomes (PROs) were demonstrated with guselkumab at Week 16. This analysis reports SPECTREM results through Week 48 from the final database lock.

Materials and Methods
A total of 338 participants were randomized 2:1 to receive guselkumab 100 mg (Week 0, Week 4, then every 8 weeks [Q8W] through Week 44) or placebo (Week 0, Week 4, and Week 12 with guselkumab crossover at Week 16, Week 20, then q8w through Week 44) by subcutaneous injection. Skin clearance (absence of/very mild disease or clear/minimal) was evaluated based on achievement of scalp-specific (ss)-IGA 0/1, facial (f)-IGA 0/1, intertriginous (i)-IGA 0/1, and static Physician Global Assessment of Genitalia (sPGA-G) 0/1 among participants with baseline site-specific IGA/PGA ≥3. Reduction in Psoriasis Symptoms and Signs Diary (PSSD) itch score, with a reduction of ≥4 points being clinically meaningful, was assessed in those with a baseline score ≥4.

Results
Among 225 guselkumab- and 113 placebo-randomized participants, improvements in skin clearance were either maintained or increased from Week 16 through Week 48 in the guselkumab group (response rates at Week 48 were: ss-IGA 0/1, 84.9% [129/152]; f-IGA 0/1, 86.7% [78/90]; i-IGA 0/1, 91.9% [102/111]; sPGA-G 0/1, 84.1% [69/82]). Improvements in PSSD itch scores were also either maintained or increased from Week 16 through Week 48 among guselkumab-randomized participants with baseline PSSD itch score ≥4 (73.6% [148/201] of participants achieved ≥4-point reductions in PSSD itch score by Week 48). Similar response rates were observed following crossover to guselkumab in the group of participants initially randomized to placebo. Guselkumab was generally

well tolerated. Safety findings through the final safety follow-up visit (Week 56) were consistent with those reported previously with no new guselkumab safety signals.

Conclusions

Guselkumab was well tolerated and provided sustained and meaningful improvements in site-specific skin clearance and PROs through Week 48 among participants with low BSA, moderate plaque psoriasis involving high-impact sites, with >80% of guselkumab-treated participants experiencing site-specific skin clearance.

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Abstract N°: 1427

Real-world, long-term persistence of guselkumab versus interleukin-17 inhibitors in patients with psoriasis: evidence through up to 6.5 years

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Introduction

Psoriasis Longitudinal Assessment and Registry (PSOLAR; NCT00508547) is a large, international, prospective, disease-based registry of patients with psoriasis (PsO) who are receiving, or are candidates for, systemic therapy. PSOLAR was initiated in 2007 and expanded in 2018 (PSOLAR-2) to include patients receiving guselkumab (GUS), an interleukin (IL)-23p19 inhibitor, or an IL-17 inhibitor (IL-17i) according to standard clinical practice.[1] Here, real-world time on therapy, and rates and reasons for discontinuation were explored for GUS and IL-17i incident user cohorts, as well as a subset of bionaïve patients.

Materials and Methods

Patients eligible for enrolment in PSOLAR-2 had a PsO diagnosis, were grouped into GUS users (any prior/current use) or IL-17i users (any prior/current use; no GUS use at/before enrolment), and were classified as incident users (starting treatment ≤30 days before/after enrolment visit; may have received prior GUS/IL-17i treatment but stopped >365 days before enrolment) or prevalent users (starting treatment >30 days prior to enrolment). Data were collected at enrolment and ~every 6 months thereafter.

Analyses presented here (data cut-off: 12 July 2025) use only data for the incident user cohort with follow-up through up to 6.5 years and censoring at treatment discontinuation or last recorded visit (whichever occurred first). Time on therapy was summarised using descriptive statistics. Kaplan-Meier (KM) curves were generated for time to GUS or IL-17i discontinuation. Discontinuations and reasons for discontinuations were described by treatment group. Analyses were repeated for the bionaïve incident subgroup.

Results

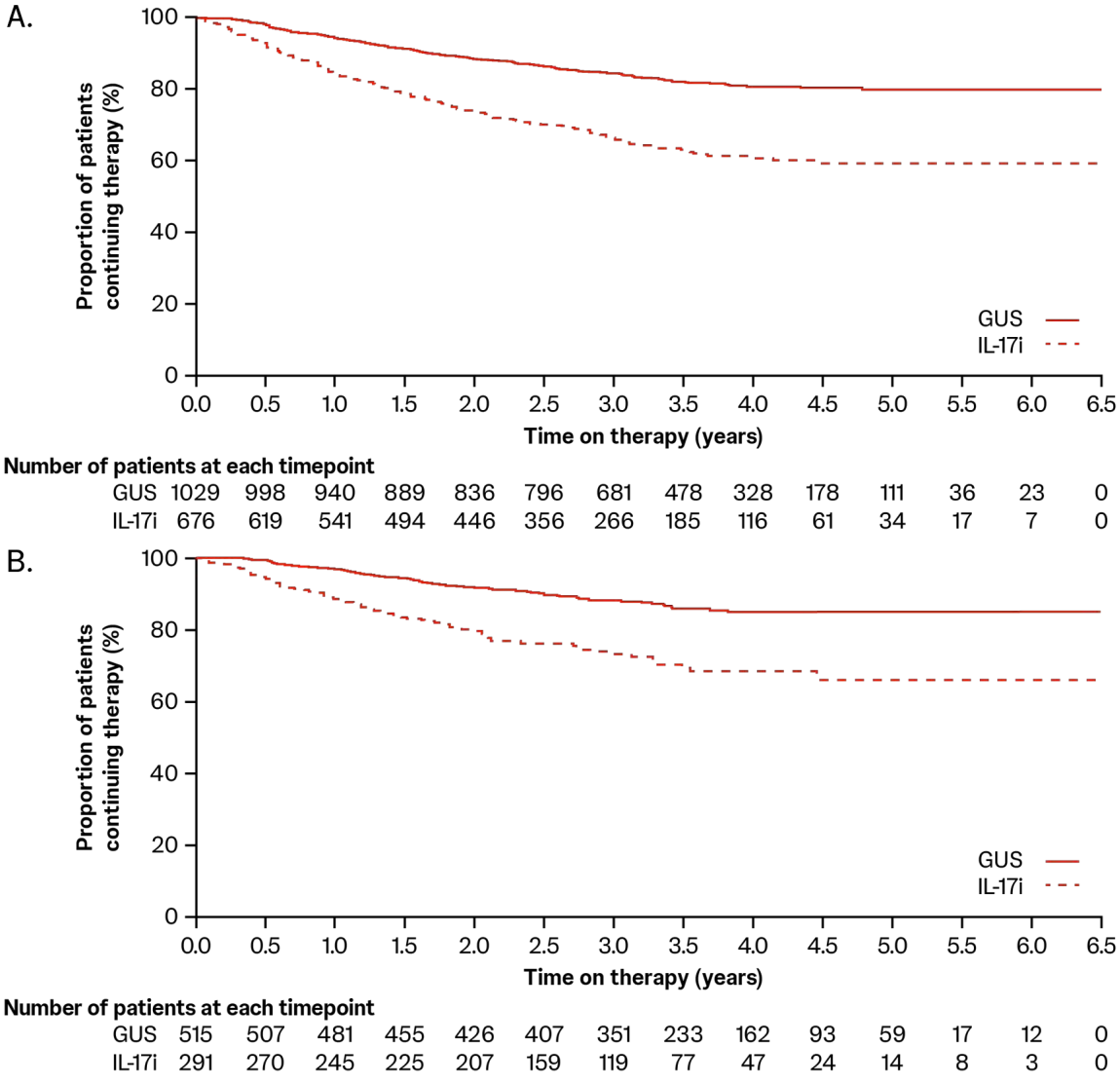
Overall, 3,501 patients (GUS: n=2,213; IL-17i: n=1,288) were enrolled across 17 countries. Baseline characteristics

were generally balanced between the GUS and IL-17i groups. The incident user cohort included 1,705 patients (GUS: 1,029; IL-17i: 676 [comprised of bimekizumab: 8.1%; brodalumab: 15.1%; ixekizumab: 44.7%; secukinumab: 32.1%]). Of these, 806 patients were bionaïve (GUS: 515; IL-17i: 291 [bimekizumab: 5.8%; brodalumab: 16.8%; ixekizumab: 42.3%; secukinumab: 35.1%]).

For the incident user cohort, mean (standard deviation [SD]) time on therapy was 3.3 (1.4) years for GUS and 2.6 (1.5) years for IL-17i. In this cohort, 178/1,029 (17.3%) patients treated with GUS stopped/switched therapy compared to 229/676 (33.9%) patients treated with IL-17i. Time to discontinuation KM curves are shown in **Figure 1A**. Of the reasons for discontinuation provided, loss/lack of efficacy was the most common given for both treatment groups (GUS: 90/158 [57.0%]; IL-17i: 136/208 [65.4%]), followed by patient choice (23/158 [14.6%]) and insurance/reimbursement issue or other (both 14/158 [8.9%]) for GUS, and adverse events (19/208 [9.1%]) and insurance/reimbursement issue or other (both 16/208 [7.7%]) for IL-17i.

For the bionaïve cohort, mean (SD) time on therapy was 3.4 (1.3) years for GUS and 2.7 (1.4) years for IL-17i. As in the incident user cohort, a lower proportion of patients treated with GUS (66/515 [12.8%]) stopped/switched therapy compared with patients treated with IL-17i (78/291 [26.8%]). Time to discontinuation KM curves are shown in **Figure 1B**. Loss/lack of efficacy was the most common reason for discontinuation in both treatment groups (GUS: 28/58 [48.3%]; IL-17i: 45/71 [63.4%]), followed by patient choice or other (both 8/58 [13.8%]) and insurance/reimbursement issue (6/58 [10.3%]) for GUS, and adverse events (9/71 [12.7%]) and other (7/71 [9.9%]) for IL-17i.

Figure 1. Kaplan-Meier curves for time to therapy discontinuation



A. Incident user cohort; B. Bionaïve cohort. GUS: guselkumab; IL-17i: interleukin-17 inhibitor.

Conclusions

GUS was associated with longer treatment persistence and lower discontinuation rates than IL-17i in both the incident user and bionaïve cohorts of PsO patients, with loss/lack of efficacy being the most common reason for discontinuation for both GUS and IL-17i.

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Bimekizumab demonstrated sustained improvements in pain and fatigue in patients with axial spondyloarthritis achieving early control of disease activity: 3-year results from two phase 3 studies and their open-label extension

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Introduction

Axial spondyloarthritis (axSpA), an interleukin (IL)-17-mediated chronic inflammatory disease mainly affecting the sacroiliac joints and spine,[1] can co-occur with other IL-17-mediated diseases such as psoriasis and hidradenitis suppurativa.[2,3]

Patients with axSpA experience hallmark symptoms such as spinal pain and fatigue, which negatively impact their daily life.[1,2] Controlling these symptoms is therefore important to maximise patients' health-related quality of life.[1]

Bimekizumab (BKZ), a humanised monoclonal IgG1 antibody that selectively inhibits IL-17F in addition to IL-17A, has demonstrated efficacy at Week 16 which was sustained to 3 years in phase 3 trials for non-radiographic (nr-)axSpA and radiographic (r-)axSpA, and their combined open-label extension (OLE).[4,5] BKZ has also demonstrated efficacy in psoriatic arthritis, plaque psoriasis and hidradenitis suppurativa.[6–8]

Here, the impact of early control of disease activity at Week 16 of BKZ treatment, as measured by the achievement of Axial Spondyloarthritis Disease Activity Score (ASDAS) low disease activity (i.e., ASDAS <2.1), on long-term pain and fatigue outcomes is reported over 3 years in patients with axSpA.

Materials and Methods

BE MOBILE 1 (NCT03928704; nr-axSpA) and BE MOBILE 2 (NCT03928743; r-axSpA) each comprised a 16-week double-blind period followed by a 36-week maintenance period.[4] Patients were randomised to receive subcutaneous BKZ 160 mg every 4 weeks (Q4W) or placebo (PBO); from Week 16, all patients received BKZ 160 mg Q4W. At Week 52, eligible patients could enter the OLE (NCT04436640) and continue receiving BKZ up to Week 164.

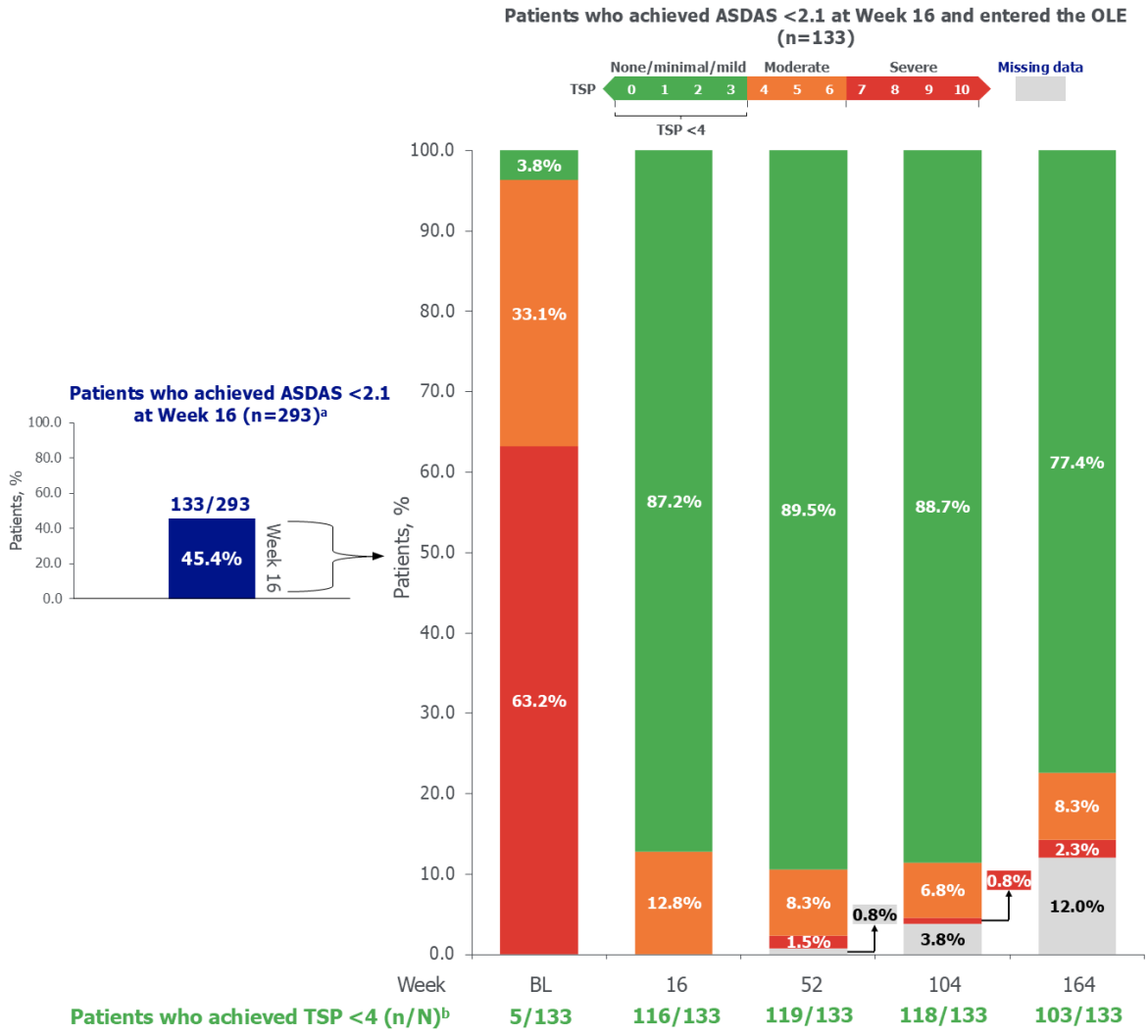
Pain and fatigue were measured by total spinal pain (TSP) and Bath Ankylosing Spondylitis Disease Activity (BASDAI) Q1, respectively. Both measures used a 0–10 point numeric rating scale, with higher scores reflecting higher levels of pain/fatigue. Proportions of patients achieving mild pain (TSP <4) or mild fatigue (BASDAI Q1 <4)[9] are presented up to Week 164 for BKZ-randomised patients (pooled across studies) who achieved early control of disease activity (ASDAS <2.1) at Week 16 and entered the OLE.

Results

In total, 128 patients with nr-axSpA and 221 patients with r-axSpA were randomised to BKZ across BE MOBILE 1 and 2, respectively (N=349). Of 293 BKZ-randomised patients who entered the OLE, 133 (45.4%) achieved early control of disease activity (ASDAS <2.1) at Week 16. At baseline, almost all patients had moderate or severe pain (TSP ≥4; 128/133 [96.2%]) or fatigue (BASDAI Q1 ≥4; 129/133 [97.0%]; **Figure 1 and 2**).

Most patients who achieved ASDAS <2.1 at Week 16 and entered the OLE achieved mild pain scores (TSP <4) at Weeks 16, 52, 104 and 164 (87.2%, 89.5%, 88.7% and 77.4%, respectively; **Figure 1**). Additionally, a high proportion of BKZ-randomised patients who achieved ASDAS <2.1 at Week 16 and entered the OLE achieved mild fatigue (BASDAI Q1 <4) at Weeks 16, 52, 104 and 164 (82.0%, 78.9%, 89.5% and 76.7%, respectively; **Figure 2**).

Figure 1. TSP score to Week 164 in BKZ-randomised patients who achieved ASDAS <2.1 at Week 16 and entered the OLE



Pooled randomised set. Only patients who were randomised to BKZ at baseline and entered the OLE

were included in this analysis. [a] At Week 16, 133/293 (45.4%) patients who entered the OLE achieved ASDAS <2.1; [b] n/N represents the proportion of patients who achieved TSP <4 at each corresponding timepoint out of all patients who achieved ASDAS <2.1 at Week 16 and entered the OLE (n=133). A missing data category is included for BKZ-randomised patients who entered the OLE and had a missing assessment at a given timepoint. ASDAS: Axial Spondyloarthritis Disease Activity Score; BKZ: bimekizumab; BL: baseline; OLE: open-label extension; TSP: total spinal pain.

Conclusions

Sustained improvements in pain and fatigue were observed through Week 164 of bimekizumab treatment among patients with axSpA who achieved early control of disease activity (ASDAS <2.1) at Week 16. These findings suggest that patients who achieve early control of disease activity with bimekizumab may experience long-term sustained benefits across domains relevant to patients, such as pain and fatigue, over 3 years.

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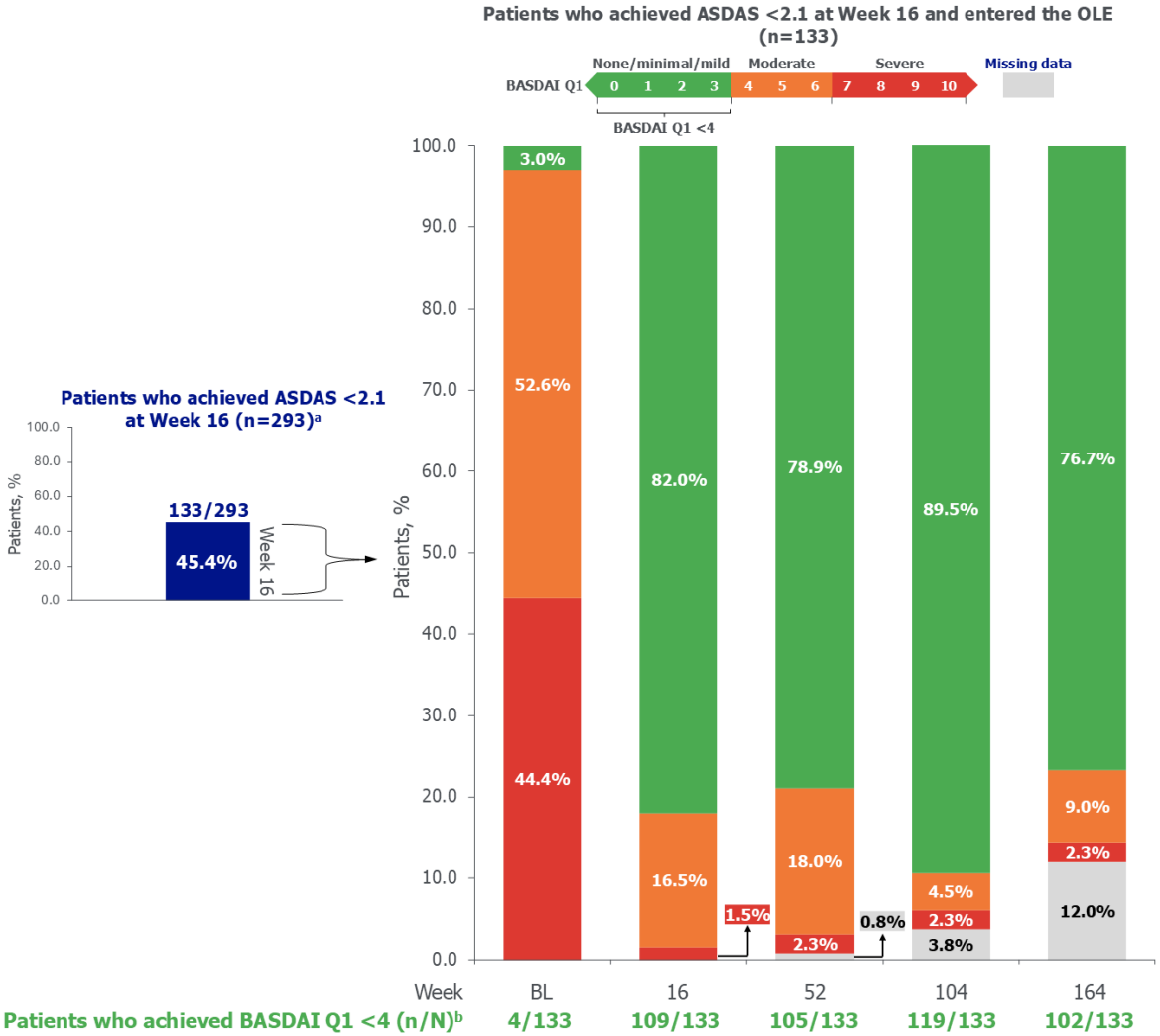
Figure 2. BASDAI Q1 (fatigue) score to Week 164 in BKZ-randomised patients who achieved ASDAS <2.1 at Week 16 and entered the OLE



Pooled randomised set. Only patients who were randomised to BKZ at baseline and entered the OLE were included in this analysis. [a] At Week 16, 133/293 (45.4%) patients who entered the OLE achieved ASDAS <2.1; [b] n/N represents the proportion of patients who achieved BASDAI Q1 <4 at each

corresponding timepoint out of all patients who achieved ASDAS <2.1 at Week 16 and entered the OLE (n=133). A missing data category is included for BKZ-randomised patients who entered the OLE and had a missing assessment at a given timepoint. ASDAS: Axial Spondyloarthritis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BKZ: bimekizumab; BL: baseline; OLE: open-label extension; Q: question.

EADV CONGRESS
VIENNA
30 SEP - 3 OCT 2026

Abstract N°: 1438

Dual Targeting of Th17 and Th2 Pathways with Secukinumab and Dupilumab in Adult Netherton Syndrome: A Case Report with Trichoscopic Diagnostic Insight

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Introduction

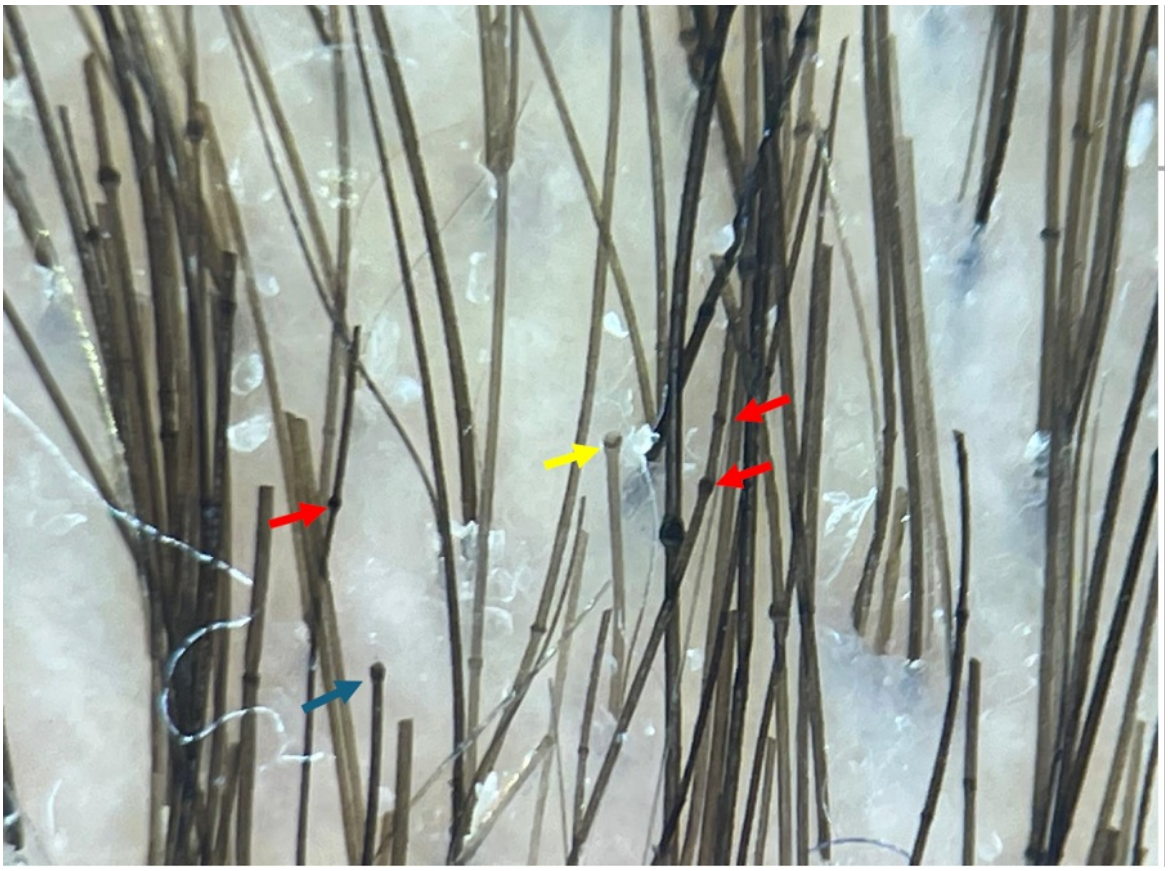
Netherton syndrome (NS) is a rare autosomal recessive genodermatosis caused by biallelic loss-of-function mutations in SPINK5, encoding the serine protease inhibitor LEKTI. LEKTI deficiency leads to unopposed kallikrein-related peptidase activity, profound skin barrier disruption, and a complex mixed inflammatory state involving the Th17/IL-36 and Th2 axes. NS is clinically defined by a diagnostic triad of ichthyosiform erythroderma, trichorrhexis invaginata ("bamboo hair"), and atopic manifestations with markedly elevated serum IgE. Despite this distinct molecular basis, NS is frequently misdiagnosed as severe atopic dermatitis, resulting in diagnostic delays spanning years to decades. Evidence for biologic combination therapy in adults remains extremely limited.

Here we report an adult patient with NS in whom trichoscopy was the decisive diagnostic tool and sustained clinical control was achieved with combined secukinumab and dupilumab therapy over approximately 10 months of follow-up.

Case Presentation

A 29-year-old male with more than a decade of misdiagnosis as atopic dermatitis presented with generalized erythema, scaling, and intractable pruritus (NRS 9/10; IGA 4; NASA 40.3/72; serum IgE 1514 IU/mL). Trichoscopy revealed the full spectrum of NS-associated hair shaft abnormalities: trichorrhexis invaginata with the pathognomonic "ball-in-cup" appearance, golf tee hairs, and matchstick hairs. The clinical diagnosis of NS was established based on the complete diagnostic triad. Compound heterozygous pathogenic SPINK5 variants were confirmed on molecular genetic analysis: a pathogenic splice site variant in intron 15

(c.1431-12G>A) and a likely pathogenic synonymous variant in exon 11 (c.891C>T), both classified according to ACMG criteria. Skin biopsy demonstrated subacute spongiotic dermatitis, underscoring the diagnostic limitation of histopathology in NS. Secukinumab (anti-IL-17A, 300 mg SC) was initiated and produced significant improvement (NRS ~5/10; IGA 2; NASA 28/72). Five months after secukinumab initiation, due to recurrence of skin lesions, dupilumab (anti-IL-4Rα, 300 mg SC q2w following 600 mg loading dose) was added, targeting the incompletely suppressed Th2 component of the patient's immune profile. Combination therapy yielded further sustained improvement (NRS 1/10; IGA 1; NASA 18/72) over approximately 10 months of real-world follow-up. No serious adverse events occurred.



Trichoscopy of the scalp demonstrating multiple pathognomonic hair shaft abnormalities of Netherton syndrome. Red arrows indicate trichorrhexis invaginata (bamboo hair) — the characteristic nodal swelling caused by intussusception of the distal hair shaft into the proximal portion. Yellow arrow indicates a "golf tee" hair — a cup-shaped proximal hair shaft remnant resulting from breakage at the invagination site. Blue arrow indicates a "matchstick" hair — a broken hair shaft with a bulging distal end. Peripilar white scaling is also present throughout.

Conclusion

Trichoscopy — including identification of the full spectrum of hair shaft abnormalities — is an essential diagnostic tool in erythrodermic patients with elevated IgE. Combined IL-17A and IL-4/IL-13 blockade provided sustained multi-measure clinical improvement in this adult NS case. To our knowledge, this represents one of the few adult real-world reports of this dual biologic combination, with a follow-up duration exceeding that of previously reported adult case.

EADV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 1440

Abrocitinib in the Treatment of Acquired Reactive Perforating Collagenosis Associated with Diabetes Mellitus: Two Case Reports

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Introduction

Reactive perforating collagenosis (RPC) is a rare primary perforating dermatosis characterized by the transepidermal elimination of degenerated collagen, first described by Mehregan et al. in 1967[1]. When developing in adulthood, this condition is frequently associated with underlying systemic diseases, and is referred to as acquired reactive perforating collagenosis (ARPC) [2-4]. The pathogenesis of ARPC remains unclear and no specific curative therapy exists for this disorder. Clinical management primarily focuses on alleviating pruritus and controlling potential complications. Abrocitinib is a Janus kinase 1 (JAK1) inhibitor that selectively inhibits JAK1 through competitive antagonism and blockade of adenosine triphosphate binding, thereby suppressing inflammatory cytokine signaling and significantly relieving pruritus. It is currently indicated mainly for the treatment of atopic dermatitis[5]. Herein, we report two cases of ARPC treated with Abrocitinib, aiming to provide new insights for the clinical management of ARPC.

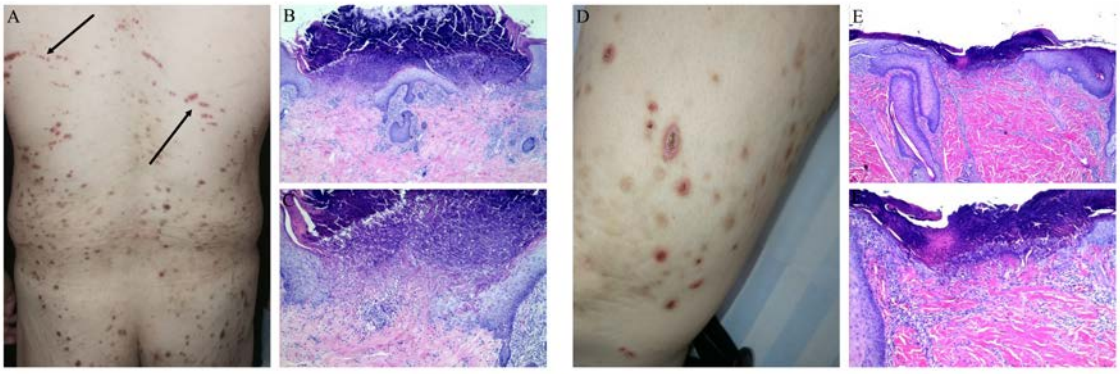
Case Presentation

Case 1: A 48-year-old male had >3 years of scattered erythematous papules on trunk/extremities with marked pruritus, worsening over the past year. He had an 11-year history of diabetes mellitus (poor glycemic control over the past year), stage 3 diabetic nephropathy for 2 years, diabetic peripheral vascular disease for 1 year, and a cerebral infarction 2 years prior. Family history of diabetes (father) but no similar skin lesions. Physical exam: densely distributed papules on extremities/trunk; some lesions had central umbilication with keratotic plugs (largest 3.6 cm). Linear arrangement suggested Koebner phenomenon. Some lesions coalesced into plaques; others resolved leaving faint brownish scars (Figure 1A). Labs: fasting glucose 10 mmol/L, postprandial 14 mmol/L, 24h urinary microalbumin >50 mg/24h, and urinary microalbumin >50 μg/mL. Histopathology: cup-shaped epidermal depression with keratotic plug, epithelial hyperplasia at margins, focal transepidermal elimination of degenerated collagen fibers with lymphocytic infiltrate (Figure 1B, C). Diagnosis: ARPC.

Case 2: A 45-year-old male had >3 years of erythematous papules on the right lower extremity with intense pruritus. He had >10 years of diabetes mellitus (poor control) and >6 years of coronary heart disease. No family history of diabetes. Physical exam: densely distributed erythematous papules on the right lower extremity; some lesions had central depression with visible plugs (max diameter 0.5 cm). Linear arrangement suggested Koebner phenomenon. Some lesions coalesced into plaques; others resolved leaving faint brownish scars (Figure 1D). Labs: fasting glucose 14.8 mmol/L. Histopathology: epidermal defect with cup-shaped depression containing abundant keratotic material, epithelial hyperplasia at edges, and focal transepidermal elimination of degenerated collagen fibers with lymphocytic infiltrate (Figure 1E, F). Diagnosis: ARPC.

Management of ARPC is challenging. Current goals are alleviating pruritus and controlling underlying conditions. Topical/systemic glucocorticoids are common. In both cases, patients had poor glycemic control, marked pruritus, and scratching. Given glucocorticoids' potential adverse effects on glucose metabolism and the antipruritic

efficacy of Abrocitinib, both patients received oral Abrocitinib (100 mg once daily) for one month after excluding contraindications, alongside standardized glycemic control. Regular follow-up for three months showed marked pruritus relief and skin lesion improvement. No recurrence or significant adverse effects were noted.



Conclusion

To the best of our knowledge, this is the first report demonstrating the therapeutic efficacy of Abrocitinib in the management of diabetes mellitus-associated ARPC. Our findings suggest that Abrocitinib may represent a potential therapeutic option for diabetes-associated ARPC.

EA DV CONGRESS
VIENNA
30 SEP - 3 OCT 2026

Abstract N°: 1481

Targeting Type 2 Inflammation in Darier Disease: Objective Clinical Response to Dupilumab in a Retinoid-Refractory Case

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Introduction

Darier disease (DD; OMIM #124200) is a rare autosomal dominant genodermatosis (prevalence 1:30,000–1:100,000) caused by loss-of-function mutations in the ATP2A2 gene encoding SERCA2, leading to impaired keratinocyte calcium homeostasis, acantholysis, and dyskeratosis. Clinically, DD manifests as chronic pruritic hyperkeratotic papules in seborrhoeic distributions, with exacerbations triggered by heat, UV, and sweating. Systemic retinoids represent the current standard of care for moderate-to-severe disease; however, their long-term use is frequently limited by dyslipidaemia, hepatotoxicity, and musculoskeletal adverse effects. Recent transcriptomic studies have identified upregulation of type 2 (IL-4/IL-13) cytokine pathways and epidermal barrier dysfunction in lesional DD skin, features shared with atopic dermatitis, providing a rationale for targeted biological therapy. Dupilumab, a fully human monoclonal antibody blocking the shared IL-4Rα subunit, has demonstrated efficacy in atopic dermatitis and, in limited reports, in related acantholytic genodermatoses.

Here we report a case of severe, biopsy-confirmed, retinoid-refractory Darier disease achieving marked clinical improvement with dupilumab, with objective outcome data at six months.

Case Presentation

A 52-year-old male presented with a 20-year history of symmetric, relapsing, pruritic hyperkeratotic papules involving the abdomen, lateral trunk, presternal region, and back — consistently worsening in summer — with a positive maternal family history. Comorbidities included COPD and hypertension, both stably managed throughout. Histopathological examination of a punch biopsy confirmed DD, demonstrating surface hyperkeratosis, intraepidermal acantholysis, and dyskeratotic cells (corps ronds). Baseline assessment: NRS 8/10, IGA 3, BSA ~18%, with nocturnal awakening due to pruritus. Acitretin 25 mg/day was initiated; after six months no clinical benefit was observed and treatment was discontinued due to hypercholesterolaemia (total cholesterol 244 mg/dL, LDL 159 mg/dL, triglycerides 169 mg/dL), elevated creatine kinase (292 U/L; reference ≤171 U/L), and back pain. Pre-biological workup was unremarkable. Dupilumab was initiated at a 600 mg loading dose, followed by 300 mg subcutaneously every two weeks. At six-month follow-up: NRS decreased from 8 to 1/10; IGA improved from 3 to 1; BSA reduced from ~18% to ~5%; nocturnal awakening fully resolved. No injection-site reactions, ophthalmological symptoms, or infectious complications were recorded.

Conclusion

This case of biopsy-confirmed, retinoid-refractory DD demonstrates clinically meaningful and objectively quantified improvement following six months of dupilumab, without adverse events. The simultaneous metabolic complications of acitretin — dyslipidaemia and CK elevation — in a patient with pre-existing cardiovascular risk factors highlight the clinical relevance of a retinoid-sparing approach. The response to IL-4/IL-13 blockade

supports the role of type 2 inflammation in DD pathogenesis and is consistent with emerging case series data. Dupilumab may represent a valuable therapeutic option in severe or retinoid-refractory DD; prospective controlled studies with standardised outcome measures are warranted.

EA DV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 1488

Targeted Systemic Therapies for Head and Neck Atopic Dermatitis: A Systematic Review and Meta-Analysis of Efficacy and Safety

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Introduction
Head and neck atopic dermatitis (AD) represents a distinct and therapeutically challenging phenotype, often characterized by persistent inflammation, increased disease severity, and reduced responsiveness to conventional therapies. This anatomical subtype is frequently associated with a higher psychosocial burden due to its visible location and impact on quality of life. Although targeted systemic therapies—including biologics and Janus kinase inhibitors—have transformed the management of moderate-to-severe AD, their effectiveness and safety specifically in head and neck involvement remain incompletely understood.

Materials and Methods
A systematic review and meta-analysis were conducted in accordance with PRISMA guidelines. Multiple electronic databases were systematically searched to identify randomized controlled trials and observational studies reporting outcomes in patients with head and neck AD treated with targeted systemic therapies, including dupilumab, tralokinumab, and upadacitinib. Primary outcomes included head and neck Eczema Area and Severity Index (H&N EASI-75) response and percentage change in EASI. Secondary outcomes included pruritus Numerical Rating Scale (NRS), Dermatology Life Quality Index (DLQI), and safety outcomes. A random-effects model was applied to account for inter-study variability. Sensitivity analyses, subgroup analyses by treatment type, and assessment of heterogeneity were performed to evaluate the robustness of finding

Results
Twelve studies were included,Overall, targeted systemic therapies demonstrated robust and clinically meaningful efficacy in head and neck AD. The pooled H&N EASI-75 response rate was **51% (95% CI: 40–62)**, indicating that approximately half of patients achieved significant clinical improvement. A marked reduction in disease severity was observed, with a pooled mean percentage change in EASI of **−72.86% (95% CI: −78.98 to −66.74)**, reflecting substantial improvement in inflammatory burden. Patient-reported outcomes also showed consistent benefit, with a mean reduction in pruritus scores of **−4.38**, indicating clinically meaningful itch relief, and a mean improvement in DLQI of **−9.97**, demonstrating significant enhancement in quality of life. Comparative subgroup analyses revealed differences across therapies, with **upadacitinib demonstrating the highest efficacy across both objective and patient-reported outcomes**, followed by dupilumab and tralokinumab. These differences may reflect variations in mechanism of action, particularly the broader cytokine inhibition profile of Janus kinase inhibitors compared to targeted biologics. The overall prevalence of adverse events was **48% (95% CI: 32–63)**, with most events classified as mild to moderate in severity. The most frequently reported adverse effects included **conjunctivitis (7%)**, predominantly associated with biologic therapy, and **herpes zoster (2%)**, more commonly observed with Janus kinase inhibitors. Despite these findings, serious adverse events were uncommon.

Conclusions
Targeted systemic therapies deliver clinically meaningful improvement in head and neck AD, a difficult-to-treat phenotype. Differences in efficacy and safety support individualized, mechanism-driven treatment selection.

However, significant heterogeneity and the absence of direct comparisons highlight the need for head-to-head trials and standardized outcomes to optimize management and guide precision-based care.

EADV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 1542

Real-world effectiveness, safety, and access of Secukinumab for moderate-to-severe psoriasis in a resource-limited setting: a retrospective case series review in a tertiary government hospital from the Philippines

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Introduction

Psoriasis is a chronic inflammatory disorder affecting 125 million people worldwide with a global prevalence of 0.33-0.6%. This is characterized by thick scaly erythematous plaques which is frequently linked to comorbidities such as psoriatic arthritis and metabolic syndrome. In the Philippines, it is one of the most common dermatologic diagnoses yet in such a resource-limited setting, access to biologics like secukinumab remains hindered by high costs, out-of-pocket payments and lengthy government aid processes. This retrospective case series describes the clinical outcomes, safety, adverse events, and access to secukinumab among psoriasis patients to inform local policy support for biologics.

Case Presentation

Eleven patients underwent secukinumab injections from 2019-2025 in our institution. The cohort included mostly females with a median age of 38 years. They had a median disease duration of 4-5 years and an estimated 4 disease flares annually. More than half presented with joint pains and almost one third had a family history of psoriasis. Prior therapies included topical medications, oral immunosuppresants and phototherapy. Baseline PASI scores had a median score of 23 (range: 12-32). All received secukinumab 300mg weekly loading dose and maintenance dose. By week 8, all achieved >30% PASI reduction with two patients achieving PASI 75. At week 24, more than half of the patients reached PASI-50 and at week 96, majority of patients achieved PASI-50 or better. During treatment course, average disease flares were reduced to 1-3 annually. No secukinumab-related adverse events. In terms of biologic access, almost all patients acquired secukinumab through patient assistance programs (PAP) and one patient had combined out-of-pocket expense/PAP. There is a median of 6 months (range: 4-10 months) from indication to biologic initiation. More than two-thirds had a 3-month interruptions due to supply issues, but all resumed without permanent discontinuation.

Conclusion

Secukinumab showed sustained effectiveness, excellent safety, and feasibility in our real-world cohort despite access delays and interruptions from PAP/supply reliance. We also highlighted baseline severity worse than regional peers due to systemic barriers. We suggested the need for policy reforms which include the following: expanded public reimbursements, pooled procurement, national formulary integration, and streamlined aid. These reforms may reduce disparities, ensure continuous biologic access, and align with universal health coverage for equitable psoriasis management in a resource-limited setting.

EADV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 1548

Macrophage Migration Inhibitory Factor and rs755622G>C Gene Polymorphism in Unstable Vitiligo: Association with Disease Activity and Treatment Response

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Introduction
Vitiligo is an autoimmune depigmenting disorder characterised by melanocyte destruction. Macrophage migration inhibitory factor (MIF) is a pro-inflammatory cytokine involved in immune regulation. This study aimed to evaluate tissue MIF levels and MIF gene polymorphism (rs755622G>C) in unstable vitiligo patients and to assess their relationship with disease activity and therapeutic response.

Materials and Methods
This case–control study included 50 patients with unstable vitiligo and 50 matched controls. Tissue MIF levels were measured using enzyme-linked immunosorbent assay. MIF gene polymorphism (rs755622G>C) was analysed using polymerase chain reaction–restriction fragment length polymorphism. Patients received narrow-band ultraviolet B combined with either oral corticosteroids or methotrexate for six months. Disease severity was assessed using the Vitiligo Area Severity Index.

Results
Lesional tissue MIF levels were significantly higher in patients compared with controls ($p=0.002$). The frequency of mutant genotypes (GC and CC) and the mutant allele (C) was significantly increased in patients ($p \leq 0.001$). Both treatment regimens resulted in significant clinical improvement and reduction in MIF levels. No significant difference was observed between treatment groups. The mutant allele (C) was associated with poorer therapeutic response.

Conclusions
Tissue MIF levels and MIF gene polymorphism (rs755622G>C) are associated with vitiligo pathogenesis and treatment response. Targeting MIF may represent a potential therapeutic strategy in unstable vitiligo.

EADV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 1553

Tissue Plexin-B2 Expression and SIRPγ Gene Polymorphism in Psoriasis: Association with Disease Severity and Treatment Response

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Introduction
Psoriasis is a chronic immune-mediated inflammatory skin disease characterized by keratinocyte hyperproliferation. Plexin-B2, a receptor involved in immune regulation and cell signaling, may play a role in psoriasis pathogenesis. This study aimed to evaluate tissue plexin-B2 expression and its related gene polymorphism (SIRPγ-rs71212732) in psoriatic patients and to assess their relationship with disease severity and therapeutic response.

Materials and Methods
This randomized controlled study included 50 patients with psoriasis vulgaris and 50 matched healthy controls. Tissue plexin-B2 levels were measured using enzyme-linked immunosorbent assay. SIRPγ gene polymorphism (rs71212732) was analyzed using real-time polymerase chain reaction. Patients received narrow-band ultraviolet B, acitretin, or combination therapy. Disease severity was assessed using the Psoriasis Area and Severity Index.

Results
Tissue plexin-B2 levels were significantly higher in patients compared with controls ($p<0.001$) and showed a positive correlation with disease severity. Significant reductions in plexin-B2 levels were observed after treatment ($p=0.005$). Combination therapy demonstrated superior clinical improvement compared with monotherapy. Mutant genotypes and allele frequencies were significantly higher in patients than in controls ($p<0.001$).

Conclusions
Tissue plexin-B2 expression and SIRPγ gene polymorphism are associated with psoriasis pathogenesis and disease severity. These findings suggest that plexin-B2 may serve as a potential biomarker and therapeutic target in psoriasis.

EADV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 1576

Serum TWEAK as a Biomarker of Treatment Response in Psoriasis and Psoriatic Arthritis: A Prospective Comparative Study

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Introduction
Psoriasis and psoriatic arthritis (PsA) are chronic immune-mediated inflammatory diseases with significant systemic impact. Tumor necrosis factor–related weak inducer of apoptosis (TWEAK) is a pro-inflammatory cytokine involved in immune regulation and tissue remodeling; however, its role in psoriatic disease remains incompletely understood.

Materials and Methods
This prospective case–control study included 100 subjects: 30 patients with psoriasis treated with adalimumab, 30 patients with PsA treated with methotrexate, and 40 healthy controls. Serum TWEAK levels were measured using ELISA at baseline and after 24 weeks of treatment. Disease activity was assessed using the Psoriasis Area and Severity Index (PASI) for psoriasis and the Disease Activity Index for Psoriatic Arthritis (DAPSA) for PsA.

Results
Baseline TWEAK levels were significantly higher in psoriasis and PsA patients compared to controls ($p < 0.001$). After 24 weeks of treatment, TWEAK levels significantly decreased in both the adalimumab and methotrexate groups ($p < 0.001$). This biochemical reduction paralleled significant clinical improvement, with PASI decreasing from 18.6 ± 5.1 to 5.2 ± 2.4 and DAPSA from 46.37 ± 8.49 to 18.59 ± 4.45. A significant negative correlation was observed between TWEAK levels and PASI after treatment, while no significant correlation was found with DAPSA.

Conclusions
Serum TWEAK levels are elevated in psoriasis and PsA and decrease following treatment, correlating with clinical improvement. TWEAK may serve as a useful biomarker for monitoring treatment response, particularly in cutaneous disease activity.

EADV CONGRESS
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Abstract N°: 1590

Application of Exposure-Response Modeling to Inform the Phase 3 Dose Selection of Povorcitinib in Nonsegmental Vitiligo

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Introduction

In a phase 2, randomized, dose-ranging study in adults with extensive nonsegmental vitiligo (NCT04818346), once-daily (QD) oral povorcitinib (Janus kinase 1 inhibitor) demonstrated substantial repigmentation without clear dose-response and was well tolerated at 15-, 45-, and 75-mg doses to Week 24 (placebo-controlled) and 45- and 75-mg doses to Week 52 (double-blind extension). Exposure-response modeling was conducted to predict response rates for doses not evaluated in phase 2 and inform povorcitinib dose selection for phase 3 studies.

Materials and Methods

The relationships between povorcitinib exposures (steady-state average concentrations) and probability of efficacy, safety, and biomarker outcomes based on observed phase 2 dose-ranging data were quantified using logistic regression models. A population pharmacokinetic model describing the time-course of concentrations following povorcitinib dosing was used to predict exposures for 30 mg QD, which served as input for efficacy and safety outcome predictions.

Results

Rates for ≥50% improvement in facial Vitiligo Area Scoring Index (F-VASI50) plateaued at exposures approximating 30 mg QD through Week 24, and Week 24 F-VASI50 correlated with achievement of ≥75% improvement in F-VASI (F-VASI75) at Week 52, the primary endpoint for phase 3 studies. All doses of povorcitinib were generally well tolerated during phase 2, with no dose-related trends for grade ≥3 adverse events. Increasing probability of acne and fatigue was predicted at exposures related to doses >30 mg QD. All doses had biological activity based on changes in biomarkers, though 15-mg povorcitinib QD was comparable to placebo for some biomarkers. The probability of achieving ≥50% decrease in C-X-C motif chemokine ligand 10 (validated vitiligo biomarker) at Week 24 increased with dose, with minimal additional gain observed at doses >30 mg QD.

Conclusions

Based on exposure-response modeling, 30-mg povorcitinib QD, a dose not tested in phase 2, was predicted to optimize the risk–benefit profile and was selected for use in phase 3 vitiligo studies.

EADV CONGRESS
VIENNA
30 SEP - 3 OCT 2026

Abstract N°: 1591

Bridging Traditional and Targeted Therapy: A Comparative Study of Methotrexate and Tofacitinib in Moderate-to-Severe Chronic Plaque Psoriasis

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Introduction
Psoriasis is a common, chronic and recurrent inflammatory skin disease characterized by the development of scaly, red, and well-demarcated plaques. Methotrexate is an effective treatment option for psoriasis, but limitations for the use include slow onset of therapeutic effect, teratogenicity and toxicities. JAK inhibitors, such as tofacitinib, have been studied for psoriasis patients, and the data are promising as a potential treatment option.

Materials and Methods
The non-randomized clinical trial was conducted at the psoriasis clinic, Department of Dermatology and Venereology, Dhaka Medical College Hospital, Dhaka, from 1st January 2022 to 30th June,2022. After fulfilling the inclusion and exclusion criteria, 32 psoriasis patients were treated with tofacitinib (Group A) and another 32 with methotrexate (Group B). The Psoriasis Area and Severity Score (PASI) was used to assess severity at the end of the 1st and 3rd months relative to baseline. Statistical analysis was done by SPSS 25.

Results
The mean age was (36.1 ± 8.3) years vs (36.3 ± 7.5) years in Group A and Group B, respectively, with no statistical significance (p=0.4). Mean baseline PASI score of Group A vs Group B was (20.9 ± 5.3) vs (18.7 ± 5.0), And Mean PASI score at the end of the 3rd month was (4.8 ± 3.2) vs (4.6 ± 3.8), respectively which were statistically not significant. In contrast, Mean PASI at the end of the 1st month was (8.7 ± 3.8) vs (12.0 ± 5.7) in Group A vs Group B, which is statistically significant (p = 0.01).

Conclusions
In this study, the PASI75 was achieved significantly in patients treated with tofacitinib in 1st month than in patients treated with methotrexate. After 3rd month, the PASI75 achievement was similar in both groups. Mild adverse effects were observed in both groups.

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Abstract N°: 1594

The dichotomy of clinical clearance and psychological burden: a 24-month follow-up of bimekizumab therapy in refractory hidradenitis suppurativa and pyoderma gangrenosum

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Introduction

Hidradenitis suppurativa (HS) is a severely debilitating chronic inflammatory skin disease. Extensive, disfiguring lesions and chronic pain frequently lead to profound psychological trauma, culminating in anxiety, depression, and profoundly reduced quality of life. Managing HS complicated by pyoderma gangrenosum (PG) is particularly challenging. Patients failing tumor necrosis factor (TNF)-alpha inhibitors represent a highly refractory population requiring potent, novel therapeutic approaches alongside comprehensive psychosocial support.

Case Presentation

We present a case of a 34-year-old female with a 20-year history of HS and histopathologically confirmed PG. Her medical history is significant for extensive comorbidities, including obesity, metabolic syndrome components, and severe anxiety disorders requiring long-term psychiatric polypharmacotherapy (including trazodone, duloxetine, and bupropion). Previous dermatological interventions included multiple surgical resections, three courses of isotretinoin, and systemic antibiotic therapies. Treatment with adalimumab was discontinued after 12 months due to secondary loss of efficacy and a catastrophic clinical flare. At the time of the biological switch, the patient presented with multiple active draining fistulae in the axillae, oozing PG ulcerations in the inframammary region, and actively inflamed nodules. Based on the clinical presentation, the baseline International Hidradenitis Suppurativa Severity Score System (IHS4) was estimated at 18. As systemic retinoids were contraindicated, targeted dual IL-17A/F inhibition with bimekizumab (320 mg subcutaneously every 2 weeks) was initiated.

Over a 24-month follow-up period, the therapy led to a profound and sustained objective clinical improvement. Physical examination revealed the complete resolution of all draining fistulae and successful healing of the PG, which transformed into a fully re-epithelialized scar. The HS inflammatory burden was 6 inflammatory nodules, corresponding to an IHS4 value of 6 (moderate activity). The patient also reported the complete absence of physical pain.

However, despite achieving spectacular healing of severe inflammatory manifestations and high physical tolerance, the psychological burden of the disease remained strikingly high. The patient's Dermatology Life Quality Index (DLQI) persisted at 15, indicating a severe ongoing impact on her daily life. The presence of extensive, irreversible retracted scarring and long-standing psychiatric comorbidities (anxiety disorders) continued to dominate her subjective perception of health.

Conclusion

Bimekizumab exhibits the potent anti-inflammatory capacity required to eliminate draining fistulae and heal persistent PG ulcerations where TNF-inhibitors fail, making it a highly effective and well-tolerated therapy for

severe, refractory HS. Nevertheless, this case emphatically highlights the dichotomy between objective dermatological clearance and subjective psychological well-being. Long-standing HS inflicts irreversible physical and mental scars; therefore, advanced biological treatments must be coupled with continuous psychiatric support to improve the patient's overall quality of life meaningfully.

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Abstract N°: 1599

Blood monitoring in patients treated with dupilumab for atopic dermatitis: is it necessary?

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Introduction
Dupilumab is a biologic agent indicated in patients with moderate to severe atopic dermatitis. Blood monitoring practices vary between centres and are not explicitly recommended by the manufacturers. The aim of this study was to evaluate the frequency and clinical significance of blood abnormalities in patients treated with dupilumab to assess whether ongoing blood monitoring is necessary.

Materials and Methods
A retrospective review was conducted of all patients commenced on dupilumab for atopic dermatitis in a single centre from 2021–2024. Collected data included demographic data, baseline laboratory values and follow-up laboratory results.

Results
Sixty-one patients were included (51 adults, 10 paediatric). The median age was 40 years (11–87 years). Adults were prescribed 300mg fortnightly and paediatric patients were prescribed 200mg fortnightly. Fifty-one patients (83.6%) had a blood test at their follow-up visit. The mean time to first follow-up blood test was 8.5 months (2–26 months). Forty patients (78.4%) had no new blood abnormalities at their follow up appointment. Eleven patients (21.6%) had developed new blood abnormalities. Five patients had transient abnormalities, including three cases of eosinophilia. Four patients had new persistent abnormalities including raised alanine aminotransferase and gamma-glutamyl transferase; thrombocytopaenia; thrombocytosis; raised alanine aminotransferase, aspartate aminotransferase, leucocytosis, and neutrophilia. No blood abnormalities were attributed to dupilumab and no patients discontinued treatment due to laboratory abnormalities.

Conclusions
No significant blood abnormalities attributable to dupilumab were identified. These findings support the cessation of routine blood monitoring in patients treated with dupilumab thus improving clinical efficiency and reducing healthcare costs.

EADV CONGRESS
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Abstract N°: 1655

Targeting type 2 inflammation: Dupilumab in refractory bullous pemphigoid and chronic spontaneous urticaria.

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Introduction

Dupilumab is a fully human monoclonal IgG4 antibody that inhibits interleukin (IL)-4 and IL-13 signaling, key drivers of type 2 inflammation. This pathway plays a relevant role in the pathogenesis of bullous pemphigoid (BP) and chronic spontaneous urticaria (CSU), and clinical trials have demonstrated its efficacy in both conditions. Dupilumab has been recently approved for the treatment of BP and CSU; however, access and positioning within the therapeutic algorithm may vary in real-world clinical practice. We present a real-word case series of patients with BP or CSU refractory to conventional systemic treatments who initiated off-label dupilumab following the approved dosing regimen. Treatment response was assessed using the Bullous Pemphigoid Disease Area Index (BPDAI) for BP, the Urticaria Activity Score over 7 days (UAS7) for CSU, and the Numerical Rating Scale (NRS) for pruritus in both conditions.

Case Presentation

Case 1: A 68-year-old male with BP and comorbid COPD, hypertension, and positive IGRA, previously treated with prednisone, dapsone, azathioprine, methotrexate, and omalizumab, achieved complete remission within 3 months of dupilumab, without adverse events.

Case 2: A 56-year-old female with BP and comorbid diabetes and hypertension, previously treated with doxycycline, prednisone, and methotrexate, achieved a favorable clinical response after dupilumab initiation.

Case 3: A 27-year-old female with CSU and a history of two HSV-1 meningoencephalitis episodes, refractory to high-dose antihistamines, intolerant to omalizumab, and previously treated with cyclosporine, achieved complete symptom resolution within 1 month, without adverse events.

Case 4: A 51-year-old female with CSU and severe asthma, refractory to antihistamines, prednisone, omalizumab, methotrexate, and cyclosporine, achieved complete disease control within 2 months. Mild eyelid eczema was observed and managed with topical corticosteroids. A relapse at 7 months required treatment intensification.

Conclusion

In our real-world experience, dupilumab appears to be an effective therapeutic option for BP and CSU refractory to multiple systemic treatments, providing rapid clinical improvement with a favorable safety profile, including in patients with relevant comorbidities.

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Abstract N°: 1687

Demographic profiles and adverse outcomes in dermatology versus non-dermatology dupilumab cohorts

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Introduction
Dupilumab is prescribed across different specialties, with potential variations in patient characteristics, indications, and safety outcomes. This study aimed to compare demographic, clinical, and adverse event profiles between dermatology and non-dermatology dupilumab cohorts.

Materials and Methods
A retrospective cohort study was conducted on 457 patients commenced on dupilumab at a tertiary metropolitan centre. Among these, 277 were managed within dermatology and 180 within other services (respiratory, allergy and immunology). Demographic variables, treatment indications, baseline clinical parameters, and adverse events were extracted from medical records. Adverse events were analysed as dichotomous outcomes. Group comparisons of categorical outcomes were performed using χ^2 or Fisher's exact tests, with non-parametric tests for continuous variables.

Results
Dermatology patients differed from non-dermatology patients in indication distribution, median age (36 vs 54 years, $p < 0.01$), gender distribution (45.5% female vs 63.3% female, $p < 0.01$), country of origin ($p = 0.01$), and Aboriginal and Torres Strait Islander (ATSI) status ($p = 0.04$) but were similar in Modified Monash Model (MMM) classification ($p = 0.27$). Adverse events were more frequently reported in dermatology patients versus non-dermatology patients (70 (25%) and 9 (5.0%) respectively) ($p < 0.01$)). Ocular surface disease (20% vs 1.7%, $p < 0.01$) and red face syndrome (2.5% vs 0.0%, $p = 0.045$) were notably more common in dermatology patients. Treatment discontinuation attributable to adverse events was infrequent in both cohorts (4.0% vs 3.9%).

Conclusions
Dermatology and non-dermatology dupilumab cohorts demonstrate distinct demographic and clinical profiles, accompanied by differing adverse event patterns. These findings highlight the necessity of speciality-specific contextualisation when interpreting safety data and tailoring patient counselling and monitoring strategies between indications.

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Abstract N°: 1776

Severe generalized pustular psoriasis in pregnancy after IL-17 inhibitor discontinuation: clinicopathological correlation and therapeutic decision-making in a high-risk scenario

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Introduction

Generalized pustular psoriasis (GPP) in pregnancy, historically termed impetigo herpetiformis, is a rare and potentially life-threatening neutrophilic dermatosis. Its significance extends beyond cutaneous involvement, as uncontrolled systemic inflammation may compromise maternal and fetal outcomes. GPP is driven by innate immune dysregulation, particularly IL-36 pathway activation, which amplifies keratinocyte cytokine release, neutrophil recruitment and epidermal pustulation. In patients with underlying plaque psoriasis, IL-23/IL-17 signaling further sustains inflammation, creating overlap between chronic adaptive immunity and acute autoinflammatory disease.

Case Presentation

We present a 35-year-old pregnant woman at 25 weeks of gestation with long-standing psoriasis vulgaris and psoriatic arthritis, previously treated with multiple systemic and biologic agents, including methotrexate, tumor necrosis factor inhibitors, IL-23 inhibition and most recently IL-17 inhibition, discontinued after pregnancy confirmation. She developed a 10-day history of rapidly progressive erythematous plaques with pustulation, severe burning pain and pruritus. Initial treatment with beta-lactam antibiotics was followed by clinical worsening, raising suspicion of drug eruption.

Examination revealed extensive, well-demarcated erythematous-violaceous plaques with thick silvery scale on the trunk and extremities, with numerous non-follicular pustules predominantly on distal lower limbs and posterior axillary regions, and confluent erythematous macules and papules on the anterior chest. A swan-neck deformity of the fifth digit supported psoriatic arthropathy. Disease burden was severe (PASI 51, DLQI 40, NAPSI 12).

Laboratory tests showed leukocytosis with neutrophilia without evidence of infection. Obstetric assessment confirmed fetal viability. Histopathology demonstrated psoriasiform hyperplasia with parakeratosis, hypogranulosis, regular acanthosis, papillomatosis, neutrophilic exocytosis and intraepidermal microabscesses, with perivascular lymphohistiocytic infiltrate containing eosinophils. No microorganisms or malignancy were identified, supporting pustular psoriasis.

Given disease severity and maternal–fetal risk, cyclosporine 3 mg/kg/day was initiated due to its rapid onset, established efficacy and greater pregnancy experience compared with newer agents. The patient improved rapidly, allowing continuation of pregnancy without complications. Notably, the flare followed IL-17 discontinuation, suggesting that withdrawal rather than exposure may have contributed to disease destabilization. In selected cases, continuation of IL-17 blockade might not require suspension and could prevent severe exacerbations.

Conclusion

This case highlights a high-risk scenario of GPP in pregnancy with multi-biologic exposure and diagnostic overlap

with toxicoderma. It underscores GPP as a systemic inflammatory emergency requiring early recognition and treatment. Importantly, it challenges the routine discontinuation of targeted therapies, suggesting that in selected patients, maintaining IL-17 inhibition may be safer than withdrawal. These findings emphasize individualized risk–benefit assessment and the need for stronger evidence to guide management of severe psoriasis during pregnancy.

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Abstract N°: 1780

Preclinical Evaluation of Rho-Associated Protein Kinase Inhibition in Keloid Disease Using a 3D Spheroid Model

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Introduction

Keloid disease (KD) is a fibroproliferative disorder characterized by excessive extracellular matrix deposition and limited therapeutic options. The absence of reliable animal models and limitations of traditional 2D culture systems restrict effective preclinical drug evaluation. This study aimed to develop a physiologically relevant 3D keloid fibroblast spheroid model and assess the antifibrotic activity of the Rho-associated protein kinase (ROCK) inhibitor AMA0825 in comparison with dexamethasone (DEX).

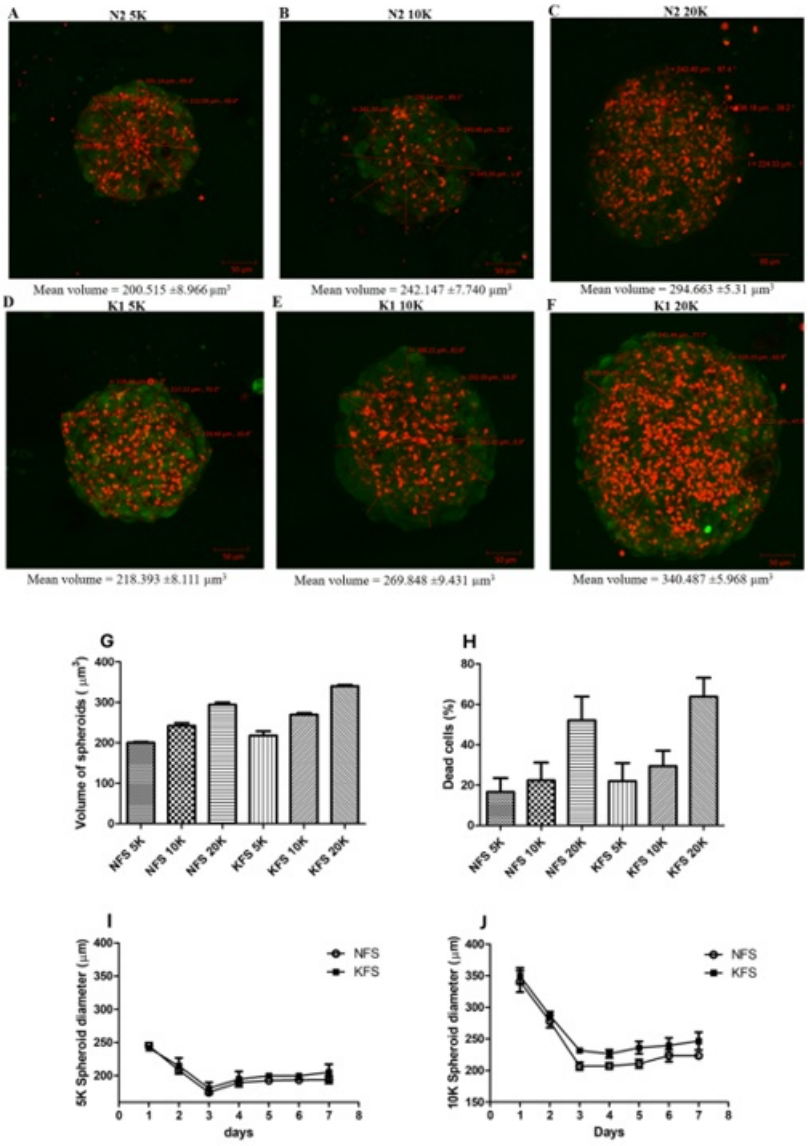

Figure 1. Generation and characterization of keloid and normal fibroblast–derived spheroids (KFS and NFS). Primary fibroblast–derived spheroids were generated from normal (N) and keloid (K) fibroblasts at varying seeding densities and analyzed by confocal microscopy, volumetric measurement, viability staining, and diameter quantification. Representative confocal images of N1-derived spheroids seeded at 5,000, 10,000, and 20,000 cells/well are shown in panels A–C, respectively. Representative confocal images of K1-derived spheroids seeded at 5,000, 10,000, and 20,000 cells/well are shown in panels D–F, respectively. (G) Quantification of spheroid volume (μm^3) for normal fibroblast spheroids (NFS; N1, N2, N3) and keloid fibroblast spheroids (KFS; K1, K2, K3). Scale bars = 50 µm. (H) Representative live/dead cell staining using Calcein-AM (live, green) and propidium iodide (PI; dead, red) to assess viability of NFS and KFS spheroids formed at 5,000, 10,000, and 20,000 cells/well. (I–J) Quantification of spheroid diameter over 7 days at optimized seeding densities of 5,000 cells/well (I) and 10,000 cells/well (J) for both NFS and KFS. Statistical analyses were performed using GraphPad Prism v5.0 with a sample size of n = 3 biological samples, each with three technical replicates. Data are presented as mean ± standard deviation (SD) from three independent experiments.

Materials and Methods

Normal fibroblasts (NFs) and keloid fibroblasts (KFs) were cultured in ultra-low attachment plates to generate spheroids at densities of 5k, 10k, and 20k cells. Spheroid morphology, size, and viability were monitored over 7 days using microscopy and Calcein/propidium iodide staining. KF spheroids were treated with AMA0825 (0.5–50,000 nM) or DEX (80 µM), and proliferation was evaluated longitudinally. Extracellular matrix (ECM) remodeling and fibrosis-associated markers (Collagen I and α-SMA) were assessed using histological staining and immunocytochemistry.

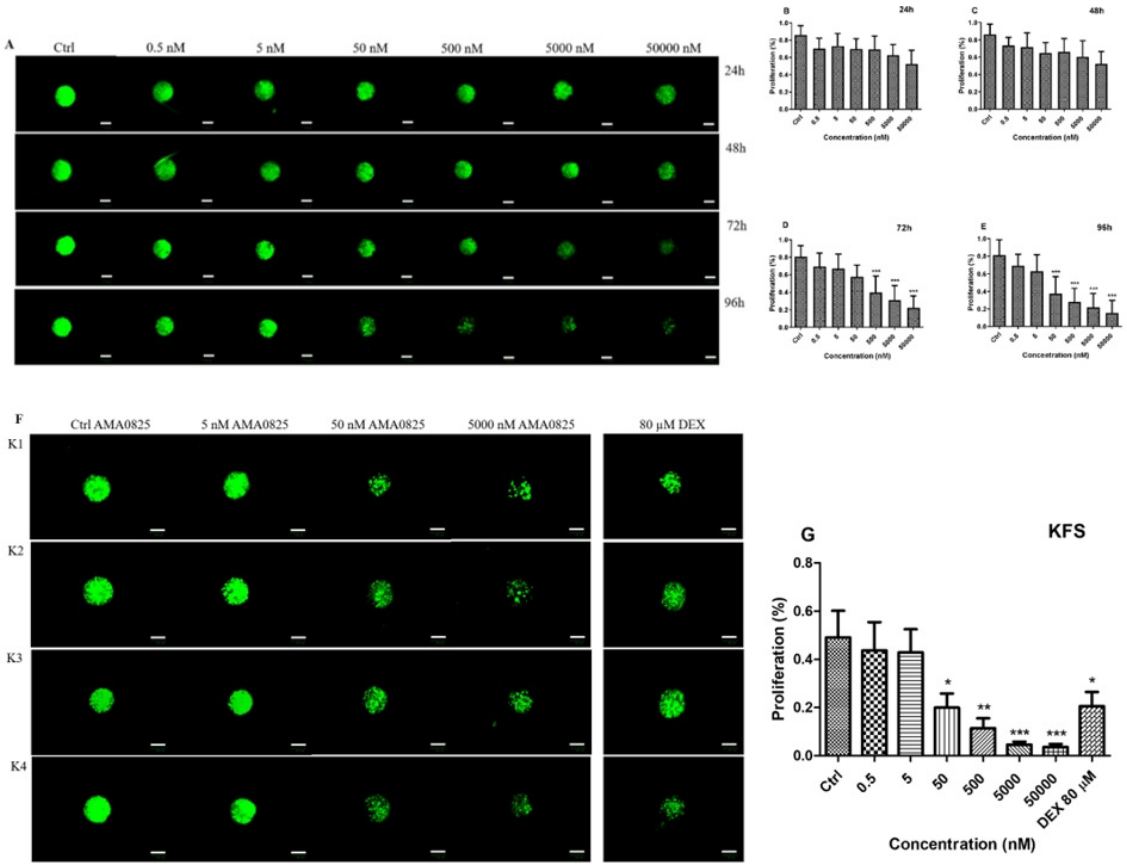

Figure 2. Optimizing AMA0825 treatment timing and evaluating its therapeutic effects with Dex in spheroid model using CyQuant and cell imaging. (A) Representative figure demonstrating the impact of AMA0825 on K1 and K4 at concentrations ranging from 0.5-50000 nM across various time points from 24 to 96h. Graph B-E representing data pertaining to K1 and K4 treatment with different concentration of AMA0825 at 24, 48, 72, and 96h (B-E). Statistics were performed on n = 8 replicates per treatment group using GraphPad Prism v5.0. (F) Representative figure illustrates the effect of AMA0825 and DEX on K1, K2, K3 and K4 at concentrations ranging from 0.5 to 50000 nM after 96h. (G) Representative data related to K1, K2, K3, and K4 treated with different concentrations of AMA0825 and 80 µM dexamethasone at 96h. Statistics were performed on sample size n=4 with 4 replicates using GraphPad Prism v5.0. In both assays, One-way ANOVA followed by Dunnett's post-hoc test used to assess statistical significance. Significance levels are indicated as follows: *P < 0.05, **P < 0.01, ***P < 0.001, Scale bars = 100 µm. Error bars represent the mean ± SEM of at least three independent experiments (n ≥ 3).

Results

Both normal fibroblast spheroids (NFS) and keloid fibroblast spheroids (KFS) formed within 24 hours, achieving circular morphology by day 2 and maximal compactness by day 3. Spheroid size increased with cell density, with KFS consistently larger than NFS. Viability remained high across all groups, although higher densities demonstrated increased necrotic regions. Based on stability and reduced necrosis, 5k spheroids were selected for downstream analysis.

AMA0825 treatment resulted in time- and dose-dependent inhibition of KFS proliferation, with maximal effects observed at 96 hours post-treatment. Significant proliferation inhibition was detected from 50 nM, reaching >90% inhibition at higher concentrations. Notably, AMA0825 demonstrated comparable or greater antiproliferative efficacy than DEX at substantially lower concentrations.

While DEX significantly reduced collagen deposition, AMA0825 demonstrated antifibrotic activity at higher concentrations, significantly reducing Collagen I and α-SMA expression across multiple donor-derived spheroids.

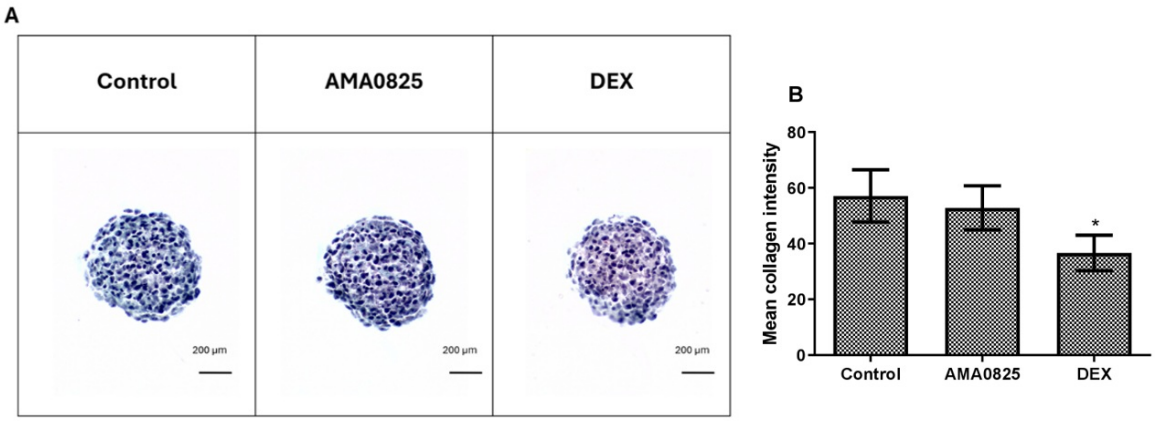

Figure 3. Effect of AMA0825/DEX on Collagen I in spheroid model assessed by Masson's trichrome staining and quantitative analysis. (A) Representative figure of Masson's Trichrome staining of spheroids from K1 donor. Collagen appears light green, cytoplasm in red, and nuclei in dark blue, illustrating collagen distribution throughout the spheroids. As collagen staining is not readily visible, image analysis was conducted using color deconvolution in FIJI-ImageJ to isolate and quantify the light green signal for collagen as described in the Methods section. Scale bar=200 µm. (B) Quantification of collagen content in K1 and K2 donors was performed on a sample size of n = 2, with 6 replicates for each sample using GraphPad Prism v5.0. One-way ANOVA followed by Dunnett's post-hoc test used to assess statistical significance. Significance levels are indicated as follows: *P < 0.05.

Conclusions

This study establishes a reproducible and physiologically relevant 3D keloid fibroblast spheroid model suitable for antifibrotic drug screening. AMA0825 demonstrated potent antiproliferative and antifibrotic effects, supporting its potential as a novel therapeutic candidate for keloid disease. These findings highlight the translational value of 3D spheroid platforms for preclinical evaluation of antifibrotic therapies in dermatological research.

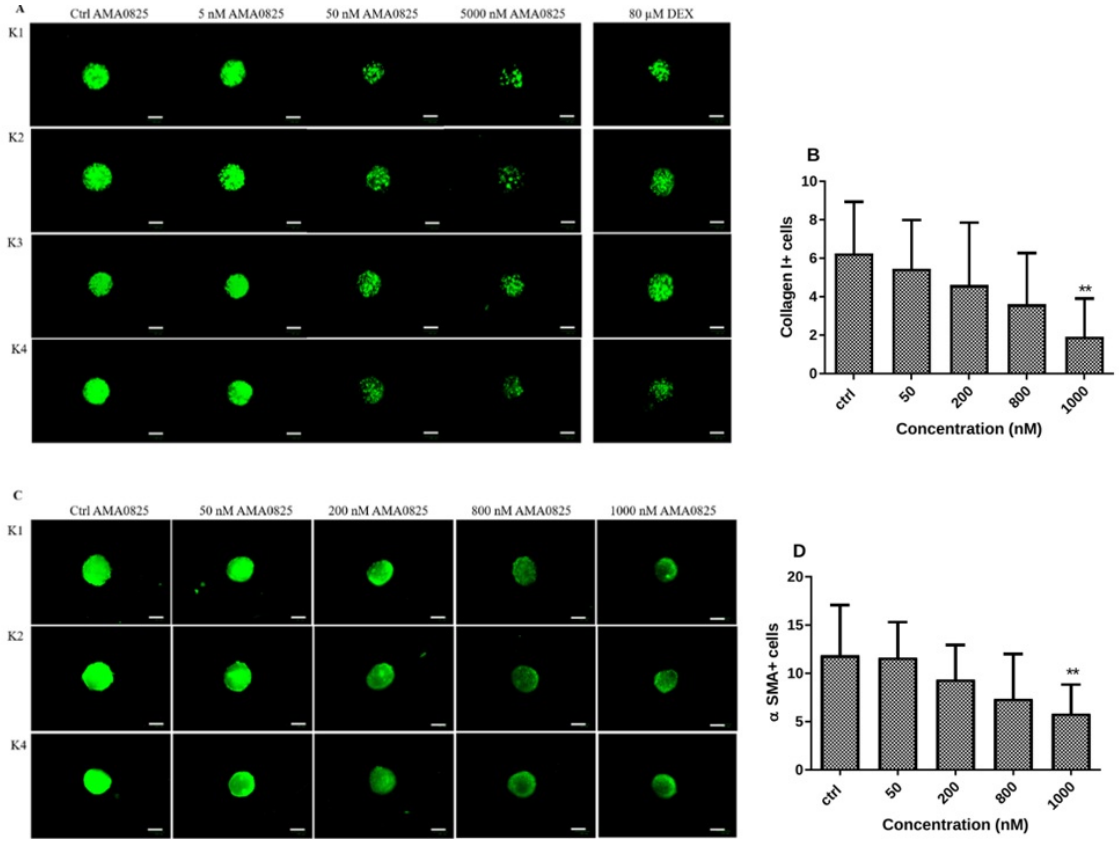

Figure 4. Effect of AMA0825 on Collagen I and αSMA expression in spheroid model assessed by immunofluorescence staining with anti-collagen I and α-SMA antibodies and quantification. (A) Representative image demonstrating the effect of AMA0825 on collagen I (green) on K1, K2, and K4 spheroids using immunofluorescence staining with anti-collagen I antibody (1:250). (B) Corresponding graph representing collagen I^+ cells. (C) Representative image showing the effect of AMA0825 on αSMA (green) using immunofluorescence staining with anti-α-SMA antibody (1:50). (D) Corresponding graph quantifying $αSMA^+$ cells. Statistical analyses were conducted on a sample size of n = 3 with 3 replicates for each sample. One-way ANOVA followed by Dunnett's post-hoc test was used to assess statistical significance. Significance levels are indicated as follows: *P < 0.05. Error bars represent the mean ± SD of two independent experiments for panel B, and three independent experiments for panel B and D.

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Abstract N°: 1819

Sonelokimab, a novel IL-17A- and IL-17F-inhibiting Nanobody, in moderate-to-severe palmoplantar pustulosis: Efficacy, safety, and translational results from the open-label Phase 2 LEDA trial

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Introduction

Palmoplantar pustulosis (PPP) is a chronic inflammatory and debilitating skin disease characterized by recurrent, painful, sterile pustules on the palms and soles that can substantially impair function and quality of life. The pathophysiology of PPP is complex, and no targeted therapy has been approved in either the EU or US. However, a critical role for Th17 signaling has been shown in PPP, with both IL-17A and IL-17F contributing to neutrophil influx and keratinocyte hyperplasia. Sonelokimab is a ~40 kDa Nanobody that binds to both IL-17A and IL-17F with a similarly high affinity. Here, we present efficacy, safety, and translational results from the LEDA trial of sonelokimab in adults with PPP.

Materials and Methods

LEDA (EUCT 2024-513305-32-00) was a Phase 2, multicenter, open-label trial. Eligible patients were ≥18 years old with moderate-to-severe PPP (PPPASI ≥12, with or without concomitant plaque-type psoriasis) inadequately controlled by previous therapy who were considered suitable for systemic therapy. All patients received sonelokimab 120 mg subcutaneously at Weeks 0, 2, 4, 6, 8, and 12. Clinical assessments were performed at the same time points (and Week 16); pain was assessed daily via an electronic diary. The primary endpoint was percent change from baseline in PPPASI score at Week 16. Key secondary endpoints were PPPASI50 and PPPASI75

response at Week 16 and safety and tolerability; additional secondary endpoints included PPP-IGA 0/1 response (with a ≥2-point improvement from baseline), pustule count, skin pain (11-point NRS), and DLQI. All statistical assessments were exploratory; missing data were imputed using a modified non-responder analysis (continuous values imputed as baseline score). Serum samples were taken at baseline, Week 4, and Week 16 for proteomic analyses using the Olink Explore HT platform.

Results

A total of 32 patients were enrolled and treated; 28 completed treatment through Week 16. Significant improvements in PPPASI scores from baseline were observed as early as Week 4, with a mean reduction from baseline in PPPASI of 64% (P<0.001) by Week 16 (**Figure 1A**). PPPASI50 and PPPASI75 responses were achieved by 70% and 39% of patients at Week 16, respectively. Early achievement of PPP-IGA 0/1 responses was observed (23% response rate at Week 4), with response rates increasing to 40% at Week 16 (**Figure 1B**). Consistent with these responses, significant reductions in fresh (–59%; P<0.001) and total (–45%; P<0.001) pustule counts were observed at Week 2 and were maintained thereafter. Similarly, significant improvements in worst skin pain were observed from Week 1 onwards (P=0.002), with 67% of patients achieving a ≥4-point improvement at Week 16. Quality of life, as measured by DLQI total score, significantly improved from Week 2 (–5; P<0.001) onwards, with 44% of patients achieving DLQI 0/1 at Week 16. Serum proteomics demonstrated a correlation between baseline PPP disease activity (PPPASI) and the IL-17 signaling pathway. Additionally, IL-36γ, IL-19, PI3, and CXCL6 correlated with sonelokimab treatment response (PPPASI). Levels of IL-19, hBD2, and PI3 were normalized after two doses of sonelokimab (Week 4) and remained stable until end of treatment. Sonelokimab was well tolerated, with no SAEs or unexpected safety findings. There were no cases of IBD, MACE, depression, suicidal ideation and behavior, or recurrent candidiasis.

Figure 1A. Mean percent change from baseline in PPPASI through Week 16

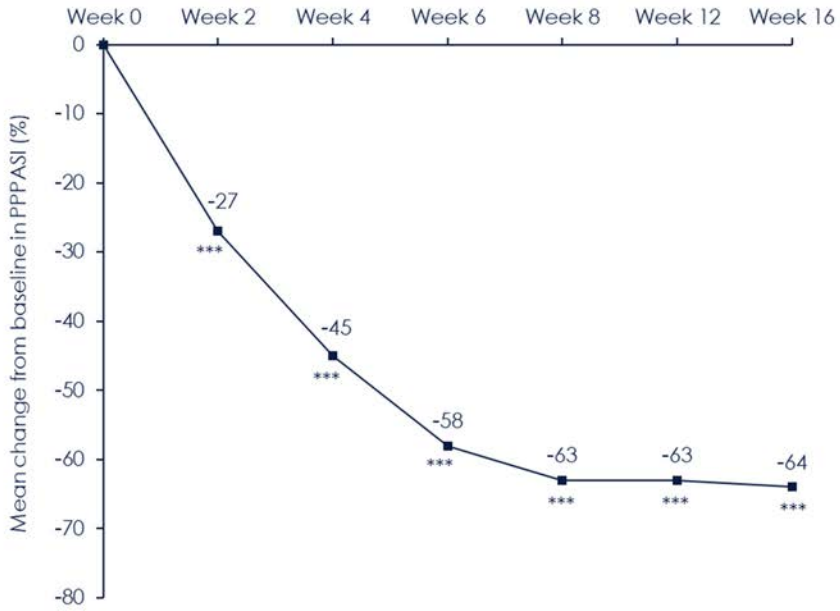
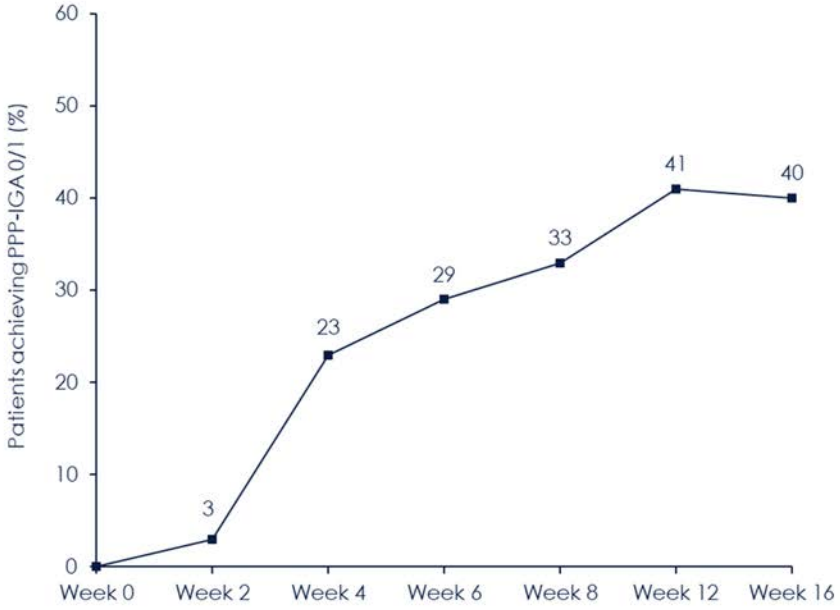


Figure 1B. Proportion of patients achieving PPP-IGA 0/1 (with a ≥2-point improvement) response through Week 16



***P<0.001. PPPASI, Palmoplantar Pustulosis Psoriasis Area and Severity Index; PPP-IGA, Palmoplantar Pustulosis Investigator Global Assessment.

Conclusions

Sonelokimab treatment resulted in rapid and clinically meaningful improvements in disease activity and patient-reported outcomes in adults with moderate-to-severe PPP. Consistent with these clinical outcomes, IL-17A and IL-17F blockade with sonelokimab was associated with objective reductions in disease-relevant and IL-17 responsive serum biomarkers. Safety was consistent with prior studies, with no new signals. These findings support further investigation of IL-17A and IL-17F inhibition with the Nanobody sonelokimab as a potential therapeutic approach for PPP.

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Abstract N°: 1826

Population Pharmacokinetic–Pharmacodynamic Modelling of Secukinumab in Real-World Psoriasis Patients to Support Model-Informed Precision Dosing

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Introduction

Secukinumab, an interleukin-17A inhibitor, is highly effective for moderate-to-severe plaque psoriasis but requires long-term administration at considerable cost. Model-informed precision dosing (MIPD) provides an alternative to fixed, one-size-fits-all dosing strategies by predicting the dosing interval most likely to achieve clear or almost-clear skin in an individual patient.

MIPD is based on population pharmacokinetic–pharmacodynamic (pop-PK/PD) models, which simultaneously describe how secukinumab concentrations change over time in a typical patient (pharmacokinetics, PK) and how these concentrations translate into improvements in Psoriasis Area and Severity Index (PASI) scores (pharmacodynamics, PD). By integrating these models with patient-specific clinical characteristics (e.g. body weight, age, concomitant medication etc.) MIPD enables individualised dosing recommendations aimed at optimising treatment outcomes while minimising unnecessary drug exposure.

However, a well-characterised real-world pop-PK/PD model for secukinumab is currently lacking. This study aimed to develop such a model on real-world datasets to support evidence-based dose optimisation.

Materials and Methods

Data were pooled from two prospective phase IV clinical studies (clinicaltrials.gov: NCT04340076 and NCT04080661), comprising 244 serum secukinumab concentrations and 262 Psoriasis Area and Severity Index (PASI) scores from 61 adult psoriasis patients. Nonlinear mixed-effects modelling was performed in R using nlmixr2 with FOCEI estimation. One- and two-compartment pharmacokinetic models were evaluated. Pharmacodynamics were described using an indirect response model with an inhibitory E_{max} (maximal drug effect) function. Allometric scaling based on body weight was applied a priori. Model performance was evaluated using goodness-of-fit diagnostics and prediction-corrected visual predictive checks (pcVPCs). Simulations of 100,000 virtual patients were conducted to derive target trough concentrations.

Results

A one-compartment model with first-order absorption and elimination best described the pharmacokinetics. The pharmacodynamic model incorporated lesion turnover, with E_{max} fixed to 1 and K_{in} (rate of lesion development) fixed to a literature value (0.62 day^{-1}). The estimated IC_{50} (concentration at which 50% of the maximal drug effect is achieved) was 2.53 µg/mL. Diagnostics and prediction-corrected visual predictive checks demonstrated

adequate model performance. Simulations identified week-24 trough concentrations of 14.5 µg/mL and 35.5 µg/mL as associated with 90% probabilities of achieving PASI ≤2 and PASI ≤1, respectively, remaining stable through week 52.

Conclusions

This real-world population PK/PD model can be integrated into an MIPD platform, such as a user-friendly R Shiny web application, in which dermatologists enter patient characteristics and, when available, measured serum secukinumab concentrations to obtain individualised dosing recommendations alongside predicted PASI trajectories over time. In addition, the model provides clinically actionable target trough concentrations that may support routine therapeutic drug monitoring in clinical practice.

Future work will focus on developing comparable pop-PK/PD models for guselkumab, ixekizumab, and risankizumab, enabling the implementation of individualised, model-based decision support across multiple biologics used in psoriasis treatment. Such tools could provide dermatologists with objective support for selecting biologic therapies, as well as for decisions regarding dose intensification, tapering, or switching, ultimately contributing to more effective and cost-conscious patient care.

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30 SEP - 3 OCT 2026

Abstract N°: 1831

Complete Remission of Dupilumab-Refractory Erythrodermic Chronic Actinic Dermatitis with Upadacitinib

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Introduction

Chronic actinic dermatitis (CAD) is a rare and often debilitating photodermatosis characterized by persistent eczematous and infiltrated lesions affecting sun-exposed areas, predominantly in elderly men. In severe cases, CAD may progress to erythroderma and can be challenging to manage. Conventional treatments, including systemic corticosteroids and immunosuppressive agents, frequently fail to achieve sustained disease control [1,2]. More recently, targeted therapies such as biologics and Janus kinase (JAK) inhibitors have emerged as potential treatment options in refractory cases [3,4]. Herein, we present a case of severe erythrodermic CAD unresponsive to conventional treatments and dupilumab, with a favorable response to upadacitinib.

Case Presentation

A 72-year-old male presented with a two-year history of intensely pruritic, erythematous, and infiltrated lesions predominantly involving sun-exposed areas, eventually progressing to erythroderma. Histopathological examination revealed epidermal hyperplasia, spongiosis, lymphocyte exocytosis, and an eosinophil-rich dermal infiltrate. Due to clinical suspicion of mycosis fungoides, repeated biopsies, immunohistochemical analysis, and T-cell receptor clonality testing were performed, which supported the diagnosis of CAD and excluded lymphoma. Phototesting demonstrated a delayed hypersensitivity reaction to UVA radiation. In addition, UVB provocation testing revealed a minimal erythema dose (MED) of 20 mJ/cm^2 at 24 hours in a patient with Fitzpatrick skin type III, suggesting increased cutaneous sensitivity to UVB.

The patient had previously been treated with cyclosporine, hydroxychloroquine, and systemic corticosteroids (prednisolone), without adequate disease control. Initial treatment with potent topical corticosteroids and antihistamines, followed by dupilumab, also failed to provide clinical improvement. Systemic corticosteroids remained insufficient in achieving sustained disease control. Given the refractory course, treatment was switched to upadacitinib 15 mg/day. Following initiation of upadacitinib, the patient showed rapid and marked clinical improvement. At 9 months, the Eczema Area and Severity Index (EASI) decreased from 11.3 to 0.3, and the pruritus Visual Analog Scale (VAS) improved from 9 to 1. Complete clinical remission was achieved without any serious adverse events.

Conclusion

Reports of upadacitinib use in CAD remain extremely limited in the current literature [8]. Recent case series have also demonstrated rapid and sustained complete remission with upadacitinib in patients with treatment-refractory CAD [8]. This case demonstrates that upadacitinib can be an effective treatment option in severe and refractory CAD, particularly in patients who fail to respond to conventional therapies and dupilumab. Given the diagnostic and therapeutic challenges associated with erythrodermic presentations, achieving rapid disease control is crucial, and JAK inhibitors may offer a meaningful advantage in this setting. The marked improvement observed in both

clinical severity and pruritus highlights the potential of JAK inhibition to target multiple inflammatory pathways involved in CAD. Although current evidence remains limited, our findings support the consideration of JAK inhibitors as a therapeutic alternative in difficult-to-treat cases. Further studies are warranted to better define patient selection and long-term outcomes.

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Abstract N°: 1846

Efficacy and Safety of Litifilimab in Cutaneous Lupus Erythematosus (CLE): 52-week results of the Phase 2 Study, AMETHYST Part A

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Introduction

Cutaneous lupus erythematosus (CLE) is a serious, autoimmune disease with chronic symptoms, which can lead to disfigurement, irreversible scarring, alopecia, and dyspigmentation. Litifilimab, a monoclonal antibody that binds to blood dendritic cell antigen 2 (BDCA2), a receptor expressed on plasmacytoid dendritic cells (pDCs), inhibits pDC-derived proinflammatory cytokines and chemokines including type I interferon. AMETHYST (NCT05531565), a randomized, double-blind, placebo-controlled, 2-part, operationally seamless Phase 2/3 study, evaluated the efficacy and safety of subcutaneous litifilimab every 4 weeks in participants with active chronic and/or subacute CLE, with or without systemic manifestations.

Materials and Methods

Participants were randomized to receive litifilimab or placebo every four weeks during Weeks 0-24 (double-blind placebo controlled [DBPC] period). At Week 24, all participants received litifilimab through Week 52 (extended treatment period) (**Figure 1**). The primary endpoint in Part A (Phase 2) was the Cutaneous Lupus Activity Investigators' Global Assessment Revised (CLA-IGA-R) erythema score of 0-1 (clear/almost clear) at Week 16. Secondary endpoints included CLA-IGA-R erythema 0-1, Cutaneous Lupus Erythematosus Disease Area and Severity Index 50 and 70 (CLASI-50/CLASI-70), and CLASI-activity (CLASI-A) 0-3 at Week 24. Here, we report data through week 52 with a focus on the extended treatment period (Weeks 24-52).

Results

A total of 93 participants were randomized and dosed in Part A. Baseline CLASI-A was well-balanced (placebo, 19.4, n=34; litifilimab, 20.2, n=59). For participants who initiated litifilimab in the DBPC period, treatment response rates continuously improved from Week 24 through Week 52 across CLA-IGA-R erythema 0-1 (19.0%, 27.2%), CLASI-70 (21.7%, 28.8%), CLASI-50 (40.8%, 46.0%), and CLASI-A 0-3 (16.3%, 16.7%). For participants who

switched from placebo to litifilimab at Week 24, the onset of efficacy was demonstrated within approximately 4 weeks after switching and the response rates at Week 52 for CLA-IGA-R erythema 0-1, CLASI-70, CLASI-50, and CLASI-A 0-3 reached 33.7%, 35.4%, 48.5%, and 22.2%, respectively. Litifilimab was generally well tolerated. In the extended treatment period, AEs were reported in 76.8% of participants who initiated litifilimab in the DBPC period and in 78.1% of participants who initiated litifilimab at Week 24. All AEs in the extended treatment period were mild or moderate, except for two AEs that were severe. Serious AEs were reported in 3.6% of participants who initiated litifilimab in the DBPC period and in 3.1% of participants who initiated litifilimab at Week 24. No serious viral infections were observed through Week 52.

Conclusions

In the AMETHYST Part A study, participants on litifilimab demonstrated continuous improvement across multiple clinical endpoints through Week 52. Participants who switched from placebo to litifilimab at Week 24 experienced a rapid onset of treatment effect. Safety data were consistent with the profile previously established in completed litifilimab studies.

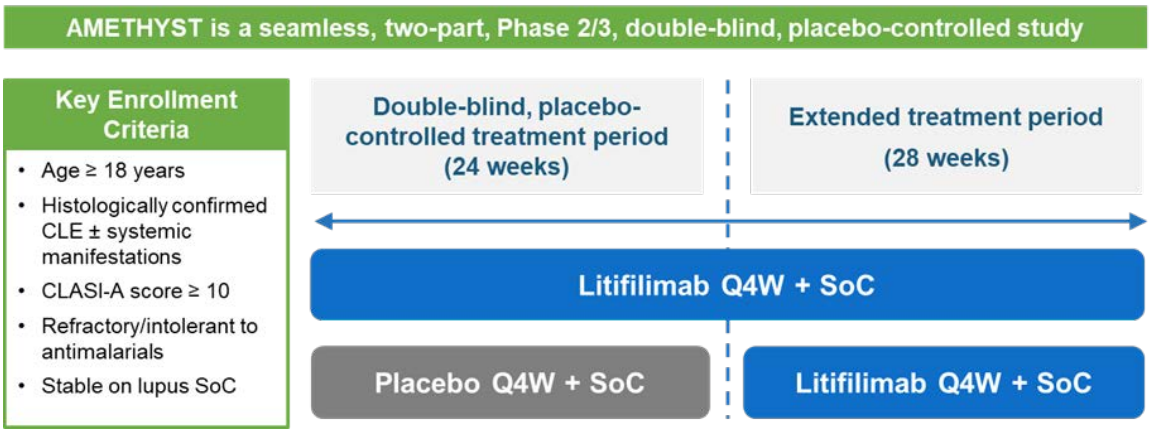

Figure 1. AMETHYST Part A Study Design. CLASI-A = Cutaneous Lupus Erythematosus Disease Area and Severity Index–Activity; CLE = cutaneous lupus erythematosus; Q4W = every 4 weeks; SoC = standard of care.

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Abstract N°: 1849

An Audit of the Effectiveness of Risankizumab in Moderate-to-Severe Psoriasis

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Introduction

Risankizumab is a self-administered, injectable biologic medication used to treat moderate-to-severe psoriasis, psoriatic arthritis, Crohn's disease and ulcerative colitis. It binds selectively to the interleukin-23 cytokine, inhibiting signalling and reducing the release of pro-inflammatory cytokines.

We have audited the use of Risankizumab in patients taking it at week 0, week 4 and every 12 weeks thereafter.

Materials and Methods

We have conducted an audit of 40 patients with moderate-to-severe psoriasis who were started on risankizumab. Baseline data collected included weight, sex, ethnicity, presence of psoriatic arthritis, and previous systemic or biologic therapies. Disease severity and quality of life were assessed using Psoriasis Area and Severity Index (PASI) and Dermatology Life Quality Index (DLQI) scores at baseline and at a 16-week follow-up. Treatment continuation, adverse effects, and service impact including monitoring requirements were also recorded.

Results

From baseline to 16 weeks, the mean PASI improved from 18.9 at baseline to 0.69, representing a 96% reduction.
94% achieved >75% reduction from baseline.
88% achieved >90% reduction from baseline.
71% achieved 100% reduction from baseline.

From baseline to 16 weeks, the mean DLQI improved from 20.4 to 2.86, representing an 86% reduction.
71% achieved a DLQI of 0.

6/40 patients (15%) reported adverse effects which were mostly mild, including fatigue, especially after the first injection, urticaria (pen related), metallic taste in the mouth.
95% of the patients included in the audit continued risankizumab.
82.5% had monitoring visits reduced.

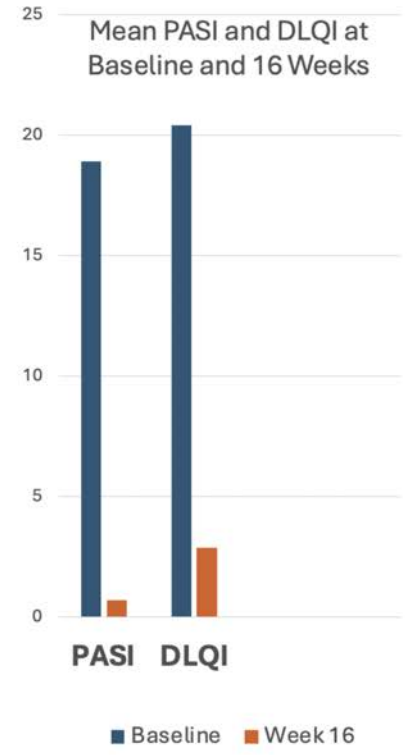

Figure 1

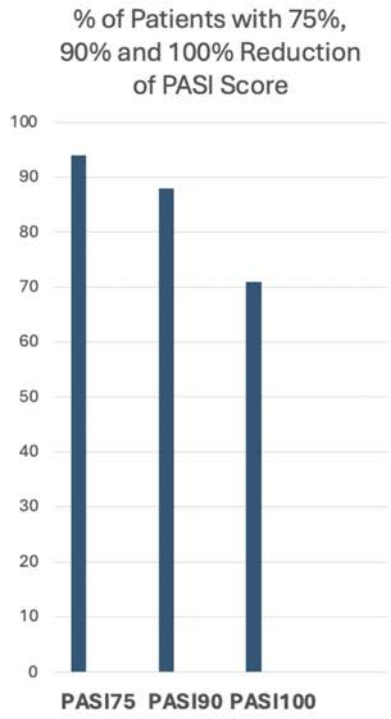

Figure 2

Figure 1: A graph demonstrating the mean PASI and DLQI of the cohort at baseline (0 weeks) and at 16 weeks. Figure 2: A graph demonstrating the percentage of patients in the cohort with a 75%, 90% and 100% reduction in their PASI score.

Conclusions

Risankizumab demonstrated a high level of effectiveness in this cohort, with significant reductions in PASI and DLQI at 16 weeks. Risankizumab was well tolerated with mostly mild adverse effects. 95% of the cohort continued risankizumab – excellent treatment persistence. There was a notable reduction in the amount of monitoring visits in this cohort, reflecting reduced demand on services.

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Abstract N°: 1850

Preclinical characterization of NAV-242, a half-life extended bispecific antibody against TNFα and OX40L

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Introduction

Hidradenitis suppurativa (HS) is a chronic, relapsing inflammatory skin disease associated with comorbidities and detrimental impact on patient quality of life. HS affects 0.4-1% of the global population and presents as painful inflammatory nodules and abscesses, which can lead to formation of oozing fistulas and permanent scarring. Targeting TNF-α has become a preferred therapy for primary HS, but this strategy fails to induce clinical responses in >50% of patients. NAV-240 is a tetravalent bispecific antibody designed to address this unmet need by blocking both TNF-α and OX40L. Phase 1 studies with NAV-240 in healthy volunteers demonstrated safety, tolerability, linear pharmacokinetics (PK) and pronounced reductions in biomarkers of target engagement, such as TARC. NAV-242 is a next-generation therapeutic based on NAV-240, further engineered for enhanced exposure and stability to enable convenient subcutaneous delivery with extended dosing intervals.

Materials and Methods

In vitro and *in vivo* assessments of NAV-242 were made to compare affinity, potency, stability and PK relative to NAV-240.

Results

Enhanced stability at high concentrations was observed upon the addition of a disulfide bond in the scFv domain and the switch from an IgG4 backbone to an IgG1 structure. NAV-242 demonstrated ~2-fold increase in NHP serum half-life (t1/2) and exposure ($AUC_{0-\infty}$) when compared to NAV-240. Binding affinity to TNF-α and OX40L were similar between both compounds. Based on PK modeling from preclinical non-human primate data, NAV-242 is predicted to support quarterly subcutaneous dosing.

Conclusions

Enhanced stability and *in vivo* PK were achieved upon generation of NAV-242, a potential best-in-class therapeutic for autoimmune conditions like hidradenitis suppurativa. Clinical studies with NAV-242 were initiated in early 2026.

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Abstract N°: 1856

Safety, PK, immunogenicity and biomarker modulation of NAV-240, an anti-OX40L/TNFα bispecific antibody, in randomized, double-blind, single and multiple ascending dose Phase 1 studies in healthy volunteers

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Introduction

NAV-240 is a tetravalent bispecific antibody targeting TNFα and OX40L being evaluated for the treatment of hidradenitis suppurativa (HS). While TNFα inhibition is a preferred therapeutic strategy for primary HS, ~50% of patients fail to achieve clinical responses. OX40L blockade complements TNFα inhibition by preventing effector T cell generation while preserving regulatory T cell activity, which may provide durable therapeutic effects extending beyond drug clearance. Combining anti-TNFα and anti-OX40L activity in one molecule addresses both the immediate inflammatory burden and the underlying immune dysregulation that drives disease persistence. In preclinical studies, NAV-240 showed synergistic modulation of inflammatory pathways. Here we present comprehensive data from the Phase 1a and Phase 1b studies of NAV-240.

Materials and Methods

Phase 1a: Single-center, randomized, double-blind, placebo-controlled, sequential single ascending dose (SAD) study of IV NAV-240 in healthy volunteers (NCT06181786), including 5 cohorts: 0.1, 0.3, 1.0, 3.0 and 10.0 mg/kg.

Phase 1b: Single-center, randomized, double-blind, placebo-controlled, sequential multiple ascending dose (MAD) study of IV NAV-240 in healthy volunteers (NCT06933043), including 3 cohorts: 2.0, 5.0 and 10.0 mg every 4 weeks (Q4W) for a total of 3 doses.

For each cohort, 8 participants were randomized (6:2) to active (NAV-240):control (matched placebo).

Safety, pharmacokinetics (PK) and immunogenicity were monitored throughout both studies. Serum samples were evaluated for serum proteomic biomarkers using immunoassays or the Olink® Target 96 Inflammation panel

Results

In total, 64 healthy volunteers were randomized to NAV-240 or placebo; 40 in the SAD study and 24 in the MAD study.

NAV-240 IV administration of a single dose or multiple doses (up to 10 mg/kg) was generally well-tolerated in healthy participants. There were no serious treatment- emergent adverse events (TEAEs) and no TEAEs leading to drug discontinuation, study withdrawal or death in either study.

NAV-240 showed linear, dose-proportional PK profiles following a single- or multiple-dose Q4W regimen. Minimal drug accumulation was observed following multiple Q4W doses.

Overall, anti-drug antibody (ADA) incidence was 36.7% and 29.4% in the SAD and MAD studies, respectively, with a trend towards lower ADA rates at higher dose levels. There was no meaningful impact on PK exposure or clearance when comparing ADA positive and negative participants.

A substantial reduction in TARC was seen as early as 5 days after NAV-240 treatment in healthy volunteers, with time- and dose-dependent TARC reduction observed following NAV-240 administration. At the highest dose of NAV-240 tested (10 mg/kg single dose), TARC suppression was sustained for 72 days beyond the treatment period. A single dose of 10 mg/kg NAV-240 also resulted in a reduction in key inflammatory proteins reported to be elevated in HS patients, including CXLCL9, IL-6, CCL4 and CCL19. This modulation was maintained for up to 10 weeks following a single dose. Biomarker data from the MAD study will be presented.

Conclusions

Treatment with NAV-240 in healthy volunteers demonstrates an acceptable safety and tolerability profile with PK exposure that supports once-monthly dosing in patients. NAV-240 demonstrated pronounced, concentration-dependent reductions in inflammatory proteins, suggesting coordinated modulation of T helper 1/T helper 2/T helper 17 cells and metabolic pathways, many of which are associated with HS. Data from both the SAD and MAD studies informed dose selection for the ongoing Phase 2 study in HS patients (NCT07384975).

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Abstract N°: 1879

Differential Impact of Body Weight and Dose Escalation on Biologic Treatment Response in Plaque Psoriasis: A Real-World Cohort Study

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Introduction

Biologic therapies have transformed the management of plaque psoriasis. Secukinumab (IL-17 inhibitor), adalimumab (TNFα inhibitor) and ustekinumab (IL-12/23 inhibitor) are internationally approved and widely used for treatment of psoriasis.

Increased body weight is associated with reduced biologic efficacy in psoriasis (Pirro et al., 2021; Xu et al., 2025), with studies suggesting a greater impact on interleukin inhibitors (Pirro et al., 2021; Rosario et al., 2014; Zweegers et al., 2016). Weight-based dosing improves outcomes with ustekinumab and dose escalation with secukinumab (Lebwohl et al., 2010; McGuire et al., 2023). These findings are reflected in current guidance: adalimumab dosing is independent of weight but may be escalated if inadequate response; ustekinumab dosing is weight-based; and secukinumab is fixed-dose, with EMA and NICE guidelines permitting weight-based escalation (BAD, 2023; FDA, 2025).

Despite established guidelines, comparative data on impact of body weight and dose optimisation across biologics is limited. This study evaluates real-world efficacy of adalimumab, secukinumab and ustekinumab, focusing on initial response, dose optimisation, and impact of weight on both.

Materials and Methods

Retrospective data were collected from a multi-site UK trust (January 2026) including patients with plaque psoriasis treated with ustekinumab (n=68), secukinumab (n=35), and adalimumab (n=69). Data collected included Psoriasis Area and Severity Index (PASI) scores, weight, and clinician- and patient-reported disease control. Treatment response was defined as achieving PASI90 or clinician-documented disease control at follow-up (16 weeks). Welch’s t-tests and exact contingency methods were used to assess the effect of body weight on treatment response at initiation and after dose escalation.

Results

Initial response rates were 83% (57/69) for adalimumab, 74% (26/35) for secukinumab, and 56% (37/68) for ustekinumab, with mean weights of 81.9 kg, 94.4 kg, and 86.3 kg, respectively [Figure 1]. Weight had the greatest impact on ustekinumab, with all patients >100 kg initially treated with 45 mg (n=5) requiring dose escalation. Secukinumab showed a partial weight effect, with 47% of patients $\geq$90 kg poorly controlled on monthly dosing and 78% of those poorly controlled on initial dosing weighing $\geq$90 kg. In contrast, adalimumab efficacy was consistent across weight ranges. Non-responders had higher mean body weight for ustekinumab (89.3 vs 82.0 kg, p=0.13) and secukinumab (98.1 vs 92.8 kg, p=0.42), but not for adalimumab (79.9 vs 82.5 kg, p=0.73).

Escalation success rates were 100% (7/7) for adalimumab, 71% (5/7) for secukinumab, and 80% (16/20) for ustekinumab; however, there was no statistically significant difference across drugs (two-tailed p=0.41). Among ustekinumab patients requiring dose escalation, 12/20 weighed >100 kg. All patients >100 kg on ustekinumab required dose optimisation and of those with follow-up, response was achieved in 91% (10/11). Adalimumab showed consistently good effect following escalation, with all patients responding, precluding statistical comparison, but showed no clear weight impact (those escalated ranging 51–133 kg). Secukinumab escalation was effective in 4/5 patients ≥90 kg and 1/2 <90 kg. Baseline weight did not significantly influence escalation success for ustekinumab (p=0.63) or secukinumab (p=0.78).

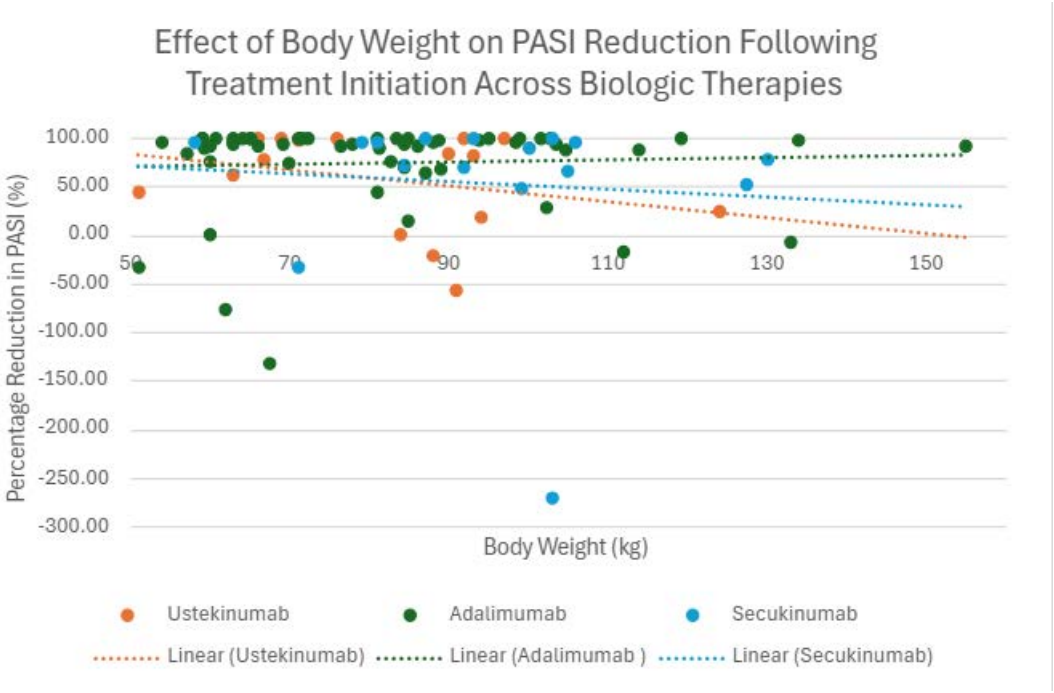

Figure 1: Percentage reduction in Psoriasis Area and Severity Index (PASI) at 16 weeks relative to pre-treatment PASI is plotted against body weight for patients treated with standard dosing of ustekinumab 45mg 12 weekly, adalimumab 40mg 2 weekly, and secukinumab 300mg monthly. Data represent response at 16 weeks post-initiation.

Conclusions

Our results suggest that bodyweight influenced treatment response differently across biologic classes, with a greater impact on interleukin inhibitors, in line with previous published reports and supporting guidelines. Dose escalation was effective across all biologics, including in higher bodyweight patients, supporting weight-informed treatment and dose optimisation in clinical practice in selective patients. Although trends were observed, these findings were not statistically significant and suggest that factors beyond bodyweight contribute to variability in biologic response.

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Abstract N°: 1910

Efficacy and safety of xeligekimab in moderate-to-severe plaque psoriasis patients with lower limbs lesions: a single-center, prospective, observational study

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Introduction

Biologic agents have greatly improved the treatment of moderate-to-severe plaque psoriasis, however, a substantial proportion of patients persist residual skin lesions and relapse due to difficult-to-treat skin lesions. Retrospective studies have identified the lower limbs as the most common site of residual disease, with reported rates ranging from 49.3% to 63.01%. The lower limbs are well-recognized difficult-to-treat anatomical site. Furthermore, lesions one the lower limbs are also the frequent site of relapse. The lower legs lesions continue to cause significant physical discomfort and have a notable negative impact on patients' quality of life. Xeligekimab, a novel IL-17A-neutralizing monoclonal antibody, has demonstrated strong efficacy in the pivotal phase III trials, and post-hoc analysis showed that xeligekimab improved lower legs lesions starting at week 4 of treatment, and 73.6% of patients achieved complete lower leg lesion clearance. Here, we investigated the therapeutic potential of xeligekimab for patients with refractory residual lower legs lesions.

Materials and Methods

We are conducting the prospective, observational, single-arm study in adult moderate-to-severe plaque psoriasis patients with lower limb lesions the enrolled patients received 200 mg xeligekimab. The primary endpoint was the proportion of patients achieveing lower limbs BSA <1% at week 12; Key secondary endpoints included the proportions of patients achieving PASI 75 , PASI 90 at week 12, and DLQI 0/1 at week 24; Other secondary endpoints included change from baseline in BSA involvement, lower limb BSA involvement, PASI score, lower limb PASI score, DLQI score at week 24.

Results

Until Apr 16, 2026, 14 patients with moderate-to-severe plaque psoriasis affecting the lower limbs were enrolled, 6 patients were male. To date, all 8 patients completed week 4 follow-up, and 3 patients completed week 12 follow-up. 5 out of 8 patients had received prior biologic therapies and without wash-out period. Both total BSA involvement and lower limb BSA involvement improved over the observation period. The mean total BSA decreased from 16.3 % at baseline to 10.3% at week 4 and 4.0 % at week 12 , and the mean lower limb BSA declined from 9.1% to 5.8% at week 4 and 2.0% at week 12. Both total PASI scores and lower limb PASI scores showed similar patterns, the mean total PASI score decreased from 12.9 at baseline to 7.3 at week 4, and 3.0 at week 12, and the mean lower limb PASI score declined from 9.0 to 4.9 at week 4, and 1.6 at week 12. DLQI scores decreased from 14 to 10 by week 4, with a further decrease to week 12 . Over the observation period, xeligekimab demonstrated a favorable safety profile.

Conclusions

In this single-arm trial, xeligekimab demonstrated significant clinical improvement and a favorable safety profile in a subset of patients with refractory lower limbs lession plaque psoriasis. Patients achived a reduction in both total BSA involvement and lower limb BSA involvement as early as Week 4, as well as PASI score. Xeligekimab is expected to become a new treatment option for moderate-to-severe plaque psoriasis with lower limb involvement, whereas its long-term efficacy requires further observation.

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Abstract N°: 1912

A Distinct Rosacea-Mimicking Facial Inflammatory Phenotype in Psoriasis: Preliminary Evidence for Synchronous Response to IL-17A Blockade with Xeligekimab

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Introduction
Psoriatic patients frequently present with facial papulopustular eruptions that clinically and dermoscopically mimic rosacea—characterized by central facial erythema, inflammatory papules, and a conspicuous absence of scaling. The nosological status of these lesions remains ambiguous. We hypothesize that such facial eruptions represent a distinct, underrecognized psoriatic facial phenotype that shares the IL-17-driven inflammatory pathway characteristic of coexisting scalp psoriasis.This study to characterize the clinical and dermoscopic features of this rosacea-mimicking facial phenotype and evaluate its response to IL-17A blockade with Xeligekimab.

Materials and Methods
We are conducting a prospective, proof-of-concept cohort study. Key inclusion criteria: (1) moderate-to-severe active scalp psoriasis requiring systemic therapy; (2) concurrent facial papulopustular lesions clinically mimicking rosacea, without scaling; and (3) documented failure of conventional rosacea-directed therapies. Patients receive standard-label Xeligekimab (anti-IL-17A, IgG4) for scalp psoriasis. Serial assessments include clinical photography, dermoscopy, multispectral vascular imaging, and collection of biospecimens for ongoing biomarker analysis.

Results
Preliminary observations from the ongoing cohort demonstrate marked and synchronous improvement in both scalp psoriasis and the rosacea-mimicking facial lesions following IL-17A blockade. To date, three patients have completed 12 weeks of therapy. Baseline dermoscopy and multispectral vascular imaging of facial lesions revealed a predominance of polygonal vessels—a pattern typically associated with rosacea—which regressed in parallel with clinical clearance. Enrollment and biomarker analyses are ongoing.

Conclusions
These preliminary findings suggest the existence of a distinct, rosacea-mimicking facial inflammatory phenotype in patients with psoriasis. This phenotype is characterized by scaling-free papulopustules, polygonal vascular patterns on imaging, and a synchronous therapeutic response to IL-17A blockade that parallels scalp psoriasis clearance. Active scalp psoriasis provides a critical clinical context for suspecting this phenotype. Complete cohort data with molecular correlates will be presented in the final analysis.

EADV CONGRESS
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Abstract N°: 1918

"An ‘innocent’ forehead lesion"

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Introduction

Blastic plasmacytoid dendritic cell neoplasm (BPDCN) is a rare and aggressive hematologic malignancy that predominantly, though not exclusively, involves the skin. It is an insidious condition that is often difficult to diagnose, initially presenting with violaceous papules or nodules with a bruise-like appearance, particularly on the trunk and face. These lesions may be solitary or multiple and are typically asymptomatic, but the disease can later evolve into an aggressive systemic condition with potential rapid leukemic dissemination.

Case Presentation

We report the case of a 56-year-old woman, previously in good health, who presented to our Dermatology Clinic in February 2021 with a cutaneous lesion appearing as a faint erythematous patch on the glabella, asymptomatic, with no reported pruritus or pain. The lesion regressed following initial treatment with topical pimecrolimus ointment but recurred in January 2022. In March 2022, a punch biopsy was performed, confirming the diagnosis of BPDCN.
At the time of diagnosis, the patient had no splenomegaly, hepatomegaly, or additional skin lesions; blood tests and bone marrow evaluation showed minimal involvement (0.8% CD123+ cells). Treatment with tagraxofusp was initiated in May 2022 for five cycles, achieving complete remission until January 2023, when a new frontal skin lesion appeared. Biopsy confirmed disease relapse. The diagnostic delay was 13 months from the patient’s initial evaluation at our center.

Conclusion

Following the fifth cycle of tagraxofusp, the patient achieved complete remission with a negative PET scan. After relapse in January 2023, complete remission was achieved with the Hyper-CVAD chemotherapy regimen followed by allogeneic stem cell transplantation. Overall survival is generally limited to a maximum of two years; however, with the advent of novel therapies—specifically tagraxofusp, a CD123-targeting fusion protein—survival may be extended. In this case, the patient remains in complete remission five years after disease onset.

EADV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 1930

Combinations of targeted therapies relevant for patients with psoriasis and associated immune-mediated diseases: a scoping review

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Introduction

Achieving adequate response with targeted monotherapy in patients with psoriasis (PsO) can still be challenging, despite the multiple available effective treatments. Combinations of targeted therapies (CTTs) may increase effectiveness, but a comprehensive evidence overview is still lacking. Therefore, the aim was to systematically map the available literature on CTTs (biologic+biologic, biologic+small molecule inhibitor (SMI), SMI+SMI) within the management of patients with PsO, also incorporating evidence from CTTs initiated for other diseases that share targeted therapies with PsO.

Materials and Methods

MEDLINE, Embase, Web of Science and Cochrane Library were searched from 2015 to October 2025 (Figure 1). Eligible studies described CTTs containing ≥1 biologic or SMI approved for the treatment of adults with PsO. When prescribed in patients with PsO, PsO effectiveness and/or safety outcomes were required. When prescribed in patients with other inflammatory diseases that share targeted therapies with PsO, safety outcomes were required. For CTTs in patients with PsO, effectiveness was categorized as improvement, stable, secondary failure, or primary failure, and adverse events (AEs) were classified using the Medical Dictionary for Regulatory Events (MedDRA). Occurrence of serious AEs (SAEs) was reported for all CTTs.

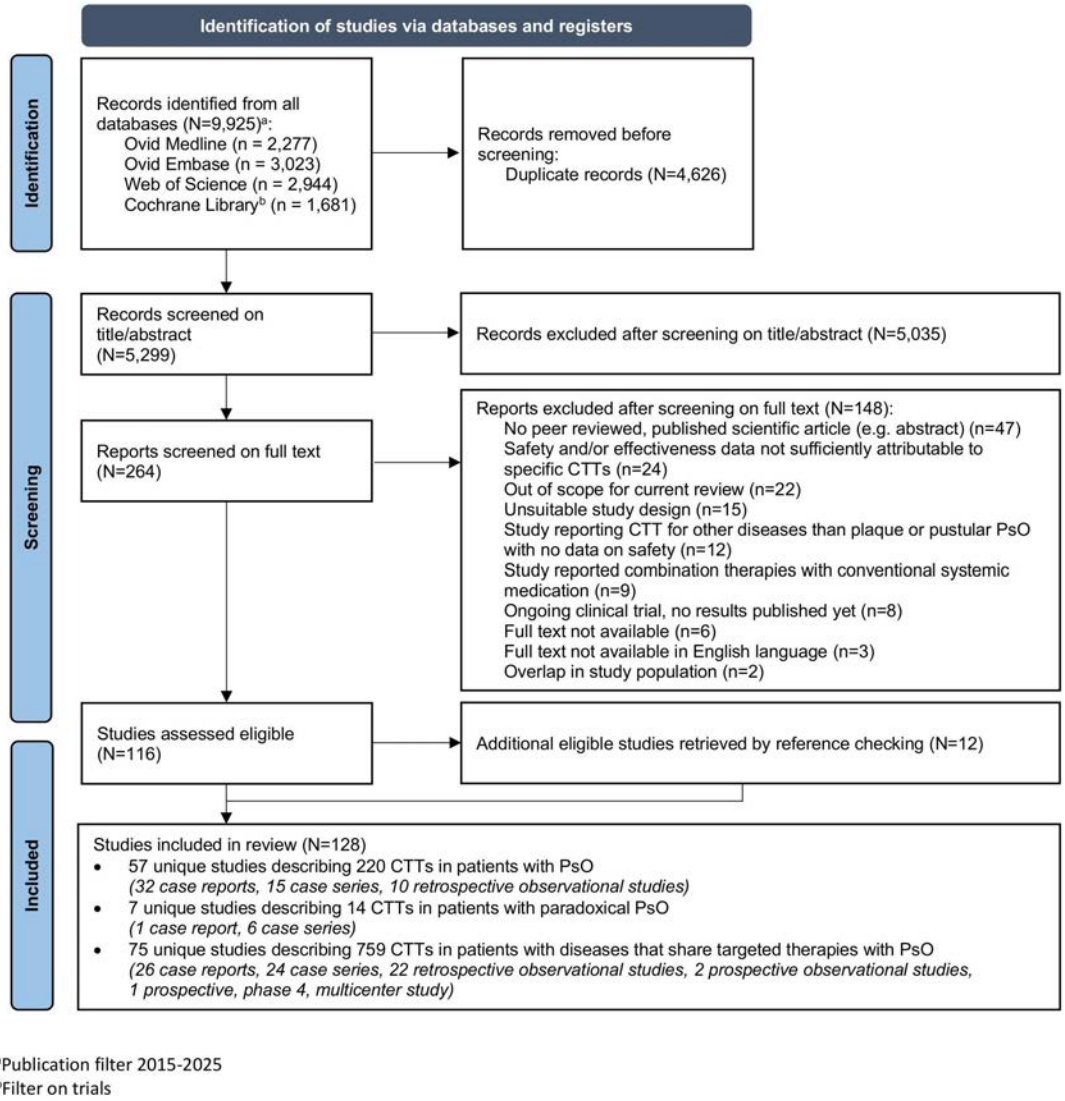

Figure 1: PRISMA flowchart illustrating the study selection process

Results

In total, 128 studies were included, of which 57 described 220 unique CTTs (113 biologic+biologic and 107 biologic+SMI) in patients with PsO. Of the 113 biologic-biologic CTTs, 47 were initiated in patients with refractory PsO (Figure 2). Of these, only 6.4% were initiated for isolated refractory PsO, whereas 93.6% were initiated for refractory PsO and a concomitant disease, predominantly psoriatic arthritis or atopic dermatitis. The most reported CTT was an IL23-inhibitor combined with dupilumab. Most dual-biologic CTTs (88.6%) initiated for refractory PsO resulted in improvement of PsO. Of the 107 biologic-SMI CTTs, mainly biologic-apremilast combinations, 56.1% were initiated for isolated refractory PsO, and 29.9% for refractory PsO and a concomitant disease. Of the biologic-SMI CTTs initiated for refractory PsO, the majority (94.0%) resulted in improvement of PsO. Across all CTTs initiated in patients with PsO (N=220), the most frequent reported AEs were *infections and infestations* (N=10) in dual-biologic CTTs and *gastrointestinal disorders* (N=20) in biologic-SMI CTTs. Only 6 (2.7%) SAEs were reported. An additional 11 CTTs initiated in patients with refractory paradoxical PsO were identified. Eight CTTs were a combination of two biologics, mainly a TNFα-inhibitor combined with ustekinumab, and 3 CTTs were a combination of a biologic with a JAK-inhibitor. Half (4/8) of the dual-biologic CTTs and all 3 biologic-SMI CTTs resulted in improvement of the paradoxical PsO. Only one mild AE (*infections and infestations*) and no SAEs were observed. Across the identified 759 CTTs in diseases sharing targeted therapies with PsO, mostly including vedolizumab for inflammatory bowel disease (IBD), unique SAE rates were also relatively low (6.6%) and seemed to reflect the underlying IBD population. Overall, no deaths were reported during all CTTs identified.

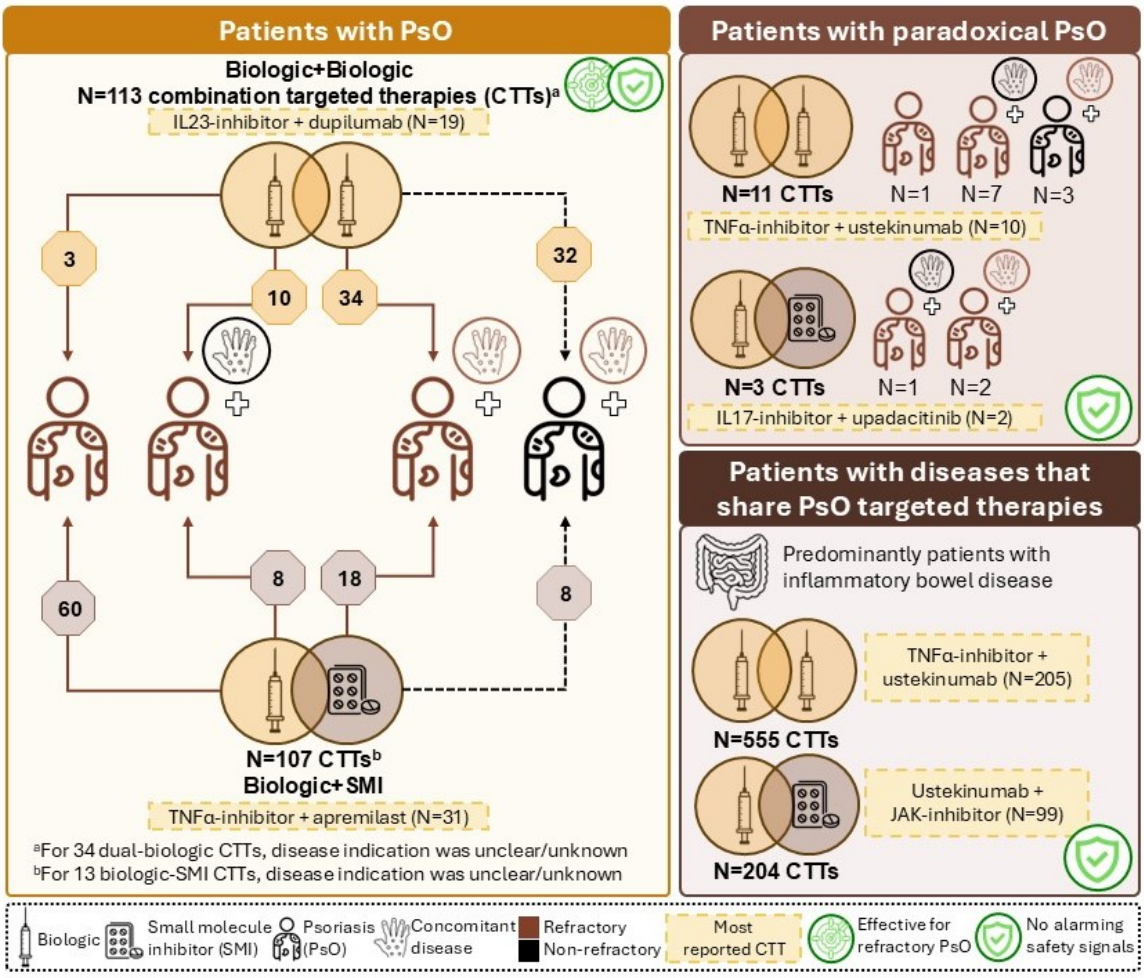

Figure 2: Overview of identified combination targeted therapies (CTTs) within available literature

Conclusions

Overall, CTTs - both biologic-biologic and biologic-SMI combinations - appeared effective when initiated for refractory PsO. In addition, no alarming safety signals were observed across all identified CTTs. Until more robust evidence on effectiveness and safety of these regimens becomes available, vigilance and monitoring is recommended.

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Abstract N°: 1940

Diffuse Cutaneous Desquamation Following Tocilizumab in an Adolescent with Multisystem Sarcoidosis

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Introduction

Tocilizumab, an anti–interleukin-6 monoclonal antibody, is increasingly used in severe pediatric sarcoidosis. Cutaneous adverse reactions are rare and poorly documented in children, particularly in the form of diffuse body desquamation.

Case Presentation

We report the case of a 14-year-old girl followed for multisystem sarcoidosis with articular, ocular, and pulmonary involvement. She had previously been treated with systemic corticosteroids, methotrexate, and infliximab, and was currently receiving tocilizumab at a dose of 400 mg/month.

In 2019, the patient received a first infusion of tocilizumab without any cutaneous reaction. However, after the second and third administrations, she developed diffuse cutaneous desquamation involving the entire body, occurring 4 days after each injection, including the palms, soles, and face. Her general condition remained stable, and she was afebrile. The reaction was not severe but caused mild functional discomfort of the hands and feet.

Management consisted of antihistamines and corticosteroid bolus premedication for subsequent infusions, allowing continuation of treatment without further adverse cutaneous events.

Conclusion

This case highlights that diffuse cutaneous desquamation, although non-severe, may occur in children receiving tocilizumab even after initial tolerance. Dermatological monitoring and appropriate premedication are essential to ensure safe continuation of therapy.

EADV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 1966

Meningococcemia under JAK inhibition presenting with retiform purpura without coagulopathy

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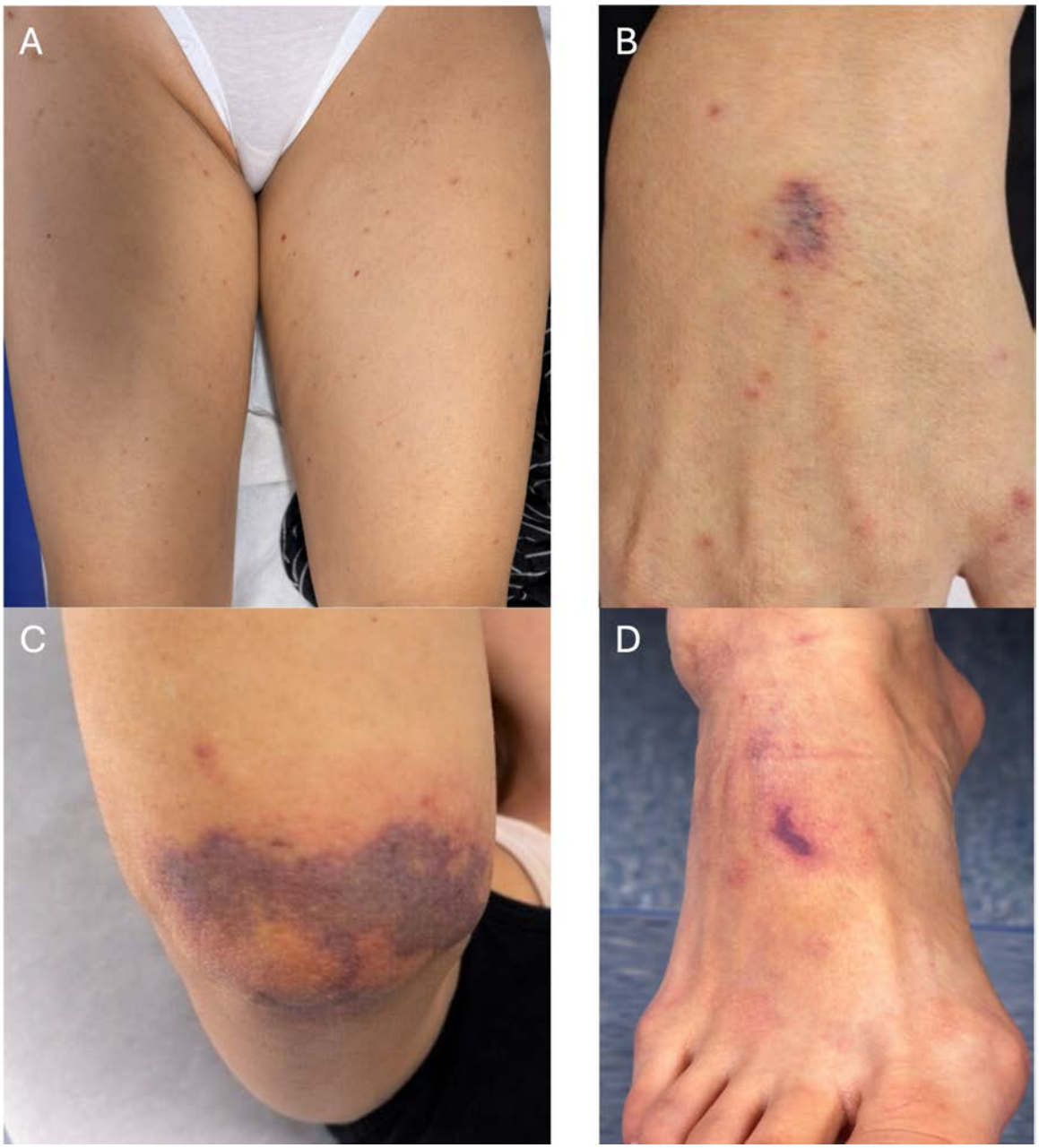
Introduction

Baricitinib is an established therapeutic option for severe alopecia areata, reflecting the expanding integration of Janus kinase (JAK) inhibitors into dermatologic practice. Although phase 3 trials and integrated safety analyses have demonstrated an overall favorable safety profile in alopecia areata, serious infections remain a recognized class-related risk of JAK inhibition.

Case Presentation

A 40-year-old woman with long-standing alopecia universalis had been receiving continuous baricitinib 4 mg daily since April 2024, with unremarkable laboratory findings. On October 19, 2025, she reported the onset of fever (>38°C), asthenia, and diffuse arthralgia. Over the following days, she developed the abrupt onset of non-tender petechial lesions on the lower extremities, which rapidly progressed over 48–72 hours into violaceous, non-blanching, angulated purpuric plaques with a retiform configuration involving the dorsal hands, feet, and elbows. No cutaneous necrosis or ulceration was observed. The rapid morphologic progression raised concern for a systemic process, prompting discontinuation of baricitinib and hospital admission on October 22, 2025. Blood cultures obtained on October 25, 2025, during a second febrile peak yielded *Neisseria meningitidis*, confirming the diagnosis. Cerebrospinal fluid analysis and neuroimaging excluded central nervous system involvement. The patient had not received prior meningococcal vaccination. Initial outpatient laboratory testing revealed leukocytosis (20 × 10^3/μL), which remained elevated at hospital admission (WBC 10.6 × 10^3/μL; neutrophils 79.9%). Coagulation parameters revealed markedly elevated fibrinogen (962 mg/dL), consistent with an acute-phase response, while platelet count (264 × 10^3/μL), prothrombin time, and activated partial thromboplastin time remained within normal limits. No laboratory evidence of disseminated intravascular coagulation was observed. Intravenous ceftriaxone was administered for 7 days in accordance with current recommendations for invasive meningococcal disease. The patient remained hemodynamically stable without requiring intensive care support and achieved complete remission of systemic and cutaneous manifestations. At follow-up evaluation in January 2026, she remained clinically stable without recurrent infectious events. Baricitinib had not been resumed, and she continues under infectious disease follow-up. JAK1/2 signaling plays a key role in antimicrobial defense and inflammatory responses. Pharmacologic inhibition is associated with increased susceptibility to infection while potentially modulating inflammatory pathways. To our knowledge, the clinical presentation of meningococcemia during JAK inhibition is not well characterized. In adults, invasive meningococcal disease may progress to fulminant septicaemia characterized by disseminated intravascular

coagulation, thrombocytopenia, and necrotic purpura fulminans, typically associated with consumptive coagulopathy and hemodynamic instability. In contrast, our patient developed retiform purpura without evidence of consumptive coagulopathy, thrombocytopenia, cutaneous necrosis, or organ dysfunction. Although causality cannot be established, the relatively preserved coagulation profile and hemodynamic stability observed in this case represent a hypothesis-generating observation. While JAK1/2 inhibition is known to increase susceptibility to infection, its potential impact on the inflammatory and coagulopathic response in invasive bacterial disease remains uncertain and warrants further investigation.



Clinical images showing non-blanching purpuric lesions with different morphologic patterns. (A) Scattered petechial lesions on the thighs. (B) Mixed petechial and purpuric lesions on the dorsal hand. (C) Confluent violaceous purpuric plaque with angulated borders on the elbow. (D) Retiform purpura on the dorsal foot.

Conclusion

Retiform purpura represents a cutaneous marker of vascular compromise requiring prompt systemic evaluation to exclude thrombotic, vasculitic, or infectious etiologies. In patients receiving targeted immunomodulators, the clinical expression of invasive infections may reflect modified inflammatory signaling, and clinicians should remain

alert to potential variability in inflammatory presentation.

EA DV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 2093

Hypereosinophilia with organ damage secondary to Dupilumab therapy: a case report and literature review

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Introduction

Dupilumab, a monoclonal antibody that targets interleukin (IL)-4 and IL-13 signaling pathways, is recommended for the treatment of moderate to severe atopic dermatitis (AD). Its safety and efficacy has been reported with high completion rates and sustained clinical improvement across studies. Dupilumab has also been associated with elevations in blood eosinophil counts, particularly in patients with higher baseline level. Although dupilumab-associated eosinophilia is usually mild and transient, severe hypereosinophilia with organ involvement is rare but clinically important. The aim of this article is to report a rare case of hypereosinophilia with organ damage secondary to dupilumab therapy in a patient with AD.

Case Presentation

A 22 years old male presented to the clinic complaining of a chronic history of childhood AD and severe itchiness. Despite repeated courses of topical steroids and calcineurin inhibitors he had recurrent relapses. Therefore, considering the chronicity of his eczema, lack of response to topicals, and the stress it caused him, dupilumab therapy was initiated.

The patient improved significantly but 5 months later he presented to the emergency department with palpitations and chest pain. His laboratory results revealed an absolute eosinophil count of 2070 cells/ul (0-400 cells/ul), while his baseline count was 860 cells/ul before the initiation of dupilumab. His troponin I level was 1.56 ng/ml (<0.04 ng/ml). A thorough cardiac work up was done including cardiac magnetic resonance which confirmed myocarditis. Based on those results a diagnosis of eosinophilic myocarditis was made by cardiology and the patient was initiated on prednisilone 40 mg and colchicine 500 mcg daily. He was then referred to hematology oncology and he was investigated for his hypereosinophilia thoroughly. Several tests ,including a whole exome sequencing, were done but did not reveal any pathological results. Therefore, he was diagnosed with hypereosinophilia secondary to dupilumab. As a result, dupilumab was discontinued which led to the return of the patient's eosinophil count to normal range (190 cells/µL) and improvement of his symptoms. A slight increase of the patient's eosinophil count (790-850 cells/µL) was noticed during his follow ups, but he was doing well and no further intervention was made as this represented the patient's baseline count.

Management approaches to dupilumab-associated hypereosinophilic organ damage include the discontinuation of dupilumab and starting systemic corticosteroids. For refractory cases, a use mepolizumab or benralizumab was reported to be successful. Kemp P et al. recommended monitoring eosinophil count for all patients on dupilumab therapy at baseline, week 4, and every 12 weeks thereafter. A more frequent monitoring, every 2 weeks, should be preserved for patients with an eosinophil count ≥

3000 cells/ul, while treatment should be halted whenever the count reaches ≥ 5000 cells/ul. We also recommend counseling hematology and immunology specialists in case of a persistently high eosinophil count or during the development of any symptom indicating organ dysfunction. In addition a complete work up should be done to exclude other causes. These include complete blood count with differential, antinuclear antibody, antineutrophil cytoplasmic antibody, renal profile, viral panel, stool test for ova and parasites, and genetic testing. Other tests and imaging modalities should be symptoms guided.

Conclusion

Dupilumab has significantly improved outcomes in moderate to severe AD cases. However, clinicians should be aware of its potential adverse effects. This case underscores the importance of thorough evaluation of new systemic symptoms and regular monitoring of eosinophil counts in patients receiving dupilumab, while maintaining a high index of suspicion for rare but serious complications.

EADV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 2101

Rapid corticosteroid tapering and steroid-sparing effect of rituximab in pemphigus: a real-world retrospective analysis

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Introduction
Pemphigus is an autoimmune blistering disease characterized by autoantibodies against desmosomal proteins. Systemic corticosteroids remain the standard first-line therapy; however, prolonged use is associated with significant adverse effects. Rituximab, an anti-CD20 monoclonal antibody, has been increasingly used in the treatment of pemphigus, with accumulating evidence supporting its effectiveness; however, real-world data on its effectiveness and steroid-sparing effect remain limited. This study aimed to evaluate the clinical effectiveness, safety, and corticosteroid-sparing effect of rituximab in patients with pemphigus.

Materials and Methods
We conducted a retrospective analysis of patients with pemphigus treated with systemic corticosteroids with or without rituximab between April 2017 and September 2025. Patients with insufficient clinical data were excluded. Outcomes included clinical remission and corticosteroid-sparing effects, assessed by cumulative prednisolone dose, maintenance dose, and time to dose reduction.

Results
A total of 19 patients were included: 9 treated with prednisolone alone and 10 treated with rituximab plus prednisolone. Baseline disease severity was comparable between groups, although patients in the rituximab group were significantly younger. All patients achieved clinical remission during follow-up. Rituximab-treated patients demonstrated significantly faster corticosteroid tapering compared with the prednisolone-alone group. Prednisolone doses were significantly lower in the rituximab group at multiple time points (3–12 months; $p=0.0335$, 0.0016, 0.0003, and 0.0070, respectively; Figure 1A). The time required to reduce prednisolone to ≤10 mg/day was significantly shorter in the rituximab group ($p<0.0001$; Figure 1B). Accordingly, cumulative prednisolone dose was higher in the prednisolone-alone group, indicating a steroid-sparing effect of rituximab (Figure 1C). Anti-desmoglein 1 and 3 antibody levels decreased over time in both groups, with a more pronounced decline observed in the rituximab group, particularly for anti-desmoglein 3 antibodies (Figure 1D). No adverse events, including infections, were observed.

Conclusions
Rituximab enabled rapid corticosteroid tapering and reduced cumulative steroid exposure in patients with pemphigus in real-world clinical practice. These findings support the early use of rituximab as a strategy to minimize corticosteroid burden while maintaining disease control.

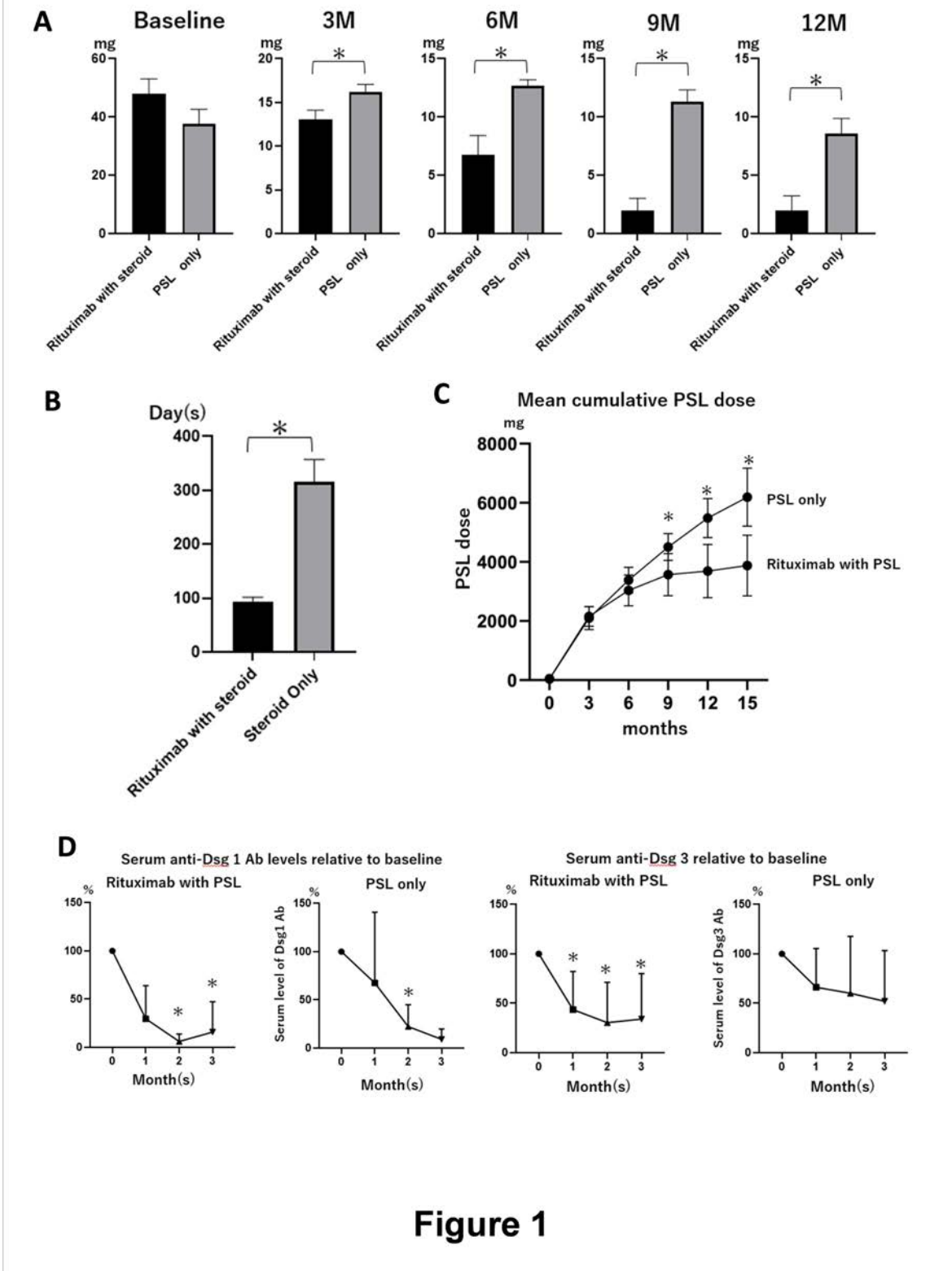

Figure 1

Figure 1. Clinical and biological responses to treatment in patients with pemphigus. (A) Time course of prednisolone dose (mg/day) in patients treated with prednisolone alone or rituximab plus prednisolone. (B) Comparison of time required to reduce prednisolone to ≤10 mg/day between treatment groups. (C) Mean cumulative prednisolone dose over time. (D) Time course of anti-desmoglein 1 and anti-desmoglein 3 antibody levels, expressed as changes from baseline. Values are presented as mean ± standard deviation where applicable. A p-value <0.05 was considered statistically significant.

EA DV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 2121

Integrating Cryosurgery and Immunotherapy for Inoperable Auricular Melanoma of Unknown Primary

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Introduction

Melanoma of unknown primary (MUP) presents as a rare metastatic melanoma with no identifiable primary lesion, complicating both diagnosis and management. We report a case of MUP of the left concha initially treated with radical auriculectomy. Subsequent presentation of metastatic melanoma on the stump was successfully managed with one year of pembrolizumab and aggressive contact cryosurgery to the metastatic lesions.

Case Presentation

73-year-old male presented with several asymptomatic, brownish-black papules on the left concha for four months (Fig. 1A). Medical history and otologic exam were unremarkable. Biopsy revealed dermal Melan A-positive atypical melanocytes without epidermal connection or an in situ component, consistent with metastatic melanoma. The tumor was MITF-positive and BRAF-mutation-negative (Figs. 1B-D). Imaging (CT, PET-CT, sentinel node) showed no primary or additional metastases, confirming MUP.

One month later, he underwent auriculectomy with complete removal of the external ear (Fig. 1E). His postoperative course was unremarkable. The following month, he began adjuvant pembrolizumab 200 mg IV every three weeks for twelve months.

Three months later, recurrence appeared as new pigmented papules on the stump (Fig. 1F). Biopsy confirmed melanoma. The patient declined further surgery, opting for conservative management.

Cryosurgery was performed on all nodules. All lesions regressed and pembrolizumab was continued. The patient’s course was uneventful.

At three-year follow-up, there was no recurrence or metastasis (Fig. 1G).

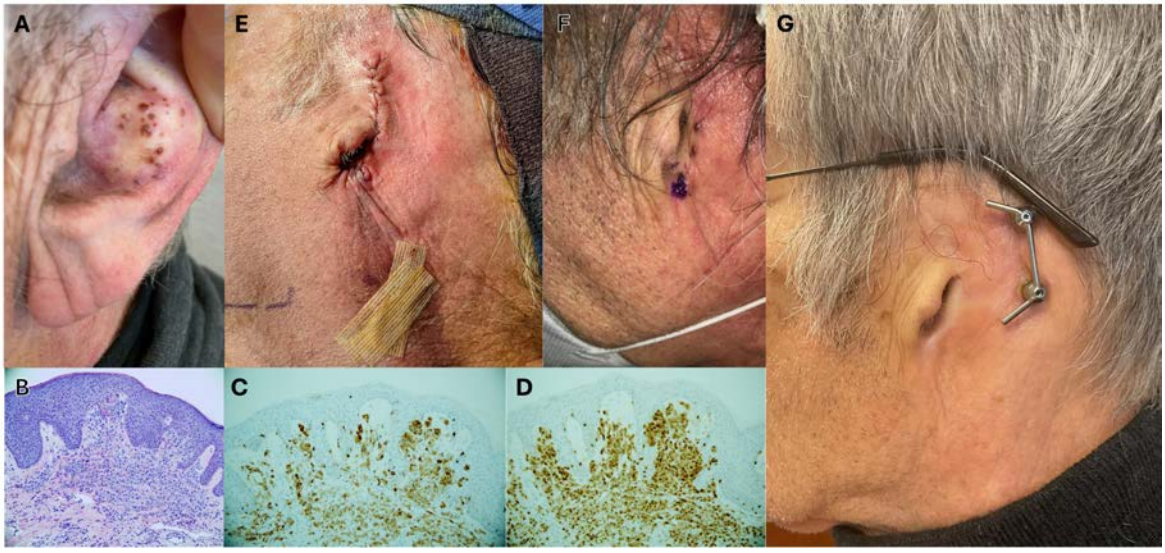

Figure 1A-G. (A) Clinical presentation revealed multiple irregular brownish-black papules on the left concha. (B) Nodular dermal proliferation of melanocytes arranged in nests beneath the epidermis (hematoxylin-eosin stain, 200×). (C) Immunohistochemistry with Melan A positivity in the dermis without an epidermal connection or in situ component (200x). (D) Immunohistochemistry with MITF positivity (200x). (E) Radical auriculectomy of the external ear. (F) Clinical presentation of recurrent brownish-black papules on the amputation site along left external auricular canal. (G) Clinical presentation at 3-year-follow up.

Conclusion

Melanoma of unknown primary (MUP) is rare, comprising only 3–4% of all melanoma cases, and is exceptionally uncommon in the auricular region. Histopathologic interpretation of MUP usually pertains to dermal and/or subcutaneous foci of atypical melanocytes with minimal perifocal inflammation in the absence of junctional activity or without any connection with the epidermis. Diagnosis requires a thorough investigation for any regressed or hidden primary lesions, which are often difficult to detect. Due to its rarity, optimal treatment strategies remain unclear. While surgery is typically the primary treatment for localized melanoma, further excision was not feasible in our case due to the anatomic location and risk of significant mutilation.Therefore, we pursued a combination of local cryosurgery and systemic immunotherapy with pembrolizumab, aiming to achieve local control while preserving function and quality of life.

Cryoablation can be used to reduce tumor burden in unresectable melanoma lesions with high metastatic load and may slow the rate of tumor spread in metastatic disease. Among various ablative techniques, cryoablation with cryoprobe application allows deeper penetration of the cryogen and more efficient cryonecrosis of malignant cells, approximately 1.3 times the peripheral freezing halo, while also inducing a systemic anti-tumor immune response. Cryonecrosis often preserves secondary and tertiary structures of internal membranous and cellular proteins, rendering them exposed to the immune system and thereby enhancing immune response to melanoma antigens. This response is further complemented and enhanced by the immunostimulatory, proapoptotic, and antiangiogenic effects of immunotherapy.

Cryotherapy is especially useful for melanomas in anatomically challenging locations (external ear, periorbital area, nasal ala), where surgery risks significant cosmetic and functional defects. It allows more precise tumor removal while sparing healthy tissue and reducing surgical morbidity compared to conventional excision.

This case demonstrates that combined cryosurgery and immunotherapy may be a promising option for inoperable and complex MUP, especially in challenging locations.

EADV CONGRESS
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30 SEP - 3 OCT 2026

Abstract N°: 2140

The current landscape of biologics provision across the United Kingdom in adult patients with psoriasis

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Introduction
There are currently 12 biologic agents and three small molecules available in the United Kingdom (UK) for the treatment of adults with plaque psoriasis. They currently fall under the high-cost drugs remit for prescribing. Inequitable access to these agents may compromise patient care and outcomes. The aim of this survey was to determine the current decision processes of prescribing biologics in adults with psoriasis across the UK.

Materials and Methods
An anonymised electronic survey was circulated via the British Association of Dermatologists (BAD) and British Dermatological Nursing Group newsletters, the Scottish Dermatology Society, Irish Association of Dermatologists and the BAD National Trainee Committee Representatives.

Results
186 complete responses were received from 175 responders between April and December 2025. In 11 cases of duplicate responses, the latter response was included in the analysis. 116/175 (66.3%) respondents were from England, 39/175 (22.3%) from Scotland, 11/175 (6.3%) from Ireland and 9/175 (5.1%) from Wales. 141/175 (80.6%) respondents were doctors, 32/175 (18.3%) were nurses and 2/175 (1.1%) were pharmacists. 133/175 (76%) respondents had a specified high-cost drugs pathway. Only 77/175 (44%) respondents had an MDT at which the eligibility of patients could be discussed. 102/175 (58.3%) respondents had a dedicated dermatology pharmacist. 119/175 (68%) respondents had a biologics nurse. The four most common decision makers were consultant (173/175, 98.9%), biologics nurse (53/175, 30.3%), registrar (48/175, 27.4%) and SAS grade (38/175, 21.7%).

Conclusions
The survey results reveal disparity in the provision of biologics for adult patients with psoriasis across the UK. Many regions do not have an MDT or a pathway which would facilitate rational selection of treatments, reduce practice variability and optimise resource utilisation. We suggest that there is merit in developing a national high-cost drug pathway to aid with decision making to improve patient outcomes.

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30 SEP - 3 OCT 2026

Abstract N°: 2164

Age-Stratified Analysis of Adverse Events Associated with Oral JAK Inhibitor Use: A Multicenter Cohort Study

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Introduction
Oral Janus kinase (JAK) inhibitors (JAKi) are widely used for indications in skin and rheumatologic disease but are associated with a spectrum of adverse events (AEs), ranging from serious outcomes such as major adverse cardiovascular events (MACE), venous thromboembolism (VTE), and malignancy, to mild AEs, such as acne, GI upset and skin infection to name a few. Age-related stratification of these adverse events has been incompletely characterized and warrants further investigation. The purpose of this study is to assess these AEs in multiple JAKi across multiple inflammatory and autoimmune indications to determine age-specific risk at more granular age strata.

Materials and Methods
We conducted a retrospective cohort study using the TriNetX Global Collaborative Network evaluating rates of known JAKi AEs among adult patients with atopic dermatitis, alopecia areata, psoriatic arthritis, Crohn's disease, ulcerative colitis, ankylosing spondylitis and rheumatoid arthritis. Patients were stratified into age groups (18–39, 40–64, ≥65 years) with further analysis done on subgrouping of older adults (65–74, 75–84, ≥85 years). Propensity score matching was performed to account for demographic differences, sources of immunosuppression, and Jaki indication matching. Cox hazard ratios were calculated for multiple AEs at 1-, 3-, and 5-year time points.

Results
MACE risk increased stepwise with age, with lower risk in young versus middle-aged (5-year HR 0.51 [0.41–0.64]) and older adults (0.20 [0.16–0.26]), and in middle-aged versus older adults (0.48 [0.44–0.53]; all $p<0.001$). VTE risk was higher in older adults compared to young (0.53 [0.35–0.80], $p=0.002$) and middle-aged adults (0.54 [0.46–0.64], $p<0.001$), with no difference between younger groups. Skin malignancy risk was lower in middle-aged versus older adults (0.39 [0.32–0.47], $p<0.001$).

Infectious AEs differed by age with more frequent SSTIs in older versus middle-aged adults at 5 years (0.86 [0.75–0.95], $p=0.003$) and less frequent herpetic eruptions in young versus middle-aged (0.78 [0.66–0.92]) and older adults (0.72 [0.58–0.89]; both $p=0.003$). URI was not significantly associated with age.

Acne was more common in young adults compared to middle-aged (1.38 [1.24–1.53]) and older adults (1.60 [1.39–1.86]; $p<0.001$), and GI upset was more frequent in young versus older adults (1.41 [1.20–1.65], $p<0.001$). Hyperlipidemia (0.76–0.77) and LFT elevations (0.80 [0.69–0.93], $p=0.003$) were less frequent in young versus older adults.

Conclusions

This large, population-level study highlights differences in both AE profile and risk across multiple indications and age categories, providing more granular insights into sequelae of medications commonly prescribed in dermatologic, rheumatologic, and gastrointestinal settings. Our findings corroborate that advanced age is a driver of serious AEs associated with JAKi, while younger patients experience milder inflammatory and mucocutaneous side effects. This study is limited by reliance on electronic health data; surveillance bias, as older patients are likely to undergo more frequent medical monitoring; and inability to capture dosing related changes in AEs. Despite this, this safety profile assessment of JAKi by age and event type indicates the importance of age-specific and outcome-specific counseling in clinical use.

EADV CONGRESS
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Abstract N°: 2173

A Randomized, Double-blind Phase III study to compare the Efficacy, Safety, Pharmacokinetics, Pharmacodynamics, and Immunogenicity of BP11 (Omalizumab biosimilar) with Reference Omalizumab in Patients with Chronic Spontaneous Urticaria Who Are Resistant to H1 Antagonist

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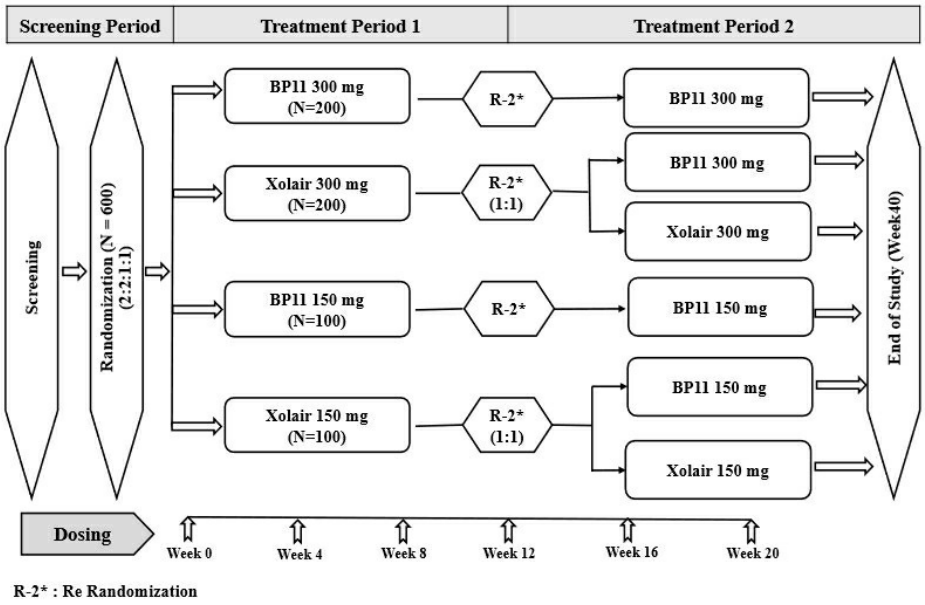
Introduction

Chronic spontaneous urticaria (CSU) is a debilitating inflammatory skin disorder characterized by the recurrent occurrence of erythematous, edematous, intensely pruritic, and occasionally painful wheals persisting for at least 6 weeks, with no identifiable external trigger and frequently driven by autoimmune mechanisms. BP11 has been developed as a biosimilar to omalizumab, a humanized monoclonal antibody targeting immunoglobulin E (IgE). By inhibiting IgE-mediated activation of mast cells and basophils, omalizumab provides effective disease control in IgE-mediated conditions, including allergic asthma and CSU. The objective of this study was to evaluate and compare efficacy, safety, pharmacokinetics, pharmacodynamics, and immunogenicity of BP11 with reference omalizumab in patients with CSU inadequately controlled with H1-antihistamines.

Materials and Methods

A total of 600 patients with CSU resistant to H1AHs were planned to be enrolled in the study consisting of two treatment periods spanning 40 weeks: Treatment Period 1 (12 weeks), followed by Treatment Period 2 (28 weeks) including a 20 weeks safety follow-up period after the last dose at week 20. Eligible patients were centrally randomized 2:2:1:1 to either of the four arms 1) BP11 300 mg (2 X 150 mg), 2) reference omalizumab 300 mg (2 X 150 mg), 3) BP11 150 mg (1 X 150 mg) or 4) reference omalizumab 150 mg (1 X 150 mg) following dose administration at baseline (Week 0), Week 4, and Week 8 during TP-1. Stratification factors included ISS7 score (<13 vs ≥13), geographic region (Europe vs India), and body weight (<80 kg vs ≥80 kg).

Prior to week 12 dose, patients assigned to reference omalizumab in TP-1 were re-randomized 1:1 to continue reference omalizumab or switch to BP11 for TP-2, while those initially assigned to BP11 continued BP11. All the randomized patients were evaluated for efficacy, safety, PK, PD, and immunogenicity. The primary endpoint was the change in the ISS7 score from baseline at week 12. Here we are presenting the results of TP-1 including the primary end point analysis.



BP11-301 Study Design

Results

A total of 609 patients were randomized across 80 sites in Europe and India and assigned to the four treatment arms; 578 patients completed TP-1 through week 12. Demographics and baseline characteristics were well balanced across all treatment arms, with approximately 70% of the enrolled patients being white. In the Full Analysis Set (FAS), the mean change in ISS7 score from baseline to week 12 was -11.53 in BP11 300 mg vs -11.94 in reference arm. The resulting mean difference was 0.41 with 95% CI of (-0.72, 1.54) which fell completely within the predefined equivalence margin (EM) of -2.5 to 2.0 demonstrating clinical equivalence. These findings were supported by the per-protocol analysis, with the 95% confidence interval also contained within the equivalence margin. The other efficacy endpoints, PK (omalizumab C_{trough}), PD (total and free IgE), TEAEs, along with anti-omalizumab antibodies were comparable between respective dose level treatment arms.

Conclusions

This phase 3 study in patients with chronic spontaneous urticaria inadequately controlled with H1-antihistamines demonstrates that BP11 is biosimilar to reference omalizumab with respect to efficacy, safety, pharmacokinetics, pharmacodynamics, and immunogenicity.

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Abstract N°: 2206

Patient-Level Persistence of On-Treatment Remission With Guselkumab: Pooled Results From VOYAGE 1 and VOYAGE 2 Participants With Moderate-to-Severe Plaque Psoriasis

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Introduction

Considering the chronic nature of plaque psoriasis (PsO), sustained clinical response leading to durable disease control is an important therapeutic goal. Guselkumab demonstrated high and durable population-level clinical response rates in the phase 3 PsO VOYAGE 1 & 2 studies.[1,2] Here, post-hoc analyses of pooled VOYAGE 1 & 2 data evaluated achievement and patient-level persistence of on-treatment PsO remission as recently defined by the National Psoriasis Foundation.[3]

Materials and Methods

Data from guselkumab-randomized (100 mg at week [W]0, W4, then every 8W [Q8W]) participants with moderate-to-severe PsO were analyzed. On-treatment PsO remission was defined as Investigator's Global Assessment (IGA)=0 for ≥6 months (i.e., ≥4 consecutive Q8W dosing visits) with continuous guselkumab use.[3] Achievement of on-treatment remission and re-achievement after initial loss of response were determined. Time to achievement and durability of on-treatment remission through W252 were assessed using Kaplan-Meier survival analysis.

Results

Among 643 guselkumab-randomized participants, 394 (61%) achieved on-treatment PsO remission through W252, with a median (95% confidence interval [CI]) time to on-treatment remission of 56.0 (40.0-64.0) weeks. Among these responders, 244 of 394 (62%) lost their initial on-treatment remission status, with a median (95% CI) time to loss of response of 104.0 (88.0-128.0) weeks; of these, 156 of 244 (64%) subsequently regained on-treatment remission during follow-up, suggesting that initial loss of response in the majority was transient. Consistent results were seen when defining on-treatment PsO remission employing PASI 100 response and

frequency of assessments mirroring real-world practice.

Conclusions

Among participants with moderate-to-severe PsO, guselkumab provided highly durable on-treatment PsO remission through 5 years.

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EA DV CONGRESS
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Abstract N°: 2216

Guselkumab Re-Treatment After Withdrawal: Rapid Regain of Disease Control in Super-Responders with Moderate-to-Severe Plaque Psoriasis - Final Week 220 Phase 3b GUIDE Findings

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Introduction
Part 3 of the GUIDE study evaluated maintenance of disease control following withdrawal from guselkumab (GUS) among adults with moderate-to-severe psoriasis.[1] Psoriasis Area and Severity Index (PASI) outcomes after GUS re-treatment among super-responders who lost disease control through Week 220 are reported here.

Materials and Methods
In GUIDE part 3, 273 super-responders (PASI=0 at Week 20+Week 28) with disease control maintenance at Week 68 (PASI <3) were withdrawn from GUS and retreated with three doses of GUS, one each at Week 0/Week 8/Week 16 (R0/R8/R16) upon loss of response (PASI >5).[2] PASI outcomes were assessed overall and in subgroups, applying non-responder imputation.

Results
Through Week 220, 227 super-responders were retreated with GUS following a median therapy-free period of 273 days; 69.2% were male, 45.8% had disease duration ≤2 years, and 91.6% were biologic-naïve, with mean baseline age of 39.5 years, body mass index of 27.1 kg/m^2, and PASI of 18.8. Following retreatment, 77.1% regained disease control (PASI<3) at R8, with increased responses at R16/R24 (86.8%/92.5%). PASI=0 response also increased over time (R8/R16/R24: 19.8%/45.8%/59.9%); mean PASI scores decreased from 7.4 at R0 to 0.5 at R24. No significant differences in R24 PASI <3 responses were observed across subgroups (GUS dosing interval in part 2 [every 8 weeks/every 16 weeks], psoriasis duration [≤2 years/>2 years], or weight [≤90 kg / >90 kg]).

Conclusions
Through 3 years post-GUS withdrawal, super-responders remained treatment-free for an extended period. The majority of super-responders who lost response, regained disease control within approximately 8 weeks after one GUS dose, with similar outcomes across subgroups. These findings support the durability of GUS response and effectiveness of retreatment, demonstrating long-term disease control and sustained reduction in disease burden.

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Abstract N°: 2218

Papulopustular and Rosacea-like Eruptions During BRAF/MEK and EGFR Inhibitor Therapy: A Case Series

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Introduction

Papulopustular, acneiform, and rosacea-like eruptions are recognized cutaneous adverse events of targeted anticancer therapies, including EGFR inhibitors and, less commonly, BRAF/MEK inhibitors.

Case Presentation

We report three cases of targeted therapy-associated papulopustular or acneiform eruptions. The first patient was a 69-year-old woman with stage IV metastatic melanoma treated with vemurafenib and cobimetinib. Approximately one month after treatment initiation, she developed a diffuse papulopustular rosacea-like eruption involving the face, clinically characterized by inflammatory erythematous papules and pustules on a background of centrofacial erythema, without comedones. The eruption was persistent and only partially responsive to treatment. She received two cycles of azithromycin 500 mg daily for three consecutive days, together with topical erythromycin. Due to incomplete response, oral doxycycline and topical ivermectin were subsequently introduced, leading only to partial benefit. Vemurafenib and cobimetinib were later discontinued, and oncologic therapy was switched to dabrafenib and trametinib. However, recurrence of the eruption was observed after the therapeutic switch. The second patient was a 39-year-old woman receiving vemurafenib and cobimetinib for stage IIIA melanoma. Approximately one month after starting therapy, she developed a facial rosacea-like papulopustular dermatitis, consisting of erythematous inflammatory papules and pustules without a comedonal component. Oral doxycycline 100 mg twice daily was prescribed, with clinical improvement and better control of the inflammatory lesions. The third patient was a 66-year-old man treated with FOLFIRI chemotherapy, including folinic acid, 5-fluorouracil, and irinotecan, combined with the EGFR inhibitor panitumumab for hepatic recurrence of sigmoid-rectal adenocarcinoma. Approximately six weeks after treatment initiation, he developed an acneiform eruption involving the beard area, neckline, upper chest, and trunk. The eruption consisted of inflammatory follicular papules and pustules distributed in seborrheic and follicle-rich areas, consistent with an EGFR inhibitor-associated acneiform eruption. Treatment with ketoconazole washes and topical clindamycin twice daily resulted in partial clinical improvement.

Conclusion

This case series highlights the clinical overlap between EGFR inhibitor-associated acneiform eruptions and BRAF/MEK inhibitor-associated papulopustular or rosacea-like reactions. In all patients, the eruptions developed early after treatment initiation and were characterized by inflammatory papules and pustules without true comedones, supporting their distinction from conventional acne. Although the clinical phenotype may be similar, the underlying mechanisms differ according to the therapeutic target. EGFR inhibition impairs keratinocyte proliferation, follicular differentiation, and epidermal barrier repair, thereby promoting follicular inflammation. In patients receiving BRAF and MEK inhibitors, interference with MAPK signaling may similarly alter follicular and epidermal homeostasis. The recurrence observed in our first patient after switching from vemurafenib/cobimetinib to dabrafenib/trametinib further suggests a class-related susceptibility to BRAF/MEK pathway inhibition rather

than a reaction to a single agent. Management remains challenging and should be individualized according to severity, distribution, symptoms, and oncologic priorities. Oral tetracyclines, topical antibiotics, ivermectin, antiseptic or antifungal washes, and anti-inflammatory topical treatments may be considered, although responses may be incomplete. In refractory or severe cases, low-dose isotretinoin may represent a potential option after multidisciplinary evaluation. Dermatologists involved in cancer care should be familiar with these patterns, as timely diagnosis and proactive management may reduce symptoms, improve quality of life, and support continuity of anticancer treatment.

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Abstract N°: 2221

Real-World Evidence That As-Needed Dosing withBimekizumab in Patients with Psoriasis Is Safe and Effectiveover Time

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Introduction
Despite substantial progress in the management of psoriasis, evidence on as-needed dosing strategies for biologic therapies remains scarce. In this context, the present study aimed to assess the effectiveness of a previously defined as needed bimekizumab (BKZ) dosing regimen in a larger cohort of patients with psoriasis, as well as to identify clinical factors associated with treatment response.

Materials and Methods
In this retrospective study, medical records of 64 patients with moderate-to-severe psoriasis treated with BKZ between May 2023 and November 2025 at a specialized psoriasis unit in Madrid, Spain, were reviewed. Patients followed an off-label, as-needed dosing regimen, consisting of two initial 320 mg doses at Weeks 0 and 4, with subsequent administrations only upon loss of a PASI 90 response. The primary outcome was the proportion of patients achieving and maintaining optimal disease control (PASI 90) over time. The duration of treatment effect was defined as the interval between the second dose and loss of PASI90 in the absence of further treatment. Concomitant topical therapies were not permitted during the study period. No patients deviated from the as-needed dosing strategy, although minor variations inherent to routine clinical practice may have occurred Safety outcomes were also evaluated.

Results
A total of 59 out of 64 patients achieved a PASI 90 response after the initial two BKZ doses, and all maintained disease control with the as-needed dosing strategy over time. Moreover (41 of 64 patients), 64% maintained a PASI ≤1 and 52 of 64 patients (81%) PASI ≤2 throughout the follow up. Over the study period, patients received on average approximately one-third of the doses that would have been expected if on-label dosing had been followed (3.46 ± 1.68 vs. 10.14 ± 3.80 doses). Figure 1 shows the number of BKZ doses received (blue), versus the doses that each patient would have received if the on-label dosing of BKZ had been followed (grey). The mean duration of treatment effect following the second dose was approximately 24 weeks. Systemic and bio-naïve patients presented the longest treatment effect duration under the as-needed dosing regimen. Oral candidiasis was reported in two patients and resolved without complications.

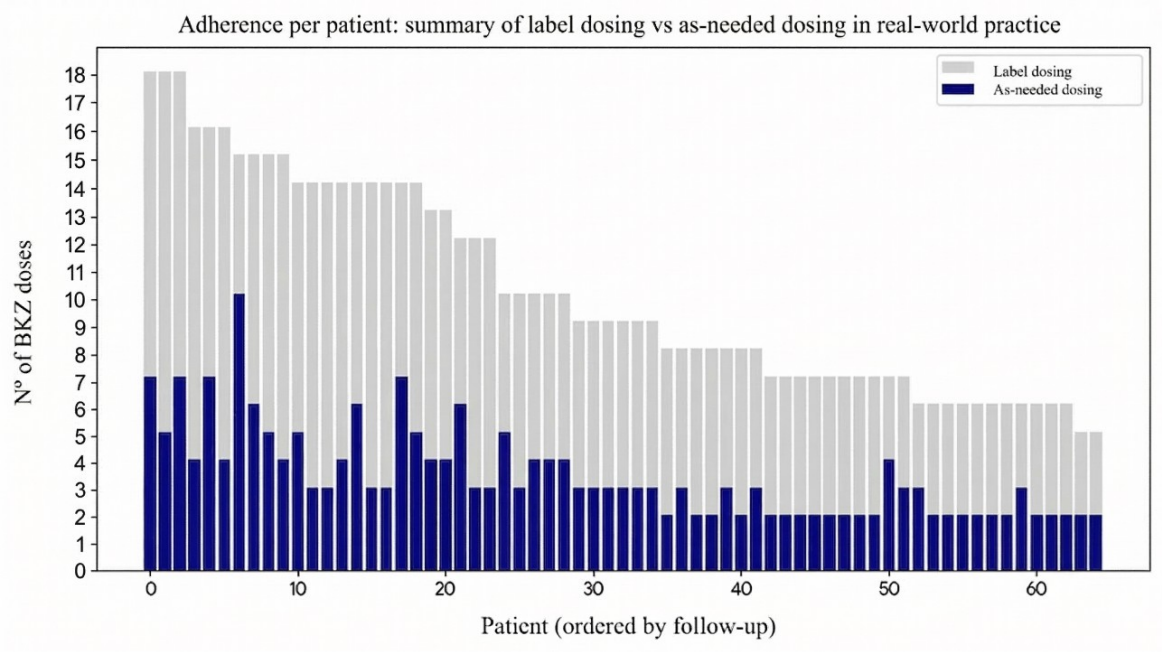

Figure 1. As-needed dosing of moderate-to-severe psoriasis patients with bimekizumab. Legend: BKZ: Bimekizumab. Blue bars: the number of BKZ doses each patient received. In grey: the number of BKZ doses they received if the standard label had been followed.

Conclusions

This study reinforces previous evidence supporting the effectiveness and safety of an as-needed BKZ dosing strategy in patients with psoriasis over time, with prior exposure to systemic or biologic therapy identified as the most important determinant of treatment effect duration. Therapeutic dose-reduction strategies such as the one described here can optimize risk-benefit considerations in in-dividual patients as well as reduce healthcare cares associated with long-term management of psoriasis.

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Abstract N°: 2222

Treatment of Plaque Psoriasis Involving High-Impact Sites With Icotrokinra, a Targeted Oral Peptide: Pooled Analyses of Four Phase 3 Placebo-Controlled Trials

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Introduction

Icotrokinra (ICO), first and only targeted oral peptide that precisely blocks the Interleukin-23 (IL-23) receptor and inhibits IL-23-pathway signaling, is being evaluated in moderate-to-severe plaque psoriasis (ICONIC-LEAD [NCT06095115], ICONIC-ADVANCE-1 [NCT06143878], and ICONIC-ADVANCE-2 [NCT06220604]) and plaque psoriasis involving difficult-to-treat, high-impact sites (ICONIC-TOTAL [NCT06095102]). ICO demonstrated significantly higher Week (W)16 skin clearance rates, including in the scalp and genital areas in ICONIC-TOTAL, and a similar adverse event profile vs placebo (PBO).[1-3] Pooled analyses were conducted across these four phase 3 trials (ICONIC-LEAD, ICONIC-ADVANCE-1, ICONIC-ADVANCE-2, and ICONIC-TOTAL) to evaluate the effect of ICO on difficult-to-treat, high-impact site (scalp, genitals, hands/feet) psoriasis.

Materials and Methods

Participants (pts) were randomized 2:1 (ICONIC-LEAD, -TOTAL, -ADVANCE-1) or 4:1 (ICONIC-ADVANCE-2) to once-daily oral ICO 200 mg or PBO through W16. Assessments among pts evaluable for efficacy included scalp-specific Investigator's Global Assessment (ss-IGA), Static Physician's Global Assessment of Genitalia (sPGA-G), and PGA of hands and feet (hf-PGA) in pts with at least moderate severity of high-impact site psoriasis (score ≥3) through W16.

Results

Among 1,863 pooled ICONIC-LEAD, -TOTAL, and ADVANCE-1 & 2 pts evaluable for efficacy, 1,340 (72%), 473 (25%), and 440 (24%) had a score of≥3 on ss-IGA, sPGA-G, and hf-PGA, respectively, and were included in applicable subgroup analyses. At W16, ICO demonstrated substantially higher rates vs PBO for achievement of clear/almost clear scalp (ss-IGA0/1: 72% vs 16%), genital (sPGA-G0/1: 76% vs 29%), and hand/foot (hf-PGA0/1: 64% vs 23%) psoriasis.

Conclusions

In these large cohorts of pts with at least moderate severity high-impact site psoriasis, ICO demonstrated high rates of clearance at W16, with >70% and >60% achieving clear/almost clear scalp/genital psoriasis and hand/foot psoriasis, respectively.

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EA DV CONGRESS
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Abstract N°: 2224

Adult Langerhans Cell Histiocytosis Presenting as Anogenital Ulceration: A Rare Case with Response to Tofacitinib

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Introduction

Langerhans cell histiocytosis (LCH) is a rare clonal proliferative disorder of CD1a-positive dendritic cells that may involve single or multiple organ systems. Although it is predominantly a pediatric disease, adult-onset LCH is increasingly recognized and often poses a diagnostic challenge because of its protean clinical manifestations. Cutaneous disease may be isolated or may represent part of multisystem involvement, and anogenital lesions are particularly uncommon in adults.

Case Presentation

A 35-year-old male presented with multiple painful ulcerated lesions over the perineum, perianal region, penis, and right inguinal fold, associated with difficulty in defecation. On examination, the lesions were well-demarcated, erythematous, and ulcerated. The remainder of the physical examination was unremarkable. Skin biopsy demonstrated a dense dermal inflammatory infiltrate composed of lymphohistiocytic cells admixed with neutrophils and occasional histiocytes showing nuclear grooves. Immunohistochemistry showed increased CD1a positivity, confirming the diagnosis of LCH. A comprehensive systemic evaluation, including radiological, hematological, and serological assessment, excluded multisystem disease.

The patient was initially treated with oral prednisolone at 1 mg/kg/day, which resulted in partial improvement followed by relapse. He was subsequently started on tofacitinib 5 mg twice daily, with complete healing of the lesions within 3 months. This response highlights the potential utility of Janus kinase inhibition in refractory cutaneous LCH.



Clinical image showing well-demarcated inguinal ulcer.

Conclusion

Adult cutaneous LCH can mimic several inflammatory, infectious, and neoplastic dermatoses, including Crohn’s disease, pyoderma gangrenosum, cutaneous tuberculosis, extramammary Paget’s disease, Behçet’s disease, and atypical herpes infection. Recognition of perianal and anogenital ulcerations as a possible presentation of LCH is essential, as early biopsy and systemic staging can facilitate timely diagnosis and organ-preserving treatment. This case underscores the importance of considering LCH in the differential diagnosis of chronic anogenital ulcers and suggests that tofacitinib may be a promising therapeutic option in selected refractory cases.



Clinical image showing healing of ulcer after 3 months of tofacitinib therapy.

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Abstract N°: 2225

Tree trunk-like acneiform eruption induced by EGFR inhibitor treatment

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Introduction

Epidermal growth factor receptor (EGFR) inhibitors are widely used in oncology for the treatment of lung, head and neck, and colorectal cancers, either as monotherapy or in combination therapy. Acneiform eruption, a pruritic papulopustular rash, occurs in 50-100% of treated patients and represents the most common adverse effect of this drug class. Although not a life-threatening side effect, it can significantly affect quality of life and in some cases lead to dose reduction or discontinuation of treatment. Management guidelines and prophylactic regimens exist in order to support patients in maintaining their EGFR inhibitor treatment, which is critical for their oncological outcome. Complications arising from non-compliance with prophylactic regimens in patients receiving EGFR inhibitor treatment remain an underreported challenge in clinical practice.

Case Presentation

A 73-year-old male was treated with capecitabine and panitumumab for colorectal carcinoma with hepatic metastases when a progressive acneiform eruption developed shortly after the initiation of treatment. He presented with hyperkeratotic crusted plaques on the forearms, scalp and beard area resembling tree trunk bark, and crusted papules on the trunk and lower extremities with gradual worsening over several weeks. Medical history was significant for hepatitis B infection and other comorbidities, with concurrent medication including entecavir, metoprolol, rivaroxaban, diltiazem and irbesartan/hydrochlorothiazide. Although prophylactic treatment with doxycycline 100mg twice daily and topical corticosteroid cream had been previously prescribed, the patient was non-compliant, taking only two tablets per week. As a result, secondary Staphylococcus aureus superinfection was confirmed by skin culture and treatment was initiated with topical vaseline-salicylic acid 5%, urea 6%-clobetasol propionate, and oral amoxicillin-clavulanate 1 g twice daily. Significant clinical improvement was noted at day 3 and day 10, amoxicillin-clavulanate was discontinued and the patient was discharged. At discharge, he was referred to his treating oncologist to resume anti-EGFR therapy with concurrent doxycycline 100 mg twice daily.

Conclusion

This case highlights the need for surveillance in patients receiving both prophylactic doxycycline and EGFR inhibitor therapy, as non-compliance with the prophylactic treatment caused secondary staphylococcal superinfection requiring management with systemic antibiotic treatment. In other cases, non-compliance can also lead to postponing or interrupting treatment with EGFR inhibitors, worsening the oncological status of the patient. Therefore, proactive patient education, close dermatological follow-up and structured interdisciplinary management algorithms should be established to support dermatology-oncology collaboration in maintaining oncological treatment and improving

patient health status.

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Abstract N°: 2227

Patient preferences for oral versus injectable therapies in psoriasis and atopic dermatitis: the impact of prior injection experience

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Introduction
Patient preference plays an important role in the selection of systemic therapies for chronic inflammatory skin diseases. In psoriasis, injectable therapies have traditionally shown higher efficacy compared to oral treatments. However, this paradigm may change with the emergence of novel highly effective oral therapies. Real-world data on patient preferences remain limited.
Objective of this study was to evaluate patient preferences for oral versus injectable systemic therapies and to assess the impact of prior injection experience in a real-world cohort.

Materials and Methods
A prospective, anonymous questionnaire-based study was conducted in patients with inflammatory skin diseases. A total of 400 questionnaires were collected, and 365 patients were included in the final analysis. Demographic data, disease characteristics, current and previous treatments, and preferences were recorded. Patients were asked about their general preference (oral vs. injectable vs. no preference) and evaluated under different treatment scenarios. Patients with psoriatic arthritis were analyzed within the psoriatic disease group. Statistical analyses included chi-square testing.

Results
The majority of patients were diagnosed with psoriatic disease (n=243), of whom 48 had concomitant psoriatic arthritis, followed by atopic dermatitis (n=66), while a smaller proportion had other chronic inflammatory skin diseases. Most patients were receiving injectable therapies (69.0%), reflecting a treatment-experienced cohort.

Overall, 194 patients (53.2%) preferred injectable therapies, 73 (20.0%) oral therapies, and 98 (26.8%) reported no preference (figure 1).

Scenario-based analyses demonstrated consistent preferences across treatment settings. When comparing daily oral therapy with an injection administered every three months by a healthcare professional, 55.4% preferred injections, 20.6% oral therapy, and 24.0% reported no preference (n=359). A similar distribution was observed for self-administered injections every 2–4 weeks (60.6%, 18.9%, and 20.6%, respectively; n=350). In both scenarios, preference for injectable therapies was significant ($p<0.001$).

Patients with psoriatic disease showed a significantly higher preference for injectable therapies compared to atopic dermatitis (65.4% vs. 47.0%, $p=0.02$). In contrast, atopic dermatitis patients more frequently preferred oral therapies (27.3% vs. 16.0%) and reported no preference more often (25.8% vs. 18.5%).

Data on prior injection experience were available for 324 patients. Patients with prior injection experience showed a higher preference for injectable therapies compared to injection-naïve patients (67.3% vs. 48.4%), while preference for oral therapies was lower (13.8% vs. 28.9%). The proportion reporting no preference was slightly reduced (18.9% vs. 22.7%), with a significant difference in overall distribution ($p=0.001$). Among injection-naïve

patients with psoriatic disease (n=96), 50 (52.1%) preferred injectable therapies, 26 (27.1%) oral therapies, and 20 (20.8%) reported no preference. Among injection-naïve patients with atopic dermatitis (n=25), 11 (44.0%) preferred injectable therapies, 7 (28.0%) oral therapies, and 7 (28.0%) reported no preference.

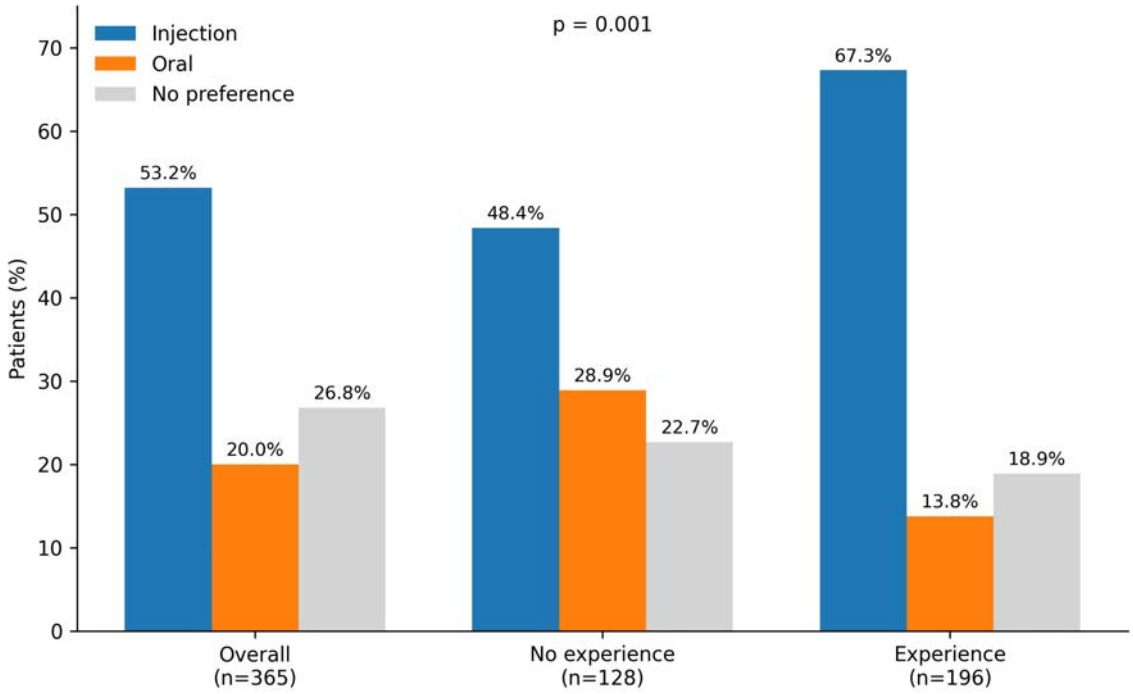

Figure 1: Patient preferences for oral versus injectable therapies overall and stratified by prior experience with self-administered injectable therapies. The overall group includes all patients with available preference data (n=365), whereas subgroup analyses are limited to those with available data on prior injection experience (n=324).

Conclusions

Injectable therapies are preferred over oral treatments in a real-world cohort, particularly in patients with psoriatic disease, and this preference remains consistent across scenarios. Patients with atopic dermatitis show a more balanced distribution of preferences. Prior injection experience is a key determinant of treatment preference. In injection-naïve patients with psoriatic disease, a relevant proportion preferred oral therapies, highlighting the importance of incorporating patient preference into shared decision-making, particularly in the context of emerging highly effective oral treatments.

EADV CONGRESS
VIENNA
30 SEP - 3 OCT 2026

Abstract N°: 2245

Differences in Gut Microbiota and Metabolism Between Rapid and Slow Responders to Omalizumab for Chronic Spontaneous Urticaria

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Introduction
To investigate the differences in gut microbiota between rapid and slow responders to omalizumab in patients with chronic spontaneous urticaria (CSU), and to analyze the potential causes underlying the different response patterns.

Materials and Methods
A total of 28 CSU patients treated with omalizumab in our dermatology department from January 2025 to December 2025 were enrolled. Demographic data, medical history, and laboratory findings were collected. Fecal and serum samples were obtained before omalizumab treatment. Patients were assigned to the rapid-responder group or slow-responder group based on UAS7 scores at week 4. Fecal samples were analyzed by 16S rRNA gene sequencing, and serum samples were analyzed by untargeted metabolomics to compare significant differences in gut microbiota and metabolites between the two groups.

Results
1. 16S rRNA sequencing yielded 2070 valid sequences with unique ASV differences: 875 in the rapid-responder group and 742 in the slow-responder group. Taxonomic analysis showed significantly higher abundance of *Clostridia*, Oscillospirales, and Ruminococcaceae in the slow-responder group ($P<0.05$), while Rikenellaceae, Leuconostocaceae, and *Weissella cibaria*were enriched in the rapid-responder group ($P<0.05$). Functional prediction revealed differences in multiple pathways including sphingolipid metabolism and AMPK signaling.
2. 2. A total of 2676 metabolites were identified by untargeted serum metabolomics, among which 197 differential metabolites were screened (29 up-regulated and 168 down-regulated), significantly enriched in fatty acid metabolism pathways ($P<0.05$).

Conclusions
There were significant differences in the abundance of microbiota including Bacteroidetes, Clostridia, Oscillospirales, and Ruminococcaceae, as well as in the fatty acid metabolism pathway, between rapid and slow responders to omalizumab in CSU patients, suggesting that gut microbiota may regulate the differential response through metabolism.

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30 SEP - 3 OCT 2026

Abstract N°: 2252

The Limits of Anti-TNF Monotherapy in Chronic Refractory Pyoderma Gangrenosum: Secondary Biologic Failure, Adjunctive Dependence, and the Price of Delayed Escalation

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Introduction

Pyoderma gangrenosum (PG) is a chronic, relapsing neutrophilic dermatosis in which delayed diagnosis and prolonged reliance on systemic corticosteroids are common, and long-term disease control often remains elusive. Anti-TNF agents have become the mainstay of treatment in corticosteroid-refractory disease, supported primarily by a single randomised controlled trial (Brooklyn et al., 2006), which assessed outcomes at week 2 and week 6, and supplemented by retrospective case series with median follow-up of 3–12 months. Long-term durability of response and risk of secondary biologic failure remain incompletely defined.

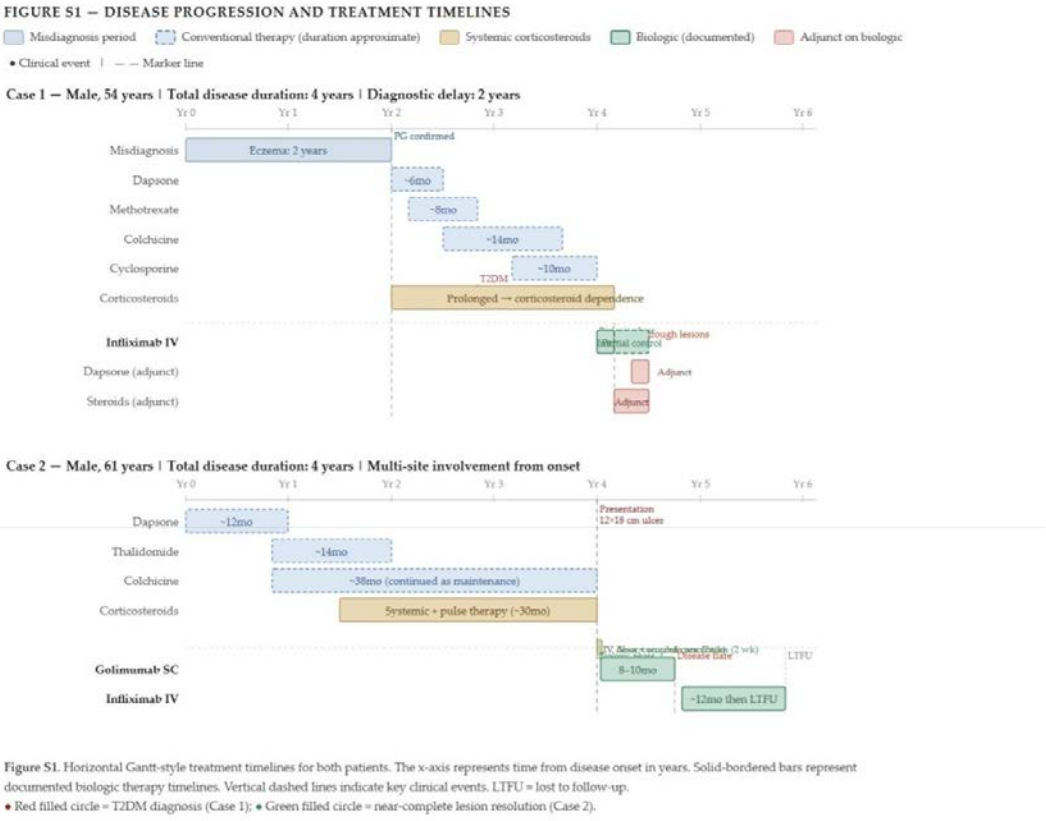

Figure S1. Horizontal Gantt-style treatment timelines for both patients. The x-axis represents time from disease onset in years. Solid-bordered bars represent documented biologic therapy timelines. Vertical dashed lines indicate key clinical events. LTFU = lost to follow-up.
• Red filled circle = T2DM diagnosis (Case 1); • Green filled circle = near-complete lesion resolution (Case 2).

Case Presentation

We report two biopsy-confirmed cases of idiopathic, multi-site, relapsing PG with four-year disease durations and sequential failure of five or more conventional systemic agents prior to biologic initiation.

Case 1: A 54-year-old male with a two-year diagnostic delay — during which PG was managed as eczema — presented with progressive multi-site ulcerative and pustular lesions. He failed dapsone, methotrexate, colchicine,

cyclosporine, and systemic corticosteroids sequentially; prolonged corticosteroid dependence resulted in new-onset type 2 diabetes mellitus. Intravenous infliximab produced initial improvement in ulcer burden, but breakthrough lesions appeared within two months, necessitating concurrent systemic corticosteroids and dapsone. Disease remained only partially controlled on triple-agent therapy.
Case 2: A 61-year-old male with recurrent multi-site PG — chest, thighs, and legs — had previously failed dapsone, thalidomide, colchicine, and pulse corticosteroids. He presented with a 12×18 cm non-healing ulcer of the posterior thigh while on maintenance therapy. Following wound stabilisation with intravenous corticosteroids, subcutaneous golimumab 50 mg four-weekly produced near-complete resolution within two weeks and sustained remission for eight to ten months. Disease flared thereafter; golimumab was discontinued, and intravenous infliximab was initiated, producing a further durable response lasting approximately twelve months before loss to follow-up.

TABLE S1 — TREATMENT RESPONSE SUMMARY: SEQUENTIAL THERAPY IN BOTH CASES

Drug / Intervention	Case 1 — Male, 54 years			Case 2 — Male, 61 years	
	Approx. duration	Response / Outcome	Reason for discontinuation / change	Approx. duration	Response / Outcome
CONVENTIONAL SYSTEMIC AGENTS					
Dapsone	~6 months	Secondary failure Initial partial response; disease progression	Reintroduced later as adjunct alongside infliximab	~12 months	Secondary failure Initial partial response; new lesions on therapy
Thalidomide	— Not used	—	—	~14 months	Secondary failure Initial response; disease relapse
Methotrexate	~8 months	Primary failure No meaningful clinical response	Progressive ulceration on therapy	— Not used	—
Colchicine	~14 months	Secondary failure Initial disease control; breakthrough ulceration	Continued as maintenance but insufficient alone	~38 months (incl. maintenance)	Secondary failure Initial control; continued as maintenance; insufficient on its own
Cyclosporine	~10 months	Secondary failure Initial response; disease recurrence	Recurrent ulceration despite continued use	— Not used	—
Systemic corticosteroids	Prolonged (~24+ months)	Steroid dependence Disease control only with ongoing steroids; unable to taper. Complicated by new-onset type 2 diabetes mellitus	Continued as adjunct throughout biologic therapy	~30 months (incl. pulse therapy)	Steroid dependence Pulse therapy used for acute flares; maintenance dose continued. No diabetes recorded
BIOLOGIC THERAPY					
Golimumab SC (50 mg Q4W)	— Not used	—	—	8–10 months	Partial → secondary failure Near-complete resolution of 12×18 cm ulcer within 2 weeks of initiation. Sustained response 8–10 months. Disease flare thereafter; golimumab discontinued
Infliximab IV (5 mg/kg)	Ongoing (at time of report)	Partial response — breakthrough on therapy Marked reduction in ulcer burden and induration initially. Breakthrough lesions at ~2 months. Partial control only with adjunctive dapsone + corticosteroids. No remission achieved as monotherapy	Dapsone and corticosteroids added as adjuncts. Disease ongoing	~12 months	Sustained remission Clinical response after switching from golimumab. Sustained remission for ~12 months on infliximab therapy; subsequently lost to follow-up

Response classifications: Primary failure = No clinically meaningful response to treatment. Secondary failure = Initial response followed by loss of efficacy. Partial/Breakthrough = Incomplete response requiring adjunctive agents for disease control. Steroid dependence = Inability to taper corticosteroids without disease flare.

Conclusion

Discussion Both cases demonstrate secondary loss of response to anti-TNF therapy in idiopathic pyoderma gangrenosum, a phenomenon well documented in inflammatory bowel disease and psoriasis but remains poorly described in the PG literature. In Case 1, infliximab controlled but never resolved disease, with adjunctive agents required throughout the treatment course. In Case 2, two sequential anti-TNF agents each produced responses lasting eight to twelve months — timeframes that already exceed the follow-up window of most published evidence. The only PG RCT assessed outcomes at two and six weeks. A semi-systematic review of 356 TNF-inhibitor-treated patients, reporting an 87% response rate, explicitly acknowledged the need for trials with "at least six-month follow-up" — a threshold our second case had already passed before losing response to his first

biologic. Adjunctive immunomodulation was required in both of our cases despite active biologic therapy. The morbidity of deferred escalation is also illustrated here: steroid-induced diabetes in Case 1 is a direct consequence of years on corticosteroids in the absence of timely biologic initiation — not an incidental comorbidity.
ConclusionThese cases raise two questions the current literature cannot answer: how long do biologics sustain remission in chronic PG, and can they reliably do so without adjunctive immunomodulation? They also make one point clearly: delayed escalation to biologics in steroid-refractory PG accrues preventable systemic morbidity. Biologics need to be positioned earlier on the PG treatment ladder, combination strategies must be anticipated from the outset rather than added reactively, and long-term follow-up data — presently absent — must become a mandatory outcome in future PG trials

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Abstract N°: 2281

Long-Term Safety of JAK and TYK inhibitors in the treatment of Psoriasis, Psoriatic Arthritis, and Atopic Dermatitis: A Systematic Review

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Introduction

Janus kinase (JAK) inhibitors and tyrosine kinase (TYK) inhibitors are oral, small-molecule immunomodulatory therapies used in the treatment of immune-mediated inflammatory diseases, including psoriasis (PsO), psoriatic arthritis (PsA), and atopic dermatitis (AD). Their use in clinical practice has been limited by safety concerns, with reported adverse events including infections (particularly upper respiratory tract infections and herpes zoster), major adverse cardiovascular events (MACE), venous thromboembolism (VTE), and malignancy, among others. These concerns are particularly relevant given the chronic nature of PsO, PsA, and AD, which require prolonged treatment exposure. This study aimed to systematically review and synthesize reported rates of adverse events, including serious adverse events, associated with JAK and TYK inhibitors in PsO, PsA, and AD.

Materials and Methods

A systematic literature review was conducted in accordance with PRISMA guidelines using PubMed, Embase, and Scopus. The search strategy combined terms related to Janus kinase inhibitors, including upadacitinib, abrocitinib, baricitinib, ruxolitinib, delgocitinib, tofacitinib, and "Janus kinase inhibitor"; TYK inhibitors, including deucravacitinib; adverse event terminology (e.g., adverse events, adverse effects, side effects, and safety); and disease indications (PsO, PsA, and AD). Studies reporting safety outcomes with a minimum follow-up duration of 48–52 weeks in patients with PsO, PsA, or AD were included. Systematic reviews, meta-analyses, and non-English publications were excluded.

Titles and abstracts were initially screened for eligibility, followed by full-text review and data extraction for included studies. Given substantial heterogeneity in study design, patient populations, and follow-up duration, adverse events were summarized descriptively using medians, interquartile ranges (IQR), ranges, and sample size-weighted means.

Results

Initial database searches identified 6,463 studies using JAK or TYK inhibitors in the treatment of PsO, PsA, and AD; these underwent title and abstract screening. Of these, 282 articles were selected for full-text review, and 86 studies met criteria for data extraction. Reasons for exclusion included limited adverse event reporting duration, incomplete reporting of specific adverse events, and lack of access to full-text articles.

Across all included studies evaluating JAK and TYK inhibitors, the median patient age was 42.8 years (range, 1.13-74.11), with a median follow-up duration of 52 weeks (range, 48–260 weeks). The sample size-weighted mean incidence of any adverse event (AE) was 37.48%. Infectious AEs had a mean incidence of 23.56% (IQR, 5.73%-41.75%) across the total study population (n = 40,881). Non-infectious AEs were less common, with mean incidences of 2.90% (IQR, 0.00%-4.79%) for musculoskeletal events, 0.98% (IQR, 0.00%-1.07%) for cardiac events, 0.14% (IQR, 0.00%-0.24%) for VTE, and 1.29% (IQR, 0.00%-2.05%) for neoplasms. Serious adverse events occurred

at a mean incidence of 2.56%, while mortality was reported in 0.15% (IQR, 0.00%-0.21%) of patients. Subgroup analyses stratified by disease state and drug class demonstrated consistent trends across groups. Across studies, median rates of ≥75% improvement in disease severity (PASI75 or EASI75) and near-complete clearance (PASI90) were 67.0% (range, 27.5%-100.0%) and 45.8% (range, 6.7%-94.6%), respectively.

Conclusions

JAK and TYK inhibitors demonstrate favorable efficacy with substantial rates of clinical response across PsO, PsA and AD, accompanied by a low incidence of serious adverse events. These findings support the overall benefit-risk profile of JAK and TYK inhibitors in immune-mediated inflammatory diseases while underscoring the importance of continued long-term safety monitoring in real-world populations.

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Abstract N°: 2289

Impact of Secukinumab on Cardiovascular Disease in Patients with Psoriasis, PsA, and Axial Spondyloarthritis: A Systematic Review of Existing Evidence

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Introduction

Psoriasis, psoriatic arthritis (PsA), and axial spondyloarthritis (axSpA) are associated with increased risk of cardiovascular (CV) disease driven by chronic systemic inflammation, with IL-17A thought to play a central pathogenic role. This inflammation contributes to endothelial dysfunction, an early and pivotal step in the development of atherosclerosis, as well as increased arterial stiffness, an important factor in its progression. Secukinumab, a selective IL-17A inhibitor approved across the three indications, may have potential implications for CV health. This review aimed to evaluate the evidence available in the scientific literature on the impact of secukinumab on CV disease in patients with psoriasis, PsA and axSpA, including its effects on vascular function, inflammatory markers, and long-term safety.

Materials and Methods

A systematic review of PubMed was performed from database inception to May 2025 according to a pre-defined search strategy (**Table 1**). Results were screened and evaluated for inclusion according to the following criteria: publications describing prospective or retrospective studies or meta-analyses; studies including at least 10 patients treated with secukinumab; studies reporting on at least one of the outcomes of interest (endothelial function, arterial stiffness, systemic inflammation, vascular imaging outcomes, and incidence of major adverse CV events [MACE] including CV death, acute myocardial infarction, stroke, unstable angina, and heart failure). Systematic reviews without meta-analysis, case studies, letters to the editor and commentaries were excluded, as were non-English language publications.

Search terms
(secukinumab)
AND
((psoriasis) OR (psoriatic arthritis) OR (ankylosing spondylitis) OR (axial spondyloarthritis))
AND
((cardiovascular disease) OR (cardiovascular death) OR (acute myocardial infarction) OR (stroke) OR (unstable angina) OR (heart failure) OR ("MACE") OR ("major adverse cardiovascular event") OR ("major adverse cardiovascular events") OR (endothelial function) OR (endothelial dysfunction) OR (arterial stiffness) OR (coronary microcirculation) OR (systemic inflammation) OR (oxidative stress) OR (subclinical atherosclerosis) OR (atherosclerotic plaque) OR (CCTA) OR ("coronary CT angiography") OR (fluorodeoxyglucose positron emission tomography computed tomography) OR (FDG PET/CT) OR (flow-mediated dilation) OR (FMD) OR (coronary artery calcium) OR (CAC) OR (GlycA) OR (hsCRP) OR (high-sensitivity C-reactive protein) OR (neutrophil to lymphocyte ratio) OR (NLR))

Table 1. Search terms

Results

From 198 articles initially returned by the search, 46 were eligible for inclusion (**Figure 1**). These comprised 9 meta-analyses or other pooled analyses, 13 randomised controlled trials, 11 retrospective studies or analyses, 9 prospective studies and 4 observational studies. Twenty publications included patients with psoriasis, 5 included patients with PsA, 11 included patients with axSpA, and 10 included multiple indications. Based on data from over twenty thousand patient-years of exposure, secukinumab did not associate with increased MACE in patients with psoriasis, PsA, or axSpA. Additionally, multiple studies reported improvements in systemic inflammatory markers including C-reactive protein, neutrophil to lymphocyte ratio and markers of oxidative stress, with others reporting favorable impact on vascular health including improvement in endothelial function, reduction in arterial stiffness, and improvement in coronary flow reserve.

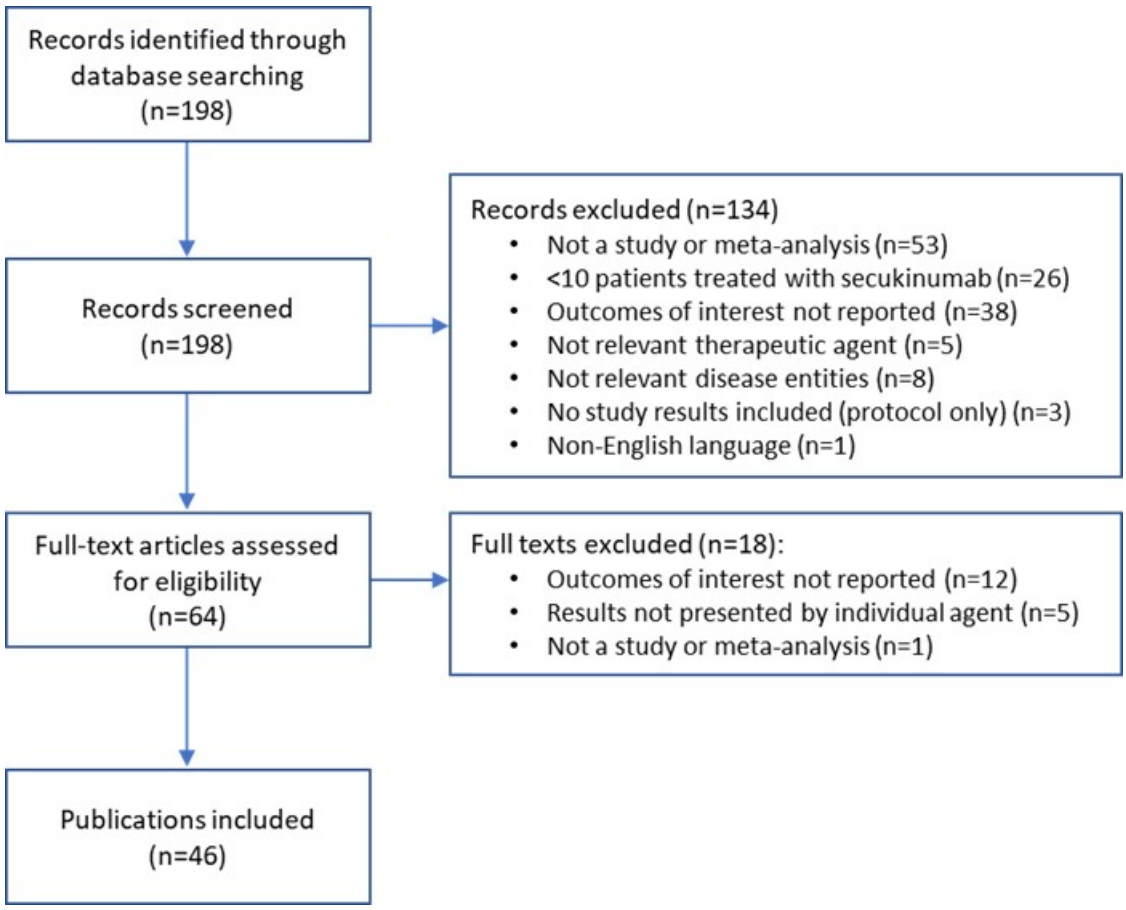

Figure 1. Flow diagram of systematic review process

Conclusions

Secukinumab was associated with a reduction in systemic inflammation and oxidative stress in multiple studies which also reported a consistent CV safety profile across psoriasis, PsA, and axSpA in terms of incidence of MACE. Emerging evidence from vascular imaging suggests that secukinumab may benefit risk factors of CV disease and markers of vascular health; however, larger, prospective randomized trials are needed to confirm this. Finally, these data suggest that secukinumab may provide benefit beyond the primary disease treatment through important reductions in systemic inflammation.

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Abstract N°: 2307

LEO 158968, a Novel Anti-IL-1RAcP Monoclonal Antibody, Demonstrates a Clear PK/PD Relationship and Strong Pharmacological Activity in Patients: Results from a Phase 1 Program

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Introduction

LEO 158968 is a high-affinity monoclonal antibody (mAb) targeting the interleukin (IL)-1 receptor accessory protein (IL-1RAcP), a co-receptor required for signaling of several IL-1 family cytokines, including IL-1α, IL-1β, IL-33, and IL-36γ.

In complex immunoinflammatory diseases with high neutrophil activation driven by IL-1 family cytokines, a single antagonistic agent with the ability to block signaling by several cytokines, such as an anti-IL-1RAcP mAb, may offer a novel approach with considerable translational benefits across multiple therapeutic areas, including dermatology, respiratory diseases, and rheumatology.

Here, we present results from a phase 1 program in healthy volunteers and in patients with acute gout, a prototypic autoinflammatory neutrophil-mediated disease in which IL-1 silencing has been clinically validated, to assess the pharmacological activity of LEO 158968.

In addition, we assessed IL-1RAcP expression in hidradenitis suppurativa (HS) skin compartments.

Materials and Methods

The phase 1 program comprised two parts. In phase 1a, the first-in-human study (NCT05682352), 120 healthy volunteers were randomized to receive intravenous or subcutaneous (SC) single ascending doses or multiple ascending doses (MAD) of LEO 158968. In phase 1b, the open-label, proof-of-mechanism study (NCT06444867), six patients with an acute gout flare received a single 600 mg SC dose of LEO 158968. Efficacy (joint pain), safety, pharmacokinetics (PK), and pharmacodynamics (PD) were assessed.

In a separate study parallel to the phase 1 program, skin biopsies from healthy donors and patients with moderate to severe HS were collected to determine levels of IL-1RAcP and its ligands in this neutrophil-driven skin disease.

Results

In phase 1a, LEO 158968 was well tolerated, with nasopharyngitis being the most common treatment-related adverse event. In MAD cohorts, it demonstrated a linear, dose-dependent increase in PK parameters. Target-mediated drug disposition was observed with clearly visible effect at low concentrations, predominantly

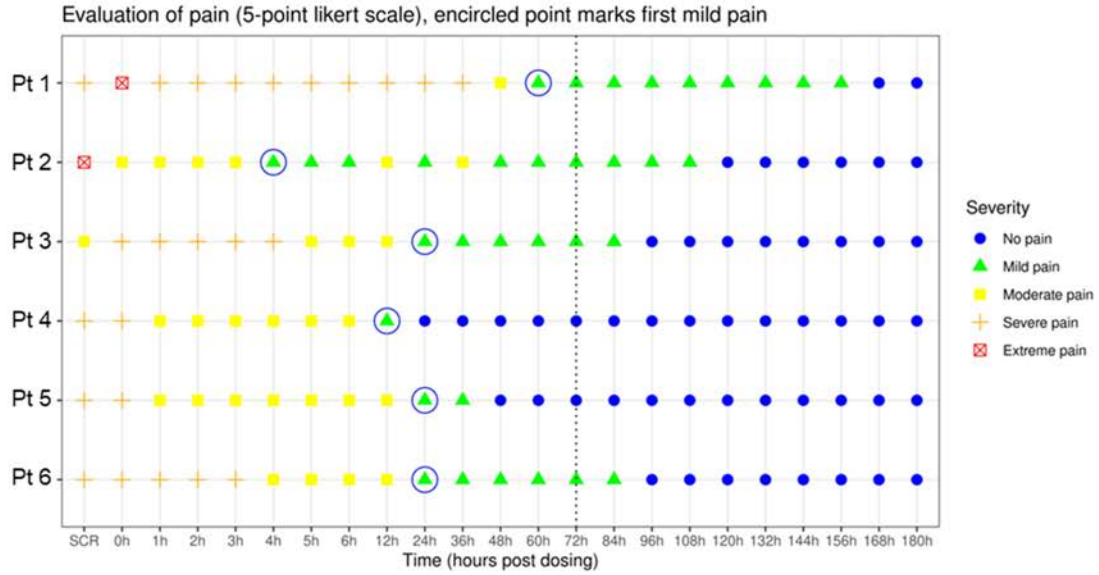
due to binding of free soluble IL-1RAcP (sIL-1RAcP). Consistently, LEO 158968 led to marked reductions in free sIL-1RAcP levels, demonstrating near-complete target engagement (TE). Following dosing, free sIL-1RAcP concentrations decreased rapidly to near-minimum levels, with higher doses prolonging the duration for which these low concentrations were sustained.

In phase 1b, a single 600 mg SC dose of LEO 158968 showed a rapid clinical effect. Most patients (5 of 6) reported mild pain within 24 h, and all patients (6 of 6) experienced a reduction in pain from extreme/severe to no/mild within 72 h post injection, as assessed on a 5-point Likert pain scale (**Fig. 1A**). This clinical effect was associated with a strong PK/PD relationship, characterized by consistent reductions in acute phase reactants, including C-reactive protein, serum amyloid A, IL-6, and neutrophil counts (**Fig. 1B**).

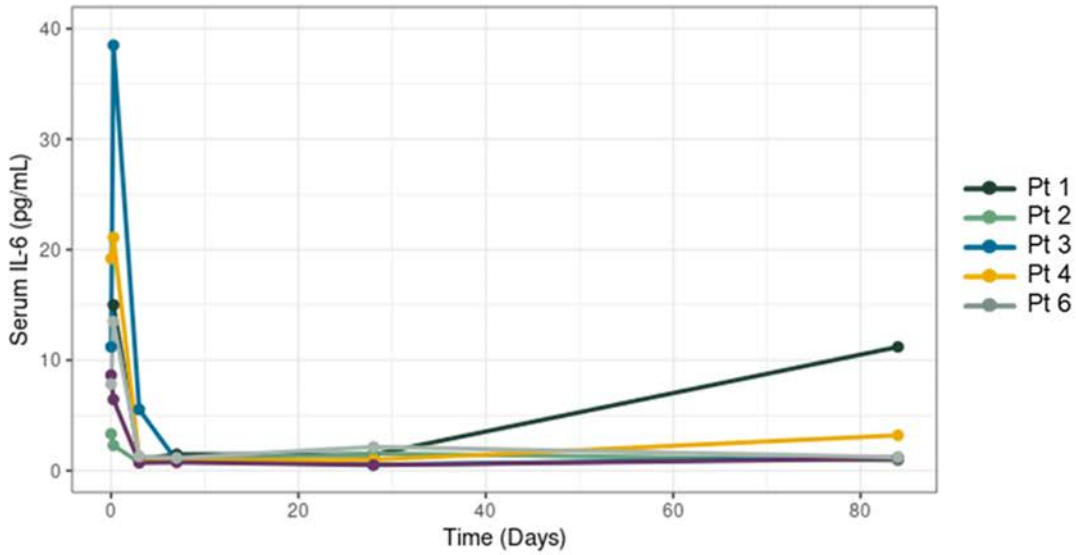
Single-cell transcriptomic profiling showed that, compared with healthy skin, HS lesions, including nodules, abscesses, sinus tracts, and scars, had increased expression and a higher proportion of cells expressing IL-1α and IL-1β in macrophages, neutrophils, keratinocytes, and plasma cells, whereas IL-1RAcP was detected in macrophages, neutrophils, keratinocytes, fibroblasts, endothelial cells, and melanocytes, and IL-36γ in keratinocytes and plasma cells.

Figure 1 Clinical benefit of LEO 158968

A Rapid effect of LEO 158968 on joint pain in patients with acute gout



B IL-6 levels in patients with acute gout after a single 600 mg SC dose of LEO 158968



IL, interleukin; Pt, patient; SC, subcoutanies; SCR, screening

Conclusions

LEO 158968 was safe and well tolerated. It showed consistent PK/PD relationships and TE and induced a rapid clinical benefit in patients with acute gout. The increased expression of IL-1 family members observed in the HS intricate inflammatory architecture suggests a potential therapeutic benefit of targeting IL-1RAcP for complex neutrophil-driven skin diseases. Together, the findings highlight the potential of LEO 158968 to provide rapid relief to patients for whom the current biologics targeting only one of the three IL-1RAcP-associated signaling pathways (i.e., IL-1, IL-33, and IL-36) remain suboptimal.

EA DV CONGRESS
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Abstract N°: 2322

VISIBLE: Guselkumab Impact on Psoriatic Arthritis Through Week 100 in Participants With Moderate-to-Severe Psoriasis and Uncontrolled Psoriatic Arthritis Across All Skin Tones

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Introduction
Underdiagnosis and undertreatment of psoriasis are common among individuals with skin of color (SoC), and scalp psoriasis can be more severe in patients with SoC. In all patients with psoriasis, scalp involvement is a risk factor for development of psoriatic arthritis (PsA). VISIBLE is a Phase 3b study that enrolled participants who self-identified as non-white with moderate-to-severe plaque (Cohort A) or scalp (Cohort B) psoriasis. This post-hoc analysis reports 12-item Psoriatic Arthritis Impact of Disease questionnaire (PsAID-12) results through Week 100 for participants with uncontrolled PsA at baseline.

Materials and Methods
Study participants were randomized 3:1 to receive guselkumab (GUS) 100 mg at Week 0, Week 4, then every 8 weeks (q8w); or placebo with crossover to GUS (placebo→GUS) at Week 16, Week 20, then q8w. Participants with PsA were identified based on a history of rheumatologist-diagnosed PsA or Psoriasis Epidemiology Screening Tool (PEST) score ≥3 at screening. Participants meeting either of these criteria were considered to have uncontrolled PsA at baseline if they had a PsAID-12 score of >4.0 (the PsAID patient-acceptable symptom state [PASS] is defined as a score of ≤4.0). PsAID-12 scores range from 0–10, and minimal clinically important improvement is defined as a reduction of at least 3.0 points.

Results
Among 205 participants (efficacy analysis set) in VISIBLE Cohorts A and B, 22.9% (N=47) had a history of rheumatologist-diagnosed PsA (n=19 [40.4%]) or PEST ≥3 at screening (n=28 [59.6%] with previously undiagnosed PsA) and PsAID-12 score >4.0 at baseline. Prior psoriasis treatments included topicals (76.6%), phototherapy (14.9%), methotrexate (21.3%), apremilast (6.4%), and biologics (any biologic [42.6%]; tumor necrosis factor [21.3%], interleukin [IL-]17 [19.1%], and IL-12/23 [19.1%] inhibitors). Mean baseline data reflect extensive skin disease (Psoriasis Area Severity Index=19.9; body surface area=23.6%) and severe impact of PsA on health (mean PsAID-12 scores of 7.3 and 8.1 in the GUS and placebo groups, respectively). From baseline to Week 16, the GUS-treated group achieved clinically important improvements in mean PsAID-12 score (least-squares mean change, −4.1 vs 0.2 with placebo; nominal $p<0.001$) and a mean score (2.9) below the PASS threshold. Mean PsAID-12 scores continued to improve and were 1.9 in the GUS group and 1.3 in the placebo→GUS group at Week 100 (least squares mean change, −5.5 and −6.9, respectively).

Conclusions
At baseline, approximately one quarter of VISIBLE participants had uncontrolled PsA (based on having a PsAID-12

score above the PASS threshold of 4.0), indicating a need for improved routine PsA screening for patients across all skin tones. Specifically, psoriasis patients with risk factors should be considered for PsA screening with PEST to enable early detection, initiation of appropriate treatment, and improved outcomes. In these GUS-treated participants with uncontrolled and often undiagnosed PsA at baseline, mean PsAID-12 scores decreased below the PASS at Week 16, indicating this group achieved clinically meaningful improvements in the physical and psychological impact of joint disease. Improvements continued and were maintained through 100 weeks of treatment.

EADV CONGRESS
VIENNA
30 SEP - 3 OCT 2026

Abstract N°: 2359

Rapid Facial Repigmentation with Tofacitinib and NB-UVB in Refractory Non-Segmental Vitiligo

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Introduction

Non-segmental vitiligo is a chronic autoimmune disorder driven primarily by the IFN-γ/JAK-STAT pathway. Activated CXCR3+ CD8+ T cells induce melanocyte apoptosis via IFN-γ secretion, establishing a pro-inflammatory feedback loop mediated by the chemokines CXCL9 and CXCL10. While topical therapies and narrowband UVB (NB-UVB) phototherapy remain first-line treatments, refractory cases pose a significant clinical challenge. JAK inhibitors have emerged as a potent alternative, particularly when combined with phototherapy; evidence suggests that light exposure is essential to stimulate melanocyte regeneration once JAK-mediated immunosuppression stabilizes the disease. We report a case of refractory non-segmental vitiligo achieving near-complete facial repigmentation through combined tofacitinib and NB-UVB therapy.

Case Presentation

A 60-year-old male (Fitzpatrick skin type III) presented with a four-year history of non-segmental vitiligo. Lesions initially appeared in the periorificial facial regions, progressing to the nasal and frontal areas, followed by involvement of the acral regions (hands and feet) and the extensor surfaces of the elbows. A relevant comorbidity included stable plaque psoriasis on the elbows and knees, well-controlled with topical corticosteroids for over five years.

After failing to respond to topical tacrolimus 0.1% (face) and clobetasol (psoriatic and vitiligo overlap lesions), the patient began combination therapy with oral tofacitinib (5 mg twice daily) and NB-UVB phototherapy (3 times a week). Notably, the patient was phototherapy-naïve. Initial repigmentation islands were observed within 15 days, primarily on the face. After 10 months, the patient achieved near-complete facial repigmentation and overall disease stabilization with no new lesions. In contrast, acral lesions showed no significant improvement. The patient reported high treatment satisfaction and improved quality of life. Minor adverse events included herpes labialis and genital warts within the first trimester, both of which resolved spontaneously without interrupting therapy.

Conclusion

JAK inhibitors are a transformative therapeutic option for refractory non-segmental vitiligo, especially when synergistic with NB-UVB. This dual-action approach—tofacitinib-led immunosuppression coupled with phototherapy-induced melanocyte stimulation—often yields superior results compared to monotherapy. Our case confirms the rapid, significant response typical of facial lesions, while also illustrating the well-documented resistance of acral areas, likely due to a depleted melanocyte reservoir. Although topical ruxolitinib is currently the only JAK inhibitor FDA-approved for vitiligo, the off-label use of tofacitinib remains a promising strategy. Systematic monitoring for infectious complications remains essential during follow-up.

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JAK1 Inhibition With Abrocitinib in Refractory Prurigo Pigmentosa Following Multiple Vaccinations

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Introduction

Prurigo pigmentosa (PP) is a rare inflammatory dermatosis classically described in young women of East Asian ethnicity, with cases in other populations likely underrecognised due to limited clinical awareness. Occurrence in elderly patients represents a rare demographic presentation. PP has been reported following COVID-19 vaccination; however, to our knowledge, neither a temporal association with multiple vaccinations nor treatment with abrocitinib has been widely reported in prurigo pigmentosa. We present an atypical case of PP in a 76-year-old non-Asian woman with a temporal association with three vaccinations, successfully treated with a selective JAK1 inhibitor (abrocitinib), highlighting an atypical demographic presentation and a potential therapeutic role for abrocitinib.

Case Presentation

A 76-year-old woman presented with a 4-month history of bilateral symmetric erythematous papules coalescing into well-demarcated plaques with fine superficial scaling on the upper limbs, associated with pruritus. She reported no systemic symptoms, no recent topical product exposure, and no changes to her diet or chronic medications. Vaccination against herpes zoster, influenza, and COVID-19 had been administered approximately 6 to 8 weeks before lesion onset, raising the possibility of a temporal immunological trigger.

Incisional skin biopsy demonstrated necrotic keratinocytes in the epidermis, vacuolar degeneration of the basal layer, pigment incontinence, perivascular mononuclear inflammatory infiltrate, and rare eosinophils, findings compatible with prurigo pigmentosa, although initially interpreted as a drug-induced eruption.

The patient had previously been treated in another center with topical and systemic corticosteroids for presumed contact dermatitis without improvement. Subsequent treatment with doxycycline also failed to control disease activity, with persistence of active inflammatory lesions. Given the clinicopathologic suspicion of prurigo pigmentosa, treatment with abrocitinib 200 mg daily was initiated. Follow-up over three weeks showed complete resolution of the active inflammatory lesions, followed by the emergence of characteristic reticulate hyperpigmentation, supporting the diagnosis of prurigo pigmentosa. Complete clearance of inflammatory activity was maintained on subsequent follow-up.

Conclusion

This case broadens the recognised clinical spectrum of prurigo pigmentosa by describing its occurrence in an elderly non-Asian patient with a temporal association with multiple vaccinations. Rapid resolution of inflammatory lesions following **abrocitinib** suggests that selective JAK1 inhibition may represent a therapeutic option in refractory prurigo pigmentosa.

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Abstract N°: 2364

Defining Clinically Meaningful Change in Cutaneous Lupus Erythematosus Disease Area and Severity Index-Activity (CLASI-A) Scores: Data from AMETHYST Part A, a Phase 2 Study of Litifilimab in Cutaneous Lupus Erythematosus

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Introduction

Cutaneous lupus erythematosus (CLE) is a serious chronic autoimmune disease, primarily affecting the skin leading to diverse manifestations. The efficacy and safety of litifilimab, a monoclonal antibody targeting blood dendritic cell antigen 2 on plasmacytoid dendritic cells, is being evaluated in the randomized, double-blind, placebo-controlled, 2-part (A and B), Phase 2/3, AMETHYST (NCT05531565) study in patients with active chronic and/or subacute CLE, with or without systemic manifestations, who are refractory or intolerant to antimalarial therapy. In AMETHYST, the Cutaneous Lupus Erythematosus Disease Area and Severity Index–Activity (CLASI-A) is used to measure treatment response. The objective of this analysis was to estimate the clinically meaningful numeric and percentage improvement in the CLASI-A Total (0-70) score using AMETHYST Part A data.

Materials and Methods

Blinded AMETHYST Part A Week 16 and 24 data were analyzed to estimate meaningful within-patient change (MWPC) for improvement of the CLASI-A Total (0-70) score. Anchor-based analyses were the main approach, with anchors selected from baseline and follow-up scores of Clinician and Patient Global Assessment of Skin (CGA, 4 questions; PGA, 3 questions), focusing on anchors of lupus-related skin activity and erythema. Anchor response options were on a 5-point scale (eg, no signs, almost no signs, mild signs, moderate signs, severe signs) and were scored as change from Baseline. Clinicians and patients were asked whether any change experienced was meaningful. MWPC estimates were derived from mean and median change within each anchor category (1 or 2 category improvement) with 95% confidence intervals, effect sizes, and receiver operating characteristic (ROC) curves (for a 1 or 2+ category anchor change), supported by cumulative distribution function (CDF) and probability density function (PDF) curves and shift tables. This was supplemented by distribution-based estimates of 0.5 standard deviation (SD), standard error of measurement (SEM), and minimal detectable change with 90%

confidence (MDC_{90}).

Results

All anchors correlated ≥0.35 with the CLASI-A Total score; CGA anchors had higher correlations than PGA anchors, and CLASI-A percentage change correlated more strongly with all anchors than numeric change. Results were consistent across all anchor-based approaches and across clinician and patient anchors; MWPC threshold estimates shown in **Table 1.** Across all methods, mean 1-category improvement numeric changes in the CLASI-A Total Score ranged from 6.7 to 9.6 at Week 16 and 6.3 to 8.8 at Week 24 (overall estimate 8.0); mean 1-category percentage changes ranged from 31.7% to 44.1% at Week 16 and 30.8% to 42.8% at Week 24 (overall estimate 40%). The anchor-based ROC gave average values across all anchors of 6.9 and 8.2 at Week 16 and 6.2 and 8.3 at Week 24 (for 1 and 2+ category improvement, respectively) for numeric change (overall estimates 7.0 and 8.0) and 35.7% and 46.9% at Week 16 and 37.0% and 51.9% for percent change (overall estimates 35% and 50%); estimates were slightly lower for perceived meaningful change. Estimates using the distribution-based approach were smaller than estimates from the anchor-based approach, indicating that anchor-based estimates show a change greater than measurement error. The CDF and PDF curves for no change, 1-point and 2-point improvement showed greater differentiation for percentage than numeric change, consistent with the higher correlations with percentage CLASI-A change.

Table 1. Distribution and Anchor-based estimates for the CLASI-A total score by anchor change category.

	Distribution-based estimates			Anchor-based estimate(s)				Final estimates
				MWPC (Within-group)				
Change	0.5 SD	SEM	MDC_{90}	ROC (1 to 2+)	ROC (meaningful)	Mean change (1 category)	Shift by 1 category from Shift tables	MWPC
Numeric	4.32	1.75	4.07	7.0 to 8.0	5.7 to 6.7	8.0	4.5 to 9.1	**7.0 to 8.0**
Percent	22%[a]	9% [a]	20% [a]	35% to 50%	32.8 to 42.9	40%	19.5 to 50.8	**35% to 50%**

[a] Using Baseline CLASI-A Total mean score of 19.88.
MWPC = meaningful within-patient change; MDC_{90} = minimal detectable change with 90% confidence;
ROC = receiver operating curve; SD = standard deviation; SEM = standard error of measurement.

Conclusions

The estimated MWPCs for improvement in the CLASI-A Total score using AMETHYST Part A data were a decrease of 7.0−8.0 for numeric change and 35−50% for percentage change. Larger correlations and greater CDF curve separation suggested that CLASI-A percentage change more precisely reflected clinician and patient perceived change. These findings support the use of CLASI-50 and CLASI-70 as endpoint in CLE clinical trials.

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Characterization of Litifilimab Treatment Response in Patients with Cutaneous Lupus Erythematosus using RNA-seq from Lilac Phase 2 Part B Blood Samples

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Introduction

Cutaneous lupus erythematosus (CLE) is a chronic autoimmune disease driven by multifactor aberrant immune responses, leading to persistent and disfiguring cutaneous lesions. The activation of innate and adaptive immune responses, including TLR sensing pathways in plasmacytoid dendritic cells (pDCs), is central to the pathogenesis of CLE. Prominent expression of blood dendritic cell antigen 2 (BDCA2) has been described on pDCs in the inflammatory infiltrate in CLE. Litifilimab, a monoclonal antibody targeting BDCA2, potently inhibits type I IFN production by attenuating TLR7 and TLR9 sensing pathways in pDCs. In the LILAC Phase 2 Part B trial, litifilimab met the primary endpoint, demonstrating significant dose-dependent reductions in CLE Disease Area and Severity Index-Activity (CLASI-A) versus placebo at Week 16 in adults with CLE with or without systemic manifestations. Despite population level clinical efficacy, a subset of participants did not respond, suggesting underlying biological heterogeneity. Here, we used whole blood from LILAC participants to perform RNA-seq to characterize the impact of litifilimab on type I IFN pathways and other inflammatory processes. We assessed transcriptomic changes associated with treatment response, and evaluated whether baseline molecular endotypes predict differential response to litifilimab.

Materials and Methods

Whole blood collected in PAXgene Blood RNA tubes from LILAC Part B participants (n=83) were demographically matched to healthy volunteers (HV; n=56). After RNA extraction, library preparation, and sequencing, transcriptomic profiles were compared 1) between participants with CLE and HV at baseline to define CLE disease signatures and 2) between litifilimab- and placebo-treated patients at Weeks 12-16 to characterize treatment effects. Differentially expressed genes (DEGs) were identified (FC > 1.2, q < 0.05), and pathway enrichment of DEGs was assessed using Ingenuity Pathway Analysis (FDR < 0.05). Clinical response was defined as ≥50% reduction in CLASI-A. Gene set variation analysis (GSVA) was applied to identify patient molecular profiles followed by K-means clustering to identify molecular endotypes as previously described (Hubbard, EL Genome Medicine 2023, 15:84).

Results

Baseline CLE patient blood transcriptional profiles exhibited >4000 DEGs compared to HV. The observed DEGs and enriched pathways were consistent with prior published profiles from CLE and SLE patient cohorts. Litifilimab treatment had a significant impact on a subset of these DEGs, with a total of 1234 (28%) being modulated towards the baseline state observed in HV (19% down, 9% up; Fig. 1). The litifilimab-downregulated genes were enriched for numerous pathways: type I IFN response, innate immune regulation, response to virus, pattern

recognition receptor signaling, complement cascade activation, macrophage alternative activation signaling, neutrophil degranulation, and macrophage-fibroblast-endothelial cell interaction. Clustering of baseline GSVA profiles partitioned patients into five molecular endotypes. Cluster 5, defined by high GSVA score for IFN, regulatory T cell, T cell, plasma cell, unfolded protein response and low pDC and cDC marker gene sets, was enriched in litifilimab nonresponders (8/9 treated participants).

Conclusions

CLE is characterized by broad dysregulation of gene expression with multiple inflammatory pathways being modulated. In participants with CLE, litifilimab treatment downregulated a broad range of immune genes centered on type I IFN and other inflammatory pathways. A treatment-refractory molecular endotype – characterized by signatures of T and B cell infiltration, regulatory T cell expansion, and reduced circulating pDC markers – was observed. These findings provide a molecular basis for differential response to litifilimab and highlight the possibility that nonresponders may exhibit unique pathobiology defined by gene expression profiles.

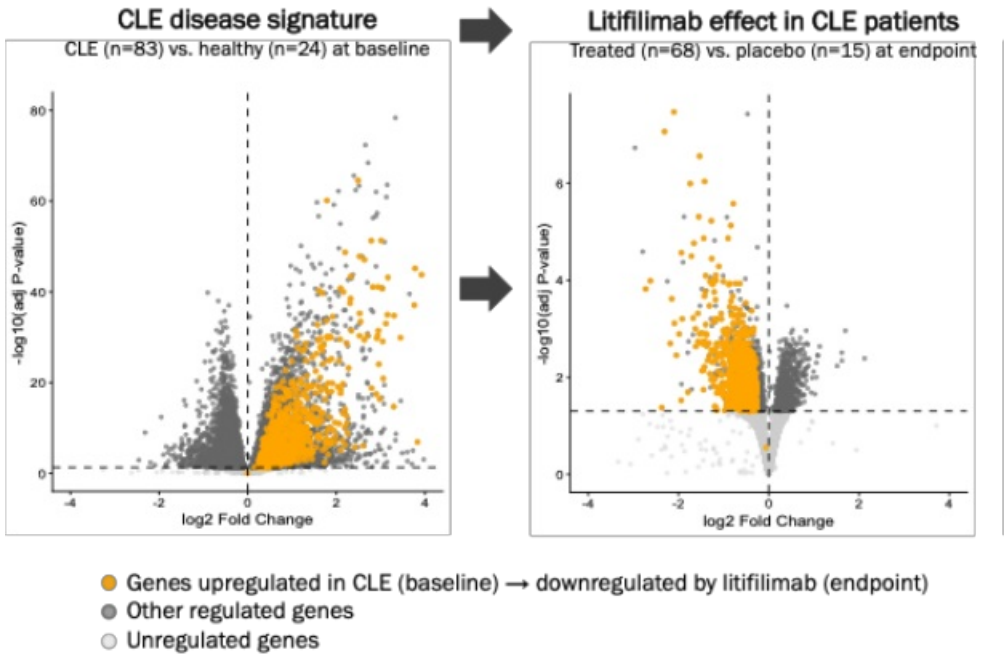

Figure 1. Whole-blood RNA-seq volcano plots depicting the CLE disease signature at baseline and litifilimab-modulated genes at endpoint

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Abstract N°: 2383

Patient thoughts and expectations when embarking on systemic therapy for atopic dermatitis: learnings from a nested interview study within the Best systemic treatments for adults with atopic eczema over the long-term (BEACON) randomised controlled trial.

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Introduction

BEACON is a UK-wide, head-to-head, multi-arm multi-stage platform randomised controlled trial comparing effectiveness, tolerability and cost-effectiveness of ciclosporin, methotrexate, dupilumab and abrocitinib in treating moderate to severe atopic eczema (hereafter 'eczema'). It includes a nested qualitative study to provide context around the quantitative study results. The decision to start a systemic therapy and weighing up the relative merits of the available options can be difficult but life changing for patients. Here we report on disease impact and patient thoughts at that moment in their journey.

Materials and Methods

BEACON participants were invited to a standalone qualitative interview in the early months after they had started taking the trial medication. Interviews explored participants' experience of both disease and treatment, including longer term expectations. Interviews were audio recorded, transcribed in full and analysed thematically.

Results

Forty participants took part in interviews, with ten patients in each treatment arm and roughly equal numbers of males/females. The sample was largely aged 18–35 (n=27, 68%), followed by 36–50 (n=9, 23%), with fewer participants aged 51–80 (n=4, 10%). Most participants were White (n=29, 73%), followed by Asian/Asian British (n=6, 15%), Black/Black British (n=2, 5%), and mixed or multiple ethnic backgrounds (n=3, 8%).

Disease Impact

Consistent with established evidence, participants described how their lives are profoundly disrupted by severe physical discomfort and substantial psychosocial distress. They spoke about unrelenting itching, skin weeping, and painful fissures. This affected their ability to function, caused embarrassment and led to low self-esteem. They described becoming housebound or bedbound and, sometimes the isolation and persistent symptoms led to suicidal thoughts.

General perceptions of systemic treatment

Participants often felt they had no option than to try systemic medications. Some expressed no medication preference, usually associated with a sense of desperation. However, others did have preferences (based on

perceived mechanisms of action, side effects and lifestyle impact) which often reflected incomplete or fragmented reasoning (via online research, or from clinicians themselves). Some participants were initially worried about injections but overall, with support, most participants said it wasn't a problem.

There was uncertainty around long term use, participants did not know whether they would have to use the systemic medication forever or whether it they would gain control of the eczema during the trial and could therefore stop the medication at the end of the 12 months.

Participants were unsure around practical issues such as such safe levels of alcohol consumption, and issues around pregnancy (some worried that methotrexate causes infertility).

Specific medication concerns

A small proportion of patients expressed a strong preference to avoid methotrexate and to a lesser extent ciclosporin. They were aware methotrexate would take longer to work. Some believed that as the newer drugs were restricted on the NHS, this meant they were better in some way.

With regards to abrocitinib, most users were mainly concerned about short term adverse effects such as nausea and tended not to mention the more concerning rarer side effects. They did not make the distinction between routine warnings and the black box warnings. A small number of participants expressed deep concerns about using the medication but felt they needed to take it.

Conclusions

Patients have very different perceptions and expectations of systemic therapies, often drawing on what they know about the terms they hear in their daily lives. Understanding and exploring individual patient narratives, which are not always aligned with those of their clinician, can enable healthcare professionals to discuss any misunderstandings and facilitate effective shared-decision making.

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Abstract N°: 2392

Janus kinase Inhibitors in Dermatology: Emerging Therapies and Clinical Considerations

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Introduction

Janus kinase (JAK) inhibitors represent a promising targeted therapy. Initially developed for systemic inflammatory disorders, they have increasingly demonstrated significant efficacy in several dermatoses such as alopecia areata (AA), atopic dermatitis (AD), and vitiligo, through their immunomodulatory and anti-inflammatory effects.

Materials and Methods

Mechanism of Action: The Janus kinase–signal transducer and activator of transcription (JAK-STAT) pathway is important for cell growth and differentiation. It comprises four types of JAK proteins (JAK1, JAK2, JAK3, and TYK2) and seven varieties of STAT proteins (STAT1, STAT2, STAT3, STAT4, STAT5a, STAT5b, and STAT6). Upon stimulated by extracellular ligands (growth factors and cytokines), the intracellular JAK proteins become activated and phosphorylate STAT proteins. These STAT proteins then dimerize and translocate to the nucleus to directly regulate gene expression. So, inhibiting this pathway results in immunomodulatory, antiproliferative, and anti-inflammatory effects.

Clinical Applications and Safety Considerations: In dermatology, the efficacy of topical and oral JAK inhibitors has been shown in many conditions, such as AD, AA, psoriasis, and vitiligo, with expanding evidence supporting their use in other disorders such as dermatomyositis and morphea. However, common adverse effects have been reported including infections (upper respiratory tract and urinary tract infections), nausea, diarrhea, headache, acne eruption, application-site burning or pruritis for topical agents and laboratory abnormalities (anemia, thrombocytopenia, neutropenia, elevated creatine kinase, and increased levels of both LDL and HDL cholesterol. Serious effects such malignacies, thromboembolic and cardiovascular events may also occur. These findings highlight the importance of carefully assessing the risk–benefit ratio of these molecules and avoiding their use in patients with active cancer, recurrent or severe infections, history or high risk of thromboembolic events, pregnancy or breastfeeding, concomitant immunosuppressive therapies, or severe organ failure. Furthermore, a complete clinical and biological evaluation should be performed before and during treatment (Table 01).

Future Perspectives: Most JAK inhibitors are non-selective, which may explain some of their adverse effects. Therefore, the development of new potent and selective JAK inhibitors represents one of the most promising future directions. Moreover, numerous studies have focused on the efficacy of these treatments in other dermatologic conditions, including bullous dermatoses, cutaneous lupus, cutaneous T-cell lymphoma, allergic contact dermatitis, and lichen planus.

Assessment	Timing	Follow-up
Complete blood count (CBC) with differential, renal and hepatic function tests	Before treatment	1 month after treatment, then every 3–6 months
Lipid panel (Total cholesterol, LDL, HDL, triglycerides)	Before treatment	3 months after initiation
Infection screening: Hepatitis B and Hepatitis C, HIV, and Tuberculosis	Before treatment	Tuberculosis screening annually if ongoing risk factors
Vaccination review (pneumococcal and Shingrix vaccines for appropriate patients)	Before treatment	

Table 01: Laboratory Monitoring Before and During Treatment With JAK Inhibitors

Conclusions

JAK inhibitors represent an emerging therapy. Therefore, dermatologists should become familiar with these molecules and carefully evaluate the risk–benefit ratio before prescribing them.

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Abstract N°: 2420

Immunogenicity of Imsidolimab for the Treatment of Generalized Pustular Psoriasis (GPP)

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Introduction

Generalized pustular psoriasis (GPP) is a rare, severe, and life-threatening inflammatory disease driven by dysregulated interleukin-36 (IL-36) signaling, frequently associated with mutations in *IL36RN*, which encodes the IL-36 receptor antagonist. Imsidolimab is a high-affinity humanized IgG4 monoclonal antibody that binds the IL-36 receptor and inhibits IL-36 signaling.

Materials and Methods

The global phase 3 GEMINI-1 study (NCT05352893) evaluated the efficacy and safety of intravenous (IV) imsidolimab in patients with GPP and was followed by the GEMINI-2 extension study (NCT05366855), which evaluated maintenance therapy with subcutaneous (SC) imsidolimab. Here, we report the immunogenicity of imsidolimab, as assessed by the development of anti-drug antibodies (ADAs). In GEMINI-1, patients with a history of GPP and moderate-to-severe flare were randomized (1:1:1) to receive one of two IV imsidolimab doses or placebo. In GEMINI-2, the same patients received monthly SC imsidolimab, placebo, or rescue therapy.

Results

In GEMINI-1, only 1 of 30 (3%) patients was ADA-positive at baseline and developed a treatment-boosted, non-neutralizing ADA after a single 750 mg IV dose. In GEMINI-2, 7 of 41 (17%) patients developed treatment-emergent ADAs, with a maximum titer of 1280 and a median time to detection of 32 weeks. Only two patients developed neutralizing antibodies. No apparent impact of ADA development on efficacy or safety was observed in either trial, nor were any effects on pharmacokinetics or a need for redosing identified.

Conclusions

Compared with the available therapeutic option for GPP, Spevigo, imsidolimab demonstrates a markedly lower ADA incidence and no requirement for redosing, supporting its potential clinical advantage as treatment for GPP.

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Abstract N°: 2421

Imsidolimab Improves Generalized Pustular Psoriasis (GPP) Pustulation as Measured by the Pustulation Rating Scale (PRS): Results from the GEMINI-1 Study

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Introduction

Generalized pustular psoriasis (GPP) is a rare, severe, and life-threatening disease characterized by debilitating flares of non-infectious pustular and erythematous skin lesions. The pathogenesis of GPP is mainly attributed to excessive activity of the interleukin-36 (IL-36) pathway, often caused by mutations in the *IL36RN* gene encoding the IL-36 receptor antagonist. Imsidolimab is a novel high-affinity humanized IgG4 monoclonal antibody that binds the IL-36 receptor and antagonizes IL-36 signaling.

Materials and Methods

GEMINI-1 (NCT05352893) was a phase 3 clinical study that evaluated the efficacy and safety of intravenous (IV) imsidolimab in GPP. The study met the primary endpoint of Generalized Pustular Psoriasis Physician Global Assessment scores of clear/almost clear. Here, we report the key secondary efficacy endpoint of achieving a Pustulation Rating Scale (PRS) score of 0 (clear) or 1 (almost clear) at Week 1. The PRS is a physician-based assessment of GPP pustulation, rated from 0 to 4 (severe). To be eligible for GEMINI-1, patients needed a PRS score ≥3 (moderate severity). Eligible patients with a GPP history and moderate-to-severe flare were randomized (1:1:1) to receive one of two IV imsidolimab doses or placebo.

Results

PRS scores improved with imsidolimab, with 67% (10/15) of patients treated with 300 mg IV imsidolimab, the intended clinical dose, achieving clear or almost clear pustulation at Week 1, versus 13% (2/15) of placebo patients (p=0.0002).

Conclusions

These findings suggest that a single IV dose of imsidolimab represents a promising therapeutic option for improving pustulation in GPP patients, as demonstrated across multiple clinical endpoints and supported by corresponding biomarker changes.

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Abstract N°: 2421

JAK-STAT Pathway Inhibition in Coexisting Autoinmune Cutaneous Diseases

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Introduction

Bullous pemphigoid (BP) is an autoimmune blistering disorder that predominantly affects elderly patients, with diagnosis in young adults being rare. BP, alopecia areata (AA), and vitiligo have each been independently associated with dysregulation of the JAK–STAT signaling pathway; however, the coexistence of all three conditions and their simultaneous therapeutic management have not been previously reported.

Case Presentation

A 35-year-old woman with a longstanding history of stable acrofacial vitiligo, which had remained untreated for several years, developed BP in 2022. The diagnosis was confirmed by histopathologic examination, direct immunofluorescence, and the detection of positive anti-BP180 antibodies. During follow-up, antithyroid antibodies were detected despite normal thyroid function. Initial treatment consisted of oral prednisone at variable doses in combination with methotrexate (10–15 mg/week), resulting in only a partial response, with persistent active blistering lesions and pruritus. In May 2024, she developed ophiasis-pattern AA, which, together with inadequate BP control, prompted the discontinuation of methotrexate and consideration of an alternative therapeutic strategy.

In February 2025, oral baricitinib at a dose of 4 mg/day was initiated, while prednisone 10 mg/day was maintained and gradually tapered over a 14 week period. Complete resolution of blistering lesions occurred within the first 4 weeks, and full hair regrowth was achieved by week 14. Following corticosteroid withdrawal, the patient remained in remission. In January 2026, 11 months after treatment initiation, the patient remained in complete remission of PA and AA, with slight improvement of vitiligo and no reported adverse events. In this context, the dose of baricitinib was reduced to 2 mg/day. After 14 months of follow-up, and following 2 months of treatment de-intensification, complete remission of AA was maintained, with only occasional sporadic PA lesions that resolved with topical corticosteroids.

Conclusion

Since 2022, fewer than 20 cases of BP treated with JAK inhibitors have been reported, the majority achieving clinical remission with a favorable safety profile; experience with baricitinib remains particularly limited, with only three published cases to date. The clinical response observed in our patient supports the presence of shared pathogenic mechanisms and highlights the therapeutic potential of JAK–STAT pathway inhibition.

We describe an exceptional case of concomitant BP, AA, and vitiligo in a young patient. Treatment with baricitinib achieved complete control of BP, resolution of AA, and successful corticosteroid withdrawal, with a favorable safety profile sustained over one year of follow-up.

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Abstract N°: 2436

Severe Scalp Psoriasis Exacerbation with Alopecia During Bimekizumab Therapy: A Diagnostic Dilemma

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Introduction

Psoriasis is a chronic, immune-mediated inflammatory skin disease characterised by erythematous, scaly plaques. Modern treatment includes biologic agents targeting the IL-17 pathway, such as bimekizumab, which are highly effective but associated with an increased risk of fungal infections due to the role of IL-17 in antifungal immunity. Paradoxical reactions, including new-onset or severe exacerbation of scalp psoriasis, have also been reported during bimekizumab therapy. We present a case of a patient who developed a significant worsening of scalp psoriasis during bimekizumab treatment, accompanied by hair loss that required differential diagnosis with other causes of alopecia.

Case Presentation

A 45-year-old woman with plaque psoriasis was admitted because of marked exacerbation of scalp lesions accompanied by hair loss while receiving biologic therapy with bimekizumab. Her previous psoriasis treatment had included topical corticosteroids and systemic therapy with acitretin and methotrexate. However, because of insufficient response to conventional systemic therapy, bimekizumab was initiated. At treatment initiation, the patient reported moderate psoriatic involvement of the scalp, which initially showed almost complete improvement during therapy. After approximately 12 months of bimekizumab treatment, the patient developed a recurrence of severe erythematous, infiltrated, and scaly lesions of the scalp associated with progressive hair shedding and the formation of alopecic patches, initially involving the occipital region and subsequently extending to the parietal scalp. She also reported scalp pruritus and intermittent serous discharge from the lesions. The patient had no prior history of severe scalp exacerbations or hair loss. Histopathological examination of a scalp biopsy demonstrated findings consistent with psoriasis. During the diagnostic workup, antinuclear antibody (ANA) testing was positive. In view of the marked hair loss with a positive hair pull test and positive antinuclear antibodies, bimekizumab was temporarily withheld and further investigations were undertaken to exclude an overlapping connective tissue disease. Direct immunofluorescence of the scalp biopsy was negative, and the remaining autoimmune workup including indirect immunofluorescence was unremarkable, with no evidence of an underlying connective tissue disease. A specimen collected from the scalp for mycological culture also yielded a negative result. Trichoscopic examination was performed, revealing regularly arranged glomerular vessels with yellow scales, suggestive of overlapping psoriasis and seborrheic dermatitis. Given these findings, empirical treatment with oral itraconazole and topical betamethasone/calcipotriol was initiated. The treatment was well tolerated and resulted in partial regression of scalp lesions together with gradual hair regrowth. Because a paradoxical exacerbation of scalp psoriasis could not be excluded, bimekizumab therapy was discontinued and treatment with an IL-23 inhibitor was subsequently initiated, which resulted in complete clinical clearance and hair regrowth within 12 weeks.

Conclusion

This case highlights the diagnostic difficulty of severe scalp lesions with alopecia developing during bimekizumab therapy. In a patient without prior extensive scalp involvement, the clinical picture raises the possibility of secondary treatment failure, a paradoxical reaction, or an overlap of psoriasis and seborrheic dermatitis, while superimposed fungal infection promoted by IL-17 inhibition and connective tissue disease must be carefully excluded. Such overlapping mechanisms

underscore the need for a comprehensive diagnostic approach integrating clinical, trichoscopic, histopathological, and laboratory evaluation. When a paradoxical reaction cannot be excluded, an interclass switch to an IL-23 inhibitor offers a mechanistically rational option that can achieve complete clearance and hair regrowth.

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Abstract N°: 2449

Dupilumab-Induced Maculopapular Exanthem: A Case Report

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Introduction

Dupilumab is a fully human monoclonal antibody directed against the interleukin-4 receptor alpha subunit (IL-4Rα), thereby inhibiting IL-4 and IL-13 signaling pathways involved in the pathogenesis of Atopic Dermatitis. In the European Union, subcutaneous dupilumab is approved for the treatment of moderate-to-severe atopic dermatitis, uncontrolled eosinophilic asthma, and chronic rhinosinusitis with nasal polyposis. Although dupilumab demonstrates a favorable safety profile, cutaneous adverse reactions may rarely occur and can represent a diagnostic and therapeutic challenge.

Case Presentation

We report the case of a 50-year-old woman with long-standing atopic dermatitis beginning in childhood, refractory to conventional therapy including emollients, topical corticosteroids, and systemic corticosteroid treatment. Due to persistent disease activity and significant impairment of quality of life, biologic therapy with dupilumab was initiated. Four days after the first administration, the patient developed an intensely pruritic maculopapular exanthem initially localized at the injection site, followed by progressive dissemination involving the trunk and extremities. Clinical examination revealed diffuse erythematous macules and papules without mucosal involvement or systemic symptoms. The eruption resolved following treatment with topical corticosteroids and short-course oral corticosteroid therapy.

To exclude an immediate hypersensitivity reaction, allergological evaluation was performed two weeks after resolution of the initial eruption. Skin prick and intradermal testing with diluted dupilumab solutions (1:100 and 1:10) as well as undiluted preparation yielded negative results at immediate readings performed after 15–20 minutes. Delayed readings at 24, 48, 72, and 96 hours also remained negative, arguing against both immediate IgE-mediated hypersensitivity and delayed-type allergic sensitization detectable by cutaneous testing.

Despite the negative allergological workup, a second administration of dupilumab was followed by recurrence of a similar cutaneous eruption. The lesions reappeared approximately 24 hours after injection, initially at the administration site, with subsequent generalized dissemination after 48 hours. The recurrent temporal association between dupilumab administration and onset of the exanthem strongly supported a drug-induced cutaneous adverse reaction. Owing to recurrence and progression of the eruption after re-exposure, dupilumab therapy was discontinued.

Conclusion

Dupilumab may rarely induce clinically significant delayed hypersensitivity reactions with complex immunopathological mechanisms. In the present case, the patient developed a generalized pruritic maculopapular eruption associated with fever, altered general status, myalgias, odynophagia, and generalized lymphadenopathy, consistent with a serum sickness-like reaction suggestive of type III delayed hypersensitivity; however, generalized adenopathies may also occur in Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS syndrome), a

type IV delayed hypersensitivity reaction, and overlap syndromes between these entities have been reported in the literature. Despite negative prick and intradermal tests, the positive reintroduction test confirmed dupilumab imputability. The occurrence of the reaction after the first administration suggests prior sensitization, most likely to an excipient such as polysorbate 80, the excipient most frequently associated with hypersensitivity reactions to biologic agents and absent from the formulation of Janus kinase inhibitors, which may represent a therapeutic alternative. This case emphasizes the importance of close clinical surveillance and multidisciplinary evaluation in patients developing systemic and cutaneous adverse reactions during biologic therapy.

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Abstract N°: 2478

Assessment of certain genetic markers polymorphism for the prediction of susceptibility to methotrexate toxicity in Egyptian psoriatic patients.

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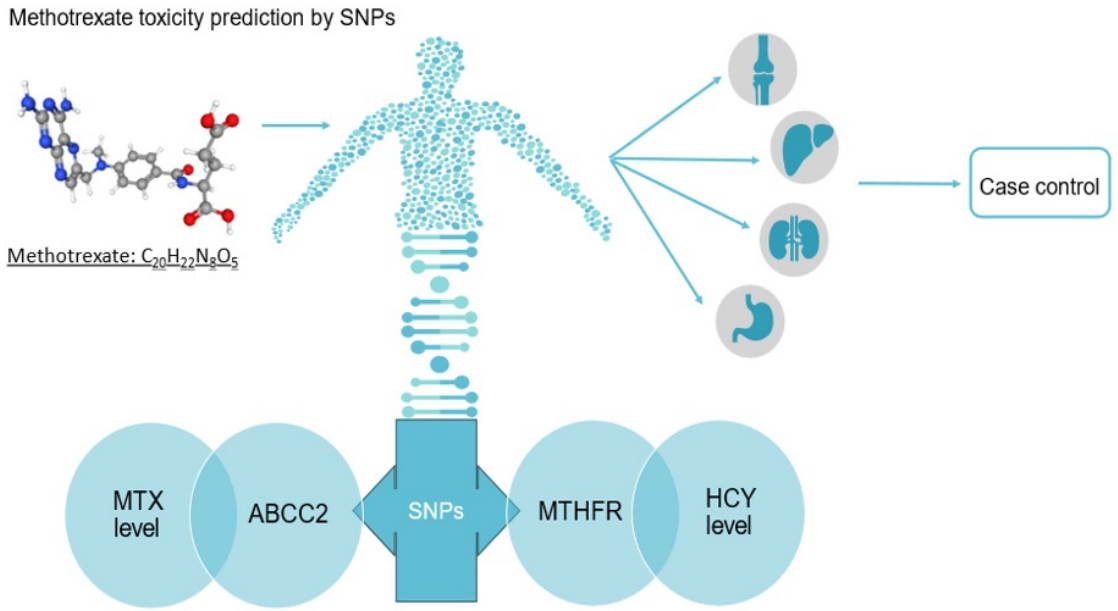
Introduction

Methotrexate (MTX) is a major drug used in most treatment protocols for cellular hyperproliferative benign and malignant disease due to its anti-inflammatory and anti-proliferative properties. It acts by inhibiting various enzymes in the synthesis pathway of folate, methionine, adenosine, and nucleotides **(Romero, et al., 2020)**.

Methotrexate is the standard treatment for most cases of psoriasis. However, it has a high percentage of toxicities. This toxicity profile varies from person to person. Various studies have reported that these interindividual differences may be due to genetic factors, such as single nucleotide polymorphisms (SNPs), which are involved in methotrexate pharmacodynamics, metabolism and mechanism of action **(Membrive et al., 2020).** These genes are involved in cellular uptake/efflux, folate antagonism, adenosine signaling, and methyl donation. Therefore, the ability to predict MTX toxicity using a selected number of reliable gene polymorphisms is of utmost interest **(Onishi, et al.,2020).**

These biomarkers which are more prone to be included in MTX toxicity are ABCB1 (rs1045642), ABCC2 (rs4148396), MTHFR 677 (rs1801133) **(Romero, et al., 2020)**.

So, the aim of the current study is to investigate certain genetic DNA markers single nucleotide polymorphism (SNPs) and measurement of the resulting proteins levels (Methotrexate and Homocysteine) in addition to clinical correlation and laboratory investigations for prediction of susceptibility to MTX toxicity among psoriases patients.



Graphical abstract

Materials and Methods

The current study was a prospective cohort study of psoriases patients receiving methotrexate therapy at Tertiary Hospital.

Patients with any liver or kidney disease before the start of treatment and patients with identifiable allergies to MTX were excluded.

MTX regimen protocol were standardized between the study groups to ensure equitable comparisons.

Recruited patients were followed to a duration of one and half years to detect the pattern of MTX toxicity and those who showed any manifestations of MTX toxicity (liver, hematological, renal, neurological, skin toxicities), were compared to an equivalent sample included patients who did not show any of the above toxicities (45 patients showed MTX toxicity)

To diagnose MTX related toxicities, a CBC with differential, liver function test results (ALT), (AST), and kidney function test results (containing serum urea and creatinine levels) were evaluated, Estimated glomerular filtration rate (eGFR) was calculated for all the patients to be correlated with MTX level and renal affection.

Methotrexate levels, homocysteine levels were assayed using an enzyme-linked immunoassay (ELISA).

The studied SNPs (MTHFR 677 (rs1801133) and ABCC2 (rs4148396)) were genotyped via real-time PCR

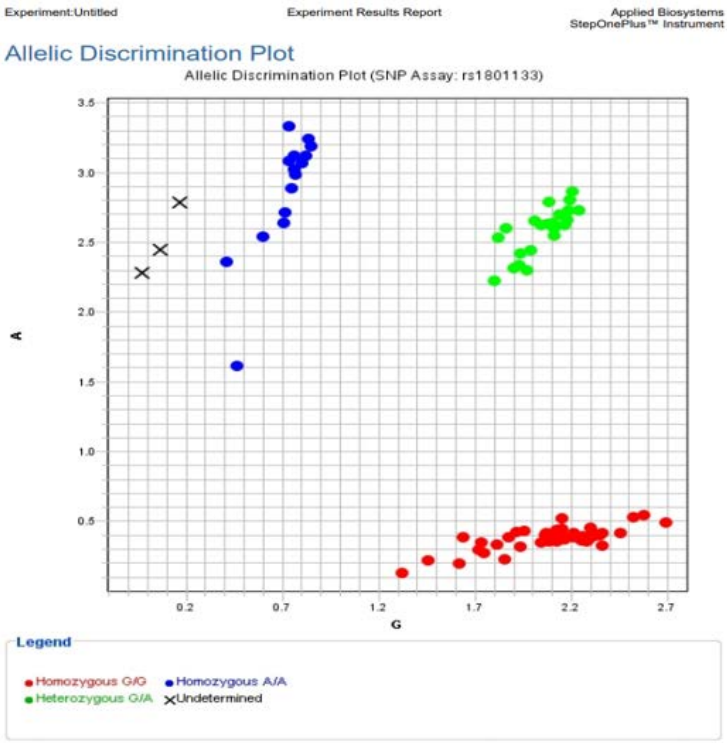


Fig. 1: Applied biosystem software revealing the allelic discrimination among patients with the MTHFR SNP (CC for GG, TT for AA, and CT for GA).

Applied biosystem software revealing the allelic discrimination among patients with the MTHFR SNP (CC for GG, TT for AA, and CT for GA).

Results

The pattern of MTX toxicity showed that GIT toxicity was in 27% of the cases, and myelosuppression in 33% percent of cases group and hepatotoxicity represented (66.7%) of the toxicity presentation of cases group. After assessment of certain genetic markers polymorphism MTHFR 677 C allele was the most prevalent in all patients (69%). The mutant homozygous (TT) allele was observed more in case group in comparison with control group (30%, 7%). ABCC2 C allele was the most prevalent (75%), while T allele presents in 25% of patients, The studied groups showed lower expression of the mutant homozygous (CT) allele in case group in comparison with control group (30%, 53%) respectively with high statistical difference between both groups. As for MTX levels it was observed to be higher in case group, while Homocysteine levels showed no difference between studied groups.

Polymorphism	MTHFR C667T			ABCC2		
	CC	CT	TT	CC	CT	TT
Hematological toxicity (n)	6	3	6	9	6	0
Yes	40%	20%	40%	60%	40%	0%
No	58.70%	28%	13.30%	53.30%	41.30%	5.3
P value	Reference	0.951	0.028*	Reference	0.795	0.999
OR		1.048	4.4		0.86	----
95% CI		(0.238-4.603)	(1.171-16.531)		(0.277-2.675)	----
Liver toxicity(n)	12	7	11	21	8	1
yes	40%	23.30%	36.70%	70%	26.70%	3.30%
No	63.30%	28.30%	8.30%	46.70%	48.30%	5%
P value	Reference	0.634	0.002*	Reference	0.042*	0.496
OR		1.304	6.967		0.368	0.444
95% CI		(0.437-3.892)	(2.015-24.085)		(0.140-0.966)	(0.043-4.581)
kidney toxicity (n)	1	1	2	2	2	0
yes	25%	25%	50%	50%	50%	0%
No	57%	26.70%	16.30%	54.70%	40.70%	4.70%
P value	Reference	0.599	0.123	Reference	0.774	0.999
OR		2.13	7		1.343	----
95% CI		(.128-35.59)	(0.59-82.993)		(.180-10)	----
GIT toxicity (n)	6	2	5	8	3	2
Yes	46.20%	15.40%	38.50%	61.50%	23.10%	15.40%
No	57.10%	28.60%	14.30%	53.20%	44.20%	2.60%
P value	Reference	0.636	0.082	Reference	0.267	0.127
OR		0.667	3.333		0.452	5.125
95% CI		(.124-3.578)	(.857-12.966)		(.111-1.839)	(.627-41.9)

Allelic distribution differences among MTHFR and ABCC2 SNPs in relation to the occurrence of organ toxicity

Conclusions

It can be concluded that toxicity is observed more in T polymorphism of MTHFR and in the C allele of ABCC2 gene. Methotrexate levels were higher in cases group than those without toxicity while no difference of homocysteine was traced between groups, so genetic testing may be required in certain patients with preexisting risk factors for mtx toxicity

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Abstract N°: 2499

When treatment worsens disease: paradoxical eczematous reaction to IL-17 inhibitors in psoriasis

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Introduction

The use of monoclonal antibodies has increased significantly in recent years, with more than 40 agents approved over the past five years, revolutionizing the management of dermatologic diseases. However, some patients may experience paradoxical reactions, which are defined as the de novo development or worsening of a pre-existing immune-mediated condition following treatment initiation.[1,2,3] Among these, eczematous reactions are the most frequently reported with an incidence of approximately 12%, particularly associated with IL-17 inhibitors.[1] It has been hypothesized that blockade of the Th1/Th17 axis may promote a shift toward Th2-mediated inflammation, favoring the development of atopic-like eczema.[1,2,3] Identified risk factors include a history of atopy, eosinophilia, raised serum IgE and IL-17 inhibitor use.[4]

Case Presentation

A 23-year-old female with a history of erythematous scaly plaques since childhood, previously diagnosed and treated as seborrheic dermatitis and atopic dermatitis, remained in clinical remission until adulthood. She subsequently developed scalp and submammary lesions refractory to multiple treatments, including antifungals, antibiotics and topical corticosteroids. Histopathologic examination was performed, confirming a diagnosis of severe psoriasis in high-impact areas. Advanced systemic therapy with IL-17 inhibitors was proposed; however, the patient declined treatment due to concerns about possible adverse effects and was lost to follow-up for two years. The patient later returned with worsening scalp disease and persistent submammary plaques, as well as new genital lesions associated with significant impairment in quality of life. She also reported a one-year history of painful nodules in the axillary and inguinal regions, as well as recurrent ingrown hairs, acne and clinical features suggestive of hyperandrogenism and insulin resistance. Therefore, at this point, a diagnosis of severe psoriasis involving high-impact areas and hidradenitis suppurativa was established (PSSI 20, DLQI 12, IHS4 2). Given the coexistence of both inflammatory diseases, the patient was considered an appropriate candidate for anti–IL-17 therapy. Despite counseling on the benefits of biologic therapy, she lost follow-up for another year and returned with disease progression. At that time, she agreed to initiate biologic therapy and bimekizumab was started. Following the second dose, there was marked improvement in scalp psoriasis and hidradenitis suppurativa (PSSI 3, DLQI 2, IHS4 0); however, pruritic eczematous plaques appeared on the neck, axillae and submammary region with clinical signs of secondary infection. Bacterial and fungal cultures were requested and empiric therapy with systemic antibiotics and topical antifungals was initiated without improvement. Cultures were reported as negative.

Given these findings, a paradoxical eczematous reaction to IL-17 inhibition was suspected.Bimekizumab was discontinued and upadacitinib was prescribed for 15 days, resulting in rapid improvement of the eczematous lesions within 72 hours but concomitant worsening of scalp psoriasis (PSSI 45, DLQI 20) and the development of new psoriatic plaques on the body. Therefore, therapy was transitioned to deucravacitinib, resulting in adequate psoriasis control and complete resolution of the eczematous lesions.



Figure 1. Clinical timeline of disease progression, therapeutic interventions and treatment-related outcomes.

Conclusion

Despite the transformative impact of biologic therapies in the management of inflammatory dermatologic diseases, they are not exempt from adverse effects, including increasingly recognized paradoxical reactions. Early identification and accurate diagnosis are essential to ensure timely and appropriate management. This case highlights the potential role of targeted small molecules as effective therapeutic alternatives in managing paradoxical eczematous reactions while maintaining control of psoriatic disease. Further studies are needed to establish evidence-based guidelines for the management of these reactions.

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Abstract N°: 2523

Superior long-term drug survival of IL-23 inhibitors over IL-17 inhibitors in psoriasis: an extended real-world treatment-episode and predictor analysis

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Introduction

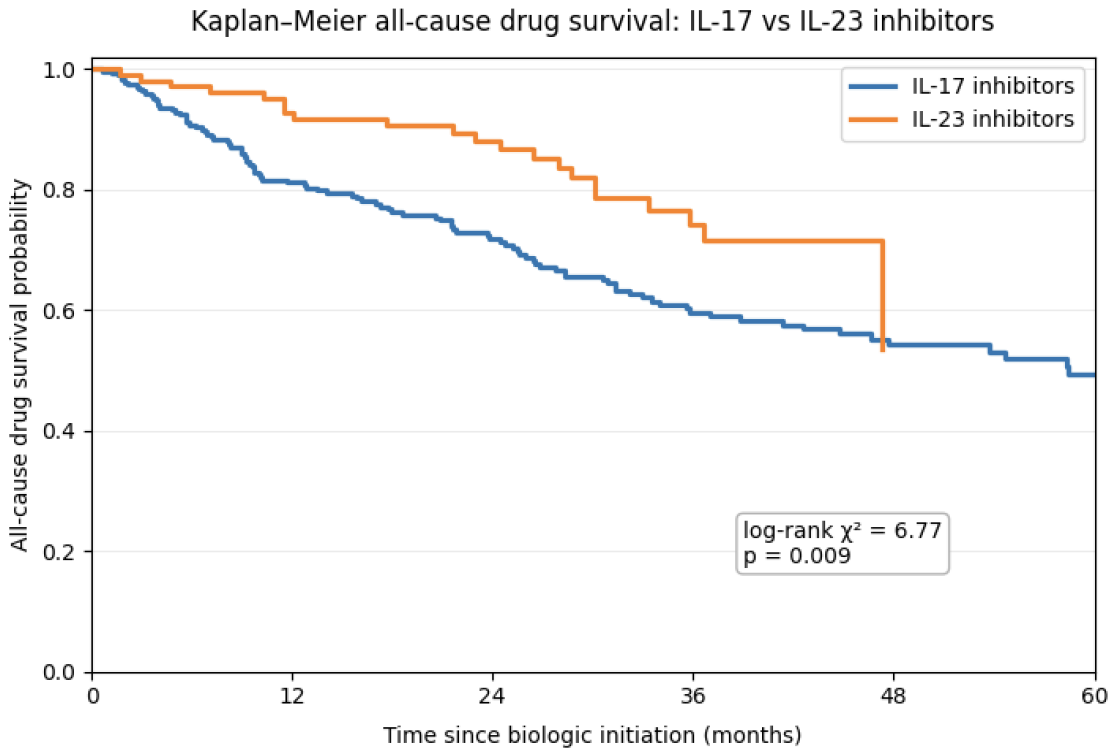
Drug survival is a key real-world outcome integrating treatment effectiveness, safety, and persistence in psoriasis. Although IL-17 and IL-23 inhibitors have demonstrated high efficacy in clinical trials, comparative long-term real-world survival data remain limited. Furthermore, the determinants of treatment discontinuation are not fully established. This study aimed to compare the drug survival of IL-17 and IL-23 inhibitors and to identify independent predictors of treatment discontinuation in a real-world psoriasis cohort.

Materials and Methods

This retrospective study included 310 patients contributing 376 biologic treatment episodes. Secukinumab and ixekizumab were classified as IL-17 inhibitors, and guselkumab and risankizumab as IL-23 inhibitors. Drug survival was defined as time from treatment initiation to discontinuation. All-cause discontinuation was the primary endpoint; a secondary analysis evaluated discontinuation due to lack of efficacy (primary non-response, secondary non-response, or PsA flare). Ongoing treatments and loss to follow-up were right-censored. Kaplan–Meier survival analysis with log-rank testing and multivariable Cox proportional hazards regression were performed. The Cox model was adjusted for age, sex, psoriatic arthritis (PsA), previous biologic exposure, treatment sequence, and comorbidity.

Results

A total of 376 treatment episodes were analyzed, including 272 (72.3%) IL-17 and 104 (27.7%) IL-23 inhibitor treatments. Overall, 124 discontinuation events were observed. Crude discontinuation rates were significantly higher with IL-17 inhibitors compared with IL-23 inhibitors (37.9% vs. 20.2%, p=0.002). Kaplan–Meier analysis demonstrated significantly superior drug survival for IL-23 inhibitors (log-rank p=0.009). At 36 months, drug survival was 74.0% for IL-23 inhibitors versus 59.5% for IL-17 inhibitors (Figure 1). Median drug survival was reached only in the IL-17 group (58.5 months). In multivariable Cox analysis, IL-17 inhibitor use was independently associated with a higher risk of discontinuation compared with IL-23 inhibitors (adjusted HR 2.06, 95% CI 1.25–3.40, p=0.005). Female sex (HR 1.45, p=0.049), presence of PsA (HR 1.74, p=0.004), and previous biologic exposure (HR 1.62, p=0.016) were also independently associated with increased discontinuation risk. In the efficacy-related analysis, IL-23 inhibitors demonstrated significantly superior survival (log-rank p=0.004). IL-17 inhibitor use (adjusted HR 2.97, 95% CI 1.52–5.80, p=0.001), PsA, and previous biologic exposure remained independent predictors of discontinuation. When individual agents were evaluated, drug survival differed significantly (log-rank p=0.003), with the most favorable persistence observed for risankizumab and the lowest for secukinumab.



Kaplan–Meier curve showing all-cause drug survival of IL-17 and IL-23 inhibitors. IL-23 inhibitors demonstrated significantly superior drug survival compared with IL-17 inhibitors (log-rank p=0.009). At 36 months, drug survival was 74.0% for IL-23 inhibitors versus 59.5% for IL-17 inhibitors.

Conclusions

In this extended real-world treatment-episode analysis, IL-23 inhibitors demonstrated significantly superior long-term drug survival compared with IL-17 inhibitors. In addition to drug class, psoriatic arthritis and prior biologic exposure were key independent determinants of treatment discontinuation. These findings highlight both the persistence advantage of IL-23 inhibition and the importance of patient-related factors in optimizing long-term biologic treatment strategies.

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Abstract N°: 2548

Nonresponse to hydroxychloroquine and rituximab in cutaneous lupus is associated with increased type I interferon responses in stromal cells

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Introduction

Cutaneous lupus erythematosus (CLE) is a chronic inflammatory skin disease that may occur either as an isolated condition or in association with systemic lupus erythematosus (SLE). Activation of type I interferon (IFN) signaling is a central driver of CLE pathogenesis and involves both stromal and immune cell populations. Hydroxychloroquine (HCQ) is the first-line therapy for CLE, with overall response rates of approximately 60%. However, a substantial proportion of patients exhibit an inadequate response, and the mechanisms underlying treatment resistance remain poorly understood. Anifrolumab, a monoclonal antibody targeting the type I IFN receptor, has shown efficacy in refractory cases, but biomarkers predicting treatment response are few.

Materials and Methods

We performed single-cell RNA sequencing (scRNA-seq) on skin biopsies of patients with active CLE prior to initiation of medication or 6 months after medication use. Patients treated with HCQ, Rituximab or Anifrolumab (n =6) were sampled and compared with healthy controls (n = 5). Paired lesional and non-lesional skin biopsies, as well as blood samples, were collected before and after treatment. Clinical response was defined as a ≥70% reduction in the Cutaneous Lupus Erythematosus Disease Area and Severity Index (CLASI70). Patients were stratified into responders (n = 4) and non-responders (n = 2), and transcriptomic profiles were analyzed across tissues following cell-type annotation based on canonical marker genes.

Results

Although all CLE samples exhibited elevated type I IFN signatures, non-responders to HCQ and rituximab demonstrated significantly higher IFN activity prior to treatment induction compared with responders and healthy controls. This effect was particularly pronounced in endothelial cells and keratinocytes. Ingenuity Pathway Analysis predicted activation of type I IFN signaling, oxidative phosphorylation, and mitochondrial dysfunction in these cell types of non-responders. In addition, lesional fibroblasts from non-responders to HCQ displayed a senescence-associated transcriptional profile that was absent in responders. Anifrolumab reduced IFN signatures back to levels of healthy controls, rescuing skin inflammation even in a non-responder to Rituximab.

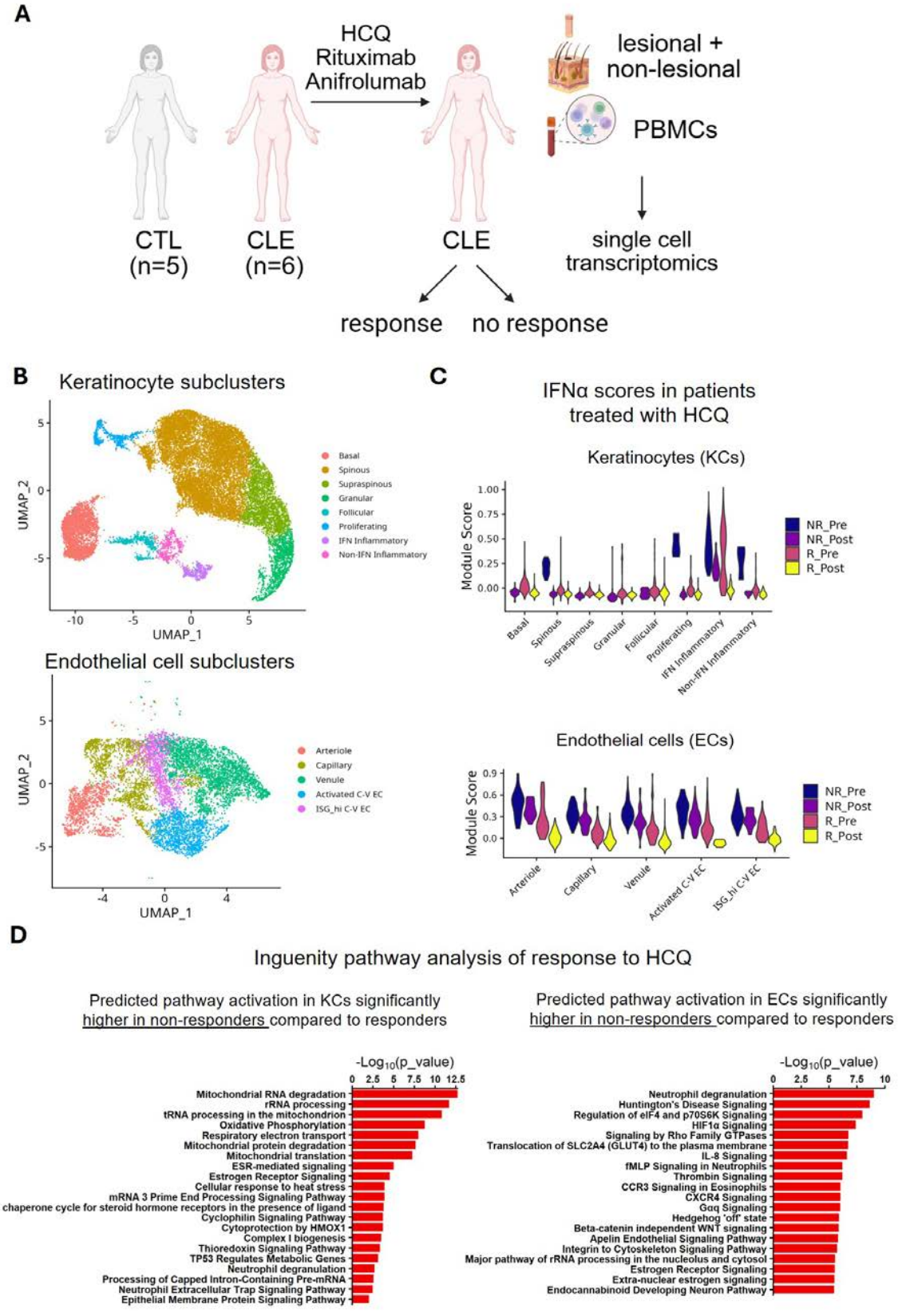

Figure 1. Higher stromal IFN signatures characterize HCQ non-responders in CLE. A. Study design. Healthy controls (n=5) and CLE patients (n=6) spent skin and blood samples at baseline and after therapy with either HCQ, Rituximab or Anifrolumab. Patients were categorized as responders by CLASI70 response. B. UMAP plots of keratinocyte and endothelial subclusters reveal 11 keratinocyte subclusters and 5 endothelial subclusters. C. Interferon module scores across keratinocyte and endothelial subclusters in baseline (pre) and post treatment (post) samples from non-responders (NR) and responders (R). D. Inguenity pathway analysis of predicted pathway activation in keratinocytes and endothelial cells of non-responders and responders highlights mitochondrial dysfunction, cytokine signaling and interferon activation in non-responders.

Conclusions

Our findings suggest that patients with a high baseline type I IFN signature in stromal cells are less likely to respond to HCQ or rituximab and may require more potent suppression of IFN signaling. These patients may represent a subgroup that could particularly benefit from targeted therapies such as anifrolumab.

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Abstract N°: 2549

Changes in biological therapies in a patient with severe hidradenitis suppurativa

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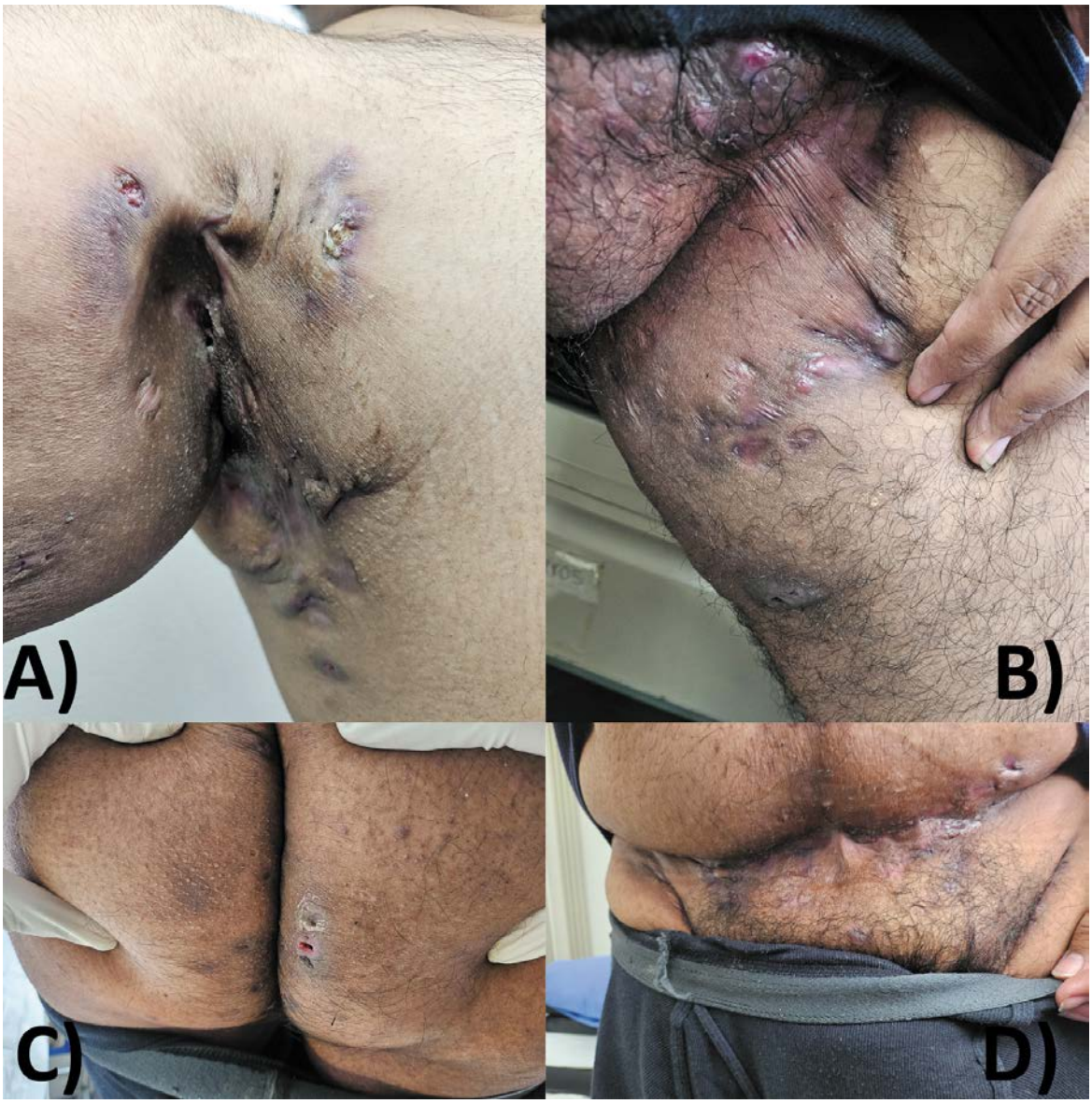
Introduction

In cases of severe hidradenitis suppurativa (HS), biologic therapies are recommended to achieve symptomatic control of the disease. These drugs are highly targeted to specific molecular targets, so their efficacy is usually good to excellent, with very few or no adverse effects. However, it is not uncommon to find patients refractory to such regimens, and in such cases, some mistakenly consider abandoning the use of these drugs altogether and returning to conventional medical management only. We present the case of a patient with severe HS refractory to conventional medical management who was started on adalimumab, without clinical improvement, so the patient was switched to secukinumab, with a very limited response, necessitating a further change to bimekisumab, with which there was a sustained favorable response after 15 days of use.

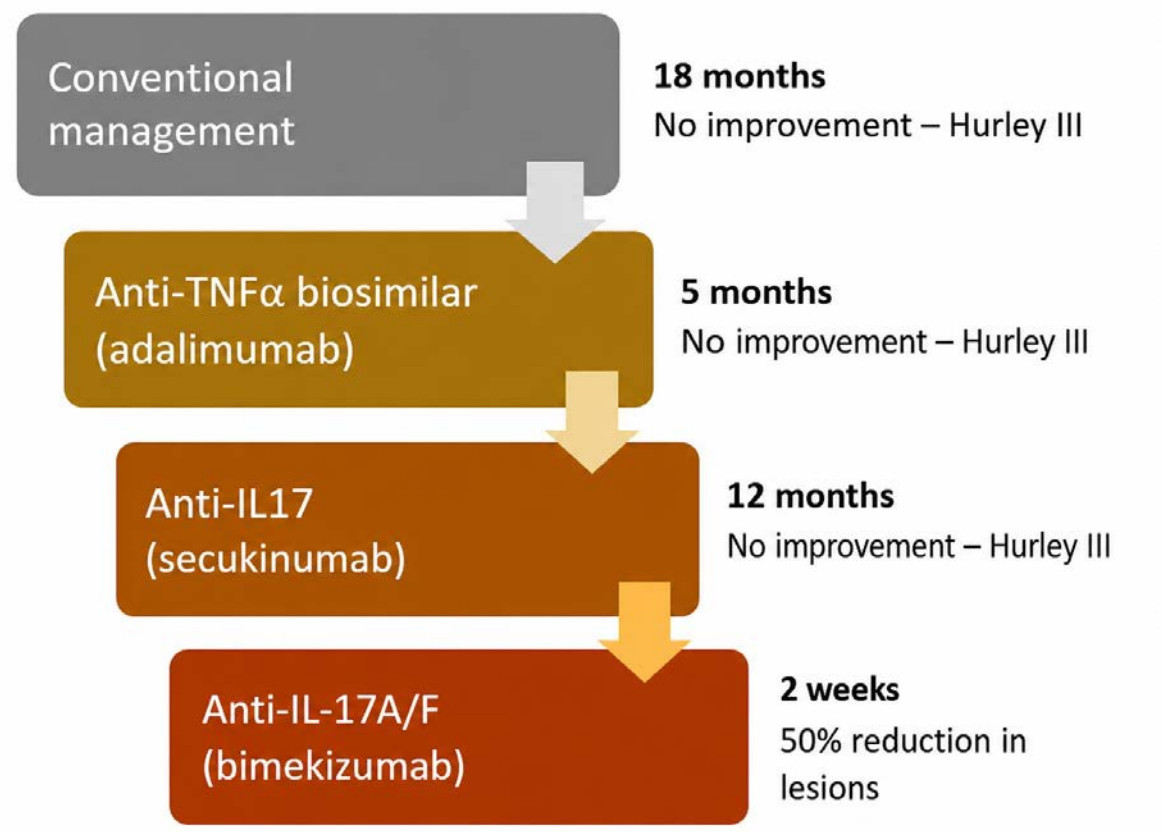


HS dermatological lesions during the initial assessment in the axillae (A), groin (B), buttocks (C), and suprapubic region (D).

Materials and Methods

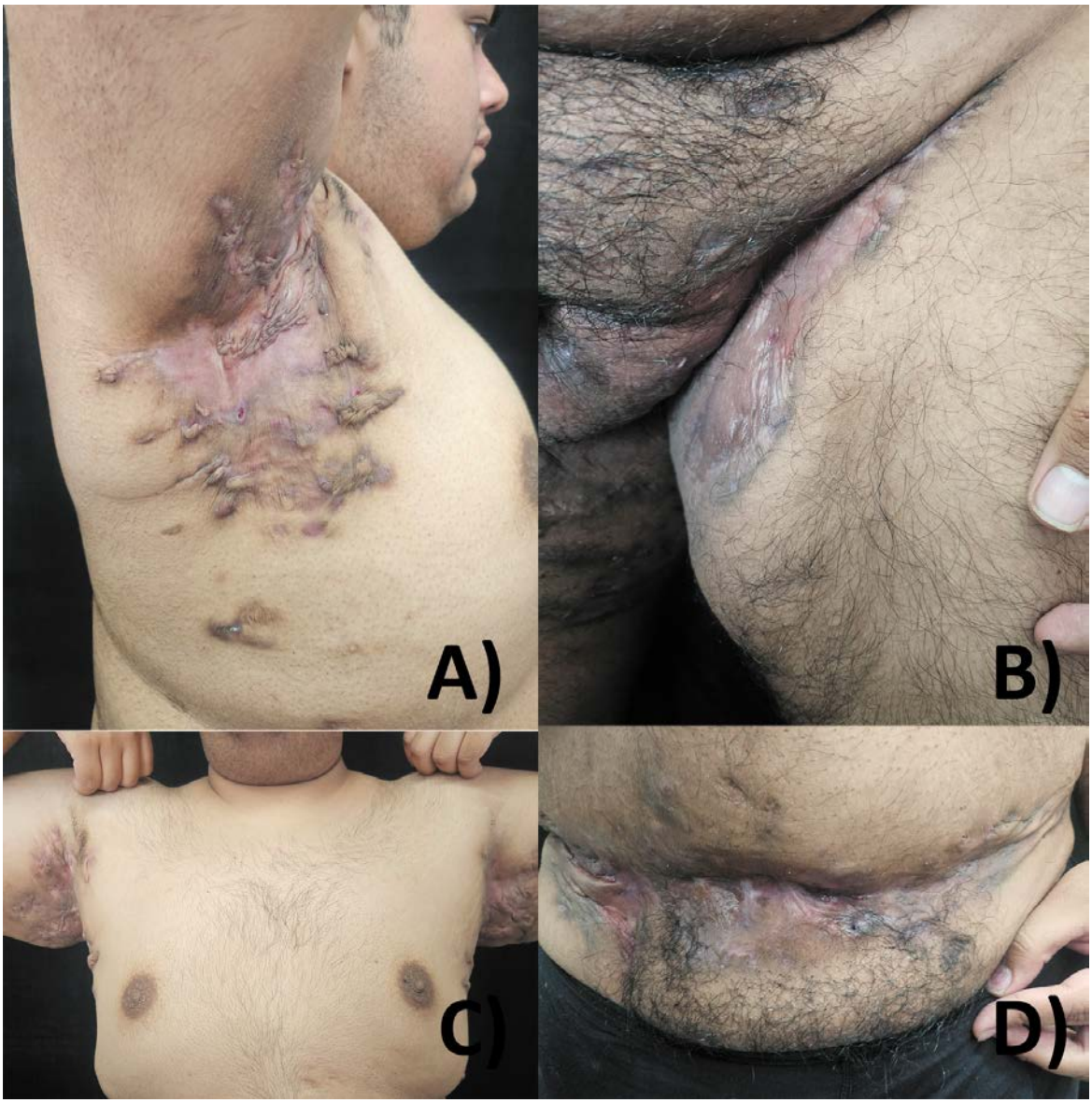
Report a case of severe HS in which therapeutic failure occurred with two different biologic therapies.

The patient is an 20-year-old male, originaly from Minatitlán, Veracruz, Mexico, currently living in Toluca, State of Mexico (2 years), actual occupation is an student (Mechatronics Engineering), with none relevant family history and has a personal medical story of bronchial hyperreactivity since childhood and grade II obesity (weight: 129 kg, height: 1.85 m, BMI: 37.69 kg/m^2).



Results

The patient initially received conventional management, with a Hurley stage III classification at the time of assessment. Despite 18 months of treatment, no clinical improvement was observed; therefore, therapy was switched to an anti-TNF-alpha biosimilar, adalimumab, for 5 months, with no significant change. The patient was subsequently referred to a tertiary-level dermatology service, where treatment with an anti-IL-17 biologic agent, secukinumab, was initiated. Mild improvement was initially observed; however, this was followed by clinical exacerbation, and treatment failure was considered after 12 months of therapy. Consequently, treatment was switched to an anti-IL-17A/F agent, bimekizumab, resulting in a 50% improvement in lesions within 2 weeks.



HS dermatological lesions at the last follow-up visit in the axillae (A), groin (B), thorax (C), and suprapubic region (D).

Conclusions

Not all patients respond to biologic therapy in the same way; in some cases, treatment adjustment is required until the molecule that provides the best clinical response is found.

The presence of risk factors such as obesity, and the proinflammatory state it triggers, represents a challenge in this type of patient.

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Abstract N°: 2551

Improved Outcomes With Early Combination Therapy of Intravenous Immunoglobulin and Cyclosporine in Pediatric SJS/TEN with Sepsis: A Retrospective Case Series

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Introduction

Stevens–Johnson Syndrome/Toxic Epidermal Necrolysis (SJS/TEN) is a severe mucocutaneous reaction, most commonly drug-induced and occasionally infection-triggered in children. Pediatric SJS/TEN with coexisting sepsis represents a particularly high-risk subgroup with significant therapeutic challenges, as concerns regarding immunosuppression often complicate management decisions. Early intervention may improve mortality and morbidity; however, standardized pediatric treatment guidelines remain lacking. Intravenous immunoglobulin (IVIG) and cyclosporine have shown benefit individually, but evidence regarding their combined early use in pediatric SJS/TEN with coexisting sepsis remains limited.

Materials and Methods

Objectives: To evaluate the clinical outcomes of pediatric SJS/TEN with sepsis treated with early with combination therapy using IVIG and cyclosporine.

Methods: We conducted a retrospective analysis of 11 paediatric patients diagnosed with SJS/TEN and concurrent sepsis, managed in Paediatric Intensive Care Unit (PICU) at a tertiary care centre. All patients were treated with IVIG at a dose of 2g/kg over 2-5 days and cyclosporine at a dose of 3-5 mg/kg/day. In addition, supportive care included hydration, infection control, barrier nursing using sterile banana leaves as an economical option.

Data collected included demographic characteristics, trigger factors, extent of body surface area involved, SCORTEN score, time to initiate therapy, duration of hospital stay, time to re-epithelialization and clinical outcomes. Safety and adverse effects related to therapy were also assessed.

Results

A total of 11 patients were included (3 = SJS, 4 = STS/TEN overlap and 4 = TEN) (mean age - 7.8 ± 3.2) M:F (1:1) approximately equal, all with clinically and/or laboratory confirmed sepsis. Mean BSA involvement was 24.9 ± 15.0 and mean SCORTEN was 1.18 ± 0.87. The most common drugs implicated were antiepileptics and NSAIDs.

Average time of initiation of therapy was 2.18 ± 0.75 days of onset. Early clinical stabilization, including cessation of new lesion formation was observed within 1–2 days, followed by a mean time to re-epithelialization of 9.45 ± 1.92 days. The mean duration of hospital stay was 17 ± 3.6 days.

Despite the severity of illness, including sepsis and PICU requirement, mortality was seen in only 1 out of 11 patients (9%)**.** No significant adverse effects attributable to IVIG or cyclosporine were observed, with outcomes that appear favourable given the severity of illness

Conclusions

Therapeutic management of pediatric SJS/TEN with concurrent sepsis is particularly challenging due to the limited availability of safe immunomodulatory options. Early combination therapy with IVIG and cyclosporine appeared to be safe and effective in our cohort, including in critically ill children with sepsis. Use within a PICU setting, alongside meticulous supportive care and low-cost biological dressings such as banana leaves, may contribute to favourable outcomes including faster re-epithelialization and shorter hospital stay. Our findings highlight the potential benefit of early combination immunomodulatory therapy in high-risk pediatric SJS/TEN. Larger prospective controlled studies are required to further validate these observations.

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Abstract N°: 2607

Characterization and Development of JADE301, a Half-Life Extended Anti-IFN-β Monoclonal Antibody for Treatment of Dermatomyositis

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Introduction

Dermatomyositis (DM) is a chronic autoimmune myopathy characterized by a strong Type I interferon (IFN) gene signature in blood, skin, and muscle, which correlates with disease activity and severity. Aberrant IFN-β signaling has emerged as a key pathogenic driver of both cutaneous and muscular inflammation in DM and specific neutralization of IFN-β has demonstrated promising clinical efficacy in DM patients, including improvement of skin lesions and overall disease activity. JADE301 is a novel, fully human IgG1 monoclonal antibody that specifically and potently neutralizes IFN-β and blocks its interaction with interferon-α/β receptor (IFNAR). JADE301 was designed for extended half-life in humans, potentially enabling convenient, infrequent, subcutaneous dosing in DM patients. The aim of these studies was to characterize the functional and pharmacokinetic properties of JADE301 to support its clinical development in DM.

Materials and Methods

The binding of JADE301 to IFN-β was assessed by surface plasmon resonance and competitive ligand blockade was determined by Enzyme-Linked Immunosorbent Assay (ELISA). Functional blockade of IFN-β signaling was assessed using complementary human cellular assays, including stimulation of an Interferon-Sensitive Response Element (ISRE) reporter cell line, inhibition of STAT1 phosphorylation and inhibition of MX-1 gene expression in human peripheral blood mononuclear cells (PBMCs). Pharmacokinetics and safety were assessed in non-human primates (NHP) administered JADE301 up to 300 mg/kg.

Results

JADE301 selectively bound IFN-β with high affinity (K_D ~ 11 pM) and strongly blocked its binding to the IFNAR beta chain (IFNAR2). JADE301 demonstrated potent, concentration-dependent inhibition of IFN-β-induced signal transduction through IFNAR in HEK293-ISRE-luc cells. Similarly, concentration-dependent and potent inhibition of STAT1 phosphorylation in human U937 cells stimulated with IFN-β was observed following incubation with JADE301. Finally, JADE301 potently inhibited expression of MX-1, a well-characterized IFN-stimulated gene (ISG), in human PBMCs after stimulation with IFN-β. In NHP, JADE301 was well-tolerated with no treatment-related adverse clinical observations, and no adverse effects on clinical chemistry or hematology. JADE301 demonstrated low, linear clearance resulting in extended exposure and half-life (> 30 days), approximately 3-fold longer than typical for a therapeutic antibody. Muscle concentrations of JADE301 ranged from approximately 1-4% of those measured in the systemic circulation.

Conclusions

JADE301 is a novel anti-IFN-β neutralizing antibody with extended half-life that potently inhibits signaling through IFNAR and subsequent expression of ISGs, consistent with robust suppression of Type I IFN signaling pathways that drive DM pathogenesis. The favorable tolerability profile observed in NHPs, together with extended half-life and potent IFN-β neutralization, supports clinical evaluation of JADE301 as a targeted therapeutic approach with

reduced treatment burden for patients with DM. The characterization of the pharmacological profile of JADE301, combined with NHP PK and tissue distribution, provides a translational framework for dose selection in DM patients to ensure strong IFN-β neutralization locally at sites of active disease in the skin and muscle.

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Abstract N°: 2613

Pemphigus Foliaceus in a Patient with Long-Standing Psoriasis Treated with Biological Therapy: A Rare Clinical Association

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Introduction

Biologic therapies have transformed the management of moderate-to-severe psoriasis and psoriatic arthritis and are generally considered safe. However, rare immune-mediated cutaneous adverse events, including autoimmune blistering diseases, may occur. The coexistence of psoriasis and pemphigus is uncommon, with psoriasis usually preceding pemphigus. Chronic inflammation, cytokine imbalance and immune modulation by biologics may contribute to autoimmunity.

Case Presentation

We report a 54-year-old woman with long-standing plaque psoriasis and psoriatic arthritis, previously treated with methotrexate, followed by secukinumab and later guselkumab. During biologic therapy she developed a progressive erosive and crusted eruption with superficial skin detachment, generalized erythema and impaired general condition. Although initial histology was non-specific, repeat biopsy demonstrated subcorneal acantholysis. Direct immunofluorescence showed intercellular epidermal IgG and C3 deposition, while serology revealed markedly elevated anti-desmoglein 1 antibodies with negative anti-desmoglein 3 antibodies, confirming pemphigus foliaceus. Biologic treatment was discontinued and systemic corticosteroids with azathioprine were initiated, leading to sustained clinical and immunological remission.

Conclusion

This case highlights the diagnostic challenge of atypical erosive or crusted eruptions in biologically treated psoriasis patients. The temporal association with secukinumab and guselkumab raises the possibility of drug-triggered pemphigus foliaceus, although coexistence related to chronic psoriatic inflammation cannot be excluded. Early biopsy, direct immunofluorescence and desmoglein antibody testing are essential for timely diagnosis and appropriate management.

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Abstract N°: 2671

Dupilumab as a Therapeutic Option for Nodular Prurigo

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Introduction

Nodular prurigo is a chronic inflammatory condition of multifactorial etiology, often associated with atopic diseases. It is currently understood as a neuroimmune inflammatory disease with a predominance of type 2 cytokines. Activation of IL-4 and IL-13 pathways contributes to the recruitment of eosinophils, mast cells, and T lymphocytes, as well as stimulation of sensory nerve fibers responsible for intense pruritus. This immunological pathway, known for its role in atopic dermatitis, is also implicated in maintaining the itch–scratch cycle in nodular prurigo.

Understanding the role of IL-4 and IL-13 cytokines in the pathophysiology of nodular prurigo has supported the use of dupilumab, originally approved for moderate-to-severe atopic dermatitis. Dupilumab is a human monoclonal antibody that blocks the alpha subunit of the interleukin-4 receptor (IL-4Rα), inhibiting the signaling of both IL-4 and IL-13. By blocking this pathway, the drug reduces Th2-mediated inflammation, the release of pruritogenic mediators, and the hyperplasia of cutaneous nerve fibers.

In the present case, the clinical improvement observed after initiation of dupilumab supports findings in the literature and demonstrates the effectiveness of selective Th2 pathway blockade in reducing pruritus and promoting lesion regression. The use of dupilumab represents an important advancement in the management of nodular prurigo, providing sustained symptomatic control and improved quality of life, with a lower risk of systemic adverse effects compared to traditional immunosuppressants.

Case Presentation

A 50-year-old previously healthy male presented with intensely pruritic erythematous-violaceous papules and nodules distributed over the trunk and limbs, with chronic evolution, causing a substantial impact on quality of life. He had previously undergone topical treatment at another facility with emollients, betamethasone, neomycin, and ketoconazole, without clinical improvement. A biopsy was performed, and histopathological analysis revealed subacute spongiotic dermatitis, consistent with the eczematous spectrum. Laboratory tests showed eosinophilia (14.4%). Based on clinical and histopathological findings, a diagnosis of nodular prurigo was established.

Treatment with dupilumab was initiated, with a 600 mg subcutaneous loading dose, followed by 300 mg after 15 days and another 300 mg 30 days after the initial dose. At follow-up, the patient reported improvement in pruritus and regression of most lesions. The treatment was well tolerated, with no adverse events reported to date.

Conclusion

This case highlights the therapeutic potential of dupilumab in the management of nodular prurigo, a chronic and debilitating condition characterized by intense pruritus and significant impairment in quality of life. The patient demonstrated marked clinical improvement, with reduction of pruritus and regression of lesions after initiation of treatment, without adverse effects. These findings reinforce the role of Th2-mediated inflammation, particularly the IL-4 and IL-13 pathways, in the pathophysiology of nodular prurigo and support the use of targeted biologic therapy as an effective and well-tolerated treatment option. Dupilumab represent a valuable alternative for patients with refractory disease, contributing to better disease control and improved quality of life.

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Abstract N°: 2679

Anti-IgE therapy with omalizumab in children with chronic urticaria

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Introduction

Chronic urticaria (CU) in children is a heterogeneous disease associated with a significant reduction in the quality of life of patients and their families. Up to 30–40% of children do not respond to standard therapy with H1-antihistamines (non-sedating antihistamines, nsAH) even when doses are increased 2–4-fold, which necessitates the search for alternative treatment strategies. Omalizumab is a humanized monoclonal antibody that selectively binds to free IgE and prevents its interaction with FcεRI receptors on mast cells and basophils, thereby inhibiting the release of inflammatory mediators. In adult patients, omalizumab is included in international guidelines as second-line therapy for resistant CU; however, data on its use in the pediatric population remain limited, especially in young children.

Materials and Methods

An open-label, single-center, uncontrolled observational study was conducted. The study included 30 children aged 4 to 16 years (mean age 11.8±3.2 years; girls 53.3%, n=16) with a verified diagnosis of chronic urticaria resistant to increased doses of nsAH. Phenotypes were distributed as follows: chronic spontaneous urticaria (CSU) — in 16 (53.3%), chronic inducible urticaria (CIndU) — in 9 (30.0%), and a combined form (CSU + CIndU) — in 5 (16.7%). Five patients had CSU with angioedema. Omalizumab was administered subcutaneously at a dose of 150–300 mg every 4 weeks for 6–12 months. Children older than 12 years received 300 mg according to clinical guidelines; preschool-aged children (4–6 years, n=5) received 150 mg off-label after approval by the hospital ethics committee/medical board. Efficacy was assessed by changes in the Urticaria Activity Score UAS7 (range 0–42; a reduction of ≥12 points was considered clinically significant) and the Urticaria Control Test UCT (sum score ≤11 — uncontrolled disease). Safety was assessed by the frequency of adverse events, including systemic and local reactions.

Results

In 28 out of 30 patients (93.3%), a decrease in urticaria activity (reduction in pruritus and number of wheals) was observed after the first injection of omalizumab. An early response (within 1–2 weeks) was seen in 22 children (73.3%), predominantly of preschool age, while a late response (4–6 weeks) was seen in 6 (20.0%). By 3 months of therapy, improvement in symptom control (median UCT 12.0 [10.0–16.0] points) was achieved in 25 children (83.3%). Complete disease control (UCT 16.0 [15.0–16.0] points) by 12 months of treatment was documented in 5 patients (16.7%). In all patients with complete control, it was possible to completely discontinue nsAH and systemic glucocorticosteroids, which had previously been used to control exacerbations. In 18 patients (60.0%), the need for nsAH was reduced by more than 50% compared to baseline. Total serum IgE levels exceeded reference values in 19 children (63.3%); however, no correlation was found between baseline IgE levels and the rate of response to therapy. Adverse events were reported in 3 patients (10%): headache (n=1), local reactions in

the form of pain and erythema at the injection site (n=2). No severe systemic reactions (anaphylaxis, angioedema, bronchospasm) were observed in any case. Therapy interruption due to side effects was not required.

Conclusions

Omalizumab in children with chronic urticaria resistant to standard antihistamine therapy demonstrates high clinical efficacy (improved symptom control in 83.3% by 3 months and complete control in 16.7% by 12 months), allows for the withdrawal or significant dose reduction of nsAH and systemic corticosteroids, and has a favorable safety profile without severe systemic reactions. These findings support its use in pediatric practice, including in preschool-aged children (off-label after approval by the hospital medical board/ethics committee).

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Extraintestinal manifestations in participants with moderately to severely active Crohn's disease: Results from the phase 3 GALAXI 2 & 3 studies

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Introduction

Extraintestinal manifestations (EIMs), including joint, skin, and ocular manifestations, are common in patients with Inflammatory Bowel Disease. The phase 3 GALAXI 2 & 3 studies evaluated guselkumab (GUS), a dual-acting Interleukin-23p19 subunit inhibitor, in participants with moderately to severely active Crohn's disease.[1,2] Here, we present EIMs reported through Week 48 in the pooled GALAXI 2/3 studies.

Materials and Methods

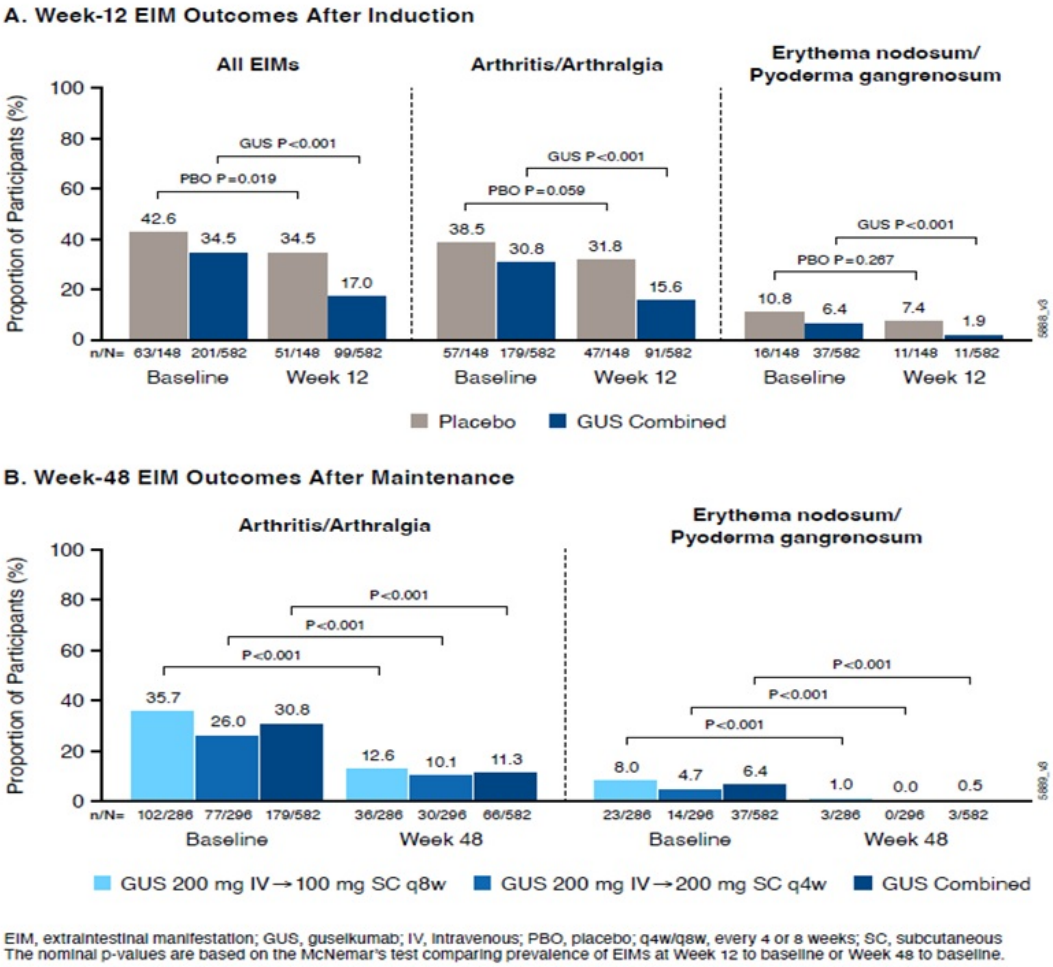
Participants were randomly assigned to GUS 200 mg intravenous at Week 0, 4, and 8, then GUS subcutaneous (SC) 100 mg every 8 weeks (q8w, N=286) or 200 mg every 4 weeks (q4w, N=296), or placebo (PBO, N=148). The studies also included ustekinumab, but the current analyses do not include ustekinumab. Corticosteroids were maintained at baseline doses through Week 12, when mandatory tapering began. EIMs (arthritis/arthralgia, erythema nodosum/pyoderma gangrenosum, and iritis/uveitis) were reported as a component of the Crohn's Disease Activity Index (CDAI) score at each visit along with individual EIM resolution and de novo EIMs at Week 12 and 48. EIM data were pooled for GALAXI 2/3.

Results

Overall, 34.5% (201/582) of GUS-treated and 42.6% (63/148) of PBO-treated participants had EIMs at baseline. Among participants with EIMs at baseline, arthritis/arthralgia was reported in 89.1% and 90.5% of GUS-treated and PBO-treated participants, respectively. Erythema nodosum/pyoderma gangrenosum was reported in 18.4% and 25.4% of GUS-treated and PBO-treated participants, respectively. Rates of iritis/uveitis were low, 5.5% and 3.2% in GUS-treated and PBO-treated participants, respectively, and were not evaluated due to small sample size. Overall, 14.4% (29/201) of GUS-treated and 17.5% (11/63) of PBO-treated participants with EIMs were receiving prednisone at baseline.

Figure A shows percentages of participants with EIMs at baseline and Week 12. Greater proportions of GUS-treated (59.2%) versus PBO-treated (42.9%) participants had resolution of their overall EIMs after induction (Week 12): arthritis/arthralgia (55.3% versus 33.3%) and erythema nodosum/pyoderma gangrenosum (75.7% versus 56.3%), respectively. Overall, rates of de novo EIMs at Week 12 were low, 2.6% and 8.1% in GUS-treated and PBO-treated participants, respectively. Greater improvements in weighted EIM CDAI component scores were reported from baseline through Week 12 (LS Means [SE]: −5.4 [0.43]) in the GUS-treated participants (both doses combined) versus PBO-treated participants (−0.70 [0.83]).

At Week 48, the proportions of participants with arthritis/arthralgia or erythema nodosum/pyoderma gangrenosum decreased from baseline in both GUS treatment groups (Figure B). LS Mean change [SE] from baseline in weighted EIM CDAI component scores at Week 48 were similar between the GUS 100 mg SC q8w (−5.5 [0.57]) and GUS 200 mg SC q4w (−6.1 [0.57]) groups.



EIM, extraintestinal manifestation; GUS, guselkumab; IV, intravenous; PBO, placebo; q4w/q8w, every 4 or 8 weeks; SC, subcutaneous
The nominal p-values are based on the McNemar's test comparing prevalence of EIMs at Week 12 to baseline or Week 48 to baseline.

Figure. A) Week-12 EIM outcomes after induction; B) Week-48 EIM outcomes after maintenance in the pooled GALAXI 2 & 3 studies

Conclusions

Participants treated with GUS versus PBO had greater EIM resolution at Week 12, and improvements continued to Week 48. Participants receiving GUS also had lower rates of de novo EIMs at Week 12 compared with participants receiving PBO. These results suggest GUS may improve and prevent EIMs in participants with Crohn's disease.

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Abstract N°: 2704

Successful Treatment of Generalized Pustular Psoriasis with Vunakizumab: A Case Series

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Introduction
Generalized pustular psoriasis (GPP) is a rare yet severe psoriasis subtype, clinically characterized by generalized or localized erythema and sterile pustules. Its pathogenesis is closely linked to epidermal recruitment of neutrophils and other inflammatory cells, induced by IL-36 binding to its receptor. Additionally, TNF-α/IL-17/IL-22 axis activation accelerates and exacerbates this inflammation, and crosstalk between IL-36 and TNF-α/IL-17/IL-22 axes jointly mediates GPP onset and progression. As a pivotal intermediate, IL-17 plays a crucial role in GPP pathogenesis. However, clinical data on IL-17 inhibitors for GPP treatment remain limited. Herein, we report five GPP patients with favorable outcomes after treatment with Vunakizumab, a humanized anti-IL-17A monoclonal antibody.

Case Presentation
Five patients with generalized pustular psoriasis (GPP) were enrolled. Demographic data, clinical history and comorbidities were documented. Patients aged <18 years received Vunakizumab 120 mg subcutaneously at weeks 0, 2, and 4 (induction phase), followed by 120 mg q4w (maintenance phase). Patients aged ≥18 years received a 240 mg dose following the same dosing schedule. Outcome measures included time to pustule clearance, Generalized Pustular Psoriasis Area and Severity Index (GPPASI) , Generalized Pustular Psoriasis Physician Global Assessment (GPPGA) , disease recurrence and adverse events.
After the first dose of Vunakizumab, all patients achieved rapid pustule drying, crusting, and regression within 2 weeks without any other treatment. By week 4, all patients achieved complete remission with 100% clearance of erythema and pustules.The GPPASI and GPPGA scores decreased to 0.Baseline Characterization and Key efficacy data are summarized in Table 1.

Case 1:The patient, a 15-year-old male, presented with generalized pustules for 10 days. The GPPASI score decreased by 88% at 2 weeks after the first injection. The treatment was discontinued after 4 months of maintenance, with no recurrence to date.

Case 2:The patient, a 11-year-old female, experienced rash worsening with generalized pustules 3 days after an upper respiratory tract infection. The GPPASI score decreased by 80% at 2 weeks after the first injection. The patient has remained on maintenance therapy with no recurrence to date.

Case 3:The patient had received two treatments with ustekinumab (an IL-12/23 inhibitor). 20 days after the second dose, new onset erythema and pustules appeared on the trunk and extremities, leading to a diagnosis of GPP. Two weeks after the first injection of vunakizumab, the GPPASI score decreased by 78%. The patient has remained on maintenance therapy with no recurrence to date.

Case 4:The patient presented with generalized pustules for 2 months. The GPPASI score decreased by 78% at 2 weeks after the first injection. The patient has remained on maintenance therapy with no recurrence to date.

Case 5:The patient presented with generalized pustules for 10 days. Medical history included erosive gastritis, chronic heart failure, and chronic obstructive pulmonary disease (COPD). The GPPASI score decreased by 73% at 2

weeks after the first injection. The patient has remained on maintenance therapy with no recurrence to date.

No severe adverse events, serious infections, allergic reactions, or treatment discontinuations were reported. No exacerbation of comorbidities (chronic renal failure, heart failure, COPD) was observed.

Baseline Characterization				Vunakizumab therapy				
Patient No./age,y/sex	Weight (kg)	Comorbidity	History of psoriasis (years)	Dose (mg)	Frequency	GPPASI Score	GPPGA Score	Total doses to date (mg)
1/15/ M	40	chronic renal failure	6	120	1st ,2nd, 3rd dose：2 weeks apart 4th-6th dose：1 month apart	Baseline:26.48 2weeks: 3.2 1month to the present: 0	Baseline: 4 2 weeks: 1 1month to the present: 0	720
2/11/ F	34	-	10	120	1st ,2nd, 3rd dose：2 weeks apart 4th-6th dose：1 month apart 7th to the present：1 and a half months apart	Baseline:25.35 2 weeks: 4.95 1month to the present: 0	Baseline: 4 2 weeks: 1 1month to the present: 0	1080
3/32/ F	48	-	23	240	1st ,2nd, 3rd dose：2 weeks apart 4th to the present：1 month apart	Baseline: 12.55 2 weeks:2.8 1month to the present: 0	Baseline: 2 2 weeks: 1 1month to the present: 0	1440
4/36/M	82	-	13	240		Baseline: 26.05 2 weeks:5.75 1month to the present :0	Baseline: 4 2 weeks: 1 1month to the present: 0	1440
5/54/M	65	Erosive gastritis, chronic heart failure, COPD	10	240		Baseline: 18.45 2 weeks:4.95 1month to the present :0	Baseline: 2 2 weeks: 1 1month to the Present : 0	1440

Table 1.Baseline Characterization and Key efficacy data

Conclusion

Vunakizumab, a humanized anti-IL-17A monoclonal antibody, rapidly induced clinical remission in patients with GPP. It was well tolerated without severe adverse events, even in patients with significant comorbidities. Vunakizumab represents an effective and safe targeted option for GPP.

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Abstract N°: 2707

Efficacy and safety of vunakizumab in the treatment of non-common subtypes of psoriasis: A case report of three Patients

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Introduction

Introduction & Objectives

Psoriasis is an immune-mediated, chronic, relapsing inflammatory skin disease. Besides classic plaque psoriasis, uncommon subtypes including erythrodermic psoriasis (EP), generalized pustular psoriasis (GPP), and palmoplantar pustulosis (PPP) are characterized by more severe clinical conditions and greatly impair quality of life. These patients frequently exhibit inadequate responses, high relapse rates, or poor tolerability to traditional systemic therapies (e.g., cyclosporine, acitretin), representing a significant unmet clinical need. Vunakizumab, a novel anti-interleukin-17A (IL-17A) monoclonal antibody, has demonstrated favorable efficacy and safety in phase III clinical trials for moderate-to-severe plaque psoriasis [1]. This study aimed to evaluate the efficacy and safety of vunakizumab in refractory uncommon subtypes of psoriasis.

Materials & Methods

We retrospectively enrolled 3 patients with non-common subtypes of psoriasis, including EP, GPP and PPP. All patients received subcutaneous injection of vunakizumab 240 mg at weeks 0, 2, and 4 for induction, followed by 240 mg every 4 weeks for maintenance. Efficacy was assessed by PASI and DLQI at baseline, week 4, 8 and 12. Adverse events (AEs) were monitored throughout treatment.

Results

All three patients achieved rapid and marked improvement in skin lesions following vunakizumab treatment. At Week 4, treatment responses reached PASI 75 for EP, GPPASI 90 for GPP and PPPASI 50 for PPP. At Week 12, responses improved further to PASI 90 for EP, GPPASI 100 for GPP and PPPASI 75 for PPP. DLQI scores decreased significantly: in the EP patient from 13 to 1, in the GPP patient from 11 to 1, and in the PPP patient from 11 to 2 at Week 12.

Keywords: erythrodermic psoriasis; generalized pustular psoriasis; palmoplantar pustulosis; vunakizumab; interleukin-17A

References

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Case Presentation

Patient 1 Generalized pustular psoriasis

A 27-year-old female presented with 4-day erythema and dense pustules on face, neck and upper trunk, preceded by upper respiratory infection. Previous antihistamines and topical corticosteroids were ineffective. The patient had a family history of psoriasis. Histopathology confirmed GPP. She was treated with vunakizumab and showed markedly improvement.

Patient 2 Erythrodermic psoriasis

A 57-year-old male presented with generalized erythema and scaling for more than 4 months. Previous topical corticosteroids, Tripterygium wilfordii and systemic corticosteroids showed limited efficacy with rapid relapse. Histopathological findings were consistent with erythrodermic psoriasis. He was treated with vunakizumab, resulting in significant improvement of skin lesions.

Patient 3 Palmoplantar pustulosis

A 23-year-old male presented with erythema, hyperkeratosis, pustules, scaling, and pruritus involving the bilateral palms and soles for 3 months. Prior topical corticosteroids, oral tofacitinib (5 mg twice daily) and deucravacitinib (6 mg once daily) showed inadequate efficacy. He was switched to vunakizumab and achieved obvious improvement of skin and nail lesions.





Patient 2
Baseline



Week 4



Week 12



Patient3
Baseline



Week 4





Week 12



Conclusion

Vunakizumab demonstrates the advantages of rapid onset, marked efficacy, and a favorable safety profile in non-common subtypes of psoriasis with poor response to traditional therapy and tofacitinib, offering an effective therapeutic option for clinically uncommon subtypes of psoriasis.

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Abstract N°: 2709

Consistency of Skin Clearance With the Targeted Oral Peptide Icotrokinra: Results From a Large Pooled Cohort of Phase 3 Study Participants With Moderate-to-Severe Plaque Psoriasis

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Introduction
ICONIC-LEAD (NCT06095115), ICONIC-ADVANCE1 (NCT06143878), and ICONIC-ADVANCE2 (NCT06220604) are evaluating the targeted oral peptide icotrokinra (ICO) for treatment of moderate-to-severe plaque psoriasis (PsO). Within each study, co-primary endpoints were met and participants achieved significantly higher rates of skin clearance with ICO versus placebo (PBO). This pooled analysis assessed skin response across baseline subgroups of interest.

Materials and Methods
Participants (≥12 years LEAD; ≥18 years ADVANCE 1 & 2) with moderate-to-severe plaque PsO (body surface area ≥10%; Psoriasis Area and Severity Index [PASI] ≥12; Investigator's Global Assessment [IGA] ≥3) were randomized (2:1 LEAD; 2:1 ADVANCE 1; 4:1 ADVANCE 2) to once-daily oral ICO 200 mg or PBO. This Week (W)16 analysis assessed proportion difference in IGA 0/1 response across sex, age (<18/18–44/45–64/≥65), body weight (BW; ≤/>90 kg), body mass index (BMI; <25/25–29/≥30 kg/m^2), PASI (</≥20), IGA (≤3/4), and PsO treatment history (biologics/systemics) subgroups, and least squares mean (LSM) difference in PASI improvement (%) from baseline in BMI and BW quartile (≤72/>72–85/>85–98.7/>98.7 kg) subgroups.

Results
Pooled efficacy analyses included 1,087 ICO- and 465 PBO-treated participants. ICO demonstrated higher IGA 0/1 response rates versus PBO at W16 (67.6% versus 9.2%); treatment effect (proportion difference: 58.0% [95%CI: 53.8–61.7%]) was consistent across subgroups (range: 51.8–64.0%). ICO also provided substantially greater PASI percentage improvements versus PBO at W16 (81.9% versus 25.8%); treatment effect (LSM difference: 56.2% [95%CI :52.9–59.5%]) was consistent across BMI and BW quartile subgroups (range: 49.2–60.3%).

Conclusions

In a large pooled-cohort of participants with moderate-to-severe plaque PsO, the robust effects of the targeted oral peptide ICO on skin clearance were consistent regardless of baseline PsO severity, sex, age, weight, or prior treatment.

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Abstract N°: 2724

The efficacy of combination therapy with the IL-17 inhibitor netakimab and a dual GIP and GLP-1 receptor agonist in patients with psoriasis and metabolic syndrome

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Introduction

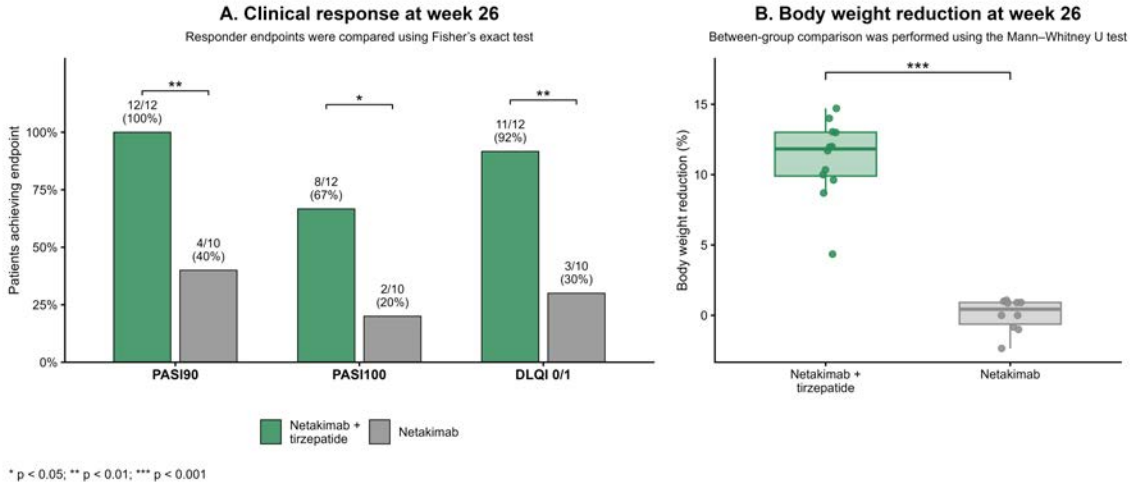
Despite the high efficacy of biologic therapies in psoriasis, the presence of metabolic comorbidities associated with systemic inflammation may reduce the likelihood of achieving a clinically meaningful response, highlighting the need for combined therapeutic strategies.

Materials and Methods

The study included 22 adult patients (≥ 18 years) with severe psoriasis and metabolic syndrome. Group 1 (n=12) received combination therapy: an IL-17a inhibitor (netakimab) and a dual GIP/GLP-1 receptor agonist (tirzepatide); Group 2 (n=10) received monotherapy with netakimab. Baseline demographic and clinical characteristics, including sex, age, height, waist circumference, body mass index, and baseline psoriasis severity, were comparable between groups. Baseline median BMI was 31.0 [30.0; 33.6] kg/m^2 in the combination therapy group and 31.7 [30.7; 33.4] kg/m^2 in the netakimab monotherapy group. The median PASI was 35.9 [19.3; 40.5] and 36.0 [16.0; 40.0], and the median DLQI was 25.0 [22.5; 27.5] and 24.5 [21.0; 26.0]. Efficacy was assessed at week 26 of treatment. The primary endpoint of the study was the proportion of patients achieving PASI 90 at week 26. Secondary endpoints included the proportion of patients achieving PASI 100 and DLQI 0/1, and DLQI ≤ 3 at week 26.

Results

By week 26, both groups demonstrated a statistically significant reduction in PASI. In the combination therapy group, median PASI decreased from 35.9 [21.1; 40.2] to 0 [0; 1], and DLQI from 25.0 [22.8; 27.2] to 0 [0; 0.2] (both p = 0.002). In the netakimab monotherapy group, the corresponding values decreased from 36.0 [17.0; 40.0] to 4.5 [3.0; 5.8] for PASI and from 24.5 [21.0; 25.8] to 3.0 [1.5; 3.8] for DLQI (both p = 0.005). This indicates a high therapeutic response in both groups. The relative reduction in PASI was significantly greater in the combination therapy group compared with netakimab monotherapy: 100% versus 83.8% (p = 0.003). The most pronounced between-group difference was observed in body weight change. Body weight decreased significantly in patients receiving combination therapy (p = 0.002), whereas no significant change was observed in the netakimab monotherapy group (p = 0.8). The percentage reduction in body weight was 11.8% [9.8; 13.0] versus 0.4% [-0.8; 0.9] (p < 0.001). The proportion of patients achieving PASI 90, PASI 100, and DLQI 0/1 was higher in the combination therapy group than in the monotherapy group: 100% (12/12) versus 40% (4/10), (p = 0.002), 66.7% (8/12) versus 20% (2/10), (p = 0.04), 92% (11/12) versus 30% (3/10), (p = 0.006), respectively. The percentage of patients achieving a DLQI ≤3 was 100% (12/12) versus 70% (7/10) (p = 0.07).



Clinical response (A) and body weight reduction (B) at week 26

Conclusions

Achieving complete control of the psoriatic process (PASI 0, DLQI 0/1) in patients with moderate-to-severe psoriasis and metabolic syndrome presents significant challenges, making a comprehensive approach essential. Combination therapy with the IL-17a inhibitor netakimab and tirzepatide was associated with significantly higher rates of achieving PASI 90, PASI 100, and DLQI 0/1 compared to monotherapy with netakimab. A dual GIP/GLP-1 receptor agonist, due to its pleiotropic effects, represents a promising treatment approach for patients with psoriasis and metabolic syndrome when combined with biologics.

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Omalizumab for Recalcitrant Morbihan Syndrome: A Case Highlighting Dose–Response Considerations

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Introduction

Morbihan syndrome is a rare, chronic condition characterised by persistent, firm facial oedema and is often refractory to standard rosacea therapies, although it may occur in the absence of other rosacea features.

Case Presentation

We report a 57-year-old woman with long-standing, recalcitrant Morbihan syndrome causing severe periorbital oedema with functional visual impairment. She failed multiple conventional therapies, including isotretinoin, antihistamines, leukotriene antagonists, and lymphatic massage. Tumour necrosis factor-α inhibition was trialled but discontinued due to recurrent infections. Omalizumab was initiated based on limited published case experience, using a loading dose of 300 mg followed by 300 mg every four weeks. At four months, facial erythema improved, while oedema demonstrated only mild response. Treatment was well tolerated without significant adverse events.

Conclusion

Omalizumab may exert benefit in Morbihan syndrome through inhibition of IgE-mediated mast cell activation, a proposed driver of oedema, lymphatic dysfunction, and progressive fibrosis. Published reports describe variable responses, with some suggesting improved outcomes using higher-dose regimens.

This case contributes to the limited literature supporting omalizumab as a therapeutic option for recalcitrant Morbihan syndrome and highlights the potential importance of dose escalation in patients with incomplete oedema response. Further studies are required to clarify optimal dosing strategies and predictors of response.

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Abstract N°: 2739

APEX: Guselkumab Response and Inhibition of Structural Damage Progression in Active Psoriatic Arthritis by Participant Baseline Characteristics

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Introduction
The ongoing phase 3b APEX study in participants with active and erosive psoriatic arthritis (PsA) met its primary endpoint, demonstrating significantly higher American College of Rheumatology ≥20% improvement (ACR20) response rates at Week (W)24 with guselkumab (GUS) versus placebo, and showed GUS provides significant inhibition of structural damage progression. Here, subgroup analyses evaluated treatment consistency in key efficacy outcomes by participant baseline characteristics.

Materials and Methods
APEX evaluated biologic-naïve adults with active PsA (swollen joint count ≥3; tender joint count ≥3; C-reactive protein ≥0.3mg/dL) and ≥2 erosive joints on hand/foot radiographs, despite previous non-biologic treatment. Randomized participants received GUS every 4 weeks (Q4W); GUS at Week 0, Week 4, then every 8 weeks (Q8W); or placebo Q4W. Primary and other efficacy endpoints were evaluated in predefined subgroups defined by baseline demographic and disease characteristics.

Results
At Week 24 in the GUS Q4W (N=273), GUS Q8W (N=371), and placebo (N=376) groups, respectively, response rates were ACR20: 66.6%, 68.3%, 47.0%; ACR50, 41.4%, 42.2%, 20.5%; Psoriasis Area and Severity Index (PASI) 90: 69.4%, 60.0%, 22.0%; minimal disease activity: 29.6%, 27.7%, 13.7%. Improvements across endpoints were consistent in GUS-treated subgroups defined by sex, body mass index, PsA disease characteristics, and concomitant methotrexate use. Inhibition of radiographic progression was also generally consistent across GUS-treated subgroups defined by baseline characteristics and radiographic features.

Conclusions

In biologic-naïve patients with active, erosive PsA, GUS provided significantly greater joint and skin improvements versus placebo at Week 24 and is the only interleukin-23 inhibitor shown to provide significant inhibition of structural damage progression. Treatment effects were generally consistent across subgroups defined by baseline characteristics and key radiographic features.

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Cutaneous toxicity from antibody-drug conjugates (ADCs): a real-world cohort of 499 patients involving 14 agents

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Introduction
Antibody-drug conjugates (ADCs) are a rapidly expanding treatment across multiple oncological disease groups. Cutaneous toxicity is common but often incompletely characterised across agents, payload classes and patient populations. Real-world data describing incidence, predictors and subtype profiles to support patients and clinicians to make informed choices is currently limited.

Materials and Methods
This single-centre retrospective cohort study included all patients who received at least one dose of ADC between March 2023 and April 2025. Cutaneous toxicity was retrospectively graded according to CTCAE v5.0 from electronic clinical records by two clinicians independently, with discrepancies resolved by consensus. Descriptive statistics, logistic regression and Fisher's exact test were used to describe and identify independent associations with cutaneous toxicity.

Results
Incidence of cutaneous toxicity was 197/499 (39.5%, 95% CI 35.3–43.8%), largely made up with grade 1 66% followed by grade 2 26.9% and grade 3 7.1%. Treatment interruption occurred in 1.6% and cessation 0.4%. Payload was overall significantly associated with cutaneous toxicity (overall Fisher p=0.011); with MMAF having significantly lower rates than all others (vs MMAE OR 0.24 p=0.002, vs DXd OR 0.24 p=0.003, vs SN-38 OR 0.19 p=0.001), consistent with linker cleavability as a novel determinant. Backbone chemotherapy agent was an independent predictor of toxicity, highlighted when comparing polatuzumab vedotin with R-CHP vs R-bendamustine (43.5% vs 9.1%, OR 7.56, 95% CI 2.05–42.37, p<0.001), despite identical molecule and payload.

The target antigen component of ADC was significantly associated overall, with Trop-2 OR 2.01, p=0.008 and CD30 OR 2.03 p=0.031, showing a higher risk of toxicity and BCMA a lower risk OR 0.30 p=0.017 vs HER2, potentially protective. Multivariate analysis highlighted thyroid dysfunction as an independent predictor of toxicity (OR 2.86, 95% CI 1.43–5.72, p=0.003). Multiple concurrent toxicities independently predict grade ≥2 severity (OR 2.16, 95% CI 1.07–4.35, p=0.031). Earlier onset was associated with higher grade toxicity (grade ≥2 median 28 days vs grade 1 median 57 days, p=0.006).

Conclusions
The largest real-world ADC-associated cutaneous toxicity cohort to our knowledge identified that payload class, antigen and chemotherapy backbone were key determinants of ADC-associated toxicity risk. Thyroid dysfunction was identified as a novel independent predictor, and earlier onset

was associated with higher-grade severity. These findings provide a framework for risk-stratified surveillance in patients receiving ADC therapy.

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Abstract N°: 2788

Dupilumab Provides Sustained Improvements in Itch, Lesions, and Quality of Life in Patients With Prurigo Nodularis

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Introduction

Relief from pruritus, healing of skin lesions, and improved quality of life (QoL) represent the primary therapeutic goals for patients with prurigo nodularis (PN, or nodular prurigo), the most common subtype of chronic prurigo. Understanding the efficacy of dupilumab treatment may help to align patient and caregiver expectations and improve treatment compliance. This post hoc analysis aims to assess the stability of clinical improvements in itch, lesions, and QoL of patients with moderate-to-severe PN receiving dupilumab.

Materials and Methods

In the PRIME/PRIME2 trials (NCT04183335/NCT04202679) 152 adult patients with PN whose disease was inadequately controlled with topical prescription therapies, or for whom those therapies were inadvisable, received dupilumab 300 mg every 2 weeks for 24 weeks. The number of patients who achieved at least a 4-point improvement in Worst-Itch Numeric Rating Scale (WI-NRS) score, an Investigator's Global Assessment of PN Stage (IGA PN-S) ≤1, or a 9-point improvement in Dermatology Life Quality Index (DLQI) score was assessed at Weeks 4, 8, and 12. Maintenance of ≥4- or ≥3-point improvement in WI-NRS, IGA PN-S ≤1 or ≤2, and ≥9-point improvement in DLQI score among responders was assessed at Week 24.

Results

A ≥4-point improvement in WI-NRS score was achieved by 27, 48, and 65 (17.8%, 31,6%, 42.8%) patients at Weeks 4, 8, and 12, respectively. IGA PN-S ≤1 was observed in 13, 24, and 45 (8.6%, 15.8%, 29.6%) patients at Weeks 4, 8, and 12, respectively. A ≥9-point improvement in DLQI score was achieved by 62, 85, and 97 (40.8%, 55.9%, 63.8%) patients at Weeks 4, 8, and 12, respectively. By study end, regardless of when improvements were first observed, >95% or >96% of responders maintained WI-NRS ≥4- or ≥3-point improvement, respectively, >77% or >92% of responders maintained IGA PN-S ≤1 or ≤2, respectively, and >87% of responders maintained DLQI ≥9-point improvement. Overall, safety was generally consistent with the known dupilumab safety profile.

Conclusions

Most patients who achieved clinically significant control of itch, lesions, and QoL improvements following dupilumab treatment were able to maintain these improvements by study end.

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Abstract N°: 2790

Sustained Multi-Domain Benefits With Dupilumab in Prurigo Nodularis: Maintenance of Itch, Lesion, and Quality-of-Life Improvements

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Introduction
Prurigo nodularis (PN, or nodular prurigo), the most common subtype of chronic prurigo, is a chronic inflammatory skin disease characterized by intensely pruritic nodular lesions lasting more than 6 weeks and impaired quality of life (QoL). Effective treatment approaches for PN require sustained disease control across all aspects of the disease, including durable improvements in itch, lesions, and QoL. This post hoc analysis aims to assess the maintenance of response to dupilumab on itch, lesions, and QoL improvements in patients with moderate-to-severe PN.

Materials and Methods
In PRIME/PRIME2 (NCT04183335/NCT04202679), 152 adult patients with PN whose disease was inadequately controlled with topical prescription therapies or for whom those therapies were inadvisable, received dupilumab 300 mg every 2 weeks for 24 weeks. The proportions of patients who achieved at least a 3- or 4-point improvement in Worst Itch Numeric Rating Scale (WI-NRS), a score of ≤1 or ≤2 in Investigator's Global Assessment of PN Stage (IGA PN-S), or a 4- or 9-point improvement in Dermatology Life Quality Index (DLQI) were assessed at every follow-up visit from Week 4 through Week 24.

Results
By Week four, 27 (17.8%) and 37 (24.3%) patients achieved at least a 3- or 4-point improvement in WI-NRS, respectively, increasing to 88 (57.9%) and 65 (42.8%) patients by Week 12. Similarly, 32 (21.1%) and 13 (8.6%) patients achieved IGA PN-S ≤1 or ≤2 at Week 4, respectively, rising to 82 (53.9%) and 45 (29.6%) patients by Week 12. A total of 110 (72.4%) and 62 (40.8%) patients achieved at least a 4- or 9-point improvement in DLQI at Week 4, respectively, increasing to 128 (84.2%) and 97 (63.8%) patients by Week 12. Among Week 4 responders, most sustained their response to dupilumab in itch (63.0%–67.6%), lesions (61.5%–87.5%), and QoL (82.3%–87.3%) at every visit through Week 24. Among Week 12 responders, improvements in itch (>81.5%), lesions (

>91.1%), and QoL (>89.7%) were maintained at every subsequent visit through Week 24. Overall, safety was generally consistent with the known dupilumab safety profile.

Conclusions

Regardless of the time of onset, the majority of responders maintained improvements in itch, lesion, and QoL through Week 24, supporting dupilumab's sustained disease control in moderate-to-severe PN.

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Bimekizumab efficacy & safety versus risankizumab in patients with active psoriatic arthritis: 16-week results from a head-to-head, multicentre, randomised, phase 3b study (BE BOLD)

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Introduction

Bimekizumab (BKZ), an IL-17A/F inhibitor and risankizumab (RZB), an IL-23 inhibitor, are approved treatments in psoriatic arthritis (PsA). Here, we report head-to-head efficacy and safety of BKZ and RZB at approved doses in patients (pts) with active PsA to 24 weeks (wks).

Materials and Methods

BE BOLD (NCT06624228) was a 24-wk, double-blinded, phase 3b study in pts aged ≥18 with active PsA who were biologic-naïve or had inadequate response/intolerance to one prior TNF inhibitor.

Pts were randomised 1:1 to BKZ or RZB up to 24 wks at approved doses (BKZ dosing based on baseline [BL] psoriasis [PSO] severity, per EMA/FDA labels). Most BKZ pts received 160 mg Q4W (final dose at Wk20); pts with PsA and concomitant moderate-to-severe PSO (body surface area [BSA] ≥10%, Investigator's Global Assessment ≥3, and Psoriasis Area and Severity Index [PASI] ≥12) received BKZ 320 mg (Q4W to Wk16 then 320 mg Q8W). All RZB pts received 150 mg (BL, Wk4 then Q12W). The final dose of BKZ 320 mg and RZB 150 mg was at Wk16.

Primary endpoint: ≥50% improvement from BL in American College of Rheumatology response criteria (ACR50) at Wk16 (noninferiority and superiority; sequentially tested). Secondary endpoints (sequentially tested, superiority): minimal disease activity (MDA) at Wk16, ACR50+PASI100 (100% improvement from BL in PASI; pts with BL BSA ≥3%) at Wk16, and ACR50 at Wk4; reported to Wk24. Additional outcomes to Wk24: PASI100 (pts

with BL BSA ≥3%), and Disease Activity Index for Psoriatic Arthritis (DAPSA) low disease activity or remission (LDA+REM; score ≤14). Missing data were imputed using non-responder imputation. Safety data reported to end of trial (Wk24 plus safety follow-up).

Table 1. Baseline Disease Characteristics

	BKZ[a] N=277	RZB[b] N=276
Age, years, mean (SD)	51.1 (13.6)	50.7 (12.7)
Sex, male, n (%)	141 (50.9)	142 (51.4)
BMI, kg/m², mean (SD)	29.1 (6.1)	29.2 (6.4)
TJC, mean (SD)	17.3 (12.7)	16.6 (12.2)
SJC, mean (SD)	10.2 (7.4)	9.5 (6.8)
PtGA-PsA, mean (SD)	59.8 (21.0)	58.5 (20.3)
Moderate to severe psoriasis, n (%)[c]	29 (10.5)	31 (11.2)
Psoriasis BSA, n (%) <3% ≥3 to ≤10% >10%	 100 (36.1) 114 (41.2) 63 (22.7)	 100 (36.2) 108 (39.1) 68 (24.6)
PASI, mean (SD)[d]	8.9 (7.5)	8.2 (7.1)
hs-CRP ≥6 mg/L, n (%)	84 (30.3)	83 (30.1)
Prior TNFi, n (%)	55 (19.9)	58 (21.0)
Any csDMARDs at baseline, n (%)[e]	191 (69.0)	195 (70.7)
Enthesitis, LEI >0, n (%)	131 (47.3)	133 (48.2)[f]
Dactylitis, LDI >0, n (%)	45 (16.2)	41 (14.9)[f]

Randomised set. [a] BKZ 160 mg Q4W and BKZ 320 mg Q4W/Q8W; [b] RZB 150 mg at baseline, Week 4 and Week 16; [c] Moderate-to-severe psoriasis defined as BSA ≥10%, IGA ≥3, and PASI ≥12; [d] In patients with baseline psoriasis ≥3% BSA: BKZ Total N=177 (BKZ 160 mg Q4W n=148; BKZ 320 mg Q4W/Q8W n=29); RZB n=176; [e] Includes methotrexate, sulfasalazine and leflunomide; [f] Data missing for 1 patient. Abbreviations: BKZ: bimekizumab; BMI: body mass index; BSA: body surface area; csDMARD: conventional synthetic disease-modifying antirheumatic drug; hs-CRP: high-sensitivity C-reactive protein; IGA: Investigator's Global Assessment; LDI: Leeds Dactylitis Index; LEI: Leeds Enthesitis Index; PASI: Psoriasis Area and Severity Index; PtGA-PsA: Patient's Global Assessment for Psoriatic Arthritis; Q4/8W: every 4/8 weeks; RZB: risankizumab; SD: standard deviation; SJC: swollen joint count; TJC: tender joint count; TNFi: tumour necrosis factor inhibitor.

Results

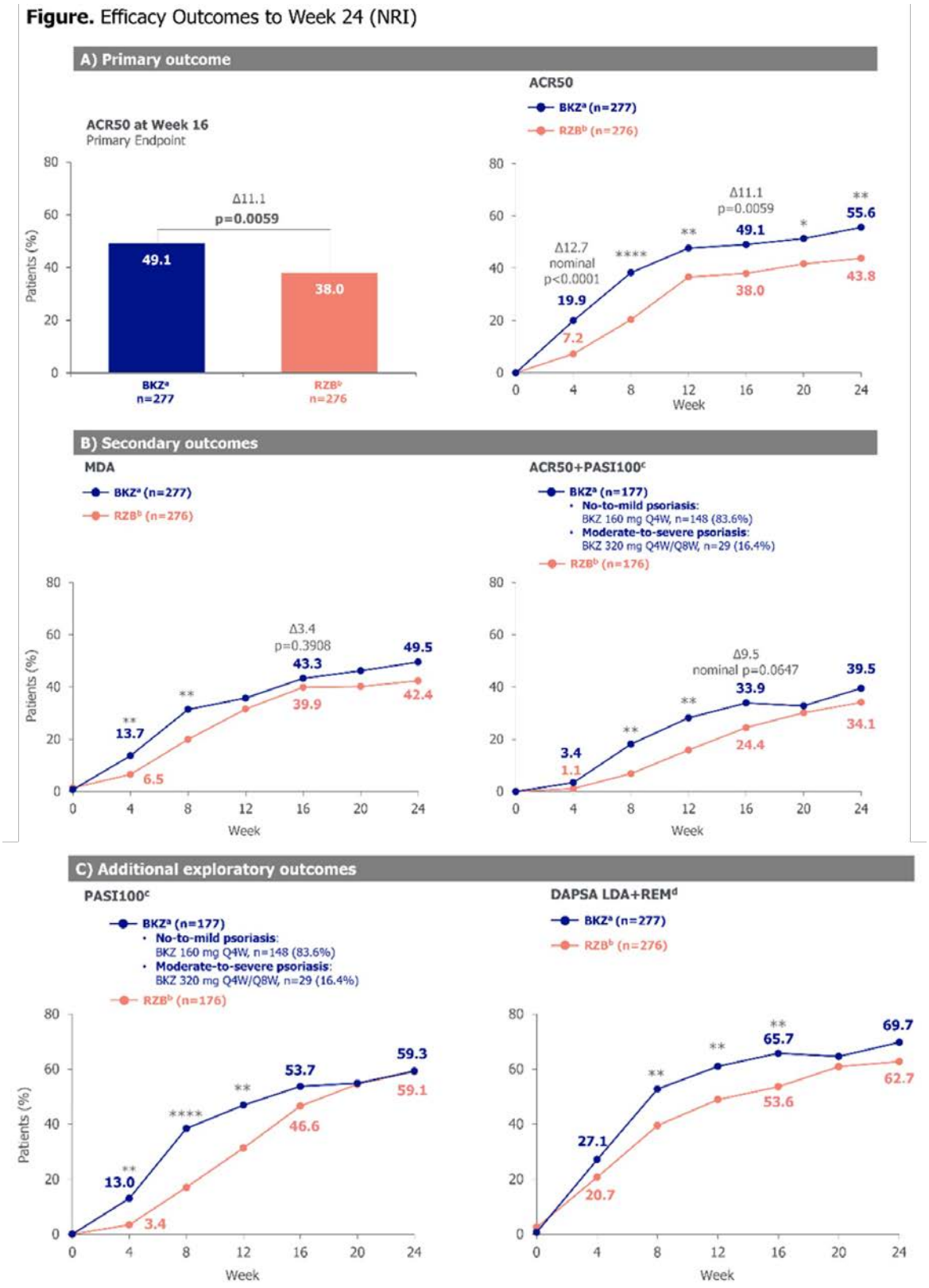
Of 553 randomised pts, 277 received BKZ, with 248 (89.5%) receiving BKZ 160 mg Q4W (no-to-mild PSO), 29 (10.5%) receiving BKZ 320 mg Q4W/Q8W (moderate-to-severe PSO), and 276 receiving RZB. 259 (93.5%) BKZ and 248 (89.9%) RZB pts completed Wk24. BL characteristics were comparable between groups (**Table 1**).

BKZ was superior to RZB in Wk16 ACR50 (49.1% vs 38.0%; p=0.0059), sustained to Wk24 (nominal p=0.0037), with greater response as early as Wk4 (nominal p<0.0001; **Fig**). The first ranked secondary endpoint (MDA to Wk16) was numerically higher (p=0.3908; sequential testing stopped at this point) and sustained to Wk24. Higher proportions of BKZ pts achieved ACR50+PASI100 to Wk16 (33.9% vs 24.4%; nominal p=0.0647), sustained to

Wk24. Numerically higher proportions of BKZ pts achieved PASI100 to Wk16 and DAPSA LDA+REM to Wk24 compared with RZB pts (**Fig**).

At end of trial, 260/277 (93.9%) BKZ and 254/276 (92.0%) RZB pts completed safety follow-up. 161/277 (58.1%) BKZ and 153/275 (55.6%) RZB pts had ≥1 TEAE; discontinuations due to TEAEs were low (**Table 2**). Serious/severe TEAEs occurred in 5 (1.8%)/5 (1.8%) BKZ and 9 (3.3%)/5 (1.8%) RZB pts. One death occurred in a pt randomised to RZB, deemed unrelated to treatment. *Candida* infections were more frequent with BKZ; all mild/moderate, none were serious, systemic or led to discontinuation. Cases of serious infection, inflammatory bowel disease, malignancy, neutropenia, hypersensitivity, hepatic and cardiovascular events were low across groups; no reported suicidal ideation and behaviour, anaphylaxis or active tuberculosis.

Figure. Efficacy Outcomes to Week 24 (NRI)



Randomised set. Pre-specified p values and differences between BKZ and RZB results are displayed for comparisons in the statistical testing hierarchy only. Other nominal p values for BKZ vs RZB: *p<0.05; **p<0.01; ***p<0.001; ****p<0.0001. [a] BKZ 160 mg Q4W and BKZ 320 mg Q4W/Q8W; [b] RZB 150 mg at baseline, Week 4 and Week 16; [c] In patients with baseline psoriasis ≥3% BSA; [d] DAPSA LDA+REM is defined as a DAPSA score of ≤14. Abbreviations. ACR50: ≥50% improvement from baseline in American College of Rheumatology response criteria; BKZ: bimekizumab; BSA: body surface area; DAPSA: Disease Activity Index for Psoriatic Arthritis; LDA: low disease activity; MDA: minimal disease activity; NRI: non-responder imputation; PASI100: 100% improvement from baseline in Psoriasis Area and Severity Index; Q4/8W: every 4/8 weeks; REM: remission; RZB: risankizumab.

Conclusions

Dual inhibition of IL-17A and IL-17F with BKZ resulted in superiority for the primary endpoint of ACR50 at Wk16 vs IL-23 inhibition with RZB, with a >10% difference, in pts with PsA. A numerically higher proportion of BKZ pts achieved key secondary joint, skin and overall disease activity outcomes vs RZB pts to Wk24. No new safety signals were identified; safety profiles were comparable between treatment arms, except for *Candida* infections which were more frequent for BKZ, as anticipated by the mechanism of action. These findings may help guide treatment decisions and inform recommendations which consider the efficacy and mechanism of action of BKZ and RZB.

Table 2. Safety to End of Trial

n (%)	BKZ[a] N=277; 164.9 PY	RZB[b] N=275; 162.1 PY
Any TEAE	161 (58.1)	153 (55.6)
Serious TEAEs	5 (1.8)	9 (3.3)
Study discontinuations due to TEAEs	4 (1.4)	3 (1.1)
Severe TEAEs	5 (1.8)	5 (1.8)
Drug-related TEAEs	42 (15.2)	31 (11.3)
Deaths	0	1 (0.4)[c]
Most common TEAEs[d]		
Nasopharyngitis	33 (11.9)	26 (9.5)
Upper respiratory tract infection	15 (5.4)	7 (2.5)
Safety topics of interest		
Serious infections	2 (0.7)	1 (0.4)
Opportunistic infections	0	1 (0.4)
Fungal infections	16 (5.8)	4 (1.5)
Candida infections	8 (2.9)	2 (0.7)
Oral candidiasis	7 (2.5)	2 (0.7)
Malignancies	1 (0.4)	2 (0.7)
Neutropenia	3 (1.1)	0
Anaphylactic reaction	0	0
Hypersensitivity	8 (2.9)	2 (0.7)
Injection site reaction	4 (1.4)	2 (0.7)
Inflammatory bowel disease	1 (0.4)[e]	0
Suicidal ideation and behaviour	0	0
Cardiovascular events	1 (0.4)	3 (1.1)
Hepatic events	12 (4.3)	9 (3.3)
Elevated liver enzymes[f]	9 (3.2)	9 (3.3)

Safety set. Safety is reported until end of trial, including the 12-week safety follow-up period; as participants were permitted to use any other medications once 28 days had passed from their final study dose, participants may have continued or discontinued the same treatment or switched to another treatment within the safety follow-up period. TEAEs were classified using the Medical Dictionary for Regulatory Activities v27.0. [a] BKZ 160 mg Q4W and BKZ 320 mg Q4W/Q8W; [b] RZB 150 mg at baseline, Week 4, and Week 16; [c] Patient without moderate-to-severe psoriasis. Deemed unrelated to treatment by the Investigator, with a reported cause of myocardial infarction in a patient with coronary artery disease, hypertension and hyperlipidaemia; [d] TEAEs occurring in >5% of either treatment arm; [e] A flare of Crohn's disease occurred in a patient with a history of Crohn's' disease; [f] Elevated liver enzymes include the following preferred terms reported as adverse events: increased/abnormal levels of alanine aminotransferase, aspartate aminotransferase, blood bilirubin, gamma glutamyltransferase, hepatic enzyme, liver function test, total bile acids or transaminases. Abbreviations: BKZ: bimekizumab; PY: patient-year; Q4/8W: every 4/8 weeks; RZB: risankizumab; TEAE: treatment-emergent adverse event.

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Abstract N°: 2832

Omalizumab for refractory eosinophilic annular erythema: first reported case and rationale for anti-IgE therapy

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Introduction

Eosinophilic annular erythema (EAE) is an uncommon eosinophilic dermatosis characterized by relapsing pruritic annular erythematous plaques with central pigmentation, and histologically by a perivascular infiltrate of lymphocytes and eosinophils without flame figures, and by a basal epidermal hyperpigmentation. The therapeutic landscape relies on case reports and small series, with no established algorithm. While corticosteroids, hydroxychloroquine, and dapsone remain first-line options, remission is often short-lived. Recent reports highlight the potential of biologics blocking IL-4/IL-13 or IL-5 signaling in treatment-resistant cases. The use of omalizumab, an anti-IgE monoclonal antibody, has not been previously described in this condition.

Case Presentation

An 18-year-old man with a personal and family history of atopy developed recurrent episodes of pruritic annular erythematous plaques over the trunk and proximal limbs following the second dose of the Moderna mRNA-1273 vaccine. Symptomatic dermographism was concurrently present. Skin biopsy revealed a perivascular infiltrate composed of lymphocytes and eosinophils in the superficial and mid dermis without flame figures; Masson Fontana staining highlighted basal epidermal hyperpigmentation, a characteristic finding of EAE. Total IgE was elevated at 177 IU/mL; complete blood count, peripheral eosinophils, and Borrelia serology were unremarkable. Sequential trials of antihistamines, topical corticosteroids, and hydroxychloroquine were unsuccessful. The patient's atopic background, raised IgE, and concurrent dermographism led to initiation of omalizumab 300 mg subcutaneously every four weeks. Skin lesions resolved completely by month four, with sustained remission at twelve months and no treatment-related adverse events.

Conclusion

EAE and Wells syndrome represent overlapping entities within the spectrum of eosinophilic dermatoses. The mechanistic rationale for omalizumab in eosinophilic skin conditions rests on IgE-mediated mast cell and basophil activation, FcεRI upregulation on tissue eosinophils, and downstream Th2 cytokine modulation. Successful outcomes with omalizumab in Wells syndrome have been reported by several groups, with mast cell activation central to the proposed mechanism. Our case extends this rationale to EAE, where the co-occurrence of symptomatic dermographism and elevated IgE in an atopic patient strengthened the therapeutic indication.

To our knowledge, this is the first documented case of omalizumab use in EAE, resulting in complete and sustained remission. Omalizumab represents a promising, well-tolerated alternative for refractory EAE in patients with an IgE-driven phenotype, warranting confirmation in further cases.

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Abstract N°: 2834

Dupi Next? Real-World Outcomes of Second- and Third-Line Systemic Therapy After Dupilumab in Pediatric Atopic Dermatitis

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Introduction

Dupilumab is the first systemic biologic for moderate-to-severe pediatric atopic dermatitis (AD), approved from 6 months of age. A subset of children fails to achieve adequate disease control or loses response over time. Several alternative systemic agents approved for AD, including the IL-13 inhibitors- lebrikizumab and tralokinumab, and the oral JAK inhibitors- upadacitinib and abrocitinib, are now approved for pediatric populations. Real-world data guiding the selection of subsequent therapy in children who have failed dupilumab remain limited. We characterized treatment trajectories and response in a pediatric cohort that switched from dupilumab to second- and third-line systemic agents.

Materials and Methods

We retrospectively reviewed pediatric AD patients treated with dupilumab at our institution, identifying those in whom dupilumab was the first systemic therapy and who switched to a different systemic agent before age 18. Recorded variables included sex, age at dupilumab initiation, dupilumab treatment duration (including interim treatment breaks), and the identity of subsequent agents. Treatment response was defined as ever achieving an Investigator's Global Assessment (IGA) of 0/1. Among treatment responders, the reason for switching to the next line was classified as loss of efficacy or adverse event. Continuous variables are reported as median (IQR) and categorical variables as n (%). Second-line agents were grouped by mechanism into IL-13 inhibitors (lebrikizumab, tralokinumab) and JAK inhibitors (upadacitinib, abrocitinib), and second-line response was compared between classes using Fisher's exact test, with odds ratio and 95% CI. Time to discontinuation of second-line therapy was estimated by the Kaplan-Meier method.

Results

Of 475 pediatric patients treated with dupilumab for AD (median age 12.7 years, IQR 7.1–17.7), 24 (5.1%) switched to a different systemic agent before age 18 and comprised the study cohort (15 female, 9 male). Dupilumab was initiated at a median age of 12.8 years (IQR 10.8–15.3) and continued for a median of 108 weeks (IQR 28.5–201.5), with the switch to second-line therapy occurring at a median age of 15.6 years (IQR 14.6–16.5). Sixteen patients (67%) required one treatment line after dupilumab, and 8 (33%) required two lines after dupilumab.

IGA 0/1 was achieved at some point on dupilumab in 7/24 patients (29%); among these dupilumab responders, switching to a second-line treatment was driven by loss of efficacy in six and adverse events in one. The remaining 17/24 (71%) were primary non-responders.

Second-line agents comprised JAK inhibitors in 13 patients (54%; upadacitinib 12, abrocitinib 1) and IL-13 inhibitors in 11 (46%; lebrikizumab 9, tralokinumab 2). Overall, 10/24 (42%) reached IGA 0/1 on second line. When comparing class-level efficacy, IGA 0/1 was achieved in 7/13 patients on a JAK inhibitor (53.8%) versus 3/11 on an IL-13 inhibitor (27.3%); the difference did not reach statistical significance (Fisher's exact, OR 3.11, 95% CI 0.43–

25.8, p = 0.24). Median time to discontinuation of second-line therapy was 35.4 weeks for JAK inhibitors and 71.4 weeks for IL-13 inhibitors. Of the eight patients who reached a third line, 2 (25%) achieved IGA 0/1.

Conclusions

In this pediatric cohort, most children switching from dupilumab were primary non-responders rather than patients who lost response, and 42% achieved IGA 0/1 on a second-line agent, supporting the clinical value of sequencing systemic therapy in refractory pediatric AD. JAK inhibitors showed a numerically higher second-line response rate than IL-13 inhibitors, though median time on therapy was longer for IL-13 inhibitors. third-line response was modest (25%). Given the small, non-randomized sample, these findings warrant confirmation in larger prospective studies.

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Abstract N°: 2835

Lichen planus or pembrolizumab-induced lichenoid drug eruption? A diagnostic challenge in the era of immunotherapy

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Introduction

Immunotherapy has revolutionized oncologic treatment, and immune checkpoint inhibitors are increasingly used in the management of a growing number of malignancies. Pembrolizumab is a monoclonal antibody targeting the programmed cell death protein 1 (PD-1) receptor, enhancing the immune response against tumor cells. It is currently used in the treatment of several cancers, including melanoma, lung carcinoma and triple-negative breast carcinoma. Due to its unique mechanism of action, this therapeutic approach has introduced new clinical challenges, particularly immune-mediated adverse events that may involve multiple organs and systems, with cutaneous manifestations being among the most frequently reported, occurring in 30-40% of patients treated with PD-1 inhibitors.

Case Presentation

We report the case of a 43-year-old woman with triple-negative invasive carcinoma of the right breast who received neoadjuvant pembrolizumab, followed by tumorectomy and subsequent adjuvant pembrolizumab therapy. Approximately nine months after initiating pembrolizumab, the patient developed multiple millimetric (1 to 3 mm), flat-topped, erythematous-violaceous non-pruritic papules arranged linearly along the periareolar surgical scar. On dermoscopy, Wickham striae were clearly evident, supporting the lichenoid nature of the eruption. The pattern of distribution suggested a Koebner phenomenon at the site of the previous surgical incision. In addition, multiple similar lesions were observed on the right breast and axilla. Histopathological examination revealed a lichenoid inflammatory infiltrate involving the papillary dermis, associated with confluent vacuolar degeneration of the basal layer of the epidermis, with formation of a prominent subepidermal cleft. Scattered colloid bodies were identified, along with pigment incontinence. The overall findings were consistent with lichen planus. No specific features suggestive of a drug-induced etiology were identified in this specimen.

Conclusion

This case highlights the diagnostic challenge of distinguishing idiopathic lichen planus from immunotherapy-induced lichenoid drug eruptions, given their overlapping clinical and histopathological features. Although classic clinical findings of idiopathic lichen planus, such as Wickham striae and lichenoid papules, were present, the absence of pruritus and the distribution pattern raise the possibility of a drug-induced etiology. Histopathological findings supported lichen planus, without features suggestive of a drug-induced etiology, further complicating the distinction. These observations underscore the importance of careful clinicopathological correlation and awareness of cutaneous immune-related adverse events in patients receiving immune checkpoint inhibitors.

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Abstract N°: 2841

Omics-Based Biomarker Discovery in Plasma and Finger Sweat for Individualized Anti-PD-1 and CTLA-4 Treatment Stratification in Melanoma

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Introduction

Reliable biomarkers for predicting and monitoring response to anti-PD-1 and CTLA-4 therapy in metastatic melanoma are still lacking. The noninvasive analysis of fingertip sweat combined with OMICS analyses from plasma represents a promising approach for capturing protein regulation and metabolic alterations across different biological compartments.

Materials and Methods

Fingertip sweat and plasma samples are collected at baseline as well as 3 weeks and 3 months after initiation of therapy with ipilimumab and nivolumab, together with clinical data. In a pilot study, fingertip sweat samples were obtained from patients receiving nivolumab immediately before and after treatment. Fingertip sweat analyses were performed using liquid chromatography–mass spectrometry (Orbitrap Exploris 480), proteomics using the Bruker timsTOF Pro platform, and plasma metabolomics using the Biocrates MXP Quant 500XL kit.

Results

In the pilot study, we investigated fingertip sweat metabolomics as a noninvasive biomarker strategy. Initial data demonstrated elevated levels of kynurenine (tryptophan metabolism), p-cresol sulfate (indicative of microbial dysbiosis), and markers of mitochondrial stress in non-responders (NR). In the longitudinal study, we were able to quantitatively demonstrate the induction of indole-3-acetic acid over time in responders (R) using fingertip sweat analysis. This metabolite is a degradation product of tryptophan and serves as an indicator of gut microbiome activity.

Plasma proteomics of this longitudinal cohort revealed differential regulation of immune-associated proteins between R and NR after three months of therapy. Responders were characterized by significant upregulation of immunoglobulins as well as the Golgi-associated protein GLIPR2. In contrast, non-responders exhibited elevated levels of SPINK1, a marker closely associated with epithelial–mesenchymal transition (EMT) and therapy resistance. Protein set enrichment analysis confirmed selective enrichment of immunological functions exclusively in responders, indicating effective systemic immune activation.

Plasma metabolomics analysis likewise demonstrated elevated kynurenine levels in non-responders after three months of immunotherapy. Additionally, triglycerides and the immunosuppressive metabolite putrescine were increased in non-responders throughout the entire observation period, whereas immune-activating metabolites

such as phosphoenolpyruvate and phosphatidylcholine were upregulated in responders. Principal component analysis (PCA) over time showed partial separation between responders and non-responders, driven in non-responders mainly by triglycerides, while phosphoenolpyruvate and indole-3-propionic acid were associated with responders. The latter is particularly relevant because this tryptophan derivative has been linked to an intact gut microbiota and improved response to immunotherapy, suggesting that the tryptophan–kynurenine pathway, in connection with the gut microbiome, appears to be an important regulator of therapeutic response.

Conclusions

The establishment of fingertip sweat analysis as a noninvasive platform in combination with plasma OMICS data offers significant potential for improving the understanding of therapy efficacy. This approach may enable early identification of non-responders as well as optimized risk stratification.

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Abstract N°: 2859

Real-world experiences with Lebrikizumab in patients with severe Atopic Dermatitis

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Introduction

Atopic dermatitis (AD) is a chronic inflammatory skin disease associated with impaired quality of life. Lebrikizumab is a monoclonal antibody that binds interleukin-13 with high affinity and a slow dissociation rate, blocking its downstream signaling and reducing inflammation. Lebrikizumab is approved in adults and adolescents ≥ 12 years with moderate-to-severe AD. Despite numerous randomized, placebo-controlled, phase 3 trials (RCT) establishing the efficacy and favorable safety profile of lebrikizumab in patients with moderate-to-sever AD, there is paucity of evidence regarding its outcomes in real-world clinical settings. This article details the effectiveness of lebrikizumab treatment for moderate-to-severe AD in clinical practice.

Materials and Methods

We prospectively followed eight patients with moderate-to-severe AD who were started on or switched to lebrikizumab treatment. Patients received lebrikizumab as a subcutaneous injection at an initial dose of 500 mg at weeks 0 and 2, followed by 250 mg every other week until week 16. Severity was assessed using the Eczema Area and Severity Index (EASI) and Numerical Rating Scale (NRS) for itch at baseline and week 16. Informed written approval was acquired from the patients for the publication of this study. The research was executed in strict compliance with the Declaration of Helsinki, guaranteeing ethical standards and safeguarding patient confidentiality. To ensure confidentiality, all data presented in this study have been anonymized, and the patients provided written consent for the publication.

Results

Six of our patients were bio-naïve and two were previously on targeted therapies (biologics or Janus kinase inhibitors). After 16 weeks of treatment with lebrikizumab, clinically significant improvements in signs and symptoms (assessed by EASI and NRS) were observed. All of our patients achieved EASI-75. Patients with difficult-to-treat areas (hands and head/neck involvement) responded particularly well. No serious adverse events were observed; mild injection-site reaction was reported in one patient and resolved without intervention or treatment discontinuation.

Conclusions

Lebrikizumab demonstrated excellent efficacy and a favorable safety profile in patients with moderate to severe atopic dermatitis. It remains an effective option for bio-naïve patients and those previously exposed to advanced systemic therapy, including biologics and Janus kinase inhibitors. These findings are consistent with pivotal phase 3 trial data and support a role for lebrikizumab in real-world clinical practice.

Patient number	Sex (M/F)	Age (Years)	EASI		NRS	
			Baseline	16W	Baseline	16W
1	M	65	20	1	6	1
2	M	30	17	1	4	0
3	F	18	16	0	4	0
4	M	60	20	2	5	0
5	F	35	15	1	6	1
6	F	20	19	0	5	0
7	M	22	25	3	6	1
8	M	28	16	1	3	1

Table 1 Clinical data of patients and results of severity AD scores (baseline) (16W) Abbreviations: W, weeks; EASI, Eczema Area Severity Index; NRS, skin itching numeric rating scale

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Abstract N°: 2863

Potential Abscopal Effect Following Intralesional BO-112 in Basal Cell Carcinoma: A Case Report

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Introduction

BO-112 is a synthetic double-stranded RNA immunotherapy designed to activate innate and adaptive immune response and immunogenic tumor-cell death. BO-112 can convert "cold" tumors into "hot" tumors by activating local innate immune sensing and driving a marked increase in intratumoral CD8+ T-cell infiltration.
By enhancing local innate immune activation and in situ tumor antigen priming, these effects may translate into immune-mediated regression signals in distant, non-treated lesions. In prior melanoma trial, BO-112 was associated with responses in non-injected lesions, suggestive of a potential abscopal effect.

Basal cell carcinoma (BCC) is typically managed with surgery, although a subset of patients are not ideal surgical candidates or seek non-surgical alternatives. BO-112 shows promising efficacy in BCC lesions, especially in cosmetically sensitive areas of head & neck. Early and substantial visual lesion shrinkage with >80% volume reduction strongly predicted complete pathological clearance. The objective of this report is to present a clinical case suggestive of a potential abscopal effect of BO-112 in an untreated BCC lesion.

Case Presentation

The clinical case was identified within SPOTLIGHT 204, a multicentre, phase 2b, single arm, two-cohort trial evaluating the efficacy, safety and tolerability of intralesional BO-112, administered once weekly for three consecutive weeks, in patients with resectable primary low- and high-risk basal cell carcinoma.

An 81-year-old woman presented with two BCCs: a high-risk nodular lesion located on the left ala of the nose (20 mm in greatest diameter), and a second lesion on the left temple. Both lesions were biopsied and the lesion on the nose was selected for treatment with BO-112.

The treated lesion was monitored through serial measurements and visual assessment. This lesion was surgically excised at week 26 in accordance with the study protocol. The non-treated lesion was additionally excised during the same surgical procedure. Both specimens were obtained using Mohs micrographic surgery and, subsequently evaluated histopathologically.

The BO-112 treated nasal lesion showed early tumor shrinkage, decreasing from 20 mm to 17 mm by week 3, with reduction in thickness from 7 mm to 4 mm. By week 7, the lesion measured 15 mm with no residual thickness, and by week 12, a complete clinical response was observed, with only residual post-inflammatory erythema. The response was maintained throughout the trial. The area of the original lesion was surgically removed at week 26, as mandated by the clinical protocol.

Histopathological evaluation of specimens obtained from Mohs surgery was performed by a local pathologist:

- Treated lesion: Complete pathological response with no residual viable tumor.

- Untreated lesion: Marked histological regression (~70%), with residual infiltrative BCC and cicatricial fibrosis; surgical margins were free of tumor.

Safety profile in this patient was characterized by a single, mild, Grade 1 transient fever which occurred on the day of the third administration and resolved within 24 hours with paracetamol. There were no other adverse events reported for this patient. The safety profile was consistent with the known safety profile of BO-112.

Conclusion

A marked histological response in a distant, untreated lesion suggests a potential abscopal effect. The BO-112 treated BCC achieved complete clinical and pathological response, with early visual assessment at week 7 enabling timely identification of response.

These findings support the hypothesis of BO-112 induced systemic antitumor immune response beyond the injected site. BCC develops in a chronically damaged, immunosuppressed microenvironment and is capable of evading immune surveillance, highlighting the need for novel immuno-oncology therapies beyond surgery.

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Abstract N°: 2868

Leukopenia associated with Janus kinase inhibitors in dermatology: a retrospective cohort study

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Introduction

Janus kinase inhibitors (JAKi) have emerged as a promising therapeutic class in Dermatology, with established or expanding indications in inflammatory skin conditions such as atopic dermatitis, alopecia areata and psoriasis. However, their use may be associated with haematological toxicity, particularly leukopenia. Current recommendations advocate regular laboratory monitoring, yet practical guidance regarding the management of JAKi-induced leukopenia, including thresholds for dose adjustment or discontinuation, remains poorly defined.

Materials and Methods

A retrospective observational cohort study was conducted at ULS São José, including all patients (adults and children) treated with JAK inhibitors (tofacitinib, baricitinib, upadacitinib and abrocitinib) for dermatological indications between 2018 and November 2025. Patients who developed iatrogenic leukopenia, defined as a leukocyte count below 4000 cells/mm^3, were identified. Demographic, clinical and laboratory data were collected, and severity was graded according to the Common Terminology Criteria for Adverse Events (CTCAE).

Results

Over the study period, 283 patients were exposed to JAKi, predominantly for atopic dermatitis, of whom 18 (6.3%) developed leukopenia, accounting for a total of 29 events. Recurrent events occurred in 9/18 patients (50%), one of whom experienced four separate episodes. Three patients (17%) developed leukopenia under two different JAKi, suggesting individual susceptibility rather than a strictly molecule-specific effect. The incidence per drug was: tofacitinib 4/46 (8.7%), baricitinib 7/82 (8.5%), abrocitinib 1/12 (8.3%) and upadacitinib 9/143 (6.3%). The mean time to the first event was 7.6 months (median 5; range 1–36), with 15/29 episodes (52%) occurring within the first six months of treatment.

Neutropenia was the predominant haematological alteration (25/29 events, 86%). Overall, most leukopenia events were mild (CTCAE grade 1; 27/29, 93%), with only 2/29 events (7%) classified as moderate to severe (CTCAE grade ≥2), and a single episode was complicated by a mild infectious complication. Baricitinib was associated with a higher proportion of lymphopenia (3/10 events, 30%), in contrast with upadacitinib (1/13). In most cases (21/29, 72%), leukopenia resolved spontaneously, without dose modification or treatment interruption.

The overall discontinuation rate among affected patients was 8/18 (44%); however, only 2/8 discontinuations (25%) were attributable to haematological toxicity, with the remaining 6/8 (75%) being due to therapeutic failure.

Conclusions

In conclusion, although leukopenia associated with JAK inhibitors is a relatively frequent adverse event, with a high recurrence rate, it is generally of mild severity and rarely requires treatment interruption. The high recurrence

rate and the occurrence of leukopenia in patients sequentially exposed to structurally distinct JAKi strongly support the hypothesis that haematological toxicity in this setting is largely driven by individual susceptibility, possibly reflecting host pharmacogenomic or immunological factors, rather than by molecule-specific mechanisms. This finding has direct clinical implications, as a previous episode of JAKi-induced leukopenia should not necessarily preclude treatment with an alternative JAKi, provided close haematological surveillance is ensured. These findings reinforce the importance of more intensive haematological monitoring during the first six months of therapy, while maintaining prolonged surveillance thereafter, and support a conservative management approach that allows therapeutic continuity in most cases. Larger, prospective studies are warranted to better characterise risk factors for recurrent or severe leukopenia and to refine evidence-based monitoring algorithms in this expanding therapeutic class.

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Abstract N°: 2905

Guselkumab Shows Better Long-term Effectiveness and Drug Survival Compared to Secukinumab Through 84 Weeks in Patients With Moderate-to-Severe Plaque Psoriasis: Results from the German Non-interventional G-REAL Study

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Introduction
Guselkumab (GUS), a p19 subunit-targeted IL-23 inhibitor, showed superior long-term efficacy compared to the IL-17A inhibitor secukinumab (SEC), in psoriasis patients in a randomized controlled trial.[1] The prospective, non-interventional G-REAL study evaluates effectiveness of GUS versus SEC in patients with moderate-to-severe psoriasis in routine practice in Germany.

Materials and Methods
This analysis includes 502 GUS/ 159 SEC patients, stratified by previous biologic therapy: bio-naïve 259 GUS/ 95 SEC and ≥1 prior biologic 243 GUS/ 64 SEC patients. Outcomes include Psoriasis Area and Severity Index (PASI) response (after applying treatment failure rules) and drug survival through 84 weeks. Treatment failure is defined as discontinuation of therapy due to lack or loss of effectiveness, adverse event of worsening psoriasis, or initiating a new psoriasis therapy. P-values are considered nominal.

Results
Baseline mean PASI scores for overall GUS and SEC cohorts were 15.1 and 14.3, respectively. At week 84, 5.4% of GUS patients experienced treatment failure versus 17.0% (SEC). PASI=0 was reached by 186 (54.1%) GUS versus 36 (36.7%) SEC patients ($p<0.01$) in the overall cohorts; 118 (67.4%) GUS versus 25 (43.9%) SEC patients ($p<0.01$) in the bio-naïve groups; and 86 (40.2%) GUS versus 11 (26.8%) SEC patients (n.s.) in the ≥1 prior biologic groups. The probabilities of drug survival at 18 months for GUS were: 84.4% (overall) and 87.1% (bio-naïve); for SEC were: 72.5% (overall) and 78.9% (bio-naïve).

Conclusions
Guselkumab demonstrated substantially higher rates of complete skin clearance and better drug-survival, especially in bio-naïve patients, compared to secukinumab through 84 weeks in real-world practice, indicating improved benefits with first-line use of guselkumab.

References:

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Abstract N°: 2927

Morbidity and Mortality Outcomes of Dupilumab for Cutaneous Immune-Related Adverse Events

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Introduction

Dupilumab is a monoclonal antibody targeting the interleukin (IL)-4 receptor α subunit, thereby inhibiting T helper 2 (Th2) immunity. It is increasingly used off-label to manage cutaneous immune-related adverse events (cirAEs) following immune checkpoint inhibitor (ICI) therapy. Unlike other immunomodulatory treaments for cirAEs, dupilumab lacks broad immunosuppressive effects, making it an optimal therapy for AD in patients with cancer and thus favored by dermatologists. While dupilumab's ability to control symptoms in patients with cirAEs is well-established, long-term outcomes remain uncertain.

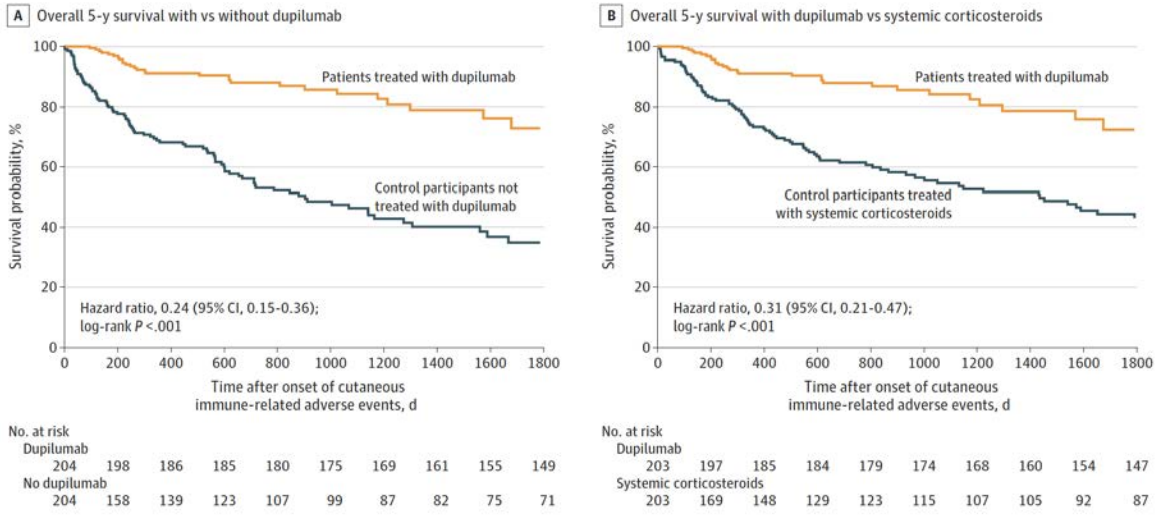
Materials and Methods

A retrospective cohort study was conducted using deidentified aggregate data from 163 healthcare organizations within the TriNetX Global Collaborative Network. We identified 204 adult patients who developed cirAEs and subsequently received dupilumab. Two 1:1 propensity score-matched comparison cohorts were established: patients with cirAEs never prescribed dupilumab (control 1) and those prescribed systemic corticosteroids without dupilumab (control 2), matched for age, sex, race, Charlson Comorbidity Index diagnoses, and use of other immunomodulatory agents; covariate balance was adequate (all standardized mean differences <0.2). Control 2 represents patients with severe cirAEs requiring systemic intervention, thereby serving as a clinically analogous comparator to dupilumab-treated patients. All-cause mortality was the primary endpoint; secondary endpoints were non-cutaneous irAEs (non-cirAEs), healthcare utilization, and infections. Hazard ratios (HRs) for Kaplan-Meier analysis and morbidity measures were calculated at 5 years post-index event, defined as cirAE onset; sensitivity analyses were performed, reindexing at 180 days post-cirAE onset.

Results

The dupilumab cohort demonstrated significantly improved survival compared to control 1 (HR 0.24, CI 0.15–0.36, p<0.0001) and control 2 (HR 0.31, CI 0.21–0.47, p<0.0001). Median survival was 903 days in control 1 and 1,438 days in control 2 but was not reached by the dupilumab cohort in either analysis. Sensitivity analyses were consistent (control 1: HR 0.37, CI 0.23–0.59, p<0.0001; control 2: HR 0.43, CI 0.27–0.68, p=0.0002). Relative risk (RR) of developing a non-cirAE was higher in the dupilumab group compared to control 1 (RR 1.46, CI 1.01–2.12, p=0.0471), specifically gastrointestinal irAEs (RR 1.76, CI 1.02–3.03, p=0.0377). Median instances of infection was also higher in the dupilumab cohort versus control 1 (3 vs. 2, p=0.0410). However, neither non-cirAE risk nor infection rates were significant after Bonferroni adjustment and did not differ significantly between the

dupilumab cohort and control 2. Rates of emergency department encounters, inpatient admissions, and intensive care unit admissions were comparable between groups in both analyses.



Kaplan-Meier curves depicting overall 5-year survival of dupilumab-treated patients versus non-dupilumab controls (A) and dupilumab-treated patients versus systemic corticosteroid-treated controls (B). Curves are shown as right-continuous step functions of survival probability from index (cirAE onset) to 1,825 days.

Conclusions

Dupilumab treatment for cirAEs was associated with improved survival compared to non-dupilumab and systemic corticosteroid-treated controls. Dupilumab use was also linked to higher rates of non-cirAEs and infections versus non-dupilumab controls, but differences lost significance after Bonferroni corrections and were absent when compared with systemic corticosteroid-treated patients. Taken together, our observations align with evidence implicating IL-4 signaling in tumorigenesis and immune evasion; notably, IL-4 blockade enhanced ICI response in murine models, with findings recapitulated in non-small cell lung cancer patients treated with dupilumab. Interpretation is limited by indexing at cirAE onset, since some dupilumab-treated patients may have previously received systemic corticosteroids, and the design introduces immortal-time bias relative to non-dupilumab controls, though mitigated by sensitivity analyses. Considering residual and unmeasured confounders, large effect sizes must be interpreted cautiously. Prospective studies are needed to validate our hypothesis-generating findings and inform algorithms for cirAE management.

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Dupilumab for bullous pemphigoid: a real-world case series from a tertiary hospital

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Introduction

Bullous pemphigoid is the most common autoimmune blistering disease and predominantly affects elderly patients with multiple comorbidities. It is mediated by autoantibodies against BP180 and BP230, leading to subepidermal blistering, pruritus and inflammatory skin lesions. Although topical and systemic corticosteroids remain the cornerstone of treatment, moderate-to-severe or refractory disease may be difficult to manage in frail patients, in whom prolonged immunosuppression is often limited by infections, metabolic complications, renal impairment or malignancy. Increasing evidence supports a relevant type 2 inflammatory signature in bullous pemphigoid, including IL-4/IL-13-driven pathways, eosinophilic inflammation and IgE-mediated mechanisms. Dupilumab, an IL-4 receptor alpha antagonist that inhibits IL-4 and IL-13 signalling, has emerged as a promising steroid-sparing therapeutic option.

The objective of this study was to describe our real-world experience with dupilumab in patients with bullous pemphigoid treated in the dermatology department of a tertiary hospital, focusing on clinical response and tolerability.

Materials and Methods

We performed a retrospective observational study including all patients with bullous pemphigoid who received dupilumab between August 2024 and January 2026 in the dermatology department of a tertiary hospital. Demographic data, comorbidities, possible triggers, previous systemic treatments, time from diagnosis to dupilumab initiation, clinical response and adverse events were collected from medical records. Dupilumab was administered as a 600 mg loading dose followed by 300 mg every two weeks.

Results

Five patients were included, three men and two women, with a median age at diagnosis of 82 years. All patients had significant comorbidities, including hypertension and dyslipidaemia in all cases, diabetes mellitus and renal impairment in 60%, and solid neoplasms in one patient. One case was probably associated with vildagliptin exposure. All patients had previously received systemic corticosteroids; two had received azathioprine and one methotrexate, which was discontinued because of infections and elevated transaminases.

Dupilumab was initiated after a median interval of 4 months from diagnosis. All patients showed clinical improvement. Complete response was achieved in three patients, occurring after 2, 3 and 5 months of treatment, respectively. Two patients achieved partial response, one of them while receiving concomitant azathioprine. No adverse events attributable to dupilumab were recorded during follow-up. Clinically, improvement was characterized by resolution of tense blisters and urticarial plaques, with residual post-inflammatory hyperpigmentation and isolated crusted areas after treatment.

Conclusions

In this real-world case series, dupilumab was associated with favourable clinical outcomes and excellent tolerability in elderly patients with bullous pemphigoid and high comorbidity burden. Our findings support the growing evidence that IL-4/IL-13 blockade may represent an effective and safe steroid-sparing alternative, particularly in patients in whom conventional immunosuppressive therapy is contraindicated, poorly tolerated or insufficiently effective. Larger prospective studies and standardized response criteria are needed to better define its optimal timing, duration of treatment and role within therapeutic algorithms for bullous pemphigoid.

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Abstract N°: 2962

Early Repigmentation and Predictive Factors of Upadacitinib in the Treatment of Advanced Non-Segmental Vitiligo: A Real-World Study Based on Generalized Estimating Equations

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Introduction
Vitiligo is a depigmenting skin disorder mediated by autoreactive CD8+ T cells, in which the IFN-γ/CXCL10 axis plays a central role. While the highly selective JAK1 inhibitor upadacitinib has shown potential in reshaping the immune microenvironment, its early clinical characteristics and predictors for repigmentation in real-world settings require systematic quantification. This study aimed to evaluate the 12-week early efficacy and safety of oral upadacitinib in patients with active non-segmental vitiligo and to identify independent predictors of early repigmentation using generalized estimating equation (GEE) models.

Materials and Methods
This retrospective study included 26 patients with active non-segmental vitiligo, involving a total of 336 target lesions, who were treated with upadacitinib for 12 weeks. The time to disease stabilization and the time to initial repigmentation were recorded for each patient, and adverse reactions were monitored. GEE models were utilized to adjust for the clustering effect among multiple lesions within the same patient and to identify independent predictors influencing marked response and overall response rates.

Results
The overall response rate was 75.0%, and the marked response rate was 53.3%. The median time to disease stabilization was 34.0 days, and the median time to early repigmentation was 29.0 days. GEE model analysis indicated that disease duration and lesion site were independent predictors of early clinical outcomes. Specifically, the success rate for lesions with a duration of <52 weeks was significantly higher than for those with a duration of ≥52 weeks (OR = 10.058, 95% CI: 2.827–35.783, P < 0.001). Using acral lesions as the reference, the success rate for limb lesions was significantly higher (OR = 7.00, 95% CI: 1.855–26.417, P = 0.004). The safety profile was favorable, with the primary adverse reactions being mild acne (6 cases) and folliculitis (2 cases); no serious adverse events occurred.

Conclusions
Oral upadacitinib demonstrates favorable early repigmentation and high safety in the treatment of progressive non-segmental vitiligo over a 12-week period. Short disease duration (<52 weeks) and lesion site are identified as independent predictors of early therapeutic response.

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Abstract N°: 2984

Budgetary Implications of Biologic Prescribing for Psoriasis in Ireland

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Introduction

Biologic therapies have transformed outcomes in moderate-to-severe plaque psoriasis but are associated with substantial financial burden. In Ireland, these agents are reimbursed through the Health Service Executive (HSE) High Tech Scheme, where increasing biologic utilisation has important implications for healthcare expenditure.

Objectives:

To compare annual maintenance-phase drug acquisition costs of commonly prescribed biologic therapies for psoriasis in Ireland using publicly available HSE High Tech Scheme reimbursement prices.

Materials and Methods

A cost-minimisation analysis was performed from the HSE payer perspective using High Tech Scheme list prices (1 December 2025). Annual maintenance-phase acquisition costs were calculated for commonly prescribed biologics used in moderate-to-severe plaque psoriasis, including TNF-α, IL-17, IL-12/23 and IL-23 inhibitors. Cost per injection was derived from pack price and dosing schedules based on a 52-week year. Costs associated with induction dosing, monitoring, administration, adverse events and indirect healthcare utilisation were excluded. A hypothetical cohort of 100 adults receiving each biologic regimen was modelled to estimate potential budget impact.

Drug	Maintenance dose & frequency	Pack price (€)	Injections per pack	Injections per year	Cost per injection (€)	Annual cost per patient (€)	Annual cost for hypothetical cohort of 100 patients (€)
Anti-TNF							
Adalimumab (biosimilar)	40mg every 2 weeks	486.08	2	26	243.04	**6,319.04**	**631,904**
Etanercept (Benepali)	50mg weekly	557.13	4	52	139.28	**7,242.69**	**724,269**
Infliximab (subcutaneous; Remsima)	120mg every 2 weeks	540.00	2	26	270.00	**7,020.00**	**702,000**
Certolizumab pegol	200mg every 2 weeks	849.18	2	26	424.59	**11,039.34**	**1,103,934**
IL-17							
Secukinumab (Cosentyx)	300mg every 4 weeks	1,111.82	1	13	1,111.82	**14,453.66**	**1,445,366**
Ixekizumab (Taltz)	80mg every 4 weeks	1,020.61	1	13	1,020.61	**13,280.93**	**1,328,093**
Bimekizumab (Bimzelx)	320mg every 8 weeks	2,152.11	2 (1 dose)	6.5	2,152.11	**13,988.72**	**1,398,872**
Brodalumab (Kyntheum)	210mg every 2 weeks	1,099.86	2	26	549.93	**14,298.18**	**1,429,818**
IL-12/23							
Ustekinumab (Stelara) 45mg	45mg every 12 weeks	1,634.66	1	4.3	1,634.66	**7,029.04**	**702,904**
Ustekinumab (Stelara) 90mg	90mg every 12 weeks	1,680.71	1	4.3	1,680.71	**7,227.05**	**722,705**
IL-23							
Guselkumab (Tremfya)	100mg every 8 weeks	2,083.95	1	6.5	2,083.95	**13,545.68**	**1,354,568**
Risankizumab (Skyrizi)	150mg every 12 weeks	3,027.21	1	4.3	3,027.21	**13,017.00**	**1,301,700**
Tildrakizumab (Ilumetri)	100mg every 12 weeks	2,714.20	1	4.3	2,714.20	**11,671.06**	**1,167,106**

Table 1. Annual drug acquisition cost per patient for selected biologic therapies used in moderate–severe plaque psoriasis based on the HSE High Tech Scheme list (1 December 2025). Annual dosing calculations are based on a 52-week year. Costs reflect maintenance dosing only and exclude induction dosing, administration, monitoring, adverse-event and indirect costs.

Results

Adalimumab biosimilar demonstrated the lowest annual acquisition cost at €6,319 per patient, followed by subcutaneous infliximab (€7,020) and ustekinumab 45mg (€7,029). In contrast, newer biologics were associated with substantially higher annual costs, including risankizumab (€13,017), guselkumab (€13,546), bimekizumab (€13,989), brodalumab (€14,298) and secukinumab (€14,454), the highest-cost agent identified. Treating a hypothetical cohort of 100 patients with adalimumab rather than risankizumab would yield estimated annual savings of approximately €579,000. Despite reduced dosing frequency with IL-23 inhibitors, higher per-dose acquisition costs offset potential savings associated with extended dosing intervals.

Conclusions

Substantial variation exists in annual maintenance-phase acquisition costs among biologic therapies used to treat psoriasis in Ireland. Biosimilar-supported TNF-α inhibitors and ustekinumab remain considerably less expensive

than newer IL-17 and IL-23 inhibitors at current Irish list prices. While treatment decisions should remain clinically driven, awareness of these cost differentials may assist biologic sequencing and healthcare resource allocation within publicly funded dermatology services.

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Abstract N°: 3000

Sarcoid -like reaction induced by adjuvant therapy in a melanoma patient

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Introduction

Immunotherapy has revolutionized cancer treatment, changing the course of several malignancies, although it is associated with immune-mediated toxicity. One of the less common immune-related adverse events is granulomatous reaction triggered by immune checkpoint inhibitors (ICIs)

Case Presentation

In January 2025, we observed a 59-year-old female patient referred after biopsy of a right calcaneus lesion which histopathological examination disclosed an ulcerated melanoma with a 3.5 mm Breslow thickness. She underwent wide local excision of the lesion and sentinel lymph node biopsy in February 2025. The right inguinal sentinel lymph node was negative and the excision specimen showed an ulcerated melanoma with a thickness of 8.7 mm and 1 mitosis/mm^2 (stage IIC). Staging examinations (thoracoabdominopelvic CT and contrast-enhanced cranial CT) showed no abnormalities. Adjuvant immunotherapy was proposed, and she started pembrolizumab 200 mg IV every 3 weeks in April 2025, with a planned duration of 1 year.

Approximately 4 months after treatment initiation, painless subcutaneous nodules appeared on the left leg and scalp. These lesions were excised, and histological examination was similar in both, showing well-circumscribed epithelioid granulomas and multinucleated giant cells without necrosis, raising the possibility of sarcoidosis. A PET scan performed in November 2025 was interpreted as compatible with pulmonary and mediastinal-hilar nodal metastases. However, biopsy of a mediastinal lymph node excluded malignancy and suggested non-necrotizing granulomatous lymphadenitis. Throughout follow-up, the patient reported moderate asthenia, without other significative associated symptoms. Following discontinuation of immunotherapy, improvement of the PET findings was observed.

Conclusion

Sarcoid-like reaction (SLR) induced by ICIs may mimic disease progression. Skin and mediastinal lymph node biopsies allowed exclusion of metastatic disease. The favorable response after treatment discontinuation, together with the patient's clinical stability and absence of laboratory abnormalities, further supported this association. We emphasize the importance of recognizing this entity in order to ensure appropriate investigation and management.

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Abstract N°: 3008

When IL-17 Blockade Backfires: Paradoxical Hidradenitis Suppurativa During Ixekizumab Therapy

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Introduction

Biologic therapies have transformed the management of refractory psoriasis, but their expanding use has revealed uncommon inflammatory adverse events with overlapping clinical phenotypes. Hidradenitis suppurativa developing during IL-17 blockade is particularly unusual. We report a case of new-onset hidradenitis suppurativa during ixekizumab treatment in a patient with refractory palmoplantar pustular psoriasis.

Case Presentation

A 38-year-old man with refractory palmoplantar pustular psoriasis since 2014, previously unresponsive to phototherapy, methotrexate, cyclosporine, systemic corticosteroids, and adalimumab, began ixekizumab in May 2025 as a second-line biologic. He achieved partial improvement of his palmoplantar lesions; however, 8 months later he developed axillary bridged comedones and pustules. Histopathology confirmed hidradenitis suppurativa (HS), classified as Hurley stage II, with an IHS4 score of 20. Based on the temporal association, a paradoxical reaction to anti–IL-17 therapy was considered, and treatment was switched to guselkumab, with subsequent clinical improvement.

Paradoxical reactions in dermatology are defined as the occurrence, during biologic therapy, of diseases that the same agents are usually able to treat. Among patients receiving biologics for inflammatory disorders, these events are uncommon: in a French cohort of almost 10,000 patients, they were reported in only 3.2%, corresponding to 7.6 per 100,000 person-years. In psoriasis, paradoxical psoriasis and paradoxical eczema are the most frequent, occurring in 5% and 1% of biologic exposures, respectively. Paradoxical HS is considerably rarer. By 2022, only 41 cases had been reported, most of them induced by anti-TNF agents, particularly adalimumab (56%) and infliximab (22%). The mean latency was 24 months, with a broad range from 1 to 72 months after treatment initiation, a timeframe compatible with our patient. To the best of our review, only one previous case of paradoxical HS associated with ixekizumab has been published. This finding contrasts with reports describing favorable responses to ixekizumab in patients with concomitant psoriasis and HS, underscoring the unusual nature of this presentation.

The mechanism underlying this reaction in the setting of IL-17 axis blockade remains unclear. Nevertheless, by extrapolating from mechanisms proposed for other biologic classes, selective cytokine inhibition may disrupt the inflammatory balance of the underlying disease and induce compensatory activation of alternative pathways. In this context, relative upregulation of TNF-α, IL-1, or IL-17F signaling could promote the clinical expression of previously silent inflammatory phenotypes, including HS. Other plausible mechanisms include reduced IL-17–dependent homeostatic activity in epidermal proliferation, antimicrobial defense, and cutaneous microbiome regulation, which may facilitate follicular inflammation and HS development in predisposed individuals .

Management is not standardized, but available reports suggest that withdrawal of the suspected causal drug should lead to improvement in most cases, and that switching to a different biologic is usually followed by partial or complete clinical response (1). In our patient, ixekizumab was discontinued and therapy was changed to guselkumab, with partial improvement. Although evidence is limited, guselkumab has previously been used successfully in paradoxical HS induced by anti-TNF therapy, achieving a reduction in IHS4 from 10 to 2 .

Conclusion

Our case adds to the scarce literature on ixekizumab-associated paradoxical HS and supports considering a change in biologic class when this adverse inflammatory phenotype appears during anti–IL-17 therapy, while maintaining control of the underlying psoriatic disease during clinical follow-up.

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Abstract N°: 3051

Intralesional BO-112 for high-risk basal cell carcinoma in cosmetically and functionally sensitive areas: first results from the phase 2b SPOTLIGHT-204 trial

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Introduction

Basal cell carcinoma (BCC) is the most common human malignancy and its incidence continues to rise globally. Mohs micrographic and conventional surgery achieve high cure rates but can result in significant scarring and functional compromise when lesions arise at cosmetically or functionally sensitive sites, particularly on the head and neck (H&N). Effective non-surgical options delivered at the point of care in dermatology practice therefore remain an unmet clinical need. BO-112 is a double-stranded RNA immunotherapy designed to activate an innate and adaptive immune response and immunogenic tumour-cell death.

Materials and Methods

SPOTLIGHT-204 is a multi-centre, phase 2b, single-arm, two-cohort clinical trial evaluating efficacy, safety and tolerability of an intralesional BO-112 injection in 50 patients (40 high-risk (HR), 10 low-risk (LR)) with resectable, histologically confirmed, high and low-risk BCC lesions. Patients received three once weekly intralesional injections followed by protocol-mandated complete surgical excision at week 24. We report results in the first 23 patients with 24 high-risk BCC lesions located on the head, with visual and pathological responses evaluated by a Central Review Committee. Visual efficacy was either 1) complete (complete lesion disappearance or scar), 2) partial (>30% reduction in lesion size), 3) no change, or 4) lesion growth (>20% increase in lesion size). Pathological response, at the time of surgical excision, was either 1) complete (no residual viable tumor (RVT), 2) major (<10% RVT), 3) partial (>10% - <50% RVT), or 4) no response (≥ 50% RVT).

Results

Out of the 23 patients, 15 (65%) were females and 8 (35%) males, with median age of 74.5 years (range 37 – 88) and predominantly Fitzpatrick skin type II (11, 48%). One patient presented with two injected lesions, majority (22, 95%) had one lesion. Out of the 24 injected lesions, 13 (54%) were nodular, followed by infiltrative subtype (5, 21%). Most of the lesions were located on the nose (9, 38%) followed by scalp (4, 17%). Median longest diameter was 13.5 mm (range 6–22 mm) with mean BO-112 cumulative exposure of 1.5 mg. All patients received 3 injections of BO-112 per lesion, one patient received optional treatment re-challenge with additional three injections. Out of 23 evaluable patients, 14 (61%) achieved a complete visual response alongside a complete (n=13) or major (n=1) pathological response, demonstrating high concordance between visual and histological assessments (Figure 1). Across all injected lesions, 19 (80%) showed ≥75% reduction in size. Notably, BO-112 was well tolerated: 91% of BO-112-related adverse events were grade 1, transient, flu-like or injection-site reactions; none were grade≥ 3 and none were serious.

		PATHOLOGICAL RESPONSE				
		Pathologic Complete Response (pCR) No RVT	**Major Pathologic Response (MPR)** ≤10% RVT	**Pathologic Partial Response (pPR)** 10%<RVT≤50%	**Pathologic Non-Response (pNR)** >50% RVT	Total # of lesions
VISUAL RESPONSE	**Complete visual clearance or scar**	**13**	**1**	0	0	14
	Partial lesion shrinkage ≥ 30% reduction volume/height/diameter	0	0	**1**	**1**	2
	No change or change smaller than Partial	0	0	0	**8**	8
	Lesion growth Increase of ≥ 20% in the longest lesion diameter	0	0	0	0	0
	Total # of lesions	13	1	1	9	24

Figure 1. Composite efficacy response on lesion level assessed by the Central Review Committee in the first 23 high-risk patients with BCC lesions on the head.

Conclusions

Intralesional BO-112 achieved high rates of complete visual and complete pathological responses with a manageable, predominantly low-grade safety profile in patients with high-risk BCC at cosmetically and functionally sensitive H&N anatomical locations. These results support further development of BO-112 as a non-surgical option that could complement surgery and address a meaningful unmet need at the dermatology point of care.

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Abstract N°: 3052

Safety and tolerability of BO-112, a novel intralesional immuno-therapy, in patients with basal cell carcinoma

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Introduction

Basal cell carcinoma (BCC) shows increasing incidence and mortality rates globally, accounting for the most common human malignancy. BCC is most commonly found in the head and neck (H&N), with 73% of all BCC located in the face. Mohs micrographic and conventional surgery achieve high cure rates but can result in significant scarring and functional compromise when lesions arise at cosmetically or functionally sensitive sites of the H&N. Effective non-surgical options delivered at the point of care in dermatology practice therefore remain an unmet clinical need. BO-112, a novel immune-oncology intralesional treatment, may provide a new treatment option for BCC patients.

Materials and Methods

SPOTLIGHT-204 is a multi-centre, phase 2b, single-arm, two-cohort clinical trial evaluating efficacy, safety and tolerability of BO-112 in patients with up to 8 resectable, histologically confirmed, high and low-risk BCC lesions. Patients receive three once-weekly intralesional injections followed by complete surgical excision of all injected lesions at week 24. An optional treatment re-challenge with additional three once-weekly injections is offered at week 12 or week 15.

Results

Here we report on the safety and tolerability of BO-112 in 50 patients enrolled in the trial (40 high-risk (HR), 10 low-risk (LR)), who received at least one study drug injection and constitute the Safety Analysis Set. Out of the 50 patients, 23 (46%) were females and 27 (54%) males, with median age of 75.0 years (min 34, max 94) and predominantly Fitzpatrick skin type II (27, 54%; min I, max IV). 60 lesions were selected for injection, most of them located on H&N (47, 78%). Median longest lesion diameter was 12.0 mm (min 5.0, max 50.0) and mean BO-112 cumulative exposure was 1.0 mg (min 0.2, max 3.0). All patients received 3 injections of BO-112, one patient received optional treatment re-challenge. 39 (78%) of patients experienced at least one adverse event (AE) with 31 (62%) patients experiencing treatment-related AEs (TRAE) (Table 1). Out of 71 TRAEs, 64 (90%) were Grade 1 (mild), 7 (10%) were Grade 2 (moderate), none of them were ≥Grade 3 (severe) and none of them were serious. The most frequent TRAEs were pyrexia (10 patients, 20%), injection site pain (8, 16%), injection site swelling (7, 14%), and headache (6, 12%). These events correlate with BO-112 safety profile observed in previous clinical trials. There were no serious adverse events, and no adverse events leading to dose reduction or discontinuation.

	Events N=132	Subjects N=50	
	n	n	%
All Treatment-Emergent Adverse Events (TEAEs)	132	39	78.0
TEAEs related to Study Drug (TRAEs)	87	31	62.0
Serious TEAEs	**0**	**0**	**0.0**
Serious TRAEs	**0**	**0**	**0.0**
TEAEs with CTCAE ≥ Grade 3	2	2	4.0
TRAEs with CTCAE ≥ Grade 3	**0**	**0**	**0.0**
TEAEs with outcome of death	**0**	**0**	**0.0**
TEAEs leading to dose reduction	**0**	**0**	**0.0**
TEAEs leading to permanent discontinuation of treatment	**0**	**0**	**0.0**

Table 1. Summary of Treatment Emergent Adverse Events and Treatment–Related Adverse Events in SPOTLIGHT-204.

Conclusions

Intralesional BO-112 safety profile in patients with BCC is characterized by a manageable safety profile of mostly mild, transient injection site reactions and flu-like symptoms. The safety profile together with preliminary promising efficacy support further development of BO-112 as a non-surgical option for BCC patients.

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Abstract N°: 3108

HL4134, a potential Best-in-Class Oral Cyclic Peptide Targeting IL-23 Receptor with Potential comparable Efficacy for Psoriasis Treatment, Compared to Leading Anti-IL-23 P19 Monoclonal Antibodies

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Introduction

Interleukin-23 (IL-23) plays a pivotal role in psoriasis pathogenesis by promoting Th17 cell differentiation and production of pro-inflammatory cytokines. While monoclonal antibodies targeting IL-23 have demonstrated significant clinical efficacy in psoriasis treatment, injection route restricted its clinical compliance. HL4134 is a novel oral cyclic peptide designed to selectively target the IL-23 receptor (IL-23R), offering the potential for oral administration with efficacy equivalent or superior to existing biologics.

Materials and Methods

Affinity assessment: SPR analysis of binding to IL-23R protein.

Protein-protein interaction inhibition: ELISA-based assessment the inhibition of IL-23/IL-23R interaction.

Cellular assays: Inhibition of IL-23-induced STAT3 phosphorylation in human PBMC;Inhibition of IL-23-induced IFN-γ in human PBMC; Inhibition of IL-23-induced IL-17A, IL-22, and IL-17F in rat whole blood

***In vitro* ADME analysis**: Metabolic stability in plasma, simulated gastrointestinal fluid, liver S9, and kidney S9; Human plasma protein binding; CYP inhibition for drug-drug interaction risk evaluation

***In vivo* PK analysis**: Intravenous and oral administration in rats, dogs, and monkeys to assess pharmacokinetic parameters

***In vivo* efficacy**: IL-23 intradermal injection-induced rat psoriasis model with oral administration; evaluation of skin thickness, histopathological psoriasiform lesions, and pharmacokinetic/pharmacodynamic relationships

Quantitative pharmacology modeling: Allometric scaling across species to predict human oral effective dose, steady-state trough concentrations (Ctrough), and tissue exposure windows relative to IC50/IC90 values.

Results

Binding affinity: HL4134 exhibits extremely high affinity for IL-23R with KD <1 pM for human, cynomolgus monkey, and dog proteins, and KD <10 pM for rat IL-23R.

PPI inhibition: inhibitopn of IL-23/IL-23R interaction with IC50 <50 pM.

Cellular activity: Inhibition of IL-23-induced STAT3 phosphorylation in human PBMCs: IC50 <5 pM;Inhibition of IL-23-induced IFN-γ secretion in human PBMCs: IC50 <100 pM; Inhibition of IL-23-induced cytokine secretion in rat whole blood: IC50 <50 pM for IL-17A, IL-22, and IL-17F

***In vitro* ADME profile**:

High metabolic stability in plasma, simulated gastrointestinal fluid, liver microsomes, liver S9, and kidney S9 fractions (no degradation observed over 24 hours); Human plasma protein binding: ~70%; observed no inhibition on 7 CYP enzyme subtypes, revealed low risk of drug-drug interactions.

***In vivo* pharmacokinetics**:

Half-life: 3.45 hours (rat), 4.72 hours (dog), 5.22 hours (cynomolgus monkey);Oral bioavailability:>0.1% (rat), 0.53% (dog), 0.57% (cynomolgus monkey); Oral exposure: Dose-proportional increases in Cmax and AUC observed across species

***In vivo* efficacy in psoriasis model**:

Significant improvement in skin thickness at 3 mg/kg/day BID oral dosing; Clear dose-dependent efficacy from 3 to 100 mg/kg/day; Histopathological analysis confirms amelioration of psoriasiform lesions

Skin tissue drug concentration approximately 10-fold higher than plasma at 1 hour post-dose;Dose-dependent inhibition of IL-17F protein expression and S100A8/S100A9 mRNA levels in rat skin tissue

Population PK modeling predictions:

Estimated oral effective dose: 50 mg QD

Estimated skin tissue exposure relative to IC50: >50 fold, comparable or superior to risankizumab or guselkumab

Conclusions

HL4134 demonstrateds exceptional in vitro activity, favorable ADME profile, promising oral pharmacokinetics, robust in vivo efficacy and superior tissue distribution. HL4134 is predicted to achieve skin tissue exposure levels relative to IC50 that are comparable or superior to anti-IL-23 monoclonal antibodies (risankizumab and guselkumab). These data suggest that HL4134 may demonstrate efficacy in psoriasis treatment equivalent or superior to existing monoclonal antibody therapies while offering the convenience of oral administration.

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Abstract N°: 3114

Discovery of the First-in-Class Oral Cyclic Peptide Targeting IL-1β for Hidradenitis Suppurativa Treatment

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Introduction

Interleukin-1β (IL-1β) is a key pro-inflammatory cytokine that plays a critical role in the pathogenesis of various autoinflammatory diseases. Blockade of IL-1β has been clinically validated to significantly improve symptoms of hidradenitis suppurativa (HS). This abstract reports the discovery of the oral peptide drug candidate targeting IL-1β, which inhibits downstream inflammatory signaling pathways and demonstrates potential therapeutic efficacy in both *in vitro* cellular models and *in vivo* surrogate efficacy models of HS.

Materials and Methods

1. **Affinity assessment**: Surface plasmon resonance (SPR) analysis to determine compound binding affinity to IL-1β protein.
2. **Cellular activity evaluation**: Inhibition of IL-1β-induced IL-6 secretion in MRC-5 cells and human PBMCs.
3. **DMPK evaluation**:
   - Metabolic stability in plasma and simulated intestinal fluid
   - Pharmacokinetic parameters including half-life, clearance, volume of distribution, oral bioavailability, and oral exposure in mice, cynomolgus monkeys, and dogs after intravenous and oral administration
4. ***In vivo* efficacy assessment**:
   - Human IL-1β injection-induced inflammatory response model in C57BL/6 mice: Evaluation of oral administration effects on IL-6 serum levels
   - MSU-induced mouse dorsal air pouch inflammation model: Assessment of neutrophil infiltration and inflammatory cytokine levels in pouch lavage fluid

Results

1. **Binding affinity**: The lead compounds exhibit extremely high affinity for human IL-1β with KD ≈ 1 pM, comparable to canakinumab.
2. **Cellular inhibition**:
   - Significant inhibition of IL-1β-induced IL-6 secretion in MRC-5 cells at very low concentrations (IC50 ≈ 1 pM, 5-20 folds more potent than canakinumab)
   - Potent inhibition of IL-1β-induced IL-6 secretion in PBMCs (IC50 < 50 pM, lower than the canakinumab)
3. **Metabolic stability**: The lead compounds showed excellent metabolic stability in plasma and simulated intestinal fluid with no significant degradation observed over 24 hours.
4. **Pharmacokinetics**:
   - Intravenous administration half-life: 2-8 hours in mice
   - Oral administration demonstrates high exposure levels
   - PK analyze in large animals (monkey and dog) is ongoing

5. ***In vivo* efficacy**:
    - In mouse PD model: 10 mg/kg oral administration shows significant inhibition (>90%) of IL-6 secretion, comparable to or better than the PD responses with clinically relevant doses of canakinumab
    - In MSU-induced air pouch model: Oral administration significantly inhibits neutrophil infiltration and reduces secretion of IL-6 and TNF-α

Conclusions

The lead highly potent oral cyclic peptides, exhibited strong affinity for IL-1β and robust inhibition of downstream inflammatory cellular functions. It demonstrates sufficient *in vivo* half-life and oral exposure, with anti-inflammatory activity observed at low doses in *in vivo* PD and efficacy models. Given the dominant role of IL-1β in hidradenitis suppurativa pathogenesis, these lead compounds represent a promising new oral therapeutic option for HS treatment.

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Abstract N°: 3137

Bimekizumab inhibited radiographic progression across baseline subgroups in biologic-naïve patients with active psoriatic arthritis: 3-year results from a phase 3 study and its open-label extension

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Introduction

Persistent inflammation in psoriatic arthritis (PsA) can drive structural damage, which may contribute to physical impairment and reduced quality of life.[1] Evaluating if treatments can durably suppress damage across patients (pts) with varying disease duration, age and sex is important, as these factors have been linked to radiographic progression.[2] Bimekizumab (BKZ), a humanised IgG1 monoclonal antibody that selectively inhibits interleukin (IL)-17F in addition to IL-17A, has demonstrated sustained inhibition of radiographic progression in pts with PsA up to 2 years.[3] Here, the impact of BKZ on radiographic progression at 3 years in biological disease-modifying antirheumatic drug-naïve (biologic-naïve) pts with PsA is reported, both overall and across predefined subgroups.

Materials and Methods

BE OPTIMAL (NCT03895203) assessed subcutaneous BKZ 160 mg every 4 weeks (Q4W) in biologic-naïve pts with PsA. The study was placebo (PBO)-controlled to Week (Wk)16, when PBO pts switched to BKZ (PBO/BKZ). Wk52 completers could enter BE VITAL (NCT04009499; open-label extension), where all pts received BKZ.

This post hoc analysis assessed radiographic progression at baseline (BL) and 3 years (Wk148) using plain radiographs of the hands and feet, scored with the van der Heijde modified Total Sharp Score (vdHmTSS; range: 0–528, higher scores indicate greater joint damage). Radiographs were centrally read by two independent, experienced, treatment-blind readers; a third adjudicator resolved discrepancies. Subgroup analyses included pts at higher risk of progression (at-risk set; high-sensitivity C-reactive protein [hs-CRP] ≥6 mg/L and/or ≥1 bone erosion at BL), and stratified by disease duration (<1, 1–5, >5 years), age (<45, ≥45 years) and sex (male, female).

Outcomes, reported for the BKZ Total group (PBO/BKZ and BKZ-randomised pts), included proportions of pts with no radiographic progression (vdHmTSS change from BL [CfB] ≤0.5) and BL vdHmTSS, vdH joint space narrowing (JSN) and vdH Erosion. Other outcomes reported for the overall radiographic set include cumulative probability of vdHmTSS CfB, and mean CfB in vdHmTSS, vdH JSN and vdH Erosion. Analyses were descriptive and based on observed cases (OC) for pts with radiographs at Wk148.

Results

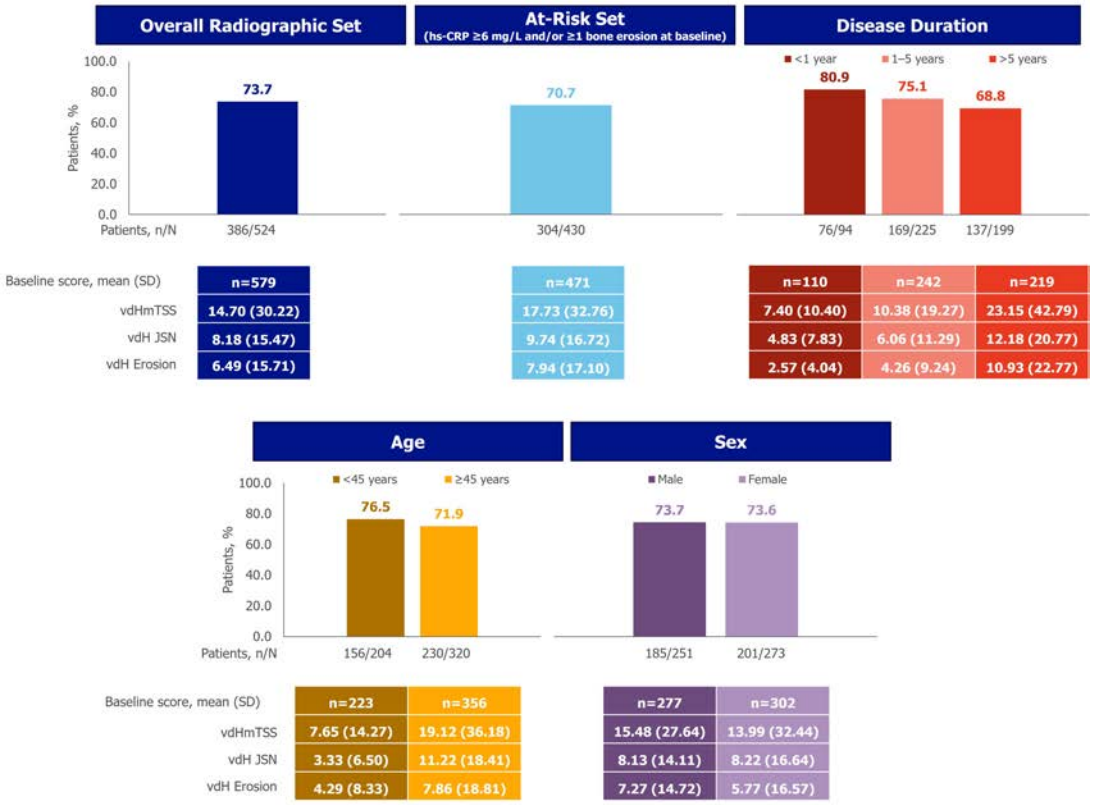
Of 712 BKZ Total pts, 579 had radiographs at BL and ≥1 post-BL timepoint, forming the overall radiographic set; of these, 524 had radiographs at Wk148. Radiographic progression was minimal to Wk148 in the overall radiographic set (mean CfB [standard deviation (SD)] vdHmTSS: 0.47 [2.69]; vdH JSN: 0.24 [1.25]; vdH Erosion: 0.27 [1.70]), and 73.7% pts had no radiographic progression at Wk148 (**Figure 1A**).

High proportions of at-risk pts and pts across subgroups also experienced no radiographic progression at Wk148 (**Figure 1A**; at-risk set: 70.7%). Higher proportions of pts with shorter disease duration vs longer disease duration had no radiographic progression at Wk148 (<1 year: 80.9%; 1–5 years: 75.1%; >5 years: 68.8%). A greater proportion of pts aged <45 years achieved no radiographic progression (76.5%) than those ≥45 years (71.9%). Proportions of pts with no radiographic progression were comparable between sexes (males: 73.7%; females: 73.6%).

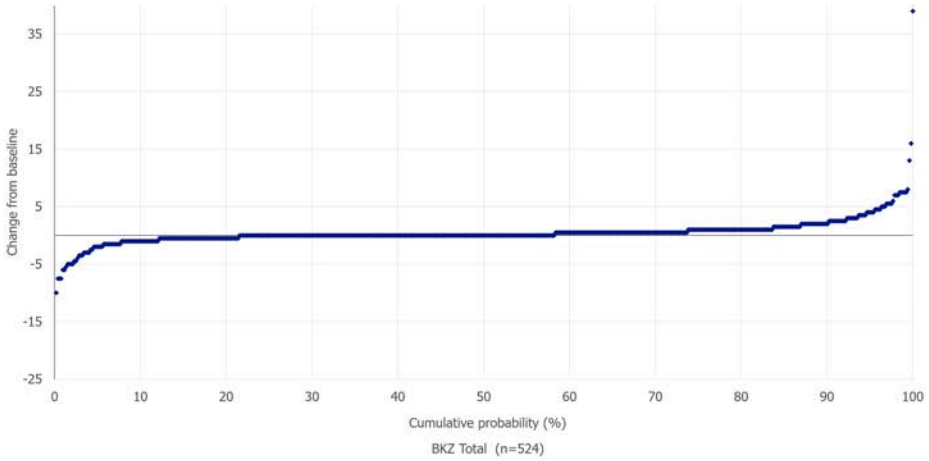
The cumulative probability of structural progression remained minimal, with most pts showing no progression (**Figure 1B**). These findings were consistent with those reported at 2 years.[3]

Figure 1. Radiographic progression in BKZ Total patients at Week 148 (OC)

A) Proportion of patients with no radiographic progression at Week 148



B) Cumulative probability of vdHmTSS change from baseline by patient in the overall radiographic set at Week 148



Radiographic set. Data are reported as observed case. Baseline values are reported for all patients with radiographs available at baseline and ≥1 post-BL timepoint (overall radiographic set: n=579). Data at Week 148 are reported for patients who had radiographs available at Week 148 (overall radiographic set: n=524). BKZ Total includes BKZ-randomised patients and PBO patients that switched to BKZ at Week 16. BKZ: bimekizumab; hs-CRP: high-sensitivity C-reactive protein; OC: observed case; SD: standard deviation; vdHmTSS: van der Heijde modified Total Sharp Score.

Conclusions

BKZ was associated with minimal radiographic progression over 3 years in biologic-naïve pts with PsA, with consistent efficacy across clinically relevant subgroups. The highest proportions of pts with no radiographic progression were among those with a shorter disease duration and aged <45 years. Overall, the likelihood of meaningful structural progression remained low over the 3-year treatment period.

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Abstract N°: 3162

Infliximab Treatment of Resistant Anogenital Ulcers in Behçet's Disease

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Introduction

Behçet's disease (BD) is a multisystemic inflammatory disease characterized by recurrent oral aphthous ulcers, genital ulcers, skin lesions, uveitis, joint involvement, and vascular lesions, with a long course accompanied by attacks, and can affect numerous organs. It was first described in 1937 by Hulusi Behçet, a Turkish dermatologist, with the trisymptom of recurrent oral aphthae, genital ulcers, and iridocyclitis with hypopyon. The disease is a significant cause of morbidity due to its involvement of the skin, mucous membranes, and other organs. On the other hand, it also increases the mortality rate. Death usually occurs due to involvement of large vessels such as pulmonary artery aneurysms, gastrointestinal system involvement, and neurological involvement. Patients with ulcers in the anogenital region are also evaluated by other departments; in particular, anal ulcers in Behçet's disease are also considered as anal fissures and fistulas. Thus, the diagnosis and treatment of the disease are delayed. The case we wanted to present was one that was both diagnosed late and resistant to treatment.

Case Presentation

The patient first applied to us in March 2022. The 45-year-old male patient had been diagnosed with Behçet's Disease at an outside center. The patient had an ulcerated lesion in the anal region that extended into the anal canal. These lesions were very painful and caused difficulties for the patient in walking and defecation. The patient, whose anal lesions began in 2016, had been evaluated by the general surgery department in 2019 as having an anal fistula and ulcer at the fistula opening. Magnetic resonance imaging had revealed "no findings suggestive of perianal fistula or abscess." Rectosigmoidoscopy had also performed, and the result had reported as "no mucosal pathology." The patient had subsequently underwent sphincterotomy, but the symptoms hadn't improved after the surgery, and the ulcerated lesion had persisted despite the operation. At an external dermatology center, a biopsy had been taken from the patient's ulcerated anal lesion due to suspicion of Crohn's ulcer and Behçet's genital ulcer, and the report had stated that it could be consistent with Behçet's disease. Upon presentation to our clinic, the patient's history revealed that they had initially been treated with methylprednisolone and colchicine at an external center. The methylprednisolone dose had reduced and then discontinued, and the patient had been followed up with colchicine for two months. Due to a lack of improvement in treatment and the presence of diarrhea, colchicine treatment had also discontinued, and azathioprine treatment had been initiated. Initially, the patient had experienced regression of lesions and improvement in symptoms with azathioprine treatment. However, upon presentation to our clinic, the patient's symptoms had worsened despite azathioprine treatment. Therefore, the patient was switched to infliximab treatment. Significant improvement in lesions was observed with infliximab treatment, and the patient is currently continuing with infliximab.

Conclusion

Genital ulcers are the second main symptom in the diagnostic criteria of Behçet's Syndrome, and the observation of scarring is particularly specific to BH. Fistula development can also be seen in 5-10% of Behçet's patients. Patients with ulcerative lesions in the anogenital region may be followed up for a long time with different

diagnoses. In our case, the patient had been followed up and treated with different diagnoses for about 5 years, and even had underwent sphincterotomy surgery, but hadn't benefit from any treatment. Finally, the patient had applied to the dermatology outpatient clinic and had been diagnosed with Behçet's disease and started treatment. In this case, we tried to emphasize the importance of appropriate treatment in improving the patient's symptoms and quality of life in a patient who presented with ulcers in the anal canal and around the anus and was resistant to treatment.

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Abstract N°: 3166

Real-World Adherence to Subcutaneous Biologics in Psoriasis: The Hidden Gap Between Drug Dispensing and Observed Response

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Introduction

Biologic efficacy in moderate-to-severe psoriasis is usually reported at nominal timepoints in clinical trials, assuming complete treatment adherence. In routine care, however, clinical response may reflect not only drug effectiveness but also real cumulative exposure, particularly for subcutaneous biologics dispensed through hospital pharmacies. The goal of this project is to assess real-world adherence to subcutaneous biologics in moderate-to-severe psoriasis and its association with clinical response.

Materials and Methods

We performed a retrospective single-centre study of adults with moderate-to-severe psoriasis who started a subcutaneous biologic between January 2021 and November 2025. Patients treated for less than 3 months were excluded. Clinical, epidemiological, treatment and outcome data were collected from routine care. Adherence was calculated in predefined time windows as the ratio between dispensed and expected units according to the approved dosing schedule. Outcomes included PASI, BSA and DLQI. Treatment groups with fewer than 30 valid cases were excluded from comparative analyses.

Results

Of 590 patients in the dispensing programme and 10,162 dispensing records, 284 patients and 361 treatment lines were included. The analysed biologics were adalimumab, guselkumab, ixekizumab, risankizumab and secukinumab. Mean age was 49.8 years, 60.2% were male, and mean BMI was 27.8 kg/m^2. Baseline median PASI, BSA and DLQI were 7.0, 6.8% and 10, respectively.

Baseline profiles differed between treatment groups. Metabolic comorbidity, previous malignancy and tuberculosis history were more frequent among patients receiving interleukin-23 inhibitors. Psoriatic arthritis was more frequent among those receiving interleukin-17 inhibitors.

Overall, 73.8% of treatment lines were ongoing at the end of follow-up. The main reasons for discontinuation were secondary failure, primary failure and adverse events. Dose modification occurred in approximately 30% of treatment lines. De-intensification rates varied across biologics.

Adherence was high during the first 6 months, but declined thereafter. It also differed substantially between treatments. Continuous correlations between adherence and PASI, BSA or DLQI were weak, probably due to ceiling effects and skewed outcome distributions during follow-up. However, in the 18–24-month window, higher adherence was independently associated with a greater probability of achieving PASI <3 at 2 years. This association remained after adjustment for biologic and baseline PASI. The estimated odds ratio was approximately 1.5 for each 0.1 increase in adherence. Dose modification was associated with poorer overall adherence, not

improved medication collection.

Conclusions

Real-world adherence to subcutaneous biologics in psoriasis is variable and declines over time. Clinical effectiveness should not be interpreted without considering actual drug exposure. Adherence monitoring may help explain suboptimal responses and guide decisions on treatment continuation, dose de-intensification or switching.

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Abstract N°: 3189

From inflammation to malignancy: Rare malignant transformation of lichen planus hypertrophicus into advanced cSCC treated with cemiplimab

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Introduction

Lichen planus is a chronic T-cell-mediated inflammatory disease affecting skin and mucosa. While malignant transformation is well-documented in mucosal forms, particularly oral lichen planus, cutaneous variants, especially *lichen planus hypertrophicus,* rarely undergo malignant transformation. Fewer than 100 such cases have been described in the global literature to date. Cutaneous squamous cell carcinoma (cSCC) is the second most common non-melanoma skin cancer, and although frequently treatable with surgery or radiotherapy, it can present as locally advanced and inoperable. Cemiplimab, a PD-1 inhibitor, is the first systemic therapy approved for such advanced cSCC. We report a unique case of advanced cSCC arising in long-standing *lichen planus hypertrophicus*, successfully treated with cemiplimab.

Case Presentation

A 58-year-old woman presented with a 10-year history of livid, hyperkeratotic plaques over the anterior and lateral aspects of both lower legs, accompanied by onychomadesis and pruritus. No previous dermatological examination had been performed, and lesions were intermittently self-treated with neutral ointment. In the last three months, she developed two rapidly growing exophytic tumor masses over the lateral malleoli (8 and 20 cm in diameter). A biopsy confirmed poorly differentiated (G3) squamous cell carcinoma. Multislice CT and PET/CT showed suspected tumor infiltration of the left calf muscles and pathological lymphadenopathy in both groins and pelvis without systemic metastases. Ultrasound-guided fine needle aspiration of the inguinal nodes revealed reactive lymphocytic hyperplasia; immunophenotyping was advised but not completed. Based on the findings and the recommendation of the multidisciplinary tumor board, systemic immunotherapy with cemiplimab was initiated. The patient received five cycles of cemiplimab over three months at three-week intervals. After the first dose, significant clinical improvement was observed, with spontaneous necrosis and detachment of ulcerated exophytic tumors. Large ulcerations (up to the size of a man’s palm) remained and were managed with local wound care. A follow-up PET/CT is planned after completion of treatment to assess therapeutic response.

Conclusion

Malignant transformation of *lichen planus hypertrophicus* into cSCC is extremely rare, with fewer than 100 cases reported in the literature. This case underscores the importance of long-term dermatologic monitoring in patients with chronic inflammatory dermatoses and emphasizes the transformative role of cemiplimab in the management of advanced inoperable cutaneous squamous cell carcinoma. The rapid and marked response to cemiplimab highlights its value in treating locally advanced, inoperable cSCC, even when arising from uncommon precursor lesions like *lichen planus hypertrophicus*.

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Abstract N°: 3298

Cutaneous Toxicity of the Dasatinib-Blinatumomab Combination: A Case Report of an Acneiform Eruption

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Introduction

The treatment of Philadelphia chromosome-positive (Ph+) Acute Lymphoblastic Leukemia (ALL) has been revolutionized by "Chemo-Free" protocols, combining Dasatinib (a BCR-ABL/SRC tyrosine kinase inhibitor) and Blinatumomab (a bispecific CD19/CD3 BiTE antibody). While this strategy achieves excellent molecular response rates, it exposes patients to a profile of cumulative toxicities that remain poorly documented. We report the case of a 49-year-old patient presenting with an acneiform eruption, which may suggest an inflammatory synergy between Dasatinib and Blinatumomab.

Case Presentation

A 49-year-old female patient, who has been diagnosed with an acute lymphoblastic leukemia Ph+, was started on a protocol including Dasatinib (140 mg/day) and Blinatumomab. Two days after the dose escalation of Blinatumomab to 28 µg/day, the patient developed an acneiform eruption consisting of monomorphic papules and pustules on an erythematous base, primarily located on the face (nose, chin, forehead) and the upper trunk (chest and back), without associated pruritus.

The clinical presentation and the chronology of the eruption were consistent with the diagnosis of an induced acneiform rash, graded as Grade 1 according to the CTCAE criteria, based on a body surface area involvement of less than 10% and the presence of associated pruritus. The patient was successfully treated with oral doxycycline, with no interruption of the treatment.

Discussion

Although Dasatinib belongs to a new era of targeted therapies renowned for lower toxicity, its activity is not strictly tumor-specific. It can generate cutaneous side effects, including acneiform rashes, which remain rare and underreported. The pathophysiology of this rash relies on the blockade of SRC-family kinases—essential relays for intracellular EGFR signaling within keratinocytes. This leads to clinically significant inflammation of the pilosebaceous unit, characterized by a typical centro-facial distribution that distinguishes it from classical anti-EGFR-induced eruptions.

Blinatumomab completes this clinical picture by acting as a powerful inflammatory catalyst. By creating an artificial synapse between effector T-lymphocytes and B-cells, it induces systemic lymphocyte activation associated with a massive release of pro-inflammatory cytokines, leading to an immunological synergy with Dasatinib: the "double-hit" theory.

Conclusion

Reporting this case is essential to document the safety profile of this new chemo-free protocol, whose synergistic toxicities are not yet well described. This observation illustrates the existence of a cutaneous double Hit, where immunotherapy reveals a molecular fragility induced by the TKI. Recognizing this side-effect profile is crucial, as severe cutaneous toxicities may necessitate treatment interruption or even permanent discontinuation.

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Abstract N°: 3351

Upadacitinib for Refractory Chronic Perianal Pruritus of Unknown Origin

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Introduction

Anal pruritus is characterized by itching of the perianal skin and affects approximately 1–5% of the adult population. When no underlying cause can be identified, chronic anal pruritus may be particularly difficult to manage and can substantially impair quality of life.[1] We report a case with chronic perianal pruritus refractory to multiple therapies who achieved sustained clinical improvement with upadacitinib.

Case Presentation

A 61 year old woman with no known comorbidities presented with 5 year history of severe pruritus confined to the perianal region. Baseline itch severity was high, with a visual analog scale (VAS) score of 8/10, numeric rating scale (NRS) score of 8/10, and a Dermatology Life Quality Index (DLQI) score of 9. The pruritus was severe enough to significantly impair her daily functioning. Dermatologic examination showed no primary lesion in perianal area. Fungal examination was negative, biopsy from the affected area showed no specific pathological findings. She had previously been evaluated by general surgery, gastroenterology, and colonoscopy findings were normal. Psychiatric evaluation revealed no contributory disorder. No underlying gastrointestinal, psychiatric, or dermatologic disorder explaining the pruritus was identified. The patient sequentially received gabapentin for 6 months, pregabalin for 6 months, cyclosporine for 3 months, aprepitant for 3 months, and dupilumab for 6 months, none of which provided clinical benefit. Repeated cryotherapy sessions and topical corticosteroids were attempted, without any improvement. Given the chronic and refractory nature of disease, upadacitinib was initiated at 15 mg once daily. Because the clinical response during the first month was limited, dose was increased to 15 mg twice daily in the second month. After dose escalation, patient experienced marked improvement, by the end of the second month her VAS, NRS, and DLQI scores had decreased to 0. Patient remains under follow up and has been receiving treatment for 7 months, with sustained benefit and no reported adverse effects.

Conclusion

Chronic perianal pruritus of unknown origin remains a diagnostic and therapeutic challenge, particularly in patients without primary skin findings and with repeatedly negative investigations.[1] A recent review suggested that the JAK/STAT signaling axis may represent a potential therapeutic target in anal pruritus, although current evidence remains limited.[1] Early experimental studies demonstrated that JAK inhibition could ameliorate pruritic inflammatory skin disease in murine models, supporting a potential role of JAK signaling in chronic itch pathogenesis.[2] Moreover, experimental data suggest that JAK1 dependent signaling in sensory neurons contributes directly to chronic itch.[3] Together, these findings provide a plausible rationale for targeting JAK1 with upadacitinib in our patient with severe refractory pruritus. These mechanisms may help explain the marked response observed despite the absence of an identifiable inflammatory dermatosis and the failure of multiple previous therapies, including dupilumab. Clinical experience with upadacitinib in chronic pruritic disorders remains limited; however, successful use has been reported in chronic pruritus secondary to polycythemia vera after dupilumab failure and in refractory chronic prurigo.[4,5] Our case suggests that JAK1 inhibition may be considered in

carefully selected patients with severe, treatment refractory chronic perianal pruritus of unknown origin. Further studies are needed to define its efficacy, optimal dosing, and long term safety in localized chronic pruritus.

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Abstract N°: 3357

Role of Azathioprine in vulvar crohn's:A Case series

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Introduction

Vulvar Crohn’s disease (VCD) is a rare extraintestinal manifestation of Crohn’s Disease characterized by granulomatous inflammation of the vulva, with or without gastrointestinal involvement. It commonly presents with vulvar edema, ulceration, fissuring, and discharge, often leading to delayed diagnosis because of its nonspecific presentation. Management remains challenging, and immunomodulators such as azathioprine play an important role in long-term disease control.

Case Presentation

Case 1: A 13-year-old female presented with vulvar perianal swelling and ulceration.Further evaluation with colonoscopy revealed ileal erosions with and on colon biopsy was suggestive of mild active ileitis and colitis . On skin biopsy showed dermal edema with mixed granulomatous inflammatory infiltrate of neutrophil, epitheliod cells,lymphocytes and giant cell in superior and deep dermis were seen.Based on clinicopathological correlation and supportive investigation diagnosis of vulvar crohn's disease was established .Treatment with azathioprine was initiated, following which marked clinical improvement was observed in vulvar swelling and ulceration.
Case 2:A 44-year-old married female presented with foul smelling discharge in the groins with deep ulcers in groins between gluteal area and lower abdomen for 4 months.She had nodular growth on vulva which was previously excised as vulvar papilloma.There was history of choroidal granuloma for which she took intravitral injections. Skin biopsy findings suggestive of vulvar crohn's disease.Based on clinicopathological correlation and supportive investigations diagnosis of vulvar crohs disease was established.Treatment with Azathioprine 2mg/kg was initiated.Following Azathioprine therapy marked clinical improvement and complete healing of ulcer was observed.

Conclusion

Conclusion:
Vulvar Crohn’s disease is an uncommon condition that may occur with or without gastrointestinal manifestations. A high index of suspicion is required for early diagnosis, particularly in young females presenting with persistent vulvar swelling and ulceration. Azathioprine appears to be an effective therapeutic option in managing vulvar Crohn’s disease. It may help achieve significant clinical improvement in both cutaneous and systemic manifestations.

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Abstract N°: 3376

Real-world experience of bimekizumab for adult patients with plaque psoriasis: A 3-year multicenter retrospective study

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Introduction
Bimekizumab is a novel interleukin (IL)-17A/IL-17F inhibitor approved for adult plaque psoriasis. Open-label extension studies have investigated the long-term efficacy and safety of bimekizumab but real-world data remains limited. Expanding upon our previously published data (weeks 16, 52 and 104), this study reports on real-world outcomes of 3-year bimekizumab use.

Materials and Methods
Our retrospective multicenter study included adult patients with plaque psoriasis from four Canadian institutions initiated on bimekizumab as per standard dosing. Effectiveness outcomes measured at 156±8 weeks included Investigator Global Assessment (IGA), Psoriasis Area and Severity Index (PASI), body surface area (BSA), and IGAxBSA scores as well as absolute PASI 3, 2, and 1 scores. Safety was assessed via treatment-related adverse events (AEs). A nonresponder imputation (NRI) analysis was used to account for missing data.

Results
This analysis included a total of 59 patients. The mean age was 46.4 (range: 19-74) years, with 64.4% (38/59) being male. At weeks 156±8 (n=59): IGA 0/1 was achieved by 96.6% (57/59); mean PASI, BSA, and IGAxBSA improvements from baseline were 94.7% (9.8 to 0.1), 94.3% (10.3 to 0.1), and 95.3% (36.6 to 0.2), respectively; 91.5% (57/59), 91.5% (54/59), and 88.1% (52/59) achieved 75%, 90%, and 100% improvements in PASI (PASI75, PASI90, and PASI100), respectively; 100% (58/58), 98.3% (57/58), and 96.6% (56/58) achieved absolute PASI scores 3, 2, and 1; and 96.6% (57/59) achieved BSA 1%. While biologic-naïve patients (n=15) had numerically lower responses than biologic-experienced patients (n=36) for IGA 0/1 (93.3% vs. 97.2%), PASI75 (86.7% vs.

94.4%), PASI90 (86.7% vs. 91.7%), and PASI100 (86.7% vs. 83.3%), and absolute PASI outcomes ≤3 (100% vs. 100%), ≤2 (93.3% vs. 100%), and ≤1 (93.3% vs. 97.1%), these differences were not significant.

Initial week 156±8 IGA 0/1, PASI75, PASI90, and PASI100 responses were maintained in 98.1% (53/54), 98.1% (52/53), 96.1% (49/51), and 93.9% (46/49) patients at week 156±8, respectively. Three patients (5.1%) utilized concomitant systemic therapy (Table 1). During the maintenance period, dose escalation from every 8 weeks to every 4 weeks was required in 6.8% (4/59) patients (mean time to dose optimization: 156.9 [range: 156.1–157.6] weeks).

In total, 21 treatment-related AEs occurred (15.3%, 9/59); most commonly oral candidiasis (10.2%, 6/59; all resolved with treatment), injection site reaction (1.7%, 1/59), molluscum contagiosum (1.7% 1/59), and pneumonia (1.7%, 1/59). There were no treatment discontinuations. No serious infections, suicidal ideation, malignancies, hypersensitivity reactions, major adverse cardiac events, or hepatic abnormalities were observed over 176.4 patient-years of safety follow-up.

Conclusions

To date, two open-label extension studies (BE RADIANT, BE BRIGHT) have evaluated bimekizumab 3-year outcomes (weeks 144 and 152). In BE RADIANT, mNRI outcomes for PASI90 and PASI100 at week 144 were 89.7% and 68.8%, respectively. In BE BRIGHT, 93.0% and 80.8% of week 16 PASI90 and PASI100 responders maintained their responses through 3 years of treatment (mNRI). Our real-world effectiveness outcomes are comparable to these results with no new safety signals. Overall, our study highlights the long-term effectiveness and safety of bimekizumab up to 3 years. Study limitations include its small sample size and retrospective nature.

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Abstract N°: 3395

Monotherapy As The Future Of Hidradenitis Suppurativa Treatment: A Meta-Review Investigating Whether Any Single Biologic Agent Can Match The Depth Of Surgical/Laser Remission And The Ethical Implications

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Introduction
Hidradenitis Suppurativa (HS) is a chronic skin condition causing inflammation, tunnel formation, and irreversible tissue damage. Biologic agents (adalimumab, certolizumab, secukinumab, bimekizumab, anakinra) help manage symptoms but do not work well for all patients. Surgical and laser treatments (deroofing, CO_2 laser, wide excision) provide longer-lasting relief by physically removing affected tissue. No single biologic has matched the depth of surgical remission. This review investigates whether any existing biologic can achieve equivalent remission and explores the ethical implications of offering medical therapy when invasive options may be superior.

Materials and Methods
A systematic meta-review of 12 randomized trials (3,247 patients), 8 real-world studies (1,892 patients), and 5 surgical series (948 patients) was conducted. Biologic outcomes were measured at 48 weeks (HiSCR50/75/90, tunnel closure). Surgical/laser outcomes were measured at 12 months (complete tunnel elimination, recurrence-free survival). A mixed-treatment comparison network meta-analysis was performed. Ethical analysis used the principlist framework (autonomy, beneficence, non-maleficence, justice).

Results
At 48 weeks, the highest-performing biologic (bimekizumab) achieved HiSCR75 in 58.3% (95% CI 52.1–64.5) and HiSCR90 in 34.7% (95% CI 29.0–40.4). Complete tunnel closure (100%) occurred in only 9.2% (95% CI 6.1–12.3%) of biologic-treated patients. In contrast, surgical/laser interventions achieved complete tunnel elimination in 89.4% (95% CI 85.6–93.2%) at 12 months ($p<0.001$). Twelve-month recurrence-free survival was 81.7% for wide excision, 74.2% for CO_2 laser, and 68.9% for deroofing. Biologic failure to achieve HiSCR90 by week 24 predicted non-response at week 48 with 86% negative predictive value. Ethical analysis revealed that most patients were not fully informed that biologics rarely achieve complete tunnel resolution, raising autonomy concerns. Justice issues emerged due to unequal access to surgical/laser care.

Conclusions
No single biologic monotherapy currently matches the depth of surgical/laser remission in HS. Biologics manage symptoms but rarely provide complete tunnel elimination, whereas surgery offers durable, near-complete disease modification. Ethical obligations require transparent shared decision-making, disclosing that biologics are suppressive, not curative. A stratified algorithm is proposed: Hurley I, biologic monotherapy first-line; Hurley II–III, surgical/laser referral within 6 months if HiSCR90 not achieved.

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Abstract N°: 3403

Dupilumab as a promising treatment for Hailey-Hailey Disease: A Five-Case Series

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Introduction

Hailey-Hailey disease (HHD), or familial benign pemphigus, is a rare autosomal dominant genodermatosis affecting epidermal keratinocyte adhesion, with an estimated incidence of 1/50,000. It is an intraepidermal blistering dermatosis characterized by painful blisters, erosions, maceration, and frequent secondary infections in flexural areas. The disease follows a chronic relapsing course with limited therapeutic options.

Case Presentation

Five patients with confirmed HHD refractory to multiple conventional treatments were treated with dupilumab. All patients showed clinical improvement, characterized by reduced maceration, exudation, and erosions, with sustained lesion control in the four patients treated for over 12 months. In one of these cases, response was delayed, with obesity and personal stress discussed as potential contributing factors. The fifth patient, on treatment for 6 months, also demonstrated clinical improvement.

Conclusion

Dupilumab is a monoclonal antibody that blocks interleukin-4 and interleukin-13 signaling. Although its exact mechanism in HHD remains unclear, inhibition of the Th2 pathway and modulation of intracellular calcium regulation may contribute to the observed clinical response. All patients reported improved quality of life and no relevant adverse events, supporting dupilumab as a potentially effective and safe option for refractory HHD. Off-label use of dupilumab in HHD has been increasing, with emerging evidence suggesting a promising efficacy and safety profile. To date, only isolated case reports and small series of up to three patients have been described. This five-case series represents the largest reported to date, further strengthening the evidence for dupilumab's role in the treatment of HHD.

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Abstract N°: 3476

Refractory mucous membrane pemphigoid successfully treated with rituximab

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Introduction

Mucous membrane pemphigoid (MMP) is a chronic autoimmune blistering disorder predominantly affecting mucosal surfaces, often requiring systemic immunosuppression. Refractory cases remain challenging, highlighting the need for effective alternatives.

Case Presentation

A 73-year-old woman with rheumatoid arthritis and hypertension presented with painful oral blisters refractory to topical therapy. Lesions involved the buccal mucosa and soft palate, with
associated odynophagia. Upper endoscopy with esophageal biopsy excluded mucosal involvement.
Histopathology showed subepidermal blistering, and direct immunofluorescence revealed linear
IgA deposition along the basement membrane zone, supporting MMP and excluding pemphigus vulgaris. Prednisone (1 mg/kg/day) with azathioprine was initiated without improvement after six months. Methotrexate was subsequently introduced, also without response. Rituximab was then administered due to refractory disease. The patient showed marked clinical improvement, with reduction of lesions and pain, enabling corticosteroid tapering. She remains stable on maintenance therapy.

Conclusion

Refractory MMP is difficult to manage, as conventional agents such as azathioprine and methotrexate may fail. Although azathioprine is widely used as a steroid-sparing agent, responses are variable. Rituximab, an anti-CD20 monoclonal antibody, targets B-cell-mediated autoimmunity and has shown efficacy in refractory autoimmune blistering diseases, including MMP. This case supports rituximab as an effective option in refractory MMP, although further studies are needed to define optimal use.
MMP may be refractory to standard immunosuppression. Azathioprine shows variable efficacy. Rituximab is effective in refractory cases. Early biologic therapy may improve outcomes

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Abstract N°: 3479

Long-Term Safety of Anti-IL-4Rα Stapokibart in Chinese Adults with Moderate-to-Severe Atopic Dermatitis: A Phase II, Multicenter, Open-Label, Single-Arm Study

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Introduction
Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease that significantly impairs quality of life in patients with moderate-to-severe forms. Current treatments face challenges regarding long-term efficacy and safety. Stapokibart is a humanized anti-interleukin-4 receptor alpha (anti-IL-4Rα) monoclonal antibody that has shown promising efficacy and safety in adults with moderate-to-severe AD. This study aimed to further evaluate the long-term safety of stapokibart in this population.

Materials and Methods
This was a multicenter, open-label, single-arm phase II trial (NCT06116565). The study included a 4-week screening period, a 52-week treatment period, and an 8-week safety follow-up period. Patients received a first dose of stapokibart 600 mg, followed by 300 mg every two weeks (Q2W). Dosing interval adjustments were made at weeks 12 and 36 based on clinical response. At week 12, fast responders (achieved ≥75% improvement from baseline in Eczema Area and Severity Index score [EASI-75] or Investigator's Global Assessment score of 0/1 [IGA 0/1]) were switched to every four weeks (Q4W), while slow responders (achieved EASI-50 but not EASI-75 or IGA 0/1) continued Q2W; nonresponders (failed to achieve EASI-50) were withdrawn from the study. At week 36, responders (achieved EASI-90 or IGA 0/1) on Q2W were switched to Q4W, responders on Q4W were switched to every eight weeks (Q8W), and non-responders maintained their regimen. The incidence and per-100-person-year incidence rate of treatment-emergent adverse events (TEAEs) were analyzed.

Results
A total of 262 patients were enrolled. Among them, 16 patients (6.1%) were classified as having poor efficacy by week 12 or withdrew early before week 12. The overall TEAE incidence was 86.3% (226/262). Treatment-related TEAEs occurred in 38.5% (101/262) of patients, all of which were mild or moderate. Serious adverse events (SAEs) occurred in 6.1% (16/262) of patients, none of which were treatment-related. The incidence of TEAEs leading to early treatment discontinuation was 1.9% (5/262), and that leading to early study withdrawal was 1.5% (4/262). No significant differences in the incidence of TEAEs were observed among the different dosing interval extension groups (Table 1). Commonly reported TEAEs (occurring in >10% of patients in at least one group) included upper respiratory tract infection (overall 26.0%, ranging from 12.5% to 35.5% across groups), hyperuricemia (7.6%, 4.3% to 16.1%), elevated creatine phosphokinase (5.0%, 2.1% to 15.6%), elevated alanine aminotransferase (4.6%, 3.2% to 12.5%), and allergic conjunctivitis (1.9%, 0% to 18.8%). The overall TEAE person-year incidence rate was 344.0 per 100 person-years, with rates varying across dosing adjustment groups: 428.7 (Q2W-Q2W-Q2W), 302.5 (Q2W-Q2W-Q4W), 305.8 (Q2W-Q4W-Q4W), and 313.4 (Q2W-Q4W-Q8W) per 100 person-years.

Conclusions
Long-term treatment with stapokibart for up to 52 weeks demonstrated a favorable and manageable safety profile in patients with moderate-to-severe AD, with a low rate of treatment discontinuation and no new safety signals. Extended dosing intervals were associated with lower incidence rates of TEAEs. These findings support the long-term safety of stapokibart as a maintenance therapy.

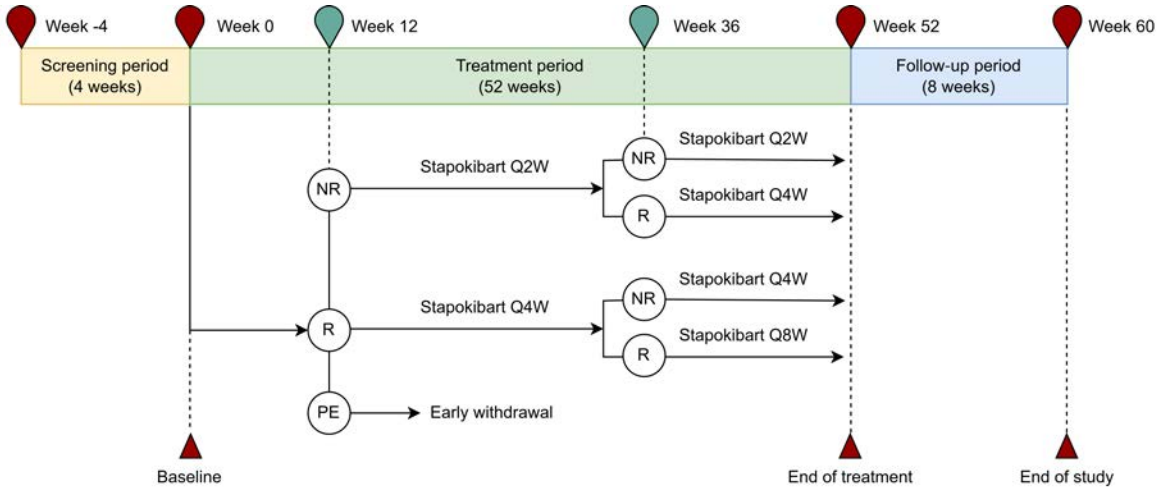

Figure 1. Schematic overview of the trial design. R, response; NR, non-response; PE, poor efficacy; Q2W, every two weeks; Q4W, every four weeks; Q8W, every eight weeks

n (%)	Early withdrawal at or before week 12 (n=16)	Q2W-Q2W-Q2W (n=31)	Q2W-Q2W-Q4W (n=28)	Q2W-Q4W-Q4W (n=32)	Q2W-Q4W-Q8W (n=141)	Total (n=262)
Any TEAEs	12 (75.0)	28 (90.3)	26 (92.9)	28 (87.5)	121 (85.8)	226 (86.3)
Mild	9 (56.3)	27 (87.1)	26 (92.9)	27 (84.4)	112 (79.4)	212 (80.9)
Moderate	7 (43.8)	14 (45.2)	11 (39.3)	9 (28.1)	52 (36.9)	98 (37.4)
Severe	2 (12.5)	3 (9.7)	0	0	3 (2.1)	8 (3.1)
Treatment-related TEAEs	6 (37.5)	14 (45.2)	12 (42.9)	11 (34.4)	51 (36.2)	101 (38.5)
Mild	5 (31.3)	13 (41.9)	10 (35.7)	11 (34.4)	46 (32.6)	92 (35.1)
Moderate	4 (25.0)	3 (9.7)	3 (10.7)	2 (6.3)	16 (11.3)	29 (11.1)
Severe	0	0	0	0	0	0
TEAEs leading to early treatment discontinuation	2 (12.5)	0	0	0	1 (0.7)	5 (1.9)
TEAEs leading to early withdrawal	2 (12.5)	0	0	0	0	4 (1.5)

Table 1. Overview of adverse events. TEAE, treatment-emergent adverse events.

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Psoriasiform Presentation of MDA5-Dermatomyositis: A Rare Case of Exacerbation Following IL-17A Blockade

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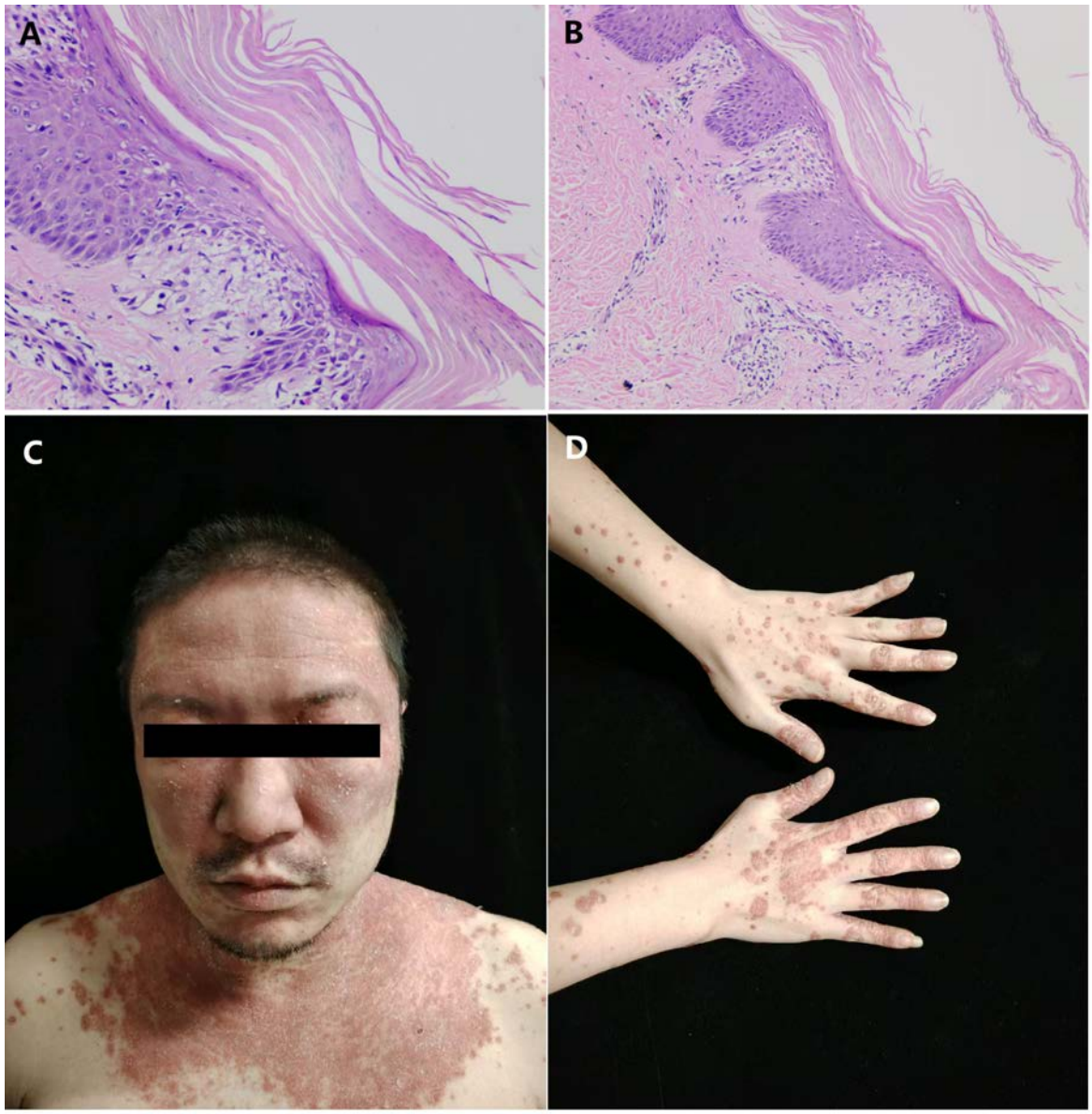
Introduction

With the advent of the biologic era, interleukin-17 inhibitors (IL-17i) have revolutionized the treatment of moderate-to-severe plaque psoriasis (PSO). However, the widespread adoption of these targeted therapies necessitates heightened vigilance regarding differential diagnoses and paradoxical reactions. We herein report a rare case of MDA5-positive psoriasiform dermatomyositis (MDA5-PDM) who developed exacerbation of the cutaneous eruption and rapidly progressive-interstitial lung disease（RP-ILD）following IL-17i administration. This case highlights that psoriasiform eruption can be an initial sign of this specific dermatomyositis subtype and biologics should be used with extreme caution before a definitive diagnosis is made.

Case Presentation

A 35-year-old man presented with a 2-year history of psoriasiform plaques, leading to an initial diagnosis of moderate-to-severe PSO, which was supported by skin biopsy showing epidermal hyperkeratosis with parakeratosis and acanthosis. (Fig. 1A) He failed topical therapies and received two doses of ixekizumab (IXE). Paradoxically, he subsequently developed worsening skin lesions, fever, and dyspnea. Examination revealed heliotrope rash, Gottron's papules with equivocal Auspitz sign. (Fig. 1C, D) Manual muscle testing revealed grade 4/5 strength in lower extremities. Re-evaluation of the initial biopsy showed interface dermatitis, inconsistent with psoriasis. (Fig. 1B) Serology showed elevated muscle enzymes and a positive ANA. Imaging confirmed myositis and interstitial lung disease. Further antibody testing returned positive for anti-MDA5 and anti-PL-7. A diagnosis of MDA5-PDM was made. Immunosuppressive therapy with corticosteroids, cyclophosphamide, and immunoglobulin led to significant improvement in pulmonary and cutaneous symptoms, allowing discharge. However, due to myositis persists, glucocorticoids were not fully withdrawn until 15 months later.



A (×100): Mild hyperkeratosis of the epidermis accompanied by small foci of mild ankeratosis. Lymphocyte infiltration around some small blood vessels in the superficial to deep layers of the skin, mainly in the superficial layer. B (×200): interface dermatitis. C: Heliotrope sign: presents as purple-red erythema on both upper eyelids and periorbital area, which may be accompanied by edema. D: Gottron sign, symmetrical, fused erythema or purplish red patches distributed on the metacarpophalangeal joints, interphalangeal joints.

Conclusion

PDM refers to dermatomyositis presenting with psoriasiform skin lesions or arthritis, but lacking other evidence of psoriasis. Due to its clinical rarity and the potential absence or delayed onset of muscle involvement, this condition is prone to misdiagnosis as PSO by dermatologists.Only three similar cases have, thus far, been described in the literature. The clinical characteristics and shared features of these cases are summarized in Table 1. The presence of Gottron's papules, heliotrope sign, positive result for anti-MDA5 antibodies and elevated muscle enzymes warrants a high index of suspicion for PDM. Although the precise mechanism by which IL-17i may exacerbate DM, in our case, remains unclear, previous studies have documented similar concerns and proposed the “cytokine transfer” hypothesis. As DM and PSO share common inflammatory cytokines, including IL-17, TNF-α and IFNs, biologics targeting one cytokine may indirectly break the inflammatory equilibrium, resulting in the accumulation of other inflammatory cytokines and an elevated risk for DM. PDM still represent a clinical challenge because of its rarity and psoriasis-mimicking skin manifestations.

Case	Gender / Age	Clinical Presentation	Physical Examination	Laboratory / Imaging Findings	Treatment	Outcome
1	F, 41	Psoriasiform lesions for 6 months, then diagnosed with psoriatic arthritis. After anti-TNF therapy, developed proximal muscle weakness and systemic symptoms.	scaly erythematous, Gottron's papules, heliotrope rash, panniculitis, mild proximal weakness, polyarthritis.	Elevated muscle enzymes (CK, LDH). Positive anti-MDA5 antibodies. Muscle biopsy: intense MHC-I expression. HRCT: ILD. CT: calcinosis cutis.	Initially: adalimumab. After diagnosis: analgesics, TCS, IVIG, TCI and alendronate.	Skin inflammation improved, but calcinosis persisted.
2	M, 20	Fever, arthritis, rash, and proximal muscle weakness for 7 months.	psoriasiform plaques. Gottron's papules, Heliotrope rash. Manual muscle testing score: 56/80.	Elevated muscle enzymes (AST, ALT, LDH, CPK). Positive anti-MDA5 antibodies.	TCS and methotrexate.	Succumbed to sudden death one month into illness.
3	F, 53	Psoriasiform rash for 3 years, followed by exertional dyspnea for 6 months. proximal muscle weakness for 3 weeks and	scaly erythematous, muscle weakness, Gottron's papules	Moderately raised inflammatory markers, markedly elevated muscle enzymes. Positive anti-Jo-1 antibodies. HRCT: ILD. Muscle biopsy: consistent with dermatomyositis.	TCS, azathioprine/mycophenolate mofetil	Complete clinical recovery of skin and muscle disease within 8 weeks.

Table 1 Clinical Characteristics of the Reported Cases

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Abstract N°: 3511

EPSTEIN–BARR VIRUS REACTIVATION IN A CHILD WITH GENERALIZED PUSTULAR PSORIASIS TREATED WITH SECUKINUMAB

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Introduction

Generalized pustular psoriasis (GPP) of von Zumbusch is a rare dermatological emergency in children, involving the IL-36 pathway. While secukinumab (anti-IL-17A) has shown rapid efficacy, its impact on the antiviral immune response and the risk of latent virus reactivation, such as EBV, remains incompletely defined.

We report a rare case of EBV reactivation occurring shortly after initiation of secukinumab in a child with severe von Zumbusch GPP, subsequently complicated by acute generalized exanthematous pustulosis induced by amoxicillin–clavulanic acid. This observation highlights the diagnostic and therapeutic challenges associated with severe pustular eruptions in immunomodulated pediatric patients.

Case Presentation

A 11-year-old girl with plaque psoriasis associated with Addison's disease was admitted for severe von Zumbusch GPP presenting with erythroderma, diffuse sterile pustules, fever, and marked impairment of general condition. Dermoscopy revealed a homogeneous dotted vascular pattern, and clinical examination showed associated scalp involvement and geographic tongue.

Initial treatment with acitretin and methotrexate was insufficient, leading to the introduction of secukinumab with rapid clinical improvement. One week after initiation, the patient developed persistent fever, asthenia, facial edema, and hepatic cytolysis. Investigations demonstrated Epstein–Barr virus reactivation confirmed by positive EBV PCR with high viral load, after exclusion of alternative infectious, autoimmune, and drug-induced causes. Secukinumab was temporarily discontinued, with favorable clinical and biological evolution.

Subsequently, the patient developed acute generalized exanthematous pustulosis induced by amoxicillin–clavulanic acid, confirmed by histopathology and pharmacovigilance assessment. Evolution was rapidly favorable after treatment withdrawal. Secukinumab was successfully reintroduced without recurrence. After one year of follow-up, the patient remains in complete remission on maintenance therapy.



Multiple small, milky, non-follicular pustules diffusely distributed over the entire skin surface, arising on an erythematous background

Conclusion

The pathophysiology of GPP, particularly in severe forms potentially linked to IL36RN mutations, differs significantly from plaque psoriasis. In this case, the GPP flare was likely exacerbated by acute EBV infection. Since IL-17 plays a role in antiviral defense, its inhibition may promote the reactivation of latent viruses. This interaction between immune activation and GPP-related inflammation highlights the diagnostic complexity of distinguishing between a viral-induced GPP flare and a drug-induced eruption like AGEP.

This case highlights the necessity of careful monitoring and prior viral screening in children treated with secukinumab. It also demonstrates that biological therapy can be safely reintroduced after managing intercurrent complications, provided there is rigorous multidisciplinary follow-up.

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Abstract N°: 3529

Disease signatures distinguishing baricitinib responders versus non-responders in alopecia areata: evidence for persistent Th1 inflammation and cytotoxic/IFN effector activity

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Introduction
Baricitinib (bari), an oral JAK1/2 inhibitor, suppresses immune-mediated follicular inflammation in alopecia areata (AA), driving hair regrowth in the majority of patients. However, 20–35% of individuals exhibit primary non-response, and the immunomolecular determinants of JAK inhibitor resistance remain poorly defined.

In this exploratory pilot study, we aim to characterize differential disease signatures in bari-treated AA patients stratified by clinical response, using transcriptomics, computational immune profiling, and immunoflourescence.

Materials and Methods
Twelve patients with moderate-to-severe AA (SALT scores 50–100) received baricitinib 4 mg/day for 24 weeks. Ten patients (4F/6M; age 22–58 years) achieved clinical response; two patients (2F; age 47 and 59 years; SALT scores 97.9 and 100) were non-responders. Scalp biopsies were analyzed by bulk RNA-sequencing (RNA-Seq), ALADIN (Alopecia Areata Disease Activity Index) computational scoring, T helper (Th) cell transcriptional profiling, and immunofluorescence (IF) for $CD3^+$, $CD8^+$, $NKG2D^+$ infiltrates and MHC class I expression.

Results
RNA-Seq identified differential regulation of innate immune/inflammatory mediators (*CCL24*, *FCGR3*) and extracellular matrix/cell adhesion genes (*COL11A2*, *THBS4*) exclusively in non-responders. ALADIN scores were elevated at baseline in non-responders; only responders demonstrated post-treatment attenuation of cytotoxic T lymphocyte (CTL) and interferon (IFN) signaling alongside increased keratin (KER) transcriptional activity. Th profiling revealed a shift from Th1/Th2 toward Th17/Th22 predominance in responders, whereas non-responders maintained persistent Th1 activity with progressive Th17/Th22 emergence and sustained Th2 suppression. IF demonstrated higher baseline $CD3^+$ and $CD8^+$ (including $NKG2D^+$) perifollicular infiltrates in non-responders. Post-treatment reduction in intra- and perifollicular T-cell density, particularly $NKG2D^+$ cytotoxic effectors, was restricted to responders. Higher baseline MHC class I expression was observed exclusively in responders compared to healthy donors, along with its downregulation, predominantly in the follicular epithelium, after drug treatment.

Conclusions

Despite the limited cohort size, these data suggest that JAK inhibitor resistance and reduced clinical efficacy arise from persistent Th1 inflammation and compensatory cytotoxic/IFN effector activity. These transcriptomic and immunophenotypic signatures may represent actionable biomarkers for patient stratification and help identify rational targets for combination strategies aimed at overcoming JAK inhibitor non-response.

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Abstract N°: 3538

Nemolizumab in prurigo nodularis: real-world efficacy and comorbidity profile - a single-centre experience

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Introduction

Prurigo nodularis (PN) is a chronic neuroimmune skin condition characterised by intensely pruritic hyperkeratotic nodules and a profound impact on quality of life. Its pathophysiology involves dysregulation of the type 2 inflammatory axis and aberrant peripheral and central sensitisation, with interleukin-31 (IL-31) playing a central role in itch signalling via sensory neurons. Nemolizumab is a monoclonal antibody targeting the IL-31 receptor A subunit, approved in 2025 in Europe for the treatment of moderate-to-severe PN in adults. Despite robust efficacy data from phase III trials, real-world experience with nemolizumab remains limited, particularly in populations with significant comorbidity that are systematically underrepresented in clinical trials.

Materials and Methods

This study is a retrospective case series of all patients with a primary diagnosis of PN initiated on nemolizumab at our centre. PP-NRS was recorded at baseline and at month 3 (M3). Demographic data, disease history, prior treatment exposure, comorbidities, dosing details, treatment persistence, and adverse events were collected. All patients received an initial loading dose of nemolizumab 60 mg subcutaneously, followed by maintenance doses of 30 mg or 60 mg every four weeks, administered according to body weigh. Atopy was defined as a documented history of atopic eczema, asthma, allergic rhinitis, or IgE-mediated food allergy. Pruritus-associated comorbidities were systematically categorised at baseline.

Results

Sixteen PN patients were included, with a mean age of 72 years (range 44–97). Mean disease duration was 52 months (range 7–132), with a mean age at onset of 68 years. Mean nemolizumab treatment duration was 5.7 months (range 1.1–11.1 months). Eight patients (50%) had at least one pruritus-associated comorbidity. Haematological conditions were the most frequent (n=4), including 2 patients with hypereosinophilic syndrome, refractory anaemia with ring sideroblasts, and essential thrombocythaemia with JAK2 mutation. Other comorbidities included liver disease (n=2), diabetes mellitus (n=2), chronic kidney disease (n=1), active or recent malignancy (n=1), and nodular pemphigoid (n=1). Six patients (38%) had an atopic background (atopic eczema n=4, asthma n=4, allergic rhinitis n=5, IgE-mediated food allergy n=1; with overlap). Prior treatment exposure was extensive: topical corticosteroids in all 16 patients (100%), topical calcineurin inhibitors in 6 (38%), phototherapy in 9 (56%), systemic corticosteroids in 3 (19%), methotrexate in 1 (6%), dupilumab in 3 (19%), and prior anti-IL-5/IL-5Rα biologics (mepolizumab n=1, benralizumab n=1) in 2 patients (13%), both treated for concurrent hypereosinophilic syndrome. Concomitant TCS were used in 4 patients at treatment initiation.

Baseline PP-NRS was available for all 16 patients (mean 9.0/10, range 7–10), reflecting a severe itch burden at treatment initiation. At M3, mean PP-NRS decreased to 1.1/10, representing a mean reduction of 7.9 points (−88%). In addition, a clinically significant reduction in nodule count was noted in all patients, though it was not formally quantified.

Nemolizumab was generally well tolerated; one patient discontinued treatment due to nummular eczema developing four months later, the only adverse event leading to discontinuation in this cohort.

Conclusions

These real-world data confirm the marked antipruritic efficacy of nemolizumab in PN, with a mean PP-NRS reduction of 88% at month 3, consistent with phase III trial outcomes and achieved in a population with extensive prior treatment failure. The favourable tolerability profile and high treatment persistence further support its use in routine clinical practice. Notably, half of this cohort presented with significant pruritus-associated comorbidities, haematological conditions in particular. These findings suggest that nemolizumab is effective and well tolerated across a broad and comorbid real-world PN population, including patient profiles typically excluded from trials.

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Abstract N°: 3550

Severe Hidradenitis Suppurativa in a Patient With Down Syndrome and Recurrent Treatment Failure: Suspected Infliximab-Induced Auditory Hallucinations

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Introduction

Hidradenitis suppurativa (HS) is a chronic, recurrent, inflammatory follicular disease that may progress to debilitating tunnels and scarring, often requiring biologic therapy in advanced stages. Patients with Down syndrome appear to have increased prevalence and severity of HS, possibly related to immune dysregulation and metabolic comorbidities, and frequently present therapeutic challenges. Although tumor necrosis factor (TNF) inhibitors are widely used in moderate-to-severe HS, neuropsychiatric adverse events such as psychosis or hallucinations have been rarely reported and remain underrecognized.

Case Presentation

We report a female patient with Down syndrome who initially presented with a predominantly follicular pattern of HS, with early nodules and limited inflammatory activity in intertriginous areas. Conservative management with topical resorcinol and clindamycin achieved only partial control, and the disease progressively evolved into a more inflammatory phenotype, approaching Hurley stage III, with extensive involvement and axillary scarring. Adalimumab was initiated, resulting in initial improvement followed by secondary loss of response. Secukinumab was then introduced, but the patient continued to worsen clinically with persistent inflammatory nodules and pain. Infliximab was started as rescue therapy, with objective improvement of HS lesions. However, from the second infusion onward, the patient's mother reported new-onset auditory hallucinations, with the patient "hearing voices". She required evaluation in the emergency department on two occasions; neurological assessment raised the suspicion of a drug-related adverse effect, and infliximab was considered the most likely trigger. Following a second episode of auditory hallucinations after infusion, infliximab was discontinued, and alternative therapeutic strategies for her advanced HS are currently under discussion.

Conclusion

This case highlights the complexity of managing severe HS in a patient with Down syndrome, with multiple biologic failures and a suspected neuropsychiatric adverse reaction to infliximab. Awareness of potential central nervous system events, including auditory hallucinations, is essential when prescribing TNF inhibitors, particularly in vulnerable populations. Careful risk–benefit assessment and close collaboration between dermatology, neurology and psychiatry are crucial when selecting systemic therapies for advanced HS with atypical adverse events.

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Abstract N°: 3556

Comparative Durability Of Response After Biologic Withdrawal: A Systematic Review Of Icotrokinra, Nemolizumab, And Bimekizumab Rebound Data. Defining A New "Treatment-Free Remission Probability Score"

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Introduction
Modern biologics for psoriasis, atopic dermatitis, and asthma achieve high response rates, but what happens after withdrawal remains poorly understood. Icotrokinra (IL-23 inhibitor), nemolizumab (IL-31RA antagonist), and bimekizumab (IL-17A/F inhibitor) show distinct rebound and relapse patterns after discontinuation. No standardized tool exists to predict which patients will maintain remission without maintenance dosing. This review compares post-withdrawal durability across these three agents and introduces a novel "Treatment-Free Remission Probability Score" (TFR-PS).

Materials and Methods
A systematic search of MEDLINE, EMBASE, and Cochrane Central (2018–2026) identified 11 clinical trials (n=2,834 patients) with published post-treatment follow-up data: icotrokinra (4 trials, n=1,047), nemolizumab (3 trials, n=672), and bimekizumab (4 trials, n=1,115). Rebound was strictly defined as ≥50% loss of PASI-75 (or EASI-75) within 12 weeks of withdrawal, while relapse was defined as loss of PASI-50 (or EASI-50) within 24 weeks. Time-to-loss-of-response curves were extracted, and Cox proportional hazards modeling identified predictors of prolonged treatment-free remission. The proposed TFR-PS integrates baseline disease severity, prior biologic exposure, and on-treatment response depth.

Results
Bimekizumab showed the fastest rebound, with 34% of patients experiencing PASI-50 loss by week 8 post-withdrawal (median time to relapse 11.3 weeks, 95% CI 9.8–12.9). In contrast, icotrokinra demonstrated the most durable treatment-free remission: only 19% relapsed by week 24 (median time to relapse 38.7 weeks, 95% CI 32.1–45.2, p<0.001 vs. bimekizumab). Nemolizumab showed intermediate durability (median relapse 22.4 weeks, 95% CI 19.3–26.1). Among patients achieving complete skin clearance (PASI-100 or IGA 0/1) at withdrawal, 58% on icotrokinra remained relapse-free at 52 weeks versus 29% on bimekizumab (p=0.002). Key predictors of longer treatment-free remission included: treatment duration >24 weeks prior to withdrawal (HR 0.47, p<0.001), absence of prior biologic failure (HR 0.61, p=0.004), and on-treatment complete response (HR 0.35, p<0.001). The TFR-PS (range 0–12) stratified patients into three risk groups: low (score 0–4, 1-year TFR probability 68%), intermediate (score 5–8, 1-year TFR 41%), and high (score 9–12, 1-year TFR 19%). External validation on two independent cohorts (n=412) showed good calibration (Hosmer-Lemeshow p=0.21) and discrimination (AUC 0.84, 95% CI 0.79–0.89).

Conclusions
Durability after biologic withdrawal varies significantly by agent and patient profile. Icotrokinra offers the longest treatment-free remission, while bimekizumab requires closer post-withdrawal monitoring. The proposed TFR-PS

provides a clinically usable tool to guide shared decisions about treatment interruption or dose de-escalation. These findings support individualized withdrawal strategies rather than uniform discontinuation protocols.

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Abstract N°: 3581

Dupilumab as a Potential Trigger for Seronegative Arthritis: A Case Report

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Introduction

Dupilumab is a human monoclonal antibody which inhibits signalling of interleukin-4 and interleukin-13, type 2 cytokines that may be important drivers of atopic or allergic diseases such as atopic dermatitis[1]. Dupilumab is highly effective in treating eczema, with patients achieving at least a 75% improvement in the Eczema Area and Severity Index by week 16[1]. It also improves other outcomes, including itch, and overall quality of life in patients with atopic dermatitis[1]. However, joint-related complications associated with dupilumab are likely underreported. This case report highlights a clinical occurrence of this adverse effect.

Case Presentation

A 46-year-old man with a long-standing history of atopic eczema was started on dupilumab for disease control. Eight weeks after initiation, he developed acute right knee pain, which was preceded by five weeks of joint swelling, prompting attendance at the emergency department. Knee aspirate, bloods (including rheumatoid factor, anti-CCP antibody and connective tissue disease screen) and x-ray were unremarkable. MRI knee showed a moderate effusion with synovial hypertrophy (Figure 1) consistent with an inflammatory or degenerative process.

His past medical history includes a right knee arthroscopy 25 years ago for synovitis, during which he underwent a washout and intra-articular steroid injection, after which he had complete resolution for two decades. His other past medical history includes asthma and hypertension.

Given the new monoarticular inflammatory symptoms, dupilumab was discontinued temporarily and he was referred to rheumatology, where he was subsequently diagnosed with a seronegative inflammatory arthritis of the right knee. Intra-articular steroid injection provided immediate relief, and his symptoms improved significantly following cessation of dupilumab. Rheumatologists felt that dupilumab could have been a contributing factor.

Of note, emerging case reports in the literature have described dupilumab-associated inflammatory arthritis and enthesitis, including early case series outlining new-onset seronegative arthropathy in patients receiving dupilumab for atopic disease[2] and mechanistic reviews proposing that IL-4/IL-13 blockade may unmask or trigger Th17-driven inflammatory pathways linked to seronegative arthritis[3].

In this context, our patient's presentation may represent dupilumab-induced monoarthritis, potentially facilitated by his prior joint pathology. Although a rare complication, there is literature linking dupilumab with synovitis and monoarthritis[2-4]. More recently, refractory cases requiring advanced immunomodulatory therapy, including successful treatment with the JAK inhibitor upadacitinib, have been reported[4]. The ongoing management plan for our patient is to initiate upadacitinib, which would address both his inflammatory arthritis and eczema[4].

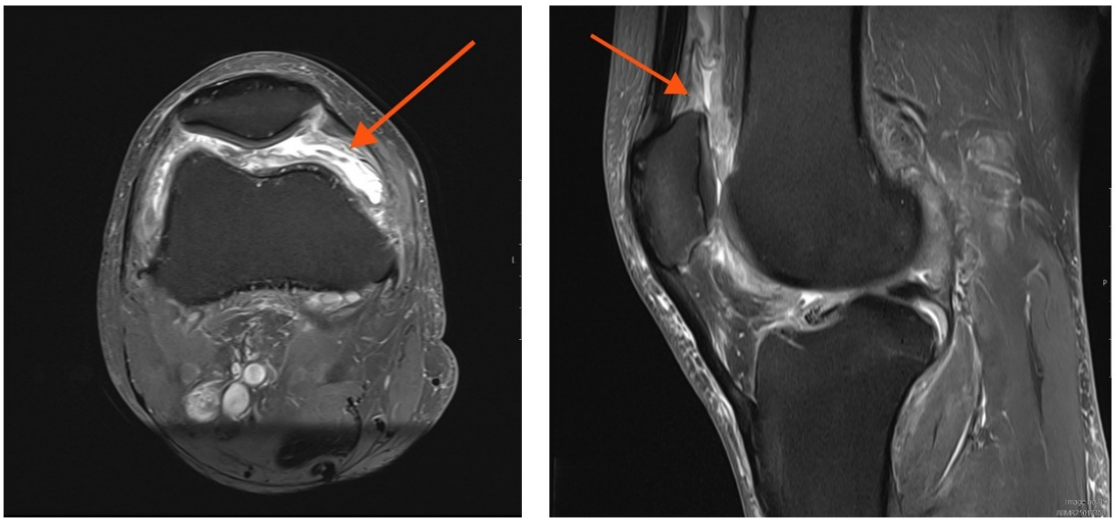

Figure 1. Short tau inversion recovery (STIR) MRI sequences of the right knee in axial and sagittal planes demonstrating joint effusion with increased T2 signal intensity (bright white) and associated synovial thickening. Prominent synovial bands and effusion are visualized, findings that are not normally present in a healthy knee joint.

Conclusion

This case highlights the importance of considering immune-modulating biologics such as dupilumab as potential triggers for new-onset inflammatory joint disease, particularly in patients with prior joint pathology.

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Exosomes in Skin of Color: Dermatological Insights, Therapeutic Potential, and Clinical Realities

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Introduction

Exosomes are nanosized extracellular vesicles released by multiple cell types and involved in intercellular communication through the transfer of proteins, lipids, messenger RNA, microRNA, and other signaling molecules. Increasing experimental evidence suggests that exosomes participate in key cutaneous processes including inflammation, melanogenesis, angiogenesis, extracellular matrix remodeling, fibroblast activation, immune modulation, wound healing, and hair follicle cycling. These biological properties have generated considerable interest in the use of exosome-based therapies in regenerative and aesthetic dermatology. Their appeal is particularly strong in India and other skin-of-color populations, where pigmentary disorders, acne scarring, alopecia, and post-inflammatory dyschromia represent common and therapeutically challenging conditions.

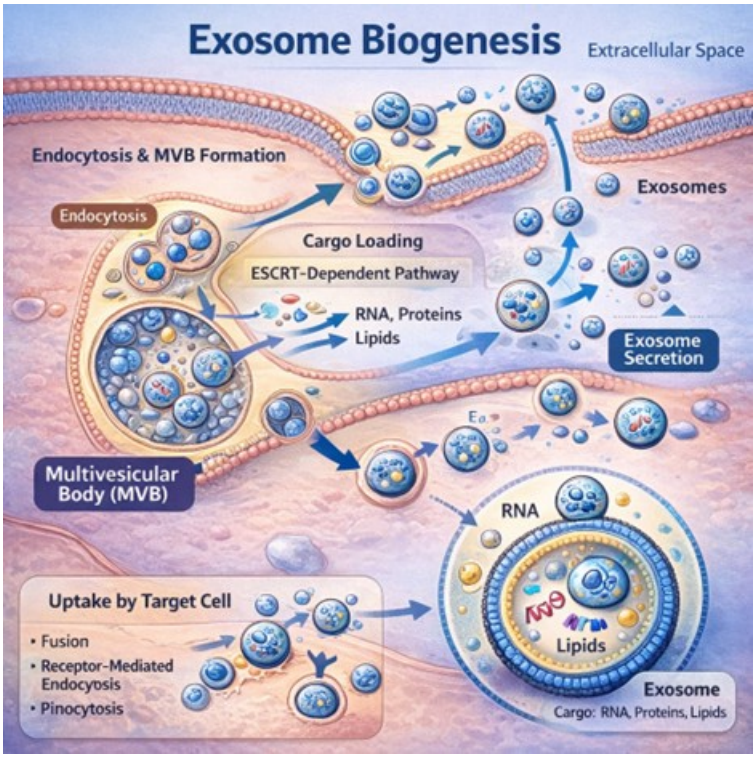

Figure 1: Exosome biogenesis and secretion pathway Schematic illustration of exosome formation within the endosomal system. Early endosomes mature into multivesicular bodies (MVBs), where intraluminal vesicles form and selectively incorporate RNA, proteins, and lipids via endosomal sorting complex required for transport (ESCRT)-dependent and ESCRT-independent mechanisms. Fusion of the MVB with the plasma membrane results in the release of exosomes into the extracellular space. Exosomes may then be taken up by recipient cells through membrane fusion, receptor-mediated endocytosis, or pinocytosis, enabling the transfer of functional molecular cargo. This is an original illustration.

Materials and Methods

A comprehensive narrative review of the literature was conducted using PubMed, Scopus, and Google Scholar databases. English-language articles evaluating exosome biology, extracellular vesicle signaling, regenerative mechanisms, dermatologic applications, safety concerns, and regulatory aspects were reviewed. Studies related to pigmentary disorders, acne scars, wound healing, hair disorders, photoaging, and inflammatory dermatoses were included. Particular emphasis was placed on studies relevant to Fitzpatrick skin types IV-VI and the Indian clinical setting, including issues related to pigmentation biology, post-inflammatory hyperpigmentation, keloidal tendency, procedural dermatology, and evolving regenerative medicine practices.

Results

Preclinical studies consistently demonstrate that exosomes derived from mesenchymal stem cells, keratinocytes, fibroblasts, platelets, and immune cells influence multiple pathways relevant to dermatology. Reported mechanisms include modulation of PI3K/Akt, Wnt/β-catenin, TGF-β, and inflammatory cytokine signaling pathways, resulting in enhanced keratinocyte proliferation, fibroblast migration, angiogenesis, collagen remodeling, melanogenesis regulation, and immune modulation. In experimental models, exosomes accelerate tissue repair and improve extracellular matrix organization. In pigmentation research, exosomal signaling has been shown to affect melanocyte activity, melanosome transfer, and tyrosinase expression, suggesting potential applications in melasma and post-inflammatory hyperpigmentation.

Early clinical reports suggest potential benefits in acne scars, skin rejuvenation, androgenetic alopecia, wound healing, and pigmentary disorders, particularly when exosomes are used as adjuncts to microneedling, laser resurfacing, or platelet-rich plasma-based procedures. High-quality randomized controlled trials remain scarce, and evidence specific to darker skin types is particularly limited. In the Indian context, translational challenges are further compounded by variability in commercially available products, lack of standardized manufacturing protocols, inconsistent disclosure of exosome source and composition, evolving regulatory frameworks, and increasing direct-to-consumer marketing.



Figure 2: Exosome-mediated intercellular communication in the skin Overview of exosome-driven signaling networks in cutaneous tissue. Keratinocyte-derived exosomes influence pigmentation pathways and barrier function; melanocyte exosomes participate in pigment transfer; fibroblast and endothelial exosomes promote wound healing and angiogenesis; and immune-cell-derived vesicles modulate inflammatory signaling. Together, these vesicle-mediated interactions contribute to pigmentation control, immune homeostasis, tissue repair, and extracellular matrix regulation. This is an original illustration.

Conclusions

Exosome-based therapies represent one of the most promising emerging frontiers in regenerative and aesthetic dermatology, offering potential applications across wound healing, pigmentary disorders, scarring, hair disorders, and inflammatory dermatoses. Nevertheless, current clinical evidence remains insufficient to support widespread routine use, particularly in skin-of-color populations where pigmentation biology and scarring responses may

differ substantially from Western cohorts. Rigorous randomized controlled trials, long-term pharmacovigilance studies, standardized manufacturing protocols, and India-specific regulatory guidance are urgently required. Until such evidence becomes available, exosome-based dermatologic therapies should be regarded as investigational and integrated into practice with scientific caution, ethical transparency, and robust patient counseling.



FIGURE 3: Ethical and regulatory pathway for responsible integration of exosome-based therapies Suggested clinical governance framework supporting safe introduction of exosome-based dermatologic therapies. Key elements include verification of product quality and source; structured risk-benefit evaluation; comprehensive patient counseling; robust informed-consent documentation; systematic monitoring and reporting of treatment outcomes and adverse events; and ongoing long-term safety surveillance. This is an original illustration

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Oral Corticosteroid Use in Patients With Bullous Pemphigoid Initiating Dupilumab: A Medicare Claims Analysis

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Introduction

Bullous pemphigoid (BP) is a rare autoimmune blistering disease that predominantly affects older adults. Oral corticosteroids (OCS) remain a cornerstone of treatment but carry significant risk of adverse effects, particularly in this medically vulnerable population with a high burden of comorbidities. Dupilumab, an IL-4Rα antagonist, is approved in the United States for the treatment of BP. This real-world study characterizes Medicare patients with BP initiating dupilumab and examines their OCS use patterns before vs. after dupilumab initiation.

Materials and Methods

This study utilized Medicare Fee-for-Service claims database (01/01/2017-09/30/2025). Adult beneficiaries (≥66 years) with ≥2 dispensations of dupilumab (1st dispensation = index date) at most 42 days apart between 2018/01/01-2024/09/30 were identified. Patients were required to have ≥1-year of enrollment in parts A, B, and D both before (baseline) and after dupilumab initiation (follow up), ≥1 inpatient or 2 outpatient BP diagnosis (L12.0), and a cumulative ≥60-day of OCS dispensation in the 1 year before dupilumab initiation. Patients with competing diagnoses (pemphigus vulgaris or other pemphigoid disorders e.g., acquired epidermolysis bullosa, linear IgA bullous dermatosis), those whose index dupilumab was prescribed by a pulmonologist or a gastroenterologist, who had prior biologic use, or had conditions requiring long-term OCS use (e.g., rheumatoid arthritis, systemic lupus erythematosus) were excluded. OCS use in the year before and after dupilumab initiation was described by daily dose (converted to mg of prednisone-equivalent and described in both OCS-treated period and whole period), changes in daily dose, number of prescriptions, and proportion of days covered (i.e., percentage of days that patient had OCS available). Proportion of patients who discontinued OCS (no additional prescription for ≥60-day after last day's supply) and median time to discontinuation during the follow up were also estimated.

Results

A total of 259 patients met the study criteria, with a mean (standard deviation [SD]) age of 80 (8) years, 55.2% female, and mean (SD) Charlson-comorbidity index of 2.8 (2.5). The mean daily OCS dose decreased from 16.4 mg to 9.2 mg (change: −7.2 mg) during the OCS-treated period and from 10.3 mg to 3.0 mg (change: −7.2 mg) across the entire pre- and post-periods. The median daily OCS dose decreased from 15.4 mg to 5.3 mg (change: −5.0 mg) during the OCS-treated period and from 7.8 mg to 1.2 mg (change: −5.6 mg) across the entire pre- and post-periods. In the OCS-treated period prior to dupilumab initiation, 68.0% and 30.5% of patients received daily OCS doses ≥10 mg and ≥20 mg, respectively. Following dupilumab initiation, these proportions decreased to 34.7% and 17.0%. Conversely, the proportion of patients receiving daily OCS doses <5 mg increased from 6.9% at baseline to 43.6% after dupilumab initiation. Proportion of days covered by OCS changed from a median of 65%

at baseline to 16% after dupilumab initiation. Total number of OCS dispensations also dropped from a median of 9.0 before to 2.0 after dupilumab initiation. Among 223 patients with OCS dispensations within 91 days prior to dupilumab initiation, average OCS daily dose went from 9.8 mg to 4.8 mg during the first 90 days and further dropped to 2.0 mg during the fourth 90-day window post dupilumab. Among 186 patients who had OCS coverage within 14 days of dupilumab initiation, 139 (74.7%) discontinued OCS during follow up (median time to discontinuation: 87 days), this included 37 patients who had no OCS dispensation after dupilumab initiation.

Table. OCS Use Before and After Dupilumab Initiation in 259 Patients With Bullous Pemphigoid in US Medicare Claims Database

Variable	Pre-Dupilumab (BP diagnosis to dupilumab initiation)	Post-Dupilumab (365-day follow-up)	Change
Daily OCS Dose (mg, prednisone-equivalent), treated period			
Median (Q1, Q3)	15.4 (8.0, 20.9)	5.3 (0.0, 14.8)	−5.0 (-14.2, -0.1)
Mean (SD)	16.4 (11.1)	9.2 (10.7)	−7.2 (13.3)
OCS dose categories, N (%)			
<5mg	18 (6.9)	113 (43.6)	+36.7
5-<10mg	65 (25.1)	56 (21.6)	−3.5
≥10mg	176 (68.0)	90 (34.7)	−33.3
Daily OCS Dose (mg, prednisone-equivalent), entire period			
Median (Q1, Q3)	7.8 (4.9, 13.2)	1.2 (0.0, 4.5)	−5.6 (-11.4, -2.3)
Mean (SD)	10.3 (8.4)	3.0 (4.2)	−7.2 (8.4)
OCS dose categories, N (%), entire period			
<5mg	67 (25.9)	198 (76.4)	+50.5
5-<10mg	91 (35.1)	41 (15.8)	−19.3
≥10mg	101 (39.0)	20 (7.7)	−31.3
Number of OCS dispensations per patient per year			
Median (Q1, Q3)	9.0 (5.6, 13.8)	2.0 (0.0, 6.0)	−5.9 (−10.0, −3.0)
Mean (SD)	10.5 (7.0)	3.7 (4.3)	−6.8 (7.1)
Proportion of days covered, %			
Median (Q1, Q3)	65 (41, 84)	16 (0, 55)	−35
Mean (SD)	62 (25)	29 (32)	−33
*OCS Discontinuation**			
N (%)	--	139 (74.7%)	--
*Time to OCS discontinuation**, days*			
Median (Q1, Q3)	--	87 (1, 164)	--
Mean (SD)	--	100 (89)	--

* Among 186 patients with OCS coverage within 14 days of dupilumab initiation. ** For patients who had no OCS dispensation during the follow up (n=37 out of 139), time to discontinuation was assigned as 1 day. BP, bullous pemphigoid; OCS, oral corticosteroid; Q1, 25th percentile; Q3, 75th percentile; SD, standard deviation

Conclusions

In this real-world study of Medicare population with BP, dupilumab initiation was associated with meaningful reductions in OCS daily dose and OCS discontinuation in three quarters of patients. These findings support the role of dupilumab as an effective steroid-sparing treatment for BP, potentially mitigating corticosteroid-related risks in this population.

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Abstract N°: 3639

Characteristics of Patients With Bullous Pemphigoid Initiating Dupilumab: A Medicare Claims Analysis

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Introduction

Bullous pemphigoid (BP) is a rare autoimmune blistering disease that predominantly impacts older patients. Dupilumab, an IL-4Rα antagonist, is approved in the United States for the treatment of BP in June 2025. This real-world study characterized BP patients initiating dupilumab in real-world clinical practice using the US Medicare database.

Materials and Methods

This study utilized Medicare Fee-for-Service claims database. Adult patients (≥18 years) with ≥2 dispensations of dupilumab (1st dispensation = index date) at most 42 days apart between 01/01/2018-09/30/2024 were identified. Patients were required to have ≥1 year of enrollment with both medical and pharmacy benefit before (baseline) dupilumab initiation and have ≥1 inpatient or 2 outpatient BP diagnoses (L12.0) in the baseline period. Patients with competing diagnoses (pemphigus vulgaris or other pemphigoid disorders, e.g., acquired epidermolysis bullosa, linear IgA bullous dermatosis) or those whose index dupilumab was prescribed by a pulmonologist or a gastroenterologist were excluded. Demographic and baseline clinical characteristics were described. Treatments received 1-year before dupilumab initiation are reported. Use of oral corticosteroids (OCS) was summarized, and all OCS dose was converted to prednisone-equivalent dosing for reporting, with average daily OCS dose was calculated over the treated period (days covered by OCS at baseline) as well as the entire period between BP diagnosis and index date.

Results

A total of 701 patients met the study criteria, with a median (Q1−Q3) age of 81 (74−87) years and 52.5% females. Median (Q1−Q3) Charlson-comorbidity index was 2 (1−4). Comorbidities were common, including hypertension (82.9%), hyperlipidemia (75.7%), diabetes (45.2%), atherosclerotic cardiovascular disease (43.9%), chronic kidney disease (35.5%), depression (27.1%), heart failure (26.5%), osteoporosis (21.0%), dementia (18.4%), and Parkinson's disease (4.9%). Additionally, 16.7% of patients had a coexisting diagnosis of atopic dermatitis and 20.5% had chronic obstructive pulmonary disease. Median (Q1−Q3) time from first ever identified diagnosis of BP (going back to 10/1/2015) to dupilumab initiation was 6.9 (2.3-20.9) months. Before dupilumab initiation, 73% (n=513) of patients received OCS; among them, median (Q1-Q3) number of prescriptions was 8.7 (4.1-14.6), daily dose was 19.6 (11.6-28.3) mg during the treated-period and 7.7 (3.8-15.0) mg during the whole baseline period; median (Q1-Q3) proportion of days covered by OCS was 50% (22%-77%) during the entire period. Overall, in the year before dupilumab initiation, 85.9% of patients received ≥1 dispensation for topical corticosteroids (TCS; 67.5% received high potency TCS), 65.6% for anti-inflammatory antibiotics, and 25.4% for immunosuppressants. Upon initiation of dupilumab (within 30-day before index date), 41.7% had dispensation for OCS, 47.4% were dispensed TCS, 33.1% were dispensed ≥1 anti-inflammatory antibiotic, 12.8% were dispensed

≥1 immunosuppressant, and 23.1% were not dispensed any of these medications before dupilumab, suggesting its use as a monotherapy.

Table. Characteristics of patients with bullous pemphigoid before initiating dupilumab in Medicare claims database

Characteristics	**All patients (N=701)**
Age at index date, years	
Mean (SD)	81 (8)
Median (Q1-Q3)	81 (74-87)
Female, n (%)	368 (52.5)
Race/Ethnicity, n (%)	
Hispanic or Latino (any race)	16 (2.3)
Non-Hispanic White	547 (78.0)
Non-Hispanic Black or African American	44 (6.3)
Asian	41 (5.8)
North American Native	< 11
Other /Unknown /Multiracial	51 (7.3)
Time from BP diagnosis date to index date, months	
Mean (SD)	16.3 (21.3)
Median (Q1-Q3)	6.9 (2.3-20.9)
Quan-Charlson Comorbidity Index score	
Mean (SD)	2.9 (2.7)
Median (Q1-Q3)	2 (1-4)
Clinical comorbidities, n (%)	
Hypertension	581 (82.9)
Hyperlipidemia	531 (75.7)
Diabetes	317 (45.2)
Atherosclerotic cardiovascular disease	308 (43.9)
Chronic kidney disease	249 (35.5)
Atrial fibrillation and flutter	192 (27.4)
Depression and other depressive mood disorder	190 (27.1)
Heart failure	186 (26.5)
Anxiety-related disorder	163 (23.3)
Osteoporosis with or without pathological fracture	147 (21.0)
History of any malignancy (except non-melanoma skin cancer)	145 (20.7)
Dementia including Alzheimer's disease	129 (18.4)
Venous thromboembolism	51 (7.3)
Parkinson's disease	34 (4.9)
Daily OCS dose (mg) in the OCS-treated period*	
Mean (SD)	21.6 (13.8)
Median (Q1-Q3)	19.6 (11.6-28.3)
Daily OCS dose (mg) in the OCS-treated period*, categories, n (%)	
<5	26 (5.1)
5-<10	77 (15.0)
10-<15	72 (14.0)
15-<20	86 (16.8)
20-<30	135 (26.3)
≥30	117 (22.8)
Daily OCS dose (mg) in the whole baseline period*	
Mean (SD)	10.9 (10.6)
Median (Q1-Q3)	7.7 (3.8, 15.0)
Daily OCS dose (mg) in the whole baseline period*, categories, n (%)	
<5	172 (33.5)

5-<10	137 (26.7)
10-<15	76 (14.8)
15-<20	50 (9.7)
20-<30	50 (9.7)
≥30	28 (5.5)
Proportion of days covered by OCS in the baseline period*, median (Q1-Q3), %	50 (22-77)
Number of OCS dispensations per patient per year in the baseline period*	
Mean (SD)	11.1 (9.8)
Median (Q1-Q3)	8.7 (4.1-14.6)

*Among patients with at least one OCS prescription during the baseline period (n=513). OCS, oral corticosteroids; Q1, 25th percentile; Q3, 75th percentile; SD, standard deviation.

Conclusions

This real-world Medicare analysis showed that patients with BP initiating dupilumab were predominantly elderly (generally aged over 80 years) and medically complex with a high burden of cardiometabolic, neurologic and psychiatric comorbidities. Most had substantial prior treatment exposure, including frequent and prolonged OCS use, despite the high prevalence of steroid-sensitive comorbidities such as hypertension, diabetes and osteoporosis. The high prevalence of comorbidities that can worsen with corticosteroid exposure underscores the need for steroid-sparing therapies such as dupilumab in this population.

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Abstract N°: 3699

Bimekizumab Associated Pemphigus Vulgaris after Treatment for Psoriasis Vulgaris: A Case Report

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Introduction

Psoriasis vulgaris is a chronic immune-mediated inflammatory dermatosis frequently requiring long-term systemic treatment in moderate-to-severe forms of the disease. The introduction of biologic agents targeting the interleukin-17 (IL-17) pathway has significantly improved disease control and patient quality of life. Despite their favorable efficacy and safety profile, biologic therapies have been associated with rare immune-mediated adverse reactions, including the development of autoimmune blistering diseases.
Pemphigus vulgaris is a rare autoimmune bullous dermatosis characterized by the formation of pathogenic autoantibodies directed against desmosomal adhesion proteins, leading to loss of keratinocyte adhesion and intraepidermal blister formation. Clinically, the disease presents with flaccid bullae and erosions affecting the skin and/or mucous membranes. Although the coexistence of psoriasis vulgaris and pemphigus vulgaris has rarely been described, the development of pemphigus vulgaris during biologic therapy remains exceptionally uncommon.
We observed a case of pemphigus vulgaris developing during biologic treatment with bimekizumab in a patient with psoriasis vulgaris.

Case Presentation

A 36-year-old male presented with blisters and erosions, involving the scalp, trunk, upper and lower extremities for over a month. He had a 9-year history of psoriasis vulgaris, which was treated with secukinumab for 2 years, but due to insufficient therapeutic response, treatment was switched to bimekizumab. Approximately after 6 months of treatment with bimekizumab, flaccid blisters, erosions and crusting were present, as well as areas of post-inflammatory hyperpigmentation. At the time of admission, he complained of painful erosions of the oral mucosa. Additionally, psoriatic plaques were absent and treatment with bimekizumab was discontinued.
Routine laboratory parameters were within the normal range. Histology showed suprabasal acantholysis consistent with pemphigus vulgaris. Direct immunofluorescence demonstrated intracellular deposition of IgG (++) within the epidermis. Based on the clinico-laboratory findings the diagnosis of pemphigus vulgaris was made. Treatment with rituximab (2 vials x 500 mg, on day 1 and 14) was initiated, following maintenance treatment with methylprednisolone 12 mg daily. Significant clinical improvement occurred. The blistering eruption responded quite favorably with no new lesions and satisfactory healing of pre-existing ones. Residual cutaneous findings were limited to post-inflammatory hyperpigmentation without active lesions or psoriatic plaques.

Conclusion

This case highlights the potential occurrence of pemphigus vulgaris during treatment with interleukin-17-targeted biologic therapy for psoriasis vulgaris. Although uncommon, autoimmune blistering diseases should be considered in patients who develop vesiculo-bullous eruptions while receiving biologic agents. Early recognition and prompt therapeutic intervention, including withdrawal of the suspected agent and initiation of immunomodulatory treatment, may contribute to favorable clinical outcomes.

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Abstract N°: 3725

Efficacy of adjuvant dabrafenib-trametinib in the treatment of resected Stage III cutaneous BRAF V600 mutated melanoma patients. Single Institute, real-life data.

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Introduction

In 2018, FDA approved dabrafenib-trametinib for the adjuvant treatment of resected, stage III melanoma with BRAF V600E and V600K mutations. 12 months of adjuvant therapy with dabrafenib plus trametinib significantly prolongs recurrence-free survival (RFS). The aim of our retrospective study was to evaluate the efficacy and safety of adjuvant dabrafenib-trametinib therapy by analyzing real-world data from our Dermato-oncology Department.

Case Presentation

Materials & Methods: We conducted datas from 111 patients who received adjuvant dabrafenib–trametinib therapy at the Department of Oncodermatology of the National Institute of Oncology, Hungary between January 2021 and October 2025. The primary endpoints were the determination of overall survival rates (OS) at 36, 48, 60, and 72 months, as well as relapse-free survival (RFS) and distant metastasis-free survival (DMFS). Adverse events (AEs) were evaluated as secondary endpoints. Our patients received dabrafenib-trametinib therapy for 12 months.

Results: Our study included 66 men (59%) and 45 women (41%) with a mean age of 56 years (26-81). The median follow-up time was 22 months (range: 5–53) from the start of therapy. 86% of patients had the BRAF V600E mutation. 42% were at stage IIIC, 31% at stage IIIB, 18% at stage IIIA, and 9% at stage IIID. Progression occurred in 14% of patients. In 9 cases (8%), patients received first-line Ipi-Nivo treatment following disease progression; among these patients, 1 (0.9%) achieved partial remission. 4 patients (3,6%) received dabrafenib-trametinib therapy due to progression occurring more than 12 months after adjuvant treatment; 100% of these patients achieved partial remisssion. The survival rate calculated from the start of therapy at 36 months was 90.99%; at 48 (4-year) months was 87.04%. RFS calculated from the start of therapy to the time of progression, at 36 months 77.41%; at 48 months (4 years) 67.56%. Distant metastasis free survival (DMFS) calculated from the start of therapy until the onset of distant metastasis was 83.69% at 36 months, and 80.52% at 48 months. Dose reduction was required in 14.4% of patients. Adverse events (AEs) occurred in 70 patients (63%), Grade 3 or 4 adverse events occurred in 24% of patients. Treatment was permanently discontinued in 4 cases (3.6%) due to reversible adverse effects, like serosus retinopathy, recurrent pyrexia. The most common adverse events were pyrexia, rash, and elevated CPK.

Conclusion

In COMBI AD trial, a 55% RFS rate was achieved at 4 years, 52% RFS rate at 5 years with a follow-up period of nearly 10 years. 12 months of adjuvant treatment resulted in better relapse-free survival (RFS) and distant metastasis-free survival (DMFS) compared to placebo in resected patients with stage III melanoma. Based on the OS analysis, the risk of death was 20% lower with the combination therapy than with placebo, but the OS benefit was not significant. In patients with BRAF V600E mutation, the risk of death was 25% lower with dabrafenib-

trametinb therapy. In the Combi AD trial, dose reduction was required in 38% of patients; the most common adverse events were fever (63%), fatigue (40%), and nausea (40%). Grade 3 adverse events occurred in 36% of patients, and treatment had to be discontinued due to adverse events in 26% of patients. Our real-world data showed better results than those reported in the literature, with a 67.56% 4-year RFS. The incidence of AE was similar to that observed in the COMBI-AD trial, but in our real-world study, treatment discontinuation occurred at a lower rate. Pyrexia was the most common adverse event, consistent with the results of clinical trials.

The introduction of new combinations and neoadjuvant therapy may offer a solution for relapses following adjuvant therapies. In the future, the introduction of biomarkers may enable more precise selection of patients who will benefit from adjuvant therapy.

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Abstract N°: 3752

Comparative study between efficacy of Excimer light with topical Tacrolimus 0.1% versus excimer light with topical Bimatoprost 0.01% in treatment of facial Vitiligo

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Introduction

Loss and absence of melanocytes due to a number of factors is responsible for vitiligo; known to be the commonest disorder of pigmentation. The aim of the current work was to compare the efficacy and safety of excimer light with topical tacrolimus ointment 0.1% versus excimer light with topical bimatoprost gel 0.01% in treatment of facial vitiligo. The study was carried out on 48 patients presented with facial vitiligo.

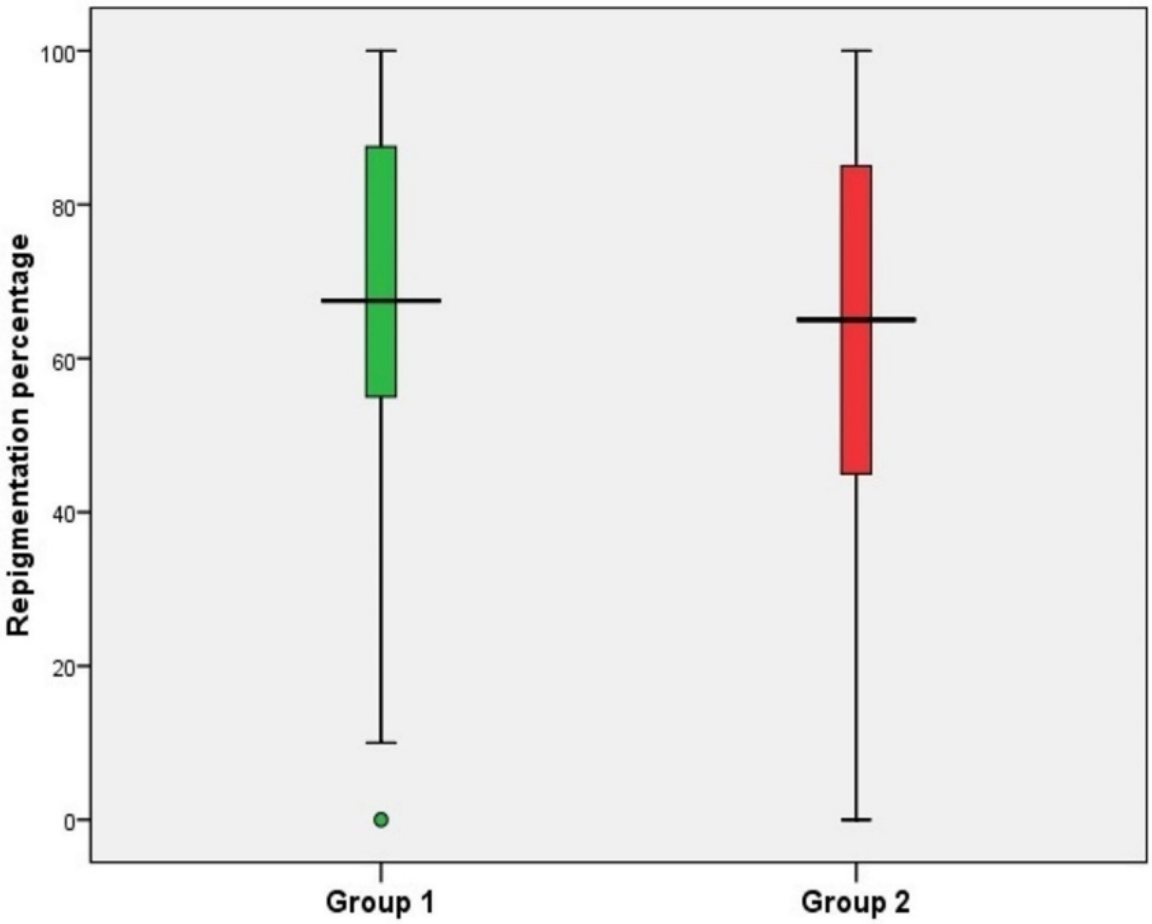
Materials and Methods

The patients were divided randomly using sealed envelope method into two groups (24 patients each). Group 1 were treated with excimer light plus topical tacrolimus ointment 0.1% and group 2 treated with excimer light plus topical bimatoprost gel 0.01%.

A 6-year old male presented with localized patch of facial vitiligo. A) At baseline, B) after 8 weeks and C) after 12 weeks of treatment with excimer light plus topical tacrolimus ointment 0.1%

Results

Clinical improvement based on the quartile grading scale at the end of treatment did not show any statistically significant difference between groups. The majority of subjects in both groups experienced good to excellent improvement. Only 20.9% of patients in group 1 and 33.3% of subjects in group 2 achieved less than 50% repigmentation ($p = 0.889$).



Box and Whisker plot showing re-pigmentation percentage among studied groups

Conclusions

Our study demonstrated that 0.01% topical bimatoprost gel in combination with excimer light is considered safe and effective as treatment of nonsegmental facial vitiligo with comparable results to 0.1% tacrolimus.

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Successful treatment of paradoxical alopecia areata totalis with upadacitinib

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Introduction

The appearance of alopecia areata in patients with inflammatory bowel diseases during the treatment with anti-TNFα agents has been widely acknowledged as an adverse event which requires special therapeutic approach. The aim to provide an individualized treatment option suitable for both clinical entities is imperative, as each patient may have a long medical history with comorbidities and treatment failures. We present a case of a patient with paradoxical alopecia areata successfully treated with the oral JAK inhibitor upadacitinib

Case Presentation

A 44-year-old woman with a history of severe Crohn's disease presented with alopecia areata totalis in November 2024 to our Dermatology department. The patient had been under treatment with adalimumab 40mg every other week for 16 months with excellent control of the inflammatory bowel disease. According to the patient, six months after the initiation of the therapy, several patches of alopecia areata developed on the scalp with gradual evolvement to alopecia areata totalis 10 months later. The patient was referred to the Dermatologist for examination. There was a Gastroentrerologist-patient-Dermatologist consensus on switching to another therapy, considering the phycological burden of the patient, who had already been on several topical treatments during the last year without any clinical response of hair regrowth. We reviewed all the available treatment options in our country, possibly suitable for our patient at that time and firstly, we excluded the anti-TNFα agents infliximab and certolizumab pegol, because alopecia areata was deemed as an anti-TNFα paradoxical reaction. Secondly, we excluded vedolizumab, an α4β7-integrin antibody, which is a gut-selective biologic agent and therefore its effectiveness for the treatment of a skin condition could not be supported. The biologic agents risankizumab and ustekinumab could offer two plausible therapeutic options, but the oral JAK inhibitor upadacitinib was finally preferred as a rapid-acting drug. The published case reports provided evidence at that time, which sufficiently supported the rationale of using a JAK inhibitor for the concurrent therapeutic assessment of alopecia areata and IBD. The patient started upadacitinib 45mg daily for 3 months, followed by upadacitinib 30mg daily afterwards. Hair regrowth was noticed a month after the initiation of the therapy. After 3 and 5 months on upadacitinib SALT scores were 30 and 10, respectively. Total hair-regrowth was achieved 2 months later. The patient did not receive any additional treatment, either topical or oral, at any timepoint of the therapy. She is still on upadacitinib 30mg with an excellent response to both Crohn's disease and alopecia areata without any reported adverse events after 18 months of treatment (May 2026, photos available at all the aforementioned timepoints, but not submitted as per submission requirements).

Conclusion

This case report could be added to the existing real-world evidence of using upadacitinib for the treatment of alopecia areata. This agent can also provide a possible option for the therapeutic challenge of controlling two inflammatory diseases in an effective and rapid-acting way.

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The Rebound Paradox Across Novel Oral Peptides: A Systematic Review of Treatment Withdrawal Effects from Icotrokinra and JAK Inhibitor Trials to Define a New Framework for "Disease Sovereignty"

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Introduction
These agents which orally aims for psoriasis and atopic dermatitis provided a high-quality control in short-term, it normalises the symptoms for many patients. But when we stop these drugs are, which indicates a reversal; the disease can outburst, sometimes this can even worse than before the treatment began. This "rebound paradox" which features a gap in our learning that how lasting are these features after stopping medication. In other words, how much "disease autonomy" the period in which a patient maintains control even without treatment so what do these therapies really offers? To address this, we compared post-withdrawal outcomes for three classes like JAK inhibitors like JAKi, TYK2 inhibitor TYK2i, and IL-23 receptor and its antagonist peptide IL-23Ri our main objective was to map out how their mechanisms of action shapes disease actions after stopping therapy.

Materials and Methods
In OUR systematic search of literatures from sources from 2019 to 2025, controlled interventional studies integrating open-label follow-up. treatment-withdrawal phases. Our focus was on adults with moderate-to-severe psoriasis or atopic dermatitis who had received abrocitinib which is a JAK1 inhibitor, deucravacitinib -a TYK2 inhibitor, or icotrokinra -an IL-23Ri peptide. For our initial results, we did analysis of the mean time to loss of disease control after stoppage and the rate of rebound, meaning flares that exceeded the original severity. For deep analysis, we used restricted mean survival time analysis on published Kaplan-Meier curves.

Results
These evidences gave us a clear picture. In atopic case of dermatitis, withdrawing abrocitinib led to a rapid loss of control TTLC hit just 4.2 weeks with 95% CI 3.5–5.0, and 28% of patients had experienced a rebound that was worse than the standard. In psoriasis, after stopping deucravacitinib (POETYK PSO-LTE) resulted in a longer TTLC of 14.8 weeks with 95% CI 12.1–17.5 and only 8% of them rebounded. Withdrawal of icotrokinra (FRONTIER 2) turned out to be even better, Rebound risk and intensity matched the scope of the cytokine blockade. JAK mean TTLC reached 18.5 weeks with 95% CI 15.9–21.1 with no observable rebound at all. inhibitors that suppress dozens of cytokines stopping suddenly caused receptors to overdo, making more intense flares. TYK2 inhibitors, blocked IL-12/23 and type I IFN pathways, which allowed a slower return of immune activity. IL-23Ri, by acting priorly on Th17 cell polarized, seems to create a more lasting immune shift essentially when it resets the disease. This gave us to a three-level Disease Sovereignty Spectrum which was like 1. pharmacologic dependence with JAKi, where disease quickly recoil 2. fractional immune reset with TYK2i, allowing some time off therapy, 3. lasting sovereignty with IL-23Ri, supporting the longest treatment-free control.

Conclusions
Broad JAK blocked almost assured a "rebound paradox" which is a fast return and sometimes even intensified disease whereas selective IL-23R antagonism breaks the cycle offering longer, steadier remission after stopping.

The Disease Sovereignty Spectrum gave us a more realistic structure for clinicians helping them to match the right drug to a patient’s goal for treatment free control.

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Abstract N°: 3775

Immune Checkpoint Inhibitor-Associated Acquired Reactive Perforating Collagenosis in a Patient With Metastatic Lung Adenocarcinoma

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Introduction

Immune checkpoint inhibitors are increasingly associated with a broad spectrum of cutaneous immune-related adverse events. Reactive perforating collagenosis is a rare perforating dermatosis characterized by transepidermal elimination of altered collagen fibers and typically presents with hyperkeratotic pruritic papules and nodules. Although acquired reactive perforating collagenosis has rarely been reported in association with immunotherapy, the underlying mechanism remains incompletely understood. Herein, we report a case of nivolumab-associated acquired reactive perforating collagenosis confirmed histopathologically in a patient with metastatic lung adenocarcinoma.

Case Presentation

A 66-year-old man with metastatic lung adenocarcinoma receiving nivolumab was referred for evaluation of widespread pruritic papulonodular eruptions involving the trunk and extremities. The patient had previously received chemotherapy, radiotherapy, and immunotherapy. Dermatological examination revealed widespread erythematous hyperkeratotic papules and nodules with central crusting and excoriation, predominantly involving the extensor surfaces of the upper extremities and trunk (Figure 1).

Closer examination demonstrated multiple crateriform hyperkeratotic papules and nodules with central keratotic plugs (Figure 2).

Histopathological examination demonstrated epidermal ulceration with orthokeratotic hyperkeratosis, inflammatory infiltrates, and transepidermal elimination of vertically oriented collagen fibers, consistent with reactive perforating collagenosis (Figure 3). Clinicopathological correlation supported the diagnosis of nivolumab-associated acquired reactive perforating collagenosis.

Conclusion

Immune checkpoint inhibitor-associated reactive perforating collagenosis is an uncommon dermatologic adverse event that may clinically mimic other pruritic papulonodular dermatoses. Recognition of its characteristic clinicopathological findings is important for accurate diagnosis. Our case further supports the expanding spectrum of cutaneous immune-related adverse events associated with nivolumab and highlights the importance of considering perforating dermatoses in patients presenting with persistent hyperkeratotic pruritic eruptions during immunotherapy.

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Abstract N°: 3789

Targeting cytokines (IL17/23/31) with a novel vaccine format: in vivo POC and positive benchmarking to marketed biologicals

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Introduction

Monoclonal antibodies that target cytokines are commonly used to treat inflammatory skin disorders such as psoriasis and atopic dermatitis. However, their use is limited to patients with more severe disease due to their high cost and the necessity of frequent administration. Tridem Bioscience has developed a novel vaccine format, termed WISIT (**Wi**n the **s**kin **i**mmunity **t**rick) with unprecedented immunogenicity. This allows the elicitation of high titer/high avidity antibody responses. WISIT vaccines are peptide/protein conjugates coupled to ß-glucans, sugar residues used to target and activate antigen presenting cells.

Materials and Methods

We identified target regions in cytokines of interest and defined short peptides representing them. These peptides were tested as part of conventional vaccines (peptide/carrier conjugate adsorbed to adjuvant) or WISIT vaccines (peptide/carrier/ß-glucan conjugates). Screening studies in mice administering vaccines 3x every 2-4 weeks (routes: conventional vaccines: s.c., WISIT vaccines: i.d.) were used to select the top candidates based on reactivity towards the targeted cytokine. Further characterization included benchmarking WISIT induced Ab specificity and avidity to the one of marketed monoclonal antibodies (e.g., ixekizumab, bimekizumab, nemolizumab) as well as in vitro and in vivo POC studies of top candidates selected.

Results

WISIT vaccines were found to be superior to conventional vaccines in inducing high titer/high avidity antibody responses to cytokines in mice. Antibody-responses induced by WISIT vaccines were dose-dependent and could be boosted. The boost injections triggered a recall-type increase in antibody titers and significantly augmented the antibody avidity.

Top candidates were further subjected to benchmarking and POC-type studies (in vitro and in vivo). Comparing the specificity and avidity of antibodies induced following 3 priming immunizations to marketed monoclonal antibodies showed comparable specificity and avidity. The biological activity of different IL31 targeting vaccines, as assessed in an in vitro inhibition assay, identified several candidates to yield inhibitory effects comparable to nemolizumab. Candidate peptides as short as 6 amino acids were shown to be effective. Furthermore, an imiquimod-based mouse psoriasis model was used to evaluate the top WISIT candidates targeting the IL17/23 axis. An initial study testing a low dose of the WISIT vaccine demonstrated in vivo POC in this model.

Conclusions

These in vivo studies demonstrate that WISIT vaccines can elicit antibodies that react with and inhibit the function of targeted cytokines in an unprecedented manner. They have the potential to

create a new, cost-efficient, and sustainable therapeutic approach.

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Abstract N°: 3790

Restoring Laminin-332 in Junctional Epidermolysis Bullosa: From Molecular Defect to Emerging Therapeutic Strategies.

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Introduction

Junctional epidermolysis bullosa (JEB) is a rare inherited blistering disorder characterized by mechanical fragility resulting from impaired dermal–epidermal adhesion. Severe phenotypes are frequently associated with pathogenic variants in LAMA3, LAMB3, and LAMC2, which encode the laminin-332 heterotrimer, a critical component of anchoring filaments within the basement membrane zone. Deficient or dysfunctional laminin-332 compromises epidermal integrity, wound healing and tissue regeneration, making its restoration a major therapeutic target in JEB.

Case Presentation

Objective

To review the role of laminin-332 deficiency in the pathophysiology of JEB and discuss emerging therapeutic strategies aimed at restoring protein expression, epidermal adhesion and regenerative capacity.

Methods

A narrative literature review was conducted using articles indexed in PubMed and LILACS published within the last five years. The search included the terms “junctional epidermolysis bullosa,” “laminin-332,” “LAMA3,” “LAMB3,” “LAMC2,” “gene therapy,” “readthrough therapy,” “cell therapy,” and “regenerative medicine.” Studies addressing molecular mechanisms, genotype–phenotype correlations and therapeutic advances in JEB were selected for analysis.

Results

Laminin-332 plays a central role in basement membrane stability, dermal–epidermal adhesion and epidermal homeostasis. Pathogenic variants affecting its subunits impair anchoring filament assembly, leading to blister formation, chronic wounds and reduced regenerative capacity (Bruckner-Tuderman; Has, 2020). Beyond its structural role, recent evidence suggests that laminin-332 also participates in signaling pathways involved in epidermal stem cell maintenance and tissue repair.

Emerging therapeutic strategies have focused on restoring laminin-332 expression or function through multiple approaches. Pharmacological readthrough therapy using aminoglycosides such as gentamicin demonstrated partial restoration of laminin-332 expression and improved wound healing in patients carrying nonsense mutations (KUECKELHAUS, M. et al., 2021). Ex vivo gene-corrected epidermal grafts targeting LAMB3 mutations have shown long-term epidermal regeneration and sustained clinical improvement in severe JEB phenotypes (HIRSCH, T. et al).

Additional approaches, including cell-based therapies and tissue engineering techniques, have also demonstrated regenerative potential in preclinical and translational models (Nyström; Bruckner-Tuderman, 2021). Nevertheless, clinical translation remains limited by mutation-specific responses, disease heterogeneity, manufacturing complexity, long-term safety concerns, cost and restricted accessibility.

Conclusion

Restoration of laminin-332 represents a biologically rational and increasingly promising therapeutic strategy in JEB. Advances in gene, regenerative and cell-based therapies may significantly change the management of severe disease by moving beyond symptomatic wound care toward targeted molecular intervention. Further clinical studies remain essential to establish long-term efficacy, safety and accessibility, particularly in genetically heterogeneous phenotypes.

EADV CONGRESS
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Abstract N°: 3794

Circulating Skin-Emigrant T Cells Characterize Dupilumab-Associated Lymphoid Reactions

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Introduction

Dupilumab, an IL-4Ra targeting monoclonal antibody, is an effective treatment for atopic dermatitis (AD). In a small subset of patients, dupilumab treatment can cause a reversible local adverse lymphoid reaction (LR), causing a burning sensation which differs from AD. Clinically, these lesions mimic mycosis fungoides (MF), raising concern for CTCL-like pathology, yet their underlying immune biology remains poorly understood. In this study we aimed to characterize the circulating (skin homing) T-cells of AD patients who developed LR during dupilumab treatment.

Materials and Methods

Peripheral blood mononuclear cells (PBMC) samples were collected from AD patients who developed LR during dupilumab treatment (n=6), AD patients with adequate response to dupilumab (DR) (n=6), patients with MF (n=6) and healthy controls (HC) (n=8). Using spectral flow cytometry, T-cells were analyzed and phenotypical as well as functional characteristics were compared between the patient groups.

Results

Unbiased clustering resulted in 20 T-cell clusters, of which 7 expressed skin-homing markers (CLA+ CCR4+). Notably, a population of CD103+ “recent skin emigrant” memory CD4+ T cells, was significantly expanded in LR patients. This population increased in 5/6 LR patients just before LR diagnosis, and exhibited elevated expression of IL-13 and IL-22, cytokines associated with AD activity. In addition, LR and MF patients shared expansion of a skin-homing $CD26^{dim}$ memory CD4+ T-cell population, in which the IL-4 production was highest in LR patients. Skin homing FOXP3+ regulatory T-cells (Tregs) were also significantly expanded in both LR and MF patients, indicating enhanced immunoregulatory responses within the circulating skin homing T-cell department. A similar trend was observed for CD103+ “recent skin emigrant” Tregs, which were most abundant in LR patients. In contrast, chronic activation/exhaustion markers and proliferation rates were similarly increased in LR and dupilumab responders, arguing against a uniquely malignant-like activation state in LR. These findings support a model in which IL-4Rα blockade induces altered trafficking and recirculation of activated skin-associated T cells rather than malignant transformation.

Conclusions

LR associated with dupilumab is characterized by a distinct expansion of activated, cytokine-producing skin-emigrant memory $CD4^+$ T cells, including Tregs. Despite its clinical resemblance to CTCL, LR lacks features of sustained malignant-like activation and instead reflects a transient, highly dynamic immune redistribution between skin and blood. Ongoing imaging mass cytometry (IMC) studies in matched skin biopsies will further define how circulating immune dynamics relate to local skin-resident T-cell organization and activation.

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Abstract N°: 3850

Long-term malignancy risk in adults with psoriasis treated with secukinumab or adalimumab: A retrospective cohort study using TriNetX.

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Introduction

Biologic therapy has become the routine care for moderate-to-severe plaque psoriasis. Thus, assessing long-term safety, particularly malignancy risk, remains critical. Secukinumab and adalimumab are among the most frequently prescribed biologics, but limited comparative data exist on their long-term malignancy risk, with no real-world comparative studies.

Our objective is to compare the long-term malignancy risk in adult patients with psoriasis treated with secukinumab or adalimumab.

Materials and Methods

Our retrospective cohort study was conducted within the Global Collaborative Network of TriNetX. Psoriasis patients aged ≥18 years treated with secukinumab (n=8,196) or adalimumab (n=18,305) were included. Patients exposed to the comparator biologic at any time were excluded from each cohort. We assessed four composite endpoints and 25 individual malignancies over 5 years after treatment initiation. Hazard ratios (HRs) were estimated using Kaplan-Meier. Confounding control was achieved by propensity score matching. Robustness was assessed through sensitivity analyses.

Results

Overall malignancy risk in patients treated with secukinumab and adalimumab was comparable between groups (HR 0.90; 95%-CI 0.76, 1.06; p=0.1916). Compared to adalimumab, secukinumab showed a lower risk of non-melanoma skin cancer (HR, 0.69; 95%-CI, 0.54, 0.89; p=0.0034), basal cell carcinoma (HR 0.71; 95%-CI 0.53, 0.95; p=0.0216), and squamous cell carcinoma (HR 0.67; 95%-CI 0.45, 0.99; p=0.0405). In secukinumab-exposed females, 18/3,875 (0.47%) developed uterine cancer, compared to ≤10/3,872 (≤0.26%) with adalimumab (HR 2.36; 95%-CI 1.03, 5.43; p=0.0372). Events were few, and counts <10 were masked. As such, this finding needs to be interpreted with care. The remaining malignancies did not differ significantly between groups.

Conclusions

Our investigation demonstrates comparable overall malignancy risk among psoriasis patients treated with secukinumab or adalimumab. However, patients receiving adalimumab should be monitored more closely for non-melanoma skin cancer.

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Abstract N°: 3872

Omalizumab in Refractory Pruritic Disorders Beyond Classical Urticaria, Atopic Dermatitis, and Bullous Pemphigoid: A Phenotype-Stratified Focused Systematic Review

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Introduction
Omalizumab is established for chronic spontaneous urticaria and severe allergic asthma, but off-label use has expanded across heterogeneous refractory pruritic disorders. Pooling these conditions as "chronic pruritus" risks obscuring biologically distinct treatment signals. We therefore evaluated whether omalizumab responsiveness clusters within specific clinicobiological phenotypes and assessed the strength of treatment attribution and available safety evidence.

Materials and Methods
PubMed and Embase were searched from inception to 3 May 2026. Original human reports, including conference abstracts, were eligible when omalizumab was used for a defined pruritic disorder and itch was an explicit baseline symptom, treatment target or extractable outcome. Chronic spontaneous or inducible urticaria, atopic dermatitis or ordinary eczema, and bullous pemphigoid were excluded as primary treated conditions. Overlapping reports were de-duplicated and mixed cohorts contributed only extractable eligible subgroups. Because phenotype, study design, outcome measurement and treatment attribution were highly heterogeneous, findings were synthesised descriptively by clinicobiological phenotype rather than meta-analysed.

Results
Forty-seven reports contributed 143 unique patients across nine broad phenotype domains; no randomised controlled trial was identified. The most coherent signals occurred in mast-cell-mediated or mastocytosis-associated pruritus, Wells syndrome or eosinophilic cellulitis, recessive dystrophic epidermolysis bullosa or DEB-pruriginosa, and myeloproliferative neoplasm-associated pruritus.

The mast-cell domain contributed 54 review-usable patients, although the largest 55-patient cohort contained only 36 patients with pruritus and primarily measured broader mediator symptoms. Eight patients with Wells syndrome showed recurrent improvement or disease control, with relapse-rechallenge support in two reports. Fourteen patients contributed to the RDEB or DEB-pruriginosa signal; in the prospective 11-patient RDEB study, 10 had good clinical outcomes and group pruritus improved by 42%. All seven patients in the MPN-associated pruritus series improved descriptively.

Supportive but less secure signals occurred in selected chronic prurigo-spectrum disease, aquagenic pruritus, eosinophilic dermatosis of haematologic malignancy, non-BP autoimmune blistering disease and treatment-related pruritic eruptions. The treatment-related domain included 24 patients and suggested a potential treatment-preserving role: in a non-BP immune-checkpoint-inhibitor subset, 8/13 responded and 11/13 continued immunotherapy uninterrupted. Counter-signals included non-response in both reported prurigo nodularis patients, inconclusive or negative Netherton syndrome evidence and no response during three months of omalizumab in severe pruritus preceding classical Hodgkin lymphoma. Safety reporting was inconsistent; the

largest mast-cell-disorder cohort reported adverse events in 16/55 patients, including five discontinuations and one severe reaction with laryngeal oedema/dyspnoea.

Conclusions

Omalizumab shows a recurrent but low-certainty, phenotype-dependent antipruritic signal outside established urticaria use. The evidence does not support a pooled efficacy estimate or broad treatment recommendation across chronic pruritus. The strongest rationale for prospective evaluation lies in biologically coherent phenotypes, particularly mast-cell-associated itch, Wells syndrome, RDEB or DEB-pruriginosa, MPN-associated pruritus and selected treatment-related eruptions where steroid avoidance or continuation of essential therapy may be clinically valuable.

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Abstract N°: 3882

Health Economic Evaluation of Dose Reduction of IL17 and IL23 inhibitors for psoriasis: A Cost-utility Analysis of the BeNeBio trial

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Introduction
Dose reduction (DR) of IL17 and IL23 inhibitors (i) by stepwise interval prolongation has recently been shown to be non-inferior to usual care (UC) with regard to the proportion of persistent flares in patients with psoriasis with stable low disease activity in the BeNeBio trial. The aim of the current study is to evaluate the cost-utility of DR compared to UC.

Materials and Methods
In an international, multicentre, pragmatic, randomized, controlled, non-inferiority trial, 244 patients with psoriasis with stable low disease activity using an IL17i or IL23i were randomized to DR by stepwise interval prolongation or UC. Every 3 months, patients completed the EuroQol-5-Dimensions-5-Levels (EQ5D5L) and Short Form-6-Dimensions version 2 (SF-6Dv2) questionnaires during a total follow-up of 18 months. At end of the study, quality adjusted life years (QALYs) were calculated based on EQ5D5L and SF-6Dv2 utility scores. Healthcare costs included the total costs of the biologic used, and the costs of the number of outpatient visits and telephone consultations during 18 months. Utilities and total costs were valued with Dutch tariffs and prices as a base case analysis, and with Belgian tariffs and prices as sensitivity analysis. Analysis was according to the principle of intention-to-treat. Missing data was imputed by multiple imputation nested within bootstrap. A cost-utility plane was analysed by bootstrapping with 5000 replications. Incremental net monetary benefit (iNMB) was analysed for willingness to accept (WTA) thresholds ranging from €20,000 to €250,000.

Results
DR saved mean costs of €7038/patient over 18 months compared to UC, and showed a loss of 0.03 QALYs with both the EQ5D5L and SF-6Dv2 utility scores (Fig.1a+b). The incremental cost-effectiveness ratio was €234,644/QALY based on EQ5D5L utilities and €234,549/QALY based on SF-6Dv2 utilities. The cost-utility plane showed that the bootstrap replications fell for 80.7% in the south-west quadrant for the EQ5D5L and for 86% for the SF-6Dv2 (Fig.1a+b) reflecting lower costs with a marginal QALY loss for DR compared to UC. The iNMB remained positive for WTA thresholds up to €100,000 for both analysis with EQ5D5L and SF-6Dv2 utilities (Fig.1c+d). Sensitivity analysis with Belgian tariffs and prices is following.

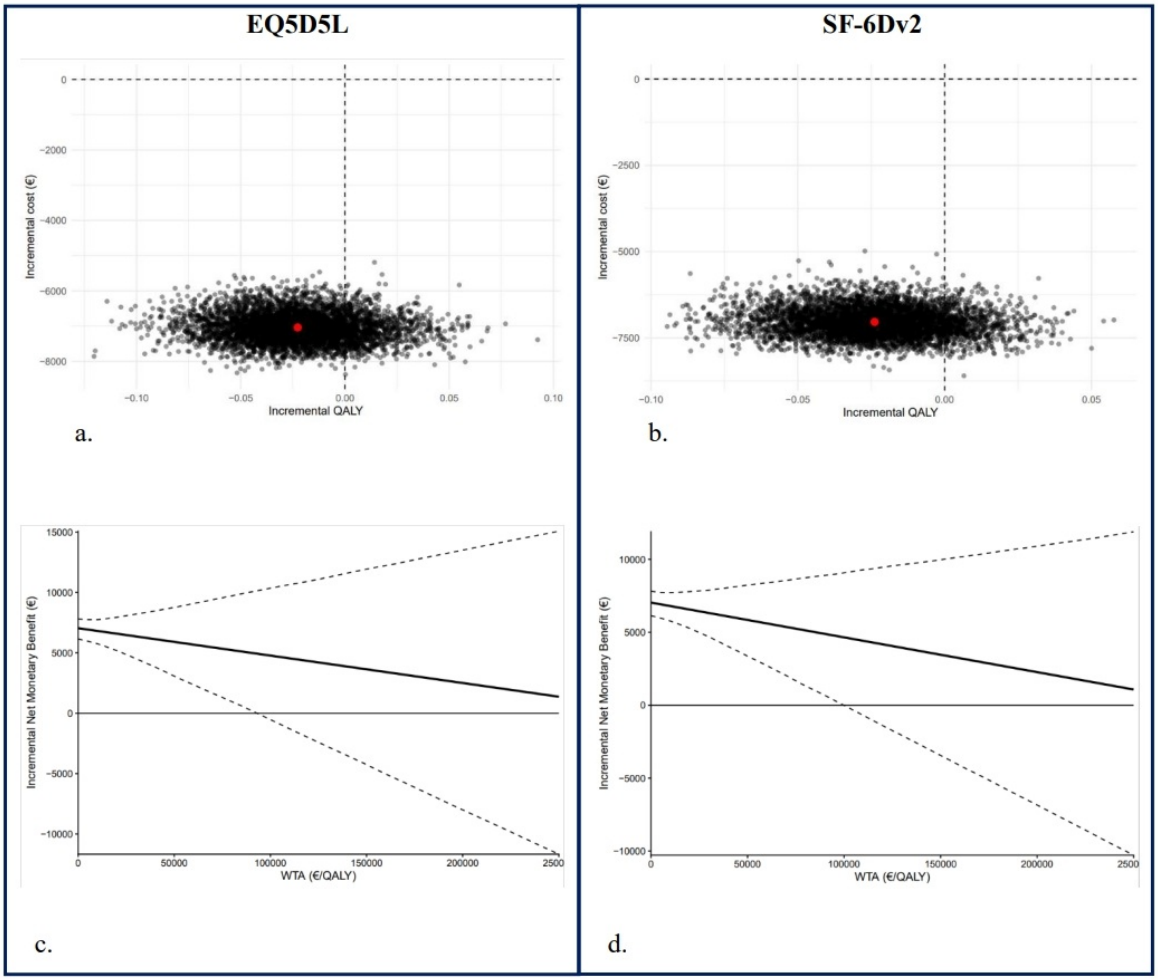

Figure 1: Results of the cost-utility analysis with Dutch tariffs and prices. In the cost-utility planes for the EuroQol-5-Dimensions-5-Levels (EQ5D5L) (a) and Short Form-6-Dimensions version 2 (SF-6Dv2) (b), each black dot represents one of the 5000 bootstrap replications; they fell for 80.7% in the south-west quadrant for the EQ5D5L (a) and for 86% for the SF-6Dv2 (b). The red dot represents the mean of all replications: DR saved mean costs of €7038/patient over 18 months compared to UC, and showed a loss of 0.03 QALYs with both the EQ5D5L and SF-6Dv2 utility scores. In the cost-acceptability curves for the EQ5D5L (c) and SF-6Dv2 (d) the incremental net monetary benefit (iNMB) for a range of willingness to accept (WT) thresholds is shown with 95% confidence intervals (dotted lines). For both the analyses with EQ5D5L and SF-6Dv2, the iNMB remained positive for WTA thresholds up to €100,000.

Conclusions

This cost-utility analysis shows that DR saves substantial costs per patient over 18 months of biologic use. It results in minimal and non-significant mean loss of 0.03 QALY. When this loss is compensated with currently widely accepted thresholds up to €100,000 per QALY, the net saving is still large. In conclusion, DR of IL17i and IL23i by stepwise interval prolongation is cost-effective compared to UC.

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Abstract N°: 3912

Indirect Treatment Comparison of Dupilumab Versus Remibrutinib on Efficacy Outcomes in Patients with Chronic Spontaneous Urticaria

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Introduction

Chronic spontaneous urticaria (CSU) is a debilitating inflammatory skin disease characterised by wheals, angioedema, or both. CSU persists for >6 weeks and imposes substantial clinical, humanistic, and economic burden on patients. Recent approvals of dupilumab (an interleukin [IL]-4/IL-13 inhibitor) and remibrutinib (a Bruton's tyrosine kinase inhibitor) have expanded the treatment landscape for antihistamine-resistant patients with CSU. However, no head-to-head trials have directly compared their efficacy to inform treatment decisions. While meta-analyses comparing dupilumab and remibrutinib exist, none has exclusively analysed Phase 3 randomised controlled trial (RCT) data. This analysis compares the efficacy of dupilumab versus remibrutinib in patients with CSU through a Bucher indirect treatment comparison (ITC) using Phase 3 clinical trial data, focusing on reduction in itch severity, hive severity, and urticaria activity.

Materials and Methods

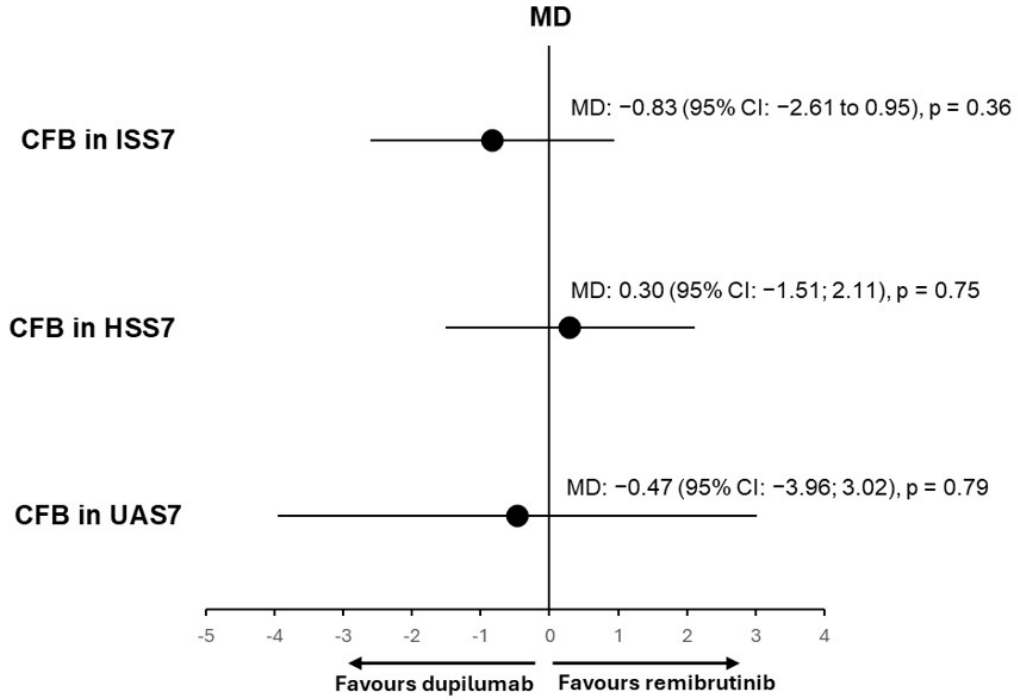
A pairwise adjusted Bucher ITC was conducted using publicly available aggregate data from the following trials in patients with CSU inadequately controlled with H1 antihistamine: LIBERTY-CSU CUPID-A and CUPID-C (NCT04180488) for dupilumab 300 mg every 2 weeks (q2w) in omalizumab-naïve patients, and REMIX-1 (NCT05030311) and REMIX-2 (NCT05032157) for remibrutinib 25 mg twice daily (bid) in omalizumab-naïve and omalizumab-exposed patients. Placebo was the common comparator. Key efficacy endpoints included change

from baseline (CFB) in the Itch Severity Score over 7 days (ISS7; range 0–21) and the Urticaria Activity Score over 7 days (UAS7; range 0–42) at Weeks 12 and 24 with higher scores indicating greater disease severity. Additional efficacy endpoints included: (i) CFB in the Hive Severity Score over 7 days (HSS7; range 0–21, higher scores indicate more severe wheals/hives) at Week 24, and (ii) the proportion of patients achieving UAS7≤6 and UAS7=0 at Week 24. Mean difference (MD) was reported for continuous measures, and odds ratio (OR) for binary outcomes. Statistical significance was determined using two-sided equivalence tests with 95% confidence intervals (CIs).

Results

Data from pooled trials included dupilumab (*N* = 144) and remibrutinib (*N* = 606), with placebo arms of *N* = 145 and *N* = 306, respectively. Populations were comparable at baseline across age, UAS7 score, and disease duration, supporting the indirect comparisons between trials, acknowledging a difference in omalizumab history between the studies. Dupilumab and remibrutinib demonstrated comparable improvements across all efficacy endpoints, with no statistically significant results for all ITC estimates. At Week 12, the MDs of dupilumab versus remibrutinib for CFB in ISS7 and UAS7 were 0.87 (95% CI: −1.02 to 2.76, p = 0.37) and 2.96 (95% CI: −0.73 to 6.65, p = 0.12), respectively. At Week 24, the MDs of dupilumab versus remibrutinib for CFB in ISS7 and UAS7 were −0.83 (95% CI: −2.61 to 0.95, p = 0.36) and −0.47 (95% CI: −3.96 to 3.02, p = 0.79), respectively. The MD of CFB in HSS7 at Week 24 was 0.30 (95% CI: −1.51 to 2.11, p = 0.75) (**Fig**.). The ORs of dupilumab versus remibrutinib in achieving UAS7≤6 and UAS7=0 at Week 24 were 0.99 (95% CI: 0.55 to 1.78, p = 0.97) and 0.90 (95% CI: 0.47 to 1.75, p = 0.75), respectively.

Fig. Indirect Treatment Comparison of Change from Baseline in ISS7, HSS7, and UAS7 at Week 24: Dupilumab Versus Remibrutinib



CFB, change from baseline; CI, confidence interval; HSS7, Hive Severity Score over 7 days; ISS7, Itch Severity Score over 7 days; MD, mean difference; UAS7, Urticaria Activity Score over 7 days.

Conclusions

In this ITC of Phase 3 RCT data, dupilumab and remibrutinib exhibit comparable efficacy at Weeks 12 and 24 in reducing itch, hive severity and, urticaria activity in patients with CSU inadequately controlled with H1

antihistamine. Given the comparable efficacy, treatment selection may be guided by other factors including safety profile, comorbidities, and patient preferences. These findings may not fully reflect real-world situations and may not be generalisable to real-life clinical practice. Additionally, differences in baseline omalizumab exposure were not accounted for due to the unavailability of patient level data from the REMIX trials. Further research is needed to evaluate comparative effectiveness in real-world settings.

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Abstract N°: 4008

Beyond Blockade, Towards Degradation: A Critical Review of Targeted Protein Degradation (STAT6) Versus Monoclonal Antibody Binding in Th2-Driven Dermatoses-Implications for Long-Term Remission Architecture

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Introduction

Blocking of IL-4 and IL-13 with dupilumab has changed that how we treat atopic dermatitis, it requires patients to manage frequent, long-term dosage revisions stop the drug and the disease tends to flash up targeted protein degradation of STAT6 which is the central transcription factor in IL-4/IL-13 signalling has completely flipped the script, transforming an historically 'undruggable' target into a promising therapeutic approach instead of just blocking the ligands outside the cell, TPD clears off the machinery inside the cell. This approach of removing the effector instead of the signals creates for us a new therapeutic philosophy. Here we examined this shift of monoclonal antibody binding to TPD, delivering into how this might reshape periods of remission, the length of time patients can go without treatment.

Materials and Methods

We collected this data from both preclinical and translational studies which was focused on proteolysis-targeting chimeras that lowers up STAT6, traditional monoclonal antibody blockers of IL-4Rα, and in-vivo models that simulates allergic inflammation. Main sources include Cohen et al, which specifies the mechanism of STAT6 degraders, with clinical pharmacokinetics and pharmacodynamics of dupilumab, also studies on how long remissions last after treatment stops in AD. All data ranges from 2019 through 2025, which was fed into a theoretical PK-PD structure that kept a track on how long target proteins lasts in the cell and how quickly it gets renewed

Results

STAT6 PROTACs got the rid of most intracellular STAT6 within the six hours. Recovery relies on creating new proteins, which have a half-life of 24 to 48 hours, while genetic activity returns to normal within four to seven days. Dupilumab works by saturating IL-4Rα receptors on both circulating and tissue-resident cells. As blood levels of the drug falls, the blockade lifts, allowing Th2 signalling to surge back. Mouse models of asthma showing that how a single, short pulse of STAT6 degradation can protect against allergen re-challenge for over eight weeks, well past the point when the drug itself is gone. Accordingly, the atopic dermatitis maintenance may permit 36- to 52-week treatment holidays, provided immune integrity is maintained. However, the long-term safety of STAT6 blockade remains a concern given its functions in helminth clearance and tumour monitoring. In mice, complete blow out increases' vulnerability to nematode infection, but when STAT6 is diminishes in adulthood, evidence suggests that immune memory is still maintained.

Conclusions

The breakdown of STAT6 could be a game-changer for therapy, offering a new approach that goes beyond just blocking it and instead removes the protein entirely, it suggests a shift from continuous ligand blockade to

regular, intracellular effector clearance. By evolving from temporary receptor suppression to deep transcriptional reprogramming, AD therapy could move toward much longer, sustained remission periods as with any new strategy, we just can’t ignore the possible risks infectious and cancer-related concerns must be closely pursued, and closed observed strategies need to be layered by age and comorbidity.

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Abstract N°: 4013

HiSCR100 as the New Benchmark. A Critical Meta-Analysis of the Gap Between Statistical Significance and Complete Disease Resolution in Contemporary Hidradenitis Suppurativa Trials (Povorcitinib, Izokibep, Bimekizumab)

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Introduction

In the most of the clinical trials for hidradenitis suppurativa (HS) relies on HiSCR specifically, HiSCR50, which means at least a 50% drop in the number of abscesses and inflammatory nodule as the main endpoint. Still at reaching HiSCR50 doesn't mean the disease is inactive. Patients often have leftover lesions, and these remnants set them up for early relapses. Now, if you raise the standard HiSCR100 that means a complete absence of inflammatory lesions you will actually start to see a benchmark which signals the possibility for the treatment-free remission. To make it more senseful we took a hard look at recent trials and critically meta-analysed the difference between accomplishing HiSCR50 or HiSCR75 and reaching full HiSCR100, exploring how this gap influences a patient's control over their disease after stopping the treatment.

Materials and Methods

We orderly meta-analysed phase 2 and 3 randomized clinical trials which studied povorcitinib which is a JAK1/TYK2 inhibitor, izokibep-an IL-17A inhibitor small molecule and bimekizumab which blocks IL-17A and IL-17F in the patients with moderate-to-severe HS, covering up publications from 2019 to 2025. Only five studies fit our inclusion criteria. From each of them we extracted rates of HiSCR50, HiSCR75, and HiSCR100 at the primary trial assessment points and that tracked post-treatment sovereignty by measuring the time it took for patients to lose HiSCR or need intensified therapy. We ran a random-effects meta-analysis to generate odds ratios for complete resolution and estimation of hazard ratios for the time to sovereignty loss by comparing HiSCR100 achievers to those settling for HiSCR50 alone, using survival data when available.

Results

When we combined this data nearly around 48–62% of patients reached HiSCR50 with these agents but out of them 32–44% made it to HiSCR75. A shocking discovery was that a mere 8–15% only achieved HiSCR100 at the main endpoint. That leaves us a glaring absolute gap that on average, a 42-percentage point difference with 95% CI 35–50 between patients hitting HiSCR50 and those who reach HiSCR100. Looking at what happens after treatment, patients who hit HiSCR50 but fell short of HiSCR100 only maintained disease sovereignty for a short period of time that is 5.6 weeks on average (95% CI 4.1–7.2) before needing any more intervention. The contrast that with HiSCR100 achievers' ones who managed 21.3 weeks (95% CI 17.8–24.9) a difference, showed up by a hazard ratio of 0.28 (95% CI 0.18–0.44). Breaking it down further by drug, bimekizumab delivered the highest HiSCR100 rate at 15%, an outcome in line with its potent dual neutralization of IL-17A and IL-17F.

Conclusions

HiSCR100 stands out as the terminal point which actually matters for sustained disease control. Settling up for HiSCR50 alone doesn’t tell you which patients can safely stop treatment, it just marks improvement, not remission. As we move forward, HS clinical trials should treat HiSCR100 as an important secondary endpoint and always report us that how long patients remain in remission off treatment, stratifying those results by the depth of response.

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Abstract N°: 4029

The "OX40 Paradox": A Systematic Review Investigating the Temporal Lag in Clinical Response for OX40/OX40L Inhibitors (Amlitelimab, Rocatinlimab)-Defining a New "Induction-Maintenance" Sequence Model

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Introduction

In OX40 and OX40L inhibitors which are amlitelimab and rocatinlimab. OX40 therapy takes time about 8-16 weeks but it offers lasting, off-treatment remissions. "The OX40 Paradox." This gap between the response and administration invalidates standard on treatment measures and suggests an immunological restructuring, rather than a fast cytokine-neutralizing mechanism of action. This review explores the mechanism of action and it then proposes an induction-maintenance treatment model.

Materials and Methods

This systematic review we analysed the data from phase 2b and phase 3 clinical trials based on the investigations on amlitelimab and rocatinlimab, with a main objective on three key aspects those are 1.how quickly the patients respond to treatment? 2.how well those responses are maintained after treatment ends? 3. what happens at the molecular level based on translational pharmacodynamic analysis? Reviewing the five primary sources published between 2023 and 2025, We evaluated a combined outcome, which also included the percentage of patients achieving EASI-75, that means at least a 75% improvement in their Eczema Area and Severity Index at weeks 4, 8, 16, and 24. To assess durability off treatment, we examined the mean time to relapse following elective discontinuation. The treatment effects on immune cell populations were then explored through repeated measurements of T cell subsets, particularly OX40+ memory T cells and regulatory T cells, to help us to clarify the drugs mechanisms of action and their potential for lasting therapeutic effects. Where more granular data were available, we then used individual patient data for restricted mean survival time analysis to quantify the durable benefits of the treatment. This approach enabled us for a comprehensive comparison between amlitelimab and rocatinlimab, shedding light on both their clinical efficacy and biological impact in the management of atopic dermatitis.

Results

EASI-75 responses reached about 15% to 20% at Week 4 with both agents comparing it to placebo. By the week 16 the responses jumped to 55%–60% and by Week 24 they hit 65%–70%. This delayed response lined up with a stable drop in circulating pathogenic Th2/Th22 memory T cells at OX40+ while FoxP3+ Treg cells increased at the same time. The treatments didn't really start regulating the immune system until after 12 weeks from the start of therapy. On average, patients who stopped drug treatment on their own stayed in remission for 26.3 weeks with amlitelimab and 22.1 weeks with rocatinlimab. That's actually the longest time to recall reported for any drug so far, nothing else, approved or investigational, has come close. Fast-acting agents bridge the 4–8-week gap, allowing slow-onset OX40-targeted therapies to deliver long-term remodelling. After that the OX40 antagonists are used for its maintenance, and then the patient eventually moves to a next period without medication.

Conclusions

The OX40 paradox is a hallmark of disease-modifying immunotherapy rather than a restriction. The tailored induction-maintenance sequencing that enables patients to bypass the lag phase while maintaining the longest observed, treatment-free intervals in AD. This model needs trial validation.

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Abstract N°: 4033

Long-Term Homeostatic Consequences of Narrow Cytokine Blockade: A Theoretical Safety Analysis of IL-22RA1 Antagonism (Temtokibart) on Tissue Repair and Microbial Defense Pathways

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Introduction

The IL22 is a pleiotropic cytokine that plays an important role in epithelial repair, induction of antimicrobial peptide (AMP), and integration of mucosal barrier. An anti-IL22 receptor that is subunit IL22RA1 which blocks antibody, Temtokibart, is currently being evaluated in atopic dermatitis. While its therapeutic benefit may be clear, chronic blockade of this homeostatic axis theoretically has the ability to impair tissue repair and predispose it to the infection. We conducted a safety analysis with combining preclinical, clinical and evolutionary data.

Materials and Methods

We built this risk structure using four of the key sources to prioritize safety and efficacy which are the following 1. Phase 1/2a clinical data for temtokibart, 2. predictive algorithms from IL-22-deficient mouse models which evaluates wound healing and bacterial infection responses, 3. human genetic data linking IL-22 receptor deficiencies to skin immunity, and the evolutionary conservation of the IL-22 pathway. From major sources which were published in years from 2019 and 2025. The main terminal points that were established in this were the delayed wound healing, the increased skin infections, and the wider consequences for skin barrier function if blockade persisted over the longer period.

Results

In the temtokibart phase 2a trial with N=123, in these two patients experienced continuous skin infections and the wound healing delay during the off-treatment phase, consistent with impaired IL-22 signalling. Without the IL-22, bacterial skin infections heal with rate 40% slower than the normal and are become more dangerous. Individuals with severe, inherited IL22RA1 defects experience continuous infections of the skin/mucous membranes (chronic mucocutaneous candidiasis) alongside the frequent, infected hair follicles (staphylococcal folliculitis). Blocking the IL-22 pathway for over 12 weeks weakens the skin's defence system by removing the unique protective signals, even though other defence proteins like IL-17 and IL-19 remains active. This extended treatment establishes a "sovereignty trap," by destroying the healthy skin bacteria and leaving patients highly vulnerable to infections even after they stop the medication. Moreover, the simulation computer models show that blocking this pathway for six months reduces the skin's ability to heal and regenerate by 15% to 20%. This loss of healing power is especially dangerous for older individuals who already have delicate skin barriers.

Conclusions

Blocking IL-22RA1 makes sense when you’re dealing with the Th22-driven inflammation, it can disorder with tissue repair and obstructs with the body’s defences against germs. So, it’s not enough to just watch safety during treatment, the closely observing needs to keep going forward, even during that vulnerable window when the

problems can show upIf our target is going to be IL-22, then our methods needs some adjustments like thinking short courses rather than persistent dosing and using biomarkers to guide when to start and stop, and being careful that not to include patients who already struggle with the infections or slow wound healing.

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Abstract N°: 4066

Real-world effectiveness of dupilumab in reducing itch and disease severity in adults with prurigo nodularis: A systematic literature review and meta-analysis

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Introduction

Dupilumab, an interleukin-4 receptor alpha antagonist, was the first biologic approved for prurigo nodularis (PN), based on efficacy and safety data from the PRIME and PRIME2 Phase 3 trials. While real-world (RW) studies have confirmed its effectiveness, inconsistent outcome measures and assessment timepoints limit their utility for informed clinical decision-making. We conducted a systematic literature review (SLR) and meta-analysis (MA) of RW evidence to assess dupilumab's effectiveness in moderate-to-severe PN assessing Itch-Numeric Rating Scale (I-NRS) and Investigator's Global Assessment of Prurigo Nodularis Scale (IGA-PN-S).

Materials and Methods

The SLR was conducted in accordance with the Cochrane methodology and PRISMA guidelines. Embase and MEDLINE® were searched (inception–June 17, 2025) to identify published RW studies assessing the effectiveness of dupilumab (monotherapy or combination therapy) in adult patients with a confirmed diagnosis of PN. Effectiveness outcomes included I-NRS score (0–10, no to worst possible itch) and IGA (0–4, higher scores reflect greater nodule burden; IGA of Chronic PN Scale [IGA-CPN-S]; IGA-PN-S and IGA Activity [IGA-A]). Conference proceedings published within 3 years prior to the search date were included.

Separate random-effects MA were conducted for short-term (12–28 weeks), mid-term (32–70 weeks), and long-term (≥84 weeks) follow-ups. Continuous outcomes were assessed as change from baseline (CFB) and dichotomous outcomes as the proportion of patients experiencing the event, each reported with 95% confidence intervals (95% CI). Studies not reporting the outcomes of interest, with insufficient sample size (n<10), or limited follow-up time (<12 weeks) were excluded from MA. A meta-regression (MR) analysis was performed using the final follow-up time as a covariate.

Results

The SLR identified 24 relevant studies from 1,260 records. Mean age of the included patients ranged from 39.3–70.0 years (17 studies). Mean scores indicating baseline disease severity (IGA or PN variants) ranged from 2.6–3.7 (14 studies) and mean baseline pruritus severity (I-NRS) ranged from 7.1–9.8 (21 studies).

I-NRS severity score substantially reduced across all time periods. Pooled mean CFB was: −6.47 (95% CI: −7.85, −5.08) at short-term (9 studies, *n*=201), −7.17 (95% CI: −8.36, −5.98) at mid-term (4 studies, *n*=70), and −7.69 (95% CI: −8.04, −7.34) at long-term (2 studies, *n*=49), and improvements were maintained or increased over time.

At short-term follow-up, clinician-assessed disease severity measured by IGA-CPN-S showed statistically significant reduction with pooled CFB: −1.61 (95% CI: −2.26, −0.97; 2 studies, *n*=84). A substantial percentage of patients achieved an IGA-PN-S score of 0/1 at short-term (2 studies, *n*=49): pooled estimate 65.16% (95% CI: 50.83%, 77.18%). Pooled percentage of patients achieving IGA-A = 0 was 63.98% (95% CI: 51.52%, 74.80%) at short-term (2 studies, *n*=67).

At short-term and mid-term follow-up, pooled percentage of patients achieving IGA = 0 was 16.48% (95% CI: 4.75%, 43.84%; 4 studies, *n*=109) and 31.04% (95% CI: 17.01%, 49.71%; 2 studies, *n*=29), respectively.

Consistent results were observed for MR, and no meaningful association was found between follow-up time and itch outcome.

Conclusions

Dupilumab was associated with substantial and sustained real-world improvements in itch and disease severity outcomes in patients with moderate-to-severe PN, consistent with findings from the PRIME and PRIME2 trials. This study provides pooled RW effectiveness estimates across clinically relevant follow-up periods and supports the long-term effectiveness of dupilumab in routine clinical practice. However, interpretation is limited by the predominance of small, uncontrolled single-cohort, and observational pre/post studies, as well as incomplete outcome reporting, missing data across timepoints, small sample sizes for selected outcomes, and heterogeneity.

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Abstract N°: 4101

Beyond Skin Clearance: Patient Priorities and Shared Decision-Making in Psoriasis Biologic Therapy

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Introduction
Introduction

Biologic therapies have transformed the management of moderate-to-severe psoriasis; however, optimal outcomes depend not only on efficacy, but also on alignment with patient expectations and treatment priorities. Understanding what patients value most when selecting biologic therapy is essential to support shared decision-making and patient-centred care. Contemporary real-world data describing patient treatment priorities remain limited. This study aimed to evaluate treatment priorities, expectations, and decision-making preferences among patients with psoriasis receiving biologic therapy in a real-world tertiary cohort.

Materials and Methods
Materials and Methods

A cross-sectional anonymous survey was conducted among adults with psoriasis receiving biologic therapy through a tertiary public dermatology service in *Australia*. A 13-item questionnaire assessed demographics, disease duration, treatment priorities (including efficacy, safety, durability, speed of onset, and injection frequency), expectations of response, safety concerns, and preferences regarding treatment decision-making. Surveys were completed during clinic visits and entered into a de-identified database for analysis. Participation was voluntary with implied consent. Descriptive statistics were used for analysis.

Results
Results

A total of 161 patients completed the survey. The cohort was predominantly male (58.4%), with psoriasis longstanding in many participants, with 44.1% reporting disease duration exceeding 20 years. Efficacy was the most important consideration when selecting biologic therapy (45.3%), followed by safety (36.6%) and durability of response (34.8%). Clear or near-clear skin was prioritised by 56.5% of respondents as the most meaningful efficacy outcome. Speed of onset was considered important or somewhat important by 64.3% of participants, while 86.3% valued long-term durability of response. Safety concerns centred on systemic health complications, malignancy risk, and infection risk. Injection frequency was of comparatively lower importance. Most patients preferred a collaborative treatment approach, with 71.8% favouring shared clinician–patient decision-making.

Conclusions

Conclusions

Patients with psoriasis prioritise efficacy, durable disease control, and safety when selecting biologic therapy, alongside a strong preference for shared decision-making. These findings reinforce the importance of aligning biologic discussions with patient-defined treatment goals and expectations to support patient-centred psoriasis care. Structured educational strategies may further support patient understanding and engagement during biologic selection.

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Abstract N°: 4108

Visual Educational Tools and Shared Decision-Making in Psoriasis Biologic Therapy

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Introduction
The management of psoriasis has been transformed by biologic therapies, with multiple agents now available that differ in efficacy, safety, durability, and dosing frequency. Selecting an appropriate biologic increasingly requires shared decision-making between clinician and patient. However, dissatisfaction with psoriasis care is often driven by communication gaps and difficulty understanding treatment options rather than treatment efficacy alone. There is limited evidence evaluating structured or visual educational tools to support biologic counselling and treatment decision-making. This study aimed to explore patient perspectives regarding biologic education, treatment priorities, and shared decision-making in a real-world psoriasis cohort.

Materials and Methods
A cross-sectional anonymous survey was conducted among adults with psoriasis receiving biologic therapy through a tertiary public dermatology service in Sydney, Australia. A 13-item questionnaire assessed demographics, disease duration, treatment priorities, expectations of response, safety concerns, and decision-making preferences. Surveys were completed during clinic visits and entered into a de-identified database for analysis. Participation was voluntary with implied consent. Descriptive statistics were used.

Results
A total of 161 patients completed the survey. Most participants prioritised biologic efficacy, safety, and durability of response when selecting treatment. Clear or near-clear skin was identified as the most meaningful efficacy outcome for the majority of respondents, while speed of onset and long-term effectiveness were also considered important. Safety concerns commonly included risks of infection, malignancy, and systemic health complications. Most patients favoured a collaborative decision-making model involving both clinician expertise and patient input. Over 70% of respondents indicated that visual educational tools, particularly when combined with clinician discussion, would be helpful in understanding biologic treatment options. Qualitative feedback suggested these resources may be particularly valuable for patients from culturally and linguistically diverse backgrounds and for those overwhelmed by the complexity of biologic treatment choices.

Conclusions
Patients commencing biologic therapy value clear, balanced information regarding efficacy, safety, and durability and strongly support shared decision-making. Visual and structured educational tools may further support patient understanding, engagement, and confidence during biologic selection. These findings support the integration of patient-centred educational strategies into routine psoriasis biologic counselling.

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Abstract N°: 4122

JAK inhibitors in the Management of Idiopathic Photodermatoses: Systematic Review of Clinical Evidence

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Introduction

Idiopathic photodermatoses is a group of disorders marked by cutaneous reactions following ultraviolet radiation exposure. These mostly present with complaints of itching and burning sensations severely affecting the quality of life of the patient in their day to day activities. Clinical features include pigmentary changes, erythema, lichenification predominantly over prephotoexposed sites. Till date there is no FDA approved medication for these conditions. Current therapeutic interventions include photoprotective measures, antihistamines, topical steroids, hydroxychloroquine and immunosuppressive medications. Janus kinase Inhibitors are novel oral drugs targeting cytokines and growth factors implicated in the pathophysiology of different immune-mediated diseases. Literature highlights that the use of this class of drugs in a range of dermatological conditions consistently results in rapid clinical response and significant improvement within days to weeks of treatment initiation. Additionally, continued treatment for months lead to sustained control of disease symptoms and cutaneous lesions.

Materials and Methods

A literature search was done on PubMed Advanced database with keywords (Photodermatoses OR Chronic actinic dermatitis OR polymorphic light eruption OR Actinic prurigo OR solar urticaria OR hydroa vacciniforme OR Juvenile spring eruption) AND (JAK inhibitors OR Tofacitinib OR Baricitinib OR Upadacitinib OR Abrocitinib OR Ritlecitinib OR Ruxolitinib OR Deucravacitinib) which yielded 30 results out of which 20 articles were selected for review.

Results

The photodermatoses where JAK inhibitors have been reported to be of use include Polymorphic light eruption(1 case series), Actinic prurigo (5 case reports) and Chronic actinic dermatitis(1 longitudinal study, 3 case series and 10 case reports). Most of the patients had long standing disease with inadequate response to treatment including conventional immunosupressive drugs such as methotrexate, cyclosporine, mycophenolate mofetil and monoclonal antibodies including dupilumab. The JAK inhibitors that have been used include abrocitinib, tofacitinib, updacitinib and baricitinib. All patients reported an improvement in symptoms especially pruritus as early as within a few days of treatment initiation and complete resolution of cutaneous lesions within 2 weeks to 16 months. No serious adverse events have been reported.

Conclusions

Thus, JAK inhibitors are a promising therapeutic intervention for idiopathic photodermatoses especially in those where conventional therapies have either failed or are contraindicated. They have demonstrated a marked improvement in cutaneous lesions and early response in resolution of pruritus, which is often the most distressing associated symptom. However, there is a lack of clinical trials to recommend them as a first line treatment option.

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Abstract N°: 4125

Psoriasis, Obesity, and Targeted Therapy: Combined Tildrakizumab and Semaglutide Treatment

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Introduction

Psoriasis is a chronic immune-mediated inflammatory disease frequently associated with obesity, insulin resistance, and metabolic syndrome. Increasing evidence highlights the bidirectional relationship between psoriasis and adipose tissue–driven systemic inflammation, particularly involving the IL-23/Th17 axis. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs), including semaglutide, have demonstrated beneficial effects not only on weight reduction and glycemic control, but also on systemic inflammatory pathways potentially relevant to psoriasis pathogenesis. Nevertheless, data regarding the concomitant use of GLP-1 RAs with biologic therapies in psoriasis remain limited.

The objectives were to review the emerging evidence regarding the role of GLP-1 receptor agonists in psoriasis management and to describe a real-world case of concomitant treatment with tildrakizumab and semaglutide in a patient with moderate-to-severe plaque psoriasis, obesity, and type 2 diabetes mellitus.

Materials and Methods

A narrative review of the literature was conducted focusing on studies evaluating GLP-1 receptor agonists in patients with psoriasis and metabolic comorbidities. Additionally, we report the case of a patient with moderate-to-severe plaque psoriasis associated with obesity and type 2 diabetes mellitus treated with tildrakizumab initiated in June 2025, followed by adjunctive semaglutide therapy introduced in April 2026 in routine clinical practice.

Results

Recent studies suggest that GLP-1 receptor agonists may contribute to psoriasis improvement through both metabolic and immunomodulatory mechanisms, including reduction of adipose tissue inflammation and modulation of the IL-23/Th17 pathway. Prospective and observational data have demonstrated improvements in psoriasis severity, cardiometabolic parameters, and systemic inflammatory burden in obese patients receiving semaglutide therapy.

In our illustrative case, combined treatment with tildrakizumab and semaglutide was associated with marked clinical improvement, with PASI decreasing from 12.4 at baseline to 1.2 during follow-up and DLQI improving from 18 to 2. The patient additionally achieved weight reduction and improved metabolic control, accompanied by a significant enhancement in quality of life, without relevant adverse events during follow-up.

Conclusions

The combination of IL-23 inhibition and GLP-1 receptor agonist therapy may represent a promising therapeutic strategy in patients with psoriasis and obesity-related metabolic dysfunction. Beyond weight reduction, GLP-1

receptor agonists may exert complementary anti-inflammatory effects that enhance overall disease control and cardiometabolic outcomes. Further prospective studies are warranted to better define the efficacy, safety, and potential synergistic effects of combined biologic and metabolic-targeted therapies in psoriasis.

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Abstract N°: 4212

Evolution of clinical response with long-term nemolizumab therapy in prurigo nodularis: From symptom relief to improvements in sleep and quality of life.

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Introduction
Prurigo nodularis (PN) is a chronic inflammatory dermatosis characterised by severe pruritus, PN-associated pain, sleep disturbance and profound impairment of daily functioning and quality of life (QoL). Nemolizumab, an interleukin-31 receptor alpha antagonist, has demonstrated sustained efficacy in patients with PN up to week (W) 148. In the OLYMPIA long-term extension (LTE) study (NCT04204616), cumulative long-term safety was the primary endpoint and has been reported previously, showing a consistent safety profile over 3 years of treatment without new safety signals. This interim analysis aims to characterize longitudinal evolution of key symptomatic, functional and QoL outcomes with nemolizumab treatment in PN up to W148.

Materials and Methods
This descriptive analysis used available efficacy data from the ongoing OLYMPIA LTE study through W148, where adults with moderate-to-severe PN received nemolizumab 30 mg/60 mg (based on body weight; <90 kg/≥90 kg) subcutaneously every 4 weeks. Detailed study design and eligibility criteria for the OLYMPIA LTE trial have been described previously. Outcomes were assessed at W16, 52, 100 and 148 and included clinically meaningful improvement in itch (≥4-point reduction in Peak Pruritus Numerical Rating Scale [PP-NRS]), itch-free or nearly itch-free state (PP-NRS <2), improvement in sleep disturbance (Sleep Disturbance Numerical Rating Scale [SD-NRS] ≥4), PN-associated pain measures, dermatology-specific QoL (Dermatology Life Quality Index [DLQI]) and overall health status assessed using the EQ-5D visual analogue scale (VAS). Observed cases are presented without imputation of missing data, with analyses conducted in outcome-specific eligible populations at each visit.

Results
At data cut-off (21-Jul-2025), clinically meaningful improvements across multiple domains of PN disease burden were observed independently over long-term treatment, with 275/508 (54%) patients completing W148 and a median exposure of 1147.5 days. Across all assessed endpoints, improvements were evident by W16, increased

further by W52, and were maintained through W100 and W148.

Core PN symptoms improved early and continued over time. Clinically meaningful itch improvement (PP-NRS ≥4) was achieved by 80% of evaluable patients at W16, increased to 84% at W52, and was sustained by 92% at W100 and 87% at W148). In parallel, the proportion of patients who were itch-free or nearly itch-free (PP-NRS <2) increased from 54% at W16 to 61% at W52 and further to 72% at W100 and 73% at W148.

PN-associated pain improved substantially, with mean pain intensity decreasing from 7.6 at lead-in baseline to 2.1 at W16, 1.7 at W52 and remaining consistently low through W100 (1.4) and W148 (1.3). The proportion of patients reporting no PN-associated pain reached 60% at W148.

Functional outcomes improved alongside symptom control. Clinically meaningful improvement in sleep disturbance (SD-NRS ≥4) was reported by 72% of patients at W16, 75% at W52, 83% at W100 and 85% at W148.

Dermatology-specific QoL improved and maintained over time. Clinically meaningful improvement in DLQI (≥4-point improvement) was observed in 88% of patients at W16, 89% at W52, 90% at W100 and 91% at W148. The proportion of patients reporting no impact of disease on QoL (DLQI 0/1) was 33% at W16, 49% at W52, 54% at W100 and 55% at W148. EQ-5D VAS scores improved from 59.3 at lead-in baseline to 75.8 at W16, 78.9 at W52, 79.4 at W100 and 80.3 at W148, indicating better overall health status.

Conclusions

In the OLYMPIA LTE study, nemolizumab demonstrated sustained improvements across key symptomatic, functional and QoL domains in patients with PN. Improvements were observed early and maintained over long-term treatment across multiple dimensions of PN disease burden, including itch, PN-associated pain, sleep disturbance and overall QoL.

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Combination Therapy With Biologics and JAK Inhibitors in Refractory Psoriasis and Psoriatic Arthritis: A Cohort Study of Eight Patients

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Introduction

Psoriasis (PSO) and psoriatic arthritis (PsA) are chronic immune-mediated diseases sharing the IL-23/Th17 inflammatory axis, yet cutaneous and articular manifestations frequently respond differentially to the same therapeutic agent. Available treatments include biologics targeting TNF-α, IL-17, IL-23 and IL-12/23, as well as oral JAK inhibitors; however, a clinically significant subset of patients with concurrent PSO and PsA fails to achieve simultaneous skin and joint disease control under any single agent after cycling through multiple lines of therapy. The differential cytokine expression between skin and joint — with stronger associations of IL-17 and IL-23 in cutaneous tissue compared with mixed profiles in the joint — may partly explain the failure of monotherapy and provides a rationale for the use of combination therapy. Evidence guiding combination b/tsDMARD strategies, particularly regarding optimal drug class combinations and safety in Latin American populations, remains scarce.

Materials and Methods

We retrospectively reviewed records of 8 patients with concurrent moderate-to-severe PSO and PsA followed at a tertiary dermatology-rheumatology outpatient clinic between 2020 and 2026, all requiring combination b/tsDMARD therapy — either two biologics from distinct drug classes, or a biologic combined with a JAK inhibitor — after failing multiple sequential monotherapies. Conventional DMARDs were not selected as combination partners due to prior failures, gastrointestinal intolerance, or hepatotoxicity. Cutaneous response was assessed by PASI and DLQI; articular response was evaluated based on patient-reported improvement in pain and functional capacity. Serious adverse events including infections, cardiovascular events and malignancies were monitored.

Results

The cohort comprised 6 women and 2 men (age range 39–66 years), with PSO duration of 16–61 years and PsA duration of 5–23 years. Joint involvement was predominantly peripheral (6/8); 2 patients had axial plus peripheral disease. Prior failed systemic therapies ranged from 1 to 9 per patient and included, across the cohort, anti-TNF, anti-IL-17, anti-IL-23, anti-IL-12/23 biologics, methotrexate, cyclosporine, leflunomide, acitretin, and oral JAK inhibitors used as monotherapy. Six patients initiated combination therapy due to persistent articular disease despite adequate cutaneous control with a biologic; 2 required combination due to uncontrolled skin disease despite articular response. Drug class combinations used were: anti-IL-23 + anti-TNF-α (2 patients), anti-IL-17 + anti-TNF-α (1), anti-IL-12/23 + JAK inhibitor (2), anti-IL-17 + JAK inhibitor (2), anti-IL-23 + JAK inhibitor (1). Six of 8 patients achieved clinically meaningful cutaneous and articular improvement; 5 reached PASI <1, including 4 with PASI 0 and DLQI 0. Two patients showed no articular improvement during the observation period. No serious adverse events — infections, cardiovascular events or malignancies — were recorded in any patient.

	P1	P2	P3	P4	P5	P6	P7	P8
Age / sex	52 / F	56 / M	63 / F	50 / M	63 / F	52 / F	66 / F	39 / F
Smoking	Former	Former	Current smoker	Former	Former (42 pk-yrs)	Never	Former	Never
PSO / PsA duration (yrs)	16 / 6	16 / 5	23 / 23	12 / 8	24 / 10	19 / 9	61 / 21	24 / 14
Joint involvement	Peripheral	Peripheral	Peripheral	Axial + periph.	Peripheral	Peripheral	Axial	Peripheral
Failed systemics (n)	5	1	6	5	4	5	9	5
Failed therapies	MTX, SEC, IFX, TOF, UPA	MTX	MTX, CsA, ACT, SEC, USTE, IFX	MTX, SEC, IFX, TOF, CsA	MTX, ADA, IFX, SEC	ADA, ETA, CsA, LEF, USTE	LEF, TOF, CsA, SEC, MTX, IFX, ADA, ETA, GOL	SEC, LEF, CsA, MTX, ADA
Failure type prompting combination	Articular	Articular	Cutaneous	Articular	Articular	Articular	Articular	Articular
Combination regimen (drug classes)	anti-IL-23 + anti-TNF-α	anti-IL-17 + anti-TNF-α	anti-TNF-α + anti-IL-23	anti-IL-12/23 + JAK inh.	anti-TNF-α + JAK inh.	anti-IL-17 + JAK inh.	anti-IL-12/23 + JAK inh.	anti-IL-23 + JAK inh.
Days to articular remission	—	90	—	90	30	30	—	110
Days to skin remission	—	—	90	—	—	—	—	—
Days on combination therapy	—	223	380	403	30	276	230	116
Skin response (PASI / DLQI)	Awaiting follow-up	PASI 0.2 DLQI 0	PASI 0 DLQI 1	PASI 0.6 DLQI 8	PASI 0 DLQI 0	PASI 0 DLQI 0	PASI 3.6 DLQI 7	PASI 0 DLQI 0
Joint response (pt-reported)	Not yet assessed	Improved	Improved	Improved	No improvement	Improved	No improvement	Improved
Adverse events	None	None	None	None	None	None	None	None

ACT, acitretin; ADA, adalimumab; CsA, cyclosporine; DLQI, Dermatology Life Quality Index; ETA, etanercept; F, female; GOL, golimumab; IFX, infliximab; inh., inhibitor; JAK, Janus kinase; LEF, leflunomide; M, male; MTX, methotrexate; PASI, Psoriasis Area and Severity Index; PSO, psoriasis; PsA, psoriatic arthritis; pk-yrs, pack-years; SEC, secukinumab; TOF, tofacitinib; UPA, upadacitinib; USTE, ustekinumab; yrs, years. (—) not achieved or not yet assessable.

Conclusions

Our data suggest that combination b/tsDMARD therapy is a promising rescue strategy for patients with concurrent PSO and PsA refractory to sequential monotherapy, offering simultaneous control of skin and joint disease in the majority of cases. Combination b/tsDMARD therapy — pairing two biologics from distinct classes or a biologic with a JAK inhibitor, across five drug class combinations — demonstrated meaningful cutaneous and articular improvement in 6 of 8 patients with refractory PSO and PsA, with an acceptable short-term safety profile. The complementary mechanisms between these therapeutic classes may result in improved disease control across both tissue compartments. As one of the first reports from a Latin American cohort, this study contributes with evidence to an area where randomised trial data remain absent, and underscores the need for prospective studies to define optimal drug class pairings, sequencing and long-term safety.

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Abstract N°: 4247

Secukinumab in hidradenitis suppurativa in clinical practice: predictors of response and minimal disease activity control at week 52 in a multicentre cohort

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Introduction

Background: Secukinumab is approved for moderate-to-severe hidradenitis suppurativa (HS), but real-world predictors of deep response and stringent low-disease-burden states remain poorly characterised, particularly in cohorts including surgery.

Objectives: To evaluate secukinumab effectiveness in routine HS care, identify predictors of response, and explore a pragmatic modified minimal disease activity (MDA) endpoint based on registry variables.

Materials and Methods

Retrospective multicentre cohort of 357 adults with HS treated with secukinumab. Outcomes were IHS4-55, IHS4-75 and IHS4-90 at weeks 16, 24 and 52. Modified MDA was pragmatically adapted to registry variables and defined as simultaneous fulfilment of IHS4 ≤2, pain VAS ≤2 or absence of flares, and no structural progression versus baseline. Multivariable logistic regression models were built for key week-24 and week-52 outcomes.

Results

Mean follow-up was 21.35 ± 12.41 months. IHS4-55 was achieved by 43.3% (114/263), 60.9% (151/248) and 71.0% (110/155) at weeks 16, 24 and 52; IHS4-75 by 19.4% (51/263), 35.1% (87/248) and 47.1% (73/155); and IHS4-90 by 9.9% (26/263), 17.7% (44/248) and 23.2% (36/155), respectively. Combined secukinumab plus surgery predicted week-24 IHS4-55 (OR 2.49, 95% CI 1.35–4.60; p=0.0035). Lower baseline IHS4 (OR 0.91, 95% CI 0.87–0.96; p=0.0008) and no previous biologic exposure (OR 0.31, 95% CI 0.12–0.77; p=0.0121) predicted week-24 IHS4-90. At week 52, 57/156 (36.5%) achieved IHS4 <4 and 25/156 (16.0%) IHS4 =0. Modified MDA was achieved by 19/117 evaluable patients (16.2%; 5.3% of the whole cohort). Component rates were 49/156 (31.4%) for IHS4 ≤2, 55/127 (43.3%) for pain VAS ≤2 or absence of flares, and 148/155 (95.5%) for no structural progression. Lower baseline IHS4 was the only stable independent predictor of modified MDA at week 52 (OR 0.87, 95% CI 0.79–0.95; p=0.0016).

Conclusions

In real-world HS practice, secukinumab achieved progressively deeper responses over 52 weeks. Lower baseline inflammatory burden was the most consistent predictor of favourable outcomes across conventional response thresholds and stringent disease-control endpoints. Modified MDA at week 52 was achievable in a clinically relevant subset of evaluable patients, supporting the feasibility of pragmatic composite targets in HS real-world studies. These findings support early intervention and the window of opportunity concept in HS, as lower baseline IHS4 was associated with a higher likelihood of achieving MDA-like status.

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Cost-utility of pre-dupilumab infectious disease screening: A retrospective cohort analysis

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Introduction

Pre-treatment infectious disease screening prior to dupilumab initiation is performed inconsistently across medical specialties in Australia. We present a retrospective cohort study to evaluate the yield, clinical impact, and cost-utility of pre-dupilumab screening, comparing dermatology-led and non-dermatology-led patient cohorts.

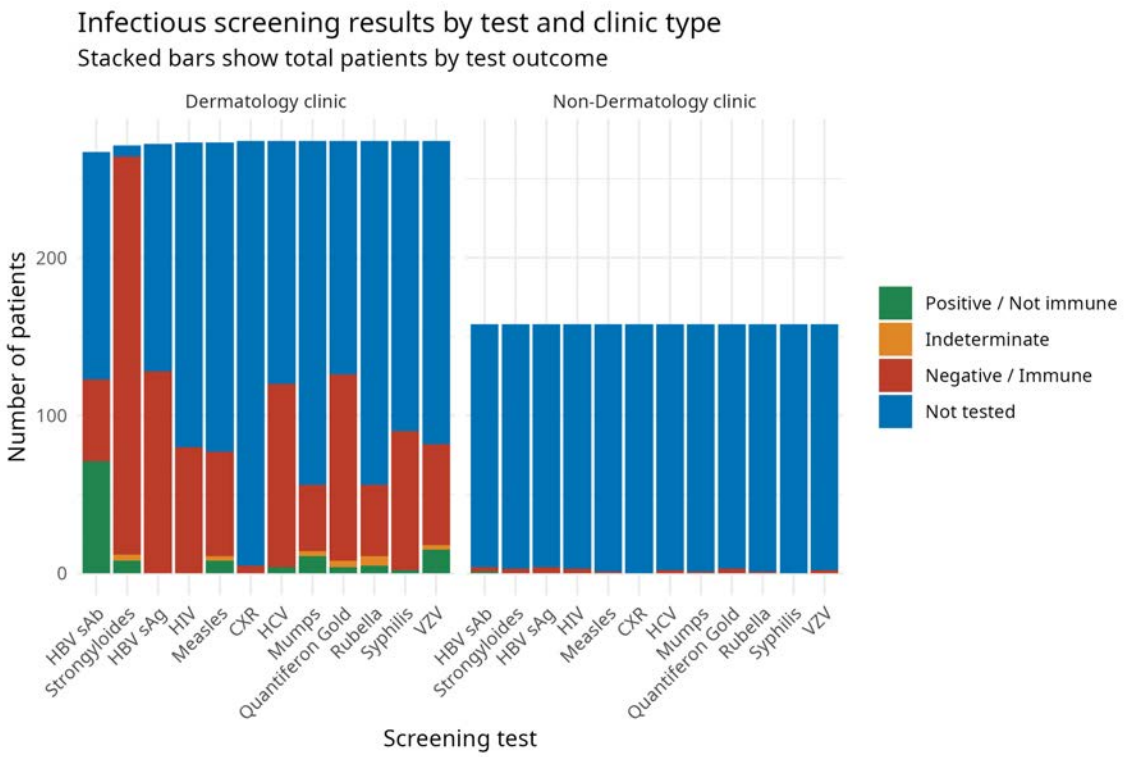
Materials and Methods

Retrospective data were collected from patients commencing dupilumab at Monash Health between 2014 to September 2025. Screening uptake, actionable management change (defined as antimicrobial treatment or vaccination updates), treatment delay, infectious complications, and costs were examined across dermatology and non-dermatology cohorts (respiratory, allergy and immunology). Screening performed for unrelated clinical indications was excluded.

Costs were derived from institutional finance data. All cost figures quoted are in Australian dollars (AUD). Comparisons were made using χ^2 or Fisher's exact tests for categorical outcomes and non-parametric tests for cost distributions. Cost-utility was expressed as cost per actionable management change, calculated as total screening cost divided by the number of actionable outcomes.

Results

Screening was performed in 271 of 274 (98.9%) dermatology patients, and 7 of 158 (4.4%) non-dermatology patients ($\chi^2 = 390$, $p < 0.01$). Actionable findings were infrequent, and occurred in 5.8% of dermatology patients, and in none of the non-dermatology patients ($p < 0.01$). The most common actionable outcome was positive Strongyloides serology driving ivermectin treatment. No cases of disseminated infection were identified during follow-up. Mean screening cost-per-patient was $71.80 AUD (median $56.50; inter-quartile range (IQR) $104) in dermatology and $2.35 (median $0; IQR $0) in non-dermatology groups. The cost per actionable outcome was $1,055.57 AUD among all patients who had at least one screening test, and $609 for patients limited only to Strongyloides testing.



Conclusions

Pre-dupilumab infectious disease screening yields low absolute rates of actionable findings, with substantial variation in cost-utility across screening strategy and clinical settings. These data suggest limited cost-effectiveness for universal screening, supporting a shift toward risk-stratified and indication-specific screening strategies to optimise resource use.

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Abstract N°: 4323

Rapid reduction in neutrophils and neutrophil percentage-to-albumin ratio upon treatment with imsidolimab in patients with GPP

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Introduction

Generalized pustular psoriasis (GPP) is a rare, severe disease characterized by debilitating flares of non-infectious pustular and erythematous skin lesions, with systemic impacts that can be life-threatening. Recurrent flares are common. The pathogenesis of GPP can be mainly attributed to excessive activity of IL-36 pathway. Mutations in the gene encoding the IL-36Ra (*IL36RN*) result in uncontrolled activation of the IL-36 pathway associated with the development of GPP, a rare and life-threatening disease. This study aimed evaluate the neutrophil percentage-to-albumin ratio (NPAR) which is an emerging inflammatory marker that reflects immune activation

Materials and Methods

In a randomized, double-blind, placebo-controlled study (Study GEMINI-1), single intravenous doses of 300 mg of imsidolimab were given to GPP patients for four weeks. Following treatment with imsidolimab, NPAR was assessed at baseline and throughout the duration of treatment in GEMINI1 clinical study.

Samples were collected from GPP patients from all over the world providing a unique opportunity to study this phenotype globally. Additional ELISA on IL36RN levels was conducted to confirm downstream effects of the identified variants. GPP Physician Global Assessment (GPPPGA) score of clear/almost clear (0/1) collectively across all GPP disease attributes (pustulation, erythema, scaling), a stringent and comprehensive characterization of disease severity was conducted to characterize this cohort of patients.

Results

Following treatment with imsidolimab in patients with GPP, significantly reduced levels of neutrophils were observed in serum on Week 4 ($p < 0.001$), and were associated with a decrease in clinical severity as assessed by a decrease in GPPPGA scores.

The NPAR is an emerging inflammatory marker that reflects immune activation . Following treatment of a single IV dose of imsidolimab on Day 1 of GEMINI-1, significantly reduced levels of NPAR were observed in serum at Weeks

1-4 ($p < 0.05$ at Week 1, $p < 0.001$ at Weeks 2 and 4) compared to baseline, and were associated with a decrease in clinical severity as assessed by a decrease in GPPPGA scores

The initial effect was observed already by week 1 of treatment, upon evaluation.

Conclusions

Consistent with the inhibition of IL-36 signaling, imsidolimab treatment decreased certain biomarkers of inflammation. A significant improvement in patients with *IL36RN* mutations following treatment with monoclonal antibody that binds the interleukin-36 receptor and antagonizes interleukin-36 signaling with 66.7% of mutant carriers achieving a GPPPGA 0 or 1 score at Week 4 vs 27.3% of non-mutant *IL36RN* carriers. This effect was consistent with reduction of neutrophils. Targeting IL-36 signaling with represents a promising therapeutic option for GPP patients, and other rare skin conditions that lead to aberrant IL36 signalling.

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Abstract N°: 4546

Safety and Efficacy of Tofacitinib in Psoriasis and Psoriatic Arthritis: A Systematic Review and Meta-Analysis of Randomized Controlled Trials

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Introduction
Tofacitinib is an oral Janus kinase inhibitor (JAKi) that modulates cytokine signalling pathways and is primarily indicated for rheumatological and inflammatory conditions including rheumatoid arthritis, psoriatic arthritis, and ulcerative colitis. As its clinical applications expand into dermatological diseases, there is a growing need for high-quality synthesis of efficacy and safety data across these conditions. This systematic review and meta-analysis aimed to evaluate the efficacy and safety of tofacitinib in dermatological diseases using randomized controlled trial (RCT) data.

Materials and Methods
This review followed PRISMA 2020 reporting guidelines and was registered on PROSPERO (CRD420261365094). MEDLINE, Embase, and Web of Science were searched from inception to January 2026 using terms for tofacitinib and synonyms combined with terms related to RCTs. Only studies involving patients with dermatologic conditions were included. Random-effects meta-analysis was conducted where feasible to compare 5 mg twice daily (BID), 10 mg BID, and placebo for efficacy and safety outcomes.

Results
A total of 5,729 unique records were screened, of which 11 RCTs comprising 4,193 participants met inclusion criteria. The included studies were predominantly of high methodological quality (72.7 %), and some with moderate risk of bias (27.3 %) based on the Joanna Briggs Institute risk of bias measure. Due to heterogeneity in outcome reporting across studies, meta-analysis was only feasible for psoriasis trials (**Figure 1)**. Compared with placebo, both 5 mg BID and 10 mg BID significantly improved Physician Global Assessment (PGA) 0/1 (RR 3.64 and 5.03, respectively) and PASI75 (RR 4.00 and 6.19, respectively; all $P<0.00001$). The risk of any adverse events (AE) was higher with 10 mg BID (RR 1.21; $P<0.0001$) but not with 5 mg BID. Serious adverse events (SAEs) did not differ significantly at either dose. Discontinuation due to AE was lower with 5 mg BID (RR 0.53; $P = 0.02$) than in the placebo arm, with no significant difference observed for 10 mg BID. Among commonly reported (≥2% or ≥5%) and special AE, upper respiratory tract infection, headache, hypercholesterolemia, and dizziness were significantly more frequent with tofacitinib compared to placebo ($P < 0.05$). Studies involving other dermatologic conditions, including systemic lupus erythematosus and systemic sclerosis, were synthesized narratively due to inconsistent outcome measures and reporting. Limitations included small total study number and short duration of exposure and AE reporting potentially underpowering rare SAEs (boxed warnings) that have been observed within this class.

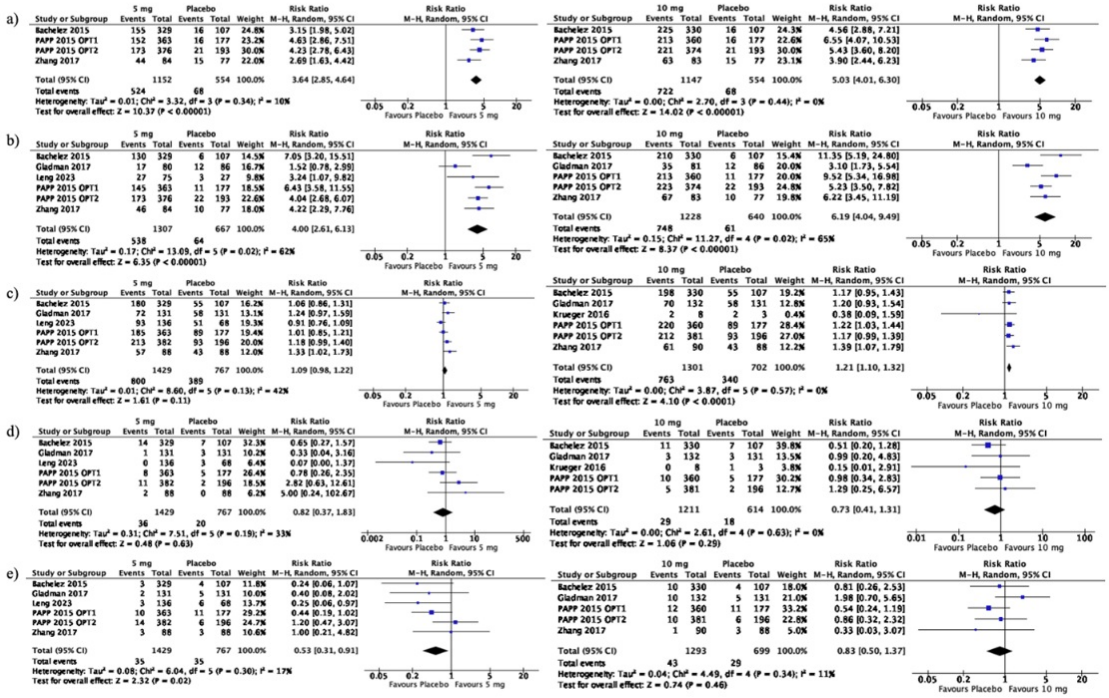

Figure 1. Forest plots of efficacy and safety outcomes for tofacitinib (5 mg and 10 mg, twice daily) compared with placebo in randomized controlled trials evaluating efficacy and safety of tofacitinib in psoriasis. Outcomes included (a) Physician Global Assessment (PGA) 0/1, (b) Psoriasis Area and Severity Index 75 (PASI75), (c) any adverse events, (d) serious adverse events, and (e) discontinuation due to adverse events. Effect estimates are presented as risk ratios (RRs) with 95% confidence intervals.

Conclusions

Overall, Tofacitinib demonstrates high efficacy in psoriasis with a generally acceptable safety profile based on randomized controlled trial evidence. Non-psoriasis dermatologic conditions remain underpowered for broader synthesis, highlighting the need for high-quality trials in non-psoriasis indications with standardized outcome reporting.

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Abstract N°: 4567

Comparative Single-Cell Analysis of Melanoma Brain Metastases & Glioblastoma – Identifying MBM-Associated Transcriptional Programmes and Therapeutic Targets

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Introduction

Melanoma is a highly lethal form of skin cancer and has one of the highest rates of brain metastases among primary malignancies. Similarly, glioblastoma is a malignant, deadly brain tumor with a survival rate of only 5% after 5 years. Melanoma brain metastases (MBM) and glioblastoma (GBM) are aggressive intracranial tumours with distinct cellular origins, yet their transcriptional landscapes at single-nucleus resolution remain incompletely compared.

Materials and Methods

Herein, snRNA-seq data was integrated from 27 MBM patients (GSE200218) and 121 GBM samples from 59 patients (GSE274546), 835,182 nuclei in total, to identify disease-specific programs and druggable vulnerabilities. After uniform quality control, all shared genes for pseudo-bulk differential expression were retained while employing a union of the top 3000 highly variable genes from each malignancy and Harmony batch correction to generate a batch-corrected embedding. Cells were annotated by module scoring of lineage markers, and pseudo-bulk counts were aggregated per sample and cell type. DESeq2 contrasting MBM versus GBM revealed 17,103 significantly altered genes in malignant cells ($|log2FC| > 0.5$, p-adj < 0.05), 8629 in macrophages, and 5209 in T-cells.

Results

As expected, malignant MBM cells were characterized by strong upregulation of melanocyte-associated transcripts (e.g., TYR, MITF, SOX10) and downregulation of glioma-defining genes such as EGFR, PDGFRA, and PTPRZ1. Oxidative phosphorylation and MYC targets were the top MBM-enriched pathways in malignant cells. Intersection with a druggable genes list from the Dec 2024 update of the Drug-Gene Interaction Database identified 6194, 3842, and 2746 actionable targets in malignant cells, macrophages, and T-cells, respectively, including EGFR, VEGFA, and KIT. Findings were robust to cohort imbalance, with 72.2% of significant genes replicating when GBM was down-sampled to 27 samples.

Conclusions

These results provide a comparative map of the MBM and GBM transcriptomes and nominate candidate therapeutic targets for pre-clinical validation, though separating tumor-specific vulnerabilities from lineage differences will require a normal-brain reference.

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Abstract N°: 4572

Comparative Efficacy and Therapeutic Selectivity of Bimekizumab, Secukinumab, and Adalimumab in Moderate-to-Severe Plaque Psoriasis: A Systematic Review

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Introduction
Plaque psoriasis is a chronic immune-mediated inflammatory disease driven predominantly by the IL-23/IL-17 signaling axis. The introduction of biologic therapies has substantially transformed disease management, beginning with tumor necrosis factor-alpha (TNF-α) inhibitors such as adalimumab, followed by selective IL-17A inhibitors including secukinumab. More recently, bimekizumab, a dual inhibitor of IL-17A and IL-17F, has emerged as a promising therapeutic option with high rates of complete skin clearance.

Materials and Methods
A systematic review was conducted using PubMed and major peer-reviewed medical journals to identify phase 3 randomized clinical trials and network meta-analyses published between 2021 and 2024. Studies comparing bimekizumab with secukinumab or adalimumab in moderate-to-severe plaque psoriasis were included. Primary outcomes analyzed were PASI 90 and PASI 100 responses at week 16, in addition to safety outcomes.

Conclusions
The available evidence demonstrated consistent superiority of bimekizumab in achieving high levels of skin clearance. In comparative studies against adalimumab, bimekizumab achieved markedly higher PASI 90 response rates, indicating enhanced control of inflammatory activity. In head-to-head comparisons with secukinumab, bimekizumab demonstrated superior PASI 100 rates, supporting the hypothesis that simultaneous inhibition of IL-17A and IL-17F may provide broader suppression of psoriatic inflammatory pathways.

The dual blockade strategy appears particularly effective in reducing residual inflammatory signaling that may persist with selective IL-17A inhibition alone. However, increased rates of mucocutaneous candidiasis were observed with bimekizumab therapy, likely related to the physiological role of IL-17 pathways in mucosal immune defense. Most infections were mild to moderate and manageable without treatment discontinuation.

Current evidence suggests that bimekizumab represents one of the most potent biologic therapies currently available for moderate-to-severe plaque psoriasis, with superior rates of complete skin clearance compared with both TNF-α and selective IL-17A inhibition. Although the safety profile remains favorable overall, careful monitoring for superficial fungal infections is recommended during treatment.

These findings reinforce the therapeutic relevance of dual IL-17A/IL-17F blockade and support bimekizumab as an important advancement in precision treatment strategies for severe psoriasis.