Abstract N°: ID-10

Topic: Adverse drug reactions, TEN

Toxic epidermal necrolysis in a complex immunocompromised patient: optimising best available medical and surgical therapies

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Introduction
Toxic epidermal necrolysis (TEN) is a rare, life-threatening mucocutaneous reaction most commonly triggered by medications. People living with HIV (PLWH) are at increased risk due to immune dysregulation and exposure to high risk drugs.

Materials and Methods
A 31 year old man presented with generalised tonic-clonic seizures. Neuroimaging revealed a ring enhancing lesion and biopsy confirmed cerebral toxoplasmosis. HIV testing was positive (nadir CD4 count = 64 cells/μL, viral load = 3.9 million copies/mL). He was commenced on pyrimethamine, sulfadiazine and folinic acid for cerebral toxoplasmosis, bictegravir/emtricitabine/tenofovir alafenamide as antiretroviral therapy (ART) and levetiracetam for seizure prophylaxis. Six weeks later he re-presented with a two day history of fever and an extensive papular rash. He was reviewed by dermatology and urgent skin biopsies were performed. On day five of admission, he developed widespread blistering and severe skin pain. Nikolsky sign was positive at multiple sites. Histopathology demonstrated features supporting the evolving clinical impression of TEN, including keratinocyte necrosis at varying levels. No single causative medication was identified, necessitating cessation and modification of multiple agents, including ART. His initial SCORTEN was 2 (BSA, HR) with 80% total body surface area involvement. He was transferred to the intensive care unit and received a stat 50mg dose of Etanercept on day two of blistering. Surgical management involved a specialised hydrosurgical debridement tool that uses a high-velocity, sterile saline jet to precisely remove damaged or necrotic tissue and application of a biosynthetic skin substitute. He showed rapid clinical improvement, with reduced pain and insensible fluid loss, and was discharged home after a one-month hospitalisation.

Results
PLWH are predisposed to TEN, particularly when exposed to sulfonamides and certain antiretrovirals or antiepileptics. Etanercept, a TNF-α inhibitor, has demonstrated benefit in TEN by reducing inflammatory cytokine activity and accelerating re-epithelialisation.

Conclusions
The use of high-risk medications in PLWH increase the risk of TEN. Etanercept was found to be safe and effective in this complex immunocompromised patient with TEN.

Abstract N°: ID-48

Topic: Adverse drug reactions, TEN

A case of severe acute generalised exanthematous pustulosis (AGEP) triggered by clindamycin

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Introduction

AGEP is a pustular drug eruption which sometimes causes toxic epidermal necrolysis (TEN). We present a case of biopsy-proven, clindamycin-associated AGEP associated with haemodynamic instability, epidermal detachment and multiorgan involvement.

Materials and Methods

A 72 year old female was prescribed a 5 day course of oral Clindamycin for a suspected vulval cyst and 24 hours after the course was completed, she developed a widespread rapidly spreading eruption. The rash began on her central trunk and spread to involve her face, scalp and thighs. On initial assessment, she had a fever of 39.2 °C with no skin fragility, blistering, or clear evidence of pustules. However, over the next 12 to 24 hours, involved areas of skin on her upper chest and face and she developed superficial desquamation and the rash on her limbs became more targetoid in appearance. Initial differential diagnoses included Erythema Multiforme, Toxic Epidermal Necrolysis (TEN) and Staphylococcal Scalded Skin Syndrome. Over the next 36 hours, she became hypotensive necessitating admission to the Intensive Care Unit (ICU) for vasopressor support. Her white cell count and neutrophil count were markedly elevated, at 30.0×10^9/L and 27.81×10^9/L respectively; the highest C-reactive protein was 421 mg/L. She developed multiorgan involvement, including grade 3 acute kidney injury and a mild transaminitis . Her eosinophil count was normal and she tested negative for COVID-19.

Results

At its most extensive, 70% of the patient's body surface area (BSA) was affected by erythema; 10-15% BSA developed serous-fluid filled blisters +/- superficial desquamation. She had a positive Nikolsky sign in places, but denied severe skin pain. Mucous membranes remained unaffected. An urgent skin biopsy was performed which showed sub-corneal, intraepidermal neutrophilic pustules, subepidermal oedema and partial epidermal necrosis with no significant interface change on initial assessment. The direct immunofluorescence was negative. On histopathology review, the subepidermal separation was interpreted as oedema instead of necrolysis. Although focal interface damage was noted, it was not established enough to support a diagnosis of Stevens-Johnson syndrome (SJS) or TEN, thus a diagnosis of severe AGEP was favoured with a EuroSCAR score of 10.

As the causative drug had already stopped prior to presentation, best supportive care was initiated. She was nursed in the ICU in a temperature-regulated private room and given intravenous fluids and analgesia. Topical emollients, an ointments and antimicrobial soap substitute and non-adherent dressings for eroded areas were provided. Large bullae were aspirated, and minimal handling was prioritised. Due to haemodynamic instability and markedly raised infection markers, she was empirically treated with vancomycin and co-trimoxazole for suspected sepsis. She did not receive oral corticosteroid treatment. Her condition improved rapidly with these measures. Topical clobetasol 0.05% ointment was later introduced to treat some affected skin areas.

Conclusions

This case illustrates the clinical challenge in differentiating severe, and rapidly progressive AGEP from TEN, particularly as confluent pustules can rapidly lead to desquamation. Clinical features supporting a diagnosis of AGEP include quicker onset following causative drug exp, the superficial nature of desquamation and an absence of mucosal involvement. Elevated inflammatory markers in the context of fever and haemodynamic instability can lead to unnecessary treatment with antibiotics. Our case highlights the value of regular re-assessment, the central role of histopathology and the use of the EuroSCAR criteria to help distinguish TEN from AGEP.

Abstract N°: ID-121

Topic: Adverse drug reactions, TEN

A Case of Allopurinol-Induced DRESS Syndrome

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Introduction

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) is a rare but serious drug-induced hypersensitivity syndrome clinically characterized by generalized erythematous-maculopapular eruptions, facial edema, fever, lymphadenopathy, and involvement of internal organs [1, 2]. Among the various implicated drugs in this syndrome, anticonvulsive agents are reported as the most common triggers (27%), followed by allopurinol (26.3%) and antibiotics (24%). Notably, individuals carrying HLA-B*58:01 allele have a significantly increased risk of developing DRESS upon exposure to allopurinol, particularly in Asian populations compared with individuals of European descent. Renal involvement is commonly observed in cases of allopurinol-induced DRESS [3, 4, 5]. It underscores the complexity and potential severity of this syndrome. Due to the heterogeneous clinical presentation and the potential for rapid progression to multi-organ dysfunction, early recognition and prompt management are crucial [4].

Materials and Methods

We report a clinically significant case of allopurinol-induced DRESS syndrome presenting with hepatic and renal involvement.

Results

A 76-year-old female developed high fever, pruritic morbilliform exanthema involving the face, trunk, and extremities, perioral edema, and odynophagia after one month of allopurinol therapy. Dermatological examination revealed confluent erythematous maculopapular lesions, most expressed on the palms, soles, and abdomen, lip desquamation, crusting, and the erosions of oral mucosa.

Laboratory findings showed leukocytosis (15.8×10^9/L) with marked eosinophilia (6.0×10^9/L), elevated C-reactive protein (76 mg/L), and acute hepatic (ALT 91 IU/L, AST 54 IU/L, GGT 238 IU/L, ALP 123 IU/L) and renal (creatinine 176 µmol/L, urea 15 mmol/L) impairment. Autoimmune (ANA) and viral (HBV, HCV) causes were excluded. Skin histologic examination demonstrated focal spongiosis, keratinocyte apoptosis, basal layer vacuolar degeneration, dermal edema, and perivascular lymphohistiocytic-eosinophilic infiltrate, consistent with DRESS. RegiSCAR's score was 7, with the disease severity graded as moderate.

Allopurinol was discontinued, and oral prednisolone 30 mg/day was initiated with gradual taper, along with antimicrobial therapy for the secondary bacterial infection. Topical therapy included only chlorhexidine and prednisolone oral rinses. This management led to rapid resolution of fever, regression of cutaneous lesions, and normalization of hepatic and renal function over subsequent weeks, leading to complete recovery without persistent organ dysfunction.

Conclusions

This case illustrates that moderate allopurinol-induced DRESS syndrome may present with significant systemic involvement yet remain manageable with oral corticosteroid therapy when accurately diagnosed. Early clinical suspicion, prompt cessation of the offending agent, and timely initiation of the proper treatment are essential in reducing morbidity and preventing long-term complications.

Abstract N°: ID-122

Topic: Adverse drug reactions, TEN

Toxic Epidermal Necrolysis (TEN) induced by Immunotherapy in an Oncology patient. A Case Report.

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Introduction

PD-1 and PD-L1 inhibitors are cancer immunotherapies that belong to the immune checkpoint inhibitors group. They work by blocking the PD-1/PD-L1 pathway, which tumors use to turn off T-cells and escape the immune system. PD-1 inhibitors block the receptor on T-cells, while PD-L1 inhibitors block the ligand on tumor cells. Even though they have a positive safety profile compared to chemotherapy, and they don`t place the lives of patents at risk, a wide spectrum of adverse events have been documented including serious cutaneous ones, such as Stevens – Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN). TEN is a rare, life-threatening skin reaction that affects people of all ages and is usually medication - induced. Signs & symptoms include widespread skin pain, spreading rash covering more than 30% of the body, blisters and large areas of peeling skin along with sores, swelling and crusting on the mucous membranes, including the mouth, eyes and genitalia. The objective of this abstract is to present an interesting TEN case induced by nivolumab treatment for squamous cell lung carcinoma (SCLC).

Materials and Methods

79-year-old male patient, diagnosed with SCLC (p40+, TTF1 -, CDX2 -, Ki27 up to 20%), was admitted in the Internal Medicine Clinic at Serres General Hospital, with a macroscopic diagnosis of TEN that was biopsy-confirmed, at the day of admission. Additionally, following ScoreTEN assessment, the result was >90%, scoring 5 risk factors:

- Age > 40
- Positive (+) History for malignancy (SCLC).
- BSA affected >10%
- Urea: 37 mg/dl> 28.
- Serum Bicarbonate levels:17 mmol/lt.

According to hospital records, patient received five chemotherapy treatment cycles, including Paclitaxel + Carboplatin + Bevacizumab, followed by two treatment cycles with PD-L1 inhibitor, Atezolizumab, for SCLC. During that time, patient experienced a cutaneous pharmaceutical reaction, that was managed with a combination of systemic & topical corticosteroids, and PD-1 inhibitor Nivolumab was introduced for four treatment cycles. Following Nivolumab`s administration, patient`s cutaneous symptoms worsened and progressed into TEN. Checkpoint inhibitor immunotherapy was discontinued. During hospitalization, patient received 500mg Methylprednisolone IV, Infliximab 5mg/kg and then 1gr/kg Immunoglobulin Gamma IV for minimization and control of TEN signs & symptoms.

Rifaximin for controlling patient`s diarrhea, along with Tazobactam-Piperacillin, Amikacin Sulfate for infection control, supportive care with Antipyretics, Oxygen, intensive intravenous Hydration, Dextrose supply and topical skin and mucousa care, were also provided.

Results

Within the first 7 days of hospitalization, TEN signs & symptoms were significantly improved macroscopically, along with a negative Nikolsky sign, indicated the ongoing skin re-epithelization, along with a decrease in the patient`s CRP serum levels.

Unfortunately, patient deceased 22 days following initial discharge, within hospital after he was admitted for severe pneumonia. Cause of death was cardiac arrest.

Conclusions

PD-1 and PD-L1 inhibitors comprise a category of drugs that has revolutionized the field of oncology. However, early diagnosis and cross-specialty collaboration for the management of associated skin toxicities, are essential for optimal patient care by improving diagnostic accuracy, coordination of treatment more effectively and overall enhancement of patient safety.

Abstract N°: ID-185

Topic: Adverse drug reactions, TEN

Keratosis pilaris–like eruption associated with ponatinib therapy: an underrecognized cutaneous adverse effect

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Introduction

Ponatinib is a third-generation tyrosine kinase inhibitor primarily used in patients with chronic myeloid leukemia harboring the BCR-ABL T315I mutation. Although highly effective, tyrosine kinase inhibitors are associated with a broad spectrum of cutaneous adverse effects. Keratosis pilaris–like eruptions are uncommon and remain poorly characterized in patients receiving ponatinib, which may lead to underrecognition and diagnostic uncertainty.

Materials and Methods

A 41-year-old female with a history of chronic myeloid leukemia with T315I mutation, treated with oral ponatinib for two years at alternating doses of 22.5 mg and 45 mg, was evaluated for a diffuse follicular eruption that developed following dose escalation. A complete dermatological examination was performed. Due to the patient's oncologic background and systemic therapy, a skin biopsy was obtained to exclude alternative diagnoses, including disease-related or drug-induced inflammatory reactions.

Results

Physical examination revealed a disseminated dermatosis involving the head, trunk, and upper extremities, characterized by multiple follicular, skin-colored punctate papules on an erythematous background, with a symmetrical distribution. Histopathological analysis showed mild, nonspecific chronic dermatitis without features of infection or severe drug reaction. After exclusion of other causes and considering the temporal relationship with ponatinib dose increase, a diagnosis of keratosis pilaris–like eruption secondary to ponatinib therapy was established. The condition was not life-threatening but was associated with patient discomfort.

Conclusions

Keratosis pilaris–like eruption represents a benign but potentially quality-of-life–impacting cutaneous adverse effect associated with ponatinib therapy. Awareness of this presentation is important to avoid misdiagnosis, unnecessary discontinuation of effective oncologic treatment, and to ensure appropriate dermatologic management. Recognition of the expanding spectrum of tyrosine kinase inhibitor–related skin reactions is essential for optimal multidisciplinary patient care.

Abstract N°: ID-190

Topic: Adverse drug reactions, TEN

Capecitabine-Induced Palmar-Plantar Erythrodysesthesia Exacerbated by Occupational Mechanical Stress: A Case Report

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Introduction

Capecitabine-induced palmar-plantar erythrodysesthesia (PPE) is a common, dose-limiting cutaneous toxicity that can significantly impair quality of life and compromise oncologic outcomes. While drug-related factors are well described, the role of occupational mechanical stress in exacerbating PPE remains under-recognized. We highlight a clinically relevant case underscoring the importance of occupational exposure in the severity and progression of PPE.

Materials and Methods

A 39-year-old woman with recurrent breast carcinoma receiving oral capecitabine monotherapy presented with progressive acral symptoms. Dermatological examination revealed symmetrical involvement of the palms and pressure-bearing areas of the soles. Palmar-plantar erythrodysesthesia was defined by painful erythema, desquamation, edema, and functional limitation affecting activities of daily living without ulceration. Severity was graded using the National Cancer Institute Common Terminology Criteria for Adverse Events (CTCAE) version 5.0. A comprehensive occupational history identified prolonged standing and repetitive mechanical stress as potential exacerbating factors. Clinical findings were documented photographically. Management included high-potency topical corticosteroids, urea- and lactic acid–based emollients, oral antihistamines, and targeted counseling to reduce mechanical stress and friction.

Results

Based on clinical presentation and CTCAE assessment, a diagnosis of Grade 2 capecitabine-induced palmar-plantar erythrodysesthesia was established. Occupational mechanical stress was identified as a significant exacerbating factor contributing to symptom severity. Following appropriate conservative dermatological management and behavioral modification, the patient demonstrated marked clinical and symptomatic improvement, with reduction in erythema, desquamation, and pain. Importantly, symptom control permitted continuation of capecitabine therapy without dose reduction or interruption.

Conclusions

Capecitabine-induced palmar-plantar erythrodysesthesia may be significantly exacerbated by occupational mechanical stress, such as prolonged standing and repetitive friction. Early recognition of aggravating factors, CTCAE-based severity assessment, and timely initiation of conservative dermatologic management can effectively control symptoms and prevent unnecessary interruption of oncologic therapy. This case highlights the importance of integrating occupational history and patient education into the multidisciplinary management of chemotherapy-related cutaneous adverse events.

Abstract N°: ID-193

Topic: Adverse drug reactions, TEN

Ocrelizumab-Induced Atopic Dermatitis-like Drug Eruption: A Case Report

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Introduction

Ocrelizumab, a humanized anti-CD20 monoclonal antibody, is used for the management of relapsing-remitting multiple sclerosis (RRMS) and primary progressive multiple sclerosis (PPMS). While psoriasiform dermatitis, thought to result from B-cell depletion and subsequent T-cell dysregulation induced by ocrelizumab, has been documented in the literature, the emergence of atopic dermatitis-like eruptions has not been previously reported. Herein, we describe a case of atopic dermatitis-like eruption associated with ocrelizumab administration.

Materials and Methods

A 67-year-old male patient presented to the neurology clinic with a one-year history of progressive gait disturbance. Neurological examination revealed bilateral clonus and mild gait ataxia. Cranial contrast-enhanced magnetic resonance imaging demonstrated multiple demyelinating plaques. Cerebrospinal fluid (CSF) electrophoresis revealed type 2 oligoclonal bands. The patient was diagnosed with primary progressive multiple sclerosis and initiated on ocrelizumab therapy. The patient had no history of taking any medications other than ocrelizumab. Six and a half months following treatment initiation and four days after administration of the third ocrelizumab infusion, the patient developed pruritic, erythematous, ill-defined eczematous papules and plaques in the antecubital and popliteal fossae. The patient was referred to the dermatology department, where a skin punch biopsy was performed.

Results

Skin biopsy revealed epidermal spongiosis and acanthosis with superficial pustule formation, accompanied by a mixed inflammatory infiltrate in the superficial dermis characterized by a dense perivascular eosinophilic infiltrate with scattered neutrophils. The histopathological findings were consistent with drug-induced eruption. The patient was prescribed emollients, topical methylprednisolone aceponate, and oral bilastine. At the two-week follow-up, complete resolution of the cutaneous lesions was observed. Given the mild severity of the eruption, discontinuation of ocrelizumab therapy was not deemed necessary.

Conclusions

The existing literature has documented various cutaneous adverse events associated with ocrelizumab therapy, predominantly psoriasiform eruptions, as well as palmoplantar pustulosis, cutaneous vasculitis, nummular dermatitis, alopecia areata, and pyoderma gangrenosum. To the best of our knowledge, this represents the first documented case of atopic dermatitis-like eruption associated with ocrelizumab administration. The pathophysiological mechanism may involve B-cell depletion induced by ocrelizumab, resulting in subsequent immune dysregulation. Although further investigation is warranted to elucidate this association, clinicians should maintain heightened vigilance for cutaneous

manifestations when administering novel biological agents.

Abstract N°: ID-229

Topic: Adverse drug reactions, TEN

Systemic Treatments for Pediatric Stevens–Johnson Syndrome/Toxic Epidermal Necrolysis: A Living Systematic Review

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Introduction
The optimal systemic treatment strategy for pediatric Stevens–Johnson syndrome (SJS)/toxic epidermal necrolysis (TEN), including the relative effectiveness of monotherapy versus combination therapy, remains unclear. This study aimed to systematically review treatment modalities for pediatric SJS/TEN.

Materials and Methods
PubMed, Embase (via OVID), and Cochrane Library databases were searched from their respective inception to April 26, 2025. Retrospective observational studies and case series or reports examining systemic treatment for pediatric patients with SJS/TEN and their clinical outcomes were included. The risk of bias of the included observational studies with case series and reports was assessed using the Risk of Bias in Non-randomized Studies of Interventions and Joanna Briggs Institute critical appraisal tools, respectively. The primary outcomes were mortality and complete remission (CR) rates in pediatric patients with SJS/TEN. These outcomes were reported as proportions based on the number of events over the total number of patients in each treatment group.

Results
Thirty-six studies (9 retrospective observational studies, 27 case series or reports) comprising 1,148 pediatric patients were included. Among the observational studies, mortality rates were 0%–33.3%, depending on the treatment modality used. Conversely, only two deaths were reported across all case series/reports. The highest CR rates were observed with steroid monotherapy and supportive care, followed by intravenous immunoglobulin (IVIG) monotherapy and the combination of steroids with IVIG. Tumor necrosis factor-alpha inhibitors, specifically etanercept, exhibited potential as a treatment option for pediatric SJS/TEN. Long-term sequelae were observed across all treatment categories, with some children experiencing complications such as dyspigmentation, corneal ulceration, dry eye, and transaminitis.

Conclusions
Current evidence supports the use of steroid monotherapy and supportive care alone as potentially effective treatment options for pediatric SJS/TEN. These findings may inform clinical decision-making and guide therapeutic strategies for this vulnerable population.

Abstract N°: ID-300

Topic: Adverse drug reactions, TEN

A case of palmarplantar erythrodysesthesia with twenty-nail dystrophy secondary to IgG myeloma managed with Itraconazole therapy

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Introduction

Palmar–plantar erythrodysesthesia (PPE) is a well-recognised cutaneous adverse effect associated with several chemotherapeutic and targeted therapies. We report a case of diffuse twenty-nail dystrophy secondary to PPE associated with Elranatamab, a bispecific antibody recently introduced for relapsed and refractory multiple myeloma. This case is significant as twenty-nail dystrophy is not an established manifestation of PPE. This case highlights the need for awareness of such manifestations in both dermatology and haematology practice.

Materials and Methods

We report a clinical case of ongoing PPE with atypical twenty-nail dystrophy, possibly caused by chemotherapy. A 73-year-old man with long-standing immunoglobulin-G kappa multiple myeloma presented to dermatology with a 5-week history of painful, erythematous, scaly eruptions on the palms and soles, accompanied by severe pruritus worsening with heat. He also developed progressive dystrophy of all 20 fingernails and toenails.

Results

He was initially treated with chemotherapy, followed by an autologous stem cell transplantation in 2015. In June 2017, he underwent further chemotherapy, a second autologous stem cell transplant, and maintenance Ixazomib until 2020. Subsequent regimens included Lenalidomide with Dexamethasone followed by Isatuximab–Pomalidomide–Dexamethasone from January 2023, before switching to Elranatamab in February 2025. Elranatamab was first given weekly, then reduced to monthly dosing. The skin and nail changes appeared approximately six months after initial administration of Elranatamab. Past medical history included coronary artery bypass grafting (2013), atrial fibrillation with complete heart block (August 2021, on apixaban), hypertension, chronic obstructive pulmonary disease, and possible primary hyperparathyroidism with mild hypercalcaemia and normal parathyroid hormone levels.

He was initially treated with Clotrimazole cream and oral Itraconazole (100 mg once daily for eight weeks), alongside an antiseptic, moisturising soap substitute and a short course of topical antifungal with Hydrocortisone. His palmoplantar scaling substantially improved, and while some of the twenty-nail dystrophy persisted, new proximal nails appeared without dystrophy on some digits. After the follow-up, He was prescribed Itraconazole but reduced to 100 mg twice weekly for t eight weeks, moisturising soap substitute, and urea-lauromacrogols emollient cream twice daily. Three months later some improvement in his nails with the Itraconazole with some fingernail growth. He was still getting areas of dry, peeling skin on his palms and soles, and more dry skin extending up into his left leg.

Apart from twenty-nail dystrophy and local injection-site erythema, no other toxicities from Elranatamab were reported in the patient. He remained on Co-trimoxazole, Aciclovir, and monthly prophylactic intravenous Immunoglobulin replacement.

Conclusions

This case represents the first reported association of Elranatamab with palmar–plantar erythrodysesthesia-like eruptions accompanied by diffuse twenty-nail dystrophy. Such a cutaneous adverse effect can mimic infection, which can potentially lead to diagnostic uncertainty and error. Thus, recognising these side effects is crucial to deliver the best quality of care to the patient. It highlights the importance for dermatologists to be aware of this condition to guide the management plan and ensure that effective systemic therapy can be continued without unnecessary interruption.

Abstract N°: ID-308

Topic: Adverse drug reactions, TEN

The case for G-CSF in the management of toxic epidermal necrolysis

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Introduction

Stevens-Johnson syndrome (SJS) and Toxic epidermal necrolysis (TEN), although rare, represent the most severe form of cutaneous drug reactions. The acute phase of the disease may be accompanied by a variety of systemic complications, including fluid and protein loss, thermo-dysregulation, high output cardiac failure, infection and neutropenia contributing to a high mortality.

Granulocyte-colony stimulating factor (G-CSF) is a safe, well tolerated medication commonly used in chemotherapy-associated neutropenia. It reduces infection-related morbidity and mortality by stimulating neutrophil production. Irrespective of neutrophil count, G-CSF additionally accelerates re-epithelialisation of cutaneous and mucosal surfaces. By promoting recovery of the skin barrier and increasing neutrocytosis G-CSF may reduce all complication rates – especially infections, hospital length of stay and mortality in SJS/TEN.

Materials and Methods

A targeted literature search was conducted using PubMed, MEDLINE and Embase for English-language articles published between January 1990 and November 2025 reporting G-CSF use in SJS and TEN. Search terms included granulocyte-colony stimulating factor, filgrastim, stevens-Johnson syndrome, toxic epidermal necrolysis and Stevens-Johnson syndrome.

Results

There have been several case reports and series in the literature that describe benefit from systemic G-CSF in TEN. A total of 13 case reports or series (USA: 3, UK: 2, India: 2, Turkey: 1, New Zealand: 1, China: 1, Singapore: 1, Denmark: 1, Poland: 1) were included, comprising 17 patients (Table 1). Ages ranged from 2-75 years; M:F ratio of 1:1 .

The duration of G-CSF therapy varied from a single administration to four daily doses followed by alternate-day dosing for eight days. Neutrophil counts increased in 16/17 patients (94%). Re-epithelialisation was achieved in 16/17 patients (94%), with seven of these reports characterising the healing trajectory as notably accelerated. In a retrospective review, Ang et al noted shorter times to re-epithelialisation in the two patients who received G-CSF compared with those who did not. In two case reports, positive outcomes were demonstrated in TEN patients with neutropenia of alternate aetiology, suggesting that G-CSF may be beneficial regardless of the underlying cause of neutropenia. Mahajan & Kanwar reported rapid epithelialisation in two patients without neutropenia, supporting the idea that G-CSF may provide benefit independent of neutrophil count. Overall survival was reported in 94% of patients (n=16). Four reports described successful treatment with supportive care and G-CSF alone, while others combined G-CSF with intravenous

immunoglobulin, tumour necrosis factor inhibition (anti-TNF), and/or ciclosporin. Table 1 summarises the characteristics of the included studies assessing the effect of G-CSF in SJS/TEN.

Table 1: Characteristics of included studies assessing the effect of G-CSF in TEN

Study, year	Location	Study design	Number of patients	Age (years)	Sex (F/M)	Inciting factor	TBSA (%)	Cytopenia if present	Dose	Duration (days)	Additional systemic therapies	Time to recovery of neutrophils (days)	Re-epithelialisation achieved (Y/N)	Time to re-epithelialisation (days)
Goulden and Goodfield., 1996	UK	Case Report	1	7	F	Erythromycin	>70	Neutropenia	500,000 units/kg	4	Hydrocortisone	2	Y	<14
Jarrett et al., 1997	New Zealand	Case Report	1	42	F	Carbamazepine	80	Pancytopenia	300µg	12	Ciclosporin Antibiotics Antifungals	2	Y	<14
Bae et al., 1997	USA	Case Report	1	32	M	Penicillin	100	Leukopenia	300µg	-	Human Growth Hormone Antifungals Antibiotics	-	Y	<14
Winfred et al., 1999	USA	Case Report	1	59	F	Captopril	>30	Agranulocytosis	300 -350µg	5	Antibiotics Hydroxyzine	3	Y	<11
Robak et al., 2001	Poland	Case Report	1	23	M	Unknown	-	Aplastic Anaemia	-	-	Ciclosporin	-	Y	'Within a few days'
Kalyoncu et al., 2004	Turkey	Case Report	1	2	M	Teicoplanin/ Amikacin	-	Neutropenia	375µg	6	IVIG Prednisolone Antibiotics	6	Y	<14
Upadya and Ruxana, 2009	India	Case Report	1	25	F	Ciprofloxacin	-	Neutropenia	-	5	Antibiotics	-	Y	<60
De Sica-Chapman et al., 2010	UK	Case Series	2	61	F	Phenytoin/ Phenobarbital	>95	Neutropenia	96 MU	5	IVIG Ciclosporin	-	Y	<15
				47	M	Sulfasalazine	80	Modest Neutropenia	60 MU	3	IVIG Ciclosporin, Antibiotics	-	Y	3
Ang and Tay, 2011	Singapore	Case Series	2	15	M	Carbamazepine	10-30	Neutropenia	300µg	1	Hydrocortisone	-	Y	1-3 days earlier than without G-CSF
				18	F	Trimethoprim-Sulfamethoxazole	10-30	Neutropenia	300µg	1	Hydrocortisone	-	Y	1-3 days earlier than without G-CSF
Pallesen et al., 2012	Denmark	Case Report	1	15	M	Unknown	25	Neutropenia	5µg/kg	3	Antibiotics	1	Y	7
Mahajan and Kanwar, 2013	India	Case Series	3	16	-	-	-	-	300µg	5	-	-	Y	Notably accelerated
				20	-	-	-	-	300µg	5	-	-	Y	Notably accelerated
				65	-	-	-	Leukopenia	300µg	5	-	-	N - died from refractory sepsis	-
Huang et al., 2024	USA	Case Report	1	48	F	Unknown	-	Pancytopenia			Steroids IVIG Infliximab Ciclosporin	14-21	Y	14-21
Zhou et al., 2025	China	Case Report	1	75	M	Tislelizumab	-	Agranulocytosis	150µg		Methylprednisolone IVIG Antibiotics	9	Y	-

Abbreviations: UK, United Kingdom; USA, United States of America; units/kg, units per kilogram; µg, microgram; MU, million units; µg/kg, micrograms per kilogram; IVIG, intravenous immunoglobulin

Conclusions

G-CSF is safe and well tolerated, reducing infection-related morbidity and mortality in chemotherapy-associated neutropenia. Evidence from case reports and series suggests G-CSF may promote rapid neutrophil recovery and favourable clinical outcomes in patients with and without SJS/TEN-associated neutropenia. Published reports describe high rates of re-epithelialisation and, in some cases, accelerated healing. G-CSF has been used alone and combined with intravenous immunoglobulin, anti-TNF, and/or ciclosporin, supporting its potential role as an adjunctive therapy. Although rare adverse events such as acute respiratory distress syndrome, capillary leak syndrome and thrombotic complications have been temporally associated with G-CSF use in other clinical contexts and comorbid conditions, no such complications have been reported in published cases of G-CSF use in SJS/TEN. Controlled studies are required to confirm efficacy, optimal dosing, and duration – however the rarity and heterogeneity of the condition are important obstacles to undertaking such work.

Abstract N°: ID-386

Topic: Adverse drug reactions, TEN

Progressive isolated lower-lip oedema after mRNA SARS-CoV-2 booster vaccination complicated by MRSA superinfection: a necrotising fasciitis mimic

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Introduction
Isolated labial oedema temporally associated with SARS-CoV-2 vaccination is uncommon. Progressive swelling with crusting or necrotic slough may prompt concern for necrotising soft tissue infection, creating an important diagnostic and management pitfall.

Materials and Methods
We report a single case with clinicopathologic correlation. Clinical history, laboratory investigations, microbiology, and histopathology were reviewed.

Results
A previously healthy 19-year-old man developed mild, isolated lower-lip swelling 3–4 hours after receiving an mRNA SARS-CoV-2 booster vaccine (active ingredient: raxtozinameran). Swelling progressed over several days with pain and crusting, peaking at approximately 96 hours. There was no history of contact allergy, trauma, piercings, cosmetic products, or recent infection. He remained haemodynamically stable throughout. Initial investigations showed normal white blood cell count and C-reactive protein, negative viral swabs, and negative serology for Epstein–Barr virus and HIV. Symptoms did not improve with empiric oral antibiotics, systemic corticosteroids, and antihistamines.

Twelve days after symptom onset, he re-presented with marked oedema and necrotic slough, raising concern for necrotising soft-tissue infection. The Laboratory Risk Indicator for Necrotising Fasciitis (LRINEC) score was 4 (low risk). Limited debridement was performed. Histopathology demonstrated hyperkeratotic, acanthotic epithelium with mixed neutrophilic and lymphohistiocytic inflammation extending into the underlying skeletal muscle. Wound culture yielded heavy growth of methicillin-resistant *Staphylococcus aureus* (MRSA). Targeted oral antibiotic therapy with co-trimoxazole resulted in clinical resolution of oedema and crusting. The patient remained well with no further labial oedema on subsequent dermatology follow up.

Conclusions
Progressive isolated labial oedema temporally associated with mRNA SARS-CoV-2 booster vaccination may represent a delayed hypersensitivity phenotype and can be complicated by secondary bacterial infection, producing a necrotising fasciitis mimic. Early microbiological sampling, clinicopathologic correlation, and prompt targeted antibiotics are important when swelling progresses and is refractory to anti-allergic therapy.

Abstract N°: ID-395

Topic: Adverse drug reactions, TEN

When Antidiabetic Therapy Affects the Skin – case report and literature review.

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Introduction

Bullous reactions represent rare but clinically significant cutaneous adverse effects of antidiabetic therapies. The literature reports an association between dipeptidyl peptidase-4 (DPP-4) inhibitors (gliptins) and autoimmune bullous diseases, particularly bullous pemphigoid, while only isolated cases have been described in relation to sodium-glucose co-transporter 2 (SGLT2) inhibitors (flozins). The underlying mechanisms remain incompletely understood; however, immune dysregulation and potential cross-reactivity with basement membrane zone antigens have been reported.

A 60-year-old male patient with a history of psoriasis and type 2 diabetes presented to the Dermatology Department with blisters and purpuric lesions (petechiae) on the hands and feet that had developed over several weeks. Additionally, he reported a three-month history of pruritic papules and erosions on the trunk. One year prior to presentation, empagliflozin had been added to his long-term metformin therapy. Initial dermatological management included systemic antihistamines and topical triamcinolone combined with tetracycline spray. Owing to the lack of clinical improvement, serological testing was performed to exclude autoimmune bullous disorders, with negative results. A referral to a diabetologist was therefore recommended to reassess and modify the antidiabetic treatment.

At the two-month follow-up visit, the patient reported that empagliflozin had been switched to dapagliflozin. Clinical examination revealed partial remission, with resolution of lesions on the hands, post-inflammatory desquamation on the feet, and persistence of a few erosive lesions and scattered papules on the trunk and lower legs. Patch testing demonstrated positive reactions to gallates and sulfur compounds. At a subsequent visit, the patient reported that dapagliflozin had also been discontinued due to the development of balanitis. The patient was maintained on metformin monotherapy. At that time, complete resolution of all cutaneous lesions was observed.

Materials and Methods

N/A

Results

In 1835, phlorizin, a dihydrochalcone isolated from apple tree bark, was identified as the first naturally occurring compound with sodium–glucose cotransporter 2 (SGLT2) inhibitor activity [1]. SGLT2 inhibitors represent a relatively novel class of oral hypoglycemic agents used in the treatment of type 2 diabetes mellitus (T2DM) [2]. This drug class includes canagliflozin, dapagliflozin, empagliflozin, ipragliflozin, luseogliflozin, and tofogliflozin [3]. SGLT1 and SGLT2 transporters, located in the proximal convoluted tubule of the kidneys, are responsible for the reabsorption of glucose and sodium from the glomerular filtrate. In patients with T2DM, upregulation of SGLT2 contributes to persistent hyperglycemia via an insulin-independent mechanism [4]. Beyond glycemic control, SGLT2 inhibitors demonstrate cardioprotective effects, including blood pressure reduction through glycosuria and natriuresis, weight loss, and a decreased risk of cardiovascular events [5]. The most commonly reported adverse effects include hypotension, genital and urinary tract infections, and diabetic ketoacidosis [6]. Dermatological adverse events associated with SGLT2 inhibitor therapy are uncommon but have been reported, including Fournier's gangrene (necrotising fasciitis), fixed drug eruptions, pruritus, Sweet syndrome, bullous pemphigoid, psoriasis, and psoriasiform vulvitis [7].

Conclusions

In the present case, blistering lesions developed after more than one year of empagliflozin therapy and showed partial remission following drug substitution and complete resolution after discontinuation of SGLT2 inhibitor treatment , suggesting a possible association with a non-autoimmune bullous reaction. This observation highlights the importance of considering SGLT2 inhibitors (flozins) in the in the differential diagnosis of bullous or blistering skin eruptions in patients with type 2 diabetes mellitus.

Abstract N°: ID-422

Topic: Adverse drug reactions, TEN

Pseudo-Pseudoxanthoma Elasticum Induced by D-Penicillamine in Wilson's Disease

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Introduction

Pseudo-pseudoxanthoma elasticum (PPXE) is a rare adverse effect associated with D-penicillamine (DPA) therapy. Cutaneous adverse effects of DPA occur in approximately 25–50% of patients. PPXE occurs more frequently in patients with Wilson's disease or cystinuria treated with long-term, high-dose DPA compared with those treated for rheumatoid arthritis. Clinically, PPXE closely resembles hereditary pseudoxanthoma elasticum (PXE); however, it is not associated with mutations in the ABCC6 gene, shows no calcification of elastic fibers, and typically occurs without systemic organ involvement. PPXE may present as an isolated condition or in association with other DPA-induced cutaneous disorders. Histopathologically, it is characterized by irregular "lumpy-bumpy" degeneration of elastic fibers, thought to result from an inability of elastic fibers to re-expand after contraction.

Materials and Methods

Herein, we present a female patient with Wilson's disease who had been treated with long-term, high-dose D-penicillamine. Clinical examination, histopathological analysis of a skin biopsy using routine and special stains, as well as ophthalmological and cardiological assessments, were performed.

Results

A 60-year-old woman with Wilson's disease, treated with D-penicillamine for 34 years (1 g/day), presented with a 12-year history of progressive skin wrinkling predominantly affecting the neck. Her medical history was otherwise notable only for arterial hypertension, and her family history was positive for Wilson's disease. Physical examination revealed coalescing, skin-colored papules with a slight yellowish hue on the neck, most pronounced on the anterior aspect. Discreet redundant skin folds were also observed in the axillary and antecubital regions.
Histopathological examination of a skin biopsy obtained from the neck revealed basophilic, irregular elastic fibers in the papillary dermis. Orcein and Elastica van Gieson staining demonstrated thickened elastic fibers with numerous lateral projections and a characteristic serrated appearance, while Von Kossa staining was negative for calcium. Based on the clinical and histopathological findings, a diagnosis of D-penicillamine-induced PPXE was established. Ophthalmological and cardiological evaluations were unremarkable.

Conclusions

PPXE is generally described as lacking systemic involvement and thus differs from hereditary pseudoxanthoma elasticum; however, elastic fiber abnormalities have been reported in organs beyond the skin. Although some authors recommend systemic evaluation and discontinuation of D-penicillamine after the onset of cutaneous manifestations, standardized guidelines for systemic assessment are currently lacking. Discontinuation of D-penicillamine does not necessarily result in regression of PPXE, and no effective treatment is currently available, as the drug primarily affects newly synthesized elastic fibers and elastin replacement is characterized by slow turnover. This case highlights a rare

cutaneous adverse effect of long-term D-penicillamine therapy and supports the potential value of multidisciplinary evaluation and systemic assessment, even in the absence of clinically evident systemic involvement.

Abstract N°: ID-431

Topic: Adverse drug reactions, TEN

Enfortumab Vedotin–Induced Cutaneous Toxicity: A Case Series

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Introduction

Enfortumab vedotin (EV) is an antibody–drug conjugate targeting Nectin-4 and has become an established therapeutic option for patients with advanced urothelial carcinoma. Cutaneous adverse events are among the most frequently reported toxicities during EV therapy and encompass a wide clinical spectrum, ranging from mild intertriginous eruptions to severe, potentially life-threatening blistering dermatoses.

The aim of this report is to describe the clinical presentation, management strategies, and outcomes of EV-induced cutaneous toxicity in two elderly patients with urothelial carcinoma.

Materials and Methods

The autors present two cases of cutaneous adverse reactions associated with enfortumab vedotin therapy in patients treated for urothelial carcinoma. Clinical features, laboratory abnormalities, dermatological interventions, and follow-up outcomes were retrospectively analyzed.

Results

The first case involved a 79-year-old woman with urothelial carcinoma of the right renal pelvis who developed erythematous and desquamative skin lesions predominantly affecting intertriginous areas following EV administration. Laboratory investigations revealed elevated inflammatory markers, leukocytosis, and mildly increased serum creatinine levels. Systemic corticosteroid therapy resulted in marked clinical improvement, allowing continuation of oncological treatment with prophylactic oral prednisone during subsequent EV cycles.

The second case concerned a 75-year-old man with urothelial carcinoma of the left ureter who presented with erythematous and infiltrative lesions in the axillary and inguinal regions, accompanied by blistering eruptions on the left forearm. No mucosal involvement was observed. A diagnosis of drug-induced exfoliative dermatitis was established, and combined systemic and topical corticosteroid therapy led to significant clinical improvement.

In both patients, skin toxicity exhibited a characteristic distribution, primarily involving large skin folds, with a potential risk of progression to more extensive and severe cutaneous reactions.

The clinical manifestations of enfortumab vedotin–induced cutaneous toxicity are illustrated in Figure 1.

Conclusions

Cutaneous adverse reactions associated with enfortumab vedotin demonstrate a characteristic clinical pattern with predominant involvement of intertriginous areas. Early dermatological evaluation and prompt initiation of corticosteroid therapy are essential to prevent progression to severe, generalized, or life-threatening skin reactions. Close

interdisciplinary collaboration is crucial to ensure patient safety while maintaining the effectiveness of oncological treatment.

Abstract N°: ID-493

Topic: Adverse drug reactions, TEN

DRESS syndrome in the setting of chemotherapy and renal failure: diagnostic pitfalls in a hemodialysis patient

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Introduction
Drug reaction with eosinophilia and systemic symptoms (DRESS) is a severe cutaneous adverse drug reaction characterized by delayed onset, systemic inflammation and potential multiorgan involvement. Diagnosis can be challenging in patients with hematological malignancies because infections, disease-related inflammation and polypharmacy may mimic DRESS.

Materials and Methods
We report a 47-year-old man with newly diagnosed multiple myeloma and end-stage kidney disease on chronic hemodialysis. He started a bortezomib-based regimen with supportive treatments including trimethoprim–sulfamethoxazole. Clinical features, laboratory findings, histopathology and outcome were reviewed. RegiSCAR criteria were applied to estimate diagnostic probability.

Results
Three weeks after treatment initiation, the patient developed a rapidly progressive pruritic morbilliform eruption beginning on the lower back and extending to the trunk and limbs. He had fever (38.4°C) and bilateral axillary and inguinal lymphadenopathy. Laboratory tests showed leukocytosis with eosinophilia (1.6×10^9/L), elevated inflammatory markers and mild hepatic cytolysis, while renal parameters remained stable under hemodialysis. Skin biopsy supported a drug-induced hypersensitivity reaction. RegiSCAR assessment was consistent with **probable** DRESS. All suspected drugs were discontinued and systemic corticosteroids (0.5 mg/kg/day prednisone equivalent) were initiated, leading to progressive clinical and biological improvement.

Conclusions
This case underlines that DRESS should be considered in hemodialysis patients receiving chemotherapy and multiple concomitant drugs. Applying validated criteria such as RegiSCAR, excluding competing diagnoses and promptly withdrawing suspected agents are key steps to prevent severe systemic complications.

Abstract N°: ID-508

Topic: Adverse drug reactions, TEN

Drug-Induced Sweet Syndrome Associated With Lorlatinib: Clinical and Pathophysiological Considerations

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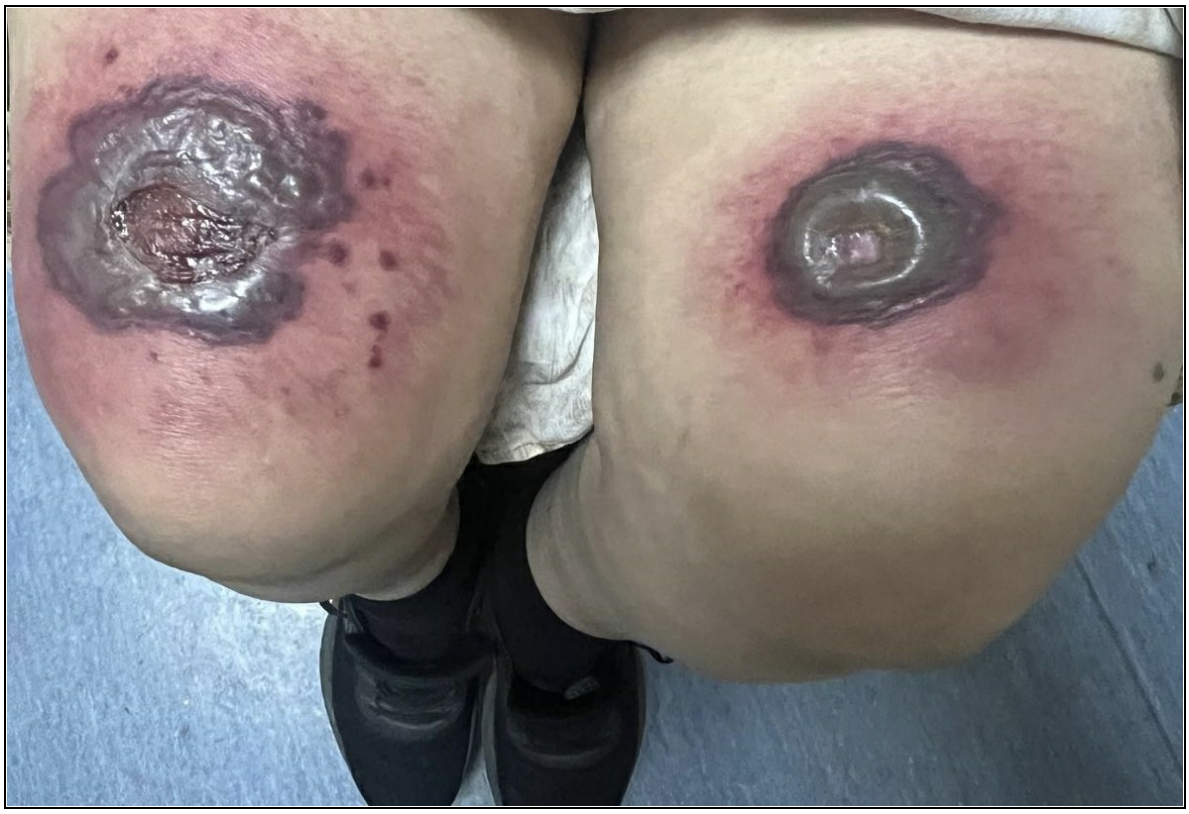
Introduction

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, is an inflammatory condition characterized by abrupt onset of fever, painful cutaneous lesions, and dense neutrophilic infiltrates on histopathology. It may occur in idiopathic, malignancy-associated, or drug-induced settings. Drug-induced Sweet syndrome has been reported with various medications, including antineoplastic agents; however, reports related to tyrosine kinase inhibitors remain limited. Lorlatinib is a third-generation anaplastic lymphoma kinase inhibitor used in metastatic lung adenocarcinoma. To date, Sweet syndrome has not been reported in association with third-generation tyrosine kinase inhibitors.

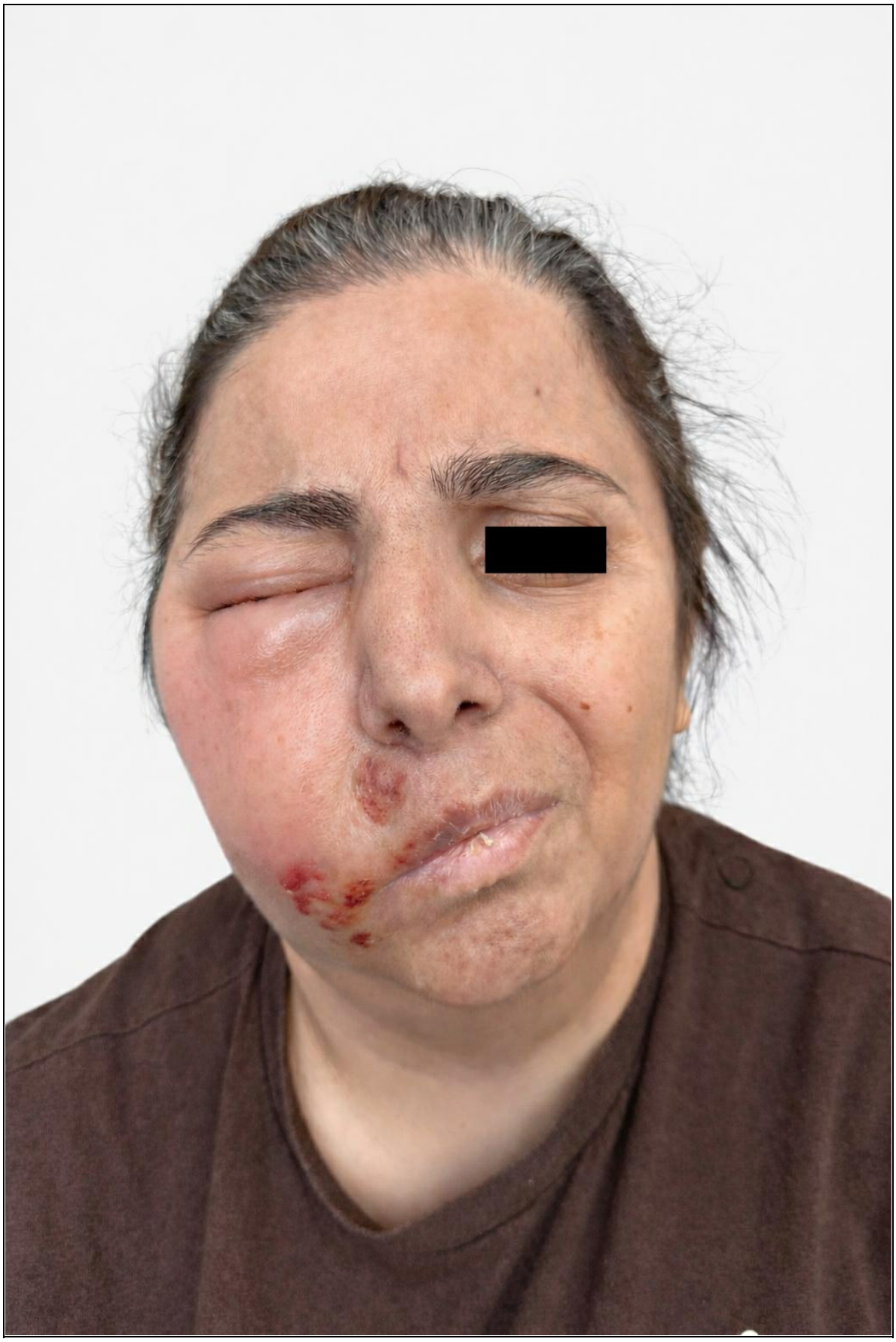
Materials and Methods

A 37-year-old woman presented with acute-onset painful, rapidly progressive cutaneous lesions and high-grade fever. Her medical history included metastatic lung adenocarcinoma in sustained radiologic remission under long-term lorlatinib therapy. Lorlatinib had been temporarily discontinued because of pancytopenia and was reintroduced at a reduced dose about eight weeks before symptom onset. Dermatologic examination revealed multiple tender violaceous plaques with central ulceration and peripheral hemorrhagic bullae on the extremities.



Results

Laboratory investigations showed elevated inflammatory markers with pancytopenia. Histopathology of punch biopsy specimens revealed a dense dermal neutrophilic infiltrate without vasculitis. Systemic corticosteroid therapy led to rapid clinical improvement and defervescence. During tapering, the patient developed right-sided facial edema with perinasal inflammatory lesions. Infectious, vascular, and angioedema-related causes were excluded. Re-escalation of systemic corticosteroids resulted in regression of facial edema; the course was complicated by transient ipsilateral facial nerve palsy, which improved under continued treatment.



Conclusions

In this patient, a drug-induced etiology was considered the most plausible explanation for Sweet syndrome. A paraneoplastic mechanism was unlikely given sustained complete oncologic remission for almost four years, whereas the close temporal relationship between lorlatinib interruption, reintroduction, and symptom onset supported a treatment-related process.

A possible pathophysiological explanation relates to lorlatinib-associated bone marrow toxicity. The patient had previously developed pancytopenia during lorlatinib therapy, followed by hematologic recovery and reinitiation; at presentation with Sweet syndrome, pancytopenia had again evolved. Increasing evidence indicates that Sweet syndrome may arise from localized, cytokine-driven neutrophilic inflammation independent of peripheral blood neutrophil counts. In this setting, pancytopenia may reflect bone marrow stress with cytokine imbalance—particularly involving IL-1β, IL-6, IL-8, and granulocyte colony-stimulating factor—creating a permissive inflammatory milieu. Such dysregulation may have contributed to disease onset and the severe inflammatory phenotype observed, including bullous lesions and marked facial edema.

Although the Naranjo Adverse Drug Reaction Probability Scale yielded a score of 3, the patient fulfilled diagnostic criteria for drug-induced Sweet syndrome. The Naranjo scale may have limited predictive value in antineoplastic drug reactions. Accordingly, a low score should be interpreted cautiously and considered alongside clinical history, temporal associations, histopathologic findings, and therapeutic response.

To our knowledge, this is the first reported case of Sweet syndrome associated with a third-generation tyrosine kinase inhibitor. This case underscores that Sweet syndrome may develop despite neutropenia. The presence of bullous cutaneous lesions and prominent facial edema highlights a severe inflammatory phenotype mimicking infectious, vascular, or angioedema-like conditions. Recognition of this adverse reaction is important for timely diagnosis and management in patients receiving targeted oncologic therapies.

Abstract N°: ID-547

Topic: Adverse drug reactions, TEN

Pemphigus or Pseudo-Pemphigus? Navigating Severe Oral Toxicities in the Era of Immunotherapy

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Introduction

The integration of immune checkpoint inhibitors (ICIs) into standard oncological care has led to a significant increase in the recognition of immune-related adverse events (irAEs). While cutaneous toxicities are frequently encountered, oral mucosal manifestations—specifically erosive and ulcerative phenotypes—remain relatively rare and diagnostically complex. Oral lichenoid mucositis represents the most common oral phenotype associated with anti-PD-1 therapy. However, when these reactions present in a severe, erosive form, they can clinically mimic immunobullous diseases such as pemphigus vulgaris or paraneoplastic pemphigus (PNP), particularly in patients with active underlying malignancies where clinical suspicion for paraneoplastic syndromes is high.

Materials and Methods

A 79-year-old woman with metastatic lung adenocarcinoma receiving pembrolizumab presented with a six-week history of progressive, painful oral ulceration that had caused marked functional impairment. Despite prior treatment for presumed oral candidiasis with both topical and systemic antifungals, her condition failed to improve. Clinical examination revealed extensive white plaques within the oral cavity, accompanied by haemorrhagic erosions and crusting of the lips and tongue. Preliminary blood tests and swabs for bacterial, viral, and fungal infections were negative. Histopathological analysis of oral biopsies demonstrated non-specific ulceration characterised by a dense, band-like lymphoplasmacytic infiltrate with occasional eosinophils. Periodic acid-Schiff with diastase staining for fungus and immunohistochemistry for cytomegalovirus (CMV) were negative.

Crucially, both direct immunofluorescence (DIF) and indirect immunofluorescence (IIF) studies were negative for intercellular or basement membrane zone autoantibodies, effectively ruling out the presence of circulating or tissue-bound antibodies against desmogleins or plakin proteins. A diagnosis of severe erosive lichenoid mucositis was made, and the patient was initiated on high-dose systemic steroids, leading to rapid clinical resolution of her symptoms. The treatment course was briefly complicated by CMV reactivation, successfully managed with antiviral therapy.

Results

Distinguishing severe oral lichenoid irAEs from paraneoplastic pemphigus is a critical diagnostic hurdle in the oncology population. While PNP is traditionally associated with refractory stomatitis and specific anti-plakin antibodies, ICI-induced lichenoid reactions are identified by their temporal relationship to immunotherapy and their typically robust response to corticosteroids. In this case, the histological features were not specific and only sparse squamous epithelium was present to evaluate intraepithelial findings. Together with the absence of intercellular IgG on DIF and the negative IIF screen, this effectively excluded pemphigus vulgaris and PNP. The patient's rapid response to immunosuppressive therapy further reinforced the diagnosis of a lichenoid-type irAE.

Conclusions

Severe erosive lichenoid mucositis is an under-recognised but significant irAE that can masquerade as more ominous immunobullous diseases. This case highlights the necessity of an early biopsy including both DIF and IIF to avoid misclassification. Recognising these severe oral reactions as potentially lichenoid rather than paraneoplastic enables early, appropriate treatment, and allows for more accurate prognostication of the patient's clinical course.

Abstract N°: ID-549

Topic: Adverse drug reactions, TEN

PLEVA associated with Ashwagandha supplementation

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Introduction

Pityriasis lichenoides et varioliformis acuta (PLEVA) is a rare skin disease of unknown origin[1]. Some reported cases have been associated with infections and drugs. Ashwagandha is an herb derived from the plant Withania somnifera, belonging to the nightshade family. Supplements based on extracts from its roots, leaves, or berries, often obtained online, have become increasingly popular in recent years for their purported effects on mood and metabolism. The herb is traditionally used to promote youthful vigour and well-being. Severe adverse effects of Ashwagandha have not yet been described.

Materials and Methods

Case report: A 63-year-old male patient was brought to my practice by ambulance in severe condition. His skin was covered with papules, nodules, and blisters. His hands and feet were swollen, causing impaired mobility. The patient complained of shortness of breath, fever, and dizziness. A biopsy showed lymphocytic infiltration mimicking lymphoma. CRP and WBC levels were elevated. Three weeks prior to the onset of PLEVA, the patient had started self-medication with an Ashwagandha supplement. He had attempted to improve his diabetic condition, along with his anxiety and sleep disorder. After he noticed significant relief from discomfort, he increased the daily dosage of his supplement from 2 to 6 capsules. The product he had purchased was not standardised. After discontinuing self-medication and receiving three weeks of prednisolone therapy, the skin lesions cleared.

Conclusions

Analysing plant extracts from Withania somnifera, more than 300 different compounds have been identified. Most of them belong to the withanolides, which have a steroid-like structure associated with lactones. These compounds are currently under intensive investigation because of their pharmacological potential[2]. Their effects both on metabolism and mood were demonstrated in double-blind trials[3]. Furthermore, nicotine was found in the plant. Dopamine release was stimulated. Serum cortisol levels were reduced. As Ashwagandha supplements are not approved as medicinal products, they are not under surveillance by health authorities. The extracts available online are not standardised. In Germany, the Bundesamt für Risikobewertung (BfR) has issued a warning regarding Ashwagandha supplements due to the lack of sufficient data.

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Abstract N°: ID-550

Topic: Adverse drug reactions, TEN

Allopurinol-Induced Toxic Epidermal Necrolysis: Case Management and Rapid Clinical Response

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Introduction
Toxic epidermal necrolysis (TEN) is a rare yet life-threatening severe cutaneous adverse reaction (SCAR), characterized by extensive epidermal detachment and mucosal involvement. Drugs remain the leading precipitating factor, with allopurinol being one of the most frequently implicated agents. Early diagnosis and prompt initiation of targeted systemic therapy are critical to reduce morbidity and mortality.

Materials and Methods
We present the inpatient management of a 77-year-old female who developed TEN following initiation of allopurinol therapy for gout. Clinical evaluation was supported by histopathological confirmation via punch biopsy. A multidisciplinary treatment approach was implemented, including systemic corticosteroids, intravenous immunoglobulin (IVIG) and adjunctive topical supportive measures.

Results

Three weeks after starting allopurinol, the patient presented to the emergency department with a rapidly progressive erythematous maculopapular eruption initially involving the trunk and subsequently becoming widespread with confluent lesions. She was initially monitored in the intensive care unit for three days. Her medical history was significant for chronic obstructive pulmonary disease, hypertension, and previous total thyroidectomy. On dermatological consultation, diffuse erythroderma, oral mucosal desquamation, and extensive crusted erosions on the face were observed. Differential diagnoses included DRESS syndrome, Stevens–Johnson syndrome, and TEN. Punch biopsy findings were consistent with TEN. The patient was transferred to our dermatology service, where she received intravenous methylprednisolone and IVIG at a dose of 0.5 g/kg, in addition to topical corticosteroids, mupirocin, local anesthetic preparations, and emollient therapy. Marked regression of cutaneous manifestations was achieved under combined systemic therapy. The major complication during hospitalization was the development of a rectus sheath hematoma, likely associated with enoxaparin prophylaxis initiated due to immobilization, while TEN-related lesions continued to improve.

Conclusions
TEN represents a dermatological emergency most commonly triggered by medications, necessitating immediate discontinuation of the offending agent and intensive supportive care. Allopurinol remains a well-established cause of SCAR, including TEN. IVIG is thought to contribute to disease control by inhibiting keratinocyte apoptosis, particularly when administered early in the disease course. In our patient, the combination of systemic corticosteroids and IVIG resulted in a rapid and significant clinical response, underscoring the importance of early recognition and aggressive multidisciplinary management in TEN.

Abstract N°: ID-559

Topic: Adverse drug reactions, TEN

Subcorneal pustular dermatosis: expanding the spectrum of dupilumab-associated cutaneous adverse effects

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Introduction

Dupilumab is a monoclonal antibody which inhibits IL-4 and IL-13 signaling, hence its use in a wide range of type 2 inflammatory diseases. Despite generally exhibiting a favorable safety profile, cutaneous adverse effects have been noted during dupilumab use, which dermatologists should be aware of. We report a rare case of subcorneal pustular dermatosis (SPD) arising in an adult patient during dupilumab therapy for chronic obstructive pulmonary disease (COPD).

Materials and Methods

A 52-year-old man presented with a 2-month history of a pruritic relapsing eruption affecting the trunk and extremities. Significant medical history included severe COPD, pulmonary and deep vein thrombosis and spinal canal stenosis. Dupilumab therapy had been initiated a year ago to treat his COPD, allowing for a slow corticosteroid tapering while successfully preventing exacerbations. Notably, a few weeks after initiation, he developed a cutaneous eruption diagnosed as acute generalized exanthematous pustulosis (AGEP) possibly related to trimetoprim/sulfamethoxazole.
The patient did not report any obvious triggers for his current skin eruption, denying the reintroduction of trimetoprim/sulfamethoxazole. Although he had achieved mild relief with mometasone furoate cream, frequent flare-ups of annular cutaneous lesions occurred. Physical examination showed round to oval erythematous plaques with pinpoint peripheral pustules distributed on his legs and poorly defined erythematous plaques lacking pustules on the chest.
Skin biopsy and laboratory investigations were performed under the clinical suspicion of SPD.

Results

Histopathological analysis of the skin biopsy revealed an intraepidermal subcorneal pustule which appeared to “sit” on the epidermis, along with a mild mixed perivascular inflammatory infiltrate in the superficial dermis, supporting the diagnosis of SPD. Direct immunofluorescence (DIF) was negative. In the light of these findings, the previous skin biopsy which led to the diagnosis of AGEP was reviewed, and it also showed features consistent with SPD. Blood laboratory investigations including serum immunoelectroforesis did not disclose any significant alterations.
Further management was discussed with the patient and his pulmonologist. Owing to the excellent respiratory response to dupilumab and acceptable control of the pruritus and the lesions with mometasone cream and tacrolimus ointment, it was decided to maintain dupilumab and topical treatment as required.

Conclusions

We report a new case of SPD that occurred in an adult patient during COPD therapy with dupilumab. We hypothesize that the first pustular eruption presented by our patient a few weeks after dupilumab initiation might have been the first flare-up of SPD. In this way, corticosteroid therapy, which was slowly tapered during the use of dupilumab, might have controlled the patient’s SPD until its complete withdrawal, leading to SPD recurrence.

A literature review on dupilumab's cutaneous adverse events (cAEs) was conducted. While head and neck dermatitis and psoriasis appeared to be the most frequent cAEs, only one case of SPD induced by dupilumab was reported. Another case of SPD successfully treated with abrocitinib was reported in a patient in whom dupilumab was started to treat suspected atopic dermatitis, although the nature of the rash might have been misdiagnosed. It has been proposed that dupilumab may result in an upregulation of Th1/Th17 pathways leading to neutrophilic dermatosis.
These results suggest SPD may be a rare cAEs of dupilumab therapy, widely used for cutaneous and noncutaneous inflammatory diseases. Our case highlights the need of a low threshold for biopsy when facing pustular and/or annular eruptions in patients treated with dupilumab, and the importance of shared decision-making in collaboration with other specialists as needed.

Abstract N°: ID-596

Topic: Adverse drug reactions, TEN

Cutaneous toxicities associated with amivantamab in patients with non-small cell lung cancer: A systematic review

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Introduction
Amivantamab is an EGFR and MET targeting bispecific antibody used for the treatment of non-small cell lung cancer (NSCLC) in patients with exon 20 mutations. Cutaneous adverse events (cAEs) are commonly reported with amivantamab, particularly in clinical trial settings. However, the overall frequency and spectrum of cAEs across clinical trials have not been systematically summarized and various non-reported toxicities have emerged with its real-world use. The aim of this systematic review was to synthesize available evidence on amivantamab-associated cAEs in patients with NSCLC.

Materials and Methods
A systematic literature search was conducted using MEDLINE/PubMed, Scopus and Web of Science libraries up to 4 December 2025, following the PRISMA guidelines (Full protocol at PROSPERO: CRD420251245455). Clinical trials and real-world observational studies reporting cAEs in patients treated with amivantamab were included. Two independent authors performed study screening and data extraction, with disagreements resolved by a third author.

Results
In total, 1343 publications were identified and after the removal of duplicates, 753 were screened based on title and abstract. Full-text screening was performed for 131 articles, from which, seven clinical trials and one real-world observational study were identified and included in the systematic review. Paronychia was the most common cAE reported across clinical trials. The rates of paronychia were higher for amivantamab combined with lazertinib [625/1090 (57.3%)] compared to amivantamab plus chemotherapy [133/281 (47.3%)] and amivantamab monotherapy [104/258 (40%)]. Grade>3 paronychia was reported in up to 12% in amivantamab plus lazertinib treated patients in the phase 3 MARIPOSA study. Acneiform rash or dermatitis was also common, affecting 73/281 (25.9%) of patients treated with amivantamab plus chemotherapy versus 260/837 (31.1%) of patients under amivantamab plus lazertinib with around 5% of them being grade ≥ 3. Importantly, the addition of chemotherapy to amivantamab and lazertinib did not substantially reduce the incidence of cutaneous toxicities, with similarly high rates of paronychia (50.6%) and acneiform dermatitis (23.6%) observed. Other frequently reported cAEs included pruritus, xerosis, and skin fissures, with severe events being uncommon (≤1%). In the phase 3 PALOMA study, subcutaneous and intravenous formulations of amivantamab combined with lazertinib demonstrated comparable rates of cAEs, indicating no relevant impact of administration route on dermatologic toxicity. In contrast to clinical trial data, real-world evidence suggests a different toxicity pattern. In one real-world observational study, acneiform rash was more frequent than paronychia (68.5% vs 30.1%).3 Moreover, erosive pustular dermatosis (15.1%) and ulcerative skin lesions (4.2%) were reported in a notable proportion of patients, toxicities that had not been adequately characterized in clinical trials.

Conclusions
Overall, available evidence indicates that amivantamab combined with lazertinib is associated with higher rates of cutaneous toxicities, particularly paronychia and acneiform rash. While this may be related to enhanced EGFR inhibition,

direct cross-trial comparisons should be interpreted cautiously due to differences in study design and patient populations. Importantly, the spectrum of amivantamab-associated cAEs appears to be expanding as real-world experience increases. Further large-scale observational and pharmacovigilance studies are needed to better define the full spectrum of amivantamab-related dermatologic toxicities and inform evidence-based management strategies.

Abstract N°: ID-665

Topic: Adverse drug reactions, TEN

Iatrogenic Cushing Syndrome Induced by Topical Corticosteroids in a Child with Generalized Pustular Psoriasis

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Introduction
Topical corticosteroids- induced Cushing syndrome is an exceedingly rare adverse effect; however, most cases reported in the literature involve pediatric patients.

Materials and Methods
We report a case of iatrogenic Cushing syndrome in a 10-year-old child following topical corticosteroid therapy for generalized pustular psoriasis.

Results
A 10-year-old girl was followed in our dermatology department for generalized pustular psoriasis. She presented with annular erythematous, scaly plaques bordered by aseptic pustules, involving approximately 80% of the body surface area. Treatment with acitretin was initiated at a dose of 0.4 mg/kg/day. Four months later, the patient was admitted to the ophthalmology department for diplopia associated with sixth cranial nerve palsy and papilledema. After extensive investigations excluded alternative etiologies, acitretin was retained as the most likely cause, and the treatment was discontinued. Following this event, the patient was lost to follow-up for one year. She subsequently returned with a complaint of weight gain and recent onset of extensive stretch marks. Physical examination revealed a moon-shaped face, truncal obesity, and violaceous striae. Further history revealed that her mother had been applying excessive amounts of potent topical corticosteroids, estimated at three to five tubes daily, to manage recurrent flares of generalized pustular psoriasis, driven by fear of systemic therapies after the ophthalmological adverse event. Morning serum cortisol levels (8 a.m.) were markedly suppressed at 3.94 nmol/L, confirming the diagnosis of iatrogenic Cushing syndrome and adrenal insufficiency. Hydrocortisone replacement therapy was initiated, and infliximab was then considered for the management of her generalized pustular psoriasis.

Conclusions
Iatrogenic Cushing syndrome and secondary adrenal insufficiency resulting from topical corticosteroid use are exceptionally uncommon adverse effects. Risk factors include the use of high-potency corticosteroids, application over large body surface areas, and prolonged treatment duration. Pediatric patients have a particular vulnerability due to increased percutaneous absorption associated with skin immaturity, a higher body surface area–to–weight ratio, and the high prevalence of dermatologic conditions requiring topical corticosteroid therapy, such as diaper dermatitis, atopic dermatitis, and pediatric psoriasis. In addition, acitretin-induced skin fragility during psoriasis treatment, as observed in our patient, may further potentiate systemic absorption of topical corticosteroids. This case highlights the need for heightened vigilance when prescribing topical corticosteroids in the pediatric population, with careful consideration of factors such as skin involvement extent, treatment duration, and corticosteroid potency, to minimize the risk of systemic absorption and iatrogenic Cushing syndrome.

Abstract N°: ID-775

Topic: Adverse drug reactions, TEN

Clozapine-Associated Papulopustular Drug Eruption: A Diagnostic and Therapeutic Challenge

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Introduction
Clozapine is reserved for refractory schizoaffective disorders but is associated with rare cutaneous drug reactions that may mimic infectious, inflammatory, or immune-mediated dermatoses. Diagnostic uncertainty is heightened in patients with concomitant chronic inflammatory skin disease.

Materials and Methods
A 47-year-old man with schizoaffective disorder developed an acute, widespread papulopustular eruption after clozapine initiation and titration. The clinical picture was complicated by severe pre-existing hidradenitis suppurativa (HS), with active abscesses and persistently elevated inflammatory markers. Differentials included guttate psoriasis, cellulitis, HS-related infection, acute generalised exanthematous pustulosis (AGEP), or clozapine-induced drug eruption. Systemic features, which could be attributed to infection or drug hypersensitivity, added further diagnostic complexity.

Microbiological investigations identified no causative pathogen. Skin biopsy demonstrated spongiotic dermatitis with subepidermal oedema and a mixed inflammatory infiltrate, consistent with a pustular drug eruption rather than psoriasis or AGEP. Dermatopathology consensus concluded this was consistent with a clozapine-associated drug reaction.

Results
Clozapine was discontinued, resulting in gradual resolution of cutaneous and systemic symptoms. Given the history of treatment-resistant psychosis and lack of alternative therapeutic options, clozapine was gradually reintroduced following multidisciplinary discussion, without recurrence.

Conclusions
This case underscores the diagnostic uncertainty surrounding clozapine-associated cutaneous reactions in the context of chronic inflammatory dermatoses. It highlights the importance of histopathology and multidisciplinary collaboration in distinguishing drug reactions and guiding management when psychiatric risk is high. The absence of recurrence after clozapine reintroduction does not exclude a drug-related cause and may reflect transient immune activation during systemic inflammation, highlighting the potential for safe re-titration under more stable and controlled clinical conditions.

Abstract N°: ID-798

Topic: Adverse drug reactions, TEN

Erythema Multiforme Induced by a Bee Sting: A Case Report

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Introduction

Erythema multiforme is an acute immune-mediated dermatological condition affecting the skin and mucous membranes. It predominantly occurs in young adults, with a slight female predominance. In approximately 90% of cases, it is triggered by an infectious cause, most commonly herpes simplex virus infection (70–80%). More rarely, medications, neoplasms, autoimmune diseases, or environmental factors may be involved.

We report the case of a patient who developed **erythema multiforme following a bee sting**.

Materials and Methods

We report the case of a **59-year-old patient** with **no significant past medical history**, including no history of herpes simplex infection, respiratory infection, or recent drug intake. The patient presented with **symmetrical target-like skin lesions topped with bullae**, predominantly involving the **acral areas and the face**, associated with **oral and scrotal erosions**, occurring **three days after a bee sting**.

Laboratory investigations revealed **microcytic hypochromic anemia** and an **elevated C-reactive protein level (91 mg/L)**. Respiratory PCR testing was negative, viral serologies were negative, and tumor markers were within normal limits.

Histopathological examination of a skin biopsy was consistent with **erythema multiforme**.

The patient was treated with **amoxicillin–clavulanic acid (1 g every 8 hours)** for secondary bacterial infection, **paracetamol for fever**, and **daily local wound care**. The clinical course was **favorable**, with marked improvement and progressive resolution of skin lesions.

Results

Erythema multiforme is a **cell-mediated immune cutaneous and mucosal reaction**, most commonly triggered by infections—particularly **herpes simplex virus** and **Mycoplasma pneumoniae**—or, less frequently, by medications. Lesions typically appear within **72 hours**, with a **symmetrical and acral distribution**.

In our case, the diagnosis of **erythema multiforme following a bee sting** was established based on the clinical history, the characteristic skin lesions, and the exclusion of infectious causes. To date, only a single case of erythema multiforme with a delayed onset of one week has been reported in the literature. These observations suggest that **insect venom, including bee venom, may act as a rare immunological trigger of erythema multiforme**.

Management remains essentially **symptomatic**, including the use of **antihistamines**, **topical or systemic corticosteroids depending on disease severity**, and **appropriate local wound care** to promote lesion resolution and prevent secondary complications.

Conclusions

Bee stings represent a **rare but possible trigger of erythema multiforme** in sensitized individuals. The diagnosis relies on the presence of **typical target-like lesions** and the exclusion of other potential etiologies, while management remains primarily **symptomatic**.

Abstract N°: ID-864

Topic: Adverse drug reactions, TEN

Nivolumab-induced lichen planus masquerading as a psoriatic flare

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Introduction
Immune checkpoint inhibitors (ICIs) have revolutionised oncology, but their cutaneous immune-related adverse events (irAEs) can present diagnostic challenges. We present a case illustrating how pre-existing skin disease can mask an alternative diagnosis, with important implications for management.

Materials and Methods
A single case report, with retrospective chart review and key learning points identified.

Results
We describe a 59-year-old woman with metastatic colorectal cancer who developed a widespread, pruritic guttate eruption with hypertrophic plaques over the dorsal hands and anterior shins ten days after her sixth cycle of nivolumab. With a history of psoriasis, the eruption was initially managed as a presumed flare with emollients and topical calcipotriol–betamethasone. At review two weeks later, scaling had improved revealing whitish streaks on the plaques. Oral Examination demonstrated lacy, reticular white patches, prompting biopsy. Histopathology showed wedge-shaped hyper-granulosis, sawtooth acanthosis, and a band-like lymphocytic infiltrate without psoriasiform features or eosinophils consistent with lichen planus. Potent topical corticosteroids and doxycycline were commenced, achieving control without interrupting nivolumab. Interval imaging confirmed partial tumour response.

Conclusions
This case highlights that ICIs can induce lichenoid eruptions closely resembling psoriasis, emphasising the value of meticulous mucocutaneous examination and histopathologic confirmation for accurate irAE classification. The presence of mucosal lesions and Wickham striae favour a diagnosis of true lichen planus over a non-specific lichenoid drug eruption. The patient-reported history of psoriasis led to an assumption of a psoriasis flare.

In the era of onco-dermatology, accurate recognition of subtle morphologic clues is essential. Careful clinicopathologic correlation not only guides appropriate treatment but also preserves vital oncologic options and optimises patient quality of life.

Abstract N°: ID-866

Topic: Adverse drug reactions, TEN

Risankizumab-Associated Lupus: A rare adverse event in IL-23 Inhibition

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Introduction

Drug-induced lupus (DIL) is an uncommon adverse reaction characterized by the development of clinical and serological features resembling systemic lupus erythematosus (SLE), triggered by exposure to certain medications. Although historically associated with drugs such as hydralazine or procainamide, cases linked to targeted biological therapies—including IL-17 and IL-23 inhibitors—have been increasingly reported over the past decade. Risankizumab, an anti–IL-23 monoclonal antibody used in psoriasis, has only rarely been associated with DIL. The coexistence of other potentially immunomodulatory treatments, such as latent tuberculosis chemoprophylaxis, may further complicate identification of the causal agent.

Materials and Methods

We describe the case of a woman with moderate-to-severe psoriasis treated with risankizumab who was also receiving chemoprophylaxis with isoniazide following a positive Quantiferon test. After several months of combined therapy, she developed inflammatory arthralgias and arthritis, marked fatigue, and laboratory evidence of hematologic involvement with lymphopenia and anemia. Immunological testing revealed positive ANA, elevated anti–double-stranded DNA antibodies, complement consumption, and a positive lupus anticoagulant, while antihistone antibodies were negative. She had no prior history of autoimmune disease. These findings raised suspicion of either drug-induced lupus or a flare of previously quiescent systemic lupus erythematosus. Risankizumab was discontinued, and antituberculosis prophylaxis was completed. To maintain psoriasis control, ustekinumab—an IL-12/23 inhibitor with a more established safety profile in this setting—was initiated.

Results

Following withdrawal of risankizumab, the patient experienced progressive improvement in systemic symptoms and laboratory abnormalities, with a trend toward normalization of immunological markers. No further lupus flares were observed during follow-up. Switching to ustekinumab allowed adequate control of psoriasis without recurrence of autoimmune manifestations. However, the simultaneous exposure to risankizumab and isoniazide complicates the ability to definitively determine which treatment acted as the primary trigger of the lupus-like syndrome

Conclusions

Risankizumab-induced lupus is an exceptional adverse event but should be considered in patients receiving IL-23 inhibitors who develop symptoms compatible with SLE. Concomitant treatments, such as latent tuberculosis chemoprophylaxis, may obscure causal attribution. Discontinuation of the suspected agent and close clinical and serological monitoring are essential for resolution. Ustekinumab may represent a safe therapeutic alternative for maintaining psoriasis control in these patients

Abstract N°: ID-910

Topic: Adverse drug reactions, TEN

Immunotherapy-induced vitiligo in metastatic melanoma

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Introduction
Immune checkpoint inhibitors targeting the PD-1 pathway have become a cornerstone in the treatment of metastatic melanoma. Cutaneous immune-related adverse events are frequent, among which vitiligo-like depigmentation is particularly associated with melanoma and may reflect a strong antitumor immune response. We aim to report a case of immunotherapy-induced vitiligo occurring during pembrolizumab treatment for metastatic melanoma and to highlight its clinical relevance.

Materials and Methods
Case report.

Results
We report the case of a 56-year-old patient treated for plantar melanoma with lymph node metastasis. The patient was started on pembrolizumab at a dose of 200 mg every three weeks. After the fifth treatment cycle, the patient developed hypopigmented facial lesions, initially localized to the temporo-frontal region. With continued treatment, the lesions progressively extended to involve the entire face and acral areas. Dermoscopic examination revealed homogenous structereless areas with sharp borders as well as multiple perifollicular white dots and leuckotrichia, without inflammatory or infectious features. The clinical course and temporal relationship with pembrolizumab supported the diagnosis of immunotherapy-induced vitiligo.

Conclusions
Melanoma-associated vitiligo represents a characteristic immune-related adverse event in melanoma patients treated with anti-PD-1 agents. Recognition of this manifestation is essential to avoid unnecessary investigations or treatment discontinuation. Importantly, vitiligo may represent a favorable prognostic marker reflecting an effective immune response against melanoma cells.

Abstract N°: ID-912

Topic: Adverse drug reactions, TEN

Atypical SDRIFE Associated with Hepatitis C Antiviral Treatment

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Introduction

Baboon syndrome, recently renamed Symmetrical Drug-Related Intertriginous and Flexural Exanthema (SDRIFE), is a flexural drug eruption characterized by symmetric erythematous lesions predominantly affecting the gluteal and intertriginous areas. It is most frequently reported with beta-lactam antibiotics, but other drugs—including certain antifungals, antivirals, and monoclonal antibodies—have been implicated. Very few cases of SDRIFE related to direct-acting antivirals (DAAs) for hepatitis C have been described to date.

Figure 1: Erythematous-squamous plaque topped with papules and hemorrhagic crusts, symmetrically located in the inguinal region.

Materials and Methods

A 38-year-old man with end-stage renal disease on dialysis and incidentally diagnosed hepatitis C developed a pruritic

rash one week after starting sofosbuvir and daclatasvir. Clinical examination revealed symmetric erythematous-squamous plaques with overlying papules and hemorrhagic crusts in axillary and inguinal folds, with satellite lesions on the abdomen and back. No mucosal involvement, facial edema, lymphadenopathy, or systemic signs were noted. Laboratory tests, including complete blood count and liver function, were normal. The diagnosis of SDRIFE was suspected based on clinical and chronological criteria. The antiviral therapy was discontinued, and the case was reported to pharmacovigilance. Symptomatic treatment with topical corticosteroids, barrier creams, and local care was initiated.

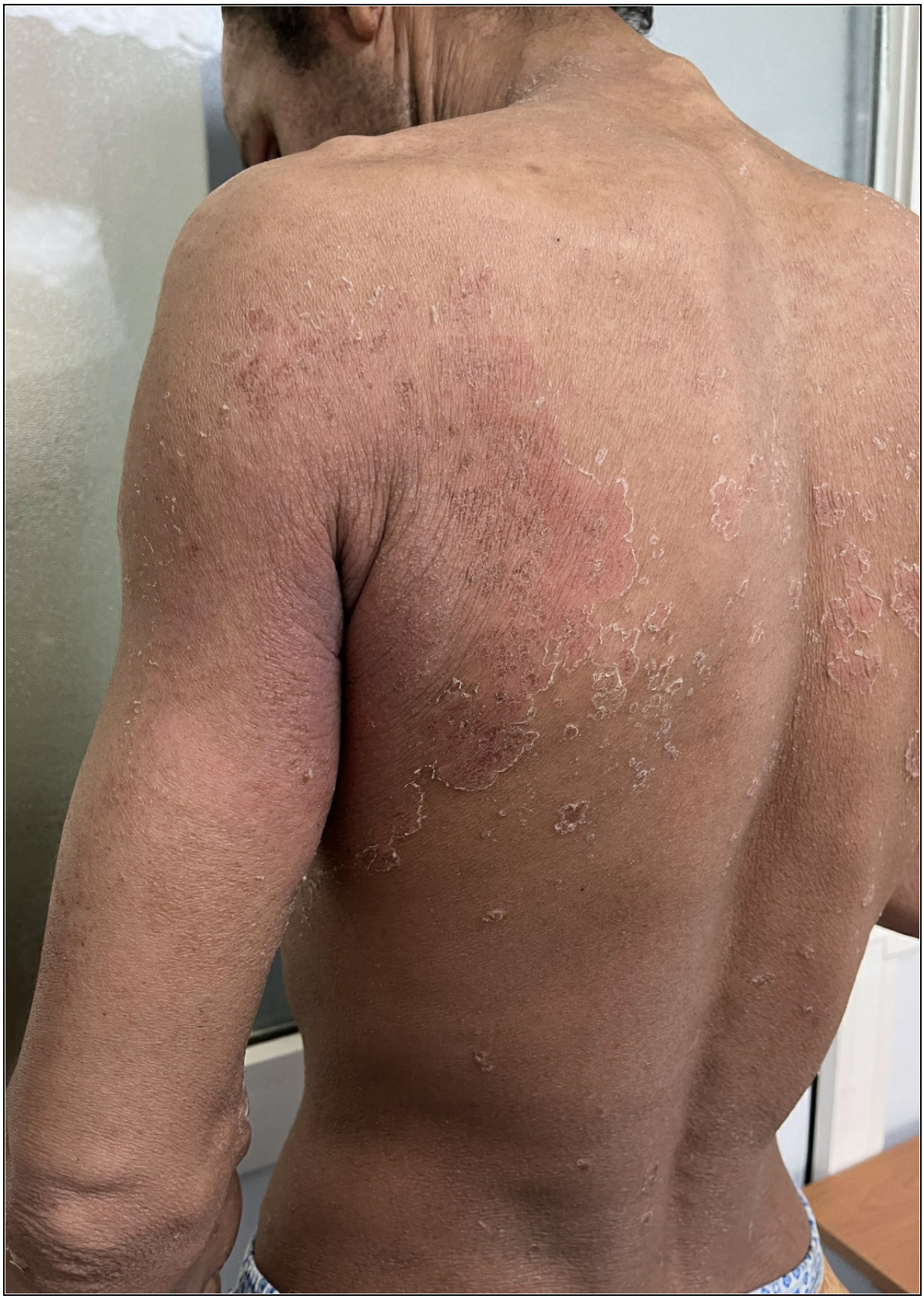

Figure 2: Erythematous-squamous plaques topped with papules and hemorrhagic crusts, symmetrically located in the axillary region, with satellite lesions on the back.

Results

Following discontinuation of the offending drugs and symptomatic management, the patient showed rapid clinical improvement with progressive resolution of the lesions. Diagnosis was based on the characteristic symmetric flexural distribution and temporal relationship with drug exposure.

Conclusions

SDRIFE should be suspected in any symmetric intertriginous eruption occurring after systemic drug exposure, even outside the typical beta-lactam class. This case highlights the need for vigilance regarding direct-acting antivirals for hepatitis C, particularly in vulnerable populations such as dialysis patients, and underscores the importance of reporting suspected reactions to pharmacovigilance to improve understanding of this under-recognized entity.

Abstract N°: ID-968

Topic: Adverse drug reactions, TEN

The Spectrum of Hypersensitivity: Two Cases of Lyell-DRESS Overlap Following Allopurinol Administration

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Introduction

Severe drug hypersensitivity reactions constitute a clinical spectrum ranging from isolated cutaneous signs to multi-organ involvement that poses a life-threatening risk. Among them, the DRESS syndrome and Lyell syndrome are two distinct entities, both pathophysiologically and clinically, but they can coexist. We report two cases of Lyell-DRESS overlap syndrome triggered by allopurinol.

Materials and Methods

Case 1 :

A 58-year-old female patient was hospitalized for a polymorphic cutaneous-mucous rash that appeared four weeks after the introduction of allopurinol. Examination revealed a maculopapular rash with purpuric areas, extensive superficial skin detachment 27%, and involvement of ocular, oral, and genital mucosa, along with fever and general condition deterioration. The suspected medication was discontinued; initial management included rehydration, appropriate antibiotic therapy, and prednisone 1 mg/kg/day. The initial assessment showed acute renal failure, severe inflammatory syndrome (CRP 202 mg/L), sepsis due to Klebsiella pneumoniae with metabolic acidosis, thoracic interstitial syndrome, and an estimated life-threatening risk of 52% (SCORTEN 4). Skin biopsy confirmed toxic epidermal necrolysis. The course of the condition was marked by angioedema, major eosinophilia, and hepatic cytolysis, leading to the diagnosis of DRESS-Lyell overlap (RegiSCAR 5), with respiratory distress requiring non-invasive ventilation ; fibroscopy revealed extensive mucosal detachment. The outcome was unfavorable, resulting in the patient's death due to intra-alveolar hemorrhage.

Case 2 :

An 80-year-old male patient was hospitalized for a polymorphic cutaneous-mucous rash that appeared three weeks after the introduction of allopurinol. Examination showed cocaroid blister lesions with superficial skin detachment 45%, and synechial ocular and oral mucosal involvement, accompanied by fever and deterioration of general condition. Allopurinol was discontinued, and hydro-electrolytic resuscitation with prednisone 1 mg/kg/day was initiated. The initial assessment indicated acute renal failure (KDIGO 2), severe inflammatory syndrome, hepatic cytolysis, and an estimated life-threatening risk of 52% (SCORTEN 4). Skin biopsy confirmed toxic epidermal necrolysis. The course was characterized by eosinophilia and proteinuria >0.5 g/24 h, leading to the diagnosis of DRESS-Lyell overlap (RegiSCAR 4). The outcome was favorable, with notable clinical and biological improvement.

Results

Allopurinol-induced Lyell-DRESS overlap may involve genetic susceptibility (the HLA-B58:01 allele) and delayed hypersensitivity combining the activation of cytotoxic CD8+ and CD4+ T lymphocytes. The initial diagnosis of Lyell syndrome was based on extensive multifocal erosive cutaneous-mucous involvement and epidermal detachment. However, the atypical progression, particularly angioedema, hepatic cytolysis, and eosinophilia, suggested an

association between Lyell and DRESS. According to the RegiSCAR group, an overlap syndrome can be suspected when a patient meets the criteria (probable or certain case) for at least two severe drug eruptions, as observed in our patients. A review of the literature identified allopurinol, carbamazepine, and sulfamethoxazole/trimethoprim as the most implicated drugs, underscoring the importance of monitoring these agents in this context. The FDA closely monitors the use of allopurinol, requiring clear information through official labels regarding potential dangers, particularly severe cutaneous reactions. Current guidelines aim to ensure the quality of formulations while proactive integration of pharmacogenetic data (such as HLA-B*58:01) is promoted by third-party organizations to secure therapy in at-risk populations.

Conclusions

Our two cases describe a rare allopurinol-associated DRESS overlap syndrome. This highlights the importance of suspecting overlaps and acting early when overlapping clinical or histopathological features are present.

Abstract N°: ID-1052

Topic: Adverse drug reactions, TEN

When drug reactions persist: a case of allopurinol-induced Lyell–DRESS overlap syndrome

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Introduction

Severe drug hypersensitivity reactions represent a clinical spectrum ranging from isolated cutaneous manifestations to life-threatening multiorgan involvement. Among these reactions, DRESS syndrome and Lyell syndrome are two well-defined entities, distinct both physiopathologically and clinically, yet they may coexist. We report a case of allopurinol-induced Lyell–DRESS overlap syndrome.

Materials and Methods

A 58-year-old woman was hospitalized for a polymorphous cutaneous and mucosal eruption that developed four weeks after the initiation of allopurinol. Physical examination revealed a widespread macular exanthema with purpuric areas, associated with superficial skin detachment involving 27% of the body surface area, as well as ocular, oral, and genital mucosal involvement, fever, and impaired general condition.

The suspected drug (allopurinol) was discontinued, and initial management included appropriate rehydration, targeted antibiotic therapy, and prednisone at a dose of 1 mg/kg/day. Initial investigations revealed acute kidney injury, a severe inflammatory syndrome (CRP 202 mg/L), Klebsiella pneumoniae septicemia with metabolic acidosis, a thoracic interstitial syndrome, and a life-threatening risk estimated at 52% (SCORTEN 4).

Skin biopsy confirmed the diagnosis of toxic epidermal necrolysis. The clinical course was marked by the development of angioedema, marked hypereosinophilia, and hepatic cytolysis, leading to the diagnosis of DRESS–Lyell overlap syndrome (RegiSCAR score 5), associated with respiratory distress requiring non-invasive ventilation. Fiberoptic examination revealed extensive mucosal detachment. The outcome was unfavorable, resulting in the patient's death due to intra-alveolar hemorrhage.

Results

Allopurinol-induced Lyell–DRESS overlap syndrome is thought to involve a genetic susceptibility, particularly the HLA-B*58:01 allele, and a delayed hypersensitivity reaction combining the activation of cytotoxic CD8+ T lymphocytes and CD4+ T cells. The initial diagnosis of Lyell syndrome was based on extensive multifocal erosive cutaneous and mucosal involvement with epidermal detachment. However, the atypical disease course, notably the occurrence of angioedema, hepatic cytolysis, and hypereosinophilia, suggested an overlap between Lyell syndrome and DRESS.

According to the RegiSCAR group, an overlap syndrome may be suspected when a patient meets the criteria (probable or definite case) for at least two severe cutaneous adverse drug reactions, as observed in our patient. A literature review of 22 reported cases identified allopurinol, carbamazepine, and sulfamethoxazole/trimethoprim as the most frequently implicated drugs, highlighting the importance of careful monitoring of these medications in this context.

Conclusions

Our case report describes a rare instance of NET–DRESS overlap attributed to allopurinol, highlighting the importance of suspecting overlaps and acting promptly when overlapping clinical or histopathological features are present.

Abstract N°: ID-1055

Topic: Adverse drug reactions, TEN

Severe mucocutaneous and systemic toxicity induced by methotrexate

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Introduction
Methotrexate (MTX) is widely used in dermatology to treat autoimmune diseases. Although generally well-tolerated at low doses, MTX can cause severe mucocutaneous and systemic toxicity, particularly in the presence of dosing errors, renal impairment, or drug interactions. This case aims to highlight the diagnostic and therapeutic challenges of MTX-induced toxicity and the need for prompt recognition and intervention.

Materials and Methods
We report a clinical case of severe mucocutaneous and systemic toxicity induced by MTX, due to a dosing error.

Results
A 90-year-old institutionalized polymorbid woman undergoing low-dose MTX treatment for long standing arthritis, presented to the emergency department with painful erosions and necrotic crusts on the oral mucosa, in addition to purpuric lesions on the face and hemorrhagic pseudovesicular papules on the trunk. Laboratory tests revealed acute pancytopenia. A comprehensive clinical evaluation and detailed anamnesis uncovered an error in MTX dosage. MTX was discontinued immediately, and rescue therapy with folinic acid was initiated in combination with supportive care.

Conclusions
Although uncommon at low doses, MTX toxicity can lead to life-threatening complications. Dermatologists should remain vigilant for early signs of toxicity, especially in elderly or polymedicated patients. Proper patient education, regular monitoring, and prompt therapeutic measures are essential to improve outcomes.

Abstract N°: ID-1059

Topic: Adverse drug reactions, TEN

Stevens–Johnson Syndrome with Acral Purpuric Targetoid Lesions: A Diagnostic Pitfall with Erythema Multiforme Major

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Introduction

Stevens–Johnson syndrome (SJS) is a rare, potentially life-threatening drug reaction. Early presentations with acral purpuric targetoid lesions and a negative Nikolsky sign may mimic erythema multiforme major (EMM), making clinicopathologic correlation essential.

Materials and Methods

A 29-year-old man with chronic kidney disease and hypertension (on stable antihypertensive therapy) and a history of recurrent herpes labialis developed acute mucosal involvement a few days after starting new medications, followed by a rapidly progressive skin eruption about one week after drug introduction. He presented with fever (38.5°C), painful oral erosions with pseudomembranes, hypersalivation and odynophagia, conjunctival hyperemia, and genital mucosal lesions. Cutaneous examination showed confluent purpuric macules with pseudo-targetoid lesions, predominantly acral with marked palmoplantar involvement; a few palmar blisters were noted. Nikolsky sign was negative. Total involved body surface area was estimated at ~20%, with <1% frank epidermal detachment (and ~2% detachable palmar areas).

Infectious work-up was negative (including HIV and viral hepatitis); HHV-6/HHV-7 PCR was negative.HSV PCR was negative. Chest X-ray was normal. Skin biopsy revealed epidermal necrosis with subepidermal cleavage and a moderate perivascular lymphohistiocytic infiltrate, consistent with SJS. Direct immunofluorescence was negative, arguing against autoimmune blistering disease.

Management and outcome

The suspected culprit drugs were discontinued and the patient received supportive care (oral rinses, local wound care, barrier/healing creams, and ocular lubrication). Clinical improvement started by day 5, with complete healing by day 25, leaving only post-inflammatory hyperpigmentation and no sequelae. Pharmacovigilance assessment considered ciprofloxacin the most likely trigger (followed by febuxostat).

Results

This case underscores a clinically important pitfall: acral purpuric pseudo-targets with a negative Nikolsky sign can strongly suggest EMM, especially in a patient with a history of herpes labialis. However, several elements favored SJS: early, multi-site mucosal involvement (oral, ocular, genital) with fever, rapid progression, and—crucially—histology showing epidermal necrosis with subepidermal cleavage, while direct immunofluorescence was negative, excluding autoimmune blistering disease. The negative HSV PCR further argued against active HSV-driven EMM, supporting a drug-triggered SJS phenotype. Importantly, this presentation illustrates that SJS may occur with minimal epidermal detachment yet significant mucositis, and should prompt immediate withdrawal of suspected drugs, supportive care, and pharmacovigilance reporting. The favorable evolution under symptomatic management, without sequelae, highlights the benefit of early recognition and appropriate multidisciplinary monitoring.

Conclusions

In targetoid acral eruptions with mucositis, SJS must be considered even when epidermal detachment is minimal. Biopsy, direct immunofluorescence, and targeted infectious work-up help secure the diagnosis and avoid misclassification as EMM.

Abstract N°: ID-1066

Topic: Adverse drug reactions, TEN

From Protection to Peril: Severe Cutaneous Drug Reaction Triggered by Co-trimoxazole in Advanced HIV

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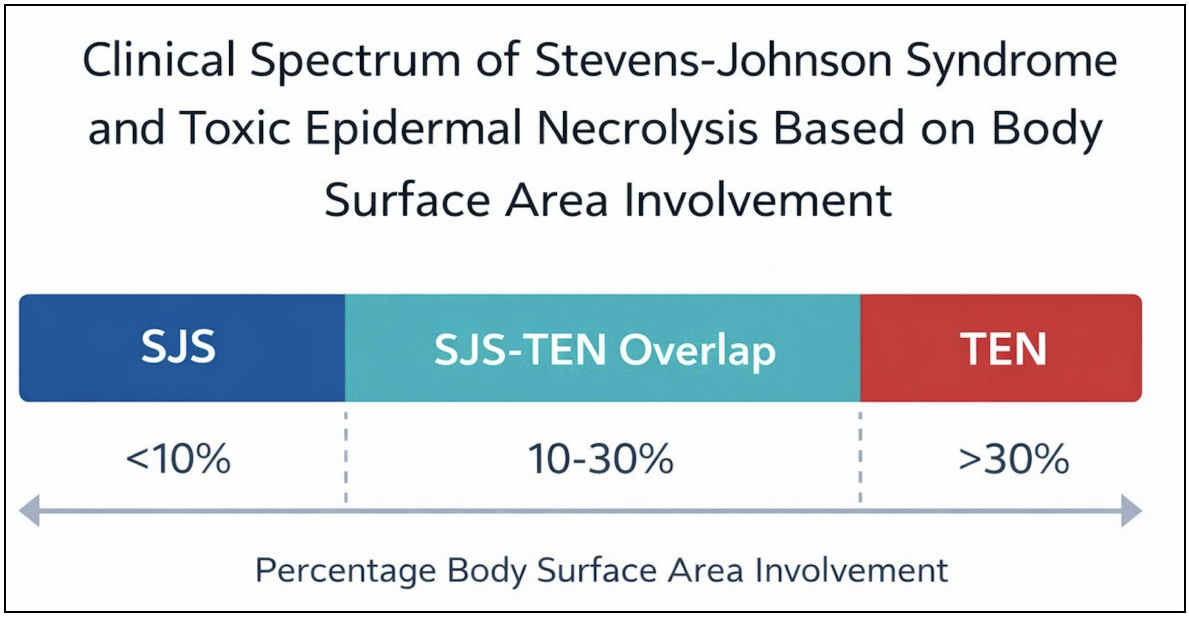
Introduction

Stevens-Johnson syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are severe, life-threatening mucocutaneous adverse drug reactions characterized by widespread keratinocyte apoptosis resulting in epidermal detachment. They are classified based on body surface area (BSA) involvement: SJS (<10%), SJS–TEN overlap (10-30%), and TEN (>30%). People living with HIV (PLHIV) demonstrate increased susceptibility to severe cutaneous adverse drug reactions due to immune dysregulation, altered drug metabolism, chronic immune activation, and polypharmacy. Sulfonamides, particularly co-trimoxazole used for Pneumocystis jirovecii prophylaxis, are commonly implicated. Early recognition and prompt withdrawal of the offending drug are critical determinants of survival.



Clinical Spectrum of Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis Based on Body Surface Area

Materials and Methods

We report a case of suspected co-trimoxazole induced SJS–TEN overlap in a patient with advanced HIV receiving Pneumocystis jirovecii prophylaxis. The patient had no known comorbidities including diabetes mellitus or hypertension. Detailed clinical history including drug exposure timeline, prodromal symptoms, mucocutaneous progression, and systemic involvement was documented. Comprehensive mucocutaneous examination was performed to assess BSA detachment and mucosal involvement. Disease severity was assessed using SCORTEN scoring at admission. Laboratory evaluation included complete blood counts, renal and liver function tests, serum electrolytes, and inflammatory markers. Histopathological examination of skin biopsy was performed. Differential diagnoses including DRESS syndrome

and erythema multiforme major were clinically considered. Drug causality was assessed based on temporal association, known drug risk profile, and clinical improvement following drug withdrawal.

STEP	DESCRIPTION
STEP 1	Advanced HIV (LOW CD4 STATE)
STEP 2	Drug exposure (Cotrimoxazole administration)
STEP 3	Abnormal drug metabolism in HIV (formation of reactive hydroxylamine metabolites)
STEP 4	Hapten formation -> Neoantigen formation
STEP 5	CD8 activation -> Granulysin release
STEP 6	Keratinocyte apoptosis
STEP 7	SJS-TEN spectrum

Proposed pathogenesis of increased sulfonamide hypersensitivity in advanced HIV

Results

A 45-year-old woman with advanced HIV infection (CD4 count 29 cells/mm^3), with no known comorbidities including diabetes mellitus or hypertension, presented with complaints of cough and low-grade fever. Clinical and laboratory evaluation raised suspicion of Pneumocystis jirovecii infection, for which co-trimoxazole prophylaxis/therapy was initiated.Within 2 days of initiation of co-trimoxazole, she developed mucocutaneous symptoms with hyperpigmented and erythematous macules associated with fever and malaise. Lesions progressed to targetoid morphology with painful oral erosions and hemorrhagic lip crusting. By day 7–8, epidermal detachment involving approximately 18–20% of body surface area was noted, consistent clinically with SJS–TEN overlap.Skin biopsy demonstrated full thickness epidermal necrosis with subepidermal split and sparse dermal inflammatory infiltrate, consistent with SJS/TEN spectrum. SCORTEN at admission was 2. Co-trimoxazole was immediately discontinued. The patient required intensive supportive care in the respiratory intensive care unit including intravenous fluids, electrolyte correction, wound care, mucosal care, analgesia, and systemic corticosteroids.Arrest of new lesion formation occurred by week 2 with complete re-epithelialization by week 3 and full clinical recovery.Temporal drug causality was strongly supported by rapid onset following exposure and clinical improvement after withdrawal

DAY	EVENT
Day 0	Co-trimoxazole prophylaxis initiated for Pneumocystis jirovecii prevention
Day 2	Onset of mucocutaneous symptoms; Initial hyperpigmented or erythematous macules; Possible fever or malaise
Day 4–5	Progression to targetoid lesions; Onset of mucosal involvement including oral erosions and lip crusting
Day 7–8	Epidermal detachment develops; Approximately 18–20% body surface area involvement; Hospital admission; SCORTEN score calculated as 2
RICU Phase	Supportive care including intravenous fluids, electrolyte correction, wound care, systemic corticosteroids, mucosal care
Week 2	Arrest of new lesion formation; Beginning of re-epithelialization
Week 3	Complete re-epithelialization; Clinical recovery and discharge

Figure: Clinical timeline of co-trimoxazole induced SJS/TEN overlap demonstrating temporal association with drug initiation, mucocutaneous progression, epidermal detachment, intensive care management, and recovery phase.

Conclusions

This case underscores the heightened vulnerability of severely immunocompromised PLHIV to sulfonamide induced severe cutaneous adverse drug reactions. Early recognition of prodromal symptoms, prompt drug withdrawal, and aggressive multidisciplinary supportive care remain the cornerstone of management. This report highlights the need for vigilant pharmacovigilance and careful risk–benefit evaluation when prescribing sulfonamides in advanced HIV. Early risk stratification and close clinical monitoring may help prevent progression to life threatening epidermal necrolysis in high-risk population.

Abstract N°: ID-1081

Topic: Adverse drug reactions, TEN

Palmar-Plantar Erythrodysesthesia under Capecitabine: Why Proactive Monitoring Saves Quality of Life

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Introduction
Capecitabine is a widely used fluoropyrimidine for the treatment of colorectal cancer and other solid tumors. Despite its efficacy, it can induce palmar-plantar erythrodysesthesia (also referred to as hand-foot syndrome), a cutaneous toxicity that may significantly impair quality of life and require temporary or permanent discontinuation of chemotherapy. This case highlights the importance of proactive monitoring of cutaneous toxicities.

Materials and Methods
We report the case of a 71-year-old patient with metastatic right-sided colon adenocarcinoma with peritoneal involvement. The patient was receiving palliative treatment with capecitabine and irinotecan and had completed seven cycles without incident. Ten days prior to consultation, he developed redness, painful fissures, and desquamation on the palms and soles, limiting daily activities. Palmar-plantar erythrodysesthesia was diagnosed, capecitabine was discontinued, and topical treatment with emollients and corticosteroids was initiated. The lesions completely resolved, and subsequent management focused exclusively on palliative care due to metastatic disease progression.

Results
Palmar-plantar erythrodysesthesia typically begins with tingling and numbness in the extremities and may progress to pain, edema, erythema, blisters, desquamation, and ulcerations. It is classically associated with fluoropyrimidines, including capecitabine and 5-fluorouracil, as well as certain cytotoxic agents such as docetaxel and liposomal doxorubicin. Risk factors include cumulative dose, duration of exposure, local trauma, and exposure to heat or chemical irritants. The pathophysiology is multifactorial, involving drug accumulation in capillary-rich areas and eccrine glands, extravasation at the microvascular level, COX-2 mediated inflammation, direct cytotoxicity of basal epidermal cells, and genetic variations affecting capecitabine metabolism. Management relies on symptomatic measures such as emollients and cold compresses for mild cases, and dose reduction or temporary treatment interruption for moderate to severe cases. Complementary strategies including vitamin E, pyridoxine, or dexamethasone have shown promising results in small studies but are not yet validated. Prevention focuses on minimizing friction and local trauma, protecting against heat and irritants, and early recognition of symptoms to reduce lesion severity and preserve patient quality of life.

Conclusions
Palmar-plantar erythrodysesthesia under capecitabine can occur unpredictably, even after several uneventful cycles, significantly affecting quality of life and treatment continuity. This case underscores the importance of proactive monitoring, prompt and appropriate management, and patient education for early recognition of symptoms. Clinical vigilance is essential, as no definitive curative strategy currently exists. Early detection and effective management not only limit lesion severity but also optimize the continuation of anticancer therapy while preserving patient comfort.

Abstract N°: ID-1114

Topic: Adverse drug reactions, TEN

Rare neurological and ocular complication of adalimumab therapy in psoriasis: aseptic meningitis coexisting with Posner-Schlossman syndrome

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Introduction
Aseptic meningitis is a condition in which cerebrospinal fluid shows increased white blood cells despite no detectable infection. It is most often caused by viral infections or drug reactions, but in some cases the cause remains unknown. Posner-Schlossman syndrome is a rare eye disorder that leads to inflammation of the anterior part of the eye and temporary increases in intraocular pressure.

Materials and Methods
A 35-year-old male patient with severe plaque psoriasis treated with adalimumab was hospitalized in the 12th week of therapy due to a three-week history of frontal headache, right ocular pain, fever, nausea, and vomiting. Diagnostic work-up included laboratory and cerebrospinal fluid tests, microbiological and molecular screening, serology, and brain MRI. Ophthalmology consultation was performed because of vision worsening.

Results
Laboratory tests showed mildly elevated C-reactive protein levels. Analysis of cerebrospinal fluid revealed lymphocytic pleocytosis with borderline protein and glucose concentrations. Extensive microbiological and molecular testing of CSF and blood, including PCR for CMV, enteroviruses, herpesviruses (HSV-1, HSV-2, HHV-6, VZV), *Cryptococcus*, and common bacterial pathogens, were all negative. SARS-CoV-2, HIV, HBV, HCV, and tick-borne encephalitis were excluded. Brain MRI showed no abnormalities. Ophthalmologic evaluation revealed unilateral acute secondary open-angle glaucoma with concomitant anterior uveitis, consistent with Posner–Schlossman syndrome. Symptomatic treatment and intraocular pressure-lowering therapy resulted in slight clinical improvement. However, after cessation of adalimumab use and switch to tildrakizumab, complete resolution of neurological symptoms was observed.

Conclusions
This case highlights a rare complication of adalimumab therapy presenting as aseptic meningitis coexisting with Posner-Schlossman syndrome. It emphasises the importance of considering drug-induced neurological and ocular adverse events in patients receiving biological therapies, particularly in the presence of atypical or persistent symptoms. Early recognition and timely modification of biological therapy may prevent further neurological and ocular complications and improve clinical outcomes.

Abstract N°: ID-1118

Topic: Adverse drug reactions, TEN

Epidemiological and Clinical Profile of Cutaneous Adverse Drug Reactions: A Series of 110Cases

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Introduction
Cutaneous adverse drug reactions (CADRs) account for 1% of all drug intakes.
In 90% of cases, cutaneous reactions are benign, but some can be life-threatening.

Materials and Methods
We conducted a retrospective study collecting data on patients
presenting with CADRs over a 20-year period at the Dermatology Department of La Rabta Hospital in Tunis. Epidemiological and clinical characteristics, as well as pharmacovigilance survey data, were recorded.

Results
Results: One hundred and ten files were identified (61 females and 49 males) with a sex ratio (M/F) of 0.8. The mean age at diagnosis was 68.7 years. A pharmacovigilance survey was performed in 100 cases (90.9%); causality was considered probable in 19 cases, likely in 35 cases, and very likely in 46 cases. The diagnosis was retained in the remaining ten cases despite the absence of a survey, based on the time to onset compatible with a CADR and/or the fact that the suspected drug is a known trigger.
Clinical manifestations included urticaria (n=19), maculopapular rash (n=20), fixed drug eruption (n=12), photosensitivity (n=12), eczema, purpura, and drug-induced acne (n=3 each). Hand-foot syndrome, mucitis, and pigmented purpuric dermatosis were each noted in 2 cases. Aggravation of pre-existing psoriasis was observed in 3 cases, while distal onycholysis and lichen planus were noted in 1 case each.
Cutaneous involvement was severe in 27 cases (24.5%): 9 cases of Acute Generalized Exanthematous Pustulosis (AGEP), including one induced by the anti-SARS-CoV-2 vaccine, 13 cases of DRESS syndrome, 4 cases of Stevens-Johnson syndrome (SJS), and one case of Toxic Epidermal Necrolysis (TEN).
The most frequently incriminated drugs were paracetamol (n=10), piroxicam (n=7), griseofulvin, tetracyclines, and allopurinol (n=6 each); pristinamycin, anticonvulsants, acetylsalicylic acid, and cephalosporins (n=4 each); hydroxychloroquine (n=3),

antidepressants, and diclofenac (n=2 each). Imatinib caused a lichenoid eruption in one case; sorafenib and capecitabine were responsible for hand-foot syndrome.
Ten patients required systemic corticosteroids, and 95 patients (86.9%) were treated with topical corticosteroids alone. For all patients, the incriminated drug was withdrawn. The outcome was favorable in all cases.

Conclusions

Consistent with literature data, our study highlighted the advanced age of patients, clinical polymorphism, and female predominance. Furthermore, a higher frequency of severe cutaneous adverse reactions (SCARs) was found in our series. The occurrence of CADRs at an advanced age could be explained by the high rate of polypharmacy in this population, leading to a higher risk of exposure. Antimicrobial agents and non-steroidal anti-inflammatory drugs (NSAIDs) were the most frequently involved drug classes.

Abstract N°: ID-1135

Topic: Adverse drug reactions, TEN

Clinical Characteristics, Management Strategies, and Outcomes of Stevens–Johnson Syndrome and Toxic Epidermal Necrolysis: A Single-Center Retrospective Study

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Introduction

Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are rare but life-threatening severe cutaneous adverse drug reactions characterized by extensive epidermal necrosis and mucosal involvement . Most published data on SJS/TEN originate from burn units, intensive care settings, or national databases, while reports from dermatology-based tertiary centers remain limited and heterogeneous . SCORTEN is a validated prognostic tool for predicting mortality in SJS/TEN; however, real-world data evaluating both admission and 48-hour SCORTEN in single-center dermatology cohorts are scarce.

The present study aimed to evaluate clinical characteristics, causative drugs, management strategies, complications, SCORTEN scores, and outcomes of SJS/TEN patients followed in a tertiary dermatology center.

Materials and Methods

A retrospective descriptive study was conducted including patients diagnosed with SJS, SJS/TEN overlap, or TEN in a tertiary dermatology center between the defined study period

Demographic data, clinical classification, extent of epidermal detachment, mucosal involvement, suspected causative drugs, treatments, complications, SCORTEN at admission and at 48 hours, histopathological findings, and outcomes were extracted from medical records. SCORTEN was calculated according to established criteria at diagnosis and reassessed at 48 hours. Descriptive statistical analyses were performed, and results were expressed as median with range or number with percentage, as appropriate.

Results

A total of 20 patients were included, with a median age of 45.5 years, and a slight female predominance was observed. Clinical classification revealed SJS in 45.0%, SJS/TEN overlap in 35.0%, and TEN in 20.0% of patients, with epidermal detachment exceeding 30% in one-fifth of cases. Mucosal involvement was present in 80.0% of patients, most commonly affecting the oral mucosa. Antibiotics, allopurinol, and anticonvulsants were the most frequently suspected causative drugs. Systemic corticosteroids were administered in 95.0% of patients, intravenous immunoglobulin in 75.0%, and cyclosporine in 25.0%, while 55.0% required intensive care unit admission. The median SCORTEN was 3.0 at admission and decreased to 2.5 at 48 hours, with SCORTEN ≥3 observed in 60.0% at admission and 50.0% at 48 hours. Sepsis and acute kidney injury were the most frequent complications, and overall in-hospital mortality was 40.0%. Skin biopsy was performed in 70.0% of patients, showing dermal lymphocytic infiltration and apoptotic keratinocytes as the most common histopathological findings.

Variable	Patients (n = 20)
Age, years, median (range)	45.5 (19–79)
Sex, n (%)	
Female	11 (55.0)
Male	9 (45.0)
Presence of comorbidities, n (%)	12 (60.0)
Presence of malignancy, n (%)	2 (10.0)
Clinical classification, n (%)	
Stevens–Johnson syndrome	9 (45.0)
SJS/TEN overlap	7 (35.0)
Toxic epidermal necrolysis	4 (20.0)
Extent of epidermal detachment, n (%)	
<10% body surface area	9 (45.0)
10–30% body surface area	7 (35.0)
>30% body surface area	4 (20.0)
Mucosal involvement, n (%)	16 (80.0)
Oral	9 (45.0)
Ocular	4 (20.0)
Anogenital	3 (15.0)
Time from drug exposure to symptom onset, days	13.5 (4–28)
SCORTEN at admission, median (range)	3.0 (1–5)
SCORTEN at 48 hours, median (range)	2.5 (0–6)
SCORTEN ≥3 at admission, n (%)	12 (60.0)
SCORTEN ≥3 at 48 hours, n (%)	10 (50.0)
Suspected causative drugs, n (%)	
Antibiotics	5 (25.0)
Allopurinol	4 (20.0)
Anticonvulsants	3 (15.0)
NSAIDs	1 (5.0)
Other drugs	7 (35.0)
Treatment modalities, n (%)	
Systemic corticosteroids	19 (95.0)
Intravenous immunoglobulin	15 (75.0)
Cyclosporine	5 (25.0)
Supportive care	20 (100.0)
ICU admission, n (%)	11 (55.0)
Length of hospital stay, days	21.5 (5–321)
Complications, n (%)	
Sepsis	8 (40.0)
Acute kidney injury	7 (35.0)
Respiratory failure	4 (20.0)
Other complications	3 (15.0)
Histopathological findings (n = 14), n (%)	
Full-thickness epidermal necrosis	7 (50.0)
Subepidermal detachment	4 (28.6)
Apoptotic keratinocytes	9 (64.3)
Dermal lymphocytic infiltration	14 (100.0)
Eosinophils	1 (7.1)
Clinical outcome, n (%)	
Recovered	12 (60.0)
In-hospital mortality	8 (40.0)

Table 1. Demographic characteristics, clinical features, causative drugs, treatment modalities, complications, histopathological findings, and outcomes in patients with Stevens–Johnson syndrome and toxic epidermal necrolysis.

Conclusions

This single-center dermatology-based retrospective study highlights the substantial morbidity and mortality associated with SJS/TEN despite intensive multidisciplinary management.
Admission and 48-hour SCORTEN values reflected disease severity and provided useful prognostic information in routine clinical practice. The high frequency of systemic complications underscores the need for early recognition, close monitoring, and prompt supportive and immunomodulatory treatment in specialized centers. Dermatology-centered real-world data contribute valuable insights into the clinical spectrum and outcomes of SJS/TEN and complement existing burn unit–based literature.

Abstract N°: ID-1151

Topic: Adverse drug reactions, TEN

Unraveling the spectrum of paradoxical Th1-mediated dermatoses under IL-4/IL-13 or IL-13 blockade: Two Cases and a Review of the Literature

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Introduction

Interleukin-4 (IL-4) and interleukin-13 (IL-13) are key cytokines in T-helper 2 (Th2)–mediated immunity, driving the pathogenesis of atopic dermatitis (AD) and related diseases (3,4). Both cytokines signal through receptor complexes containing the IL-4 receptor alpha subunit (IL-4Rα), activatingthe JAK/STAT pathway and promoting type 2 inflammation. Dysregulated IL-4 and IL-13 signaling is therefore a hallmark of Th2-driven inflammatory disorders, including AD, asthma, and related atopic conditions(2). Moreover, IL-4 and IL-13 exert important immunoregulatory effects by suppressing T-helper 1 (Th1) and T-helper 17 (Th17) pathways, inhibiting Th1Th1 differentiation, reducing Th1-associated chemokines, and modulating interferon-γ production (6,7). Dupilumab (anti-IL-4Rα) and more recently the selective IL-13 inhibitors tralokinumab und lebrikizumab have been introduced as an additional targeted therapeutic option, by reducing type 2 inflammation while maintaining a favorable safety profile (1,2,5,6). However, blockade of the IL-4/IL-13 axis may disrupt immune homeostasis, potentially triggering paradoxical Th1-mediated inflammatory conditions. We report two cases and review the literature on paradoxical Th1-driven dermatoses emerging under IL-4/IL-13 and IL-13 blockade. (8).

Materials and Methods

We present two patients with manifestations of moderate-to-severe AD treated with IL-4/IL-13 or IL-13 inhibitors who developed new-onset Th1-mediated inflammatory dermatoses. Clinical presentation, histopathological findings, and therapeutic outcomes were documented and presented in a consolidated format. A comprehensive literature review was performed to identify similar paradoxical cutaneous reactions reported under dupilumab, tralokinumab, and lebrikizumab therapy.

Results

Both patients developed paradoxical Th1-mediated dermatoses following initiation of biologic therapy targeting the IL-4/IL-13 axis. Clinical manifestations included psoriasiform eruptions and other inflammatory skin conditions consistent with Th1/Th17 immune activation. Histopathology supported a shift toward Th1-driven inflammation. Literature review revealed increasing reports of similar paradoxical reactions, including psoriasis, alopecia areata, vitiligo, and granulomatous dermatitis, occurring under IL-4/IL-13 blockade. Management strategies varied from continuation of therapy with adjunctive treatment to biologic discontinuation. NanoString-based transcriptomic profiling of the skin biopsies revealed the immunopathogenic signatures underlying both conditions.

Conclusions

Paradoxical Th1-mediated dermatoses are an emerging complication of IL-4/IL-13–targeted therapies for atopic dermatitis and related disorders. Our two cases, supported by NanoString- based gene expression profiling of lesional biopsies, provide insights tp the postulated shift from Th2- to Th1/Th17-skewed immune signatures under IL-4/IL-13 or IL-13 blockade, providing mechanistic insight into these reactions. These findings underscore the need for vigilant clinical monitoring, awareness of paradoxical inflammatory events, and individualized management rather than automatic

discontinuation of biologic therapy. Larger prospective studies are warranted to define risk factors, clarify immunopathogenic pathways, and guide safer use of IL-4/IL-13 inhibitors in clinical practice.

Abstract N°: ID-1205

Topic: Adverse drug reactions, TEN

Severe Carbamazepine-Induced DRESS Syndrome with Multiorgan Involvement Complicated by Macrophage Activation Syndrome

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Introduction

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) syndrome is a severe cutaneous adverse drug reaction related to delayed hypersensitivity, characterized by febrile skin eruption, lymphadenopathy, and multiorgan involvement, typically occurring 2–8 weeks after initiation of the offending drug. Its pathophysiology involves immune dysregulation, altered drug metabolism, genetic susceptibility, and herpesvirus reactivation, particularly human herpesvirus 6. We report a case of carbamazepine-induced DRESS syndrome with multiorgan involvement complicated by macrophage activation syndrome (MAS).

Materials and Methods

A 33-year-old woman with a history of depressive disorder was admitted for a febrile cutaneous eruption occurring three weeks after initiation of carbamazepine therapy. Clinical examination revealed fever up to 39.5 °C, petechial purpuric macules on the lower limbs, mild erythema of the trunk and upper limbs, and inguinal, cervical, and axillary lymphadenopathy, without facial edema or bullous lesions. The patient also reported acute chest pain; electrocardiography showed diffuse ST-segment elevation, and transthoracic echocardiography demonstrated pericardial effusion, consistent with acute pericarditis.

Laboratory investigations showed marked hepatic cytolysis, proteinuria of 0.52 g/24 h, and findings suggestive of MAS, including normocytic normochromic anemia, severe agranulocytosis, hyperferritinemia (2,806 ng/mL), elevated lactate dehydrogenase (911 IU/L), and moderate hypertriglyceridemia (2.03 g/L), without splenomegaly. Bone marrow examination revealed reactive changes. The RegiSCAR score was 4, supporting a diagnosis of probable DRESS syndrome complicated by MAS and multiorgan involvement.

Carbamazepine was discontinued, and systemic corticosteroid therapy at 1 mg/kg/day was initiated, combined with colchicine for pericarditis management, resulting in favorable clinical and biological evolution.

Results

DRESS syndrome is a severe drug-induced reaction frequently associated with aromatic antiepileptic drugs, particularly carbamazepine, with symptom onset typically occurring between two and eight weeks after drug exposure.

Complication by macrophage activation syndrome during DRESS is rare but associated with a poorer prognosis. In the present case, the presence of severe cytopenias, marked hyperferritinemia, elevated LDH levels, and moderate hypertriglyceridemia was consistent with probable MAS, despite the absence of splenomegaly and bone marrow hemophagocytosis, as incomplete forms are commonly described in adults.

Febrile agranulocytosis was also observed, an exceptional complication of DRESS. This may be related to MAS; however, direct carbamazepine-induced hematologic toxicity or an immune-mediated mechanism associated with DRESS cannot be formally excluded.

The association of hepatic, renal, hematological involvement and acute pericarditis highlights the severity of the clinical presentation and underscores the need for close monitoring of target organs. Favorable outcome following withdrawal of the offending drug and initiation of systemic corticosteroid therapy confirms the importance of early diagnosis and prompt management.

Conclusions

DRESS syndrome is a severe cutaneous adverse drug reaction with a polymorphic presentation and potential progression to life-threatening multiorgan involvement. Early recognition, immediate discontinuation of the offending drug, and appropriate treatment are crucial for prognosis.

Abstract N°: ID-1225

Topic: Adverse drug reactions, TEN

Title: Clinical Characteristics of severe adverse reactions (SCARs) induced by antifungal drugs at a Tertiary Dermatology Center in Hungary

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Introduction

Dermatomycoses are generally easily treatable conditions; however, systemic antifungal therapy may be required in refractory cases. Commonly used agents include terbinafine, itraconazole, fluconazole, and, in selected cases voriconazole may be considered.

Although antifungal drugs are usually well tolerated, they may induce severe cutaneous adverse reactions (SCARs). These include delayed-type hypersensitivity reactions such as maculopapular exanthema, drug reaction with eosinophilia and systemic symptoms (DRESS), acute generalized exanthematous pustulosis (AGEP), and Stevens–Johnson syndrome/toxic epidermal necrolysis (SJS/TEN). AGEP is most frequently associated with terbinafine, while voriconazole is particularly linked to photosensitivity reactions. Furthermore, it has been established that certain autoimmune diseases, including lupus erythematosus, and inflammatory diseases, such as psoriasis, have been observed to be triggered or exacerbated by antifungal medications. Among local allergic reactions, contact dermatitis and fixed drug eruption are noteworthy, but various irritant allergic reactions may also occur.

Materials and Methods

A review of the literature between 2015 and 2025 identified 13 cases of AGEP, 7 cases of SJS, and 2 cases of DRESS related to antifungal therapy. A thorough search of our database revealed two documented cases of terbinafine-induced AGEP, necessitating hospitalisation within our institution.

Results

Hereby we present the clinical manifestations and courses observed in the two patients.

Conclusions

These cases highlight the importance of awareness of antifungal-related adverse events, careful patient education, prompt drug discontinuation, early assessment of systemic involvement, and timely initiation of appropriate local and systemic immunosuppressive therapy are essential for optimal management.

Abstract N°: ID-1243

Topic: Adverse drug reactions, TEN

Acute Generalized Exanthematous Pustulosis Triggered by H. pylori Eradication Therapy: A Case Report

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Introduction

Acute generalized exanthematous pustulosis (AGEP) is a rare, acute, usually drug-induced skin eruption, with an annual incidence of 1–5 cases per million population [Creadore A, et al. 2022]. Over 90 % of cases are drug-induced, with antibiotics (especially β-lactams and macrolides) being the most frequently implicated agents [Tetart F, et al. 2024]. Proton-pump inhibitors such as pantoprazole have also been reported in isolated cases to trigger AGEP [Schmitz B, et al. 2018]. AGEP typically presents with an acute onset of erythema, mainly affecting large skin folds, with multiple sterile non-follicular pustules, frequently accompanied by fever, neutrophilia and elevated C-reactive protein (CRP). Patients with suspected AGEP should be evaluated according to expert recommendations to ensure accurate diagnosis and appropriate management [Tetart F, et al. 2024]. Although the condition is self-limited in many cases, recognition and prompt withdrawal of the causative agent are essential to prevent progression and systemic complications.

Materials and Methods

We report a case of a 67-year-old female who developed an AGEP after initiating triple therapy for *H. pylori* eradication (amoxicillin, clarithromycin, pantoprazole).

Results

Five days after starting *H. pylori* eradication therapy, the patient developed a maculopapular exanthema that progressed over the following week. Previous outpatient treatment with clemastine and intramuscular dexamethasone was ineffective. On admission, the patient was hemodynamically stable but reported low-grade fever and pruritus (VAS 3–4). Dermatologic examination showed confluent erythematous plaques with non-follicular pustules on the trunk, extremities, and gluteal region, accompanied by mild palmar and plantar erythema. Severity scoring based on the clinical assessment indicated BSA 30% and DLQI 8.

Lab findings demonstrated leukocytosis (14.3×10^9/l) with neutrophilia (11.3×10^9/l) and no eosinophilia, elevated CRP (32 mg/L), and mild renal function impairment (creatinine 124 µmol/l). Liver function, electrolytes and total IgE were normal. Skin histologic examination revealed multiple subcorneal pustules containing neutrophils, epidermal spongiosis, and a dermal perivascular infiltrate rich in neutrophils and eosinophils - fully compatible with AGEP. Direct immunofluorescence was negative. EuroSCAR's AGEP validation score was 10, confirming a definite diagnosis.

All suspected medications (amoxicillin, clarithromycin, pantoprazole) had already been discontinued at symptom onset. Due to disseminated lesions, systemic treatment with oral prednisolone 30 mg/day was initiated for 5 days with a

gradual taper. Additional supportive measures, such as intravenous fluids and emollients were provided. Significant clinical improvement occurred within several days, with reduction of inflammation and gradual skin improvement, resolution of pustules.

During follow-up, the patch and intradermal testing with all suspected drugs were negative. Although no culprit drug was identified, the patient was advised to avoid all the suspected drugs. Alternative options were suggested, including cefuroxime, third- or fourth-generation cephalosporins, while macrolides should be avoided.

Conclusions

This case highlights the diagnostic complexity of AGEP in patients exposed to multiple concurrent medications, as is common in *H. pylori* eradication regimens. Early recognition, prompt withdrawal of potential offending agents, and histopathologic confirmation remain essential for favorable outcomes. Systematic and comprehensive evaluation, including allergological testing, is essential to identify potential triggers and guide the safe selection of future therapeutic options for affected patients.

Abstract N°: ID-1245

Topic: Adverse drug reactions, TEN

Systemic psoriasis treatments and oncologic outcomes in immune checkpoint inhibitor–induced psoriasis: a multicenter study from the EADV Task Force “Dermatology for Cancer Patients”

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Introduction

Psoriasis is an increasingly recognized immune-related adverse event of immune checkpoint inhibitors (ICIs). Its management poses a critical clinical dilemma: controlling cutaneous disease without attenuating antitumor immunity. Although expert consensus supports the use of several systemic psoriasis therapies in this context, robust clinical data regarding their oncologic safety remain limited.

Materials and Methods

We conducted a multicenter retrospective cohort study within the EADV Task Force “Dermatology for Cancer Patients.” All consecutive adult patients who developed ICI–induced psoriasis and were managed at participating centers between 2020 and 2024 were included. Systemic treatments were categorized as none, acitretin, apremilast, methotrexate, systemic corticosteroids, and biologics (anti–TNF, anti–IL-17, anti–IL-23, anti–IL-12/23). Primary outcomes were progression-free survival (PFS) and overall survival (OS), measured from the time of psoriasis diagnosis. Multivariable Cox regression analyses were performed using both time-constant and time-dependent exposure models, adjusted for age, sex, cancer stage, duration of ICI therapy, oncologic regimen, and psoriasis severity.

Results

A total of 217 patients were included (mean age 63.3 years; 71% male), most with advanced malignancy (56.2% stage IV). The most common cancers were non–small cell lung cancer (39.6%) and melanoma (20.3). Nearly one in five patients (17.9%) required interruption or discontinuation of oncologic therapy due to cutaneous toxicity. In multivariable Cox regression analyses, systemic corticosteroid exposure was independently associated with significantly worse PFS (HR 2.49; 95% CI 1.26–4.93; $p<0.01$). Acitretin showed a borderline association with inferior PFS (HR 1.76; 95% CI 1.00–3.11, $p=0.05$). In contrast, no significant associations with impaired PFS or OS were observed for apremilast, methotrexate, or biologic therapies, providing reassuring evidence regarding their oncologic safety in real-world practice. Notably, apremilast exposure showed a trend toward improved OS (HR 0.32; 95% CI 0.09–1.21), suggesting a potentially favorable profile that warrants further investigation.
Cancer stage remained the strongest independent prognostic factor for both PFS (stage IV vs stage II: HR 5.84; 95% CI 2.02–16.91) and OS (stage IV vs stage II: HR 5.00; 95% CI 1.17–21.36). Longer duration of ICI therapy was consistently associated with improved outcomes (PFS: HR per month 0.92; 95% CI 0.89–0.94; OS: HR per month 0.93; 95% CI 0.90–0.95). Interestingly, patients with moderate psoriasis severity (10–30% BSA) demonstrated better oncologic outcomes compared to those with mild or severe disease, supporting the concept that a certain degree of immune activation may correlate with enhanced antitumor response.

Conclusions

This large multicenter real-world cohort provides clinically actionable evidence for the management of ICI-induced psoriasis. Systemic corticosteroids appear to negatively impact oncologic outcomes and should be avoided whenever possible, supporting a clear steroid-sparing approach. In contrast, most commonly used systemic therapies—including biologics, methotrexate, and apremilast—appear oncologically safe. These findings support a shift toward individualized, mechanism-driven management and underscore the need for prospective studies.

Abstract N°: ID-1278

Topic: Adverse drug reactions, TEN

Severe DRESS induced by sulfasalazine with high HHV-6 viral load and absence of chromosomal integration: *relevance of targeted virological assessment*

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Introduction

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) is a severe, delayed drug-induced hypersensitivity reaction with potentially life-threatening systemic involvement. Its pathophysiology involves inappropriate immune activation and, in a significant proportion of cases, reactivation of human herpesvirus 6 (HHV-6). High HHV-6 viral loads may also result from chromosomally integrated HHV-6 (ciHHV-6), a constitutional condition that can lead to misinterpretation of virological findings. Distinguishing true viral reactivation from ciHHV-6 is therefore crucial, as it directly impacts therapeutic decisions, particularly the indication for antiviral therapy.

Materials and Methods

We report a case of severe DRESS with systemic involvement. Clinical, biological and cardiac assessments were performed. HHV-6 viral load was measured using quantitative polymerase chain reaction (PCR) in peripheral blood. In the presence of marked viremia, chromosomal integration of HHV-6 was investigated using hair follicles and nail samples.

Results

A 33-year-old woman treated with methotrexate and low-dose corticosteroids for an undifferentiated inflammatory rheumatic disease developed a rapidly progressive morbilliform eruption 26 days after initiation of sulfasalazine. The eruption evolved into a diffuse infiltrated erythroderma associated with facial edema, painful cervical lymphadenopathy and high fever (40 °C). Laboratory investigations revealed hepatic cytolysis, and cardiac evaluation showed mild pericardial involvement. The RegiSCAR score was 6, consistent with definite DRESS.

Initial treatment with intravenous methylprednisolone pulses resulted in partial improvement. Forty-eight hours after switching to oral prednisone (1 mg/kg/day), a severe rebound occurred, characterized by recurrent high fever, worsening erythroderma and marked hepatic cytolysis reaching 30 times the upper limit of normal. Quantitative PCR revealed an HHV-6 viral load exceeding 6 log copies/mL. Investigation for ciHHV-6 using hair and nail samples was negative, confirming true viral reactivation. Continued systemic corticosteroid therapy led to rapid clinical and biological improvement, and antiviral therapy was not initiated.

Conclusions

This case highlights the importance of systematically assessing chromosomally integrated HHV-6 in DRESS patients presenting with high HHV-6 viral loads, particularly in severe or corticosteroid-refractory cases. Integrating targeted virological investigations into the diagnostic approach helps avoid unnecessary antiviral therapy and supports more accurate, individualized management of severe DRESS.

Abstract N°: ID-1326

Topic: Adverse drug reactions, TEN

Benign maculopapular exanthema: Drug profile of a common cutaneous adverse reaction

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Introduction
Benign maculopapular exanthema is the most common type of cutaneous drug reaction, characterized by a diffuse erythematous and pruritic rash. Although benign, accurate diagnosis is essential to avoid unnecessary discontinuation of essential treatments. This study aimed to describe the drug profile and clinical characteristics of benign maculopapular exanthema in order to improve its recognition and management.

Materials and Methods
A single-center retro-prospective study was conducted over a two-year period, including patients who consulted for benign maculopapular exanthema.

Results

A total of 48 patients were included. The mean age was 39 ± 13 years, with a female predominance (62%). The most frequently implicated drugs were antibiotics (42%), mainly beta-lactams and fluoroquinolones. Allopurinol accounted for 10% of cases. Antiepileptic drugs were involved in 8% of exanthemas, while nonsteroidal anti-inflammatory drugs accounted for 6%. Other medications were less frequently implicated, including chemotherapy agents (8%), immunosuppressants (6%), proton pump inhibitors (4%), alpha-amylase (4%), and beta-blockers (2%).

The median time to onset of the exanthema after drug initiation was 6 days. Clinically, lesions were diffuse, erythematous, maculopapular eruptions, predominantly affecting the trunk and limbs in 88% of cases, with flexural accentuation in 72%. Pruritus was reported by 78% of patients, with no mucosal involvement or systemic manifestations. Management consisted of discontinuation of the suspected drug in all patients, combined with symptomatic treatment, including oral antihistamines in 80% of cases, emollients in 92%, and low- to moderate-potency topical corticosteroids in 68%. The outcome was favorable in all patients, with complete resolution of lesions within 7 to 10 days, without complications or early recurrence.

Conclusions
Benign maculopapular exanthema is a common cutaneous drug reaction. Prompt recognition allows for simple and effective management based on withdrawal of the causative drug and appropriate symptomatic treatment.

Abstract N°: ID-1340

Topic: Adverse drug reactions, TEN

Imipenem–Cilastatin–Associated Monodactylic Erythromelalgia: A Rare Drug-Induced Reaction

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Introduction

Erythromelalgia is a rare neurovascular acrosyndrome characterized by episodic erythema, increased local temperature, and burning pain, typically affecting the distal extremities. It is classified as primary (idiopathic or genetic) or secondary to systemic disease or medications. Localized or monodactylic forms are exceptional and may mimic infectious, inflammatory, or ischemic digital disorders, creating diagnostic uncertainty.

Materials and Methods

An 82-year-old man was admitted to urology for urinary sepsis requiring bilateral ureteral stent management. Medical history included hyperthyroidism, bilateral lower-limb deep venous thrombosis, and prostate adenocarcinoma treated with radiotherapy complicated by a rectovesical fistula requiring diverting colostomy. Intravenous imipenem–cilastatin was initiated. Several days after treatment onset, the patient developed abrupt inflammatory symptoms localized to the right thumb and index finger, consisting of congestive erythema, marked warmth, and burning pain, without preceding trauma or cold exposure. There were no bullae, necrosis, discharge, or portal of entry. Peripheral pulses were preserved, and no livedo or Raynaud phenomenon was observed. Differential diagnoses included cellulitis, crystal-induced dactylitis, complex regional pain syndrome, and digital ischemia. The characteristic clinical triad and absence of supporting findings for alternative diagnoses favored localized secondary erythromelalgia. Symptomatic management, including analgesia and cautious cooling measures, was provided while imipenem–cilastatin therapy was continued due to the septic context. Rapid spontaneous regression of erythema, warmth, and pain was observed.

Results

Secondary erythromelalgia has been described in association with myeloproliferative disorders and drug exposures, including calcium channel blockers, bromocriptine, and cyclosporine. Carbapenems are generally well tolerated, and cutaneous adverse reactions are rare and typically nonspecific. To our knowledge, a localized monodactylic erythromelalgia temporally associated with imipenem–cilastatin has not been previously reported. The focal distribution further contributes to the atypical presentation and diagnostic challenge.

Conclusions

This case illustrates a rare localized erythromelalgia presentation temporally associated with imipenem–cilastatin exposure, with favorable evolution despite continuation of therapy. Recognition of atypical erythromelalgia is important to prevent misdiagnosis and unnecessary escalation of treatment when clinical findings are consistent with a benign, reversible neurovascular process.

Abstract N°: ID-1354

Topic: Adverse drug reactions, TEN

Unusual drug reaction: Bullous variant of Erythema multiforme minor induced by doxycycline

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Introduction

Erythema multiforme (EM) is an acute, immune-mediated mucocutaneous hypersensitivity reaction most commonly triggered by infections (especially *HSV-1*) and, less frequently, drugs. Drug-induced *EM* accounts for <10% of cases; commonly implicated drugs include sulfonamides, NSAIDs, penicillins, trimethoprim, barbiturates, and carbamazepine, while antibiotics such as cephalosporins and fluoroquinolones have also been reported. Tetracyclines (doxycycline/minocycline) can cause cutaneous reactions including phototoxicity and fixed drug eruptions, but reports linking doxycycline with classic *EM* are rare.

Materials and Methods

We report a single patient case of a 27-year-old female patient who developed a disseminated cutaneous eruption following doxycycline exposure after a dental procedure. Clinical features, laboratory workup, therapeutic interventions, and the temporal association with doxycycline were analysed. Extensive infectious (HBV, HCV, HIV, EBV, adenovirus, *Mycoplasma pneumoniae*, and CMV including serology, IgG avidity testing, and PCR) and immunologic testing (ANA, ENA, ANCA, immunoglobulin levels) were conducted to exclude alternative etiologies.

Results

Three weeks prior to hospital admission, the patient underwent a dental procedure, after which doxycycline was prescribed. Several days later, erythematous papules appeared on the thighs and were initially interpreted as insect bites (*Ictus insecti*). However, the eruption progressively worsened. Persistent pruritus and clinical suspicion of scabies led to treatment with topical sulfur-based therapy. Despite this treatment, erythematous plaques subsequently developed on the gluteal region and face, accompanied by multiple target lesions on the buttocks and tense vesicles and bullae on the palms and soles. No mucosal involvement was observed, which is consistent with *EM minor*. Outpatient therapy included systemic corticosteroids (1 mg/kg), oral antihistamines, gastric protection, and cefalexin. However, the eruption progressed despite therapy given. Throughout hospitalisation, systemic steroids were maintained, with addition of potent topical corticosteroids and standard care of bullous lesions, leading to clinical improvement. Patient was in good general condition.

Leukocytosis with neutrophilia/monocytosis and reactive CMV IgM/IgG with high avidity (77%) indicated past CMV exposure, but PCR CMV was negative. Other serologic and immunologic tests were unremarkable. Given the timing after doxycycline and exclusion of common infectious triggers, drug-induced bullous variant of EM was considered most likely.

Note on CMV avidity: High CMV IgG avidity (>60–70%) typically indicates infection >3 months prior, making acute CMV

unlikely; negative PCR CMV supports this interpretation.

Conclusions

This case highlights an atypical course of *EM minor* triggered by doxycycline. While drug induced *EM* is less frequent than infectious triggers, clinicians should consider antibiotic triggers in patients with targetoid eruptions after drug exposure. Doxycycline more commonly causes photosensitive reactions and fixed drug eruptions, but classic *EM* remains rare, underscoring the importance of careful evaluation. Awareness of such rare reactions may aid to early identification and management, especially for evolving acral lesions.

Abstract N°: ID-1361

Topic: Adverse drug reactions, TEN

Efficacy of Cyclosporin in the Management of Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis: A Systematic Review

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Introduction

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are severe, life-threatening mucocutaneous conditions that often result in significant morbidity and mortality. These conditions are frequently triggered by drugs, and early intervention is crucial for improving survival rates. Cyclosporin, an immunosuppressive agent, has been explored as a potential treatment due to its ability to modulate immune response. This systematic review evaluates the efficacy and safety of Cyclosporin in the treatment of SJS/TEN.

Materials and Methods

A comprehensive review of literature was conducted, including studies published between 2005 and 2024. Data were extracted from clinical trials, case reports, and retrospective studies that assessed the use of Cyclosporin in managing SJS/TEN. The primary outcomes evaluated included survival rates, the extent of skin detachment, and complications. Secondary outcomes included the duration of treatment and any adverse effects related to Cyclosporin therapy. The risk of bias in the studies was assessed using the Cochrane risk of bias tool.

Results

The review included 12 studies involving a total of 185 patients. Cyclosporin treatment led to a significant reduction in skin detachment in the majority of cases, with an overall survival rate of 84%. Most patients showed rapid improvement in the acute phase, particularly when Cyclosporin was administered within the first 24–48 hours of onset. The use of Cyclosporin was associated with fewer complications and a shorter hospital stay compared to traditional treatments such as corticosteroids. However, the therapy was also linked to minor adverse effects, including renal toxicity and hypertension, which were generally manageable with dose adjustments.

Conclusions

Cyclosporin appears to be an effective and relatively safe treatment for managing SJS/TEN, particularly when administered early in the course of the disease. It significantly improves survival rates and reduces skin detachment. However, its use should be closely monitored for potential side effects, particularly renal toxicity. Further large-scale, randomized controlled trials are needed to confirm its long-term efficacy and safety in the treatment of these severe dermatological conditions.

Abstract N°: ID-1364

Topic: Adverse drug reactions, TEN

Cutaneous immune-related adverse events of immune checkpoint inhibitors: diagnostic challenges and therapeutic strategies

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Introduction

Cutaneous immune-related adverse events (irAEs) are the most frequent complications of immune checkpoint inhibitor (ICI) therapy, occurring in up to 40% of patients, with severe forms reported in approximately 5%. Despite their wide clinical spectrum, cutaneous irAEs often serve as an early marker of systemic immune activation and have been associated in several studies with improved oncologic survival outcomes. The lack of standardized diagnostic and therapeutic approaches poses a significant clinical challenge.

Materials and Methods

A structured literature search and analysis of original studies and review articles published over the last five years was conducted using PubMed, Scopus, ScienceDirect, and Elsevier databases. Publications focusing on clinical manifestations, diagnostic strategies, and treatment of cutaneous irAEs were included.

Results

Cutaneous toxicities of ICIs range from mild, self-limiting eruptions to severe bullous and life-threatening reactions. The most common presentations include rash, pruritus, vitiligo-like depigmentation, and psoriasis-like lesions. Major challenges include differentiating irAEs from infections or other drug reactions, as well as the absence of standardized severity grading criteria. Management of mild cases typically involves topical corticosteroids and symptomatic therapy, while severe presentations require systemic corticosteroids or immunosuppressants. In refractory cases, targeted biologic agents (e.g., anti–IL-17, anti–IL-6, JAK inhibitors) have shown promising results. A multidisciplinary approach involving both dermatologists and oncologists remains essential for optimal patient care.

Conclusions

Cutaneous irAEs represent a significant clinical concern due to their high prevalence, heterogeneity, and prognostic relevance. Despite growing experience, no international consensus guidelines currently exist for their diagnosis and management. Establishing multicenter registries and developing expert-driven recommendations are critical to improving patient outcomes and integrating cutaneous irAEs into oncologic prognostication.

Abstract N°: ID-1379

Topic: Adverse drug reactions, TEN

SJS/TEN–DRESS Overlap Triggered by Allopurinol: A Case Report

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Introduction

Severe cutaneous adverse reactions are classically classified into distinct entities such as Stevens–Johnson syndrome/toxic epidermal necrolysis (SJS/TEN) and Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS). However, increasing reports describe patients fulfilling diagnostic criteria for both conditions, a rare presentation that poses important diagnostic and therapeutic challenges. We report a case of allopurinol-induced overlap between SJS/TEN and DRESS in an elderly patient.

Materials and Methods

An 81-year-old man was started on allopurinol for gout. Two weeks after treatment initiation, he developed fever followed by a rapidly progressive generalized eruption.

Dermatological examination revealed a diffuse erythematous-violaceous maculopapular rash with purpuric lesions. Marked facial and palpebral edema was present. Mucosal involvement included severe erosions of the oral and genital mucosa. The Nikolsky sign was positive, with epidermal detachment involving approximately 12–15% of the body surface area, consistent with Stevens–Johnson syndrome/toxic epidermal necrolysis (SJS/TEN) overlap.

Laboratory investigations showed eosinophilia (1.8×10^9/L), lymphocytosis and hepatic cytolysis (ALT 185 IU/L, AST 142 IU/L). Infectious work-up was negative. According to the RegiSCAR scoring system, the patient scored 5 points, corresponding to probable DRESS, supporting a rare overlap between SJS/TEN and DRESS.

Allopurinol was immediately discontinued. The patient was treated with systemic corticosteroids (prednisone 60 mg/day), systemic antibiotics and daily wound care. A rapid and favorable outcome was observed, with progressive re-epithelialization and normalization of laboratory abnormalities.

Results

Overlap between severe cutaneous adverse reactions (SCARs) is increasingly recognized but remains exceptionally rare. SJS/TEN and DRESS share a T-cell–mediated pathophysiology and are frequently triggered by the same medications, particularly allopurinol, one of the most common culprits in both conditions. The coexistence of epidermal detachment and severe mucosal involvement with systemic features such as eosinophilia, facial edema and hepatic cytolysis should raise suspicion of an overlap presentation.

Recognition of overlap SCAR is clinically important because management strategies differ between SJS/TEN and DRESS. While the use of systemic corticosteroids in SJS/TEN remains debated, they represent the cornerstone of DRESS treatment and are increasingly reported to be beneficial in overlap cases. Our patient showed a rapid and favorable response to systemic corticosteroid therapy following prompt withdrawal of the culprit drug.

Conclusions

This case highlights the importance of recognizing overlap SCAR presentations in patients with severe drug eruptions. Early diagnosis, prompt withdrawal of the culprit drug and appropriate systemic therapy can lead to favorable outcomes.

Abstract N°: ID-1384

Topic: Adverse drug reactions, TEN

WHEN TREATMENT WORSENS THE CONDITION: AZATHIOPRINE HYPERSENSITIVITY IN A PATIENT WITH ACQUIRED EPIDERMOLYSIS BULLOSA

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Introduction

Acquired epidermolysis bullosa (EBA) is a rare autoimmune blistering disease mediated by autoantibodies directed against type VII collagen. Its management is challenging and relies on the combination of several therapeutic agents. Azathioprine is commonly prescribed; however, its use may be limited by adverse effects. Among these, hypersensitivity reactions remain exceptional, reported in only 1–2% of exposed patients across all clinical settings.

Results

We report the case of a patient with EBA confirmed clinically, histologically, by direct immunofluorescence, and by salt-split skin testing. First-line treatment with dapsone proved ineffective. Azathioprine was subsequently introduced; however, a few days after treatment initiation, the patient developed fever and a diffuse exanthema with skin detachment involving less than 10% of the body surface area, associated with peripheral blood eosinophilia, without visceral involvement. Immediate discontinuation of azathioprine resulted in a gradual and complete clinical recovery.
Hypersensitivity reactions to azathioprine are rare and largely anecdotal. The literature reports only a limited number of isolated cases, which may manifest as systemic symptoms such as fever, cutaneous involvement, or, more rarely, multiorgan involvement. These reactions are unpredictable and dose-independent, underscoring the need for close clinical monitoring during azathioprine therapy.
The present case is distinguished by a limited form, characterized by mild systemic symptoms, restricted cutaneous involvement, and isolated eosinophilia without visceral impairment. The early onset of symptoms, their idiosyncratic nature, and the requirement for immediate drug discontinuation emphasize the need for heightened vigilance when initiating azathioprine.
The use of alternative immunosuppressive agents or biologic therapies (mycophenolate mofetil, rituximab, intravenous immunoglobulins) remains essential in refractory or intolerant cases.

Conclusions

This case illustrates the therapeutic complexity of EBA, marked by dapsone inefficacy and the occurrence of a rare azathioprine hypersensitivity reaction limited to diffuse exanthema with eosinophilia. It highlights the importance of rigorous clinical follow-up, awareness of the rarity yet potential severity of this complication, and the consideration of individualized alternative therapeutic strategies.

Abstract N°: ID-1391

Topic: Adverse drug reactions, TEN

A Red Flag: Symmetric Intergluteal Erythema (SDRIFE) After Amoxicillin–Clavulanate

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Introduction
Symmetrical drug-related intertriginous and flexural exanthema (SDRIFE), also known as “baboon syndrome,” is a characteristic benign drug eruption presenting with sharply demarcated, symmetrical erythema predominantly affecting the gluteal/intergluteal region, usually after systemic drug exposure—most commonly β-lactam antibiotics. Early recognition helps avoid misdiagnosis as infection and prevents re-exposure.

Materials and Methods
A 42-year-old woman underwent right adnexectomy for an ovarian cyst. Postoperatively, she received amoxicillin–clavulanate (3 g/day), paracetamol, nefopam, metoclopramide, and enoxaparin. She reported previous non-documented urticaria after amoxicillin and acetylsalicylic acid. Approximately 12 hours after treatment initiation, she developed a pruritic rash. She was clinically stable and afebrile. Examination revealed a **sharply demarcated, symmetric erythematous indurated plaque** centered on the **intergluteal cleft and buttocks**, mildly warm, associated with two erythematous plaques on the back. There was **no genital mucosal involvement** and no inguinal or axillary involvement. No pustules or vesicles were observed.

Investigations: A complete laboratory work-up was normal. Pharmacovigilance assessment identified **amoxicillin–clavulanate** as the most likely culprit.

Management and outcome: Amoxicillin–clavulanate was discontinued and replaced with an alternative antibiotic. The patient received an oral antihistamine and topical corticosteroids for one week, with **marked clinical improvement**.

Results
The rapid onset after systemic drug exposure, the typical symmetric intergluteal/buttock distribution with sharp borders, and the absence of systemic symptoms strongly supported SDRIFE. In the postoperative setting, SDRIFE may be mistaken for cellulitis, irritant/contact dermatitis, candidiasis, or fixed drug eruption; however, symmetry and the temporal relationship to a β-lactam are key diagnostic clues. Documentation of the culprit drug and consideration of allergy evaluation are important to prevent recurrence.

Conclusions
SDRIFE should be suspected in patients developing sharply demarcated symmetric intergluteal/buttock erythema shortly after systemic drug exposure, particularly amoxicillin–clavulanate. Prompt drug withdrawal and symptomatic therapy lead to rapid resolution.

Abstract N°: ID-1475

Topic: Adverse drug reactions, TEN

Cutaneous effects of biologic treatments: a systematic review

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Introduction
Biologic therapies targeting specific cytokines and immune pathways have revolutionized the management of chronic inflammatory skin diseases such as psoriasis, atopic dermatitis, and hidradenitis suppurativa. Despite their favorable safety profile, these agents can cause a wide range of cutaneous adverse effects that may mimic disease flares or new dermatologic conditions. Accurate recognition is crucial to prevent misdiagnosis or inappropriate treatment changes. The objective of this review is to evaluate cutaneous reactions to commonly used biologics and to propose a structured diagnostic reasoning framework to guide clinical management.

Materials and Methods
A mini-systematic narrative review was conducted using PubMed databases. Articles published between January 2005 and December 2024 were included. Search terms combined “biologic therapy,” “cutaneous adverse effects,” “paradoxical reactions,” “TNF inhibitors,” “IL-17 inhibitors,” “IL-23 inhibitors,” and “dermatology.” Eligible publications included clinical trials, observational studies, case series, and review articles. Data extraction focused on type of biologic agent, latency to reaction onset, clinical presentation, dermoscopic features when available, and therapeutic outcomes.

Results
The literature demonstrates that injection site reactions represent the most frequent adverse event across biologic classes, typically presenting as transient erythema and edema. Paradoxical inflammatory reactions, including psoriasis induced by anti-TNF agents and eczematous eruptions associated with IL-17 inhibitors, have been consistently reported. Other manifestations include alopecia areata-like patterns, lichenoid eruptions, and neutrophilic dermatoses. Latency periods and lesion morphology vary according to the targeted pathway, providing useful diagnostic clues. This review highlights the importance of structured diagnostic reasoning when evaluating new cutaneous lesions in patients receiving biologic therapies. Differentiating drug-induced reactions from disease relapse or coincidental dermatoses requires careful integration of temporal relationships, lesion morphology, patient history, and treatment response. Dermoscopy may assist in selected cases by revealing patterns inconsistent with the underlying disease. A reasoned approach allows clinicians to individualize management, balancing continuation, temporary interruption, or switching of biologic agents while maintaining disease control.

Conclusions
Cutaneous adverse effects associated with biologic therapies are diverse but often reproducible when interpreted through structured clinical reasoning. Early recognition facilitates optimal management and improves therapeutic adherence. Dermatologists should maintain a high index of suspicion for biologic-related cutaneous reactions and apply systematic diagnostic frameworks to guide therapeutic decisions.

Abstract N°: ID-1481

Topic: Adverse drug reactions, TEN

Bullous Erythema Multiforme Induced by a "Natural" Dietary Supplement Purchased via Social Media: A Case Report

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Introduction
Erythema multiforme (EM) is an acute immune-mediated mucocutaneous disorder most commonly triggered by infections, particularly herpes simplex virus and *Mycoplasma pneumoniae*, and less frequently by medications. Dietary supplements, which are not subject to the same regulatory controls as pharmaceutical drugs, may contain undeclared active substances and represent a growing source of iatrogenic adverse reactions, especially when promoted through social media platforms.

Materials and Methods
We report a clinical case of bullous erythema multiforme in an elderly patient following the use of a dietary supplement purchased online. Clinical examination, drug history, infectious investigations, and therapeutic management were analyzed.

Results
A 70-year-old man with type 2 diabetes mellitus treated with oral antidiabetic agents and benign prostatic hyperplasia presented with an acute erythematous bullous eruption two weeks after initiating a "natural" dietary supplement intended to improve urinary symptoms. Cutaneous examination revealed typical target lesions predominantly involving acral areas, associated with mucosal bullae, consistent with bullous erythema multiforme. Infectious workup was negative, and no other new medications had been introduced. The dietary supplement was discontinued, and systemic corticosteroid therapy was initiated at a dose of 1 mg/kg/day, leading to a favorable clinical outcome.

Conclusions
This case highlights dietary supplements as a potential and often overlooked cause of severe cutaneous adverse reactions. It emphasizes the importance of systematically investigating supplement use in patients presenting with erythema multiforme, particularly in elderly, diabetic, and polymedicated individuals. Increased patient awareness and stricter regulation of online-marketed supplements are essential to reduce iatrogenic risks.

Abstract N°: ID-1482

Topic: Adverse drug reactions, TEN

Preventing Anti-Tuberculosis Treatment Interruption Through Early Dermatologic Management: A Prospective Study

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Introduction

Anti-tuberculosis (anti-TB) therapy is long and frequently associated with cutaneous adverse drug reactions (CADRs), which may compromise treatment adherence and lead to interruption. Data focusing on the role of dermatologic management in preventing treatment discontinuation remain limited, particularly in high-burden tuberculosis settings. This study aimed to evaluate the frequency and spectrum of CADRs related to anti-TB drugs and to assess the impact of early dermatologic intervention on treatment continuation.

Materials and Methods

We conducted a prospective descriptive study at Mohammed V Military Teaching Hospital, Rabat, Morocco, between January and August 2025. Patients with bacteriologically confirmed tuberculosis underwent a baseline dermatological examination before initiation of anti-TB therapy and were followed every three months. Patients with pre-existing dermatological diseases were excluded. Demographic, clinical, and therapeutic data were collected. Cutaneous adverse reactions were recorded, classified, and managed according to severity.

Results

Among 221 patients receiving anti-TB treatment, 31 developed CADRs, corresponding to a frequency of approximately 14%. The median age was 32 years, with a marked male predominance (84%). All patients were immunocompetent and received standard first-line anti-TB regimens in 97% of cases.

The most frequent manifestations were acneiform eruptions (61%), followed by urticarial lesions (26%), pruritus (10%), and DRESS syndrome (3%). Most reactions were mild to moderate and occurred within the first months of treatment. Thanks to early dermatological assessment and tailored management, anti-TB therapy was maintained in 97% of affected patients. Symptomatic treatments included topical retinoids and benzoyl peroxide for acneiform eruptions, antihistamines for urticaria and pruritus, and immediate drug withdrawal with systemic corticosteroids for DRESS syndrome, with definitive discontinuation of the suspected drug.

Conclusions

Cutaneous adverse reactions to anti-tuberculosis drugs are common but mostly benign. Early dermatologic evaluation and appropriate management allow continuation of anti-TB therapy in the vast majority of patients. Dermatologists play a key role in preventing treatment interruption and potentially limiting drug resistance, highlighting the importance of multidisciplinary collaboration in tuberculosis care.

Abstract N°: ID-1484

Topic: Adverse drug reactions, TEN

DRESS syndrome during chemotherapy in a patient on chronic hemodialysis: diagnostic pitfalls in the setting of polypharmacy

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Introduction

Le syndrome d'hypersensibilité médicamenteuse avec éosinophilie et symptômes systémiques (DRESS) est une réaction cutanée indésirable grave aux médicaments, caractérisée par un début retardé, une inflammation systémique et une atteinte multiorganique potentielle. Le diagnostic peut s'avérer complexe chez les patients atteints d'hémopathies malignes, car les infections, l'inflammation liée à la maladie et la polymédication peuvent simuler un DRESS.

Materials and Methods

Nous rapportons le cas d'un homme de 47 ans atteint d'un myélome multiple nouvellement diagnostiqué et d'une insuffisance rénale terminale sous hémodialyse chronique. Il a débuté un traitement à base de bortézomib associé à des traitements de soutien, notamment le triméthoprime-sulfaméthoxazole. Les caractéristiques cliniques, les résultats de laboratoire, l'histopathologie et l'évolution ont été analysés. Les critères RegiSCAR ont été appliqués pour estimer la probabilité diagnostique.

Results

Trois semaines après le début du traitement, le patient a présenté une éruption morbilliforme prurigineuse rapidement progressive, débutant au bas du dos et s'étendant au tronc et aux membres. Il avait de la fièvre (38,4 °C) et une lymphadénopathie axillaire et inguinale bilatérale. Les analyses biologiques ont révélé une hyperleucocytose avec éosinophilie (1,6 × 10^9/L), une élévation des marqueurs inflammatoires et une cytolyse hépatique modérée, tandis que les paramètres rénaux restaient stables sous hémodialyse. La biopsie cutanée a confirmé une réaction d'hypersensibilité médicamenteuse. L'évaluation RegiSCAR était compatible avec un syndrome DRESS **probable** . Tous les médicaments suspectés ont été arrêtés et une corticothérapie systémique (0,5 mg/kg/jour d'équivalent prednisone) a été instaurée, entraînant une amélioration clinique et biologique progressive.

Conclusions

Ce cas souligne l'importance de prendre en compte le syndrome DRESS chez les patients hémodialysés recevant une chimiothérapie et plusieurs médicaments concomitants. L'application de critères validés tels que RegiSCAR, l'exclusion des diagnostics différentiels et l'arrêt rapide des agents suspectés sont des étapes clés pour prévenir les complications systémiques graves.

Abstract N°: ID-1489

Topic: Adverse drug reactions, TEN

An Unexpected Cutaneous Eruption Associated with Cemivil Therapy in Accelerated Phase Chronic Myeloid Leukemia

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Introduction

Cutaneous eruptions are common adverse events associated with tyrosine kinase inhibitors (TKIs) used in the management of chronic myeloid leukemia (CML). These manifestations may represent a drug reaction or, conversely, reflect the underlying progression of the disease itself. We report the case of a patient who developed a macular rash while receiving Cemivil for CML in the accelerated phase.

Materials and Methods

Mrs. F., a 57-year-old patient followed for chronic myeloid leukemia in the accelerated phase, was undergoing treatment with Cemivil and allopurinol. She presented with an erythematous macular eruption that had emerged one week prior. The rash was localized to the trunk and upper limbs, was non-pruritic, and showed no clinical signs of dermatological severity.

Clinical examination revealed moderate tachycardia and palpable splenomegaly, though her overall general health remained stable. Laboratory investigations showed marked leukocytosis, anemia, elevated LDH levels, and a positive BCR-ABL transcript. While the temporal proximity between the initiation of treatment and the eruption suggests a benign cutaneous drug reaction (toxidermal reaction) to Cemivil, a paraneoplastic origin could not be entirely ruled out given the clinical context.

Results

Tyrosine kinase inhibitors, such as Cemivil, are known to induce skin rashes in 15% to 30% of cases. These are typically mild and rarely necessitate the discontinuation of therapy. However, any eruption occurring in a patient with CML must also raise suspicion of a paraneoplastic cutaneous manifestation, particularly during advanced or accelerated phases. In this specific case, the absence of "red flag" symptoms and the close chronological link between starting Cemivil and the onset of lesions point toward a drug-induced rash as the most probable etiology.

Conclusions

This case highlights the necessity of vigilant dermatological monitoring for patients treated with Cemivil. Even a seemingly subtle rash can serve as a clinical signpost for either a treatment reaction or disease evolution. A close collaboration between hematologists and dermatologists remains essential to optimize patient care and refine management strategies without compromising therapeutic efficacy.

Abstract N°: ID-1499

Topic: Adverse drug reactions, TEN

Multiple Ingrown Nails Induced by Anti-EGFR Therapy: A Rare yet Debilitating Adverse Effect

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Introduction

The advent of tyrosine kinase inhibitors (TKIs) targeting the EGFR receptor—such as osimertinib (Tagrisso®)—has fundamentally reshaped the management of non-small cell lung cancer (NSCLC) harboring activating mutations. While their safety profile is generally favorable, cutaneous and nail toxicities can emerge, sometimes appearing late in the course of treatment or following a therapeutic switch. Although dermatological side effects are well-documented, nail complications like ingrown nails (onychocryptosis) remain underreported, particularly in the elderly population. We report a clinical case of multiple ingrown nails affecting both the toes and fingers, occurring rapidly after a medication substitution.

Materials and Methods

We present the case of a 90-year-old patient with a medical history of hypertension managed by amlodipine and insulin-dependent diabetes. She is currently being treated for EGFR-mutated NSCLC. Her initial regimen consisted of osimertinib (Tagrisso®), one tablet daily, which she tolerated well for nine months without notable incidents.

Following a change in therapeutic strategy, the patient was transitioned to Miralta® (a generic form of erlotinib). Within fifteen days of starting this new treatment, she developed escalating pain in her extremities accompanied by localized inflammation. Clinical examination revealed multiple ingrown nails involving not only the toes but also the fingers, presenting as painful paronychia. Notably, there was no fever or signs of systemic infection. A dermatological consultation was requested, and a local regimen of anti-inflammatory and antiseptic treatments was initiated to alleviate her discomfort and manage the lesions.

Results

Nail-related side effects induced by anti-EGFR therapies are significantly less frequent than cutaneous rashes or xerosis; however, their functional impact and the associated pain can be substantial, particularly in elderly and frail patients. The rapid onset of lesions following the transition from osimertinib to erlotinib suggests a strong temporal correlation. While both molecules belong to the same class, their toxicity profiles differ: osimertinib is generally better tolerated from a dermatological standpoint than first-generation inhibitors like erlotinib.

In this patient's case, the convergence of advanced age, diabetes, and likely impaired vascularization—combined with the switch in targeted therapy—undoubtedly predisposed her to such extensive nail complications. The simultaneous involvement of both fingers and toes compounds the functional burden, particularly regarding activities of daily living. This case underscores the vital importance of vigilant monitoring whenever a treatment is modified, even when staying within the same therapeutic class.

Conclusions

Nail-related side effects induced by anti-EGFR therapies are significantly less frequent than cutaneous rashes or xerosis; however, their functional impact and the associated pain can be substantial, particularly in elderly and frail patients. The rapid onset of lesions following the transition from osimertinib to erlotinib suggests a strong temporal correlation. While both molecules belong to the same class, their toxicity profiles differ: osimertinib is generally better tolerated from a dermatological standpoint than first-generation inhibitors like erlotinib.

In this patient's case, the convergence of advanced age, diabetes, and likely impaired vascularization—combined with the switch in targeted therapy—undoubtedly predisposed her to such extensive nail complications. The simultaneous involvement of both fingers and toes compounds the functional burden, particularly regarding activities of daily living. This case underscores the vital importance of vigilant monitoring whenever a treatment is modified, even when staying within the same therapeutic class.

Abstract N°: ID-1539

Topic: Adverse drug reactions, TEN

Acyclovir-Associated Delirium in an Older Adult With Herpes Zoster Ophthalmicus

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Introduction
Acyclovir is commonly used for herpes zoster, including herpes zoster ophthalmicus, and is generally well tolerated. However, neuropsychiatric adverse effects such as delirium and encephalopathy may occur, particularly in older adults and in the setting of impaired renal function. We report a case of acute delirium developing during intravenous acyclovir treatment.

Materials and Methods
A 73-year-old man was hospitalized with herpes zoster ophthalmicus and started on intravenous acyclovir (10 mg/kg, three times daily). He had no prior cognitive impairment, psychiatric disorder, or neurologic disease. On treatment day 3, he developed acute disorientation, agitation, and incoherent speech. A focused delirium work-up was initiated, including laboratory assessment and neuroimaging.

Results
Laboratory investigations revealed stage 1 acute kidney injury, with serum creatinine rising to 1.7 mg/dL from a baseline of 0.84 mg/dL. Electrolyte levels were within normal limits. To exclude organic central nervous system pathology, diffusion-weighted magnetic resonance imaging (MRI) was performed, which revealed no abnormalities. Following a psychiatric consultation, symptomatic treatment with haloperidol was initiated. Suspecting acyclovir-induced neurotoxicity, the intravenous treatment was discontinued. After cessation, the patient's mental status improved gradually, with complete resolution of delirium observed within three days. No residual neuropsychiatric symptoms were noted during the follow-up period.

Conclusions
This case emphasizes acyclovir-associated delirium as a rare but clinically significant adverse effect, especially in elderly patients, even with mild renal impairment. Clinicians should maintain a high index of suspicion for antiviral-induced neurotoxicity when acute mental status changes occur during therapy. Prompt recognition and discontinuation of the offending agent are crucial for rapid and complete recovery.

Abstract N°: ID-1542

Topic: Adverse drug reactions, TEN

Drug reaction with eosinophilia and systemic symptoms induced by Interferon Beta-1a: A case report

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Introduction

Drug reaction with eosinophilia and systemic symptoms (DRESS) is a potentially severe acute drug reaction, with an estimated mortality rate of approximately 10%. Interferon beta-1a is an immunomodulatory agent widely used in the treatment of multiple sclerosis to reduce relapse frequency and slow disease progression. This report aims to describe the clinical presentation and therapeutic management of a patient who developed DRESS syndrome induced by interferon beta-1a.

Materials and Methods

We report the case of a 29-year-old woman followed for multiple sclerosis who developed DRESS syndrome after initiation of interferon beta-1a therapy, with a favorable outcome under appropriate treatment.

Results

A 29-year-old woman with a recent diagnosis of multiple sclerosis was treated with weekly intramuscular injections of interferon beta-1a at a dose of 30 micrograms. Five weeks after the first injection, she developed fever up to 39 °C, bilateral lower limb edema, and a diffuse erythematous–violaceous morbilliform maculopapular rash, with pseudotargetoid lesions in some areas. There was no skin detachment, and the estimated body surface area involved was 72%. Mucosal examination was normal. Physical examination revealed right axillary and left inguinal lymphadenopathy of lenticular size. Pulmonary examination was unremarkable.

Laboratory investigations showed marked eosinophilia associated with impaired liver and renal function tests. Stool examination for parasites and viral serologies (HBV, HCV, EBV, CMV) were negative. A diagnosis of DRESS syndrome was established with a RegiSCAR score of 5, implicating interferon beta-1a, with an extrinsic imputability score of B3 and an intrinsic score of I3 according to the French causality assessment method. The patient was treated with systemic corticostotherapy (prednisone 0.5 mg/kg/day), resulting in clinical improvement, skin desquamation from day 5 of treatment, and progressive normalization of biological abnormalities.

Conclusions

DRESS syndrome should be considered a rare but possible complication of interferon beta-1a therapy. Its use requires careful clinical monitoring, and clinicians should remain vigilant for severe cutaneous adverse drug reactions, particularly in the early weeks of treatment.

Abstract N°: ID-1577

Topic: Adverse drug reactions, TEN

Acute generalized exanthematous pustulosis induced by Panitumumab: A case report drug hypersensitivity and allergology

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Introduction

Panitumumab, an anti-EGFR monoclonal antibody, is widely used in metastatic colorectal cancer. Cutaneous adverse effects are common, mainly papulopustular follicular rash, considered a marker of therapeutic efficacy. In contrast, acute generalized exanthematous pustulosis (AGEP) is an exceptionally severe drug reaction under anti-EGFR therapy, with very few cases reported. We describe the clinical, biological, and histological features of AGEP induced by panitumumab and the patient's evolution after treatment discontinuation.

Materials and Methods

We report the case of a 60-year-old patient hospitalized for AGEP following panitumumab therapy.

Results

The patient, treated for left-sided colon adenocarcinoma with peritoneal carcinomatosis, received palliative surgery followed by biweekly intravenous panitumumab. Five days after initiation, he developed a generalized morbilliform macular rash over the trunk and upper limbs, topped with numerous non-follicular pinhead-sized pustules, without mucosal involvement, lymphadenopathy, or fever. Laboratory tests revealed leukocytosis with neutrophilia; other parameters were normal. Histopathology showed spongiosis with neutrophilic infiltration of the epidermis and papillary dermal edema, confirming the diagnosis of AGEP. Panitumumab was immediately discontinued. Lesions began to regress, with desquamation starting five days after treatment cessation.

Conclusions

AGEP induced by panitumumab is rare but possible. Early recognition and careful clinical monitoring are essential for appropriate management. This case highlights the need to document rare drug reactions to guide clinical practice and improve patient safety.

Abstract N°: ID-1587

Topic: Adverse drug reactions, TEN

Acneiform rash induced by Panitumumab: Role of allergy assessment

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Introduction

Panitumumab, an anti-EGFR monoclonal antibody, is frequently associated with cutaneous toxicities, particularly acneiform rash, which can be mistaken for drug hypersensitivity. Misinterpretation may lead to unnecessary treatment interruptions or inappropriate management. This study aims to describe the clinical characteristics of panitumumab-induced acneiform rash and to evaluate the role of allergy assessment in differentiating this toxicity from true drug hypersensitivity, thereby optimizing patient management.

Materials and Methods

A retrospective descriptive study was conducted on 14 patients treated with panitumumab who were referred to an allergy consultation for a suspected drug-related skin eruption. Demographic, clinical, chronological, biological, therapeutic, and outcome data were analyzed.

Results

All patients (100%) were receiving panitumumab for solid tumors, including 71% for colorectal cancer and 29% for other digestive cancers. The cohort included 10 men (71%) and 4 women (29%), with a mean age of 61 ± 8 years. The rash appeared between 7 and 21 days after the first dose in 86% of patients, with a median onset of 12 days. Lesions were papulo-pustular in all cases, affecting the face (100%), neck (86%), scalp (43%), and trunk (57%), without mucosal involvement or systemic signs. Mild pruritus was reported in 29% of patients. No eosinophilia or clinical/biological evidence of an allergic mechanism was observed. Management consisted of topical emollients and corticosteroids in all patients, with local or systemic antibiotics in 57%. No permanent discontinuation of panitumumab was required, and clinical outcomes were favorable in all cases.

Conclusions

Acneiform rash induced by panitumumab is a common cutaneous toxicity that may mimic drug hypersensitivity. Allergy assessment is valuable for confirming the diagnosis, avoiding unnecessary therapy interruptions, and optimizing patient care.