Abstract N°: ID-5

Topic: Biologics, immunotherapy, targeted therapy

Oral monotherapy with JAK inhibitors in Bolivian patients with refractory vitiligo

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Introduction
Vitiligo is one of the most common autoimmune diseases seen in dermatology clinics. It is caused by the destruction of melanocytes and clinically manifests as a loss of skin pigmentation, which has a significant psychosocial impact on the patient. Given the often unsatisfactory results of conventional therapies, we undertook a study with JAK inhibitor drugs, which yielded excellent results with fewer adverse effects, better tolerance, and improved treatment adherence.

Materials and Methods
A prospective study was conducted on 20 patients with refractory vitiligo at our private clinic in La Paz, Bolivia, between December 2024 and October 2025. Patients who had undergone multiple treatments with steroid creams, calcineurin inhibitors, oral vitamins, meladinin, and laser therapy without improvement were included. After a detailed analysis of each patient, an innovative therapy was proposed using oral JAK inhibitors: oral tofacinib 11 mg (10 patients) and baricitinib 2 mg (10 patients), administered once daily for 9 months.

Results
An evaluation was conducted that considered the VASI score and clinical parameters over 9 months with quarterly follow-up periods. Twenty patients were included; 10 received tofacitinib and the other 10 received baricitinib. The VASI score decreased from 16.37 ± 14.33 (tofacitinib) and 10.16 ± 9.78 (baricitinib) at month 1. The improvement at month 3 was 25.28 ± 18.55 (tofacitinib) and 25.29 ± 11.77 (baricitinib). The improvement at month 6 reached 34.72 ± 6.42 (tofacitinib) and 35.15 ± 12.16 (baricitinib), and the final progress was 43.82 ± 8.42 (tofacitinib) and 46.15 ± 18.12 (baricitinib) at month 9. Regarding clinical parameters, the trichrome and confetti signs were observed and gradually improved. No adverse effects were reported.

Conclusions
We report the first study conducted in Bolivian patients with vitiligo refractory to multiple treatments, who opted for a new, effective therapy with good tolerability and a broad safety profile. However, while the results and the use of JAK inhibitors show promise, further clinical studies are needed

Abstract N°: ID-18

Topic: Biologics, immunotherapy, targeted therapy

Efficacy and safety of tofacitinib in treatment of moderate to severe alopecia areata

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Introduction
Alopecia Areata (AA) is a common immune-mediated condition that presents as non scarring patches of hair loss with a wide range of severity and tremendous impacyon patient's quality of life. Targete immunomodulators, like JAK inhibitors, have emerged as novel promising therapeutic options with favorable safety profile.

Materials and Methods
An open label prospective interventional study.
Patients with moderate to severe alopecia areata (defined as a SALT score above 20) above 2 years of age were treated with tofacitinib. The starting dose in adults was 5 mg BID, while in paediatric patients, a weight adjusted dose was used (2.5 mg if body weight <20kg, 5mg if body weight 20-40 kg and 10 mg if body weight >40kg).

patients were assessed at regular intervals with consideration for dose increments if insufficient response was observed.

Efficacy was evaluated using SALT score
Sacety was assessed by patient's symptoms and laboratory evaluation
A 50% reduction in baseline SALT score was considered the primary endpoint of the study

Results
55 patients were enrolled in the study. 45% had alopecia universalis.

Mean SALT score dropped from 74.2 to 29.4 at the end of the study (week36)
72.7% of patients achieved 50% or more reduction in SALT score
41.8% of patients achieved 90% or more reduction in SALT score
17.5% of patients required dose escalation to achieve the desired response
Female sex, shorter disease duration and lower baseline SALT score were predictors of a favorable response
Acneform eruptions and upprr respiratory tract infections were the most commonly reported adverse effects
Elevated liver enzymes qnd leukopenia requiring treatment interruption were recorded in 2 patients

Conclusions
Tofacitinib is an effective and relatively safe treatment for moderate to severe alopecia areata with disease duration and baseline SALT score being significant predictors of response

Abstract N°: ID-72

Topic: Biologics, immunotherapy, targeted therapy

Immunotherapy in Melanoma: A Dynamic Frontier in Cancer Treatment

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Introduction

Melanoma remains one of the most aggressive and treatment-resistant forms of skin cancer. Recent advances in immuno-oncology and molecular biology have revolutionized its management, putting immunotherapy at the top of novel therapeutic approaches. This study aims to explore the evolving role of immunotherapy in melanoma, with a focus on molecular targets, mechanisms of action, and the integration of precision medicine into clinical practice.

Materials and Methods

A comprehensive literature review was conducted across multiple databases up to 2025. The search focuses on immunotherapy treatments in Melanoma, *Immune checkpoint inhibitors, Oncolytic Viruses, mRNA Vaccines,* Resistance Mechanisms to Immunotherapy in Melanoma (*Lack of T-cell Infiltration, Impaired Antigen Presentation, Role of BRAF and MEK inhibition to overcome resistance to immunotherapy).*

Results

Highly focused treatments have been developed as a result of significant genetic mutations, such as those in BRAF, NRAS, and KIT, as well as changes in signaling pathways, such as MAPK and PI3K/AKT/mTOR. Immune checkpoint inhibitors have become a mainstay treatment because they provide sustained therapeutic implications and greatly increasepatient survival. Additionally, novel approaches like mRNA-based vaccinations and oncolytic viruses are expanding the range of therapeutic options. These advances have made it possible to move toward individualized treatment plans, which have improved long-term results and decreased toxicity. An important advancement in oncology, the incorporation of molecular profiling into routine melanoma care made immunotherapy a viable and sustainable cancer treatment option.

Table 1: Novel strategies to overcome resistance to immunotherapy in melanoma					
Target	Mechanism of resistance	Study population	Intervention	Outcomes of inhibtion	Reference
LAG-3	T-cell exhaution via negatively regulating mitochondrial activity in CD4+ T cells Enhance the function of Tregs cells (115).	Melanoma patients who had PD after anti–PD-1/PD-L1 (± anti–CTLA-4 or BRAF/MEK inhibitors).	BMS-986016 (LAG-3 inhibitor) + nivolumab	ORR: 16%, DCR: 45%	(116)
TIM-3	Tregs which express TIM-3 have more potent immunosuppressive effects. Suppress intracellular TLR-induced activation in dendritic cells Promoting suppressive macrophage phenotype (117)	Patients with melanoma stage III	randomized (1:1) to neoadjuvant dostarlimab (arm A) or dostarlimab + cobolimab (anti-TIM-3) (arm B) for 2 cycles prior to surgery.	MPR rate was 33.3% (Arm A) and 51.9% (Arm B) (p = 0.0029). 1-year EFS rate Arm B (92%) vs Arm A (82%).	(118)
TIGT	Prevent T cell activation, modest inhibition of CD4+ T cells priming and NK cell killing (119, 120)	Patients with high-risk melanoma stage III	Arm A: vemurafenib, cobimetinib, and atezolizumab Arm B: cobimetinib, atezolizumab. Arm C: atezolizumab, tiragolumab	--------	NCT03554083
LAG-3: lymphocyte activation gene-3, CD4: cluster of differentiation 4, PD: programmed cell death protein 1, PD-L1: programmed death-ligand 1, CTLA-4: cytotoxic t-lymphocyte–associated antigen 4, ORR: objective response rate, DCR: disease control rate, TIM-3: t-cell immunoglobulin and mucin-domain containing-3, TLR: toll-like receptor, MPR: major pathologic response, EFS: event-free survival, TIGIT: t-cell immunoreceptor with Ig and ITIM domains, NK: natural killer.					

Table 1: Novel strategies to overcome resistance to immunotherapy in melanoma

Conclusions

In conclusion, melanoma's genetic landscape has greatly improved, allowing precision medicine to replace traditional methods. Targeted therapy has been promising since mutations like BRAF, NRAS, and KIT have been found, as well as changes in important signaling pathways, including MAPK and PI3K/AKT/mTOR. Novel approaches, including oncolytic viruses and mRNA vaccines, as well as immunotherapy, particularly immune checkpoint inhibitors, have increased treatment options and enhanced long-term results concurrently. These developments have made it possible for physicians to customize treatments to each patient's unique tumor profile, which has increased survival rates, decreased toxicity, and produced more robust responses. As precision medicine has been successful in improving patient outcomes and establishing a new benchmark for melanoma therapy, the incorporation of molecular diagnostics into routine melanoma care represents a significant turning point in oncology.

Abstract N°: ID-107

Topic: Biologics, immunotherapy, targeted therapy

Efficacy and Safety of Secukinumab in the Treatment of Elderly Patients with Psoriasis and Comorbid Chronic Metabolic Diseases or Cardiac Dysfunction

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Introduction

Psoriasis is a common systemic inflammatory condition that is frequently associated with chronic cardiometabolic conditions, such as diabetes, metabolic syndrome, and chronic heart failure, especially in the elderly. The safety and effectiveness of interleukin-17A inhibitor Secukinumab in this patient population remains largely undefined. This study evaluated the real-world efficacy and safety of secukinumab in this patient population.

Materials and Methods

A retrospective study was performed to include 58 elderly patients (≥ 65 years) with moderate-to-severe psoriasis and significant cardiometabolic comorbidities treated during October 2021 and May 2024. Cardiometabolic conditions included type 2 diabetes, gout, hypertension, and chronic heart failure. Another 67 age- and sex-matched elderly patients without cardiometabolic conditions served as the control group. Primary endpoints were PASI 75 response at week 16, 1-year Secukinumab survival rate, and the incidence of adverse events.

Results

Secukinumab demonstrated high efficacy in both groups, with 84.5% and 88.1% of patients in the study group and control group achieving PASI 75, respectively, at week 16 (P = 0.56). The 1-year survival rate for Secukinumab was also comparable in the study group as compared to the control group (47/58 vs 56/67, P = 0.71). No serious adverse events occurred in both groups. Adjusting for baseline characteristics, the study group showed similar risk of secukinumab discontinuation (OR=1.06, 95% confidence interval 0.92-1.17).

Conclusions

Secukinumab showed robust efficacy and safety profiles in elderly psoriasis patients with chronic metabolic diseases or cardiac dysfunction, supporting its use as a suitable treatment option in this vulnerable group.

Abstract N°: ID-116

Topic: Biologics, immunotherapy, targeted therapy

Effects of Biologic Agents on Nailfold Dermoscopic Features in Patients with Psoriasis: A Three-Year Observational Study

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Introduction

Psoriasis is a chronic inflammatory dermatosis characterized by microvascular changes associated with systemic comorbidities **(1,2)**. Nailfold dermoscopy has been used for psoriasis treatment follow-up and for the diagnosis of comorbidities such as psoriatic arthritis **(3,4)**. In our study, we used nailfold dermoscopy to evaluate the effects of biological agent treatment on microvascular changes in psoriasis.

Materials and Methods

Thirty-five patients scheduled to start treatment with biological agents for psoriasis were studied. Nailfold capillary loops were observed and recorded using dermoscopy in 2022. Nailfold dermoscopic findings of 13 patients who continued their regular biological treatment were re-evaluated and recorded in January 2025. 21 psoriasis patients with a PASI score <10 who did not receive systemic treatment to date were included in the study as a control group. The findings of the patients were compared pre- biological after three years and between the pre-biological and control groups.

Results

The number of capillary ectasia findings on nailfold dermoscopy was significantly higher in the pre-biological group than in the control group. ($p<0.05$). No significant difference was found in the capilloscopic findings between the pre- and post-biological groups. However; in three patients who initially had capillary ectasia and neovascularization, these capillary findings regressed after three years.

		Pre-biologic	Control	Post-biologic
Mean Age		44.49	37.29	
Female/Male		19/16	8/13	
Mean PASI score		15.6	4.2	
Psoriatic arthritis		13	-	
Treatment	Anti-TNF	12	-	
	Anti IL-17	9	-	
	Anti IL12/23	4	-	
	Anti IL-23	10	-	
Dermoscopic findings	Capillary ectasia	21	4*	8
	Microhemorrhage	7	-	4
	Decrease in capillary density	18	9	7
	Anormale morphology	14	6	8
	Avascular area	3	4	-
***p>0,05; TNF, tumour necrosis factor; IL, interleukin**				

Table 1. Comparison of demographic characteristics, clinical features, treatments, and nailfold dermoscopic findings between pre-biologic, post-biologic, and control groups.

Conclusions

Nailfold dermoscopy is a rapid, simple, and noninvasive technique that can be effectively used to evaluate microvascular alterations in patients with psoriasis. Our findings suggest that nailfold dermoscopic assessment may provide valuable insights into systemic microvascular involvement, particularly in patients requiring biological therapy. Moreover, nailfold dermoscopy may serve as a useful tool for monitoring the effects of biological treatments—especially anti-TNF agents—on microvascular abnormalities and related comorbidities in psoriasis **(5)**. Larger prospective studies are warranted to further clarify its role as an adjunctive biomarker in the assessment of treatment response and cardiovascular risk modulation.

Abstract N°: ID-231

Topic: Biologics, immunotherapy, targeted therapy

Immune-induced scarring alopecia: About two cases

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Introduction

Over the last decade, immunotherapy has revolutionized the management of advanced cancers. However, the anti-tumor response triggered by the immune system is often accompanied by immune-mediated adverse events affecting various organs. Cutaneous manifestations are among the most common, occurring in approximately 40% of patients undergoing treatment. These include pruritus, xerosis, and maculopapular eruptions. Immune-induced alopecia has occasionally been reported, with alopecia areata being the most frequently described form, whereas scarring alopecia remains rarely documented.

We report two cases of scarring alopecia occurring in patients treated with Nivolumab for metastatic melanoma.

Materials and Methods

N/A

Results

Observation n°1: A 53-year-old woman was treated with Ipilimumab-Nivolumab for stage IV melanoma. A significant regression of secondary lesions was observed following the initial treatment, which was subsequently continued with nivolumab monotherapy. After two years, she developed pruritic plaques of the scalp, associated with hair loss. Clinical examination revealed a diffuse reduction in hair density, with erythematous and squamous plaques on the vertex. Dermoscopy showed loss of follicular openings, perifollicular scaling and erythema, and hair casts. The rest of the examination revealed keratotic papules and dystrophic hair on the limbs, and pubic and axillary hair loss. The diagnosis of lichen planopilaris, in the context of Graham-Little-Lasseur syndrome, was considered. Histology showed perifollicular lymphocytic infiltrate and interface dermatitis. Clonality testing ruled out folliculotropic mycosis fungoides. Treatment with doxycycline 200 mg/day and very potent topical corticosteroids was initiated. After three months, the lesions on the limbs showed significant improvement. However, the alopecic plaques on the scalp continued to progress, presenting as ivory-white areas without follicular openings, with persistent dermoscopic signs of activity at the periphery of the plaques.

Observation n°2: A 66-year-old woman was treated with two administrations of Ipilimumab Nivolumab for stage IV melanoma, followed by a temporary discontinuation of treatment due to a favorable oncological response and the occurrence of grade III immune-mediated hepatitis. Six months later, disease progression was observed, and Nivolumab monotherapy was resumed. After 18 months of monotherapy, she developed a pruritic alopecic plaque on the frontal scalp. Examination revealed a unique, well demarcated, atrophic, erythematous, keratotic plaque of the frontal scalp. Histopathological examination showed a deep perifollicular lymphocytic infiltrate with a few keratotic plugs. Direct immunofluorescence and systemic evaluation were negative. The diagnosis of cutaneous discoid lupus erythematosus was retained. Treatment with potent topical corticosteroids was initiated. The patient declined treatment with synthetic antimalarial agents. After three months, pruritus had resolved. Clinical examination showed a slight reduction in erythema and hyperkeratosis, with no extension of alopecia.

Conclusions

The hair follicle is an immune-privileged site characterized by reduced expression of Major Histocompatibility Complex I molecules. Typically, the immune system is in a state of immune-modulation, preventing inflammatory or autoimmune responses to the autoantigens of the hair follicle. This function is dependent on regulatory T cells. Pharmacological inhibition of PD-1 and CTLA-4 can lead to the collapse of this immune privilege, resulting in follicular toxicity and related skin manifestations. Characterizing immune-induced alopecia, particularly distinguishing whether it is scarring or non-scarring, is crucial for informing patients about the potential psychological impact of hair loss in an oncological context. Furthermore, the possible association between therapeutic efficacy and follicular toxicity warrants further investigation.

Abstract N°: ID-306

Topic: Biologics, immunotherapy, targeted therapy

Descriptive effectiveness of b/tsDMARDs, including ixekizumab, at 12 months for patients with psoriatic arthritis in the German cohort of the PRO-SPIRIT observational study

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Introduction

PRO-SPIRIT is a multinational, 2-year observational study investigating real-world effectiveness and persistence of biologic/targeted synthetic disease-modifying antirheumatic drugs (b/tsDMARDs), including ixekizumab (IXE), in the treatment of psoriatic arthritis (PsA).

Objectives: To describe baseline (BL) demographic/disease characteristics and the effectiveness of IXE and other b/tsDMARDs at 12 months (M) for patients (pts) with PsA from the PRO-SPIRIT German cohort.

Materials and Methods

Pts with PsA who initiated or switched to a new interleukin-17A inhibitor (IL-17Ai) (IXE or secukinumab [SEC]), tumour necrosis factor inhibitor (TNFi), IL-12/23i, IL-23i, Janus kinase inhibitor (JAKi), or phosphodiesterase-4 inhibitor (PDE4i) during December 2019-June 2022 were evaluated across Germany, Italy, Spain, France, UK, and Canada. Pt characteristics and clinical/pt-reported outcomes were collected at BL and 12M, including swollen-joint count, tender-joint count, change from baseline (CFB) in Clinical Disease Activity Index for PsA (cDAPSA) and PsA-affected body surface area (BSA), and proportion of pts achieving BSA <3%, cDAPSA remission (REM), and minimal disease activity (MDA). This post hoc analysis presents descriptive data for the German on-label population. Mixed models for repeated measures assessed CFB. Missing data were handled via multiple imputation.

Results

Of 1134 PRO-SPIRIT pts, 270 (23.8%) were enrolled in Germany.* Based on descriptive data only, pts taking IXE had longer disease duration (8.7 vs 6.9 years), more previous b/tsDMARD failures (69.8% vs 38.3%), more monotherapy use (72.9% vs 63.3%) and higher BSA ≥3% involvement (51.0% vs 46.7%) at BL than those taking TNFi (Table 1). At 12M, some IXE joint effectiveness measures were higher (unadjusted CFB in cDAPSA, –17.3 vs –11.5), more pts achieved cDAPSA REM (38.9% vs 27.0%) and MDA (46.9% vs 36.8%), and skin improvement was higher (BSA shift to <3%, 31.3% vs 25.0%) than what was observed with TNFi. At BL, pts with IXE had higher skin involvement (BSA ≥3%, 51.0% vs 42.5%) than pts with SEC. At 12M, IXE joint effectiveness was similar (unadjusted CFB in cDAPSA, –17.3 vs –16.8), more pts achieved cDAPSA REM/MDA (cDAPSA REM, 38.9% vs 11.5%; MDA, 46.9% vs 33.7%), and skin improvement was higher (BSA shift to <3%, 31.3% vs 25.0%) than what was observed with SEC. BL characteristics of pts taking IXE, IL-12/23i and IL-23i were similar, except for lower mean BSA (IXE 5.9% vs IL-12/23i 10.5% vs IL-23i 10.3%) and lower proportions of BSA ≥3% (IXE 51.0% vs IL-12/23i 66.7% vs IL-23i 71.4%) for IXE. At 12M, IXE joint effectiveness was similar (unadjusted CFB in cDAPSA, –

17 0 .3 vs –17.7), more pts achieved cDAPSA REM/MDA (cDAPSA REM, 38.9% vs 15.4%; MDA 46.9% vs 29.2%), and skin outcomes were similar (BSA shift to <3%, 31.3% vs 33.3%) to those observed with IL-23i. BL characteristics of pts taking IXE and JAKi were similar, except more pts taking IXE used monotherapy (72.9% vs 56.8%), had b/tsDMARD experience (69.8% vs 56.8%), or had BSA ≥3% (51.0% vs 34.1%). At 12M, IXE joint effectiveness was higher (unadjusted CFB in cDAPSA, –17.3 vs –12.9) and more pts achieved cDAPSA REM with IXE (38.9% vs 14.3%) while achievement of MDA (46.9% vs 44.2%) and skin outcomes (BSA shift to <3%, 31.3% vs 31.8%) were similar to what was observed with JAKi (Table 1).

*PDE4i group is not presented due to low pt numbers (32/1134 overall; 3/270 German pts).

Table 1: Baseline characteristics and effectiveness of IXE and other b/tsDMARDs in the German PRO-SPIRIT cohort at 12 months

Baseline	IXE	SEC total[a]	TNFi	IL-12/23i	IL-23i	JAKi	OVERALL[b]
n (%)	96 (35.6)	40 (14.8)	60 (22.2)	6 (2.2)	21 (7.8)	44 (16.3)	270 (100.0)
Age, years, mean (SD)	55.5 (12.1)	52.4 (11.1)	48.9 (13.9)	53.2 (14.8)	50.9 (11.4)	54.5 (12.4)	53.0 (12.5)
Female, n (%)	59 (61.5)	27 (67.5)	39 (65.0)	3 (50.0)	11 (52.4)	29 (65.9)	169 (62.6)
Time since PsA diagnosis, years, mean (SD)	8.7 (8.0)	7.6 (8.0)	6.9 (8.6)	8.8 (8.5)	6.1 (7.0)	7.1 (5.4)	7.6 (7.7)
b/tsDMARD experienced, n (%)	67 (69.8)	30 (75.0)	23 (38.3)	4 (66.7)	16 (76.2)	25 (56.8)	165 (61.1)
No csDMARD use (monotherapy)	70 (72.9)	28 (70.0)	38 (63.3)	6 (100)	15 (71.4)	25 (56.8)	185 (68.5)
SJC (0-66), mean (SD)	5.9 (6.5)	4.3 (4.5)	3.1 (5.3)	1.7 (2.7)	3.4 (3.2)	4.4 (4.9)	4.5 (5.5)
TJC (0-68), mean (SD)	9.5 (7.6)	9.6 (7.2)	5.5 (8.8)	6.0 (8.7)	11.0 (11.1)	8.8 (7.7)	8.5 (8.3)
cDAPSA, mean (SE)	27.5 (1.4)	26.4 (1.5)	19.7 (1.8)	15.8 (5.9)	26.6 (3.4)	24.9 (1.7)	24.8 (0.8)
BSA (%), mean (SD)	5.9 (9.6)	6.1 (13.4)	9.1 (16.6)	10.5 (14.8)	10.3 (11.8)	4.1 (9.5)	6.8 (12.3)
BSA ≥3%, n (%)	49 (51.0)	17 (42.5)	28 (46.7)	4 (66.7)	15 (71.4)	15 (34.1)	131 (48.5)
Effectiveness at 12M							
n (%) on-label	54	26	37	5	13	21	158
MDA[c], n/N[d] (%)	25 (46.9)	8 (33.7)	11 (36.8)	1 (25.0)	4 (29.2)	8 (44.2)	59 (40.1)
cDAPSA REM[c] (score ≤4), n/N[d] (%)	21 (38.9)	3 (11.5)	10 (27.0)	2 (40.0)	2 (15.4)	3 (14.3)	42 (26.6)
Unadjusted CFB in cDAPSA, mean (SE)	-17.3 (1.8)	-16.8 (2.3)	-11.5 (2.3)	-10.2 (4.3)	-17.7 (4.2)	-12.9 (2.8)	-15.2 (1.1)
12M cDAPSA, mean (SE)	9.2 (1.3)	11.1 (1.5)	8.8 (1.2)	8.4 (4.7)	9.9 (2.1)	9.7 (1.8)	9.4 (0.7)
Adjusted (MMRM) CFB in cDAPSA	-15.3 (1.1)	-13.7 (1.5)	-15.3 (1.3)	-15.7 (3.8)	-15.8 (2.2)	-14.5 (1.8)	-15.1 (0.7)
CFB in BSA (%), mean (SE)	-3.4 (1.0)	-3.8 (1.9)	-5.2 (1.4)	-5.5 (2.6)	-5.7 (1.9)	-3.2 (1.5)	-4.1 (0.6)
12M BSA (%), mean (SE)	2.5 (0.5)	2.3 (0.7)	3.9 (1.1)	5.0 (4.4)	4.6 (1.4)	0.9 (0.3)	2.7 (0.4)
Adjusted (MMRM) CFB in BSA, %	-3.9 (0.5)	-4.2 (0.7)	-3.4 (0.6)	-2.5 (2.0)	-2.9 (1.0)	-5.1 (0.7)	-3.9 (0.3)
12M shift from BSA ≥3% to <3%, n (%)	30 (31.3)	10 (25.0)	15 (25.0)	3 (50.0)	7 (33.3)	14 (31.8)	81 (30.0)

All data are presented as mean (SE) unless otherwise specified. [a]26 pts received SEC 150 mg and 14 pts received SEC 300 mg. [b]Includes data for 3 pts in the PDE4i group, which are not presented separately due to low pt numbers. [c]Pts who already met the criteria for MDA or cDAPSA REM at baseline were excluded from this analysis. [d]The percentage is not always equal to n/N due to the use of MI.

Abbreviations: **12M**, 12 months; **BSA**, body surface area; **b/ts DMARDs**, biologic or targeted synthetic disease-modifying anti-rheumatic drugs; **cDAPSA**, clinical Disease Activity index for Psoriatic Arthritis; **CFB**, change from baseline; **csDMARDs**, conventional synthetic disease-modifying anti-rheumatic drugs; **IL-12/23i**, Interleukin 12/23 inhibitors; **IL-23i**, Interleukin 23 inhibitors; IL-17Ai, interleukin 17A inhibitor; **IL-12/23i**, interleukin-12/23 inhibitors; **IXE**, ixekizumab; **JAKi**, Janus kinase inhibitors; **M,** month; **MDA**, minimal disease activity; **MI**, multiple imputation; **MMRM**, mixed models for repeated measures with least squares mean (SE); **PDE4i**, Phosphodiesterase-4 inhibitors; **REM**, remission; **SD**, standard deviation; **SE**, standard error; **SEC total**, the pooled group of pts who initiated secukinumab (an IL-17Ai) 150mg or 300mg; **SJC**, swollen joint count; **TJC**, tender joint count; **TNFi**, tumor necrosis factor inhibitors.

Treatment groups drugs: TNFi (adalimumab, etanercept, infliximab or biosimilar), IL-12/23i (ustekinumab), IL-23i (guselkumab, risankizumab), JAKi (tofacitinib, upadacitinib).

Conclusions

These real-world data suggest that IXE may provide similar joint effectiveness for some related measures but greater skin improvement than TNFi, despite longer disease duration, more prior b/tsDMARDs, and greater skin involvement at BL. IXE may lead to higher rates of cDAPSA REM than SEC, IL-23i and JAKi and similar skin improvement to IL-23i. In general, these descriptive trends in the German cohort align with findings from the overall PRO-SPIRIT population [1,2], supporting well-balanced joint and skin improvements with IXE in PsA.

References:

1. Kristensen LE, Ng KJ, Ngantcha M, et al. Comparative early effectiveness across 14 PsA drugs and 5 classes of PsA treatment: 3-month results from the PRO-SPIRIT study. RMD Open. 2024 Sep 20;10(3):e004318. 2. Marzo-Ortego M, Sewerin P, Selmi C, et al. Comparative effectiveness and persistence of ixekizumab and other b/tsDMARDs in patients with PsA from a real-world setting: 12-month results from PRO-SPIRIT study. Accepted for publication in RMD Open on 13 January 2026.

Abstract N°: ID-325

Topic: Biologics, immunotherapy, targeted therapy

Sequential Treatment With Tofacitinib, Baricitinib, And Upadacitinib In Alopecia Areata: A Real-World Cohort Study

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Introduction

Alopecia areata (AA) is an immune-mediated, non-scarring hair loss disorder where the JAK–STAT pathway plays a central role in pathogenesis. While various JAK inhibitors have demonstrated significant hair regrowth in moderate-to-severe AA, a subset of patients may experience inadequate response or relapse. This study aims to evaluate treatment responses, side effect profiles, and patient satisfaction in patients who sequentially used tofacitinib, baricitinib, and upadacitinib due to various clinical reasons in a real-world setting.

Materials and Methods

This retrospective study included patients with moderate-to-severe AA who were treated sequentially with three different JAK inhibitors (tofacitinib, baricitinib, and upadacitinib). Demographic data, treatment durations, and adverse event records were analyzed. Clinical response was evaluated based on SALT scores using standardized photographs at 3-month visits. Patient satisfaction and quality of life parameters were collected through questionnaires and medical records.

Results

Ten patients (70% male, mean age 32.7) were included, with a mean disease duration of 13.4 years.

- **Tofacitinib:** With a mean duration of 21.5 months, the SALT score decreased from 81.0 to 49.0. Continued shedding was observed in 70% of patients, with a satisfaction score of 4.6.
- **Baricitinib:** With a mean duration of 17.6 months, the SALT score decreased from 64.0 to 17.0. The response rate in the beard/eyebrow area was 40%, and the satisfaction score was 7.1.
- **Upadacitinib:** With a mean duration of 9.3 months, the SALT score decreased from 52.0 to 10.0. A 90% response rate was observed in beard, eyebrow, and eyelash areas. No hair loss was observed during this period, and the satisfaction score was the highest (8.4). Weight gain (20%) was observed in the baricitinib and upadacitinib groups; no adverse events were reported for tofacitinib.

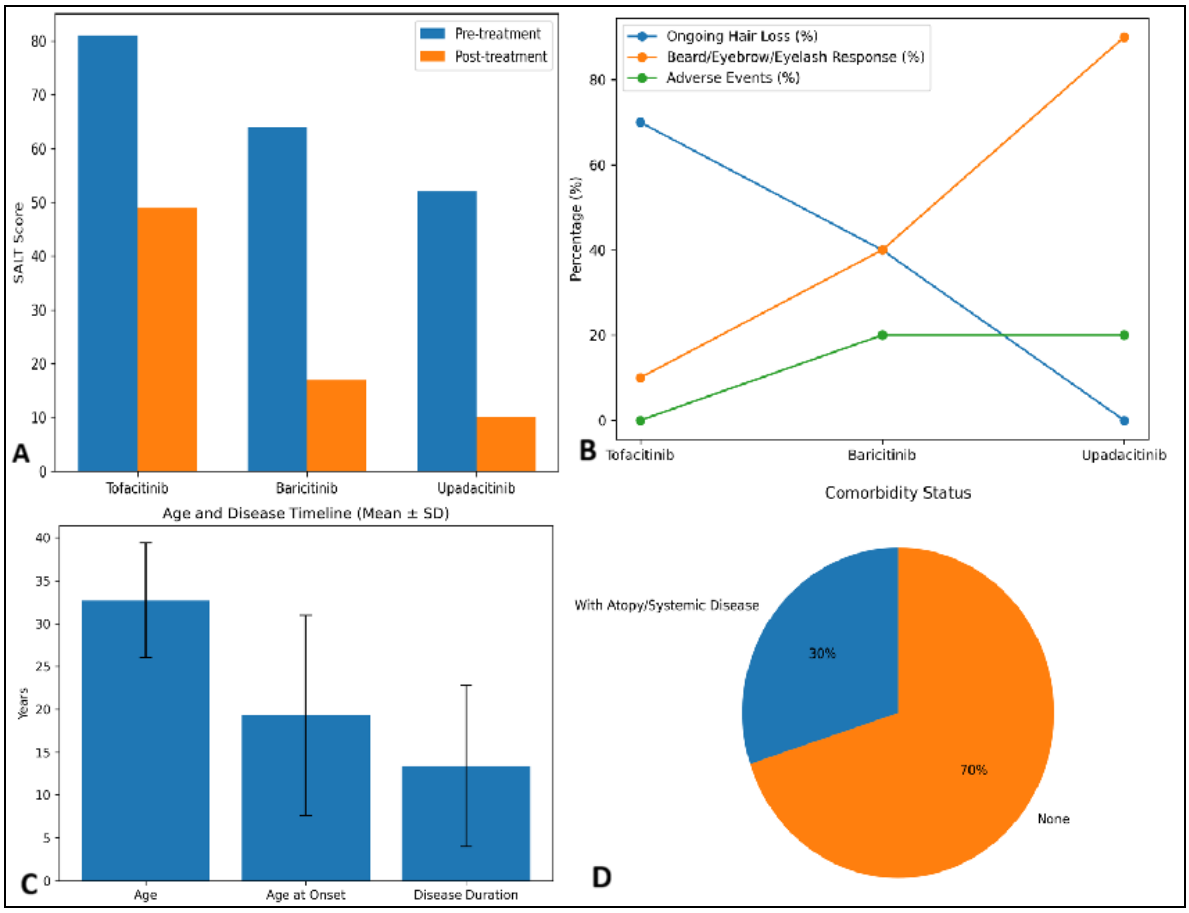

Figure A. Pre- and Post-Treatment SALT Score Changes Across Sequential JAK Inhibitor Therapies Mean Severity of Alopecia Tool (SALT) scores before and after treatment with tofacitinib, baricitinib, and upadacitinib are shown. Bars represent mean values for each treatment period, illustrating a progressive reduction in SALT scores across sequential JAK inhibitor therapies, with the most pronounced improvement observed during the upadacitinib treatment period. Figure B. Comparison of Clinical Outcomes Across Sequential JAK Inhibitor Treatments Clinical outcomes observed during treatment with tofacitinib, baricitinib, and upadacitinib are compared. Parameters include the proportion of patients with ongoing hair loss, the presence of beard, eyebrow, or eyelash regrowth, and the occurrence of adverse events. While ongoing hair loss decreased and cosmetic area involvement improved across sequential therapies, the highest rates of cosmetic response and complete suppression of hair loss were observed during upadacitinib treatment. Demographic Figure C. Age Distribution and Disease Timeline of the Study Cohort The figure illustrates the mean age of the patients, mean age at disease onset, and mean disease duration, expressed as mean ± standard deviation. The data highlight a young age at onset and a prolonged disease duration within the cohort, reflecting a population with long-standing and chronic alopecia areata. Demographic Figure D. Prevalence of Atopy and/or Systemic Comorbidities in the Study Cohort The proportion of patients with atopy and/or concomitant systemic diseases is shown. Atopic or systemic comorbidities were present in 30% of the patients, emphasizing the immunologically complex background of the study population.

Conclusions

Our study indicates that switching agents in AA cases with inadequate or partial response to the first JAK inhibitor can provide significant clinical improvement. Notably, upadacitinib showed more pronounced improvements in both SALT scores and patient-reported outcomes. These heterogeneous response profiles may be attributed to differences in JAK subtype selectivity. These findings warrant further support from larger, prospective studies.

Abstract N°: ID-364

Topic: Biologics, immunotherapy, targeted therapy

An Eight-Patient Case Series Detailing a Single Centres Real-World Experience with Dual Dupilumab and Omalizumab Therapy

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Introduction

Dual biologic therapy is increasingly used in the management of atopic and inflammatory conditions. In Australia, dupilumab is approved for atopic dermatitis (AD), asthma, and allergic rhinitis with nasal polyps, while omalizumab is indicated for chronic spontaneous urticaria (CSU) and asthma. Given their overlapping indications and the high rate of multimorbidity among atopic diseases, combined use is likely to increase in the future. However, current safety and efficacy data remains limited to a small number of case series and reports.

Materials and Methods

A retrospective case series was performed by searching the hospital electronic medical records for patients prescribed both dupilumab and omalizumab.

Results

A total of 8 patients were identified. Dupilumab was prescribed for AD in 6 patients and asthma in 2 patients. Omalizumab was prescribed for CSU in 6 patients and asthma in 2 patients. The average duration of dual therapy was 23.3 months (range 10.9-51.5), with six of the eight patients currently continuing dual therapy. Dual therapy was well tolerated in the majority of patients. Two separate adverse events were recorded; mild dry eyes following dupilumab commencement (prior to omalizumab commencement) and ocular surface disease leading to one patient self-ceasing dupilumab. AD and asthma patients maintained clinical response, however two CSU patients discontinued omalizumab due to secondary loos of efficacy which developed 12 and 24 months into therapy.

Conclusions

Findings from this series support the safety of dual omalizumab and dupilumab therapy. More research is necessary to give data regarding efficacy of dual therapy.

Abstract N°: ID-410

Topic: Biologics, immunotherapy, targeted therapy

Relationship of Genetic Variants of TYK2 (rs2304255) and DDX58 (rs34085293) Genes, Psoriasis Course Features and Response to Treatment with IL-17A Inhibitor

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Introduction
The data on the association of single nucleotide polymorphisms of TYK2 (rs2304255) and DDX58 (rs34085293) genes and the maximal clinical effect – achievement and long-term retention of PASI100 with interleukin (IL) 17A blockade, involvement of these genes in the pathogenetic axis of IL-23/IL-17 psoriasis development – strongly indicate the importance of studying the impact of single nucleotide polymorphisms of these genes at therapy effectiveness.
Purpose. To analyze the presence of TYK2 (rs2304255) and DDX58 (rs34085293) gene polymorphisms in patients with psoriasis and to evaluate the relationship of these single-nucleotide polymorphisms and therapeutic efficacy of IL-17A inhibitor netakimab.

Materials and Methods
70 patients (45 males and 25 females, median age 45 years) with moderate to severe forms of psoriasis vulgaris (n = 60) and pustular psoriasis (n = 10) were enrolled in the study. They were treated with IL-17A inhibitor netakimab. Clinical parameters were registered at weeks 0, 6, 24, 48 and 72 of the treatment. Polymerase chain reaction was used to determine allelic variants of TYK2 (rs2304255) and DDX58 (rs34085293) genes. Data statistical processing was done to find out the effectiveness of the studied genotypes at the psoriasis course and netakimab effectiveness.

Results
The overall incidence of TYK2 (rs2304255 C > T) allele was 18.33%; 15% of patients with psoriasis vulgaris were heterozygous for DDX58 (rs34085293 T > G). DDX58 (rs34085293 G/G) allele incidence was 1%. By the basic indicators of psoriasis severity and activity (Psoriasis Area and Severity Index, PASI and Physician Global Assessment, PGA), polymorphism presence or absence in the studied genes did not significantly influence the type of psoriasis severity. A statistically significant difference was found in TYK2 (rs2304255 C > T) polymorphism incidence in male patients ($p = 0.03$); the presence of DDX58 (rs34085293 T > G) polymorphism did not produce any statistically significant deviations in the male population ($p > 0.05$). In heterozygous individuals with TYK2 (rs2304255 C > T) alleles, tissue hyperexpression due to injury was a provoking factor in the psoriasis development; in individuals with DDX58 (rs34085293 T > G) polymorphism, an infectious process is thought to be an indicator of the increased risk. When assessing PASI and PGA dynamics in patients with TYK2 (rs2304255C > T) polymorphism, differences were registered in groups with homozygote (C/C) and heterozygote (C > T), which demonstrates a high efficacy of netakimab therapy (PASI 100) and retention of the effect of at least PASI 90 until therapy week 72 in patients with heterozygous genotype TYK2 (rs2304255 C > T) ($p < 0.001$). While comparing netakimab effectiveness by PASI index in groups with homozygous genotype DDX58 (rs34085293 T/T) and heterozygous genotype DDX58 (rs34085293 T > G), a significant difference was found at weeks 6, 24, and 48; in heterozygous patients, the effect of netakimab therapy was higher ($p < 0.05$).

Conclusions

The obtained pharmacogenomic research data – demonstrating the effectiveness of psoriasis therapy – will improve predictive diagnostics so as to assess the expected effectiveness of psoriasis therapy. In future, it could be used for the accurate interpretation of the individual patient’s profile and its genetic predictors so as to develop a personalized management plan.

Abstract N°: ID-418

Topic: Biologics, immunotherapy, targeted therapy

Concurrent Dual Biologic Therapy for Complex Treatment-Refractory Dermatologic Disease: A Single-Center Retrospective Analysis

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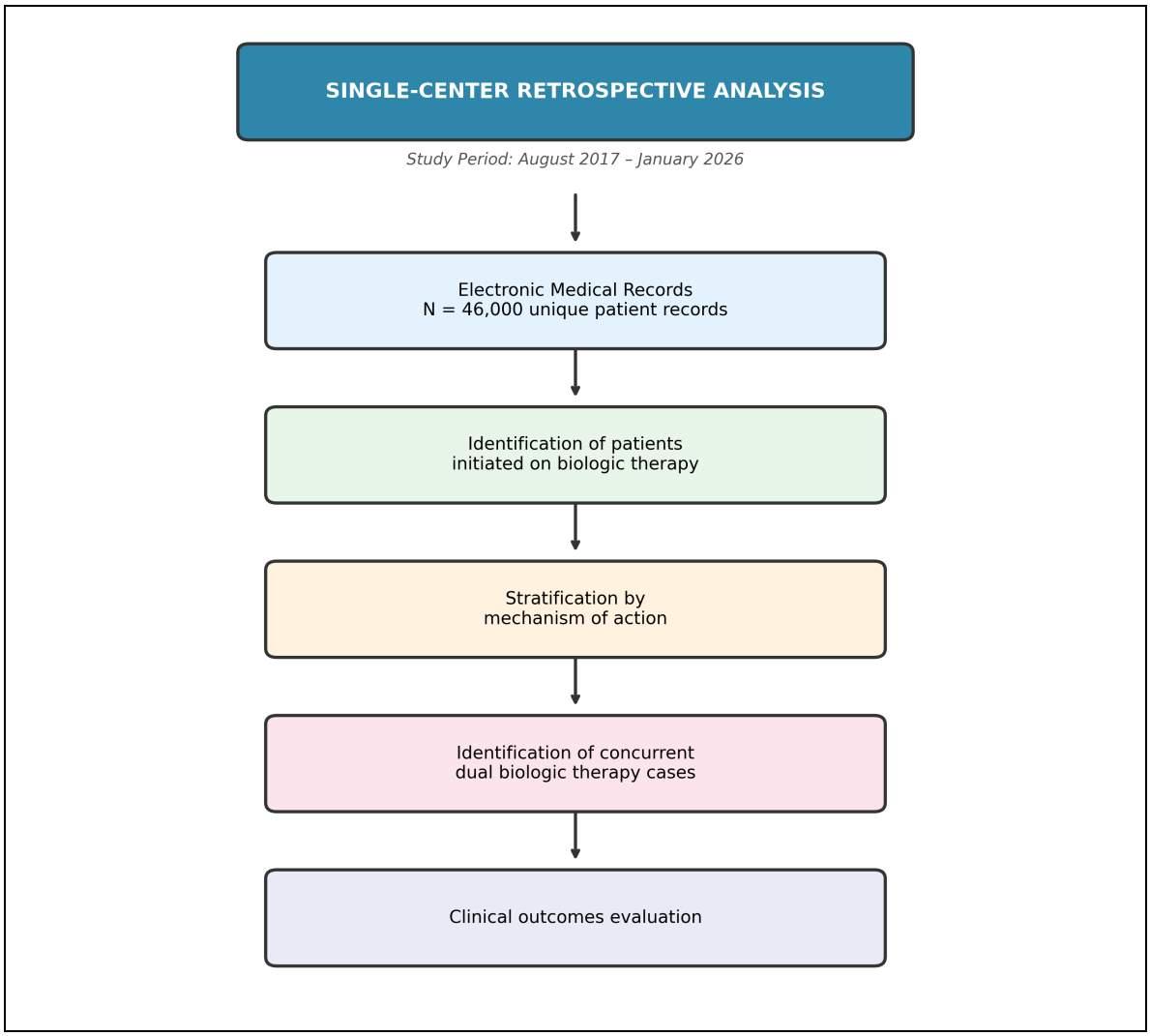
Introduction

Biologic therapies have transformed the management of inflammatory dermatoses. However, concurrent dual biologic therapy may be required when patients have coexisting systemic conditions requiring distinct immunomodulation. The complexity is significantly increased when two biologic response modifiers (BRMs) are used concurrently, or when an additional BRM is administered to mitigate adverse effects caused by the primary therapeutic BRM in situations where discontinuation of the culprit agent is not feasible due to clinical indications. Evidence guiding such combination regimens remains limited. We aimed to determine the incidence and outcomes of concurrent dual biologic therapy in a real-world dermatology practice.

Materials and Methods

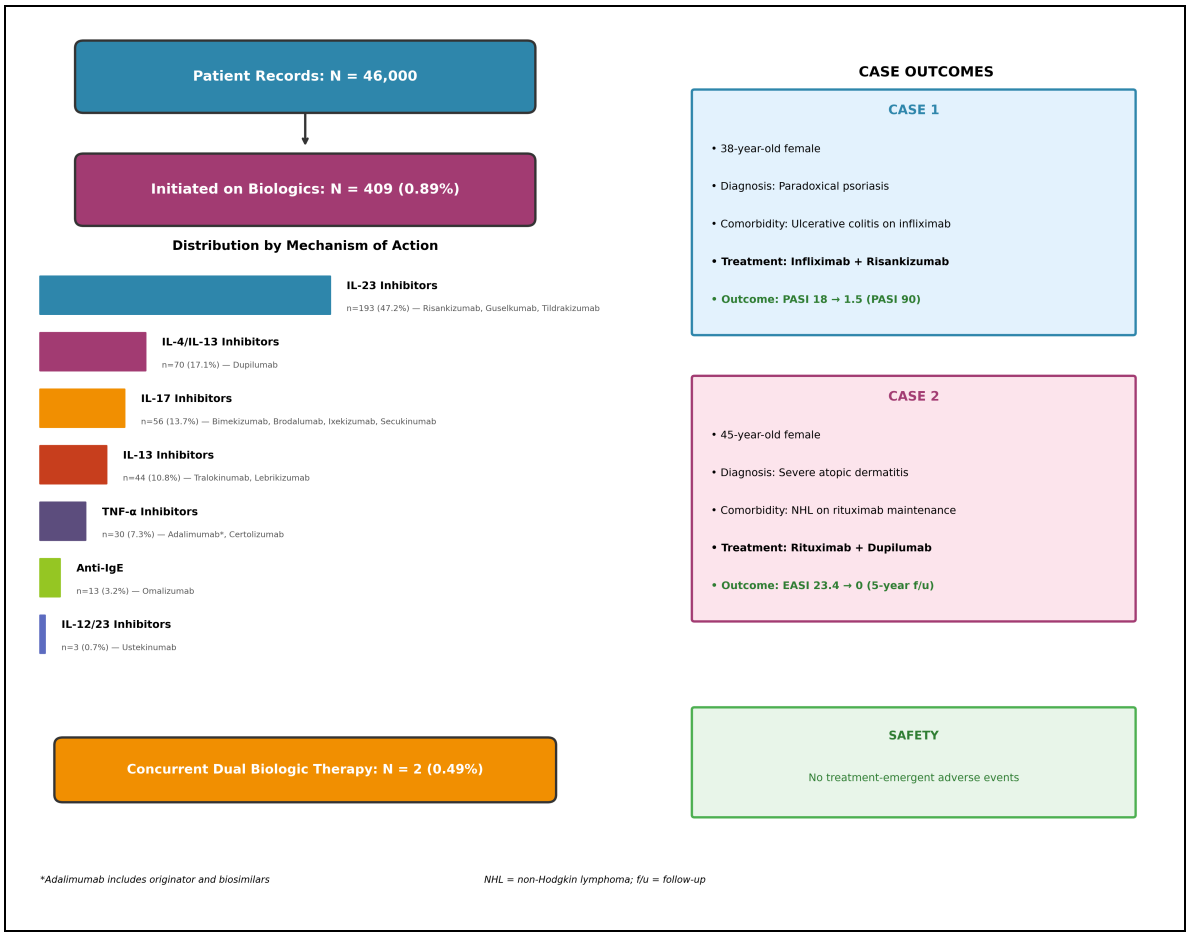
We conducted a single-center retrospective analysis of 46,000 unique patient records at the outpatient dermatology practice from August 2017 to January 2026. All patients initiated on biologic therapy were identified and stratified by mechanism of action (IL-23 inhibitors, IL-4/IL-13 inhibitors, IL-17 inhibitors, IL-13 inhibitors, TNF-α inhibitors, anti-IgE, and IL-12/23 inhibitors). Cases of concurrent dual biologic therapy were identified, and clinical outcomes were evaluated.



Study design. Single-center retrospective analysis of 46,000 patient records (August 2017 – January 2026) with stratification by mechanism of action and identification of dual biologic therapy cases.

Results

A total of 409 patients (0.89%) were initiated on biologic therapy across the following categories: IL-23 inhibitors (n=193, 47.2%; risankizumab, guselkumab, tildrakizumab), IL-4/IL-13 inhibitors (n=70, 17.1%; dupilumab), IL-17 inhibitors (n=56, 13.7%; bimekizumab, brodalumab, ixekizumab, secukinumab), IL-13 inhibitors (n=44, 10.8%; tralokinumab, lebrikizumab), TNF-α inhibitors (n=30, 7.3%; adalimumab and biosimilars, certolizumab), anti-IgE (n=13, 3.2%; omalizumab), and IL-12/23 inhibitors (n=3, 0.7%; ustekinumab). Two cases (0.49%) of concurrent dual biologic therapy were identified. Case 1: A 38-year-old woman with ulcerative colitis developed paradoxical psoriasis on infliximab; concurrent risankizumab achieved PASI 90 (PASI 18→1.5) with no adverse events. Case 2: A 45-year-old woman with severe atopic dermatitis on rituximab maintenance for non-Hodgkin lymphoma achieved complete clearance (EASI 23.4→0) with dupilumab over 5 years of co-therapy without treatment-emergent adverse events.



Patient flow and outcomes. Of 409 patients initiated on biologics across 7 MOA categories, IL-23 inhibitors were most prescribed (47.2%). Two patients (0.49%) received dual biologic therapy, both achieving excellent disease control (PASI 90; EASI 0) without adverse events.

Conclusions

Concurrent dual biologic therapy is rare in clinical practice (<0.5% of biologic-treated patients) but may be a viable strategy for managing complex, treatment-refractory dermatologic disease when monotherapy is insufficient and discontinuation of essential systemic therapy is not feasible. In carefully selected patients with multidisciplinary oversight, dual biologic regimens can achieve durable disease control with acceptable safety profiles.

Abstract N°: ID-426

Topic: Biologics, immunotherapy, targeted therapy

EFFECTIVENESS AND SAFETY OF BARICITINIB IN PATIENTS WITH SEVERE ALOPECIA AREATA

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Introduction

Severe Alopecia Areata (AA) is an autoimmune disease that causes patchy hair loss on the scalp, face and body.Baricitinib is an oral, selective, reversible janus kinase 1 and 2 (JAK1 and JAK2) inhibitor that is approved to treat severe AA in adults (>18 years).

The objetive is evaluate the effectiveness and safety of Baricitinib in patients with severe Alopecia Areata (AA) with Severity of Alopecia Tool (SALT) score of 50 or higher.

Materials and Methods

Observational, retrospective and multicentre study of patients treated forsevere Alopecia Areata (AA) with Baricitinib of Dermatology Services in two third-tier hospitals from November 2022 to October 2025.

Data were obtained from the Electronic Clinical History (DRAGO-AE®) and the Pharmacy Service Managing Software (FARMATOOLS®).

Severe AA is considered a Severity of Alopecia Tool (SALT) score of 50 or higher where 0 represents no scalp hair loss and 100 represents complete scalp hair loss.

Clinical variables collected were: sex, age, SALT ≥50, Baricitinib posology, SALT≤20 at week 36, adverse events, other autoimmune diseases and suspension of Baricitinib.

Baricitinib is funded in patients with severe AA with a current episode of more than 6 months.

Results

42 patients were included in the study, mean age 38 years old (27 women, 15 men) with a current episode of more than 6 months of severe AA (hair loss encompassing ≥50% of the scalp). Of these 42 patients, 3 of whom were adolescents (13-16 years), Baricitinib was used off-label.

4% of the patients had other autoinmune diseases (6: atopic dermatitis, 1:systemic lupus erythematosus, 1: vitiligo, 1: rheumatoid arthritis).

The recommended dose of Baricitinib is 4 mg once daily. 100% of the patients had a dose of Baricitinib 4 mg/24 hours.

The study assessed the proportion of patients who achieved a SALT score of ≤20 (80% or more scalp coverage with hair) at week 36. The results were: 6% of the patients assessed SALT ≤20 (11 women, 3 men).

No new safety concerns were identified, with reported adverse effects neutropaenia, upper respiratory tract infections and nausea (1,26% each) which did not lead to discontinuation of treatment. The therapy with Baricitinib was discontinued in 4,6% (11 patients) due to lack of response.

Conclusions

The effectiveness of Baricitinib observed in our patients was similar than reported in pivotal clinical trials, with no new safety alerts.

In view of these results, Baricitinibis an effective and safe treatment option for patientswith severe Alopecia Areata.

Abstract N°: ID-473

Topic: Biologics, immunotherapy, targeted therapy

Durable Inhibition of Structural Damage Progression and Improvements in Joint Disease Activity With Guselkumab in Active and Erosive Psoriatic Arthritis: Week 48 Results From APEX

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Introduction

APEX (NCT04882098) evaluates guselkumab (GUS), a fully-human mAb able to bind CD64 and selectively inhibit the IL-23p19-subunit, in participants (pts) with active and erosive PsA. At Week(W)24, GUS demonstrated significantly higher ACR20 rates (primary endpoint) and radiographic progression inhibition vs placebo (PBO; major secondary endpoint). Here we report W48 findings.

Materials and Methods

APEX enrolled adults with active PsA (≥3 tender, ≥3 swollen joints; CRP ≥0.3 mg/dL) and ≥2 erosive joints on radiographs of hands/feet, despite previous non-biologic therapy. The modified full analysis set comprised 1020 randomized pts (273 GUS 100mg Q4W; 371 GUS 100mg at W0/W4 then Q8W; 376 PBO-to-GUS Q4W at W24). Key endpoints through W48 included ACR20/ACR50 rates and least squares mean (LSM) change in PsA-modified van der Heijde-Sharp (vdH-S) score per reading session 2 (W0/24/48 radiographs). Exposure-adjusted incidence rates of adverse events (AEs) per 100 pt-years (100PY) [95% CI] are reported through W48.

Results

GUS Q4W/Q8W ACR20 rates increased from W24 (67%/68% vs 47% PBO; both p<0.001) to W48 (71%/74%). GUS ACR50

rates increased from W24 (41%/42% vs 20% PBO; both nominal-p<0.001) to W48 (51%/56%). At W48, 71% and 48% of PBO-to-GUS Q4W pts achieved ACR20 and ACR50, respectively.

Reading session 2 results indicated continued suppression of radiographic progression with GUS Q4W/Q8W from W0-24 (LSM changes in PsA-modified vdH-S score: 0.36/0.46) throughout W24-48 (0.24/0.32). Radiographic progression in the PBO group from W0-24 (0.96) was curtailed by GUS W24-48 (0.41).

Through W24, AE incidence rates with GUS Q4W/Q8W (168[146-191]/163[145-183]) and PBO (174[155-195]) were similar. Incidence rates did not increase with continued GUS (W0-48: 147 [132-163]/148 [136-162]), or after PBO-to-GUS transition (W24-48: 156 [138-176]).

Conclusions

In biologic-naïve adults with active and erosive PsA, inhibition of radiographic progression and joint disease activity improvements with GUS were durable through W48 without increased AE incidence, further substantiating GUS benefit for preserving joint health.

Abstract N°: ID-528

Topic: Biologics, immunotherapy, targeted therapy

Selective Facial Microbiome Changes Following JAK Inhibitor Therapy Without Global Dysbiosis

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Introduction
Acneiform eruptions are a recognized adverse event associated with Janus kinase (JAK) inhibitor therapy, yet their underlying mechanisms remain incompletely understood. Given the importance of the facial skin microbiome in inflammatory dermatoses, we investigated whether JAK inhibitor treatment is associated with compositional changes in the facial bacterial and fungal microbiome. This study aimed to assess age-stratified, paired changes in the facial skin microbiome before and after treatment with JAK inhibitors.

Materials and Methods
Facial skin samples were collected from 31 patients receiving JAK inhibitors, yielding 62 paired samples obtained before and after treatment. Participants were stratified into younger and older (≥50 years) age groups. Bacterial and fungal communities were analyzed using next-generation sequencing. Alpha diversity, beta diversity (principal coordinates analysis), taxonomic composition, and differential abundance were assessed, including LEfSe analysis with an LDA threshold >2.0.

Results
Beta-diversity analyses demonstrated substantial overlap between pre- and post-treatment samples in both age groups, indicating no major community-wide restructuring of the facial microbiome. In alpha-diversity analyses, significant pre/post differences were observed in the fungal community of the older group for phylogenetic diversity (p=0.013) and observed features (p=0.037), while bacterial alpha diversity remained unchanged in both age strata. Differential abundance analysis identified selective changes in low-abundance taxa. Notably, *Penicillium digitatum* was enriched after treatment in both age groups. Several fungal taxa displayed age-dependent directional changes. In the bacterial dataset, enrichment of *Neisseria, Streptococcus,* and *Rothia* was observed after treatment in younger patients, whereas no significant discriminant bacterial taxa were identified in the older group.

Conclusions
JAK inhibitor therapy was associated with selective, age-modified changes in specific facial bacterial and fungal taxa without evidence of global microbiome dysbiosis. These findings suggest that JAK inhibitor–associated acne may be related to immune-mediated alterations in host–microbe interactions rather than broad compositional changes of the facial skin microbiome.

Abstract N°: ID-529

Topic: Biologics, immunotherapy, targeted therapy

Successful Treatment of Facial Pseudolymphoma With Intravenous Rituximab: A Case Report

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Introduction

Pseudolymphoma is a benign lymphoproliferative disorder triggered by varied stimuli that closely mimics malignant lymphoma clinically and histologically.
Cutaneous lymphoid hyperplasia typically presents as persistent erythematous nodules or plaques, primarily on the head, neck, and upper limbs.
Accurate diagnosis requires comprehensive histopathology and immunohistochemical staining to distinguish dense lymphocytic infiltrates from true malignancies.
Standard treatments range from topical corticosteroids and excision to phototherapy, though some cases regress spontaneously while others remain refractory.
This report highlights intravenous Rituximab as an effective targeted therapy for treatment-resistant cases, offering a solution when traditional methods fail.

Materials and Methods

A 42-year-old man developed spreading facial nodules and pruritic plaques after dental implant surgery, which were initially misdiagnosed as lupus erythematosus.
Despite various treatments including oral prednisolone, hydroxychloroquine, and topical therapies, the lesions remained refractory and sensitive to sun exposure.
A specialized biopsy revealed dense dermal lymphocytic infiltration and follicular structures, suggesting a differential diagnosis of pseudolymphoma or sarcoidosis.
Immunohistochemical staining and PCR analysis confirmed a polyclonal B-cell population, effectively ruling out malignancy and light chain restriction.
The final diagnosis of cutaneous B-cell pseudolymphoma was established through this comprehensive clinical, histopathological, and molecular evaluation.

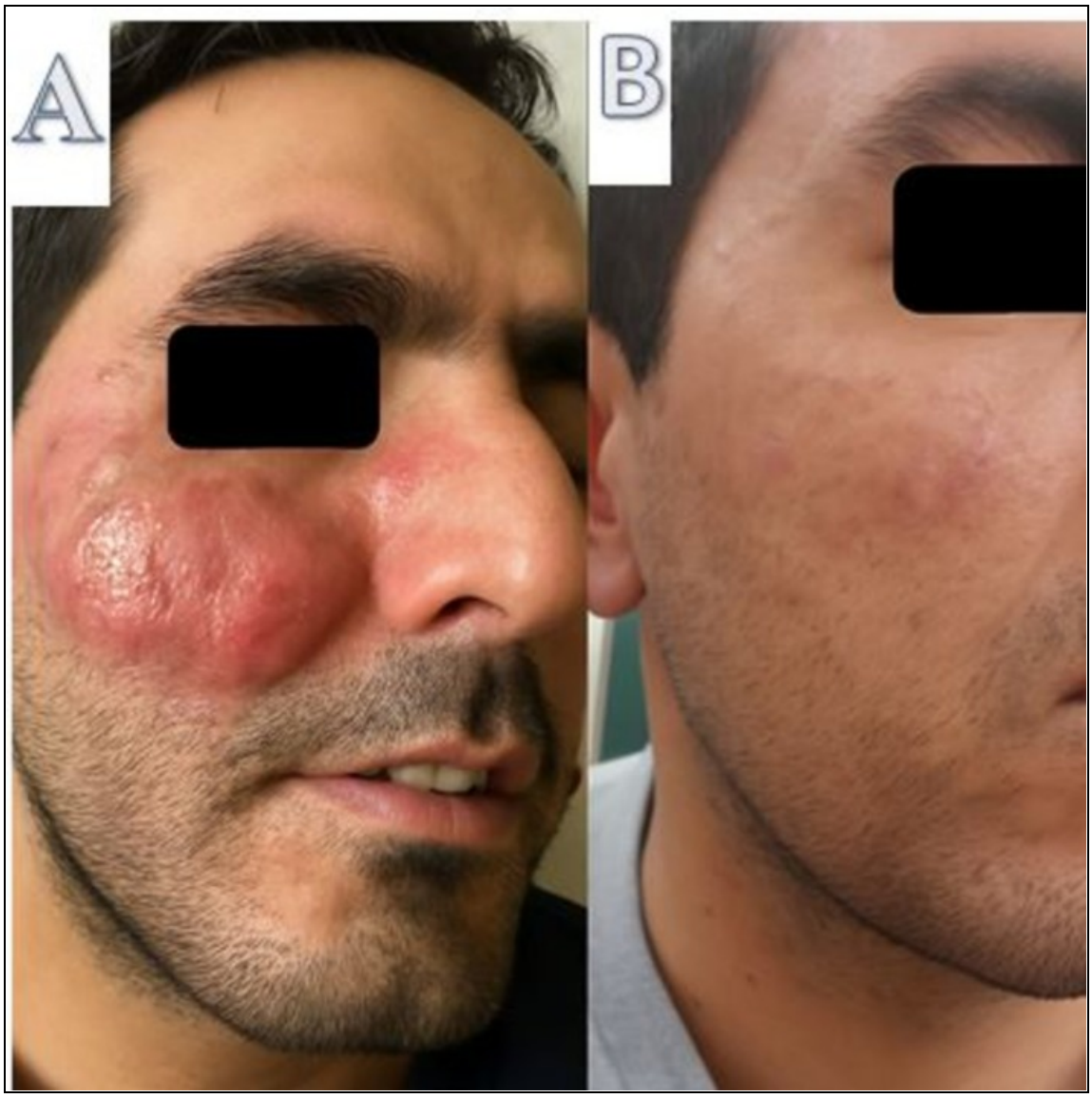

FIGURE 2 Significant healing of facial lesions following treatment with Rituximab. (A) Before, (B) After.

Results

The introduction of intravenous rituximab (500 mg weekly for 4 weeks) led to a marked reduction in lesion size and erythema after the second infusion. Two additional rituximab injections were administered at four-month intervals, resulting in significant disease regression. By the end of the 4-week course, the lesions had significantly diminished, and no recurrence was observed during 1 year of follow-up (Figure 2).

The patient's peripheral B-cell population was effectively depleted following rituximab induction, as confirmed by CD19+ and CD20+ flow cytometry showing < 1% B-cells at 1 month. Immunoglobulin levels remained stable across all timepoints up to 12 months post-treatment. Additionally, HBV reactivation screening and TB surveillance remained negative, and no laboratory abnormalities or infections occurred during the monitoring period.

This case highlights the challenges in diagnosing and managing cutaneous pseudolymphoma, particularly in patients with treatment-resistant disease. The patient's condition remained refractory to multiple immunosuppressive and antiinflammatory therapies, including corticosteroids, antimalarials, immunomodulators, and biologics. However, intravenous rituximab demonstrated substantial efficacy, leading to a significant reduction in lesion burden. These findings suggest that rituximab may be a viable treatment option for refractory cases of cutaneous pseudolymphoma. Regular follow-up is essential to monitor disease recurrence and assess long-term treatment outcomes.

This case adds to the limited but expanding clinical experience with rituximab in cutaneous pseudolymphoma. Future studies with larger cohorts and longer follow-up periods are necessary to establish optimal dosing regimens and to evaluate the durability of treatment response.

Conclusions

This case adds to the limited but expanding clinical experience with rituximab in cutaneous pseudolymphoma. Future studies with larger cohorts and longer follow-up periods are necessary to establish optimal dosing regimens and to evaluate the durability of treatment response.

EA SYMPOSIUM DV 2026
ATHENS 7 - 9 MAY

Abstract N°: ID-530

Topic: Biologics, immunotherapy, targeted therapy

Darier's Disease Successfully Treated with Tofacitinib: A Case Report

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Introduction

Introduction

Darier's disease (DD) is a rare autosomal dominant genodermatosis caused by mutations in the ATP2A2 gene, leading to impaired keratinocyte adhesion and chronic relapsing disease. Management is challenging, particularly in refractory cases, and effective long-term treatments are limited. Janus kinase (JAK) inhibitors have recently emerged as potential therapeutic options for difficult-to-treat dermatologic conditions.

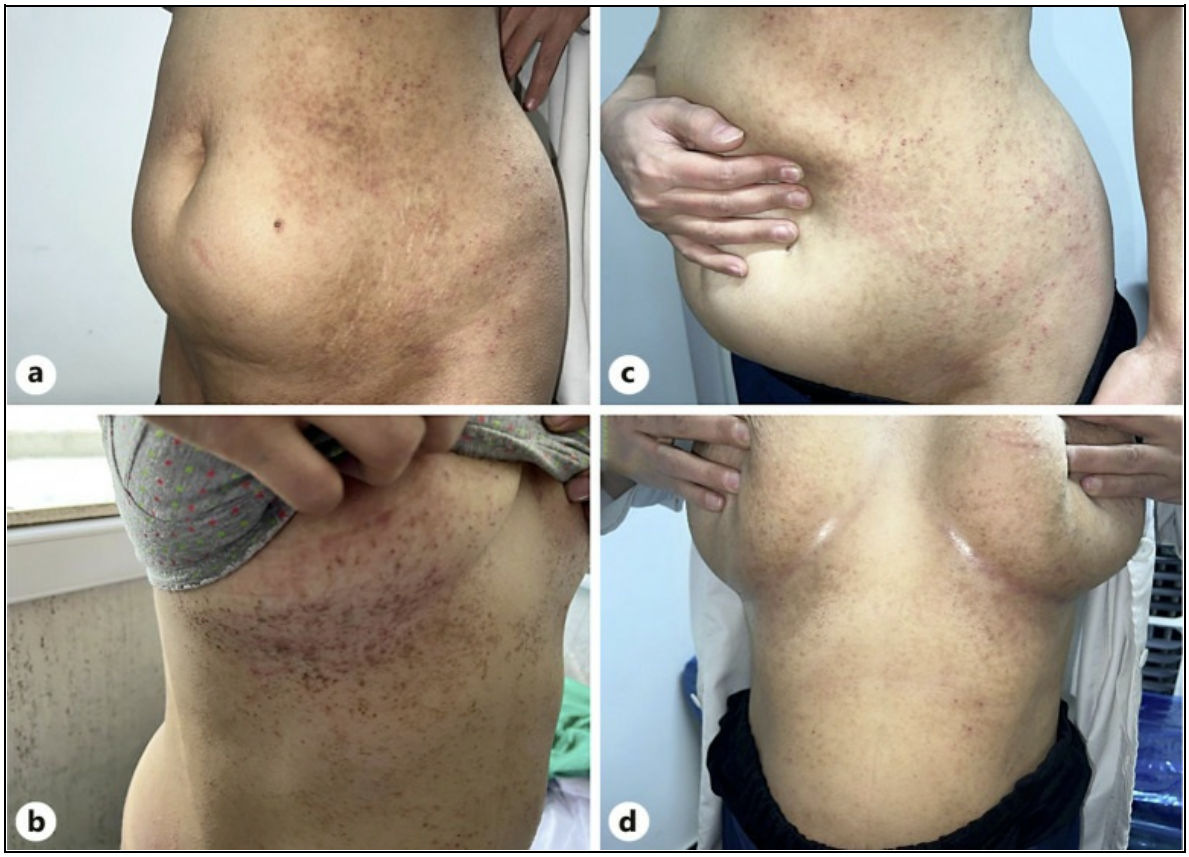

Fig. 1. Hyperkeratotic follicular brown papules with excoriations on the trunk (a, b), showing flattening of the lesions 1 month after treatment (c, d).

Materials and Methods

Materials and Methods

We report a 42-year-old woman presenting with pruritic, hyperkeratotic brown papules involving the trunk, neck, inframammary area, and groin, with disease exacerbation during warm weather. Histopathological examination demonstrated full-thickness epidermal acantholysis and dyskeratosis, confirming the diagnosis of Darier's disease. The patient was initially treated with adalimumab (40 mg biweekly) without clinical improvement. Following informed

consent and baseline laboratory evaluation, oral tofacitinib was initiated at 10 mg daily.

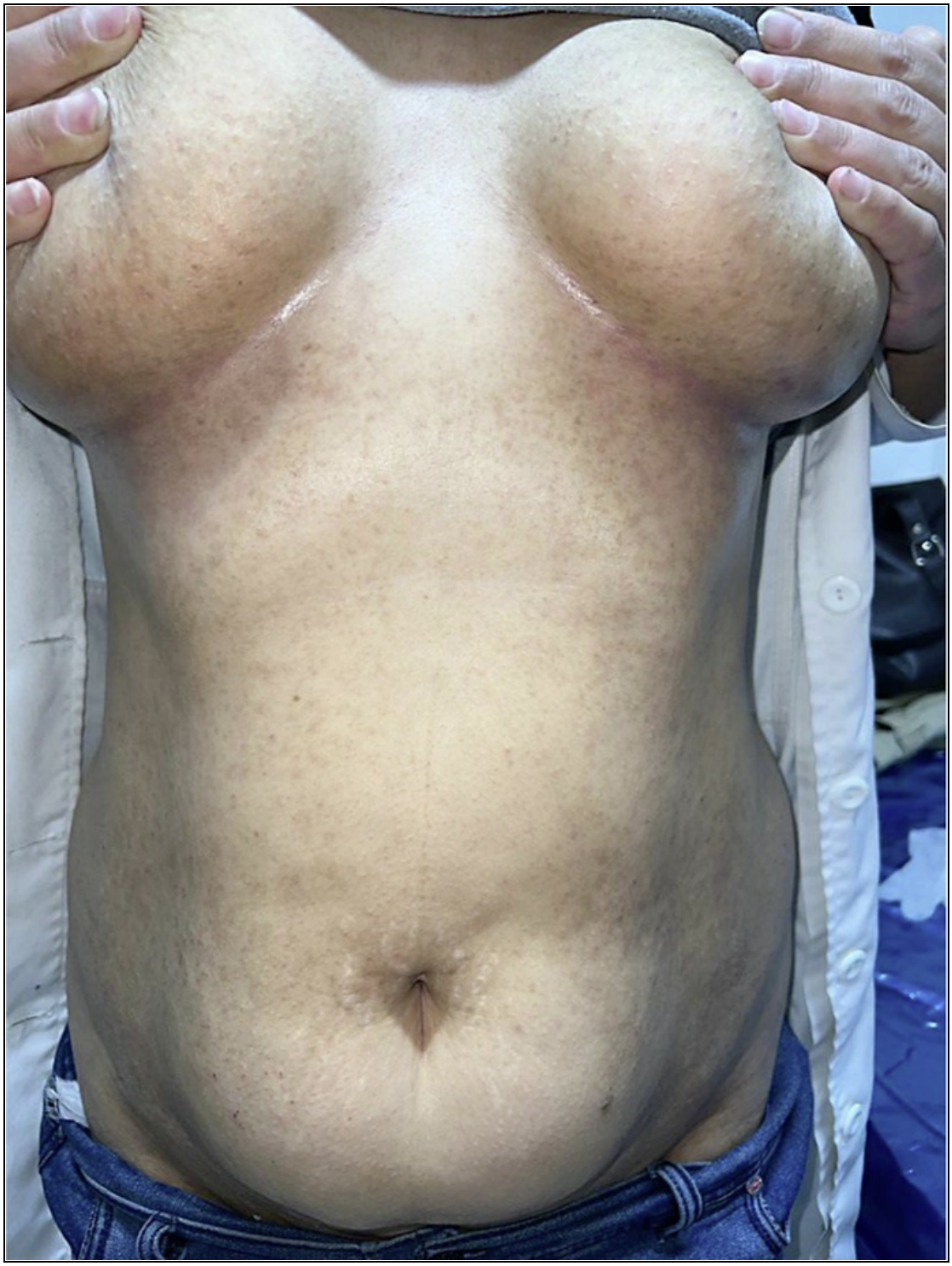

Fig. 3. Photograph of the trunk 3 months after treatment, showing remission of lesions and excoriations.

Results

Results

Treatment with tofacitinib led to disease stabilization; however, persistent pruritus and new lesions prompted dose escalation to 15 mg daily. Within one month, approximately 90% clinical improvement was observed, with marked reduction in pruritus and lesion burden. After three months of sustained response, the dose was reduced to 10 mg daily, maintaining disease control. No adverse effects or laboratory abnormalities were detected during follow-up, and the patient reported high satisfaction with treatment outcomes.

Conclusions

Conclusion

This case highlights the potential efficacy and safety of tofacitinib as a targeted therapeutic option for refractory Darier's

disease. JAK inhibition may represent a promising alternative in patients unresponsive to conventional therapies. Further studies are needed to clarify long-term outcomes and optimal treatment strategies.

Abstract N°: ID-555

Topic: Biologics, immunotherapy, targeted therapy

Acquired Hyperhidrosis Following Dupilumab Therapy for Atopic Dermatitis: A Case Report

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Introduction

Dupilumab, a monoclonal antibody targeting interleukin-4 *(IL-4)* and interleukin-13 *(IL-13)* signaling, is an effective treatment for moderate-to-severe atopic dermatitis (AD). While its safety profile is generally favorable, the emergence of paradoxical adverse effects warrants careful observation.

Materials and Methods

This report details the case of a 34-year-old Saudiwoman with chronic, moderate-to-severe AD who developed new-onset, localized hyperhidrosis and flushing over her chest and back approximately two years into Dupilumab therapy. Despite AD often being associated with reduced sweating, this patient experienced excessive perspiration.

Results

Causality assessment using the Naranjo Adverse Drug Reaction Probability Scale indicated a probable link between Dupilumab and the hyperhidrosis.

Conclusions

This case highlights the importance of recognizing atypical adverse drug reactions, even those seemingly contradictory to the underlying disease pathophysiology, to enhance pharmacovigilance and guide clinical practice.

Abstract N°: ID-570

Topic: Biologics, immunotherapy, targeted therapy

Biologic switching in psoriasis: a national cohort study under a standardized national formulary

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Introduction
Biologic therapies targeting tumor necrosis factor (TNF) and interleukin (IL)-12/23, IL-17, and IL-23 pathways have transformed treatment of moderate-to-severe psoriasis. However, real-world biologic switching remains common and is influenced by both clinical and non-clinical factors. Most prior studies of biologic switching rely on commercial or Medicare claims data, where formulary restrictions, prior authorizations, and coverage changes may confound clinical decision-making. The U.S. Veterans Health Administration (VHA), with its nationally standardized formulary and medically complex population, provides a unique setting to examine biologic initiation, persistence, and switching patterns with reduced insurance-related variability. We aimed to characterize biologic initiation and switching among patients with psoriasis, compare switching patterns across biologic classes, and identify demographic and clinical factors associated with switching.

Materials and Methods
We conducted a retrospective national cohort study using VHA electronic health record data from fiscal years 2016–2023. Patients with incident psoriasis were identified using validated ICD-10 diagnostic codes. Patients were followed for two years after diagnosis to assess biologic initiation and, among biologic initiators, for an additional two years to evaluate class switching and discontinuation. Biologic classes included TNF, IL-12/23, IL-17, and IL-23 inhibitors. Switching was defined as a change in biologic mechanism-of-action class, and discontinuation was defined as no biologic prescription within the final 120 days of follow-up. Demographic characteristics, healthcare utilization, and comorbidities (including psoriatic arthritis, obesity, cardiovascular disease, inflammatory bowel disease, and psychiatric diagnoses) were assessed using structured EHR data. Bivariate comparisons between were performed using chi-square tests and Student's t-tests.

Results
Among 114,759 patients with newly diagnosed psoriasis (mean age 62.2 years; 92.1% male), 9.4% of biologic-naïve patients initiated biologic therapy within two years of diagnosis. TNF inhibitors were the most common initial class (84.2%). Among biologic initiators, 16.5% switched biologic classes and 29.1% discontinued therapy within two years. Female patients were more likely to initiate biologics (13.3% vs. 9.1%), initiate later, and both switch (23.0% vs. 15.8%) and discontinue therapy (34.2% vs. 28.7%). Switching rates varied by initial biologic class, with the highest rates observed among IL-12/23 inhibitors (25.5%) and the lowest among IL-23 inhibitors (14.8%). Median time to first switch was 355

days and did not differ by biologic class.

Patients who switched biologics were younger and more likely to have obesity, psoriatic arthritis, major depressive disorder, and post-traumatic stress disorder compared with non-switchers. Inflammatory bowel disease was less common among switchers. At the end of follow-up, 70.9% of patients remained on biologic therapy, with lower persistence observed among women.

Conclusions

In this national VHA cohort with a standardized formulary, biologic switching and discontinuation was lower than prior U.S. commercial and Medicare claims studies but remained substantial. Persistence differed by biologic class, and switching was associated with demographic, metabolic, rheumatologic, and psychiatric factors. Given the high cost of biologics, each switch may represent substantial therapeutic waste and resource utilization, reinforcing the need to optimize initial drug selection. These findings highlight the multifactorial nature of biologic treatment modification and underscore the importance of incorporating comorbidity burden and psychosocial factors into personalized psoriasis management. Future studies incorporating disease severity and patient-reported outcomes are needed to further clarify drivers of biologic persistence in real-world settings.

Abstract N°: ID-579

Topic: Biologics, immunotherapy, targeted therapy

Beliefs About Medicines and Adherence to Biologic Therapy in Patients with Moderate-to-Severe Psoriasis: A Cross-Sectional Study

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Introduction
Psoriasis is a chronic immune-mediated inflammatory disease that requires sustained treatment to achieve long-term disease control. Although biologic therapies, particularly interleukin-17 and interleukin-23 inhibitors, provide high levels of efficacy, their real-world effectiveness is influenced by patients' adherence and beliefs about medicines. Data linking adherence to both general and treatment-specific medication beliefs in psoriasis are scarce. The objective of this study was to evaluate adherence to biologic therapy and to analyse its association with medication beliefs in patients with moderate-to-severe psoriasis.

Materials and Methods
This cross-sectional study was conducted between March and August 2025 at a tertiary referral dermatology centre. Adult patients with moderate-to-severe psoriasis receiving biologic therapy with interleukin-17 or interleukin-23 inhibitors were consecutively recruited during routine outpatient visits. Medication adherence was assessed using the Medication Adherence Report Scale (MARS-5). Patients' beliefs about medicines were evaluated using validated Croatian versions of the Beliefs about Medicines Questionnaire–General (BMQ-G) and Beliefs about Medicines Questionnaire–Specific (BMQ-S) and interpreted according to the Necessity–Concerns Framework. Sociodemographic and clinical data were collected from medical records and patient questionnaires. Statistical analyses included correlation analyses and group-based comparisons to explore associations between medication beliefs, adherence, and sociodemographic characteristics.

Results

A total of 122 patients were included in the analysis. Overall self-reported adherence to biologic therapy was high, with 96.7% of participants reporting use of medication as prescribed and 84.4% reporting consistent adherence to dosing instructions. Strong treatment necessity beliefs predominated across the cohort; however, treatment-related concerns were frequently reported. Concerns regarding long-term effects were expressed by 40.2% of patients, while 15.6% reported concerns about dependence. More negative general beliefs about medicines were associated with less favourable treatment perceptions and adherence. Higher BMQ-G Overuse scores were significantly correlated with lower adherence ($r = -0.27$; $p = 0.003$) and with higher BMQ-Specific concern scores. Based on the Necessity–Concerns Framework, 31.1% of patients were classified as Distrustful, characterised by low necessity beliefs and high concerns, and this group demonstrated the highest scores for perceived medication harm and overuse. Lower educational attainment was significantly associated with more negative general beliefs about medicines.

Conclusions

Self-reported adherence to biologic therapy was high in this real-world cohort of patients with moderate-to-severe psoriasis treated with IL-17/IL-23 inhibitors, yet a substantial proportion of patients reported persistent concerns about treatment, particularly regarding long-term effects and dependence. More negative general beliefs about medicines, especially perceptions of overuse and harm, were associated with lower adherence and higher treatment-specific concerns, and were more frequent among patients with lower educational attainment. These findings support the routine evaluation of both general and treatment-specific medication beliefs in clinical practice and indicate that targeted, belief-oriented communication and patient education could help address concerns and reinforce long-term adherence to biologic therapy.

Abstract N°: ID-581

Topic: Biologics, immunotherapy, targeted therapy

Therapeutic Efficacy of Mesenchymal Stem Cell–Derived Extracellular Vesicles for Wound Healing and Skin Regeneration: A Systematic Review of Meta-Analyses

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Introduction

Background:

Studies examining the therapeutic potential of mesenchymal stem cell–derived extracellular vesicles (MSC-EVs), including exosomes, in wound healing and skin regeneration have expanded rapidly. Preclinical evidence suggests that MSC-EVs exert regenerative, pro-angiogenic, and immunomodulatory effects across multiple phases of wound repair. However, before clinical translation, a systematic understanding of therapeutic strategies, experimental designs, and overall efficacy is required. While most evidence remains preclinical, recent meta-analyses of human studies evaluating MSC-derived exosomes in cutaneous applications provide early translational insight. This study aims to systematically review and synthesize findings from published meta-analyses to evaluate the overall impact of MSC-EV–based therapies on wound healing and skin regeneration.

Materials and Methods

PubMed was searched using the Boolean strategy: (exosomes OR extracellular vesicles) AND (wound healing OR skin regeneration). Following screening and eligibility assessment, 16 meta-analyses were included. These comprised 15 preclinical meta-analyses synthesizing data from over 440 primary experimental animal studies (exceeding 4,300 reported animals) and one meta-analysis of human clinical trials evaluating MSC-derived exosomes in cutaneous applications. Outcomes assessed included wound closure rate, angiogenesis, re-epithelialization, collagen deposition, scar formation, skin quality parameters, and inflammatory modulation.

Table 1- Methodology, Studies Included.

Study (Reference Link)	Number of Papers Reviewed	Total Number of Animals Tested
PubMed ID: **36185607**		
https://pubmed.ncbi.nlm.nih.gov/36185607/	68 studies (44 non-diabetic, 28 diabetic)	>1843 animals (many studies did not report exact numbers)
PubMed ID: **38849563**		
https://pubmed.ncbi.nlm.nih.gov/38849563/	43 studies	530 animals
PubMed ID: **40128791**		
https://pubmed.ncbi.nlm.nih.gov/40128791/	83 studies (36 non-diabetic, 39 diabetic)	>560 animals
PubMed ID: **34461128**		
https://pubmed.ncbi.nlm.nih.gov/34461128/	60 studies	>46 animals
PubMed ID: **36669689**		
https://pubmed.ncbi.nlm.nih.gov/36669689/	21 studies	323 animals
PubMed ID: **39066806**		
https://pubmed.ncbi.nlm.nih.gov/39066806/	6 studies	72 animals
PubMed ID: **33893619**		
https://pubmed.ncbi.nlm.nih.gov/33893619/	10 studies	136 animals
PubMed ID: **39076515**		
https://pubmed.ncbi.nlm.nih.gov/39076515/	21 studies	Not specified
PubMed ID: **37102269**		
https://pubmed.ncbi.nlm.nih.gov/37102269/	13 studies	13 animals
PubMed ID: **39038766**		
https://pubmed.ncbi.nlm.nih.gov/39038766/	22 studies (18 preclinical, 4 clinical without results)	18 animals
PubMed ID: **38970763**		
https://pubmed.ncbi.nlm.nih.gov/38970763/	12 studies	Not specified
PubMed ID: **37015903**		
https://pubmed.ncbi.nlm.nih.gov/37015903/	29 studies	381 animals
PubMed ID: **40598532**		
https://pubmed.ncbi.nlm.nih.gov/40598532/	27 studies	Not specified
PubMed ID: **40311161**		
https://pubmed.ncbi.nlm.nih.gov/40311161/	19 studies	264 animals
PubMed ID: **39812853**		
https://pubmed.ncbi.nlm.nih.gov/39812853/	9 studies	128 animals
MDPI Article		
https://www.mdpi.com/2571-841X/8/4/268	6 studies	Clinical human studies (no animals)

Results

Among the included meta-analyses, six evaluated general wound healing and skin regeneration, nine focused on diabetic wound healing, and one synthesized human clinical trial data. Preclinical meta-analyses consistently demonstrated significant improvements in wound closure, neovascularization, re-epithelialization, collagen deposition, and scar reduction following MSC-EV or exosome therapy in both diabetic and non-diabetic models. Modified EVs—including microRNA-enriched vesicles, vesicles derived from preconditioned or genetically modified cells, and vesicles incorporated into biomaterial scaffolds—showed superior efficacy. Favourable immunomodulatory effects were observed, with reduced pro-inflammatory cytokines and increased anti-inflammatory mediators.

The human meta-analysis reported improvements in cutaneous outcomes such as scar characteristics, skin elasticity, and overall aesthetic parameters, with no major safety concerns identified, although study heterogeneity and limited sample sizes constrained definitive conclusions.

Conclusions

Evidence from 16 meta-analyses, encompassing both preclinical and human studies, supports the regenerative potential of MSC-EV and exosome-based therapies for cutaneous wound healing and skin regeneration. While preclinical efficacy is consistent and robust—particularly in diabetic wound models—human evidence remains early and limited. Standardized methodologies and high-quality randomized clinical trials are essential to bridge the translational gap and support clinical implementation.

Abstract N°: ID-593

Topic: Biologics, immunotherapy, targeted therapy

Paradoxical Vitiligo Outcomes with Dupilumab Use: A Systematic Review

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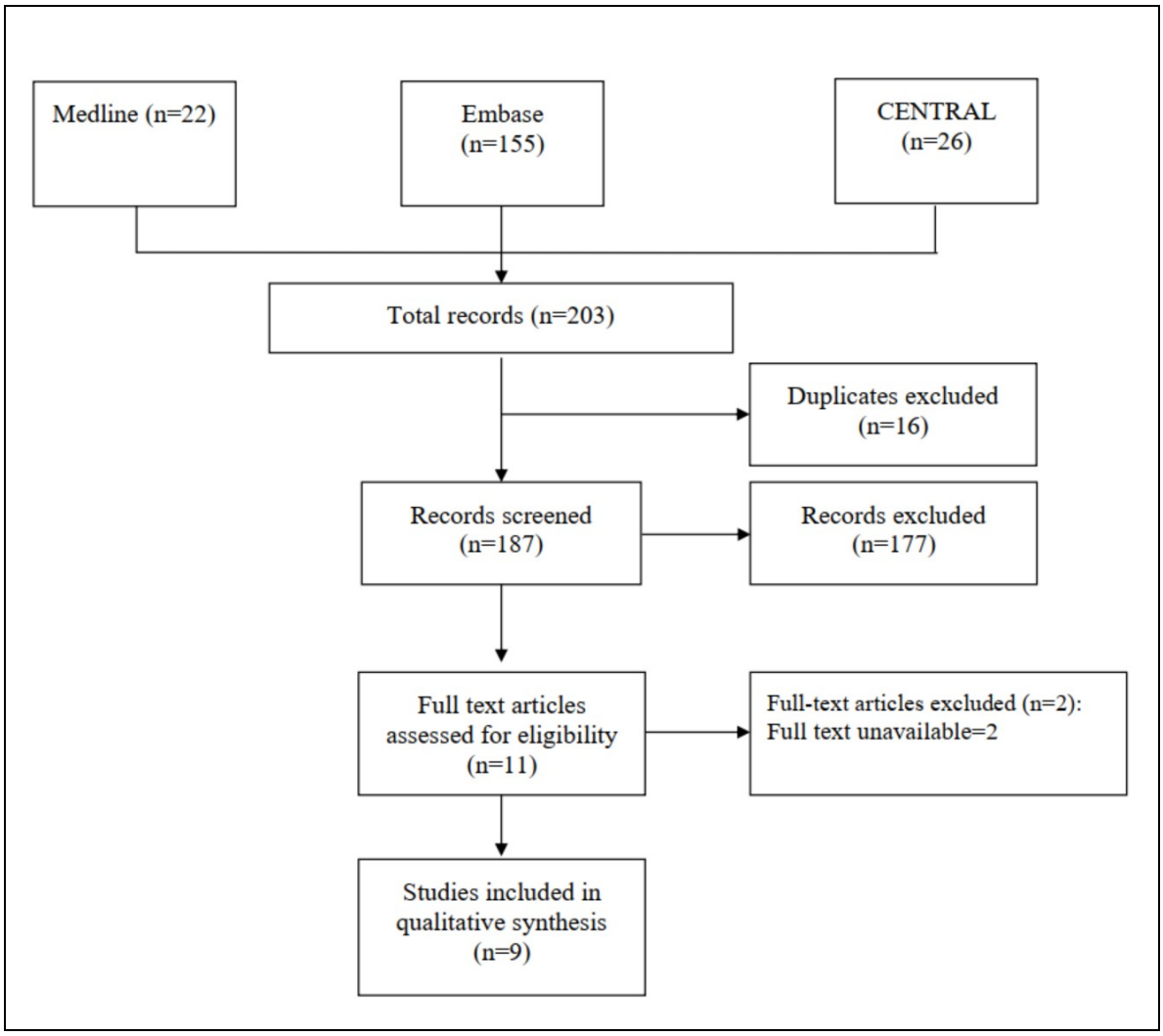
Introduction
Dupilumab, an IL-4Rα antagonist approved for various inflammatory conditions, has been linked to pigmentary changes. However, its role in inducing, worsening, or improving vitiligo remains unclear.

Materials and Methods
A PRISMA-guided systematic review was conducted using MEDLINE, EMBASE, and CENTRAL through November 2025. Eligible studies reported vitiligo outcomes in patients on dupilumab monotherapy. Data were extracted and reported on clinical characteristics and outcomes.



Results

We included 9 articles (with a total of 21 patients where vitiligo either developed or worsened, and 2 patients where repigmentation of vitiligo occurred) [8-16]. Among the reported cases, 13 patients without preexisting vitiligo developed new depigmented lesions after initiating dupilumab, primarily for atopic dermatitis (AD) [8-11]. The onset of vitiligo varied, occurring between 1 to 24 months after treatment initiation (mean ~5.15), with lesions commonly appearing on the scalp, neck, hands, feet, and trunk [8-14]. While some patients continued dupilumab despite vitiligo progression, others discontinued it due to worsening depigmentation [11]. Various treatments were introduced, including topical corticosteroids, tacrolimus 0.1% ointment, triamcinolone acetonide 0.1% ointment, and narrowband UVB (NBUVB) phototherapy [8-14]. Outcomes of vitiligo following treatment are not all available, however, in four cases where vitiligo has newly developed, complete remission of both vitiligo and AD was observed despite continued dupilumab use [8, 14]. In another case, discontinuation of dupilumab did not lead to lesion regression [10].

In contrast to the cases above, only two cases reported repigmentation of vitiligo occurring after one year in one patient and within 5 months in another [12,13]. These, to our knowledge, remain the only documented cases of repigmentation, emphasizing the variability in patient outcomes. Table I provides a summary of all included patients, covering the demographics, indication for Dupilumab use, associated side effects (induction or repigmentation), time to outcome, treatments received for vitiligo, and their outcomes.

Conclusions

The relationship between dupilumab and pigmentary changes remains unclear, with reports indicating both induction or worsening of vitiligo and, in rare cases, repigmentation. While most documented cases suggest that dupilumab may contribute to vitiligo development by altering immune pathways, the exact mechanisms behind these effects are not fully understood. Conversely, the few cases of repigmentation raise the possibility of a different immune response in certain individuals. Given the limited number of reported cases and the absence of controlled studies, no definitive

conclusions can be made regarding the role of dupilumab in vitiligo onset or treatment. Further research including larger studies and better investigations is needed to clarify its impact on melanocyte function and identify potential risk factors for pigmentary changes in patients receiving dupilumab.

Abstract N°: ID-681

Topic: Biologics, immunotherapy, targeted therapy

Anti-TNF–induced paradoxical psoriasis in a patient treated for inflammatory arthritis: a clinicopathological case report

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Introduction

Paradoxical psoriasis is a recognized adverse effect of tumor necrosis factor (TNF) inhibitors. It may present with atypical clinical and histopathological features, creating diagnostic challenges in patients treated for inflammatory rheumatic diseases and often requiring therapeutic adjustment.

Materials and Methods

We report the case of a middle-aged man treated with adalimumab for chronic inflammatory arthritis, without previous personal history of psoriasis. Methotrexate had been discontinued because of hepatotoxicity. The patient presented with a three-week history of multiple pruritic annular erythematous plaques involving the trunk and extremities.

A complete clinical, dermoscopic, and histopathological evaluation was performed. Differential diagnoses included psoriasis, subacute cutaneous lupus erythematosus, dermatophytosis, and cutaneous T-cell lymphoma. Skin biopsy, periodic acid–Schiff staining, and direct immunofluorescence were carried out.

Results

Clinical examination showed multiple well-demarcated erythematous psoriasiform plaques on the trunk and limbs. Dermoscopy revealed dotted vessels and peripheral scaling.

Histopathology demonstrated epidermal acanthosis, hyperkeratosis with parakeratosis, and a superficial perivascular lymphohistiocytic infiltrate with mild epidermal exocytosis. Periodic acid–Schiff staining was negative and direct immunofluorescence showed no immune deposits, supporting the diagnosis of paradoxical psoriasiform dermatitis induced by anti-TNF therapy.

Topical corticosteroids were initiated and adalimumab was discontinued. After multidisciplinary discussion, treatment was switched to an interleukin-23 inhibitor. Complete clinical resolution of the lesions was achieved after induction and maintenance therapy.

Conclusions

Paradoxical psoriasis is an important adverse effect of anti-TNF therapy that may occur in patients without prior psoriasis. Atypical clinical

presentation requires careful clinicopathological correlation. Early recognition and therapeutic switching to a biologic agent with a different mechanism of action can result in rapid and complete remission.

Abstract N°: ID-692

Topic: Biologics, immunotherapy, targeted therapy

An Atypical Prurigo Nodularis-Like Presentation of Anti–PD-1–Induced Late-Onset Bullous Pemphigoid

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Introduction

Programmed cell death protein-1 (PD-1) inhibitors, which are widely used immune checkpoint inhibitors (ICIs) in the treatment of various malignancies, are most commonly associated with cutaneous adverse events as the earliest and most frequent complications. These dermatologic events may present with a broad spectrum of clinical manifestations, ranging from acute-onset adverse events such as toxic epidermal necrolysis to late-onset events, including alopecia. Bullous pemphigoid is typically reported within 3-4 months after treatment initiation. Although cases of bullous pemphigoid developing as late as three years after anti–PD-1/PD-L1 therapy have been reported, the median time to blister onset is 27.5 weeks after treatment initiation.

Materials and Methods

Herein, we report a case of a patient with non-small cell lung cancer who had been receiving PD-1 inhibitor therapy for three years (40th cycle) and initially developed prurigo nodularis–like lesions, followed by blister formation.

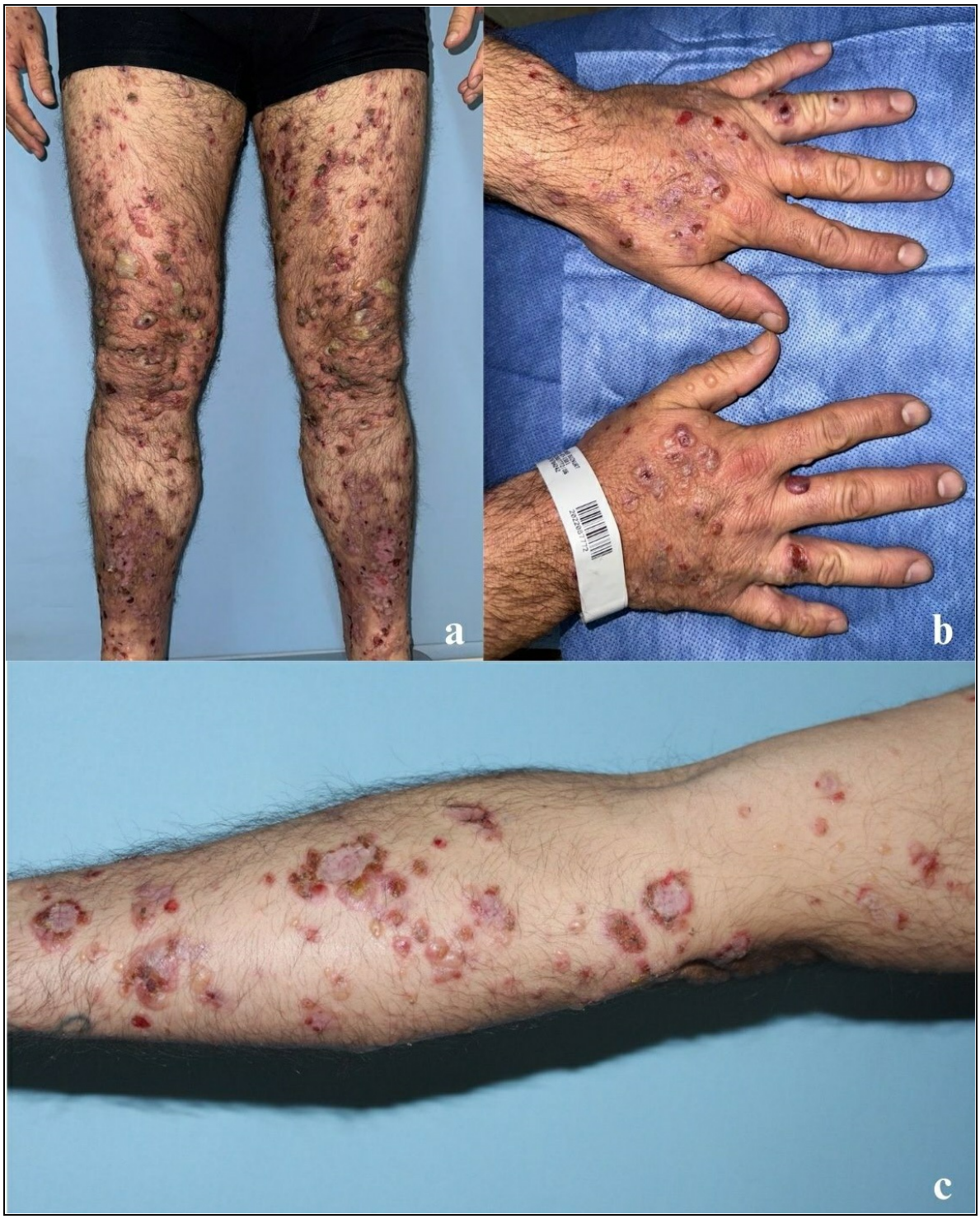

Figure 1. Clinical images. (a) Lichenified violaceous plaques with serous bullae on the legs. (b) Excoriated plaques with hemorrhagic bullae on the hands. (c) Erythematous plaques with annular blisters on the arms.

Results

A 43-year-old man with stage IV non-small cell lung cancer (poorly differentiated adenocarcinoma), who had been receiving anti–PD-1 immunotherapy for three years, presented with mildly generalized pruritus, predominantly involving the anterior tibial regions. The pruritus started during the 40th cycle of immunotherapy. Dermatologic examination revealed lichenified, excoriated violaceous plaques consistent with prurigo nodularis. Due to the nonspecific appearance of the lesions, scabies was considered in the differential diagnosis, and empirical treatment with topical permethrin and antihistamines was initiated, without significant improvement. Laboratory evaluation revealed a markedly elevated total IgE level (4163 kU/L), while other hematologic and biochemical parameters were within normal limits. Despite exclusion of infectious and parasitic causes, the symptoms progressed. During follow-up, at the 43rd cycle of anti–PD-1 therapy, new bullous lesions developed, accompanied by excoriated plaques (Figure 1a,b,c). Histopathologic examination of a punch biopsy specimen obtained from the antecubital fossa demonstrated focal subepidermal clefting and eosinophil-rich perivascular inflammation (Figure 2a,b,c). Direct immunofluorescence revealed linear IgG and C3 deposition along

the basement membrane zone, consistent with bullous pemphigoid (Figure 2d). Following multidisciplinary evaluation, anti–PD-1 therapy was temporarily discontinued, and systemic corticosteroid therapy was initiated. The patient showed rapid clinical improvement and was placed under close follow-up.

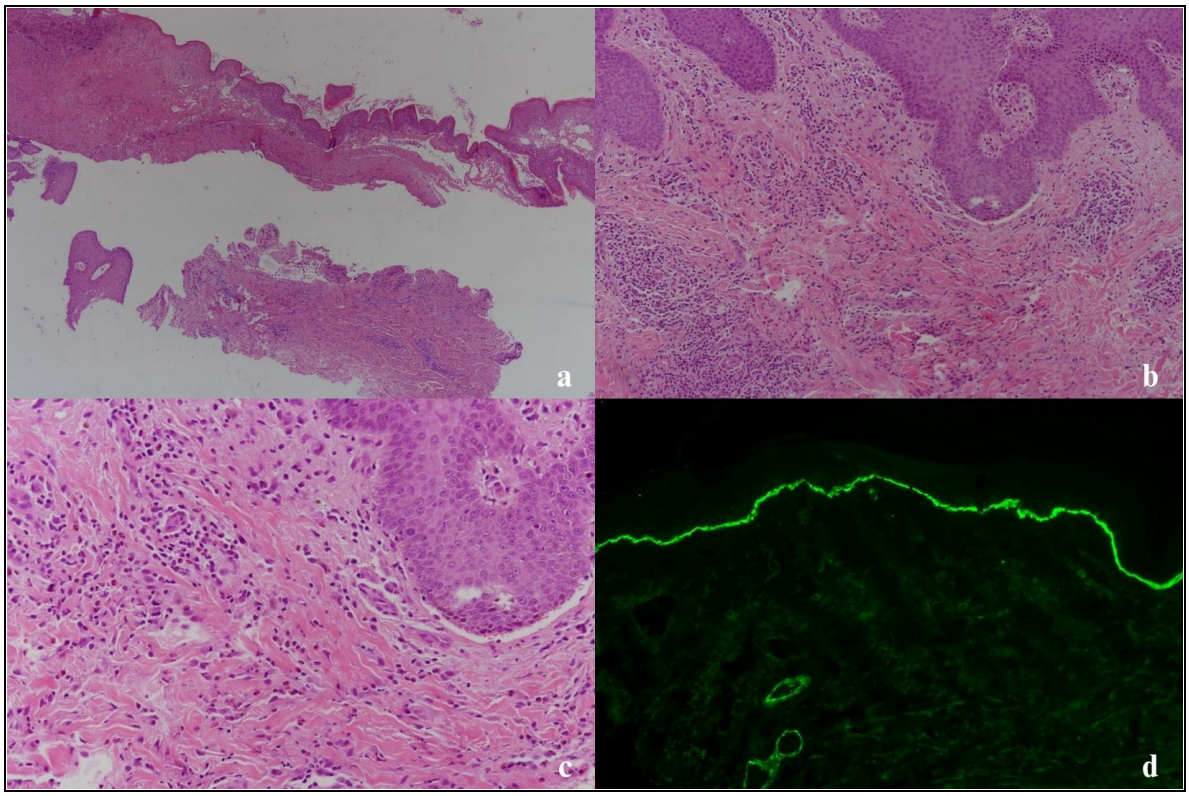

Figure 2. Histopathologic and immunofluorescence findings. (a) H&E, ×40: subepidermal blister. (b,c) H&E, ×100: focal subepidermal blister with mixed inflammatory infiltrate. (d) DIF, ×40: linear IgG and C3 deposition at the dermoepidermal junction.

Conclusions

Bullous pemphigoid is an autoimmune subepidermal blistering disorder characterized by autoantibodies against hemidesmosomal proteins. In recent years, its association with immune checkpoint inhibitors, particularly PD-1/PD-L1 inhibitors, has been increasingly recognized, although it remains a relatively rare adverse event. Cutaneous adverse events related to ICIs usually occur within the first few months after treatment initiation, and most reported cases of bullous pemphigoid develop 3-4 months after treatment initiation. Although ICI-associated bullous pemphigoid is generally considered an adverse event, only a limited number of late-onset cases have been reported. Clinically, bullous pemphigoid most commonly presents with tense bullae. However, our patient initially exhibited a prurigo nodularis–like morphology, which represents a rare reported clinical variant. This case highlights a late-onset presentation with atypical morphology and contributes to the existing literature. Taken together, these findings emphasize the long-term clinical implications of immunotherapy-related cutaneous adverse events and highlight important considerations for patient management and follow-up. Although bullous pemphigoid is a well-recognized complication of immune checkpoint inhibitor therapy, clinicians should be aware that bullous pemphigoid-like cutaneous adverse events may develop even at very late stages of treatment.

Abstract N°: ID-758

Topic: Biologics, immunotherapy, targeted therapy

Clinical Predictors of Omalizumab Dose Requirement and Corticosteroid Dependence in Bullous Pemphigoid: A Real-World Cohort Study

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Introduction
Bullous pemphigoid (BP) is a severe autoimmune blistering disease of the elderly, often requiring prolonged systemic corticosteroids. Omalizumab has emerged as an effective steroid-sparing treatment; however, predictors of treatment intensity and clinical response remain unclear.

Materials and Methods
We retrospectively analyzed 22 consecutive BP patients treated with omalizumab at a tertiary dermatology center. Demographics, comorbidities, disease distribution, mucosal involvement, BPDAI and pruritus VAS scores, laboratory parameters (IgE, eosinophils, CRP, LDH), systemic corticosteroid use, and omalizumab dosing (300 mg vs 450–600 mg/month) were recorded. Within-group changes were analyzed using repeated-measures models or Wilcoxon tests, and between-group comparisons using Mann–Whitney U or Fisher's exact tests.

Results
Mean age was 74.23 ± 10.26 years and 68.2% were female. Diabetes mellitus was present in 77.3%, cardiovascular disease in 63.6%, hyperlipidemia in 81.8%, and 54.5% had prior DPP-4 inhibitor (gliptin) exposure. Mucosal involvement was observed in 45.5%, scalp involvement in 22.7%, extremity involvement in 95.5%, and trunk involvement in 90.9%.

Mean disease duration was 49 months, with a 5.7-month interval from disease onset to treatment initiation. Mean BPDAI and VAS scores improved significantly from 66.9 and 23.3 at baseline to 13.8 and 6.1 after treatment ($p < 0.001$ for both). Patients received a mean of 15 omalizumab injections.

Baseline CRP levels were significantly higher in patients requiring high-dose omalizumab compared with those treated with standard-dose therapy (adjusted mean 17.02 vs 8.17 mg/L; $p = 0.045$). Total IgE levels showed a significant time-dependent increase during omalizumab therapy, rising from 520.5 IU/mL at treatment initiation to 1355.6 IU/mL at last follow-up ($p = 0.002$).

Patients with prior gliptin exposure exhibited a distinct clinical phenotype. Baseline pruritus severity was higher in this group ($p < 0.017$), and although pruritus improved significantly in both groups, the magnitude of VAS reduction was greater in gliptin-exposed patients (26.5→8.25 vs 19.5→3.60; $p < 0.05$). These patients also required lower systemic corticosteroid doses than gliptin-naïve patients (0.33 mg vs 3.40 mg; $p = 0.041$) and showed a higher prevalence of chronic kidney disease (58.3%), cardiovascular disease (91.7%), and scalp involvement (41.7%).

Systemic corticosteroids were discontinued after a mean of 8 omalizumab doses, with a mean steroid exposure of 11.8

months. Dose escalation to 450–600 mg/month was required in 31.8% of patients, but reductions in BPDAI and VAS were similar between standard- and high-dose groups ($p > 0.05$). Mucosal involvement was not associated with corticosteroid requirement.

Table 1. Within-group changes in pruritus severity (VAS) according to prior DPP-4 inhibitor (gliptin) exposure

Group	Baseline VAS	Last VAS	p (within-group)
Gliptin (+)	26.5	8.25	<0.001
Gliptin (−)	19.5	3.60	<0.001

The magnitude of VAS improvement was significantly greater in the gliptin-exposed group ($p \leq 0.05$).

Conclusions

Omalizumab is an effective and steroid-sparing treatment for bullous pemphigoid regardless of dose. Elevated baseline CRP identifies patients requiring dose escalation, while prior DPP-4 inhibitor exposure defines a pruritus-dominant phenotype with enhanced treatment response. Mucosal involvement does not predict corticosteroid dependence.

Abstract N°: ID-810

Topic: Biologics, immunotherapy, targeted therapy

Safety of guselkumab treatment in patients with moderate-to-severe plaque psoriasis combined with latent tuberculosis or inactive hepatitis B virus infection: a retrospective multicenter observational study

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Introduction
The infection risk of biologics requires special attention in patients with psoriasis combined with latent tuberculosis infection (LTBI) or inactive hepatitis B virus (HBV) infection.The burden of tuberculosis and HBV is particularly high in the Chinese mainland. There is currently a lack of evidence on the safety of biologics targeting inter-leukin (IL) IL-23 in Chinese patients with psoriasis combined with LTBI or inactive HBV infection.

Materials and Methods
This retrospective, multicenter, observational study was conducted in three centers in China. In total, 220 Adult patients with moderate to severe plaque psoriasis were treated with guselkumab for 64 weeks. Screening of tuberculosis and hepatitis were performed at baseline and week 64.

Results
In this multicenter study, 41 patients with psoriasis and LTBI and 53 patients with psoriasis and inactive HBV infection were treated with guselkumab. Furthermore,14 patients with inactive HBV infection or LTBI did not receive antituberculosis or antiviral hepatitis B prophylaxis during treatment. During the 1-year follow-up period, HBV reactivation and LTBI were not observed in treatment group.

Conclusions
This preliminary study confirms the safety of guselkumab in Chinese patients with psoriasis combined with LTBI or inactive HBV infection. Further validation in other groups of patients of different ethnic back- grounds will be important to provide evidence for better biologics options in these psoriasis patients.

Abstract N°: ID-937

Topic: Biologics, immunotherapy, targeted therapy

JAK Inhibitors in Lichen Planus and Folliculitis Decalvans

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Introduction

Janus kinases (JAKs) are intracellular protein tyrosine kinases that associate with type I and type II transmembrane cytokine receptors, transmitting signals from a wide range of cytokines and growth factors. These signaling pathways play key roles in normal immune function as well as in autoimmune disorders. The JAK–STAT pathway consists of a transmembrane receptor associated with an effector protein kinase known as JAK and an intracellular signaling protein called STAT. Involved in both inflammatory and autoimmune mechanisms, the JAK–STAT pathway has emerged as a key therapeutic target, as its modulation can help suppress pathological immune activation. Lichen planus (LP) is a chronic inflammatory disease marked by a dense, band-like infiltrate of T lymphocytes in the superficial dermis. Folliculitis decalvans is a rare chronic inflammatory disorder of the scalp hair follicles. Progressive inflammation leads to hair loss and subsequent scarring, resulting in permanent cicatricial alopecia.

Materials and Methods

A narrative review of the literature was conducted, focusing on published studies, case reports, and clinical data evaluating the use of JAK inhibitors in LP and FD. Relevant articles addressing pathogenesis, mechanism of action, clinical outcomes, and safety were analyzed.

Results

Studies and evidences suggest that JAK inhibitors may reduce inflammatory activity in LP by modulating Tcell mediated immune responses. Limited but growing data also indicate potential benefit in FD through suppression of inflammatory pathways involved in follicular destruction. Reported outcomes include clinical improvement in erythema, scaling, pruritus, and disease progression, with an acceptable safety profile in most cases.

Conclusions

JAK inhibitors represent a promising therapeutic option for selected patients with LP and FD resistant to treatment. Although current evidence is limited and mostly based on small studies and case reports, the mechanistic rationale and early clinical outcomes support further investigation through larger, controlled trials.

Abstract N°: ID-943

Topic: Biologics, immunotherapy, targeted therapy

Acneiform eruption secondary to upadacitinib in ulcerative colitis: two cases and dermatological management

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Introduction

Upadacitinib is a selective Janus kinase 1 (JAK1) inhibitor increasingly used in inflammatory diseases, including ulcerative colitis. Acneiform and rosaceiform eruptions have been reported as cutaneous adverse events, with variable clinical presentation and time to onset.

Materials and Methods

We report two clinical cases of women with ulcerative colitis who developed facial acneiform eruptions after initiation of upadacitinib. Clinical presentation, treatment, and outcomes were analysed.

Results

The first case involves a 35-year-old woman with ulcerative colitis treated with upadacitinib 30 mg/day who, two months after the initiation of the treatment, developed inflammatory facial papules associated with pruritus and a burning sensation. A progressive worsening was subsequently observed, with an increase in the number of lesions and with development of eczematous plaques, predominantly affecting the frontal, perioral, and preauricular regions, leading to dermatological evaluation. Topical treatment with fusidic acid 2% cream and hydrocortisone 1% cream twice daily was initiated, in combination with oral doxycycline 100 mg every 12 hours for one week, followed by 100 mg daily. Due to gastrointestinal intolerance, oral doxycycline was discontinued after three weeks and replaced by oral azithromycin 250 mg for three consecutive days per week for eight weeks, associated with topical erythromycin 2% cream. At follow-up, complete resolution of papules and pustules was observed, with persistence only of centrofacial erythema consistent with a rosaceiform phenotype.

The second case concerns a 37-year-old woman with ulcerative colitis who started treatment with upadacitinib 45 mg/day and, after one week of treatment, developed a facial acneiform eruption characterised by closed comedones localised on the chin and frontal region. Topical treatment with azelaic acid 20% cream once daily was initiated, with evident clinical improvement at two-month follow-up.

Conclusions

These cases demonstrate that acneiform eruption associated with upadacitinib may present with different temporal patterns and degrees of severity, and that targeted dermatological management allows effective control of cutaneous manifestations without the need to discontinue the drug in most cases.

Abstract N°: ID-946

Topic: Biologics, immunotherapy, targeted therapy

Trastuzumab Deruxtecan-Associated Vitiligo Treated with Topical Janus Kinase Inhibitor in Human Epidermal Growth Factor Receptor 2-Low Metastatic Breast Cancer: A Case Report

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Introduction

Trastuzumab deruxtecan (T-DXd) is an antibody–drug conjugate composed of a humanized anti–Human Epidermal Growth Factor Receptor 2 (anti-HER2) monoclonal antibody linked via a cleavable linker to a potent topoisomerase I inhibitor. It is approved for the treatment of unresectable or metastatic HER2-low breast cancer, including hormone receptor–positive disease, after progression on standard therapies. The most frequently reported adverse events include nausea, fatigue, myelosuppression, alopecia, gastrointestinal toxicity, and interstitial lung disease. Cutaneous adverse effects are usually mild and nonspecific.

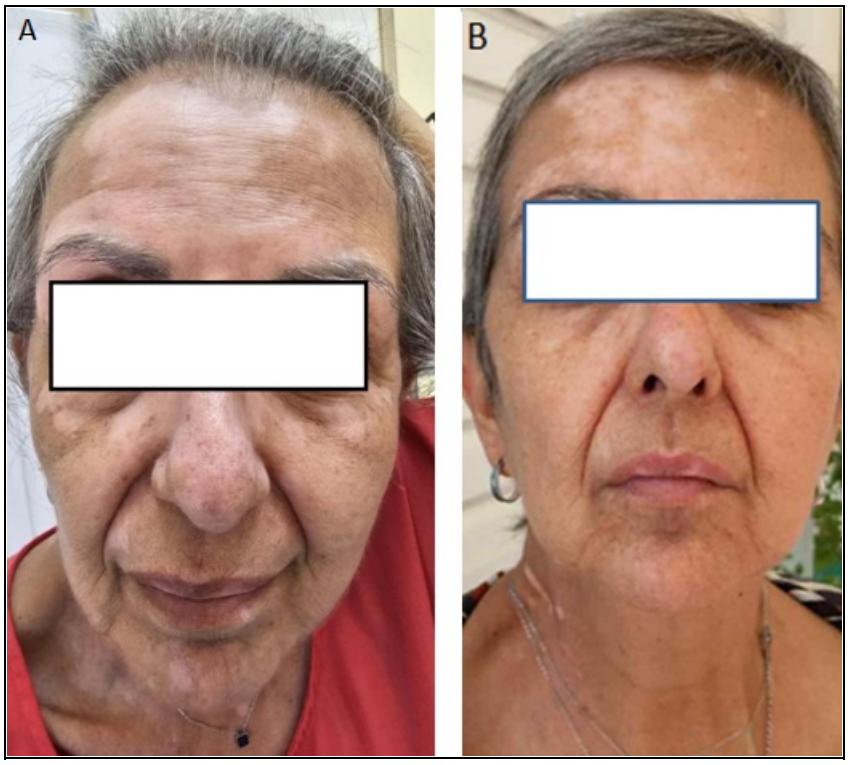
Vitiligo is an acquired depigmenting disorder caused by the loss of melanocytes, commonly related to autoimmune processes or immune-modifying treatments. To our knowledge, vitiligo has not previously been reported as an adverse effect of T-DXd. We describe a case of vitiligo occurring shortly after initiation of T-DXd in a patient with metastatic breast cancer

Materials and Methods

Clinical data were collected retrospectively from the medical records of a 60-year-old woman with metastatic HER2-low breast cancer and vitiligo treated with T-DXd. Dermatological examination, therapeutic interventions, and clinical follow-up were documented over a two-year period. The causality between drug exposure and skin manifestations was assessed based on temporal relationship, clinical evolution, and exclusion of alternative etiologies

Results

A 60-year-old woman with estrogen receptor–positive, HER2-low metastatic breast cancer with liver involvement was started on T-DXd at a dose of 5.4 mg/kg every three weeks. One week after the first treatment cycle, she developed well-defined depigmented patches on the face, suggestive of vitiligo. She had no personal or family history of vitiligo or autoimmune disease. She was initially treated with topical mometasone furoate cream, without improvement. One year after starting topical corticosteroids, vitiligo had spread to the neck, chest, upper back, and upper limbs. Topical calcineurin inhibitor therapy with ointment tacrolimus 0.03% was added twice weekly but proved ineffective. Given the progression of the disease, topical ruxolitinib cream 1.5% was subsequently initiated twice daily. Ruxolitinib is a JAK1/2 inhibitor that suppresses interferon-γ–mediated immune pathways implicated in the melanocyte destruction. After 12 weeks, perifollicular repigmentation was observed with partial repigmentation over the subsequent months. Treatment was well tolerated, and allowed the continuation of T-DXd therapy



Vitiligo-like depigmentation following trastuzumab deruxtecan therapy A) with partial repigmentation B) following ruxolitinib treatment

Conclusions

This case suggests a potential association between T-DXd and vitiligo, given its rapid onset following the initiation of T-DXd treatment and progressive course. The response to topical ruxolitinib indicates a JAK-STAT–mediated mechanism. Early dermatologic intervention may help manage this rare adverse effect and allow continuation of anticancer therapy. Further reports are needed to clarify its pathophysiology

Abstract N°: ID-950

Topic: Biologics, immunotherapy, targeted therapy

Six Years of Biologic Therapy for Moderate to Severe Plaque Psoriasis: A Real-World, Single-Center Study

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Introduction

Psoriasis is a chronic, immune-mediated systemic disease affecting more than 125 million people worldwide. Patients with moderate to severe disease often require long-term systemic therapy to maintain stable disease control. Although psoriasis is typically manageable rather than curable, modern treatment strategies, including biologic therapy, can achieve sustained symptom control over prolonged periods. This study aimed to evaluate the real-world effectiveness, drug survival and switching patterns of biologic therapy in patients with moderate-to-severe psoriasis.

Materials and Methods

A retrospective study included 210 patients with moderate to severe psoriasis, who were treated with biological therapy between 2018 and 2023. Baseline data included demographics, comorbidities, prior treatments, Psoriasis Area and Severity Index (PASI) scores, Dermatological quality of life index (DLQI) scores and treatment-related adverse events.

Results

Of the 210 patients, 60.0% were male (n = 126), with a mean age of 48.3 ± 13.6 years in men (range 16–74) and 48.4 ± 13.6 years in women (range 17–79). Mean PASI at initiation of biologic therapy was 15.0 ± 8.1 and decreased to 3.3 ± 4.7 at 1 year, 2.7 ± 4.0 at 3 years, and 2.8 ± 3.3 at 5 years.

Drug discontinuation varied between therapies: etanercept had a higher hazard of discontinuation than ustekinumab (hazard ratio (HR) 2.55, 95% confidence interval (CI) 1.17–5.52; p = 0.018), infliximab (HR 0.36, 95% CI 0.13–0.97; p = 0.043) and adalimumab (HR 0.47, 95% CI 0.23–0.98; p = 0.045) (**Figure 1**).

Overall, 154 patients remained on their initial drug without switching. The most common reason for switching was insufficient efficacy (93.8%), with the most frequent switch from adalimumab to ustekinumab (35.7%, n = 20).

Five-year drug survival rates were highest for risankizumab (95.8%) and guselkumab (88.0%), intermediate for adalimumab (56.0%), ustekinumab (43.5%), secukinumab (42.9%) and infliximab (33.3%), and lowest for etanercept (10.0%) and ixekizumab (0%).

Median time to achieve ≥50% reduction from the baseline in PASI scores (PASI 50) was 4.4 weeks for adalimumab and 13.0 weeks for guselkumab, risankizumab and ustekinumab. The highest PASI 50 response rates were observed with risankizumab, infliximab, and ixekizumab (100%; $n = 24$, $n = 12$, and $n = 2$, respectively). ≥75% reduction from the baseline in PASI scores (PASI 75) were highest for infliximab (91.7%, $n = 11$) and risankizumab (87.5%, $n = 21$), with lower rates for guselkumab (76.0%, $n = 19$), adalimumab (72.6%, $n = 61$) and secukinumab (71.4%, $n = 5$).

Median time to achieve ≥90% reduction from the baseline in PASI scores (PASI 90) was 26.1 weeks for adalimumab, risankizumab, and ustekinumab, and 39.1 weeks for guselkumab. PASI 90 response rates were higher for guselkumab (68.0%, $n = 17$) and infliximab (66.7%, $n = 8$) and lower for risankizumab (62.5%, $n = 15$), secukinumab (57.1%, $n = 4$), and

adalimumab (53.6%, *n* = 45). Median time to reach ≥100% reduction from the baseline in PASI scores (PASI 100) ranged from 39.1 weeks (adalimumab, guselkumab, risankizumab) to 52.1 weeks (ustekinumab). PASI 100 response rates were similar across adalimumab (38.1%, *n* = 32), risankizumab (37.5%, *n* = 9), and guselkumab (36.0%, *n* = 9).

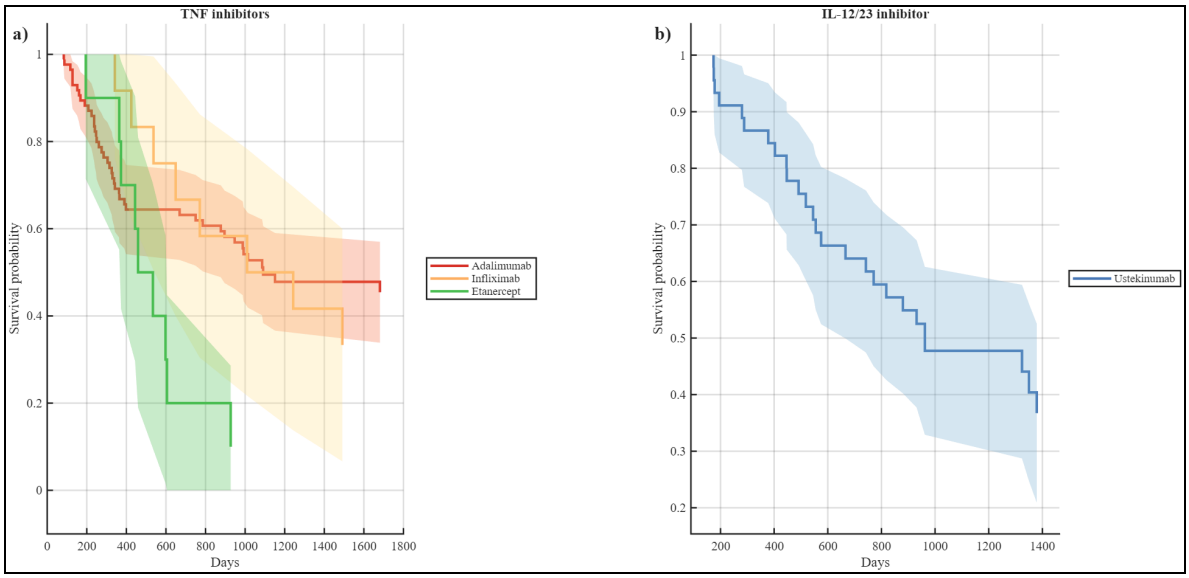

Figure 1. Kaplan–Meier drug survival curves grouped by biologic classes of all patients, a) Tumor necrosis factor (TNF) inhibitors, b) Interleukin (IL)-12/23 inhibitors.

Conclusions

In our study, biologic therapy was associated with substantial improvement over time, with PASI decreasing markedly across follow-up. While average PASI reductions were similar across drugs, PASI trajectories varied by treatment over time. Drug survival was initially high for all agents but separated over time, with etanercept showing the lowest long-term persistence and a higher risk of discontinuation. Switching was predominantly driven by insufficient efficacy and often restored response in subsequent therapies.

Abstract N°: ID-1073

Topic: Biologics, immunotherapy, targeted therapy

Successful Treatment of Concomitant Psoriasis and Morphea With Bimekizumab After Failure of Conventional Therapies: A Case Report

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Introduction

Psoriasis is a chronic immune-mediated inflammatory skin disease, whereas morphea is a localized fibrosing disorder with distinct immunopathogenic mechanisms. The coexistence of psoriasis and morphea in the same patient is rare and may pose diagnostic and therapeutic challenges, particularly in cases refractory to conventional systemic treatments. Evidence regarding biologic therapies effective for both conditions remains limited.

Materials and Methods

A 42-year-old female with a history of psoriasis since the age of five had been followed at an external center prior to her first presentation to our clinic in 2019. She presented with annular, atrophic plaques on the trunk. Skin biopsies were obtained from these atrophic plaques with preliminary diagnoses of mycosis fungoides and morphea, as well as from psoriatic plaques to confirm the diagnosis of psoriasis. Histopathological examination revealed findings consistent with morphea in the atrophic plaques and psoriasis in the psoriatic lesions.Based on these findings, methotrexate therapy was initiated in 2020 at a dose of 7.5 mg/week and subsequently increased to 10 mg/week during follow-up. The patient was lost to follow-up in 2021. She re-presented in May 2025 with exacerbation of both morphea and psoriatic lesions, reporting irregular methotrexate use at doses up to 15 mg/week. According to the patient's history, the cumulative methotrexate dose was approximately 1.5 g. There was no nail or joint involvement. Dermatological examination revealed violaceous, shiny plaques with central atrophy consistent with morphea, along with active psoriatic plaques on the trunk. The Psoriasis Area and Severity Index (PASI) was 6.7, and the Dermatology Life Quality Index (DLQI) score was 18.

Results

Due to active disease under conventional therapy and significant impairment in quality of life, transition to biologic therapy was planned. Given the patient's difficulty with treatment adherence and reduced quality of life related to frequent injections with methotrexate, bimekizumab was initiated following appropriate screening. The treatment regimen consisted of 320 mg administered subcutaneously every four weeks for the first 16 weeks, followed by maintenance dosing every eight weeks.At the second month of follow-up, complete clearance of psoriatic lesions was achieved (PASI 0), along with a marked reduction in induration and atrophy of existing morphea lesions. During the eight-month follow-up period, no new morphea lesions developed, and improvement in both conditions was sustained. No adverse events or laboratory abnormalities were observed.

Conclusions

This case demonstrates the successful and well-tolerated use of bimekizumab in a patient with long-standing psoriasis and concomitant morphea refractory to conventional systemic therapy. Dual inhibition of IL-17A and IL-17F may

represent a promising therapeutic option for managing overlapping inflammatory and fibrosing skin disorders while preventing new morphea lesion development.

Abstract N°: ID-1157

Topic: Biologics, immunotherapy, targeted therapy

Targeted immunomodulatory and biologic therapies for treatment-resistant granuloma annulare: a systematic review

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Introduction
Granuloma annulare (GA) is a chronic, inflammatory granulomatous skin disease. The incidence of GA is estimated at 0.04%, with a predilection for women during their fifth decade of life. In many cases, particularly in generalized forms, GA is resistant to currently used standard therapies, which are largely based on case reports and clinical experience, and no evidence-based guideline exists for the management of generalized GA. Increasing insight into GA pathogenesis, including the role of Th1- and Th2-mediated cytokine signaling via the JAK-STAT pathway, supports the rationale for targeted immunomodulatory and biologic therapies. The aim of this systematic review was to summarize and critically assess the available literature and evidence on innovative immunomodulatory and biologic treatment options for GA.

Materials and Methods
This systematic review was conducted according to PRISMA guidelines and registered in PROSPERO. A search of PubMed, Embase and Cochrane databases was performed for studies published between January 2020 and December 2025, using relevant MeSH terms and keywords related to GA and targeted or biologic therapies. Original studies, case reports, and case series involving adults treated with immunomodulatory or biologic agents were included. Eligibility assessment and data extraction were performed independently by two reviewers.

Results
Seventeen publications were included, comprising 42 patients with GA treated with biologic or targeted therapies. The majority were female (71%), aged 26-79 years, with generalized disease refractory to prior treatment in most cases. Janus kinase (JAK) inhibitors were the most frequently administered therapy, used in 36/42 patients (87%). The analysis included tofacitinib, upadacitinib, abrocitinib, baricitinib, and deucravacitinib. Most patients treated with JAK inhibitors achieved significant clinical improvement, typically within 2-6 weeks, with complete or near-complete clearance reported in the majority of cases. Biologic agents were used less frequently. Tumor necrosis factor-alpha inhibitors were administered in three patients and were also associated with clinical improvement, although relapses were reported. Dupilumab, apremilast, and tildrakizumab were each reported in single patients, with variable efficacy. Adverse events were uncommon and mild. Laboratory abnormalities, mainly hyperlipidemia, were reported in three patients, with pharmacologic management required in one case. No serious adverse events leading to treatment discontinuation were observed.

Conclusions
Targeted biologic and immunomodulatory therapies, particularly JAK inhibitors, appear to be effective and well-tolerated options for patients with GA resistant to conventional treatment. Most reported patients achieved relevant clinical improvement, with complete or near-complete clearance. However, current evidence is limited to case reports and

small case series, emphasizing the need for further research, especially prospective controlled studies

Abstract N°: ID-1169

Topic: Biologics, immunotherapy, targeted therapy

Intralesional Purified Protein Derivative as Immune-Targeted Therapy for Condyloma Acuminata: Clinical Evidence of Cell-Mediated HPV Clearance

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Introduction
Condyloma acuminata remains a therapeutic challenge due to persistent Human Papillomavirus (HPV) infection and high recurrence rates following destructive treatments. Conventional modalities primarily eliminate visible lesions without addressing the underlying viral persistence. Clearance of HPV is largely dependent on effective cell-mediated immunity (CMI), particularly Th1-driven cytotoxic responses. Intralesional immunotherapy has emerged as an immune-targeted approach capable of inducing both local and systemic antiviral immune activation. Purified Protein Derivative (PPD), a well-characterized delayed-type hypersensitivity antigen, has demonstrated promising results in the treatment of viral warts.

Materials and Methods
We report a young female adult aged 28 years presenting with multiple anogenital verrucous warts involving the vaginal introitus and surrounding genital area. Clinical examination revealed multiple verrucous nodules with a positive acetowhite reaction. Laboratory and serological evaluations tested negative for other sexually transmitted infections. Following confirmation of tuberculin sensitization, intralesional PPD (0.2 mL) was administered weekly into the largest lesion without adjunctive destructive or topical therapy. Treatment was administered for 5 weeks. Clinical response, lesion regression, and adverse events were systematically evaluated.

Results
Clinical regression was observed within the first week of treatment, with progressive reduction in lesion size and number over subsequent sessions. By week 5, near-complete resolution was achieved, with only minimal residual lesions after five treatment sessions. No new lesions developed during therapy. Treatment was well tolerated, with only mild, transient injection-site discomfort and no systemic adverse effects. The rapid and sustained response, including regression beyond the injected site, supports the induction of a systemic cell-mediated immune response rather than a purely local effect. Complete clinical clearance was achieved and maintained, and at 3-month post-treatment follow-up, the patient remained asymptomatic with no evidence of recurrence or development of new anogenital lesions.

Conclusions
Intralesional purified protein derivative is an effective and safe immune-targeted therapeutic option for condyloma acuminata. PPD enhances cell-mediated immunity by acting as a delayed-type hypersensitivity antigen, thereby inducing a Th1-dominant immune response with increased production of interferon-γ and interleukin-2. This immune activation facilitates cytotoxic T lymphocyte and macrophage mediated clearance of HPV infected keratinocytes, resulting in both

local lesion regression and systemic clearance of distant untreated lesions. Intralesional PPD may therefore be considered a valuable therapeutic modality, particularly in patients with recurrent or treatment-resistant anogenital warts. Sustained lesion clearance without recurrence at the 3-month follow-up further highlights the potential of intralesional PPD as an immune-mediated therapeutic option for controlling HPV infection

Abstract N°: ID-1190

Topic: Biologics, immunotherapy, targeted therapy

Paradoxical Alopecia Areata Linked to Adalimumab-atto: case series

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Introduction

Tumor necrosis factor-α (TNF-α) inhibitors are widely used in inflammatory diseases and are generally effective and safe; however, paradoxical immune-mediated adverse events remain incompletely characterized. Adalimumab-atto, a biosimilar of adalimumab, demonstrates comparable efficacy and safety to the originator, yet reports of rare dermatologic paradoxical reactions are scarce. Alopecia areata (AA) is an uncommon and underrecognized paradoxical effect of TNF-α inhibition and has not previously been reported with adalimumab-atto. Borderline paradoxical AA refers to new-onset or worsening AA during TNF-α inhibitor therapy, despite a theoretical immunologic rationale and lack of established therapeutic benefit. A temporal relationship, characteristic trichoscopic findings, and exclusion of alternative causes support a drug-induced mechanism, likely related to disruption of hair follicle immune privilege (1,2). Increased recognition of this phenomenon is essential as biosimilar use expands.

Case Report Objectives and Novelty
- Report the first known case of alopecia areata associated with adalimumab-atto
- Highlight AA as a rare, borderline paradoxical reaction to TNF-α inhibition
- Emphasize diagnostic features supporting a drug-induced mechanism
- Increase clinician awareness of paradoxical reactions with TNF-α biosimilars

Materials and Methods

Patient 1 is a 43-year-old woman with well-controlled hypothyroidism and severe plaque psoriasis (PASI 12) and psoriatic arthritis had a remote history of a single self-resolving episode of patchy alopecia areata (AA) eight years earlier . Family history was notable for vitiligo and psoriasis. She achieved sustained psoriasis control with ustekinumab and narrowband UVB phototherapy. After three years, ustekinumab was discontinued for financial reasons and replaced with adalimumab-atto. Six weeks later, she developed new-onset non-scarring patchy alopecia of the left parietal and vertex scalp, accompanied by worsening psoriasis. Trichoscopy showed yellow and black dots, exclamation-mark hairs, and short vellus hairs without scarring or scale **(Figure 1. A-D).** Adalimumab-atto was discontinued, and topical clobetasol propionate 0.05% ointment was initiated.

Patient 2 is a 10-year-old boy with immune thrombocytopenia was followed for erythrodermic juvenile pityriasis rubra pilaris (PRP). There was no personal or family history of AA, psoriasis, or other autoimmune disease. His disease was refractory to oral corticosteroids, cyclosporine, and acitretin but achieved control for 43 weeks with ustekinumab, which was discontinued due to financial constraints. After relapse, adalimumab-atto was initiated in combination with cyclosporine. Eight weeks later, he developed new-onset patchy, non-scarring alopecia of the vertex scalp without nail or other hair involvement. Trichoscopy revealed yellow dots and short vellus hairs **(Figure 1. E-F).** Adalimumab-atto was continued with close monitoring, cyclosporine was tapered and discontinued, and topical therapy for alopecia with

alternating mometasone furoate lotion and tacrolimus 0.1% ointment was started.

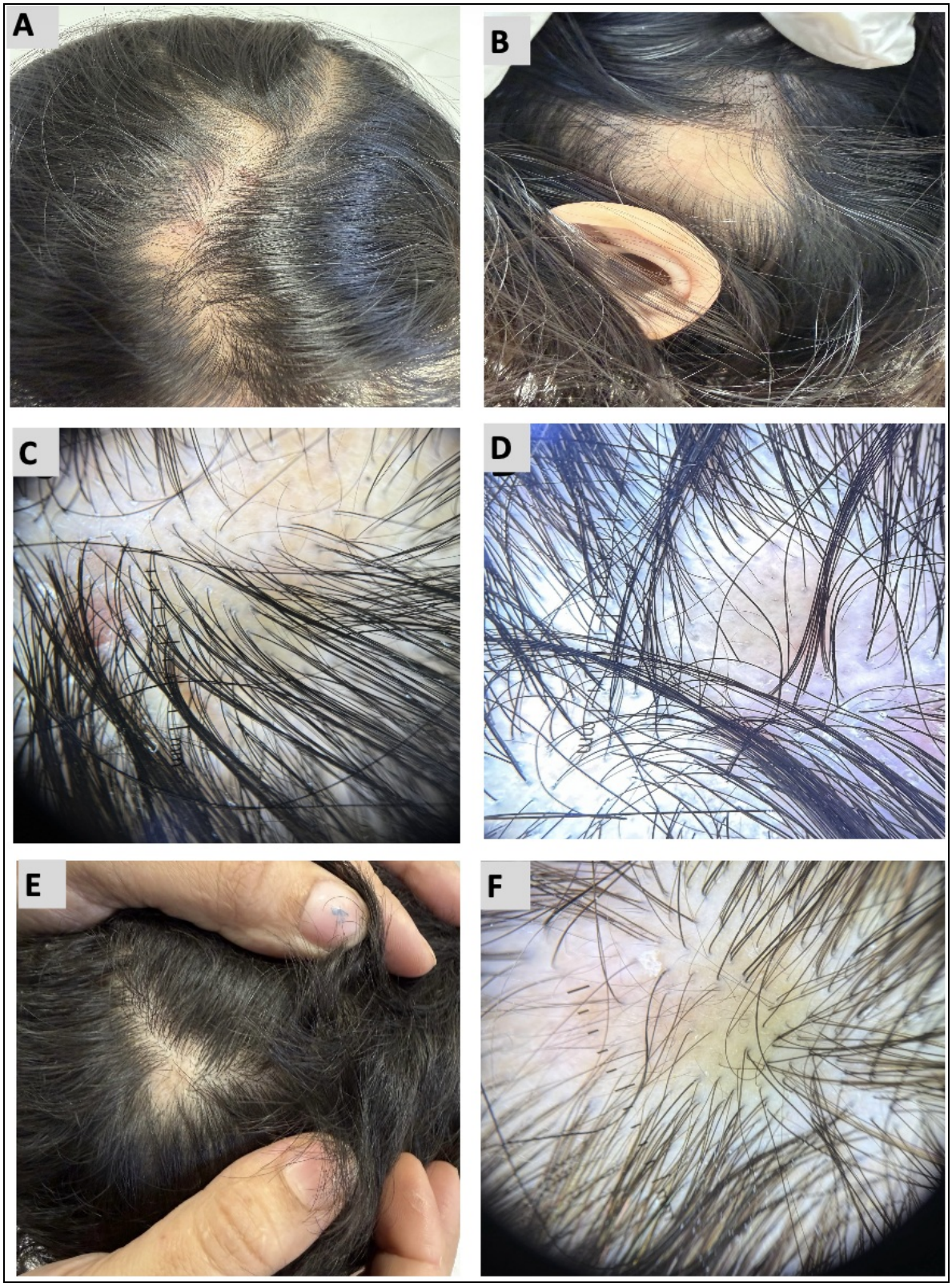

Figure 1. Clinical and trichoscopic features of paradoxical alopecia areata associated with adalimumab-atto. (A–D) Patient 1: Clinical photographs show well-demarcated, non- scarring alopecic patches involving the left parietal and occipital scalp, with smooth, normal-appearing skin, preserved follicular openings, and absence of scale, erythema, or psoriasiform inflammation. Trichoscopic examination reveals multiple yellow dots corresponding to dilated follicular ostia filled with keratin and sebum, along with black dots, exclamation-mark hairs, and short regrowing vellus hairs, consistent with active alopecia areata. (E–F) Patient 2: Clinical image demonstrates a localized, well- circumscribed, non-scarring alopecic patch over the vertex scalp with normal underlying skin and no inflammatory or scaly changes. Trichoscopy shows yellow dots and numerous short vellus hairs, compatible with alopecia areata in an early regrowth phase, without features of psoriasiform or cicatricial alopecia. Examination of the palms reveals diffuse, symmetric, yellow-orange waxy

hyperkeratosis with accentuated skin markings, consistent with palmoplantar involvement of pityriasis rubra pilaris, and the trunk and neck demonstrate controlled pityriasis rubra pilaris at the time of alopecia onset.

Results

N/A

Conclusions

- Borderline paradoxical alopecia areata is a rare but clinically significant immune-mediated adverse effect of TNF-α inhibitors, including the biosimilar adalimumab-atto
- Early recognition of new-onset hair loss enables prompt evaluation and individualized risk–benefit assessment
- Awareness of this paradoxical reaction supports informed therapeutic decision-making and optimized patient outcomes
- Increased clinician recognition is essential as biologic and biosimilar use continues to expand

Abstract N°: ID-1238

Topic: Biologics, immunotherapy, targeted therapy

Late-Onset Erythema Nodosum-like Panniculitis Associated with Nivolumab in Metastatic Malignant Melanoma: A Case Report

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Introduction

Immunotherapies targeting cytotoxic T-lymphocyte–associated antigen 4 (CTLA-4) and the programmed cell death 1 (PD-1) receptor and its ligand (PD-L1) have demonstrated substantial therapeutic benefit in patients with advanced solid malignancies. Despite their clinical efficacy, these agents are frequently associated with immune-related adverse events resulting from nonspecific immune activation. Cutaneous toxicities are among the most common immune-related adverse events. Although rash and pruritus are frequently observed, less common inflammatory dermatologic reactions, including panniculitis, have been increasingly recognized with the expanding use of immune checkpoint inhibitors. Among these, erythema nodosum–like panniculitis remains a particularly rare manifestation, with only a limited number of cases reported in the literature.

Results

A 91-year-old woman with metastatic malignant melanoma had been receiving nivolumab monotherapy every three weeks since April 2024. Following the 28th treatment cycle, she developed painful erythematous subcutaneous nodules localized to the anterior and posterior aspects of the left tibial region. Dermatologic examination revealed tender subcutaneous nodules consistent with panniculitis. No accompanying systemic symptoms, including fever or arthralgia, were observed. Histopathological evaluation demonstrated findings compatible with erythema nodosum. Laboratory investigations, including antinuclear antibodies, C-reactive protein, erythrocyte sedimentation rate, hepatitis profile, tuberculosis screening, serum angiotensin-converting enzyme levels, and streptococcal infection, were all negative. Chest radiography was unremarkable, with no evidence of bilateral hilar lymphadenopathy. There was no history of newly initiated medications or recent infectious episodes.
Erythema nodosum–like panniculitis developed after prolonged exposure to nivolumab, occurring following the 28th treatment cycle. Topical corticosteroid therapy with diflorasone diacetate ointment was administered for four weeks, resulting in partial clinical improvement. Given the absence of systemic symptoms and the limited severity of cutaneous involvement, nivolumab therapy was continued without dose modification or interruption. Upon recurrence of the nodules, treatment was escalated to clobetasol propionate 0.05% cream for four weeks, leading to marked clinical improvement with significant reduction in erythema, pain, and nodularity. No systemic immunosuppressive therapy was required, and no additional immune-related adverse events were observed during follow-up.

Conclusions

This case highlights erythema nodosum-like panniculitis as a rare and underrecognized immune-related cutaneous adverse event associated with nivolumab therapy. Given the limited number of cases reported to date, this presentation adds to the existing literature and underscores the expanding and heterogeneous spectrum of immune checkpoint inhibitor-related dermatologic toxicities. Importantly, this case demonstrates that erythema nodosum-like panniculitis may occur after prolonged exposure to immunotherapy and can be effectively managed with topical corticosteroids, allowing for the continuation of life-prolonging immune checkpoint inhibitor treatment without interruption. Increased awareness of this uncommon adverse event is essential to ensure timely diagnosis, avoid unnecessary treatment discontinuation, and optimize patient outcomes.

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Abstract N°: ID-1299

Topic: Biologics, immunotherapy, targeted therapy

Efficacy and Safety of Oral IL-23 Receptor Blockade With Icotrokinra in Plaque Psoriasis: Meta-analysis of Randomized Controlled Trials

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Introduction

Plaque psoriasis is a chronic, immune-mediated inflammatory skin disease characterized by erythematous, scaly plaques and associated with psychological and systemic comorbidities. Although biologic agents targeting the IL-23 pathway achieve high rates of skin clearance, their use is limited by injectable administration. Advanced oral therapies such as apremilast and deucravacitinib offer greater convenience but demonstrate lower efficacy and tolerability compared with biologics. Icotrokinra is a first-in-class targeted oral peptide that selectively binds the IL-23 receptor, in oral formulation. We performed a meta-analysis of ICONIC-LOAD and ICONIC-ADVANCE 1 and 2 to provide estimates of efficacy and safety and to clarify the therapeutic role of icotrokinra in moderate-to-severe plaque psoriasis.

Materials and Methods

We systematically searched PubMed, Embase, Cochrane for English-language studies involving comparison of treatment effects between Icotrokinra and Placebo, and reported at least one relevant outcome. Three studies met inclusion criteria. Data were pooled using the inverse variance weighting method with a random-effects model in R Studio. Confidence intervals and heterogeneity were assessed, and forest plots were generated to visualise the pooled effect estimates.

Results

Three randomized controlled trials involving 1,555 patients were included. Analysis of Icotrokinra versus placebo at week-16 demonstrated significantly higher IGA 0/1 achievement (RR=7.21; 95% CI: 5.40-9.61; p<0.0001) and IGA 0 responses (RR=22.98; 95% CI: 10.96-48.17; p<0.0001). Scalp-specific IGA 0/1 also favored Icotrokinra (RR=4.08; 95% CI: 3.29-5.06; p<0.0001). For PASI outcomes, PASI75 (RR=6.58; 95% CI: 5.07-8.55), PASI90 (RR=13.44; 95% CI: 8.23-21.95), and PASI100 (RR=31.17; 95% CI: 11.70-83.04) all significantly favored Icotrokinra (all p<0.0001). Total adverse events were comparable (RR=0.90; 95% CI: 0.77-1.05; p=0.18), as were serious adverse events (RR=0.69; 95% CI: 0.27-1.71; p=0.42) and discontinuations due to adverse events (RR=0.72; 95% CI: 0.20-2.59; p=0.62).

Conclusions

In moderate to severe plaque psoriasis, icotrokinra markedly improved IGA and PASI responses including scalp clearance versus placebo, with similar safety profile through 16 weeks. Future work should confirm durability and long-term safety and define comparative effectiveness in head-to-head and real-world studies.

Figure 1a: Patients achieving Investigator's Global Assessment (IGA) 0 or 1

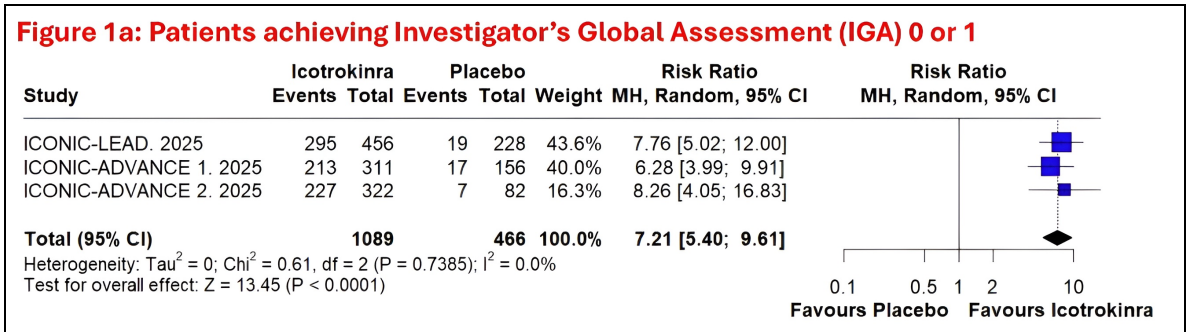


Figure 1b: Patients achieving Investigator's Global Assessment (IGA) 0

Study	Icotrokinra Events	Icotrokinra Total	Placebo Events	Placebo Total	Weight	Risk Ratio MH, Random, 95% CI
ICONIC-LEAD. 2025	152	456	3	228	42.8%	25.33 [8.17; 78.55]
ICONIC-ADVANCE 1. 2025	114	311	3	156	42.9%	19.06 [6.16; 59.01]
ICONIC-ADVANCE 2. 2025	118	322	1	82	14.4%	30.05 [4.26; 211.90]
Total (95% CI)	**1089**		**466**		**100.0%**	**22.98 [10.96; 48.17]**

Heterogeneity: $Tau^2 = 0$; $Chi^2 = 0.21$, df = 2 (P = 0.9005); $I^2 = 0.0\%$
Test for overall effect: Z = 8.30 (P < 0.0001)

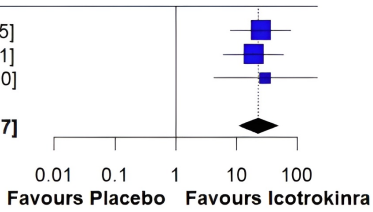


Figure 1c: Patients achieving Scalp-specific Investigator's Global Assessment (IGA) 0 or 1

Study	Icotrokinra Events	Icotrokinra Total	Placebo Events	Placebo Total	Weight	Risk Ratio MH, Random, 95% CI
ICONIC-LEAD. 2025	293	405	30	200	41.0%	4.82 [3.45; 6.74]
ICONIC-ADVANCE 1. 2025	189	261	28	134	40.4%	3.47 [2.47; 4.86]
ICONIC-ADVANCE 2. 2025	199	270	13	71	18.7%	4.03 [2.45; 6.61]
Total (95% CI)	**936**		**405**		**100.0%**	**4.08 [3.29; 5.06]**

Heterogeneity: $Tau^2 = 0$; $Chi^2 = 1.87$, df = 2 (P = 0.3918); $I^2 = 0.0\%$
Test for overall effect: Z = 12.84 (P < 0.0001)

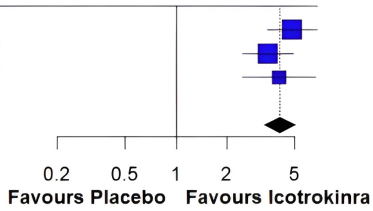


Figure 2a: Patients achieving PASI 75

Study	Icotrokinra Events	Icotrokinra Total	Placebo Events	Placebo Total	Weight	Risk Ratio MH, Random, 95% CI
ICONIC-LEAD. 2025	315	456	25	228	48.8%	6.30 [4.33; 9.17]
ICONIC-ADVANCE 1. 2025	231	311	18	156	35.5%	6.44 [4.15; 9.99]
ICONIC-ADVANCE 2. 2025	249	322	8	82	15.7%	7.93 [4.09; 15.35]
Total (95% CI)	**1089**		**466**		**100.0%**	**6.58 [5.07; 8.55]**

Heterogeneity: $Tau^2 = 0$; $Chi^2 = 0.37$, df = 2 (P = 0.8308); $I^2 = 0.0\%$
Test for overall effect: Z = 14.10 (P < 0.0001)

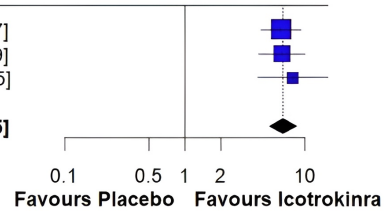


Figure 2b: Patients achieving PASI 90

Study	Icotrokinra Events	Icotrokinra Total	Placebo Events	Placebo Total	Weight	Risk Ratio MH, Random, 95% CI
ICONIC-LEAD. 2025	226	456	10	228	57.7%	11.30 [6.12; 20.86]
ICONIC-ADVANCE 1. 2025	171	311	6	156	36.1%	14.30 [6.48; 31.53]
ICONIC-ADVANCE 2. 2025	184	322	1	82	6.3%	46.86 [6.66; 329.43]
Total (95% CI)	**1089**		**466**		**100.0%**	**13.44 [8.23; 21.95]**

Heterogeneity: $Tau^2 = 0.0107$; $Chi^2 = 2.10$, df = 2 (P = 0.3504); $I^2 = 4.6\%$
Test for overall effect: Z = 10.39 (P < 0.0001)

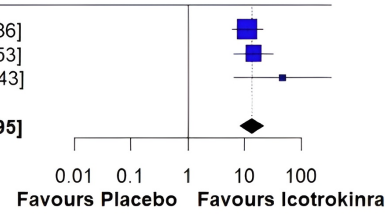


Figure 2c: Patients achieving PASI 100

Study	Icotrokinra Events	Icotrokinra Total	Placebo Events	Placebo Total	Weight	Risk Ratio MH, Random, 95% CI
ICONIC-LEAD. 2025	123	456	1	228	25.0%	61.50 [8.65; 437.26]
ICONIC-ADVANCE 1. 2025	97	311	2	156	49.9%	24.33 [6.08; 97.37]
ICONIC-ADVANCE 2. 2025	102	322	1	82	25.1%	25.98 [3.68; 183.41]
Total (95% CI)	**1089**		**466**		**100.0%**	**31.17 [11.70; 83.04]**

Heterogeneity: $Tau^2 = 0$; $Chi^2 = 0.64$, df = 2 (P = 0.7258); $I^2 = 0.0\%$
Test for overall effect: Z = 6.88 (P < 0.0001)

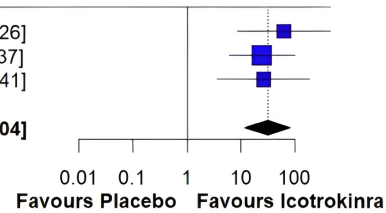


Figure 3a: Adverse events

Study	Icotrokinra Events	Icotrokinra Total	Placebo Events	Placebo Total	Weight	Risk Ratio MH, Random, 95% CI
ICONIC-LEAD. 2025	225	456	112	228	37.9%	1.00 [0.85; 1.18]
ICONIC-ADVANCE 1. 2025	144	310	92	155	35.1%	0.78 [0.66; 0.93]
ICONIC-ADVANCE 2. 2025	159	322	44	82	26.9%	0.92 [0.73; 1.16]
Total (95% CI)	**1088**		**465**		**100.0%**	**0.90 [0.77; 1.05]**

Heterogeneity: $Tau^2 = 0.0101$; $Chi^2 = 4.23$, df = 2 (P = 0.1209); $I^2 = 52.7\%$
Test for overall effect: Z = -1.33 (P = 0.1821)

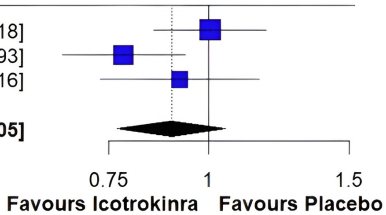


Figure 3b: Serious Adverse events

Study	Icotrokinra Events	Icotrokinra Total	Placebo Events	Placebo Total	Weight	Risk Ratio MH, Random, 95% CI
ICONIC-LEAD. 2025	6	456	6	228	50.2%	0.50 [0.16; 1.53]
ICONIC-ADVANCE 1. 2025	5	310	4	155	39.8%	0.62 [0.17; 2.29]
ICONIC-ADVANCE 2. 2025	0	322	0	82	9.9%	4.86 [0.29; 82.65]

ICONIC-ADVANCE 2. 2025	9	322	0	82	9.9%	4.86 [0.29; 82.65]
Total (95% CI)	**1088**		**465**	**100.0%**	**0.69 [0.27; 1.71]**	

Heterogeneity: Tau^2 = 0.1086; Chi^2 = 2.36, df = 2 (P = 0.3080); I^2 = 15.1%
Test for overall effect: Z = -0.81 (P = 0.4187)

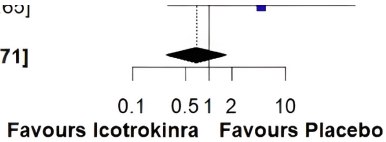
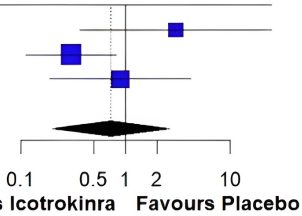


Figure 3c: Discontinuations due to Drug Adverse events

Study	Icotrokinra Events	Icotrokinra Total	Placebo Events	Placebo Total	Weight	Risk Ratio MH, Random, 95% CI
ICONIC-LEAD. 2025	6	456	1	228	22.9%	3.00 [0.36; 24.77]
ICONIC-ADVANCE 1. 2025	6	310	10	155	44.9%	0.30 [0.11; 0.81]
ICONIC-ADVANCE 2. 2025	7	322	2	82	32.2%	0.89 [0.19; 4.21]
Total (95% CI)	**1088**		**465**	**100.0%**	**0.72 [0.20; 2.59]**	

Heterogeneity: Tau^2 = 0.6879; Chi^2 = 4.37, df = 2 (P = 0.1125); I^2 = 54.2%
Test for overall effect: Z = -0.50 (P = 0.6175)



Abstract N°: ID-1338

Topic: Biologics, immunotherapy, targeted therapy

Sequential paradoxical inflammatory dermatoses in hidradenitis suppurativa following TNF-α and IL-17 pathway inhibition

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Introduction

Paradoxical inflammatory reactions are increasingly recognised adverse events of biologic therapy and represent a significant clinical challenge in the management of immune-mediated dermatoses. Hidradenitis suppurativa (HS), characterised by complex cytokine dysregulation involving TNF-α, IL-17 and innate immune pathways, may predispose patients to such reactions[1].

Materials and Methods

We report the case of a 46-year-old woman with HS diagnosed in 2013, who previously achieved excellent disease control with oral clindamycin and rifampicin. Approximately nine years later, adalimumab was initiated for disease recurrence. Shortly after treatment commencement, she developed rapid and severe clinical deterioration with inflammatory nodules, abscesses and ulcerated lesions consistent with HS flare in areas previosuly unaffected by HS. In the absence of infection, immunogenicity or secondary loss of response, this presentation was considered paradoxical HS induced by TNF-α inhibition[1]. The flare responded promptly to systemic prednisolone and amoxicillin–clavulanate and adalimumab was discontinued.

The patient was subsequently transitioned to secukinumab in January 2025 with a concomitant corticosteroid taper and later escalated to bimekizumab 3 months later, due to persistent disease activity. After approximately six months of IL-17A/F inhibition, she developed new-onset, well-demarcated erythematous scaly plaques clinically consistent with paradoxical psoriasis, without any personal or family history of psoriasis[3]. This was followed in early November 2025 by the onset of painful, tender subcutaneous nodules on the lower limbs, with histopathology confirming erythema nodosum.

Results

Mechanistically, TNF-α blockade may induce paradoxical inflammation through unchecked activation of plasmacytoid dendritic cells and increased type I interferon signalling, leading to aberrant cutaneous immune responses[2]. Similarly, inhibition of the IL-17 pathway may disrupt cytokine homeostasis, with compensatory activation of alternative inflammatory circuits, potentially contributing to multisystem inflammatory manifestations including psoriasiform disease and panniculitis[2,3]. This case is notable for the occurrence of sequential and overlapping paradoxical inflammatory dermatoses across different biologic classes in a single HS patient.

Conclusions

Recognition of paradoxical reactions is essential to avoid misdiagnosis as disease progression or therapeutic failure. This

case highlights the importance of immunopathogenic understanding when selecting and sequencing biologic therapies in HS.

Abstract N°: ID-1346

Topic: Biologics, immunotherapy, targeted therapy

Emerging Treatments for Pediatric Dermatologic Diseases: A Systematic Review of Biologics and Small Molecule Inhibitors

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Introduction
Recent advancements in pediatric dermatology have introduced biologics and small molecule inhibitors (SMIs) as targeted therapies for treating conditions like atopic dermatitis, psoriasis, and alopecia areata. This systematic review evaluates the effectiveness and safety of these therapies for dermatologic conditions in infants, children, and adolescents, focusing on randomized clinical trials.

Materials and Methods
A comprehensive search was conducted on PubMed, Scopus, and Web of Science databases following PRISMA guidelines. Studies included were those focusing on systemic biologics and SMIs used for dermatologic diseases in patients under 18. Data were extracted on treatment regimens, patient demographics, efficacy, adverse effects, and follow-up details. Bias assessment was performed using the Cochrane Risk of Bias Tool.

Results
Out of 1,454 initial studies, 49 articles met the inclusion criteria, covering 6,372 cases. Biologics, including Dupilumab, and JAK inhibitors such as Abrocitinib and Upadacitinib, demonstrated significant efficacy in treating pediatric atopic dermatitis and psoriasis. While most adverse events were mild to moderate, serious side effects were noted with certain treatments.

Conclusions

Biologics and SMIs offer promising therapeutic alternatives in pediatric dermatology, providing more targeted and effective treatments compared to traditional therapies. However, further research is essential to evaluate their long-term safety, particularly regarding developmental impacts in younger patients.

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Abstract N°: ID-1356

Topic: Biologics, immunotherapy, targeted therapy

LONG-TERM EFFECTIVENESS AND SAFETY OF TRALOKINUMAB IN PATIENTS WITH SEVERE ATOPIC DERMATITIS

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Introduction

The introduction into clinical practice of biological treatments with a highly specific focus on the IL-13 immune axis has revolutionized the landscape of Atopic Dermatitis (AD). Therefore, the objective of this study was to evaluate the medium- to long-term efficacy and safety of Tralokinumab in patients with AD at a tertiary hospital.

Materials and Methods

Retrospective observational study including all patients with AD treated with Tralokinumab from January 2022 to January 2026. The following demographic and clinical variables were evaluated based on data collected through the Farmatools electronic program: age, sex, duration of treatment, concomitant pathologies and previous use of biological treatment.

Treatment effectiveness was evaluated based on changes in the Eczema Area and Disease Severity Index (EASI), the Peak Pruritus Numerical Rating Scale (PP-NRS), and the Investigator Global Assessment (IGA) at weeks 0, 16 and 52. Effectiveness was defined as achieving at least EASI75, a reduction in PP-NRS of >3 points and an IGA of 0-1. The results were compared with baseline values.

The safety profile was determined by collecting adverse effects and classifying the degree of toxicity according to the Common Terminology Criteria for Adverse Events v. 5.0 scale.

Results

During the study period, a total of 24 patients were included (54.2% men (n=13); mean age: 39.5 years). Treatment with Tralokinumab lasted a mean of 16.8 months. Fifty percent of patients (n=12) had a concomitant condition: 29.2% rhinitis (n=7), 25% asthma (n=6), and 4.1% urticaria (n=1). Fifty-four point two percent of patients were biologic-naive (n=13).

At week 0, the mean EASI score was 24.9 and the PP-NRS score was 7.6 points. 81.2% (n=13) had an IGA score of 3-4 at baseline.

28.5% of patients (n=4) achieved an EASI75 and 21.4% (n=3) achieved an EASI 90 at week 16. At week 52, 77.8% of patients (n=7) achieved an EASI 75 and 66.7% (n=6) achieved an EASI 90.
61.5% of patients (n=8) achieved a significant decrease in PP-NRS at week 16 and 66.7% (n=6) at week 52. Likewise, 54.5% (n=6) and 83.3% (n=5) achieved an IGA of 0-1 at week 16 and 52, respectively.

16.5% (n=4) experienced ocular toxicity during treatment, of which 25% (n=1) was grade III/IV. 4.1% of patients (n=1) had to discontinue treatment due to severe toxicity.

Conclusions

This study demonstrates that Tralokinumab is an effective and safe medium- to long-term alternative for the treatment of moderate to severe AD. More comprehensive studies with larger sample sizes will be needed in the future to confirm the results obtained.

Abstract N°: ID-1363

Topic: Biologics, immunotherapy, targeted therapy

Paradoxical reactions to biologics in dermatology: clinical spectrum and management

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Introduction
The expanding use of biologic therapies in dermatology has been accompanied by an increasing recognition of paradoxical reactions, defined as the onset of new or exacerbation of pre-existing dermatoses (e.g., psoriasis induced by anti-TNF agents, eczema under dupilumab therapy). These reactions represent a significant diagnostic and therapeutic challenge, often necessitating treatment modification and negatively impacting adherence. Despite the growing body of evidence, standardized approaches to classification and management remain lacking.

Materials and Methods
A comprehensive literature review was conducted in PubMed, Scopus, ScienceDirect, and Elsevier databases over the past five years. Eligible sources included original research articles, systematic reviews, and clinical case series addressing paradoxical reactions in patients receiving biologic therapies for dermatological conditions. Clinical patterns and management strategies were systematically analyzed and categorized.

Results
The analysis identified several major clinical patterns of paradoxical reactions: psoriasiform eruptions with anti-TNF agents; eczematous and eosinophilic reactions with IL-4/IL-13 inhibitors; acneiform and neutrophilic reactions with IL-17 inhibitors; exacerbation of systemic autoimmune manifestations with IL-23 inhibitors. In most cases, complete discontinuation of the biologic was not required; reactions could be managed effectively with topical therapy or by switching to an alternative biologic class. However, predictive tools and standardized algorithms for management remain underdeveloped.

Conclusions
Paradoxical reactions are an emerging clinical concern in dermatology. The proposed categorization of clinical patterns and therapeutic strategies may facilitate optimization of patient management. Future efforts should focus on developing consensus-based algorithms and exploring biomarkers for risk stratification and prediction of such reactions.

Abstract N°: ID-1366

Topic: Biologics, immunotherapy, targeted therapy

Comparative Efficacy and Safety of Biologics and Small Molecule Inhibitors in the Treatment of Moderate-to-Severe Psoriasis: A Systematic Review

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Introduction
Psoriasis is a chronic, immune-mediated inflammatory skin condition affecting a significant portion of the global population. The condition results in substantial impairment of patients' quality of life and is associated with several comorbidities. The development of biologics and small molecule inhibitors has revolutionized the treatment landscape, offering targeted therapies with improved efficacy compared to traditional treatments. This systematic review evaluates the comparative efficacy and safety profiles of biologic therapies and small molecule inhibitors for the management of moderate-to-severe psoriasis.

Materials and Methods
A systematic review was performed across multiple databases, including PubMed, Web of Science, and Scopus, to identify randomized controlled trials (RCTs) comparing biologic agents and small molecule inhibitors in the treatment of moderate-to-severe psoriasis. The primary focus was on assessing treatment efficacy, as measured by the Psoriasis Area and Severity Index (PASI), and safety outcomes. Secondary endpoints included long-term treatment durability and patient-reported outcomes.

Results
The review included 22 head-to-head RCTs, involving over 50,000 patients. Among biologics, IL-17 inhibitors (Secukinumab, Ixekizumab) and IL-23 inhibitors (Guselkumab, Risankizumab) demonstrated superior efficacy in achieving PASI 90 and PASI 100 responses when compared to TNF-α inhibitors (Adalimumab, Etanercept). In particular, Secukinumab and Guselkumab exhibited higher rates of complete skin clearance (PASI 100) at Week 48. Among small molecule inhibitors, Deucravacitinib was found to be more effective than Apremilast in achieving PASI 75 and Static Physician Global Assessment (sPGA) responses. The safety profiles of these therapies were generally comparable, with mild injection-site reactions and nasopharyngitis being the most common adverse events. IL-17 inhibitors, however, were associated with a higher incidence of Candida infections.

Conclusions

IL-17 and IL-23 inhibitors show superior long-term efficacy compared to TNF-α inhibitors in treating moderate-to-severe psoriasis, with IL-23-targeting agents offering enhanced disease control. Small molecule inhibitors like Deucravacitinib present promising alternatives, especially for patients seeking effective oral therapies. Further research is required to compare TYK2, JAK, and PDE4 inhibitors with IL-17 and IL-23 agents in head-to-head trials, to refine clinical treatment strategies for psoriasis.

Abstract N°: ID-1372

Topic: Biologics, immunotherapy, targeted therapy

Effectiveness of treatment with lenalidomide, bortezomib, and dexamethasone in a patient with scleromyxedema associated with monocolonal gammapathy

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Introduction
A 57-year-old woman had a 9-months history of progressive skin oedema and thickening.

Materials and Methods
The patient also complained of difficulty opening her mouth and fatigue. The condition was initially located on her shoulders, arms and legs but progressed to most parts of her body. She was previously diagnosed with cutaneous vasculitis, and she was administered with 25 mg of oral prednisolone per day, 1000 mg sulfasalazine per day, 200 mg hydroxychloroquine per day for 4 months without improvement. Previously histopathologic study showed features of the granuloma annulare; immunoelectrophoresis demonstrated monoclonal spikes IgG kappa (κ) (4.9 g/L) in blood samples, but not in urine samples.

Results
Physical examination revealed erythematous plaques composed of numerous small light-red to bright-red waxy papules on her trunk and extremities. The skin on the face, hands, and forearms exhibited diffuse erythema, edema, and scleroderma-like induration. There were deep accentuating folds and furrows on dorsal side of her hands. Repeated histopathological examination of the lesional skin revealed increased fibroblasts, thickened collagen bundles in the dermis; toluidine blue staining demonstrated the deposition of mucin in the dermis. Immunophenotyping of blood cells revealed a population of clonal plasma cells with the CD138+CD38+ CD56-CD10-CD117-kappa immunophenotype. 2% of plasma cells were found in the myelogram. Immunophenotyping of bone marrow cells by flow cytometry revealed 0.144% of plasma cells with the aberrant immunophenotype CD38dimCD138+CD319+CD19-CD45+/-CD56+/- CD27+/-CD117-/+CD200+CD20-/+. No criteria for symptomatic or smoldering multiple myeloma were found. Patient was diagnosed with scleromyxedema associated with a monoclonal gammapathy of clinical significance.
Patient was administered 5 cycles of therapy with lenalidomide 25 mg orally days 1–14; bortezomib 1.3 mg/m^2 weekly subcutaneously (SC) on days 1, 4, 8, and 11; and dexamethasone 20 mg intravenously on days 1, 2, 4, 5, 8, 9, 11, and 12. Treatment was resumed every 22nd day of therapy. After 5 cycles of therapy, the patient's skin lesions resolved, and only a trace secretion of monoclonal IgG-kappa remained. Thereafter, autologous stem cell transplantation was performed.

Conclusions
Thus, we demonstrate the effectiveness of treatment with lenalidomide, bortezomib, and dexamethasone in a patient with scleromyxedema associated with monocolonal gammapathy of clinical significance.

Abstract N°: ID-1464

Topic: Biologics, immunotherapy, targeted therapy

Off-label use of Omalizumab for therapy-refractory adult-onset IgA vasculitis: a case report.

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Introduction

Immunoglobulin A vasculitis (IgAV) is a small-vessel vasculitis and the most common form of vasculitis in childhood, typically characterized by a self-limiting and uncomplicated course. In contrast, adult-onset IgAV is rare and is often associated with a more severe and complex disease course, frequently involving systemic manifestations. While childhood IgAV is often preceded by an upper respiratory infection, adult-onset disease is commonly associated with autoimmune disorders, malignancies, or medication exposure. Hereby we report a case of therapy-refractory adult-onset IgAV with severe cutaneous and systemic involvement successfully treated with off-label omalizumab.

Case Report

A 67-year-old female presented to our outpatient dermatology clinic in December 2021 with generalized, slightly elevated, intensely pruritic vasculitic skin lesions persisting for several weeks. Pruritus was rated as 7 on a 10-point visual analogue scale (VAS). In addition, she reported painful, recurrent scleritis occurring for approximately 4 years, as well as migratory joint swelling, bilateral auricular polychondritis and ulcerative colitis. Clinical examination revealed polymorphic, palpable purpura on the trunk and extremities, partly necrotic and confluent, ranging from 1 mm to several centimeters in diameter. These lesions were particularly pronounced on the lower extremities, where they were partially associated with blister formation, ulcerations and scarring. Histopathological analysis demonstrated perivascular lymphohistiocytic infiltrates with numerous leukocytoclastic neutrophils and occasional eosinophils. Direct immunofluorescence revealed linear IgA deposits in small dermal vessels, confirming the diagnosis of IgA vasculitis. Laboratory investigations showed anemia, relative neutropenia with eosinophilia and basophilia, reduced complement C3 levels, elevated total IgE, increased inflammatory markers and a positive antinuclear antibody titer (1:320) with a fine speckled nuclear pattern. After a prolonged, therapy-refractory course of the vasculitis with cutaneous, ocular, articular, renal, and gastrointestinal involvement, the patient experienced recurrent episodes of severe pruritus and skin lesions despite extensive immunosuppressive treatment, including high systemic corticosteroids, methotrexate, adalimumab, azathioprine, mycophenolate mofetil, and 5 cycles of cyclophosphamide. Treatment with omalizumab 300mg s.c. every 4 weeks was initiated in May 2024, resulting in rapid and sustained improvement of pruritus and skin manifestations. Importantly, under omalizumab therapy, IgA vasculitis remained clinically stable, with no evidence of relapse or progression of cutaneous or systemic involvement, including ocular, renal, musculoskeletal, and gastrointestinal manifestations. This case suggests that omalizumab may represent a therapeutic option for patients with therapy-refractory adult-onset IgA vasculitis.

Discussion & Conclusion

Adult-onset IgA vasculitis is rare and often associated with a severe, therapy-refractory disease course and multisystem

involvement. In this case, extensive immunosuppressive treatment failed to achieve sustained control of pruritic cutaneous disease. Initiation of omalizumab led to rapid and durable improvement of pruritus and skin manifestations without relapse or progression of systemic involvement. Although the precise mechanism remains unclear, IgE-mediated pathways and mast cell activation may contribute to disease activity in selected patients, particularly those with elevated IgE levels. While limited by its single-case design, this report suggests omalizumab as a potential therapeutic option in therapy-refractory adult-onset IgA vasculitis.

Abstract N°: ID-1477

Topic: Biologics, immunotherapy, targeted therapy

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. The Th1 axis has classically been implicated in GA pathogenesis; however, recent evidence suggests involvement of the Th2 pathway. Urticarial vasculitis (UV) is an immune-mediated disorder characterized by persistent urticarial lesions and vasculitic histopathology. The coexistence of GA and UV is rare, and optimal treatment strategies remain undefined. We report a complex case of generalized GA with coexisting urticarial vasculitis successfully treated with dupilumab, highlighting its potential as a novel therapeutic option.

Materials and Methods

Clinical, laboratory, histopathological, and treatment data of a patient diagnosed with generalized GA and UV were reviewed.

Results

A 65-year-old woman presented with a 7-year history of persistent, non-blanchable wheals lasting longer than 24 hours, along with petechiae, involving the trunk and extremities. Her medical history included goiter and cardiac arrhythmia. An initial skin biopsy was consistent with urticarial vasculitis. Laboratory investigations, including ANA and complement levels (C3, C4), were normal, and no systemic involvement or malignancy was identified. Despite treatment with systemic corticosteroids, methotrexate, hydroxychloroquine, and azathioprine for at least three months each, lesions followed a waxing and waning course. In 2023, she developed new erythematous annular plaques with central atrophy, predominantly on the extremities. Histopathological examination revealed granulomatous inflammation with degenerated collagen bundles, confirming granuloma annulare. The presence of more than 10 lesions supported a diagnosis of generalized GA. Paraneoplastic causes were excluded. A subsequent biopsy obtained from annular plaques revealed granulomatous inflammation with degenerated collagen bundles, confirming granuloma annulare. Given the presence of more than 10 plaques, the disease was classified as generalized GA based on clinicopathological correlation. Paraneoplastic causes were excluded. Subsequent therapies, including intralesional and systemic corticosteroids, narrowband UVB phototherapy with acitretin, omalizumab, and cyclosporine, were ineffective. In 2025, a new biopsy due to treatment resistance again demonstrated features of UV. Based on clinicopathological correlation, a diagnosis of UV with concomitant GA was established. Dupilumab was initiated to target both conditions, with a 600 mg loading dose followed by 300 mg administered biweekly, resulting in marked clinical improvement after three months without adverse events.

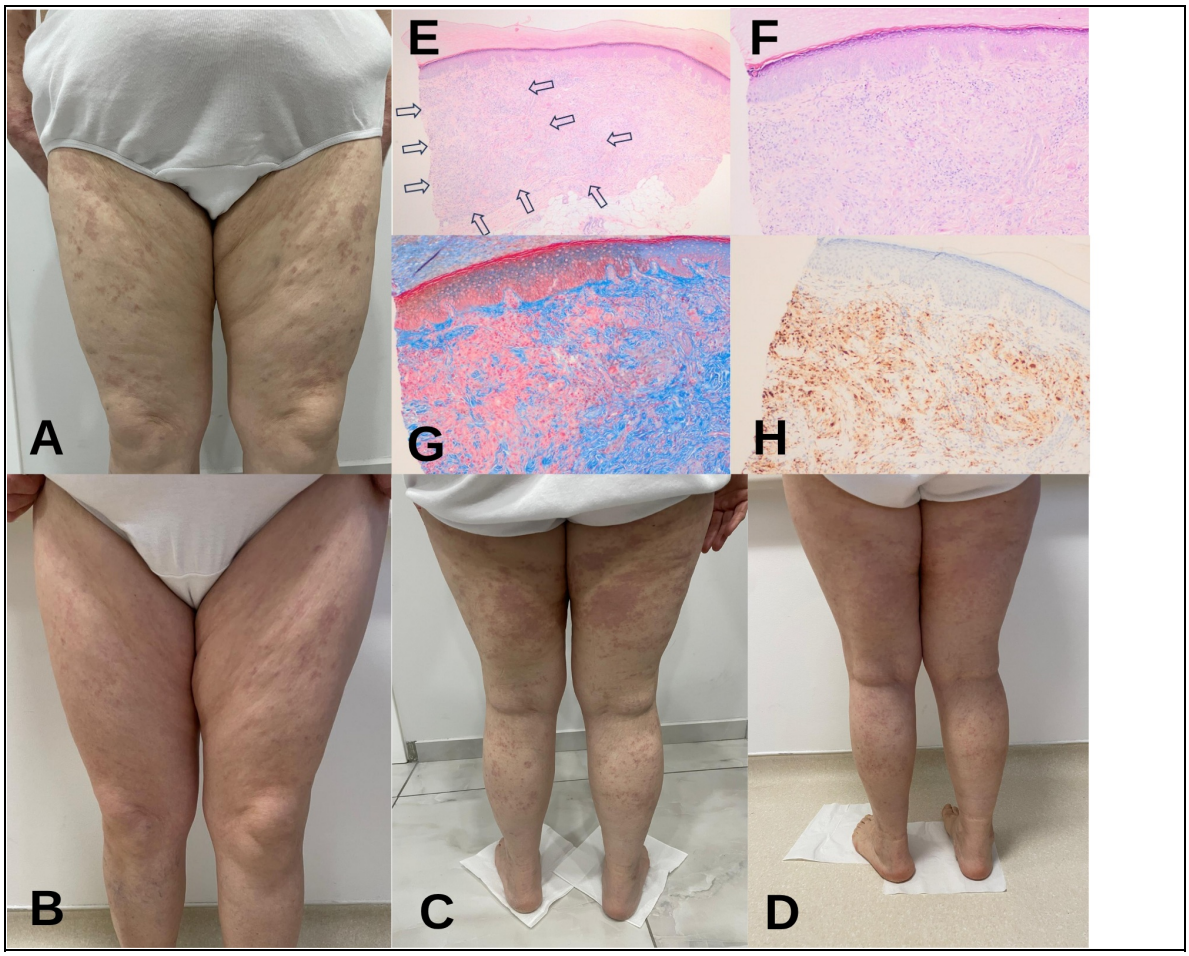

Figure 1: Erythematous annular plaques on the thighs before dupilumab therapy (A, C); marked clearance of lesions after 3 months of dupilumab treatment (B, D). Histological sections of the skin tissue. Dermis showing a granulomatous reaction with collagen degeneration (arrows) (H&E, ×40). (E); Higher magnification of panel E, demonstrating collagen fibers and histiocytes with abundant eosinophilic cytoplasm (H&E, ×100). (F); Collagen degeneration highlighted by eosinophilic staining (Masson's trichrome, ×100). (G); CD68 positivity in histiocytes forming the granulomatous reaction (immunohistochemistry, ×100). (H)

Conclusions

Dupilumab demonstrated efficacy and safety in the management of generalized GA and urticarial vasculitis. To our knowledge, this is the first reported case documenting successful dupilumab treatment in both conditions, suggesting a potential role for Th2-targeted therapy in selected refractory cases.

Abstract N°: ID-1491

Topic: Biologics, immunotherapy, targeted therapy

Nivolumab-Induced Vitiligo in Hodgkin Lymphoma: An Emerging Immune-Related Event Beyond Melanoma

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Introduction

Immune checkpoint inhibitors, particularly monoclonal antibodies targeting the PD-1 receptor such as nivolumab, have transformed the management of relapsed or refractory Hodgkin lymphoma. These agents restore antitumor immune responses by inhibiting tumor-induced immune tolerance pathways. However, this immune reactivation may be associated with immune-related adverse events, especially cutaneous manifestations. While dermatologic adverse effects are well characterized in patients with melanoma, they remain rarely reported in hematologic malignancies.

Materials and Methods

A 24-year-old patient with classical nodular sclerosis Hodgkin lymphoma was treated with brentuximab vedotin, followed by nivolumab at a dose of 100 mg administered every two weeks. Six months after initiation of immunotherapy, the patient reported the progressive onset of asymptomatic hypopigmented lesions, with no identified triggering factors or preceding local inflammatory episodes. Dermatological examination revealed well-demarcated hypochromic macules, round to oval in shape, located on the anterior aspect of the left wrist. Under Wood's lamp examination, the lesions showed homogeneous white fluorescence, consistent with depigmentation due to melanocyte loss. The patient had no personal or family history of autoimmune disease, including vitiligo. The medical history was otherwise unremarkable, with no additional systemic manifestations or treatment-related adverse events.

Results

PD-1 inhibitors such as nivolumab may induce various immune-related cutaneous adverse events, including vitiligo, which is well documented in melanoma, with an incidence reaching up to 25% in the metastatic setting. In contrast, this type of depigmentation is rarely described in hematologic malignancies, particularly in Hodgkin lymphoma. The underlying pathophysiology remains unclear. In melanoma, vitiligo is thought to result from an immune response directed against melanocytes through antigenic mimicry. In non-melanoma tumors, mechanisms such as nonspecific T-cell activation or the unmasking of an underlying autoimmune susceptibility have been proposed. In our case, the clinical presentation was typical, with no systemic involvement or identifiable autoimmune background. Nivolumab therapy was maintained. Management consisted of topical high-potency corticosteroids or topical calcineurin inhibitors. Phototherapy may be considered in cases of progression or significant psychological impact.

Conclusions

This case highlights the occurrence of immunotherapy-induced vitiligo in a non-melanoma context and underscores the importance of dermatologic monitoring in patients receiving immune checkpoint inhibitors, regardless of the underlying malignancy. Recognition of these adverse effects allows appropriate management without compromising the efficacy of oncologic treatment.