Abstract N°: 179

Exosomes derived from adipose tissue-derived mesenchymal stem cells (ASCE) for the treatment of dupilumab-related facial redness (DFR) in patients with atopic dermatitis

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Introduction & Objectives:

Atopic dermatitis (AD) is a chronic, pruritic, and inflammatory dermatosis affecting approximately 20% of children and 10% of adults worldwide. Dupilumab facial redness (DFR) is gaining attention as additional cases are coming to light in the medical literature. Recent studies have reported promising results of exosomes for skin aging. Exosomes are nano-sized (30 - 200 nm in diameter) lipid bilayered vesicles secreted by most cell types. Adipose stem cell exosomes (ASCE) are emerging as effective cell-free therapeutic agents to treat a variety of skin aging.

To understand that adipose stem cell exosome (ASCE) can be a paradigm shift in regenerative aesthetics & therapeutics

To find the clinical efficacy and safety of adipose tissue stem cell-derived exosomes as adjuvant therapy after the application of micro-needling for dupilumab-related facial redness

Materials & Methods:

Twenty patients on dupilumab for AD and diagnosed with DFR were enrolled in a 12-week prospective clinical study. investigated whether topical application of adipose stem cell exosomes could reduce dupilumab facial redness in patients. ASCE was applied to each entire face of patients and prism sonophoresis for effective delivery. This topical application of exosomes was performed weekly for five consecutive weeks

Results:

The average investigator global assessment (IGA) and clinical erythema assessment (CEA) scores significantly decreased from week 2 until week 12. Skin erythema was measured at the forehead, chin, and right and left cheek. Erythema index (EI) decreased in all four areas after ASCE treatment from week 4. Skin hydration was increased and TEWL was decreased after ASCE treatment at four different anatomical areas. The mRNA expression of IL-1a was significantly decreased in stratum corneum samples, while FLG (filaggrin) and VEGF (vascular endothelial growth factor) were significantly increased.

Conclusion:

ASCE downregulated inflammation and increased skin barrier-related proteins and angiogenesis. In addition, ASCE can result in a therapeutic effect by topical application which is effective for DFR manifests since DFR is localized skin lesions. Thus, Adipose stem cell exosomes may serve as effective management to treat dupilumab facial redness.

Abstract N°: 225

A Rare Clinical Encounter: DRESS Syndrome Complicated by Acute Pancreatitis

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Introduction:

DRESS syndrome is a rare but potentially life-threatening drug-induced hypersensitivity reaction. It usually presents 2-6 weeks after drug exposure with fever, widespread skin eruptions, lymphadenopathy, eosinophilia, and multi-organ involvement, primarily affecting the liver, kidneys, and lungs. Mortality rates range from 10% to 20%. Pancreatic involvement is uncommon and often underrecognized, presenting as acute pancreatitis or, less commonly, fulminant type 1 diabetes mellitus. Data on its exact incidence remain limited, but it is considered a rare gastrointestinal manifestation of DRESS. Here, we report a rare case of DRESS syndrome complicated by acute pancreatitis

Case Report:

A 37-year-old female with no medical history was admitted for DRESS syndrome following exposure to phenobarbital, chlorpromazine, amitriptyline, and sertraline. She developed fever (39°C), facial edema, lymphadenopathy, and a morbilliform rash covering 90% of her body, sparing the palms and soles. Laboratory findings showed eosinophilia (2,000 cells/µL), AST 1,000 U/L, ALT 600 U/L, and 27% atypical lymphocytes. A RegiSCAR score >5 confirmed the diagnosis, and corticosteroid therapy (1 mg/kg/day) was initiated. Two weeks later, the patient developed severe epigastric pain, bilious vomiting, and tenderness. Serum lipase was elevated (1,800 U/L), and abdominal ultrasound ruled out biliary obstruction and gallstones. The pancreas appeared normal without peripancreatic fluid collections, leading to a diagnosis of acute pancreatitis. Management included IV rehydration, electrolyte correction, IV methylprednisolone, and multimodal analgesia with subcutaneous morphine (5 mg/6h). The patient achieved satisfactory pain control (VAS <3). Oral feeding was initially poorly tolerated but successfully resumed by day 3 without recurrence of symptoms.

Discussion:

Hepatic involvement is the most common in DRESS (up to 80%), while pancreatic manifestations remain rare. The pathogenesis of DRESS-associated pancreatitis is not well understood but is likely immune-mediated. Drug-induced pancreatitis is a known adverse effect of medications such as phenobarbital, making it difficult to differentiate from DRESS-related pancreatic involvement. In this case, the temporal association suggests DRESS as the primary cause.

Conclusion:

This case highlights the importance of considering acute pancreatitis as a rare but significant complication of DRESS syndrome. Clinicians should maintain high suspicion for pancreatic involvement in DRESS patients with gastrointestinal symptoms. Early recognition and appropriate management are crucial for improving patient outcomes.

Abstract N°: 248

N-Acetylcysteine as an Adjuvant Therapy in Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis Overlap Syndrome with Chronic Kidney Disease: A Case Report

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Introduction & Objectives:

Stevens-Johnson syndrome/toxic epidermal necrolysis (SJS/TEN) overlap syndrome is an acute and life-threatening mucocutaneous reaction, characterized by epidermal necrolysis of 10-30% of the body surface, accompanied by mucosal lesions. There is a hypothesis that SJS/TEN overlap syndrome is caused by drug or drug metabolites that are toxic, accumulated in epidermal cells due to disturbances in metabolic pathways. N-acetylcysteine (NAC) act as an antioxidant and has a detoxifying effect, may be useful for the treatment of SJS/TEN overlap syndrome, especially in patients with chronic kidney disease (CKD). We aim to study the effect and safety of NAC for the patient with SJS/TEN with CKD.

Materials & Methods:

This is a case report of a 53-year-old female patient with CKD stage IV and hypertension, admitted with SJS/TEN overlap syndrome caused by recent use of allopurinol for 14 days. SCORTEN on first day was 3. Estimated glomerular filtration rate (eGFR) at the first day of admission was 21.5, but she refused the hemodialysis treatment. We stopped the allopurinol, treated the SJS/TEN overlap syndrome with intravenous (IV) dexamethasone 5 mg iv 3 times/day; oral NAC 200 mg 3 times/day, and topical treatments. She also received IV ceftriaxone 2 g/day and her routine hypertension medication. After day-6 of treatment, new lesion were not found, the corticosteroid was tappered off.

Results:

Reepithelialization occurred in 8 days of treatment, the patient's condition was stabile when she was discharged. There was no adverse effect found on follow up day-10. No sequelae was found on the second month follow up, the eGFR was 22.4. There were reports regarding the use of NAC in treating SJS and TEN but its use in patients with kidney disease is not well established.

Conclusion:

Treatment with oral NAC were effective and safe as an adjuvant for SJS/TEN overlap syndrome, even in patients with CKD. Further studies with more samples are needed.

Abstract N°: 324

Efficacy and safety of a topical cream containing adipose tissuederived stem cell-derived exosomes in mitigating radiation dermatitis after adjuvant radiotherapy for breast cancer

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Introduction & Objectives:

Acute radiation injury of the skin is a common and severe side effect among breast cancer patients undergoing RT (radiotherapy). Although RT preferentially targets tumor cells, damage to healthy tissue in the path of radiation beams is inevitable. The skin is especially sensitive to the toxic effects of RT because of the high cellular turnover rate. Studies by Harper et al. and Lopez et al. have reported radiation dermatitis in up to 90% of patients treated with RT for breast cancer, respectively. Radiation dermatitis causes considerable discomfort and limits daily activities. Moreover, severe radiation dermatitis leads to increased fibrosis and pigmentation, adversely affecting cosmetic outcomes and thus the overall quality of life following breast-conserving surgery.5 Therefore, although it is impossible to completely prevent radiation dermatitis, it is crucial to devise strategies to mitigate radiation dermatitis.

This study aimed to assess the efficacy and safety of a topical cream containing adipose tissue-derived mesenchymal stem cell-derived exosomes (ASC-EXOs) in mitigating radiation dermatitis in breast cancer patients undergoing RT.

Materials & Methods:

An 8-week prospective, single-center pilot study was conducted to evaluate the clinical efficacy and safety of a cream containing ASC-EXOs. A total of 15 patients undergoing RT applied the cream to the radiation exposure site, starting the night prior to the initiation of RT and continuing until 2 weeks following the conclusion of RT. Radiation dermatitis was evaluated at baseline and at weeks 2, 4, 6, and 8 using a 5-point grading system advocated by the Radiation Therapy Oncology Group (RTOG).

Results:

The RTOG score was the highest at week 6, consistent with the point at which the effects of radiation treatment were most cumulative. During the study, no patient had acute toxicity with a grade of 3 or 4. Evaluation of the maximum RTOG scores showed radiation dermatitis with maximum grades of 0, 1, and 2 in 6.7% (1/15), 40% (6/15), and 53.3% (8/11) of patients, respectively. Transepidermal water loss (TEWL) was increased at week 6 but was decreased by week 8 to a point that was even lower compared with the baseline.

Conclusion:

The overall incidence of radiation dermatitis in our study was notably lower than that previously documented, demonstrating the positive effects of the cream containing ASC-EXOs in mitigating radiation dermatitis. Furthermore, the results of TEWL analysis indicated that the topical application of the ASC-EXO-containing cream was effective in maintaining the integrity of skin barrier function.

Abstract N°: 365

Chlorpromazine-Associated Cutaneous and Ocular Pigmentary Changes: A Case Report

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Introduction & Objectives:

Chlorpromazine, a typical antipsychotic of the phenothiazine class, is widely used in the treatment of psychiatric disorders. Phenothiazines are known to cause cutaneous hyperpigmentation, particularly with prolonged, high-dose use. This side effect, though rare, is well-documented. Studies suggest the underlying mechanism involves photosensitizing properties and oxidative metabolites leading to pigment formation. This hyperpigmentation can be progressive, often requiring a switch to alternative medication.

Materials & Methods:

N/A

Results:

This report details a 57-year-old male patient with psychotic depression who developed diffuse skin hyperpigmentation, predominantly in sun-exposed areas, after long-term, high-dose chlorpromazine treatment. Due to limited response to other therapies, the patient was prescribed chlorpromazine at doses up to 800 mg/day for approximately eight years. Abnormal skin pigmentation developed after about six years of treatment, progressively worsening over time. Dermatological examination revealed increased pigmentation with bluish-gray discoloration, concentrated in sun-exposed areas, including the malar region, nasal dorsum, glabella, and scalp, sparing the periorbital, nasolabial folds, and wrinkles. The patient did not report any associated pain or pruritus. A skin biopsy from the forehead showed histiocytes containing brownish pigment in the superficial dermis. Fontana-Masson staining confirmed the presence of melanin in these pigments. The patient was also evaluated by the ophthalmology team, which revealed fine grayish punctate deposits in the cornea, without abnormalities in the fundoscopic examination, i.e., no retinal changes were observed. The patient denied any ophthalmologic complaints.

Conclusion:

The pathophysiology of chlorpromazine-induced cutaneous hyperpigmentation is multifaceted and not fully understood. A chlorpromazine metabolite, 7-hydroxychlorpromazine, is suspected to play a role, as it turns purple upon exposure to sunlight and is oxidized by melanoproteins to form a melanin-like pigment. Ultrastructural studies indicate that the pigment is likely a complex formed by chlorpromazine (or a metabolite) with melanin. These pigmentary products can elicit a mild inflammatory response, resulting in the presence of pigment-containing macrophages in the dermis, as observed in the patient's biopsy. Early diagnosis is crucial to prevent progression and minimize negative impacts on the patient's quality of life. Based on the patient's history and biopsy findings, the decision was made to gradually reduce the chlorpromazine dose and transition to an atypical antipsychotic, given their improved safety profile. Rigorous photoprotection measures, including sunscreen use and avoidance of direct sun exposure, were also recommended. Resolution of hyperpigmentation can be slow, sometimes taking months or even years. This case underscores the importance of monitoring patients on long-term chlorpromazine therapy for this adverse effect, including dermatologic and ophthalmologic evaluations.

Abstract N°: 380

Cutaneous and Mucosal Manifestations of Immune Thrombocytopenia Following Hepatitis B Vaccination: A Rare Adverse Event and Clinical Challenge

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Introduction: Immune thrombocytopenia (ITP) is an autoimmune disorder characterized by the production of autoantibodies that target and destroy platelets, leading to isolated thrombocytopenia. The most common symptoms of ITP include bleeding, ranging from mild petechiae and epistaxis to severe intracranial hemorrhage. Although ITP following vaccination is rare, it has been recognized as a potential adverse effect, particularly following the MMR and varicella vaccines. The association between the hepatitis B vaccine and ITP is infrequently reported in the literature.

Case report: We report the case of a 30-year-old male who presented with a 24-hour history of skin rash, oral bleeding, and epistaxis. His medical history was unremarkable, and he had no history of medication use. Physical examination revealed disseminated purpuric macules and petechiae, most noticeable at pressure points (Koebner phenomenon), as well as hemorrhagic bullae on the buccal mucosa, hard palate, and tongue. Upon admission, the patient had a markedly reduced platelet count of 4×10^9/L, while other blood parameters remained within normal limits. The sternal puncture revealed no pathological changes, and the abdominal ultrasound was unremarkable. Immunological testing showed a positive antinuclear antibody (ANA) titer of 1:80, but tests for anticardiolipin IgM/IgG, Beta-2 glycoprotein IgM/IgG, and ENA screen were negative. Viral testing showed elevated hepatitis B surface antigen (HBsAg > 1000 mIU/ml) antibodies, indicating prior exposure to hepatitis B, while tests for other acute viral infections (HIV, HCV, CMV, VZV, Parvo B19, EBV) were negative. Upon further history-taking, the patient disclosed receiving the second dose of recombinant hepatitis B vaccine 8 weeks before symptom onset, raising suspicion for vaccine-induced ITP. The patient was initially treated with high-dose intravenous corticosteroids and intravenous immunoglobulin (IVIG) over four days, leading to a significant increase in platelet count. He was continued on oral prednisolone at a dose of 100 mg daily with a gradual tapering regimen, and his dosage was adjusted several times in response to fluctuations in platelet levels; two months later, azathioprine was initiated to further manage his condition. Long-term follow-up was recommended to monitor progress and adjust treatment as necessary. Given the potential risk of exacerbation, the patient was advised to avoid the third dose of the hepatitis B vaccine.

Discussion: In the literature, to our best knowledge, there have been only 48 reported cases of ITP following hepatitis B vaccination, predominantly in children. According to the Adverse Drug Reaction (ADR) Probability Scale, our patient scored 5, classifying the ITP as a probable adverse reaction to the hepatitis B vaccine. Although rare, vaccine-induced ITP should be considered as a potential cause of thrombocytopenia, particularly in individuals without a prior history of platelet disorders. The widespread use of the hepatitis B vaccine suggests that such cases may be underreported.

Conclusion: This case highlights the rarity and potential severity of vaccine-induced ITP following widely used hepatitis B vaccination. Early recognition and intervention are crucial for positive outcomes. Although underreported, this adverse reaction underscores the need for heightened awareness, cautious management, and long-term follow-up to prevent complications and ensure optimal care.

Abstract N°: 469

Clinical, Biological, and Histological Features of Severe Drug Reactions: A Retrospective Study of 10 Cases

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Introduction & Objectives:

Severe drug-induced skin reactions, such as DRESS (Drug Reaction with Eosinophilia and Systemic Symptoms), Lyell's syndrome, and pseudo-Lyell reactions, are serious conditions often associated with systemic organ involvement, which can be life-threatening. This study aims to analyze the clinical, biological, and histological characteristics of 10 patients presenting with severe toxidermia.

The objectives of this study were : -To describe the clinical, biological, and histological characteristics of severe toxidermia in a Tunisian cohort. -To identify the medications frequently implicated and associated risk factors -To evaluate systemic complications and clinical outcomes

Materials & Methods: This is a descriptive, retrospective study spanning 8 years (2016-2024), collecting medical records of patients hospitalized in the internal medicine department of Habib Thameur Hospital in Tunis for severe toxidermia. Clinical, biological, and histological data were analyzed.

Results:

Among the 10 patients (8 women, 2 men), the mean age was 60.4 years. Diagnoses included 7 cases of DRESS syndrome, 1 case of Lyell's syndrome, 1 case of pseudo-Lyell, and 1 unspecified toxidermia. Cutaneous manifestations included pruritic hyperpigmented plaques in 4 patients, skin detachment in 1 patient, pseudo-cocarde lesions in 1 patient, and non-specific skin lesions in 3 patients. Biological findings included hepatic cytolysis (6 patients), cholestasis (5 patients), and renal failure (4 patients). Histologically, 6 patients showed lymphocytic and eosinophilic infiltration, 3 had associated neutrophilic infiltration, and 3 presented with keratinocyte necrosis. One patient exhibited orthokeratotic hyperkeratosis without specific lesions.

Four female patients had systemic lupus erythematosus (SLE), with two developing associated ganglionic tuberculosis. Two SLE patients developed DRESS syndrome due to pyrazinamide. One case of pseudo-Lyell and one unspecified toxidermia were noted in the other two patients. Three patients with rheumatoid arthritis developed DRESS syndrome in response to methotrexate, erythromycin, and Non-steroidal anti-inflammatory drugs (NSAIDs). One patient with multiple autoimmune syndrome developed Lyell's syndrome following Bosentan and died. Another patient with psoriatic arthritis developed DRESS syndrome due to fusidic acid.

Conclusion:

This study highlights the clinical and histological diversity of severe drug reactions, with lymphocytic and eosinophilic infiltration being the most common findings. The presence of neutrophilic infiltration and keratinocyte necrosis may indicate more severe forms. Visceral involvement, such as hepatocellular damage, cholestasis, and renal failure, was frequent, underscoring the need for rapid multidisciplinary intervention. Patients with systemic diseases, particularly SLE and rheumatoid arthritis, appear to be at higher risk, especially when

treated with immunosuppressive agents or antibiotics. Severe drug reactions represent medical emergencies requiring heightened vigilance, particularly in high-risk populations. A better understanding of the immunological mechanisms involved could improve prevention and treatment strategies

Abstract N°: 544

Nicolau syndrome and acute localised exanthematous pustulosis secondary to intramuscular injection of stanozolol

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Introduction & Objectives:

Nicolau syndrome, or embolia cutis medicamentosa, is a rare local complication resulting from the intramuscular, intra-articular, or subcutaneous administration of treatments. It is characterised by the immediate onset of pain following administration, followed by erythema, purpura, and ultimately a necrotic plaque. This occurs due to damage to small- and medium-calibre dermohypodermic vessels, leading to cutaneous infarction.

Acute localised exanthematous pustulosis (ALEP) is a toxicodermatosis clinically characterised by the appearance of sterile pustules on an erythematous base, occurring 48–72 hours after the administration of the responsible drug.

The objetice is to describe a case of Nicolau syndrome (embolia cutis medicamentosa) and acute localised exanthematous pustulosis (ALEP) secondary to the intramuscular injection of stanozolol.

Materials & Methods:

Patient’s medical history

Results:

A healthy 36-year-old male presented to the dermatology department with a large retiform purpura on the right arm, accompanied by overlying pustules, which had been present for five days. He did not report fever but experienced functional impairment primarily due to pain. Symptoms had begun immediately after the intramuscular administration of stanozolol for the purpose of increasing muscle mass; it was the third weekly cycle. Laboratory tests revealed no coagulation abnormalities. Microbial cultures from the pustules were negative. A skin biopsy demonstrated a subcorneal pustule, necrotic keratinocytes, vascular thrombosis in the mid-dermis, and necrosis of the eccrine glands. A diagnosis of embolia cutis medicamentosa an ALEP following intramuscular stanozolol administration was established. Symptomatic treatment was prescribed, leading to gradual resolution and recovery of limb function within four weeks.

Stanozolol is a synthetic derivative of dihydrotestosterone, exhibiting androgenic virilizing effects, promoting protein synthesis, and enhancing muscle development. Although it has certain medical applications, it is also used by some athletes to improve body composition, and its use is considered doping.

Conclusion:

We present the first reported case in the literature of Nicolau syndrome and ALEP secondary to intramuscular stanozolol administration. Dermatologists should be aware of this complication and these drugs due to their increasingly widespread use among individuals who engage in sports or intense physical exercise.

Abstract N°: 585

Menstrual Fixed Drug Eruption : A Case Report

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Introduction & Objectives:

Fixed drug eruption (FDE) is a cutaneous adverse reaction most commonly associated with nonsteroidal anti-inflammatory drugs (NSAID), analgesics, antibiotics, and anticonvulsants. Its occurrence during the menstrual period, even in the presence of medication use, should raise suspicion for other differential diagnoses.

Materials & Methods:

We report a case of periodic FDE occurring during menstruation triggered by NSAID.

Results:

A 25-year-old female patient with no notable pathological history consulted for erythematous plaques evolving in an intermittent course over the past five months. The medical history revealed recurrent episodes coinciding with menstruation and resolving spontaneously afterward, leaving hyperpigmented scars. Clinical examination revealed the presence of three slightly infiltrated erythematous-violaceous plaques located on the buttocks, the left thigh, and the nape of the neck. The rest of the dermatological examination was unremarkable. Upon further questioning, the patient reported taking an NSAID during her periods due to intense pain and the diagnosis of FDE was confirmed. A pharmacovigilance investigation was conducted, identifying the NSAID as the causative agent, leading to its contraindication for future use. The outcome was marked by the healing of the lesions and the absence of recurrence following discontinuation of the treatment.

Conclusion:

Our study highlights a case of menstrual-induced FDE triggered by the use of an NSAID. This observation leads us to consider an important differential diagnosis: autoimmune progesterone dermatitis (APD), a rare cyclic premenstrual reaction to progesterone produced during the luteal phase of the menstrual cycle, whose clinical and histological symptoms overlap with those of other dermatoses, such as FDE. In this condition, clinical signs appear during the premenstrual period, when progesterone levels are elevated, making it essential to consider APD in the absence of drug intake. The diagnosis must then be confirmed by a positive intradermal progesterone sensitivity test. In our case, the onset of lesions during menstruation, the history of NSAID use, and the absence of recurrence after discontinuation of the causal treatment strongly support the diagnosis of FDE.

Abstract N°: 713

Efficacy of Vitamin D3 in Preventing Chemotherapy-Induced Hand-Foot Syndrome: A Pilot Study

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Introduction & Objectives:

Toxic erythema of chemotherapy (TEC) is an uncommon but impactful dermatologic condition in chemotherapy patients, that in severe cases may require cessation of chemotherapy, negatively affecting disease prognosis. Previous studies have shown that high doses of vitamin D (hdvD) can play a role in the treatment of TEC.

The present study explores the preventive role of hdvD using a standardized dose and monitoring creatinine and electrolytes to assess the effectiveness and safety of the treatment.

Materials & Methods:

This single-center, randomized, controlled, and blinded clinical trial aims to evaluate the effect of hdvD in the primary prevention of TEC in a tertiary care hospital in Mexico City. Ethical approval was obtained from the hospital's committee. From 250 patients 30 met the inclusion criteria. Using a standardized dose of 100,000 IU of vD on Day 1 and Day 7 of the first cycle of chemotherapy in patients aged 18 years and older who were naïve to treatment, blood tests were performed to measure serum levels of vD, and electrolytes before and after the interventions (Day 0 and Day 8). (Fig. 1). The primary outcome was defined as a reduction in the incidence TEC by at least 40% with the administration of vitamin D3 compared to a control group without treatment in patients undergoing high-risk chemotherapy. Fisher's exact test assessed associations between categorical variables, while the Mann-Whitney U test was applied for continuous variables. Statistical significance was defined as $P < 0.05$.

Results:

From August 2023 to May 2024, 30 patients were recruited as candidates to receive hdvD for the prevention of TEC. A comparative analysis of demographic and baseline characteristics (Table I), as well as serum levels of vitamin D, calcium, phosphorus, creatinine, albumin, and ionized calcium before and after the interventions, was conducted between the hdvD prevention and placebo groups (Table II). Statistical analysis of these variables showed no significant differences between the groups , except for the notable increase in serum vitamin D levels ($p < 0.001$), with no increase in creatinine levels or the other measured serum electrolytes. The median follow-up was 123 days (21-309).

Of 30 patients, 4 developed TEC. 3 in the placebo group. Statistical analysis of variables showed no significant differences between the groups, except for the notable increase in serum vitamin D levels, with no increase in creatinine levels or the other measured serum electrolytes. (Table III)

Conclusion:

In 2022, Nguyen et al. reported on six patients with TEC who were treated with hdvD, finding a reduction in the time to TEC improvement from 2-4 weeks to 1-4 days. In January 2024, two more cases were published, showing

excellent results in the treatment of radiodermatitis with hdvD.

This study evaluated the role of hdvD in the prevention of TEC, It is known that vD has a significant impact on the regulation of autophagy of M2 macrophages, which improves epidermal regeneration and suppresses the inflammatory response once triggered. Although our study did not demonstrate a preventive role for hdvD in the development of TEC, it confirmed its safety in patients undergoing high-risk chemotherapy. The study is limited by its single-center design. It appears that the risk of TEC is associated with the nature of chemotherapy itself, and the role of vD may be therapeutic rather than preventive.

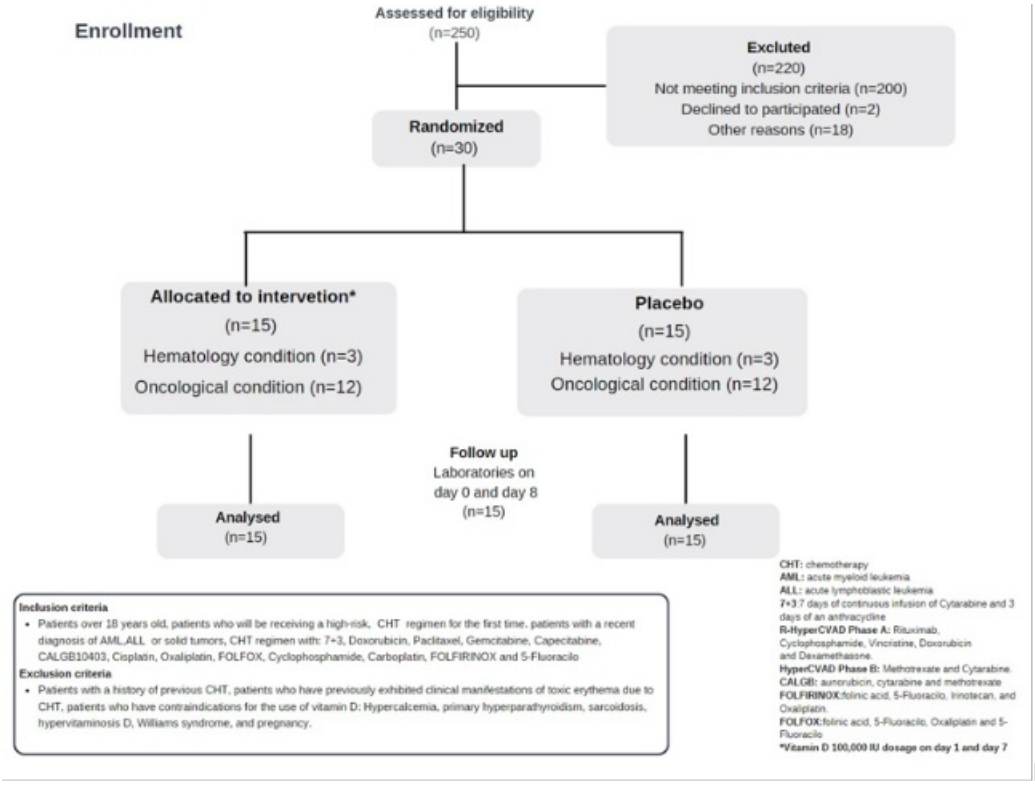

Fig. 1

Table 1. Sociodemographic and clinical traits in placebo and prevention groups

Sociodemographic and clinical traits in placebo and prevention groups			
	Placebo n=15	Vitamin D n=15	Total n=30
Age (years)	55.5 (23 - 82)	55.5 (19 - 71)	55.5 (19 - 82)
Sex n (%)	M: 5 (16.6%) F: 10 (33.4%)	M: 6 (20 %) F: 9 (30%)	M: 11 (36.6%) F: 19 (63.4%)
Primary hemato-oncology diagnosis			
Hematological	n=3 (10%)	n=3 (10%)	n=6 (20%)
ALL	2 (6.66%)	3 (10%)	5 (16.66%)
MPAL	1 (3.34%)	-	1 (3.34%)
Oncological	n=12 (40%)	n=12 (40%)	n=24 (80%)
Colon cancer	3 (10%)	3 (10%)	6 (20%)
Breast cancer	3 (10%)	3 (10%)	6 (20%)
Pancreatic cancer	2 (6.66%)	-	2 (6.66%)
Cholangiocarcinoma	2 (6.66 %)	-	2 (6.66%)
Others	2 (6.66 %)	6 (20%)	8 (27%)
Chemotherapy regimen			
Hematology	n=3 (10%)	n=3 (10%)	n=6 (20%)
	2 = CALGB 10403 (6.66%)	2 = CALGB 10403 (6.66 %)	4 = CALGB 10403 (13.34%)
	1 = R-HyperCVAD Fase A (3.34%)	1 = R-HyperCVAD Fase A (3.34%)	2 = R-HyperCVAD Fase A (6.66%)
Oncology	n=12 (40%)	n=12 (40%)	n= 24 (80%)
	1= Doxorubicin + cyclophosphamide (3.33%)	1= Doxorubicin + cyclophosphamide (3.33%)	2 = Doxorubicin (6.66%)
	1= Paclitaxel + carboplatin (3.33%)	2=Doxorubicin (6.66%)	2 = Doxorubicin + cyclophosphamide (6.66%)
	1=Docetaxel + carboplatin (3.33%)	1 = Paclitaxel + doxorubicin + cyclophosphamide (3.33%)	1 = Paclitaxel + doxorubicin + cyclophosphamide (3.33%)
	1=Docetaxel + Oxaliplatin + 5-Fluorouracil (3.33%)	2=Paclitaxel + Carboplatino (6.66 %)	3 = Paclitaxel + carboplatin (10%)
	1=Gemcitabine (3.33%)	1 Paclitaxel + Gemcitabine + cisplatin (3.33%)	1 = Paclitaxel + Gemcitabine + cisplatin (3.33%)
	1= Capecitabine + Oxaliplatin (3.33%)	1= Docetaxel + carboplatin (3.33%)	2 = Docetaxel + carboplatin (6.66%)
	1 = Capecitabine (3.33%)	1=Capecitabine (3.33 %)	1 = Docetaxel + Oxaliplatin + 5-Fluorouracil (3.33%)
	1= Oxaliplatin (3.33%)	1=Oxaliplatin (3.33 %	1 Gemcitabine (3.33%)
	3 = FOLFOX (10%)	2=FOLFOX (6.66 %)	2 Capecitabine (6.66%)
	1= FOLFIRINOX (3.33%)		1 Capecitabine + Oxaliplatin (3.33%)
			2 Oxaliplatin (6.66 %)
			5 FOLFOX (16.66%)
			1 FOLFIRINOX (3.33%)

Table 2. Laboratory test results on Day 0 and Day 8 for placebo and prevention groups

Laboratory test results on Day 0 and Day 8 for placebo and prevention groups				
Baseline laboratories on Day 0 *				
	Placebo n=15	Vitamin D n=15	Total n=30	P value
Serum calcium (mg/dL)	9.06 (7.38-10.42)	8.91 (7.27-9.69)	8.94 (7.27-10.42)	> 0.99
25-hydroxyvitamin D (ng/mL)	23.10 (8.80-35.20)	22.00 (9.30-35.70)	22.55 (8.80-35.70)	> 0.99
Ionized calcium (mg/dL)	4.77 (4.02-5.16)	4.65 (4.40-5.09)	4.69 (4.02-5.16)	> 0.99
Serum phosphorus (mg/dL)	3.40 (2.83-4.90)	3.71 (1.97-4.96)	3.43 (1.97-4.96)	.466
Serum creatinine (mg/dL)	0.71 (0.37-1.16)	0.77 (0.47-1.12)	0.72 (0.37-1.16)	.466
Serum albumin (mg/dL)	3.88 (2.69-4.65)	3.83 (2.34-4.67)	3.85 (2.34-4.67)	> 0.99
Corrected calcium (mg/dL)	9.18 (8.40-10.92)	9.04 (8.14-9.50)	9.15 (8.14-10.92)	.715
Laboratory test on 8th day *				
Serum calcium (mg/dL)	8.75 (7.63-9.32)	8.74 (7.94-9.64)	8.74 (7.63-9.64)	> 0.99
25-hydroxyvitamin D (ng/mL)	18.00 (7.20-33.90)	45.70 (18.50-63.40)	27.81 (7.20-63.40)	p < 0.001
Ionized calcium (mg/dL)	4.90(4.67-5.30)	4.81 (4.45-5.96)	4.89 (4.45-5.96)	.715
Serum phosphorus (mg/dL)	3.70 (0.79-4.56)	4.14 (3.39-4.53)	3.88 (0.79-4.56)	.143
Serum creatinine (mg/dL)	0.69 (0.32-0.93)	0.70 (0.49-1.34)	0.69 (0.32-1.34)	> 0.99
Serum albumin (mg/dL)	3.60 (2.86-4.16)	3.80 (2.89-4.42)	3.67 (2.86-4.42)	.466
Corrected calcium (mg/dL)	8.96 (8.54-9.66)	9.09 (8.60-9.99)	8.98 (8.54-9.99)	> 0.99

Table III. Clinical traits in placebo and prevention groups

	Placebo n=3	Vitamin D n=1	Total n=4
Presented TEC/HFS	3 patient	1 patients	4 patients
Cycle	Cycle 1 – 1 patient Cycle 2- 1 patient Cycle 3 - 1 patient	Cycle 3- 1 patient	1 = cycle 1 1= cycle 2 2 = cycle 3
Type of cancer	2 = Colon cancer 1= Breast cancer	Colon cancer	1 = Breast cancer 3 = Colon cancer
Type of chemotherapy	1=Capecitabine + Oxaliplatin 1=Doxorubicin + Cyclophosphamide 1 = FOLFOX	Capecitabine	1 = Capecitabine 1 = capecitabine + oxaliplatin 1=Doxorubicin + Cyclophosphamide 1 = FOLFOX

Abstract N°: 723

Severe Cutaneous adverse reactions in a local hospital setting in Bangladesh: A 5-year retrospective study

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Introduction & Objectives: *Cutaneous Adverse Drug Reactions (CADR) are common adverse drug reactions that can manifest in many ways and range in severity. The most typical drug allergy symptom is skin manifestation, underscoring how crucial it is to identify the offending medication and stop using it doing so could even save your life in some cases. This study objectives was to investigate the prevalence of severe cutaneous adverse reactions (SCARs) in hospitalized Bangladeshi patients.*

Materials & Methods: *A total of 50 patients were included in the current study. Data were gathered retrospectively from inpatient records of patients who were treated for the disease at the inpatient department of the Combined Military Hospital(CMH), between January 2012 and December 2016. These patients had severe cutaneous adverse effects of drugs. The study comprised 50 patients with SCAR diagnoses in total. The intended sample size of 50 samples was reached using information gathered from patient data collected throughout the research period.*

Results: *Clinical evaluation of the study participants revealed that 46% of cases were SJS(Stevens Johnson Syndrome), 29% were TEN(Toxic Epidermal Necrolysis), 16% were DRESS(Drug Reaction with Eosinophilia and Systemic Symptoms), and 10% were AGEP(Acute Generalized Exanthematous Pustulosis). Drug classes that contain anticonvulsants may have the highest prevalence of SCARs. Carbamazepine caused 22% of patients' SCARs, followed by phenytoin in 16% of instances and phenobarbital in 14% of cases.*

Conclusion: *SCARs have been a serious issue in healthcare for decades. The majority of SCAR is caused by medications that prescribed by doctors and healthcare providers. SCARs were more frequently observed with anticonvulsants from the carbamazepine and phenytoin categories. Continuous monitoring of SCARs is necessary to develop preventive measures.*

Abstract N°: 839

In-vitro detection of suspected drug in maculopapular drug reaction to antibiotics using secreted cytokines from drug-specific activated T cells

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Introduction & Objectives:

Maculopapular drug eruptions (MPE) to antibiotics are associated with enhanced expression of Th1 cytokines like IFN gamma or Th2 cytokines like IL-5. Identifying the culprit drug usually involves re-challenge, which may not be forthcoming. Memory lymphocytes remain responsive to the culprit drug long after the reaction has resolved. Upon reactivation in-vitro, there is increased secretion and expression of certain markers which provides us with an opportunity to predict the causal drug.**

The study aimed to assess drug-specific cytokine production (IL-5 and IFN-gamma) in peripheral blood mononuclear cell culture supernatants to predict the causal antibiotic in maculopapular drug eruptions.

Materials & Methods:

Peripheral blood mononuclear cells of 20 patients who developed MPE to various antibiotics and 11 drug-matched healthy controls were incubated for five days with the respective drugs at two different concentrations. Cytokines secreted were measured in the supernatants at 6 hours, day 2 and day 5 using enzyme-linked immunosorbent assay (ELISA) for IL-5 and IFN-gamma.

Results:

Drug-specific IL-5 and IFN-gamma production could be demonstrated in 65% and 73.6% of the cases, respectively. Maximal secretion of IL-5 and IFN-gamma were observed on day 5 and day 2 of incubation, respectively. Based on the ROC curves, the cut-off Delta values, which is the difference in cytokine concentration of the drug-stimulated and unstimulated sample, were 4 pg/ml and 6 pg/ml for IL-5 and IFN-gamma, respectively. (Figure 1 & 2)

Conclusion:

The measurement of drug-specific secretion of IL-5 and IFN-gamma using ELISA is a valuable method for detection of antibiotic-induced maculopapular exanthema.

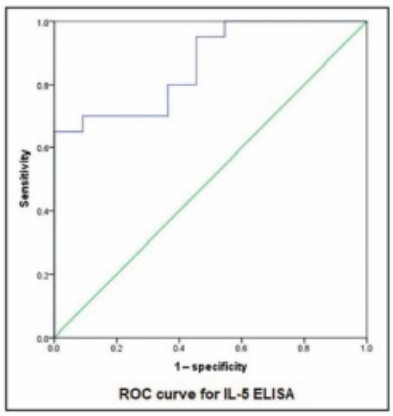

Figure 1 Receiver operating characteristic (ROC) curve for delta value of interleukin (IL)-5 enzyme-linked immunosorbent assay (ELISA) on day 5 from culture supernatant with drug causality algorithm as the reference.

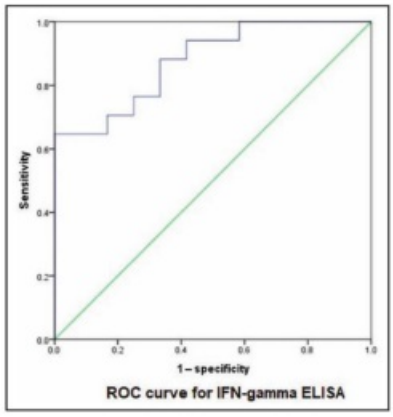

Figure 2 Receiver operating characteristic (ROC) curve for delta value of interferon (IFN)-gamma enzyme-linked immunosorbent assay (ELISA) on day 2 from culture supernatant with drug causality algorithm as the reference.

Abstract N°: 1032

Erythema Multiforme Induced by Albendazole: A Case Report

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Introduction & Objectives:

Drug-induced erythema multiforme is a rare and poorly described variant of erythema multiforme. It is most often triggered by HSV infections and rarely by adverse drug reactions. Albendazole is an antiparasitic drug from the benzimidazole class, widely used to treat helminth infections. Although it has a very high safety profile, hypersensitivity reactions have been reported with various clinical manifestations.

Materials & Methods:

We report a case of erythema multiforme induced by albendazole in a patient.

Results:

A 50-year-old patient with end-stage renal disease undergoing hemodialysis three times a week was diagnosed with a hepatic hydatid cyst one month prior and was prescribed albendazole at a dose of one tablet per day for 10 days. He presented to the emergency department with pruritic erythematous papular lesions on the limbs, which had been evolving for five days before consultation, occurring 20 days after taking albendazole. The patient had no history of herpes, pneumonia, or other infections. On physical examination, multiple well-defined erythematous maculopapular targetoid lesions, some confluent into plaques, measuring 1 to 4 cm in diameter, were symmetrically distributed on the upper and lower limbs, with no mucosal involvement. The rest of the somatic examination was unremarkable. Laboratory tests revealed no abnormalities. A skin biopsy showed a pericapillary lymphohistiocytic infiltrate in the superficial dermis, without vascular wall abnormalities, along with mild lymphocytic exocytosis in the epidermis. The diagnosis was established based on anamnesis and clinical findings. A report of the drug reaction was submitted to the pharmacovigilance center. The clinical course was marked by significant improvement, with spontaneous regression of the skin lesions over 20 days.

Conclusion:

Erythema multiforme is an acute skin and mucosal eruption typically triggered by infections or allergic drug reactions. It is classified as a type IV hypersensitivity reaction, in which T-cell-mediated immune mechanisms cause skin inflammation. Common triggers include viral infections, particularly herpes simplex virus, and bacterial infections such as *Mycoplasma pneumoniae*, as well as certain medications.

Clinically, erythema multiforme presents with a spectrum of severity, from mild forms to severe forms with mucosal involvement. The eruption often manifests as red macules or papules, sometimes merging, and is often accompanied by target lesions, which are characteristic of the condition. Histologically, it begins with swelling of endothelial cells and dermal edema, followed by an inflammatory infiltrate mainly composed of lymphocytes and histiocytes around blood vessels. In later stages, epidermal destruction occurs, with partial or total epidermal necrosis, changes at the dermo-epidermal interface, and the formation of blisters either within the epidermis or

beneath it.

In most cases, erythema multiforme resolves spontaneously within three to five weeks without leaving permanent skin damage, though recurrences can occur, particularly upon re-exposure to the triggering agent. In the case reported, albendazole, an antiparasitic drug, appears to have induced erythema multiforme. Although it is generally well-tolerated, albendazole can cause hypersensitivity reactions ranging from mild rashes to more severe conditions such as toxic epidermal necrolysis.

Abstract N°: 1075

Etanercept and topical Silver Nitrate in Stevens Johnson Syndrome: a pilot study.

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Introduction & Objectives: in the management of Stevens Johnson Syndrome (SJS) there is a consensus concerning only the supportive care, topical and systemic therapies are still subject of controversies. Our objective was to assess in a series of SJS patients the efficacy and tolerance of subcutaneous TNF-alpha blocker etanercept associated with topical silver Nitrate in spray, associated with supportive care.

Materials & Methods: in an open multicentric pilot clinical study we included patients with SJS of all etiologies, with no previous treatment. Beside the basic supportive care, patients received topical silver nitrate at 0.5% sprayed on involved skin areas, associated with single a dose of etanercept 50 mg SC.

Results: were included seven adult women (mean age 38 y.o.) with the diagnosis of SJS presenting mucosal and skin lesions covering a mean body surface area (BSA) of 4.5 % (3-5%). The SJD was induced by drugs (n=5) and by infections (n=2). Each patient received supportive care, etanercept was administrated SC in single dose 50 mg at day 0 of admission (D0), topical silver Nitrate 0.5% was sprayed 2X/day on affected skin areas, until the epithelization of the skin areas treated. A significant improvement of skin and mucosal lesions was seen at day 7 (D7) versus D0, with mucosal and skin complete clearance at D14.

The review of the literature on the management of SJS and Toxic Epidermal Necrolysis (TEN) or overlap SJS/TEN did not reveal a consensus on the management of these two severe skin reactions (except the consensus on supportive care).

Conclusion: in a series of seven SJS patients, supportive care associated with topical Silver nitrate spray at 0.5% and etanercept single dose showed a good therapeutic response with a clearance of lesions at week two. Larger series of patients are necessary to better assess the effects of the association etanercept single dose and silver nitrate 0.5% spray.

Abstract N°: 1240

Skin adverse effects during treatment of hepatitis C infection by directly acting antiviral agents: a prospective cohort study

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Introduction & Objectives:

Chronic hepatitis C virus (HCV) infection is a major health problem that infects about 185-million individuals worldwide. In Egypt, HCV and its complications represent a major health problem with high rates of prevalence. In 2015, an Egyptian demographic survey study stated that 6.3% of the population have been tested HCV Antibody (HCV Ab) positive and with infection rate increasing with age reaching 27.6% in individuals among the age group 55–59 years. Among the extrahepatic manifestations, skin manifestations significantly add to the morbidity of the disease. Now, treatment of HCV has become easier and tolerable to all patients with the discovery of directly acting antiviral (DAA) agents which target HCV at specific steps within its life cycle. Cutaneous adverse events can be a potential concern during HCV treatment, and can be mistaken for cutaneous manifestations of HCV infection

Due to the scarcity of evidence on DAAD cutaneous side effests, this study was designed to assess the frequency and clinical pattern of cutaneous adverse events in patients receiving directly acting antiviral agents for treating chronic hepatitis C.

Materials & Methods:

Following sample size calculation using EPI, a target population of 140 HCV positive adult patients were recruited. We excluded patients who have any dermatological manifestation wither as extrahepatic HCV cutaneous involvement or any primary skin disease, patients CHILD C cirrhosis showing moderate to severe liver impairment, those with platelet count <50 000 mm3 as moderate to severe thrombocytopenia is a common finding in decompensated liver disease with portal hypertension, patients with epatocellular carcinoma except for 4 weeks after intervention aiming at a cure with no evidence of activity by dynamic imaging (CT, MRI), extrahepatic malignancy except after 2 years of disease-free interval, pregnant or lactating womrn or inability to use proper contraception, inadequately controlled diabetes mellitus or any other systemic disease with potential dermatological manifestations. Patients were assined to one of 2 treatment protocols: protocol 1 (SOF+DAC regimen) or protocol 2 (SOF+DAC+Ribavirin regimen)

Full dermatological examination including hair, nails and mucous membrane before starting the treatment and every 2 weeks for the whole treatment period and during the follow up for 3 months follwing the end of the treatment.

Results:

Observed cutaneous effects are listed in Table 1

Conclusion: Directly acting antiviral therapy might yield some dermatological adverse effects. Most of them are reversible and don’t require stopping of the treatment. Yet dermatologists and other physicians should be aware of the potential side effects to reassure their patients.

	Protocol 1 (N=105)	***Protocol 2 (N=35)***	***P***
Itching	**Yes**	**NO**	**Yes**
	22 (21%)	83(%79)	4(11.4%)
Rash	2 (1.9%)	103(%98.1)	1 (2.9%)
Hyper pigmentation	2 (1.9%)	103(%98.1)	1 (2.9%)
Mouth dryness	11 (10.5%)	94(%89.5)	3 (8.6%)
Joint stiffness	7 (6.7%)	98(%93.3)	3 (8.6%)
Increasing acne & folliculitis	2 (1.9%)	103(%98.1)	1 (2.9%)
Hair fall	7 (6.7%)	98(%93.3)	6 (17.1%)
Ecchymosis	0	105(%100)	1 (2.9%)

Abstract N°: 1313

Methotrexate-Induced Lymphoproliferative Disorder in a Patient with Granulomatosis with Polyangiitis: A Case Report

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Introduction

Methotrexate (MTX) is widely used for autoimmune conditions but has been associated with methotrexate-induced lymphoproliferative disorder (MTX-LPD), often linked to Epstein-Barr virus (EBV) reactivation. EBV promotes B-cell proliferation in immunosuppressed individuals, though not all cases are EBV-positive. EBV presence increases the likelihood of spontaneous remission upon methotrexate withdrawal. This report describes a patient with MTX-LPD primarily affecting the skin, confirmed via Epstein-Barr encoding region (EBER) in situ hybridization. The patient's lesions regressed significantly after methotrexate withdrawal without additional intervention, emphasizing the need for awareness of EBV-associated MTX-LPD in long-term methotrexate therapy.

Background

The World Health Organization (WHO) classifies MTX-LPD under "Other Iatrogenic Immunodeficiency-Associated Lymphoproliferative Disorders."1 While some cases resolve after discontinuing methotrexate, rare malignancies may persist. MTX-LPD is often of B-cell origin and associated with latent EBV infection.

Case Presentation

A 79-year-old male with granulomatosis with polyangiitis and renal vasculitis was on low-dose methotrexate (10 mg weekly) for nine years, alongside low-dose azathioprine. The patient developed a non-healing plaque on the left ear and an ulcerated plaque on the left calf, both persisting for six months. Systemic examination was unremarkable.

Histopathology revealed a pleomorphic lymphoid infiltrate of small-to-medium-sized atypical cells with vesicular nuclei and amphophilic cytoplasm. Immunohistochemical staining showed CD20, CD79a, and MUM1 positivity, with high proliferation (MIB-1: 80%). The cells were strongly positive for CD20 and EBER, confirming EBV reactivation. EBV serology was also positive.

Based on histological findings and longterm methotrexate use, the patient was diagnosed with methotrexate-induced lymphoproliferative disorder affecting the skin.

Discussion

Prolonged methotrexate use has been linked to lymphoproliferative disorders, including cutaneous presentations2. EBV reactivation contributes to B-cell proliferation, leading to MTX-LPD. After discontinuing methotrexate, the patient's skin lesions significantly regressed within weeks, without requiring chemotherapy or radiation therapy.

The patient also developed biopsy proven squamous cell carcinoma on the scalp and left ear, coinciding with the presentation of MTX-LPD. However, it's uncertain whether this was directly related to methotrexate or due to long-term immunosuppression. Interestingly, the ear lesion clinically regressed after stopping Methotrexate.

Conclusion

MTX-LPD is an important complication of longterm methotrexate therapy, particularly in autoimmune conditions. EBV reactivation plays a key role in disease development and may predict remission upon drug withdrawal. Clinicians should maintain a high index of suspicion for MTX-LPD in patients with atypical skin lesions during methotrexate therapy.

References

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Abstract N°: 1436

Universal Alopecia as a Sequela of Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) Syndrome

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Universal Alopecia as a Sequela of Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) syndrome

Introduction & Objectives:

Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome is characterized by a morbilliform skin eruption, systemic involvement, and multi-organ dysfunction. Although its pathogenesis is not yet fully understood, T-cell activation has been implicated, contributing to various immunological sequelae.

Objective: To describe a case of universal alopecia as a sequela of DRESS and explore its underlying pathophysiological mechanisms.

Materials & Methods:

We present a case of universal alopecia as a sequela of DRESS in a patient who attended our clinic within the past year.

Results:

A 27-year-old male with a history of tonic-clonic seizures due to epilepsy diagnosed in 2023 developed DRESS secondary to carbamazepine in February 2024. He presented with a disseminated dermatosis affecting all four body regions except for the head, abdomen, and thighs. The skin eruption was characterized by a maculopapular exanthem with lamellar scaling and hepatic involvement.

Initial laboratory tests showed AST 87 U/L, ALT 152 U/L, LDH 382 U/L, leukocytes 16,600/mm^3, and eosinophils 4,200/mm^3. A skin biopsy revealed superficial perivascular dermatitis. He was treated with a gradual dose reduction of prednisone, leading to dermatosis remission and normalization of liver function tests.

However, in September 2024, he developed progressive hair loss, initially on the scalp, later extending to the eyebrows and eyelashes.

Examination revealed alopecic involvement of the scalp, eyebrows, eyelashes, mustache, and beard. Additional lesions were observed on the anterior thorax, periumbilical region, pubic area, thighs, and legs, consisting of multiple well-defined alopecic plaques of varying sizes, some with leukotrichia. Dermoscopy findings included yellow dots, black dots, and cadaveric hairs. Nail examination showed pitting.

A diagnosis of universal alopecia was confirmed by scalp biopsy. Autoimmune polyglandular syndrome type 3 and other autoimmune disorders were ruled out. Histopathological findings confirmed the diagnosis, prompting the initiation of systemic calcineurin inhibitors and corticosteroids.

Throughout the disease course, the patient experienced periods of remission and exacerbation. Mycophenolic acid was introduced, and multiple intralesional steroid injections were administered. Currently, the patient has shown scalp hair regrowth but continues to exhibit active disease in the eyebrows, with a Severity of Alopecia Tool (SALT)

score of 1. He remains on systemic calcineurin inhibitor therapy.

Conclusion:

Universal alopecia is characterized by complete hair loss across the entire body, a retrospective cohort reported that 11.5% of patients with DRESS syndrome experienced sequelae, including alopecia in 2% of cases (Figure 1). The pathogenesis involves a T-cell-mediated response that directly targets the hair follicle. Among the sequelae of DRESS, universal alopecia is one of the most common autoimmune complications, attributed to an immune-mediated mechanism.

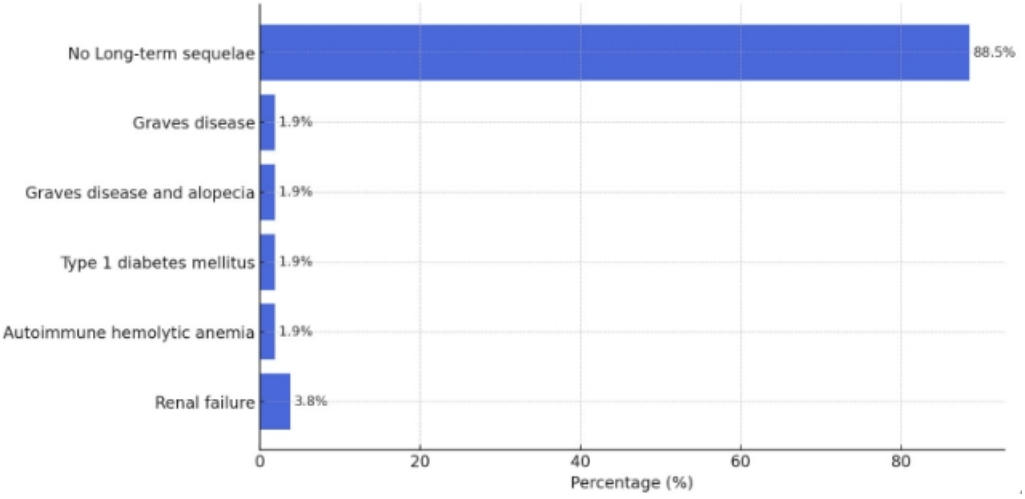

Figure 1. Distribution of Long-term Outcomes in Patients

Abstract N°: 1442

Disseminated cutaneous vasculitis associated with Durvalumab treatment – case report

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Introduction & Objectives:

Durvalumab is an IgG1 monoclonal antibody that has efficacy in many advanced-stage cancers, especially in small-cell lung cancer. The efficacy of durvalumab can be enhanced by chemotherapy. We present a rare clinical case of a cutaneous side effect due to durvalumab treatment, which was not presented in the scientific literature.

Materials & Methods:

We present a clinical case of a female patient aged 61, who is a smoker, weighing 65 kg, diagnosed in 2021 with small cell lung carcinoma exhibiting mediastinal extension, specifically stage III B, cT3N2M. The diagnosis was established following a CT scan and bronchial mucosa biopsy, conducted subsequent to a prior SARS-CoV-2 infection. The patient initiated a five-cycle treatment comprising Durvalumab DT 1500 mg, Carboplatin AUC5 DT 638 mg, and Etoposide DT 178 mg, demonstrating good tolerability and no acute toxicity. Subsequently, the patient continued with monotherapy using Durvalumab, administered once every four weeks, intravenously for a duration of seven months, showing a favorable response. Shortly thereafter, the patient sought consultation at the Dermatology outpatient unit, due to the emergence of multiple round/oval, papulovesicular, and papule- necrotic lesions dispersed across the lower back, abdomen, upper, and lower limbs, with size between 3 mm and 1 cm, with a total of more than 50 lesions

Results:

The lesions were associated with moderate pruritus and pain. Moderate xerosis of skin was also present, particularly on the lower limbs' area. No nail involvement was found. A suppositional diagnosis of cutaneous vasculitis was made. Laboratory results evidenced only a slight increase in inflammation markers and discrète anemia. No internal organ involvement were seen. A cutaneous biopsy revealed cutaneous leukocytoclastic vasculitis presenting a dense perivascular neutrophilic infiltration, fibrinoid necrosis of the vessel walls, leukocytoclastic, and red blood cell extravasation. She underwent treatment with methylprednisolone at a dosage of 0.5 mg/kg for three weeks, and locally topical Clobetasol propionate ointment at 0.1*/u once daily, yielding lesion remission. Importantly, the Durvalumab treatment was not discontinued. After another cycle of Durvalumab, the lesions reappeared. We ruled out alternative reasons for cutaneous vasculitis through clinical and laboratory observations, and so the reappearance of the cutaneous lesions after the treatment with Durvalumab confirmed the etiology. In consultation with the oncologist, we opted to discontinue the Durvalumab treatment and reassess the patient for alternative follow-up therapies.

Conclusion:

As we found in the literature, there was only one case of cutaneous vasculitis de- scribed in association with the chronic use of Durvalumab, more specifically the blue toe syndrome, a more localized clinical form. In our case, we

present the association between Durvalumab and a generalized form of cutaneous vasculitis. To the best of our knowledge, our case is the first published case of disseminated cutaneous vasculitis as a result of Durvalumab treatment. In the searched databases, we did not find any cases presenting this kind of side effect, due to other immune checkpoint inhibitors. Frequent dermatological evaluation is thus mandatory in the case of atypical, persistent/recurrent or severe cutaneous lesions due to these therapies.

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Abstract N°: 1556

Exfoliative Dermatitis as a Manifestation of Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) Secondary to Phenobarbital in an Adolescent Filipino Male

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Introduction & Objectives:

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) is a rare but potentially life-threatening, idiosyncratic adverse drug reaction characterized by delayed onset of cutaneous eruptions, hematologic abnormalities, and internal organ involvement. Cutaneous manifestations, though often nonspecific, are the most common, ranging from maculopapular to exfoliative dermatitis. Phenobarbital, a commonly used aromatic antiepileptic agent, is among the high-risk drugs associated with DRESS. This case aims to describe a Filipino adolescent male who developed exfoliative dermatitis as a manifestation of DRESS syndrome secondary to phenobarbital and to emphasize the importance of early recognition and long-term monitoring of complications.

Materials & Methods:

This is a single-patient case report involving a 17-year-old Filipino male who was prescribed phenobarbital 60 mg/day after a single episode of generalized tonic-clonic seizure. A detailed review of clinical presentation, laboratory findings, histopathologic features, management, and follow-up data was performed. Diagnostic confirmation was based on clinical features and RegiSCAR criteria. Histopathology was obtained via skin punch biopsy. Relevant literature was reviewed to contextualize this case.

Results:

Four weeks after starting phenobarbital, the patient developed erythematous papules and plaques that progressed into generalized exfoliative dermatitis, fever, lymphadenopathy, and difficulty in ambulation. Physical examination revealed generalized erythematous plaques with fissuring, erosions, and a positive Nikolsky sign. Laboratory findings showed leukocytosis with absolute eosinophilia (9.57 × 10^9/L), elevated liver enzymes, and hypoalbuminemia. Chest radiography showed right lower lung opacities. A skin punch biopsy revealed lichenoid dermatitis. RegiSCAR scoring yielded 6 points, confirming a definite case of DRESS. A drug chart (Fig. 1) supported the temporal association with phenobarbital. Treatment with methylprednisolone (0.64 mg/kg/day) and topical corticosteroids led to significant improvement. Follow-up showed residual desquamation and onychomadesis. At two years, no recurrence or autoimmune sequelae was observed.

Conclusion:

This case illustrates the exfoliative cutaneous presentation of DRESS syndrome triggered by phenobarbital in an adolescent. Early recognition, prompt withdrawal of the offending drug, and initiation of systemic corticosteroids were critical in achieving favorable outcomes. Given the waxing and waning nature of DRESS and its risk for delayed autoimmune sequelae, long-term follow-up every 6–12 months for up to four years is strongly recommended

Abstract N°: 1586

A rare case of multiple endocrine abnormalities in Drug Reaction with Eosinophilia and Systemic Symptoms syndrome

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Introduction & Objectives:

Drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome, is recognised as one of the severe cutaneous adverse reactions and can be potentially life-threatening due to its acute complications and its long-term sequela after initial resolution.

A growing number of reports have documented the occurrence of newly developed diseases weeks or months after DRESS resolution, especially autoimmune diseases.

Here is a rare case of co-occurrence of type 1 diabetes mellitus, thyroiditis and sialadenitis in a woman with DRESS syndrome.

Materials & Methods:

A 50 yo female with a history of carbamazepine intake 37 days back, was admitted with complaints of fever, extreme fatigue, pruritic maculopapular erythematous rash, swelling of the whole body, including palms, soles and facial edema that began one week prior. She was subsequently diagnosed as a DRESS according to the Regi-SCAR criteria.

Results:

The laboratory tests at admission showed an elevated HbA1C:11,1% with a 4.3 g/dl of glycemia and elevated liver enzyme.

On the 6th day of hospitalization, the patient presented a fever with bilateral cervical swelling which was corresponding on the ultrasound to enlarged cervical lymph nodes with swelling of the parotid and sub-mandibular glands.

On the 7th day, the patient presented a tachycardia and palpitations, the level of TSH was low at 0.07 with elevated T3 and T4 which were all normal at admission, the ultrasound showed an enlarged thyroid gland with doppler hypervascularity.

The patient was started on a potent local corticosteroid preparation along with antihistamines, as well as insulin therapy and synthetic thyroid hormones with a slow and gradual improvement.

Conclusion:

Patients with a suspicion of DRESS syndrome should undergo extensive evaluation not only to establish diagnosis and assessment of severity but also for the short-term and long-term monitoring for signs or symptoms suggestive of autoimmune diseases which can be fatal.

Abstract N°: 1696

Rosacea-like folliculitis associated with tralokinumab therapy: a diagnostic challenge

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Introduction & Objectives:

Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by pruritus, eczematous lesions and lichenification. During the last years new drugs, including biological agents, have been launched and it has changed completely the therapeutic scenario of the disease

Materials & Methods:

Case report and literature review

Results:

We present a 35-years-old female with medical history of AD who experienced severe AD outbreaks since 2010.

At the beginning, she was treated with topical corticosteroids and calcineurin inhibitors, without any significant improvement. Then, azatioprine was prescribed at 50mg/day with good response. It was discontinued due to the patient´s gestational desire. Eight months later, the patient suffered an important AD outbreak. Cyclosporine was prescribed at 300 mg/day and it led to initial improvement, but the patient was pregnant and suffered from severe edemas on extremities and on the face, so the drug was stopped.

One year later, the patient worsened, reaching an Investigator Global Assessment (IGA) score of 3, an Eczema Area Severity Index (EASI) of 25 and a Body Surface Affected of 30%. Due to the worsening of her dermatitis, tralokinumab was prescribed. After that, EASI 0 was achieved, with a significant decrease in pruritus (IGA 0). Four weeks after the beginning of tralokinumab, the patient started with a papulopustular eruption on the face, located on the mandibular and chin areas. The lesions appeared as papules that tended to form yellowish crusts. She was treated with doxycycline 100mg per day and PCR samples were taken for varicella zoster virus, herpes simplex virus 1 and 2, as well as swabs for bacterial and fungal cultures. All samples were negative, and a complete remission was achieved after one month with oral doxycycline.

Finally, tralokinumab was discontinued due to a new pregnancy.

Conclusion:

According to the scientific evidence, main factors described in the pathogenesis of AD are dysfunction of the epidermal barrier and dysregulation of the immune system. There is an imbalance between T helper 1 (Th1) and Th2 lymphocytes, with predominance of Th2 expression1.

Rosacea is a chronic inflammatory dermatosis mainly affecting the face. Multiple factors have been involved in its pathogenesis, including environmental changes, immune dysregulation and increased density of demodex 2, 3.

Tralokinumab is a fully human IgG4 monoclonal antibody that blocks the binding of IL-13 to two receptors, avoiding the activation of the signaling cascade involved in AD1, 4.

Among its most frequent adverse events are headaches, conjunctivitis, injection site reactions, upper respiratory tract infections4 and “red face”5, with no cases of rosacea-like folliculitis reported so far. However, it has been previously described after the use of dupilumab2,3. These patients presented with a new-onset rosacea-like folliculitis characterized by erythema, flushes and papulopustules in the centrofacial area2,3. Moreover, it was demostrated an increased number of demodex per follicle3. Some authors have postulated that the immune dysregulation with alteration of Th1/Th2 balance through Th2 blockade could explain the development of this rosacea-like eruption2.

On the whole, the exact mechanism leading to these reactions is not fully understand, and further investigations will be necessary to elucidate the role of Th2 and IL-13 inhibition in the development of rosacea-like folliculitis.

Abstract N°: 1702

Drug-induced linear igA bullous dermatosis: A systematic review of the literature

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Introduction & Objectives:

Linear IgA bullous dermatosis (LABD) is a rare autoimmune blistering disorder marked by linear IgA deposition along the basement membrane. LABD can be idiopathic or drug-induced. Evidence on differences in presentation and management between the two forms is limited. This systematic review aims to identify key differences in clinical features and treatment approaches.

Materials & Methods:

A systematic review of published LABD cases was conducted.

Results:

1,945 cases were included: 487 (25.0%) drug-induced, 1,458 (75.0%) idiopathic. Vancomycin and beta-lactams were the most frequent triggers. Drug-induced LABD presented ~12 days post-exposure and showed more erythematous plaques (27.5% vs. 5.1%), Nikolsky's sign (4.7% vs. 0%), and higher mucosal involvement. Dapsone, corticosteroids, and drug withdrawal were commonly used treatments.

Conclusion:

Drug-induced LABD shows distinct features and strong links to specific drugs. Drug cessation is key. More research is needed on underlying mechanisms.

Abstract N°: 1724

Mucositis and exanthema marker of marrow toxicity due to methotrexate intoxication.Case Report.

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Introduction & Objectives:

Methotrexate is an antimetabolite analogous to folic acid and its mechanism of action is to inhibit the de novo synthesis of purines and pyrimidines required for DNA and RNA synthesis. It acts in the S phase of cell division and this is the reason for its antiproliferative action and its adverse effects.

We present the case of methotrexate intoxication, a drug used in several areas with a good safety profile but with serious adverse effects, highlighting its effects on the skin as a marker of spinal cord damage, with the need for vigilance during the application of the treatment to avoid complications.

Case Report

A 42-year-old woman with history: arterial hypertension under treatment; chronic kidney disease KDIGO 5 of 1 year of diagnosis without therapy; rheumatoid arthritis of 1 month of diagnosis under treatment with metrotexate 2.5 mg Saturday and Sunday with last administration every 24 hours for 4 consecutive days.

She presented with abdominal pain, fever, mucositis and dermatosis of 3 days of evolution, assessed by a physician indicating treatment with 3rd generation cephalosporins and corticosteroid without clinical improvement, presenting neurological deterioration. Physical examination revealed disseminated dermatosis on face, neck, thorax and asymmetric extremities with macular morphology, erosions and necrotic crusts of approximately 5 days of evolution accompanied by uremic frosting, with presence of mucositis and bleeding.

Laboratories with pancytopenia (severe anemia, profound neutropenia) with suspicion of bone marrow aplasia treated with transfusion of blood products, granulocyte colony stimulating factor, antimicrobial and antifungal agents without improvement, evaluated by internal medicine with a diagnosis of methotrexate pharmacoderma and started management with folic acid 15 mg IV every 6 hours. She presented severe myelosuppression with death on the 2nd day.

Results:

There are facilitating factors of cytotoxicity: overdosage due to dosage error, infection, folic acid deficiency, hypoalbuminemia, renal hypofunction, recent initiation of methotrexate, dose increase and/or reintroduction after its interruption. It affects hematopoietic proliferating cells, medullary cells, gastrointestinal epithelium and keratinocytes. Biopsy is rarely required. Histology shows edematocytic keratinocytes with pale cytoplasm and nuclei, vacuolated or dyskeratotic cells and even epidermal necrosis. The treatment of choice for methotrexate poisoning is folinic acid, analogous metabolite at doses of 10-25 mg/m2 intravenously every 6 hours.

Conclusion

The importance of this pharmacoderma lies in the proper physical examination of the skin, being a marker of spinal cord damage. Early recognition and proper management

are crucial to improve patient outcomes and avoid complications.

Abstract N°: 1816

Fixed Pigmentary Erythema triggered by dipyrone in a Pediatric Patient

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Introduction & Objectives:

Fixed drug erythema (FDE) is an adverse drug reaction characterized by macular or erythematous-grayish plaques. In cases of reexposure to the causative agent, the lesions reappear at the same location, justifying the term "fixed" due to the specific reactivity in a particular skin area.

This article reports a rare pediatric clinical case of FDE associated with dipyrone, a widely used medication in clinical practice in Latin America, in a patient who is seropositive for herpes simplex. It also discusses the possible etiopathogenic association of this condition with viral infection.

Materials & Methods:

A comprehensive review of the literature was carried out for this case, noting that a search for "Fixed Pigmented Erythema" AND "Dipyrone" yielded only 14 published articles in indexed medical database.

Results:

A 12-year-old male patient presented a purplish macule that evolved into a grayish color at the left corner of his lip over the past three months. Serological tests confirmed positivity for herpes simplex.

On physical examination, a grayish macule was also observed on the left forearm. After investigating the lesion, the mother denied any history of vesicular-bullous lesions in the area and reported recurrent use of dipyrone to treat migraine episodes. FDE was suspected, and a biopsy of the lesion was performed, revealing lichenoid dermatitis with evident pigment incontinence, confirming the diagnosis.. The dipyrone contact test was positive. Therefore, it was recommended to permanently discontinue the medication.

Conclusion:

FDE accounts for 16-21% of drug-induced skin manifestations and 14-22% of drug-induced skin reactions in children. The most frequently associated drugs include nonsteroidal anti-inflammatory drugs.

The pathophysiology of FDE involves a delayed cutaneous hypersensitivity reaction. Studies suggest that after viral infections, such as herpes simplex virus (HSV), effector memory T cells retained in specific areas of the skin may cross-react with drug antigens, resulting in the characteristic FDE lesions. In fact, most patients with FDE are seropositive for HSV, even without a prior history of clinical herpetic lesions.

FDE manifests as well-demarcated cutaneous and/or mucosal lesions, initially erythematous-violet, which evolve into a persistent grayish color. In some cases, the lesions may be bullous, associated with itching and burning sensations.

The diagnosis of FDE is clinical and may be supplemented by biopsy, histopathology, contact testing, and oral provocation testing. A safer and more practical alternative is the contact test, which is positive in approximately 43% of patients.

FDE is generally a self-limiting condition. Treatment consists of identifying and permanently discontinuing the causative agent, along with symptomatic management.

This case highlights the importance of detailed anamnesis and diagnostic tests in identifying the causative agent and contributes to understanding the condition, emphasizing the possible relationship between FDE and herpes simplex, suggesting an immunological role in the pathogenesis of the disease.

Abstract N°: 2056

Treating apalutamide-induced toxic epidermal necrolysis with adalimumab and baricitinib in a patient with prostate cancer

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Treating apalutamide-induced toxic epidermal necrolysis with adalimumab and baricitinib in a patient with prostate cancer

Introduction & Objectives:

Apalutamide, a next-generation androgen receptor antagonist, is rarely associated with severe cutaneous adverse reactions. Toxic epidermal necrolysis (TEN) as a life-threatening complication, likely triggered by delayed T-cell-mediated hypersensitivity. Early recognition and aggressive immunomodulation were critical. The combination of IVIG, corticosteroids, and targeted biologics (adalimumab/baricitinib) aligns with emerging evidence supporting dual inhibition of TNF-α and JAK-STAT pathways in refractory TEN. Here, we present a case of TEN induced by apalutamide.

Case report

A 75-year-old male presented with a two-weeks history of generalized diffuse erythematous blisters accompanied by fever (peak 40.1°C). He had initiated treatment with apalutamide 240 mg daily for prostate cancer. Physical examination revealed generalized dusky erythematous patches with diffuse distribution, demonstrating extensive denudation and erosions localized to the cephalocervical region, extremities, and dorsal aspects. Scattered vesicles and bullae were present with marked tenderness upon palpation. Nikolsky's sign was elicited as positive. Notable edema involved both the facial region and extremities, accompanied by mucosal involvement. A series of diagnostic investigations including blood and sputum next-generation sequencing blood cultures, total prostate-specific antigen (TPSA), free-PSA and chest CT were systematically conducted to exclude malignant and infectious etiologies as potential causes of the persistent high-grade fever. A diagnosis of TEN was established. A multidisciplinary therapeutic strategy was instituted comprising high-dose intravenous immunoglobulin (20 g/day) for immunomodulation, pulse methylprednisolone (80 mg IV twice daily) as the initial anti-inflammatory intervention, subcutaneous adalimumab (80 mg weekly) for targeted TNF-α blockade, and oral baricitinib (2 mg twice daily) to inhibit JAK1/2-mediated signaling pathways. Concurrent antimicrobial prophylaxis was achieved through sterile application of topical 1% sulfadiazine silver cream to denuded skin areas on alternating days. Notably, cutaneous manifestations demonstrated rapid stabilization within 10 days, permitting cessation of JAK inhibition therapy (baricitinib) and initiation of a systemic corticosteroid taper protocol (methylprednisolone 80mg BID→40 mg BID→40 mg QD→30 mg QD→20 mg QD). Maintenance therapy with TNF-α blockade (adalimumab 40 mg biweekly) was sustained to prevent disease recrudescence. Subsequent clinical surveillance revealed complete re-epithelialization of denuded surfaces by Day 30, achieving >95% body surface area recovery without adverse drug reactions.

Conclusion:

This case demonstrates TNF-α inhibitor/JAKi combination therapy effectively manages refractory TEN in cancer patients, through synergistic immunomodulation and neutralizing cytokine storms. The regimen achieved rapid re-epithelialization with reduced steroid dependence, addressing malignancy-associated immune dysregulation. Large-scale clinical studies are still needed to explore the clinical potential of TNF-α inhibitor and JAKi in cancer

patients with TEN.

Abstract N°: 2136

Facial redness in a dupilumab-treated population: characterization and management

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Introduction & Objectives:

Dupilumab has been approved for the treatment of moderate to severe atopic dermatitis (AD) in patients with or over 6 months of life, demonstrating effectiveness in all anatomic regions (including face and neck). Post-marketing surveillance data identified paradoxical facial erythema (PFE) as an uncommon, but troublesome side effect of dupilumab in patients with AD. From our experience, this emergent and potentially immune-related side effect remains yet elusive and in need of further characterization.

Materials & Methods:

An observational, retrospective study was carried out to identify and characterize PFE in our dupilumab-exposed AD cohort. We also aimed to identify the implemented therapeutic strategies and their effectiveness. The ones who reported new or worsening facial and neck erythema after starting treatment were selected.

Results:

In a population of 283 dupilumab-exposed AD patients, 26 (9%) were diagnosed with PFE. The median onset time of PFE was at 8 weeks (interquartile range 17) of treatment. Twenty-one (81%) had facial eczema before treatment. Most had a complete (62%) or partial (31%) AD clearance from the neck down. The periorbital, malar and neck regions were the most affected. Approximately 77% were symptomatic - itch (62%) and burning (58%) were the most common symptoms. More than half had partial or near-complete improvement with medical therapy, including topical calcineurin inhibitors (TCI), topical and systemic corticosteroids (SCS, TCS), doxycycline (2/2) or itraconazole (2/6); with phototherapy (2/4); by avoiding allergens identified on patch tests (2/3); and after spacing dupilumab (5/6; to every 4-8 weeks). PFE led to dupilumab withdrawal in 11 (42%) cases, of which 8 (31%) initiated other drugs (baricitinib, upadacitinib, tralokinumab and lebrikizumab) and 3 (12%) stopped treatments altogether. Of these, 7 have reported improvement so far, after an average of 6.5 ± 3.8 weeks (minimum 4 and maximum 12).

Conclusion:

Although uncommon, dupilumab-related PFE remains a hard-to-treat side effect, as 42% of patients who developed it discontinued the drug. Prolonging dupilumab administrations seems to oftenly work in mitigating PFE, allowing to continue the biologic without significantly compromising the response from the neck down. When this is not possible or is ineffective, switching to other drugs, such as other anti IL-13 inhibitors or iJAK may be considered.

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Abstract N°: 2260

Long-Term Safety of Ruxolitinib Cream in Pediatric and Adult Patients: an Analysis of 7 Phase 3 Clinical Trials in Atopic Dermatitis and Nonsegmental Vitiligo

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Introduction & Objectives: Janus kinase (JAK) inhibitors are effective therapeutic agents for dermatologic conditions, such as atopic dermatitis (AD) and vitiligo; class warnings are present in US and Canadian labels for serious infections, malignancies, thromboembolic events, major adverse cardiac events (MACE), and mortality, originating from safety concerns with a systemic JAK inhibitor in patients with rheumatoid arthritis. The present analysis investigates the incidence of these adverse events in phase 3 clinical trials of topical ruxolitinib in patients with AD or nonsegmental vitiligo.

Materials & Methods: Exposure-adjusted incidence rates (EAIRs) among patients who applied twice-daily 1.5% ruxolitinib cream were calculated using data from 4 clinical studies in mild to moderate AD (Investigator's Global Assessment score of 2/3 and 3%−20% affected body surface area [BSA; excluding scalp] for up to 1 year in patients aged ≥2 years [applied as needed after Week 8]) and 3 studies in vitiligo (3%−10% affected BSA for up to 2 years in patients aged ≥12 years). EAIRs were calculated as the number of patients experiencing the event per 100 person-years (PY) of exposure.

Results: In patients with AD aged 2−11 years who applied 1.5% ruxolitinib cream (n=154; 112.88 PY), the EAIR (95% CI) for serious infections was 0.89 (0.02−4.94) patients/100 PY; no other events of interest occurred in this population. In patients with AD aged ≥12 years who applied 1.5% ruxolitinib cream (n=701; 545.65 PY), EAIRs (patients/100 PY [95% CI]) were 0.92 (0.30−2.14) for serious infections, 0.37 (0.04−1.32) for nonmelanoma skin cancer (NMSC), 0 (0−0.68) for other malignancies, 0.18 (0−1.02) for MACE, and 0.37 (0.04−1.32) for thromboembolic events. In patients with vitiligo aged ≥12 years who applied 1.5% ruxolitinib cream (n=637; 867.90 PY), EAIRs (patients/100 PY [95% CI]) were 0.46 (0.13−1.18) for serious infections, 0.12 (0−0.64) for NMSC, 0.35 (0.07−1.01) for other malignancies, and 0.23 (0.03−0.83) for thromboembolic events. No fatalities occurred in any clinical study.

Conclusion: EAIRs of serious infections, malignancies, thromboembolic events, and MACE were low among

patients with AD or nonsegmental vitiligo who applied 1.5% ruxolitinib cream twice daily; no fatalities occurred.

Abstract N°: 2350

Dramatic Interaction: Drug-Induced Toxic Epidermal Necrolysis in Measles in a Child

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Introduction:

Lyell's syndrome, or toxic epidermal necrolysis (TEN), is a severe and rare drug-induced dermal and mucosal condition, with an incidence of 0.1% in adults and even less in pediatrics. It requires urgent and specific management to reduce morbidity and mortality. This report presents the case of a 3-year-old girl who developed TEN after a medication treatment in the context of confirmed measles.

Observation:

A 3-year-old girl was referred to the pediatric emergency department for a skin eruption that developed 72 hours after the administration of ibuprofen and paracetamol, which had been prescribed in the context of measles, confirmed by PCR.

At admission, clinical examination revealed an asthenic and febrile patient (temperature: 40 °C), presenting with a widespread macular exanthema and skin detachment estimated at 90% of body surface area — including 50% of already detached skin and 40% at risk of detachment. Nikolsky's sign was positive, and the skin appeared as "wet laundry".

Additionally, the patient exhibited erosive cheilitis, conjunctivitis complicated by palpebral synechiae, and hypovolemic shock. Pediatric intensive care management was initiated immediately, including intubation with sedation, fluid resuscitation according to Karjaval's formula, and daily wound dressings. Initial bacteriological samples were negative.

Initial laboratory workup revealed thrombocytopenia, hyponatremia, and hypokalemia. A skin biopsy showed keratinocyte necrosis with intraepidermal cleavage, leading to the diagnosis of toxic epidermal necrolysis.

discussion:

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are severe drug-induced conditions, with less than 10% skin detachment for SJS and over 30% for Lyell's syndrome. Mortality rates range from 5 to 30%, often due to electrolyte imbalances and infectious complications. Commonly involved drugs in children include antibiotics, anticonvulsants, and nonsteroidal anti-inflammatory drugs (NSAIDs) like ibuprofen. Although paracetamol is rarely involved, cases of TEN have been reported, and in 2013, the FDA issued a warning regarding the risk of SJS and TEN related to paracetamol, reporting 91 cases in the United States between 1969 and 2012. However, this warning was not supported by data from the French national pharmacovigilance network. Both SJS and TEN are rare (1 case/million), but severe, with mortality rates between 5% and 30%. The risk is increased with certain NSAIDs such as oxicams, but also with ibuprofen. Specific HLA phenotypes are implicated, although it is not possible to identify at-risk individuals. Advocacy groups call for better labeling of over-the-counter NSAIDs, warning about the danger of continuing treatment if skin symptoms arise. To date, such warnings are not included on packaging. Treatment involves discontinuing the medication, rehydration, and infection prevention. The use of intravenous immunoglobulins and corticosteroids remains controversial, with no proven benefit on mortality.

Conclusion:

It is essential to inform clinicians, particularly pediatricians, about the risk of severe hypersensitivity reactions linked to commonly prescribed drugs like paracetamol and ibuprofen. Re-exposure to the causative medication carries a risk of potentially fatal complications.

Abstract N°: 2395

Multiple drug hypersensitivity

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Introduction & Objectives: Multiple drug hypersensitivity is a rare syndrome characterized by confirmed hypersensitivity reactions to at least two structurally distinct medications. Here, we report a new case.

Materials & Methods: A 66-year-old man was hospitalized for a generalized pruritic rash started a week ago. The patient had undergone a surgical evacuation of a chronic subdural hematoma 7 weeks earlier and had been treated with phenobarbital since then. On clinical examination, the patient presented an erythroderma with facial edema and a fever up to 39 °C. Mucous membranes appeared normal, and there were no palpable lymph nodes. Laboratory tests showed leukocytosis (13,600/µL) with eosinophilia (1560/µL), elevated ALT levels (2.17 times the normal value), and elevated CRP (70 mg). Based on these findings, a diagnosis of Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) was made. Corticosteroid therapy at a dose of 1 mg/kg/day was initiated, and phenobarbital was replaced with sodium valproate. A week later, the patient developed bullous skin detachment on the elbows, face, back, buttocks, and chest, with a 'wet cloth' appearance and a positive Nikolsky sign, involving approximately 30% of the body surface area. He also developed blisters and erosions on the oral and genital mucosa. The skin biopsy confirmed the diagnosis of toxic epidermal necrolysis, showing a blistering lesion with dermoepidermal splitting and nearly complete keratinocyte necrosis.

Results: Despite the short interval of one week between the two eruptions, the different clinical and paraclinical presentations suggest two distinct drug eruptions, ruling out a flare-up of DRESS. Our patient experienced two types of drug eruptions, each induced by medications from different pharmacological classes. The causality of these drugs was established using the French causality assessment method. This likely represents a case of sequential multiple drug hypersensitivity, distinguished from cross-reactive reactions to structurally similar drugs and flare-ups (mini-DRESS or flare-up), which are transient reactions to a second drug, usually tolerated once the initial reaction resolves. This syndrome is rare, and its mechanism remains poorly understood, but it is believed to be triggered by severe T lymphocyte-mediated reactions (primarily severe exanthems and DRESS), which seem to have a lasting effect on the patient's immune system. The drugs involved are the same as those implicated in DRESS. Clinical presentations vary widely, and there are currently no validated diagnostic criteria, but the culprit drugs can be identified through positive skin tests or in vitro testing.

Conclusion: Multiple drug hypersensitivity is a rare entity that should be considered in patients who experience two or more episodes of drug eruptions involving medications from distinct pharmacological classes.

Abstract N°: 2426

Drug-induced Bullous Pemphigoid Following Ixekizumab Therapy: A Case Report

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Introduction & Objectives:

Bullous pemphigoid is a chronic autoimmune subepidermal blistering disorder that primarily affects the elderly population. It may be triggered by various external factors, including pharmacologic agents such as biologic therapies. Although ixekizumab, an anti-IL-17A monoclonal antibody, is commonly used in the treatment of psoriasis vulgaris, its association with the onset of bullous pemphigoid remains rare. This case report aims to describe the development of bullous pemphigoid in a patient treated with ixekizumab for psoriasis vulgaris.

Results:

We present the case of a 78-year-old female with a history of histologically confirmed psoriasis vulgaris and previously treated middle rectal adenocarcinoma. She developed widespread erythematous plaques, excoriations, hemorrhagic crusts, and tense blisters involving the limbs, intergluteal region, and posterior trunk. The patient had previously received topical corticosteroids, ciclosporin, and phototherapy with insufficient clinical control. Due to persistent disease activity, ixekizumab was initiated following thorough evaluation by dermatology, oncology, and pulmonology specialists.

Histopathological examination revealed a subepidermal blister accompanied by a dermal infiltrate rich in eosinophils, lymphocytes, and plasma cells. Direct immunofluorescence of perilesional skin showed linear deposits of C3 and IgG along the basement membrane zone. Serological testing confirmed the diagnosis with positive anti-BP180 and anti-BP230 antibodies.

Systemic corticosteroid therapy was introduced after the failure of topical management, alongside gastric protection and potassium supplementation. The patient exhibited progressive clinical improvement. However, following the reintroduction of ixekizumab for psoriasis control, a relapse of bullous pemphigoid was observed, which was subsequently managed with topical corticosteroids.

Conclusion:

This case highlights a rare but clinically significant adverse effect of ixekizumab in the form of drug-induced bullous pemphigoid. Although IL-17 inhibitors may be considered therapeutic options in certain autoimmune bullous dermatoses, their potential to paradoxically induce bullous pemphigoid must be recognized. Close clinical surveillance is warranted, especially in elderly patients with multiple comorbidities, to ensure prompt identification and appropriate management of such adverse events.

Abstract N°: 2625

A case of Pemphigus vulgaris presenting as Toxic epidermal necrolisis

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A case of Pemphigus vulgaris presenting as Toxic epidermal necrolysis

Itroduction:

Toxic Epidermal Necrolysis (TEN) is a severe form of adverse drug reaction and life-threatening condition that is often manifested with widespread skin and mucous membranes erosions.

TEN is a dermatological emergency, therefore acute measures to identify the culprit drug should be rapidly taken.

The diagnosis of TEN is primarily clinical, based on typical skin findings. Histopathological examination is often needed to confirm the diagnosis and help to differentiate from other similar skin conditions mimicking TEN such as Pemphigus vulgaris, Staphylococcal Scalded Skin Syndrome (SSSS), Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS), and Acute Graft Versus Host Disease (GVHD).

Case report:

A 49-year-old male healthy, medicated occasionally with Omeprazole due to stomach issues, presented to the emergency department due to rapidly progressive and intensely painful extensive skin erosions located on the scalp, thorax, and extremities. Patient was acutely transferred at the burn unit center.

He reported pruritic skin rash, 3-4 weeks prior to skin exfoliation. These symptoms were self-attributed to bed bugs infestation and were self-medicated with a medicine that was obtained from patients' native country, which was later confirmed as Amoxicillin.

Dermatological examination revealed targetoid lesions on the lower extremities with central necrosis, extensive denuded skin covering aproximately 50% of the body surface area, as well as diverse mucous membrane involvement such as scrotum, penis, oral cavity, nasal cavity, and eyes. Nikolsky's sign was positive.

Laboratory findings upon admission showed: WBC 11.4, bicarbonate 27, CRP 155, GFR > 90, albumin 18. Lesional skin biopsies and one perilesional skin biopsy for immunofluorescence were obtained, and circulating autoantibodies were also assessed to rule out bullous diseases.

The patient was initiated on high-dose methylprednisolone therapy (500 mg/day for three days) until skin biopsy and laboratory test were available. During hospitalization, the patient rapidly developed new vesicles, bullae, and extensive erosions in the back and extremities, leading to sedation due to severe pain.

On the third day of methylprednisolone treatment, we received a preliminary pathology report favoring diagnosis of pemphigus vulgaris, although serological results for circulating autoantibodies were still pending. Treatment was continued with oral prednisolone (2 mg/kg) and topical application of high-potency corticosteroids to the erosive lesions. Subsequent serological testing confirmed the presence of circulating autoantibodies. Rituximab infusions (1000 mg) were administered with two weeks apart. Rapidly clinical improvement was seen, allowing for gradual tapering of prednisolone.

Discussion:

TEN is a dermatological emergency, therefore immediate evaluation of the patient should be done. Differential diagnosis sometimes can be challenging, as several skin conditions may mimic TEN, as demonstrated in our case report where extensive erosions characteristic of pemphigus vulgaris were initially misinterpreted as TEN. Thus, an appropriate evaluation of the patient should be done in order to ensure accurate diagnosis but also to prevent treatment delay.

Abstract N°: 2713

Onychomadesis and palmoplantar erythema secondary to talquetamab in a patient with refractory multiple myeloma: a case report.

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Introduction & Objectives: Talquetamab is a humanized bispecific antibody that simultaneously targets the CD3 receptor on T cells and the G protein-coupled receptor class C group 5 member D (GPRC5D), expressed in malignant plasma cells and in hard keratinized tissues. In clinical trials and real-world studies, the most frequent adverse events include cytokine release syndrome, an increased risk of infections, as well as mucocutaneous and nail toxicity.

Materials & Methods: presentation of a clinical case and review of the literature.

Case Report: we present the case of a 64-year-old woman diagnosed with lambda light chain multiple myeloma (MM), refractory to four prior lines of treatment. Talquetamab was initiated as a fifth-line therapy, achieving a partial response both serologically and radiologically after two cycles. However, the patient developed grade I cytokine release syndrome (CRS), accompanied by palmoplantar erythema, onychomadesis, onycholysis, and onychodystrophy. Nail involvement was symmetrical and predominantly proximal, with progressive loss of several nails and associated dystrophic changes, leading to significant functional limitation. Due to this cutaneous-nail toxicity attributed to the treatment, Talquetamab was discontinued and a sixth-line therapy was initiated. High-potency topical corticosteroids and a short course of oral corticosteroids were administered, with progressive clinical improvement, although dystrophic nail changes and Beau's lines persisted at six-month follow-up.

Conclusion: this case illustrates a frequent cutaneous-nail adverse effect associated with Talquetamab. Although the pathogenesis of onychomadesis induced by this drug has not been fully elucidated, it is proposed that the expression of GPRC5D in nail matrix cells makes them susceptible to T-cell-mediated cytotoxicity. This interaction, facilitated by Talquetamab, may transiently impair the proliferative function of the matrix, leading to Beau's lines or, in more severe cases, to onychomadesis. The concomitant presence of palmoplantar erythema supports the concept of targeted toxicity toward keratinized epithelial structures.

Abstract N°: 2878

Acute Lymphoblastic Leukemia Following Etanercept Therapy in a Patient with Psoriasis

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Introduction & Objectives: Tumor necrosis factor-alpha (TNF-α) inhibitors represent a pivotal advancement in the treatment of autoimmune diseases such as rheumatoid arthritis, ankylosing spondylitis, psoriasis, and psoriatic arthritis. Etanercept, a soluble TNF receptor fusion protein, is an effective option for patients with chronic moderate-to-severe plaque psoriasis who are unresponsive to conventional therapies. Although generally well-tolerated, TNF-α inhibitors have been associated with serious adverse effects, including infections, lupus-like syndromes, congestive heart failure, demyelinating diseases, and malignancies. In recent years, there have been increasing reports of hematologic malignancies, such as lymphoma, acute myeloid leukemia (AML), and acute lymphoblastic leukemia (ALL), following anti-TNF-α therapy. Here, we report a case of ALL in a patient with psoriasis treated with etanercept.

Materials & Methods: A 48-year-old male with an eight-year history of weekly etanercept therapy (50 mg/week) for plaque psoriasis presented to the emergency department in November 2024 with abdominal and chest pain persisting for 1.5 months. Laboratory tests revealed a white blood cell (WBC) count of 28.83 × 10^9/L and an absolute lymphocyte count of 17.11 × 10^9/L. Suspecting acute leukemia, he was admitted to internal medicine. Peripheral blood smear showed numerous small, atypical lymphocytes. Flow cytometry confirmed B-cell ALL, with CD10 and CD19 positivity. Etanercept was discontinued, and the patient was referred to oncology for chemotherapy initiation.

Results: Acute leukemia following anti-TNF-α therapy is less commonly reported than lymphoma. The first case of Philadelphia chromosome-positive ALL during TNF-α inhibitor treatment was reported in 2003, followed by a second case in 2010 involving adalimumab. Over 40 leukemia cases have since been associated with etanercept. The Uppsala Monitoring Centre (UMC) reported 121 leukemia cases linked to TNF inhibitors, 39 of which involved etanercept; 37 were classified as single suspect drug cases. While the leukemogenic mechanism remains unclear, decreased TNF-α levels may promote carcinogenesis via direct effects or chemokine dysregulation. Interestingly, TNF-α inhibitors are also being explored in hematologic malignancies at risk for blast transformation.

Conclusion: The potential link between anti-TNF-α therapy and malignancy remains controversial. Variability in reported outcomes may reflect factors such as disease duration, family history, concomitant medications, and individual genetic or environmental risks. This case highlights the need for careful hematologic evaluation prior to initiating TNF-α inhibitors. A complete blood count (CBC) should be obtained at baseline, with ongoing monitoring throughout treatment. If abnormalities suggest bone marrow involvement, further investigation with bone marrow aspiration or biopsy is warranted to exclude malignancy.

Abstract N°: 3008

Hydroxychloroquine in the treatment of lichen planus-like cutaneous immune-related adverse events

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Introduction & Objectives:

In patients with advanced cancer, the use of immune checkpoint inhibitors (ICIs) has revolutionized cancer therapy. By allowing for T-cell activation, the body's own immune system is able to fight cancer. Common adverse events of ICI use are immune cell-mediated, and can include a variety of cutaneous immune-related adverse events (cirAEs), including lichen planus-like (LPL) eruptions. CirAEs are associated with a good prognosis, but can have significant effect on quality-of-life, and may commonly result in discontinuation of life-saving ICIs. Goals of the oncodermatologist include managing any cutaneous toxicities of cancer therapy so that patients can remain on cancer therapy. Hydroxychlorquine is a systemic therapy commonly used for lichenoid dermatitides, but is not included in guidelines for management of cirAEs. The objective of this study was to characterize our patient cohort and perform a systematic review of those who have developed LPL cirAEs during ICI and who were managed with hydroxychloroquine.

Materials & Methods:

Demographics and clinical characteristics of two patients from our clinic who developed LPL cirAEs where recorded. Similar demographics and clinical characteristics of three reported cases in literature were also recorded in order to be presented as a cohort.

Results:

Five patients with LPL cirAEs who were successfully managed with hydroxychloroquine as systemic monotherapy are presented in Table 1, with three reported in literature and two new cases reported herein. Case 1 is a 72 year-old man with advanced prostate adenocarcinoma who developed a LPL cirAE after 15 weeks of ICI use. Case 2 is a 76 year-old woman with advanced colon cancer who developed a LPL cirAE after 9 months of ICI use. The onset of LPL cirAEs varied, from less than one week into ICI therapy to nine months afterwards. All five patients' LPL cirAEs were managed with hydroxychloroquine, allowing for them to continue on ICI therapy. Only two patients required a brief interruption of ICI (one week and two months). In all of five cases, hydroxychloroquine was well tolerated.

Table 1 – Summary of reported and presented cases of hydroxychloroquine used in the management of lichen planus-like cutaneous immune-related adverse events secondary to immune checkpoint inhibitor use in cancer management. Case 1 and Case 2 are not yet published and are presented in this abstract.

Case report	Age & sex	Cancer type & stage	Time to rash onset from ICI initiation/ cycle #	ICI interruption
Case 1	72 M	Prostate adenocarcinoma stage IIIc	15 weeks/C5	1 week
Case 2	76 F	Colon adenocarcinoma stage IIIc	9 months/C13	None
Alnajjar et al.	67 F	Endometrial carcinoma stage IV	1 month/C1	2 months
Coscarart et al.	78 F	Lung adenocarcinoma stage II	<1 week/C1	None
Headd et al.	60 M	Renal cell carcinoma stage IV	32 weeks/C16 (1) 18 months* (2)	None

(#) = order of instance, *After ICI discontinuation. M=male; F=female; ICI=immune checkpoint inhibitor.

Conclusion:

While there is no standardized management for LPL cirAEs, hydroxychloroquine is a safe and effective steroid-sparing therapy in this context. With the increasing prevalence of ICI use, the development of steroid-sparing therapies for cirAE management is imperative. In all of five known cases, use of hydroxychloroquine allowed for treated patients to continue with ICI therapy and further resaerch will assess the long-term efficacy and safety of this therapy.

Abstract N°: 3092

life-threatening adverse effects of dapsone in patients of leprosy: a case series

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Introduction & Objectives:

Dapsone has been used for the treatment of leprosy since 1945 and its adverse effects range from mild hemolysis to several life-threatening adverse reactions. Here we describe a series of 6 patients of leprosy who were started on dapsone as a part of WHO Multidrug therapy for multibacillary leprosy (MDT-MB) and presented later with life-threatening adverse effects.

Materials & Methods:

All patients of leprosy who presented with signs and symptoms suggestive of adverse effects of dapsone were evaluated thoroughly. After detailed history taking, clinical examination and appropriate investigations, dapsone was stopped and patients were managed appropriately.

Results:

We encountered 6 cases (all males) of leprosy who presented to emergency with life-threatening adverse effects of dapsone over a period of 1 year. Their age ranged from 22 to 72 years. All these cases had been previously diagnosed with leprosy and were being treated with WHO-MDT MB. Duration of MDT varied from few days to 9 months. The cases included two** cases of severe methemoglobinemia and one each of hemolytic anemia, drug induced liver injury, generalized fixed drug eruption (FDE) and drug rash with eosinophilia and systemic symptoms (DRESS). Dapsone was stopped in all cases and they were additionally treated as per individual adverse reaction. Five out of these cases were successfully managed and were later treated with modified MDT (without dapsone); while one case of DRESS succumbed to severe liver injury. None of the patients was glucose-6-phosphate dehydrogenase deficient.

Conclusion:

All components of MDT but particularly dapsone carries highest risk of life-threatening reactions. Dapsone has wide spectrum of adverse effect ranging from insignificant degree of hemolysis to life threatening complications like methemoglobinemia, agranulocytosis and dapsone hypersensitivity syndrome. A keen observation and remaining vigilant after starting MDT is essential for early diagnosis and successful treatment of such adverse effects.

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Abstract N°: 3102

Acute generalized exanthematous pustulosis induced by cefazolin: An atypical cross-reactivity involving the “cephem” ring

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Introduction & Objectives:

Acute generalized exanthematous pustulosis (AGEP) is a sever cutaneous drug reaction presenting with rapid-onset sterile pustules on edematous erythema, mainly attributed to drugs. Antibiotics are implicated in 80% of these cases, mostly penicillins and macrolides. There are few cases associated with cephalosporins. We report a original case, in which cefazoline induced AGEP and displayed an unique cross-reactivity to all tested cephalosporins. Tolerance to penicillins were confirmed with Patch tests (PT) and negative

oral challenges.

Materials & Methods:

NA

Results:

A 53-year-old male, with a three-year history of recurrent erysipelas, for which he was treated with long-term preventive penicillin G. He was Hospitalised for management of another episode of recurrent erysipelas and a treatment with cefazolin was initiated. Night days after ongoing cefazolin, a pustular eruption, appeared initially on flexural regions then generalized symmetrically to the extremities and trunk without mucosal involvement. Leucocytosis (21.970 cells/µl) with an elevated neutrophil count (10.840 cells/µl) were objected on laboratory tests. Anatomopathological findings revealed spongiform subcorneal pustules, oedema in the papillary dermis and perivascular inflammatory infiltrate consisting of neutrophils, findings consistent with AGEP. cefazolin was withheld and topical corticosteroids and emollient were prescribed for symptomatic relief. The clinical feature was resolved within ten days. Three months later, PT with cefazolin was performed, yielding negative result. Howeve, an intradermal test (IDT) to cefazolin was positive at the 48-hour reading. To access cross-reactivity among betalactam. We performed IDT to ceftriaxone, cefotaxime, ceftazidime, (2 mg/mL), benzylpenicillin, amoxicillin and oxacillin (20 mg/mL). These tests were positive to ceftriaxone, cefotaxime and ceftazidime at 48-hour reading and negative to benzypenicillin, amoxicillin, and oxacillin. Subsequently, a sequel gradual oral provocation test (OPT) to amoxicillin and penicillin V were carried out with no incidence.

Conclusion:

According to the Naranjo Causality Scale, the accountability of both drugs was found to be probable (score of ‘6’). This patient experienced an AGPE induced by cefazoline and confirmed by a positive IDT. Allergological workup was relevant for a cross-reactivity toward all tested cephalosporins and tolerance to penicillins. In light of these considerations, it could be argued that the cross-reactivity to cephalosporins observed in our patient would be related to the 6-membere dihydrothiazine (cephem) ring bound to the β-lactam ring, which is common to all cephalosporins.

Abstract N°: 3113

Clinical Audit on the Current Management of Stevens–Johnson Syndrome and Toxic Epidermal Necrolysis in Adults at a Tertiary Care Centre

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Introduction:

Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are rare but life-threatening mucocutaneous reactions, most commonly caused by medications. These conditions are associated with significant morbidity and mortality, and optimal outcomes depend on early recognition, prompt withdrawal of the culprit agent, and multidisciplinary care. Despite existing guidelines, real-world management remains inconsistent across institutions.

Objective: To evaluate current clinical practices in the management of adult patients with SJS/TEN at a tertiary care center and compare these practices to international management guidelines.

Materials & Methods:

A literature review using Ovid MEDLINE and Embase was conducted to identify national and international guidelines on SJS/TEN management published between 2000 and 2025. In the absence of Canadian guidelines, protocols from five countries—United Kingdom, United States, France, Germany, and India—were reviewed and synthesized to develop audit standards. A retrospective chart review was performed on adult patients diagnosed with SJS/TEN between January 2020 and December 2024. Data were collected on SCORTEN documentation, initial assessments (ocular, oral, urogenital), drug causality, systemic treatments used, location of inpatient care, and discharge planning elements such as medical alert recommendations and follow-up referrals.

Results:

Six adult cases were identified. The audit revealed variability in documentation and adherence to recommended care practices. While the withdrawal of suspected causative agents was consistently performed, other measures, including SCORTEN scoring, mucosal site assessments, and interdisciplinary evaluations, were inconsistently documented. Discharge instructions varied, with some lacking key recommendations such as MedicAlert identification and referral to specialized follow-up services.

Conclusion:

The audit highlights significant variability in SJS/TEN management and documentation at the study site. There is a clear need for standardized care protocols and structured documentation tools to ensure comprehensive, multidisciplinary care. Future directions include expanding the audit sample, collaborating with specialty clinics, and implementing standardized care pathways to enhance clinical outcomes and continuity of care for patients with SJS/TEN.

Abstract N°: 3171

Paradoxical cutaneous reactions in the treatment of hidradenitis suppurativa: a case report

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Introduction & Objectives:

Biologic therapies targeting pro-inflammatory cytokines such as TNF-alpha and IL-17 generally exhibit favorable safety profiles. Nevertheless, immune-mediated adverse effects are not uncommon. We present a clinical case highlighting paradoxical cutaneous reactions during the treatment of hidradenitis suppurativa (HS), with the aim of contributing to the understanding of such events and exploring alternative therapeutic strategies.

Materials & Methods:

We report the case of a 51-year-old woman with a medical history of dyslipidemia, hypertension, active smoking, and grade 1 obesity. She presented with a worsening of HS, initially diagnosed the previous year. On physical examination, she showed multiple fistulas and inflammatory nodules in axillary, inguinal, and submammary regions. Treatment was initiated in May 2019 with a biosimilar of adalimumab, alongside occasional surgical intervention and antibiotics for flare-ups

Results:

In November 2021, the patient visited the emergency department due to a new outbreak of erythematous and pustular plaques on the trunk and limbs, including the palms, along with severe scalp hyperkeratosis. A skin biopsy confirmed psoriasis, and adalimumab was discontinued due to suspicion of a paradoxical reaction. Secukinumab was initiated, leading to significant improvement in both psoriasiform lesions and HS.

However, 2–3 weeks later, she developed erythematous-crusted plaques on the lower limbs and knee arthralgia. A second biopsy revealed leukocytoclastic vasculitis. Secukinumab was withdrawn, and oral prednisone was prescribed, leading to improvement.

Subsequently, guselkumab was introduced at psoriasis-standard dosing, initially combined with cyclosporine. Currently, the patient remains on guselkumab monotherapy, showing good control of both psoriasis (only isolated scalp lesions) and HS (no new fistulas or abscesses).

Conclusion:

Paradoxical psoriasis has been reported in over 15% of HS patients treated with adalimumab. In contrast, vasculitis as an adverse reaction to secukinumab is rare, with only one other documented case linked to HS treatment. Literature on the use of guselkumab in HS is limited and suggests modest efficacy, potentially due to its application in severe, treatment-resistant cases or suboptimal dosing. This case highlights the importance of monitoring for paradoxical reactions during biologic therapy and suggests guselkumab may be a viable alternative in selected patients.

Abstract N°: 3266

Early use of tumor necrosis factor antagonist shortens course of Stevens-Johnsonsyndrome/toxic epidermal necrolysis

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Introduction & Objectives:

Stevens-Johnson syndrome (SJS) / toxic epidermal necrolysis (TEN) are life-threatening drug reactions with high mortality rate. However, the treatment for SJS/TEN remained controversial. In this study, we explored early use of tumor necrosis factor (TNF) α inhibitors in SJS/TEN and identified potential cytokines predicting clinical course.

Materials & Methods:

Patients of acute SJS/TEN were retrospectively reviewed and time from onset to treatments were recorded. Serum levels of interleukin (IL)-2, IL-4, IL-6, IL-10, TNF-α and interferon (IFN) γ were measured in the acute phase. Factors that related to acute stage and skin healing were analyzed.

Results:

A total of 64 patients were included, among which 47 received combination therapy with methylprednisolone and TNF inhibitor, 17 were treated with steroid monotherapy. In patients treated with TNF inhibitor, median time of acute stage was 5 (IQR 4-7) days, median time for skin healing was 10 (IQR 6-15) days. Linear regression analysis showed that baseline score of SCORTEN (β=0.543, 95% CI 1.097-3.292, p<0.001) and onset to TNF treatment time (β=0.263, 95% CI 0.002-0.349, p=0.047) were associated with skin healing time. Elevated serum IL-6 and TNF- α were observed in acute stage of SIS/TEN, and skin re-epithelization time was positively correlated with IL-6 level in SJS/TEN patients (r=0.326, p=0.015).

Conclusion:

Early use of TNF-α inhibitor was beneficial for patients with SJS/TEN. Serum IL-6 level might be a promising marker for clinical course.

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Abstract N°: 3344

A Case of Benidipine-Induced Lichenoid Drug Eruption

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Introduction & Objectives:

Lichenoid drug eruptions (LDE) are rare adverse cutaneous reactions mimicking lichen planus (LP) both clinically and histologically. However, key distinctions such as the absence of Wickham striae and characteristic histological findings, including eosinophilic infiltrates and vacuolar degeneration, aid in differentiating LDE from idiopathic LP.

Although various drugs, including beta-blockers and angiotensin-converting enzyme inhibitors, are commonly implicated, this case represents the first reported instance of LDE induced by benidipine, a calcium channel blocker with known renoprotective and cardioprotective properties.

Materials & Methods:

A 76-year-old female presented with a three-week history of intensely pruritic erythematous and violaceous papules and plaques primarily localized to the lower extremities. The eruption developed 3 weeks after initiating benidipine for the management of hypertension. A punch biopsy confirmed the diagnosis of LDE, revealing interface vacuolar degeneration and band-like lymphocytic infiltration. Considering the temporal relationship, benidipine was discontinued based on the suspicion of benidipine-induced LDE and doxazosin was initiated. Since the patient had widespread lesions, systemic prednisolone treatment (40 mg daily) was started in addition to the discontinuation of benidipine and complete resolution of the lesions was observed within two months. No recurrence was observed in the 5-month follow-up.

Results:

Although LDE may have several clinical and histopathological findings that help in the differential diagnosis from idiopathic LP, these two entities still represent very similar clinical pictures, making the diagnosis of LDE challenging. LDE has a characteristic delayed onset, often appearing weeks to months after initiating the offending medication. In the present case, the latent period was approximately three weeks, which aligns with the variability reported in the literature. The diagnosis of LDE was supported by the temporal association between the onset of symptoms and initiation of benidipine, as well as the resolution of lesions following discontinuation of the drug and the introduction of systemic corticosteroid therapy. Histopathological findings, including band-like lymphocytic inflammation with sparse eosinophils and interface vacuolar degeneration, further corroborated the clinical diagnosis. Treatment for LDE generally involves cessation of the causative drug, with most cases resolving within weeks to months. In severe or persistent cases, systemic corticosteroids may be required, as was employed successfully in this patient. The absence of new lesions and complete resolution of existing lesions at the two-month follow-up underscores the importance of early recognition and prompt discontinuation of the offending agent.

Conclusion:

This case expands the spectrum of adverse cutaneous reactions associated with calcium channel blockers, underscoring the importance of prompt recognition and management of LDE. Clinicians should remain vigilant for

LDE in patients presenting with compatible eruptions following antihypertensive therapy, particularly when involving less commonly implicated agents like benidipine.

Abstract N°: 3386

Squamous Cell Carcinoma Induced by Adalimumab Therapy for Psoriatic Arthritis: A Diagnostic and Therapeutic Challenge

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Introduction & Objectives:

Psoriatic arthritis (PsA) is a chronic, heterogeneous, immune-mediated inflammatory disease affecting the musculoskeletal system. Adalimumab, a fully human monoclonal antibody targeting tumor necrosis factor-alpha (TNF-α), is widely used for the management of PsA and other chronic inflammatory conditions. While effective, long-term TNF-α inhibition may carry a risk of malignancy, particularly non-melanoma skin cancers (NMSC).

We report a rare case of cutaneous squamous cell carcinoma (CSCC) in a PsA patient treated with adalimumab, without other classical risk factors, to emphasize the importance of monitoring for cutaneous malignancies during biologic therapy.

Materials & Methods:

A 74-year-old male with a 10-year history of adalimumab therapy for PsA and chronic plaque psoriasis presented with a 2-year history of a skin lesion on the first left interdigital space. He had Fitzpatrick skin type V, no history of intense sun exposure, phototherapy, or smoking.

Clinical examination revealed a 1×1.5 cm ulcerated, scaly, verrucous nodule with an indurated base. A skin biopsy confirmed a well-differentiated CSCC. Radiologic evaluation, including whole-body CT and regional ultrasound, showed no signs of lymph node involvement or metastasis. Surgical excision with clear margins was performed. The lesion was staged pT1N0M0 (AJCC 8th edition).

Results:

Adalimumab was discontinued following pharmacovigilance guidance and replaced by symptomatic treatments. At three months follow-up, there was no local recurrence or metastasis. The patient’s PsA and cutaneous psoriasis remained stable with no disease flare.

Conclusion:

This case highlights a potential link between long-term adalimumab therapy and CSCC development in the absence of traditional risk factors. Although TNF-α inhibitors have transformed the treatment of PsA, vigilance for malignancies, particularly skin cancers, is warranted. Dermatologic monitoring should be incorporated into long-term management plans for patients receiving anti-TNF-α therapies, with interdisciplinary collaboration ensuring optimal care.

Abstract N°: 3464

Folliculitis decalvans secondary to treatment with tumor necrosis factor alpha inhibitors

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Introduction & Objectives:

In recent years, a wide spectrum of adverse reactions associated with the use of tumor necrosis factor alpha inhibitors (anti-TNFa) has been recognized, including paradoxical psoriasis and, rarely, alopecia.

Materials & Methods:

A 29-year-old woman with a 15-year history of severe psoriasis, with extensive involvement the scalp and palmoplantar areas, refractory to multiple topical treatments and methotrexate, was started on biological therapy with adalimumab.

After two months of treatment with adalimumab, the patient developed a scarring alopecic plaque on the scalp with abundant suppuration, along with psoriasiform plaques on the limbs and trunk. Trichoscopy reveled a milky-red alopecic plaque with destruction of the follicular openings. At the periphery, numerous tufted hairs and perifollicular keratosis were observed.

Histological examination showed a psoriasiform epidermal pattern and a dermis with a lymphoplasmacytic infiltrate interstitial and perifollicular with abundant neutrophils. Scarring fibrosis was also observed in the interfollicular dermis, along with follicles showing multiple hair shafts (tufted hairs) and some hair shafts devoid of follicular epithelium, surrounded by neutrophils and foreign-body giant cells.

Based on these findings, a clinico-pathological diagnosis of folliculitis decalvans was made. Adalimumab was immediately discontinued and doxycycline 100mg every 24 hours was started. After one month of treatment, a marked improvement was observed, with absence of suppuration, less erythema and regrowth of some vellus hairs.

Results:

Among the cutaneous reactions described using anti-TNFa therapy, alopecia has been documented in the form of alopecia areata and psoriasiform alopecia.

Recently, a new type of alopecia secondary to anti-TNFa therapy has been described, which combines features of both entities. Histopathologically, these cases demonstrate psoriasiform epidermal changes along with dermal alterations like those in alopecia areata, including follicular miniaturization, increased number of catagen and telogen hairs, and peribulbar infiltrate in terminal hairs.

In this report, we present a different form of alopecia secondary to anti-TNFa therapy: folliculitis decalvans. To our knowledge, only two cases of this entity have previously been described in the literature.

Conclusion:

We present a case of folliculitis decalvans secondary to adalimumab use, a different entity from the alopecia induced by anti-TNFa described in the literature.

Abstract N°: 3474

Eczematous rash associated with Pembrolizumab: Successful Management with Phototherapy

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Introduction & Objectives:

Immune checkpoint inhibitors (ICIs) frequently cause immune-related adverse events (irAEs), with skin toxicities affecting up to 50% of patients 1. While many are mild, severe eruptions can significantly impair quality of life and complicate oncology treatment decisions. This case highlights pembrolizumab-induced eczematous rash, successfully managed with adjunct phototherapy, enabling continuation of immunotherapy.

Materials & Methods:

An 81-year-old man with metastatic squamous cell lung carcinoma (PD-L1 90%) was initiated on pembrolizumab 400mg every 6 weeks. Comorbidities included COPD, hypertension, and hypercholesterolemia. After Cycle 2, a widespread, pruritic eczematous rash appeared, affecting 60% of body surface area (BSA). Prednisolone 50mg (0.5mg/kg) was commenced. Despite treatment the rash affected 70% of BSA after cycle 3 causing significant pruritus and sleep disruption. Pembrolizumab was paused. Ten weeks later the rash persisted on doses of prednisolone under 20mg.

Skin biopsy demonstrated spongiotic dermatitis with lymphocyte exocytosis, dyskeratotic keratinocytes with scant neutrophils, consistent with drug toxicity. Autoimmune serology was negative.

A Dermatology/Oncology MDT was held to determine the best treatment. Narrowband UVB (NB-UVB) phototherapy was initiated alongside a tapering course of prednisolone, leading to significant improvement.

Results:

Pembrolizumab was re-started after a 10 week break at a dose of 200 mg every 3 weeks. Within 3 weeks, the rash recurred and progressed with associated impetigo by week 5. Skin swab confirmed *Staphylococcus aureus* infection. It was managed with flucloxacillin, topical steroids, and restarting prednisolone at 10 mg.

Three months later, the patient remained on pembrolizumab, 5 mg prednisolone, and biweekly phototherapy.

The multidisciplinary decision to commence phototherapy alleviated cutaneous symptoms and psychological distress for the patient, while allowing immunotherapy to continue.

Conclusion:

Spongiotic dermatitis is a recognised ICI-related cutaneous irAE. NB-UVB phototherapy effectively controlled the eruption and reduced steroid reliance. Evidence supports phototherapy in ICI-related pruritus with or without a rash 1. Balancing clinical benefit with immunotherapy-related skin toxicity was especially challenging, necessitating careful multidisciplinary decisions.

Literature supports continuation of ICIs in most cutaneous irAEs unless life-threatening 2. Collaborative management was critical in balancing oncologic benefit and dermatologic risks, allowing pembrolizumab rechallenge and improving quality of life.

Severe cutaneous irAEs like spongiotic dermatitis can be effectively managed with adjunctive therapies such as phototherapy, allowing continuation of life-prolonging immunotherapy. This case underscores the importance of multidisciplinary care in managing severe skin toxicity while preserving oncologic outcomes and patient wellbeing.
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Abstract N°: 3594

Terlipressin-induced ischemic skin necrosis: a case series from a secondary-level hospital in Mexico City

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Introduction & Objectives:

Terlipressin, is widely used for esophageal variceal bleeding and hepatorenal syndrome-acute kidney injury. Its vasoconstrictive action can cause ischemic complications, like cutaneous necrosis. We report four cases of terlipressin-induced ischemic necrosis in patients with hepatic dysfunction.

Materials & Methods:

Case report 1:

A 65-year-old woman with metabolic dysfunction-associated liver disease developed retiform erythematous-violaceous macules and blisters on the foot and scalp 3 days after starting terlipressin at 4 mg loading dose, then 2 mg every 4 hours for 5 days. Scalp biopsy showed extensive epidermal and adnexal necrosis. Lesions resolved, with hair regrowth, after drug discontinuation and wound care.

Case report 2:

A 65-year-old man with suspected alcohol-related liver disease and upper gastrointestinal bleeding, started to develop widespread violaceous macules and flaccid, hemorrhagic, blisters affecting the trunk, scrotum, penis, and lower limbs within 48 hours of terlipressin initiation, same dosing as Case 1. Histopathology confirmed extensive epidermal necrosis. The patient died secondary to hypovolemic shock.

Case report 3:

A 65-year-old man with Child-Pugh B liver disease received terlipressin at 4 mg over 24 hours, for variceal bleeding, which was later modified to 2 mg every 6 hours for 24 hours. Two days after stopping the medication, he developed hemorrhagic blisters and necrotic eschars on the scalp, scrotum, and penis. Management included interruption of drug therapy and wound care with adequate re-epitelization.

Case report 4:

A 63-year-old man with hepatitis C–related cirrhosis developed multiple ulcers with hemorrhagic crusts on the scrotum, penile shaft, and left lower limb, two days after initiating terlipressin at 2 mg every 6 hours for 5 days. He responded well to wound care but died following a second episode of variceal bleeding.

Results:

Terlipressin-induced skin necrosis is an infrequent complication, characterized by the onset of painful necrotic skin lesions; in our series of four cases, lesions developed 48 to 72 hours after terlipressin administration, consistent with the median onset described in previous reports. Described as retiform erythematous-violaceous macules,

often with flaccid blisters or necrotic eschars, affecting lower limbs, scrotum, trunk, scalp, and penile shaft. While previous literature identifies the abdomen, lower limbs, and scrotum as the most frequently involved areas (Cases 2 and 4), our cases contribute to the spectrum by documenting scalp involvement (Cases 1 and 3) and localized acral necrosis (Case 1), both of which are infrequently reported.

Histopathological findings in two of our patients, confirmed the diagnosis, revealing epidermal and adnexal necrosis, occasionally accompanied by dermal thrombi. These findings support the hypothesis that terlipressin's vasoconstrictive action especially in patients with underlying hepatic dysfunction and compromised perfusion may precipitate cutaneous ischemia.

Conclusion:

This study provides further insight into the pathophysiology, risk factors, and anatomical variability, emphasizing the need for individualized therapies and suggest that early recognition and timely drug discontinuation remain essential in preventing irreversible ischemic skin damage in high-risk patients receiving terlipressin therapy.

Abstract N°: 3756

Evolving Skin Symptoms and Systemic Involvement in DRESS Syndrome: Insights from a Case of Coexisting PRP and Crystal Arthropathy

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Introduction & Objectives:

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) is a rare, potentially life-threatening hypersensitivity reaction, triggered by antiepileptic drugs, sulfonamides and antibiotics such as vancomycin. The syndrome is characterized by fever, hematologic abnormalities, systemic involvement and polymorphic cutaneous eruptions. Diagnosis is challenging due to the delayed onset of symptoms, which may overlap with other severe cutaneous adverse reactions and clinically mimic sepsis.

Materials & Methods:

A 58-year-old male underwent left total hip arthroplasty in June 2023 for advanced coxarthrosis. Three weeks postoperatively, signs of infection (fever, purulent drainage, elevated inflammatory markers) indicated DAIR surgery with implantation of vancomycin- and gentamicin-loaded stimulan beads. MRSE was cultured, so intravenous vancomycin in combination with oral rifampicin was initiated for two weeks, followed by oral trimethoprim-sulfamethoxazole upon discharge.

Four weeks later the patient developed high fever and cutaneous eruptions on the limbs and back, laboratory tests revealed elevated inflammatory markers. Due to concern for reinfection, vancomycin, rifampicin and meropenem were administered. CT indicated periarticular abscess and hepatosplenomegaly. Repeated DAIR surgery was performed with reimplantation of antibiotic beads. Postoperatively, faint erythematous papules on the chest and extremities rapidly progressed into confluent livid-red exanthema. Laboratory findings including eosinophilia, elevated liver enzymes, lymphadenopathy, persistent fever and hepatomegaly raised suspicion for DRESS syndrome, which diagnosis was supported by biopsy. The entire prosthesis and the antibiotic beads were removed and high-dose systemic corticosteroids were initiated and tapered over three months, resulting in clinical improvement.

Results:

In December 2023, one week after steroid cessation, the patient presented with fever, anemia and new, coin-sized erythematous scaling plaques, mainly on the flexural areas. Histology confirmed pityriasis rubra pilaris (PRP). Treatment with corticosteroids and cyclosporine led to permanent improvement.

In early 2024, the patient noted swelling over the left hip, accompanied by CRP flares. MRI revealed a multi-compartmental joint effusion communicating with the prosthetic joint. Rheumatology consultation and ultrasound-guided joint aspiration followed by crystal analysis confirmed crystal arthropathy. Febuxostat, colchicine and low-dose corticosteroids were introduced, leading to remission of inflammation. LTT was positive for vancomycin, stimulan and trimethoprim-sulfamethoxazole.

In July 2024, the final prosthesis was implanted under linezolid coverage. Although the LTT result for rifampicin was negative, its reintroduction led to a DRESS relapse. With corticosteroid therapy and rheumatologic management, full remission was achieved.

Conclusion:

This case highlights the importance of recognizing locally applied vancomycin as a prolonged antigen source, possibly contributing to the relapse of DRESS. It illustrates a hyperergic immune response manifesting through multiple clinical phenotypes. Joint swelling and crystal arthropathy were effectively managed through targeted rheumatologic therapy. Multidisciplinary collaboration enabled appropriate diagnosis, management, and successful rehabilitation.

Abstract N°: 3896

A Thin Line Between Cure and Harm: The Role of Patient Adherence in Preventing Skin Complications

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A Thin Line Between Cure and Harm: The Role of Patient Adherence in Preventing Skin Complications

Introduction & Objectives:

Imiquimod is an immune response modifier used in the treatment of cutaneous malignancies and premalignant lesions, exerting its effect through the induction of localized inflammation that facilitates lesion clearance. The most frequently reported adverse effects of imiquimod are localized reactions at the site of application. These drug-related reactions commonly manifest as erythema, burning, pruritus, and pain localized to the treatment area. A secondary infection may develop in rare instances, particularly in cases of non-adherence to treatment and skincare guidelines.

Objectives: to describe the clinical manifestations resulting from the use of topical imiquimod on healthy skin when applied without physician guidance.

Materials & Methods:

A case involving the self-directed use of topical imiquimod on intact skin, leading to a severe local inflammatory response and the development of a secondary infection.

Results:

A 45-year-old male presented to the dermatology clinic with inflammatory eruptions on the facial skin.**Clinical Examination**: On physical examination, extensive hyperkeratotic, brown-yellow crusted plaques measuring approximately 3 cm thick and 4-5 cm in diameter were observed bilaterally in the infraorbital regions, with an erythematous base. Additionally, erythematous, scaly plaques with serous crusts were noted on the nasal tip and both temples. **Patient History**: One month prior to presentation, the patient had been prescribed imiquimod 5% cream to be applied three times per week for one month to target actinic keratoses on the nasal tip and temporal regions. Contrary to medical instructions, the patient self-initiated daily application of the cream to the bilateral nasolabial areas, where no clinically evident lesions were present. The patient reported progressive crust formation in the cheeks over the course of treatment, with gradual plaque enlargement. He refrained from disturbing the crusts and avoided cleansing the affected areas. **Diagnosis**: A diagnosis of impetiginization, resulting from inappropriate use of topical imiquimod on unaffected skin, was established. **Management and Clinical Course**: Initial treatment consisted of twice-daily application of disinfectant wet dressings, a topical zinc bacitracin–neomycin sulfate complex, and a glycerol-based emollient. Several days later, following spontaneous detachment of the crusts, the patient observed pustular lesions. Systemic antibiotic therapy with clindamycin 300 mg twice daily was initiated for six days. At a two-week follow-up, residual erythema was noted in previously affected areas, with no signs of ongoing infection or further complications.

Conclusion:

This case underscores the potential for serious local adverse reactions and secondary infections resulting from the unsupervised or inappropriate use of topical immunomodulatory agents without physician prescription. It

highlights the critical need for thorough patient education regarding the precise application of prescribed treatments and the importance of clinician follow-up, especially when using agents associated with strong local inflammatory responses.

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Abstract N°: 4055

Erythema Multiforme: clinical presentation and triggering factors

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Introduction & Objectives:

Erythema multiforme (EM) is an acute condition affecting the skin and mucous membranes, usually triggered by infections or certain medications. The lesions are typically “target-like” with variable severity of mucosal involvement. The objective of this study was to describe the clinical, demographic, and histopathological features of 25 cases of EM.

Materials & Methods:

This retrospective study analyzed 25 cases of EM, confirmed by histopathology, treated in the dermatology department between 2016 and 2024. The clinical data collected included age, gender, triggering factors, skin and mucosal involvement, severity of lesions, and treatment outcomes.

Results:

The study included 25 patients, with a mean age of 34 years and a male-to-female ratio of 0.32. The majority of the cutaneous lesions were localized to the extremities, and mucosal involvement was observed in 24% of cases. Severe lesions, including extensive mucosal erosions, were noted in 52% of cases. Histopathological examination revealed interface dermatitis with keratinocyte necrosis. The most frequent cause was post-herpetic, affecting 60% of patients, followed by Mycoplasma infections, confirmed in 28% of cases, while the remaining 12% had a drug-induced etiology. Hospitalization was required in 42% of cases. Although most patients responded well to symptomatic treatment, severe cases required systemic corticosteroid administration.

Conclusion:

This study is consistent with the data reported in the literature. The observed male-to-female ratio in our study is similar to other series, where a female predominance is often noted. The mean age of patients also aligns with the findings of other studies, indicating that EM primarily affects young adults. The identification of triggering factors in this study supports the literature, which highlights a frequent association with viral infections, particularly herpes simplex. Mycoplasma infections were also well-documented as triggering factors. Rapid and appropriate management, based on early detection and accurate assessment of severity, is essential to minimize complications. EM is predominantly triggered by viral infections, with herpes simplex being the most common cause. Early diagnosis and appropriate treatment are key to managing the condition and preventing complications. Further research is needed to better understand triggers and optimize treatment approaches for improved patient outcomes.

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Abstract N°: 4067

When antibiotics get hairy: a rare case of black hairy tongue linked to co-amoxiclav

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Introduction & Objectives: Black hairy tongue (BHT), also known as *lingua villosa* or *melanoglossia*, is a rare, benign, and self-limiting condition. It is characterized by a black discoloration and a hair-like appearance on the dorsal surface of the tongue, resulting from the elongation of filiform papillae. Here, we present an unusual case of BHT induced by co-amoxiclav, emphasizing the importance of recognizing this adverse drug reaction.

Materials & Methods: NA

Results: A 22-year-old female with a history of allergic rhinitis was prescribed oral co-amoxiclav for bronchitis. Nine days after starting the antibiotic, she presented to our department with a black-brownish coating on her tongue. Upon examination, the pigmentation was primarily observed on the dorsum of the tongue, with the buccal mucosa remaining unaffected. The patient denied smoking, alcohol consumption, or excessive intake of coffee or tea, which are common risk factors for BHT. The mycological examination of the oral cavity and the HIV serology were both negative. Based on the characteristic clinical presentation, a diagnosis of antibiotic-induced BHT was made. The patient was reassured and advised to maintain good oral hygiene, including regular tongue brushing and adequate hydration, while completing the prescribed two-week course of antibiotics. Notably, the discoloration improved significantly within three days and completely resolved one week after finishing the antibiotic therapy.

Conclusion: This case highlights a rare association between co-amoxiclav and BHT. While the exact pathophysiology remains unclear, previous studies suggest that certain antibiotics may disrupt the oral microbiome, leading to the overgrowth of chromogenic bacteria and fungi that produce porphyrins, resulting in pigmentation. Additionally, impaired desquamation of keratinized cells on the dorsal tongue surface leads to the accumulation of elongated filiform papillae, further contributing to the “hairy” appearance. Various antibiotics, including amoxicillin-clavulanate, metronidazole, doxycycline, erythromycin, linezolid, tetracycline, moxifloxacin, and imipenem/cilastatin, have been associated with BHT. Although asymptomatic in most cases, BHT can be distressing due to its striking appearance. Some patients may also experience halitosis, nausea, or a burning sensation. The condition typically develops 1–2 weeks after exposure to predisposing factors, including smoking, poor oral hygiene, excessive coffee/tea consumption, alcohol intake, antibiotic use, malignancy, and immunosuppression. In our case, the absence of conventional risk factors, aside from co-amoxiclav use, supports its role in the development of BHT. The prognosis is favorable, as the condition is self-limiting and resolves with appropriate oral hygiene. While first-line management consists of eliminating contributing factors and maintaining good oral hygiene, second-line treatments, such as oral and topical retinoids or antifungal agents, have been explored but lack strong clinical evidence. This case underscores the importance of recognizing co-amoxiclav as a potential, though rare, cause of BHT. Awareness of this benign and self-limiting condition is essential to avoid unnecessary discontinuation of antibiotics and to alleviate patient anxiety. Emphasizing proper oral hygiene remains the cornerstone of management, ensuring timely resolution.

Abstract N°: 4219

A case of nevirapine-associated Toxic Epidermal Necrolysis in a neonate

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Introduction & Objectives: Drug-induced epidermal necrolysis (DEN), including Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN), is a rare, life-threatening reaction, especially in infants with only isolated neonatal cases reported globally. Nevirapine, a non-nucleoside reverse transcriptase inhibitor, is the preferred antiretroviral for preventing vertical HIV transmission in neonates in South Africa. While its role in severe cutaneous adverse reactions (SCARs) is established in adults, its implication in neonatal TEN is poorly documented. HIV-infection, immunocompetence with CD4 >15% and HLA-C*04:01 have been found to be associated with an increased risk of nevirapine-induced SJS/TEN in sub-Saharan African populations. The objective of this cae report is to highlight an exceptionally rare instance of nevirapine-induced TEN in a neonate.

Materials & Methods:

We report a 17-day-old HIV-exposed but uninfected neonate receiving nevirapine for prevention of vertical HIV transmission who developed a rapidly progressing erythematous and blistering rash accompanied by fever on day 16 of exposure. Initial differential diagnosis included neonatal sepsis, herpes simplex virus (HSV), varicella-zoster virus (VZV), and staphylococcal scalded skin syndrome. As the rash progressed to involve mucosal surfaces and widespread skin stripping, and infectious causes were excluded (negative blood cultures, HSV PCR, VZV PCR, Tzanck smear and HIV PCR) a cutaneous adverse reaction was suspected. Skin biopsy demonstrated subepidermal blistering with full-thickness epidermal necrosis and RegiSCAR criteria supported a diagnosis of TEN based on fever, targetoid lesions, mucosal erosions, and >30% body surface area involvement. The ALDEN drug causality assessment algorithm supported nevirapine as the definitive causative agent and HLA typing was also supportive confirming carriage of HLA-C*04:01. The infant was managed with systemic corticosteroids and multidisciplinary supportive care.

Results:

Blistering ceased by day four with complete re-epithelialization by day 10. At 6-month follow-up, the infant remained well, HIV PCR negative, with residual post-inflammatory hyperpigmentation.

Conclusion: This case illustrates an exceptionally rare instance of genetically predisposed nevirapine-induced TEN in an HIV-negative neonate carrying the HLA-C*04:01 risk allele. DEN is an HLA-class I restricted CD8+ T cell dependent reaction. The occurrence of DEN in this neonate with an immature T cell immune system is highly unusual and highlights not only the importance of excluding infectious mimics, prompt drug withdrawal and comprehensive supportive care but also offers unique insights into the immunopathogenesis of DEN in early life.

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Abstract N°: 4247

DRESS syndrome induced by imatinib

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Introduction & Objectives:

Drug reaction with eosinophilia and systemic symptoms (DRESS) is a severe cutaneous adverse drug reaction, with rare cases being imatinib-induced. The main treatment is the withdrawal of the causative drug. We report an rare case of DRESS syndrome associated with imatinib in a 62-year-old man with acute myeloid leukemia (AML).

Materials & Methods:

A 62-year-old male developed a generalized pruritic maculopapular exanthema, more pronounced on the forearms, lower limbs, and neck, along with lichenified and excoriated lesions, facial edema, and multiple axillary and inguinal lymphadenopathies measuring greater than 1 cm, accompanied by prolonged fever. These symptoms appeared 9 weeks after initiating imatinib and 5 weeks following the commencement of azacitidine for acute myeloid leukemia (AML). The peripheral absolute eosinophil count increased to 1,900 cells/µL. The remainder of the work-up was unremarkable, including normal liver and kidney function tests. The chest X-ray and ECG were also free of abnormalities.

A diagnosis of DRESS syndrome was established based on the RegiSCAR criteria for severe cutaneous adverse reactions. The imputability study, in collaboration with the pharmacovigilance department, implicated imatinib more strongly than azacitidine. The treatment was immediately discontinued, and he was started on dermocorticoids, leading to complete resolution of the rash and eosinophilia.

Results:

Drug rash with eosinophilia and systemic symptoms (DRESS syndrome) is a severe, potentially life-threatening drug-induced hypersensitivity reaction characterized by cutaneous eruptions, fever, diffuse lymphadenopathy, along with eosinophilia and elevated liver enzymes. The severity and potential organ damage associated with DRESS mandate withdrawing the offending drug and providing a suitable replacement.

Imatinib is an oral tyrosine kinase inhibitor. The clinical tolerance of imatinib is excellent. DRESS syndrome is a rare adverse drug reaction associated with imatinib; only ten cases have been published. However, no case of DRESS syndrome induced by azacitidine has been described. In consultation with pharmacovigilance, imatinib has been incriminated.

Conclusion:

This case highlights the importance of recognizing DRESS syndrome as a rare but serious adverse reaction to imatinib. Early identification and prompt withdrawal of the offending agent are critical to prevent potentially life-threatening complications.

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Abstract N°: 4297

Durvalumab's cutaneous footprint: a refractory lichenoid eruption

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Introduction & Objectives: Immunotherapy is a novel approach for the treatment of various types of cancer. Among its adverse effects, cutaneous manifestations are not uncommon and should not be overlooked. Durvalumab is a fully human monoclonal antibody that targets programmed death-ligand 1 (PD-L1). It is approved for the treatment of patients with unresectable stage III non-small cell lung cancer and locally advanced or metastatic urothelial carcinoma, although it is currently being investigated for other indications. Its safety profile is consistent with that of other immune checkpoint inhibitors (ICIs), with cutaneous adverse events being among the most frequently reported. The most common cutaneous toxicity described is a psoriasiform eruption. We present a rarely reported case of a treatment-resistant lichenoid eruption.

Materials & Methods: Clinical data and histopathological findings were retrospectively reviewed from the patient's medical records. Relevant images and complementary tests were included.

Results: We report the case of a 64-year-old male patient with no relevant past medical history, except for stage IIIB lung adenocarcinoma treated with chemoradiotherapy. The chemotherapy regimen included cisplatin–vinorelbine, followed by maintenance therapy with durvalumab for one year. Three months after initiating durvalumab, the patient developed bilateral forearm lesions that were unresponsive to low-dose systemic corticosteroids (10 mg/day) and topical corticosteroids. He was referred to dermatology outpatient care for further evaluation. On examination, erythematoviolaceous, slightly infiltrated plaques with Wickham's striae on the surface were observed on both forearms and the dorsum of the hands. A skin biopsy was performed with a differential diagnosis of lichenoid drug eruption or acute psoriasiform lupus. Treatment was initiated with calcipotriol/betamethasone, followed by maintenance therapy with topical tacrolimus 1%. Histopathology revealed a lichenoid dermatosis with basal vacuolar degeneration, consistent with a drug-induced etiology. The eruption persisted despite treatment; however, as it remained asymptomatic, discontinuation of immunotherapy was not considered. Complete resolution of the lesions was observed during the final follow-up visit, coinciding with the completion of durvalumab therapy.

Conclusion: Lichenoid reactions are a recognized dermatologic toxicity of immune checkpoint inhibitors, including PD-1 and PD-L1 inhibitors. However, literature specifically linking lichenoid eruptions to durvalumab remains scarce. Although these reactions can be asymptomatic, severe forms such as bullous lichenoid eruptions have been reported, which can lead to significant morbidity and may necessitate treatment interruption. Awareness of these adverse effects, along with early diagnosis and appropriate management, is essential to optimize patient quality of life and ensure continuation of effective oncologic therapy. Dermatologists should remain alert when evaluating patients undergoing durvalumab therapy, as cutaneous adverse events may be the first or only sign of immune-related toxicity.

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Abstract N°: 4313

A Rare Case of Oral Methylphenidate–Related Leukoderma in a Pediatric Patient

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Introduction & Objectives: Chemical leukoderma is an acquired hypopigmented dermatosis following repeated chemical exposure. It is often misdiagnosed as vitiligo, especially in children. While typically associated with transdermal contact, few cases are linked to systemic drugs, and those involving oral formulations are extremely rare. Transdermal methylphenidate-induced leukoderma has been reported, but oral-related cases remain very limited.

Materials & Methods: We present a female adolescent with attention deficit hyperactivity disorder who developed hypopigmented skin lesions following oral methylphenidate use.

Results: A 14-year-old female patient presented with newly developed hypopigmented patches on her forearms and hands, which had appeared over the past two weeks while she had been receiving oral methylphenidate for four months. She had no personal or family history of vitiligo, nor any previous drug use, infection, or trauma. A dermatological exam revealed two hypopigmented patches with irregular borders on the left dorsal forearm and one on the right wrist and hand. Wood lamp examination confirmed hypopigmentation. Laboratory tests including vitamin B12, iron, CBC, TSH, T3, T4, and anti-TPO were unremarkable. skin biopsy is not typically helpful in differentiating vitiligo from chemical leukoderma, so it was not performed. methylphenidate-induced leukoderma was diagnosed based on clinical features and exclusion of other causes. According to the Naranjo scale, the case was classified as a “probable” adverse drug reaction. Following psychiatric consultation, methylphenidate was discontinued and replaced with atomoxetine. Dermatological treatment included topical tacrolimus and a topical corticosteroid. Partial repigmentation was observed within two months, with no new lesions developing.

Conclusion: Chemical leukoderma presents with acquired hypopigmented macules and patches following repeated exposure to chemicals via skin contact or systemic routes such as oral administration. Methylphenidate, a presynaptic norepinephrine and dopamine reuptake inhibitor, may trigger melanocyte apoptosis by generating reactive oxygen species, as dopamine and its metabolites can induce oxidative stress leading to mitochondrial dysfunction and caspase activation. Although rare, leukoderma can occur with oral methylphenidate use. Rash, pruritus, and urticaria are common cutaneous side effects, yet chemical leukoderma should be considered when hypopigmented lesions are observed. Early drug withdrawal and topical immunomodulators or corticosteroids may yield favorable outcomes. This case contributes to the limited literature on methylphenidate-related cutaneous adverse effects and highlights the need for clinical awareness.

Abstract N°: 4401

Erythema of the folds and chemotherapy: What is the link?

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Introduction & Objectives:

Toxic erythema of chemotherapy (TEC) is a rare adverse effect of chemotherapy which usually develops two or three weeks following the initiation of chemotherapeutic drugs. It is important to recognize the clinical presentation of this entity and be aware of the chemotherapeutic agents associated with this cutaneous side effect to prevent misdiagnosis. Herein, we present a case of TEC associated with a combination of Ifosfamid and doxorubicin therapy.

Materials & Methods:

Results:

A 60-year-old patient with a medical history of locally advanced leiomyosarcoma , presented with a symmetrical indurated erythema in the popliteal fossae associated with pruritus, which appeared on the second day following the first cycle of chemotherapy. The cutaneous reaction reappeared during the second cycle administered one month later. This temporal correlation suggests a chemotherapy-induced toxic erythema. The chemotherapeutic drugs were interrupted which led to the resolution of the lesions.

Conclusion:

Toxic erythema of chemotherapy is an infrequently reported cutaneous condition. Malignant intertrigo , also known in the literature as intertriginous eruption associated with chemotherapy is one of the clinical variants of TEC. It often develops two days to three weeks after the initiation of chemotherapeutic drugs as erythematous plaques which can be scaly , crusted or erosive over intertriginous areas. A presumed pathogenesis of TEC involves the secretion of chemotherapeutic agents through eccrine glands, leading to a localized cytotoxic effect. Consequently, the most frequently involved regions are those with high eccrine gland density ,such as the palms and soles ,as well as occlusive areas including intertriginous zones. TEC typically resolves spontaneously within one to four weeks, however recurrence with re-administration of the chemotherapeutic agent is common. Diagnosis is based predominantly on clinical presentation and temporal correlation. Doxorubicin is one of the most chemotherapeutic agents frequently reported to be in cause. The cessation of the chemotherapeutic agents leads to significant improvment. However , a numerous cases reported the use of topical/oral corticosteroids with chemotherapy dose reduction. Finally, TEC is a rarely reported cutaneous adverse effect of chemotherapeutic agents. Its crucial that healthcare professionals feel comfortable identifying this skin eruption in order to initiate appropriate management.

Abstract N°: 4425

Drug Profile of Severe Toxicodermias in the Dermatology Department, Bab El Oued University Hospital, Algiers

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Introduction & Objectives: ** Toxicodermias encompass a wide range of clinical presentations; some are severe, posing a risk to life, and represent true dermatological emergencies. These include acute generalized exanthematic pustulosis (AGEP), drug reaction with eosinophilia and systemic symptoms (DRESS), and toxic epidermal necrolysis (TEN), which includes Stevens-Johnson syndrome (SJS) and Lyell's syndrome.

The objective of this study was to investigate the drug profile of patients with severe toxicodermia.

Materials & Methods:

We conducted a retrospective, descriptive, monocentric study in the Dermatology Department of Bab El Oued University Hospital, spanning a 6-year period from January 2019 to January 2025, including all cases of severe toxicodermia.

Results:

We included 51 patients with severe toxicodermia. Of these, 22 patients (43%) had acute generalized exanthematic pustulosis (AGEP); 18 patients (35%) had drug reaction with eosinophilia and systemic symptoms (DRESS); and 11 patients (21.5%) had Stevens-Johnson syndrome or Lyell's syndrome, or a combination of both.

The average age was 45.29 years, with a range from 7 to 86 years, with a female predominance (female-to-male ratio of 1.48). Amoxicillin was the most frequently implicated drug (31.8% of cases) in patients with AGEP, followed by hydroxychloroquine (27.3%), terbinafine (13.6%), and various other medications in 27.3% of patients. The most commonly implicated drugs in DRESS patients were antibiotics (38.88%) (amoxicillin +/− clavulanic acid, cefotaxime, ciprofloxacin, vancomycin, Bactrim, and azithromycin), followed by aromatic antiepileptics (33.33%) (carbamazepine, phenobarbital, lamotrigine, sodium valproate), and allopurinol (3 cases, 16.7%), with sulfasalazine and acetazolamide.

In patients with Stevens-Johnson syndrome/Lyell's syndrome or a combination, the most implicated drugs were allopurinol, hydroxychloroquine, and lamotrigine (18% for each), followed by antibiotics (sulfamide, secnidazole, cefalexin, cefotaxime, amoxicillin) (9.1% each).

In AGEP, the clinical-biological resolution occurred in an average of 15 days (range: 4-30 days), and no deaths were recorded. In DRESS, clinical-biological resolution occurred in an average of 11.06 days (range: 3-20 days), with no deaths reported. In toxic epidermal necrolysis, the mortality rate was 18.2%.

Conclusion:

Antiepileptics and antibiotics remain the most frequently implicated drugs. The hierarchy of inducing drugs varies depending on the type of toxicodermia, which may be explained by the genetic predisposition of patients, exogenous factors, and the pharmacodynamics of the drugs.

Abstract N°: 4431

Between Cure and Complication: The Ocular Price Of Antimalarials

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Introduction & Objectives:

Synthetic antimalarials (SMAs), particularly hydroxychloroquine (HCQ) and chloroquine (CQ), are widely used in the treatment of autoimmune diseases such as systemic lupus erythematosus and dermatomyositis. While generally well tolerated, their most feared complication is retinal toxicity, which may lead to irreversible vision loss. This study aimed to identify risk factors for SMA-induced maculopathy, evaluate the effectiveness of diagnostic tools, and underline the importance of interdisciplinary collaboration in long-term patient monitoring.

Materials & Methods:

This retrospective study included 216 patients treated with synthetic antimalarials (chloroquine or hydroxychloroquine) for more than two years between 2017 and 2023. Conducted jointly by the dermatology and ophthalmology departments of Mohammed VI University Hospital in Marrakech, the study involved patients followed for autoimmune systemic diseases. Inclusion criteria were long-term SMA treatment and dermatological follow-up; patients with pre-existing macular or retinal pathology were excluded. All participants underwent comprehensive ophthalmologic evaluation, including assessment of visual symptoms, fundus examination, automated 10-2 visual field testing (Humphrey), macular OCT (Spectralis), and mfERG in selected cases. Abnormal visual fields were defined by specific point losses. Data analysis was performed using Excel for statistics and Microsoft Word for documentation. Patient anonymity and confidentiality were strictly maintained in accordance with ethical medical research standards.

Results:

Toxic maculopathy was observed in 17 patients (8%), all aged over 60 years, including two with chronic renal failure. All had received high cumulative doses of synthetic antimalarials (>1000 g for hydroxychloroquine, >500 g for chloroquine) and prolonged treatment durations (>5 years for HCQ, >3 years for CQ). Reported symptoms included decreased visual acuity, visual fog, and subjective annular scotoma. Fundus examination revealed characteristic bull's eye maculopathy with annular RPE damage in all confirmed cases. OCT findings ranged from early "flying saucer" appearance to advanced macular atrophy. Visual field testing demonstrated annular or tubular defects with impaired reliability indices. A single mfERG showed perifoveal amplitude reduction. Treatment was discontinued in all cases. Functional deterioration persisted in those with macular atrophy, while early-diagnosed patients experienced stabilization and slight improvement in visual symptoms.

Conclusion:

In conclusion, synthetic antimalarials (hydroxychloroquine and chloroquine) are cornerstone treatments for many systemic diseases, especially systemic lupus erythematosus. Their therapeutic profile, combining efficacy and good tolerance, makes them a preferred option. However, retinal toxicity—although rare at recommended doses (risk <1% at 5 years)—requires heightened vigilance, particularly in at-risk patients. Systematic ophthalmologic screening and close collaboration between dermatologist and ophthalmologist are essential to prevent irreversible vision loss.

Abstract N°: 4472

A Novel Dermatologic Adverse Reaction Associated with Secukinumab: A Case of Normocomplementemic Urticarial Vasculitis

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Introduction & Objectives:

Secukinumab is a monoclonal antibody targeting interleukin-17A, approved for the treatment of dermatologic conditions such as plaque psoriasis and hidradenitis suppurativa, as well as rheumatologic disorders including, axial spondyloarthritis and psoriatic arthritis. Although generally well-tolerated, emerging reports suggest potential for novel immune-mediated adverse events.

Materials & Methods:

Case report.

Results:

We report the case of a 49-year-old female with a history of axial spondyloarthritis who presented to the emergency department with new-onset cutaneous and gastrointestinal symptoms. She had previously failed treatment with adalimumab and etanercept for disease control. Secukinumab 150 mg subcutaneously was initiated with a standard loading regimen.

By week 2 (after three injections), she developed generalized pruritus, facial edema, migratory skin lesions, and gastrointestinal symptoms including diarrhea and abdominal pain. Clinical examination revealed disseminated, circinate, urticariform lesions with central clearing, non-scaly appearance, and intense pruritus, primarily on the trunk and lower extremities. Lesions persisted for over 48 hours in the same location.

Differential diagnoses included urticaria, urticarial vasculitis, erythema annulare centrifugum, and drug-induced subacute lupus. Laboratory findings showed normal complement levels, positive ANA (1:160), and negative anti-dsDNA, ANCA, and anti-histone antibodies. Skin biopsy demonstrated dermal edema with a predominantly neutrophilic perivascular infiltrate, along with frequent leukocyte margination and infiltration of capillary walls—findings consistent with urticarial vasculitis.

Secukinumab was discontinued at week 2, prior to a definitive diagnosis. The skin symptoms resolved within two months following secukinumab discontinuation, aided by antihistamines for pruritus control. Additionally, the patient developed an intestinal perforation with peritonitis, requiring emergency segmental enterectomy. Histopathological analysis of the resected bowel revealed ulcerative enteritis of unknown etiology. The leading hypothesis explaining the event is an inflammatory bowel disease (IBD), possibly masked by prolonged anti-TNF therapy and exacerbated by secukinumab—an association supported by existing literature however, a systemic component of urticarial vasculitis contributing to the perforation cannot be excluded.

Conclusion:

To our knowledge, this is the first reported case of normocomplementemic urticarial vasculitis associated with secukinumab. Clinicians should remain vigilant for atypical dermatologic and gastrointestinal manifestations in patients receiving IL-17A inhibitors, particularly those with a complex autoimmune history.

Abstract N°: 4483

DRESS Syndrome: A Series of 18 Cases

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Introduction & Objectives:

DRESS syndrome (Drug Reaction with Eosinophilia and Systemic Symptoms) is a severe drug-induced hypersensitivity reaction that can be life-threatening, with an estimated mortality rate ranging from 2 to 6%. The severity of this condition is mainly related to systemic involvement, which can lead to multiorgan failure.

The most commonly implicated drugs are limited to a few classes, including allopurinol, antiepileptic agents, sulfasalazine, sulfadiazine, and minocycline.

The objective of our study was to clarify the epidemiological, clinical, and chronological characteristics of DRESS syndrome and to identify the different causative medications.

Materials & Methods:

This was a retrospective, descriptive study of patients hospitalized for DRESS syndrome in our department between March 2019 and October 2023. Statistical analysis was performed using IBM-SPSS software, version 25

Results:

A total of 18 patients were included (8 men and 10 women). The mean age was 48.4 years (range: 18–86 years). The responsible medication was identified in all cases.

The most frequently implicated drugs were:

- Antibiotics in 88% of cases (amoxicillin ± clavulanic acid, cefotaxime, ciprofloxacin, vancomycin, cotrimoxazole, and azithromycin);

- Aromatic antiepileptic drugs in 33.3% of cases (carbamazepine, phenobarbital, lamotrigine, and sodium valproate);

- Allopurinol in 16.7% of cases;

- Sulfasalazine and acetazolamide in one case each.

The average latency period between drug exposure and symptom onset was 12 days (ranging from 4 to 30 days).

Our findings are consistent with those reported in the literature, particularly regarding the most frequently implicated drugs (antiepileptics, allopurinol, sulfasalazine, and acetazolamide). Antibiotics, although commonly prescribed for early symptoms, were strongly implicated in our series due to the delayed onset (≥12 days), which supports a true DRESS diagnosis.

Typically, DRESS syndrome develops within the first two months after initiating the causative medication, most often between 2 to 6 weeks. However, symptoms can appear earlier and be more severe in cases of re-exposure. In our cohort, the average latency was 11 days, with extremes of 4 days for allopurinol and 30 days for

carbamazepine.

Conclusion:

DRESS syndrome is a rare but potentially life-threatening severe cutaneous adverse reaction. Viral reactivation, triggered by certain medications in genetically predisposed individuals, seems to play a central role in its pathogenesis.

Our series highlights the high frequency of facial edema, blood eosinophilia, and hepatic cytolysis as major features. Early diagnosis and prompt corticosteroid therapy remain the cornerstones of management.

Abstract N°: 4577

Tender Nodules On Palms After Amoxicillin-Clavulanate And Ciprofloxacin Treatment

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Introduction & Objectives: Neutrophilic dermatosis of the dorsal hands (NDDH) represents a rare, localized variant of Sweet's syndrome (SS). Although NDDH typically affects the dorsal aspects of the hands, palmar involvement is considered an atypical manifestation. This case is atypical presentation of a NDDH patient with palmar involvement after amoxicillin-clavulanate and ciprofloxacin treatment.

Materials & Methods: A 72 year old male presented with painful, violaceous, tender nodules on the palms for 3 days. The patient had been using amoxicillin-clavulanic acid and ciprofloxacin for the past seven days for gastroenteritis. Lesions on the palms began to appear on the fourth day of treatment. Dermatological examination revealed multiple violaceous and tender papules and nodules with no systemic symptoms. Laboratory investigations showed a total leukocyte count of $9.44 \times 10^3/\mu L$, with a neutrophilic predominance (72%). Histopatological evaluation revealed mixed-type inflammatory infiltrate predominantly composed of neutrophilic leukocytes, with occasional eosinophils in the mid-dermis. Additionally, vascular changes characterized by inflammatory cell infiltration within vascular walls were detected. Based on the clinicopathologic features, the patient was diagnosed with NDDH. Topical clobetasol propionate was used twice daily for 2 weeks and then tapered over 3 weeks. After the treatment, all lesions resolved.

Results: NDDH has been associated with hematologic disorders, solid organ tumors, sarcoidosis, seropositive arthritis, and inflammatory bowel disease. Moreover, lenalidomide, thalidomide, filgrastim and vaccinations may cause NDDH.

Cases of NDDH with palmar involvement have been rarely reported in the literature. Del Pozo et al. presented a retrospective study of eight cases of neutrophilic dermatosis of the hands. Joshi et al. described a case of a man with Felty syndrome who developed NDDH with sudden onset, painful blisters located on both the dorsal and palmar aspects of his bilateral hands after initiating filgrastim treatment. Panigrahi et al. reported a case of a man who presented with multiple tender, erythematous to slightly violaceous plaques symmetrically distributed over the palmar surfaces of both hands. Sahu presented a case of a 55 year old female with painful erythematous to violaceous, tender oedematous plaques and nodules over bilateral palms. Additionally, in a review by Micallef et al. involving 123 patients with NDDH, palmar involvement was reported in only 4.1% of cases.

Antibiotics have been well-documented as potential triggers in several reported cases of Sweet syndrome. Yuksek *et al.* described two patients who developed histiocytoid Sweet syndrome triggered by recent antibiotic use. One case was attributed to levofloxacin and the other to amoxicillin-clavulanate. Similarly, Cook *et al.* reported a case of acute febrile neutrophilic dermatosis attributed to amoxicillin.

Conclusion: These cases emphasizes how crucial it is to keep a high index of suspicion for Sweet syndrome when a patient is taking an trigerring medication, even in atypical presentations. Additionally, in the context of drug exposure, involvement of the palms may indicate an NDDH and does not rule out the diagnosis of neutrophilic dermatosis.

Table 1. Clinical characteristics of 9 cases

Study, Year of Publication	Patient	Sex	Age	Location of lesion	Treatment
Pozo et al, 2007	1	N	N	Palms	N
	2	N	N	Palms	N
	3	N	N	Palms	N
	4	N	N	Dorsal and palmar aspects of hands	N
	5	84	F	Dorsal and palmar aspects of hands	Topical corticosteroids
Sahu, 2021	6	F	55	Palms	Oral prednisolone 30 mg for 2 weeks
Joshi et al, 2023	7	M	64	Dorsal and palmar aspects of hands	No treatment beacause of comorbidities
Panigrahi et al, 2021	8	M	45	Palms	Oral prednisolone 40 mg for 1 week
K. Kodalak et al,2025	9	M	72	Palms	Topical clobetasol propionate

N:absent

Abstract N°: 4587

Lamotrigine-induced Rowell syndrome

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Introduction & Objectives:

Rowell syndrome is a rare condition characterized by the coexistence of lupus erythematosus (LE) and erythema multiforme (EM)-like lesions, accompanied by positive ANA and anti-Ro/SS-A antibodies. While its etiology remains unclear, drug-induced cases have been reported. Lamotrigine, a widely used anticonvulsant, is known to trigger autoimmune and hypersensitivity reactions, yet its role in Rowell syndrome is rarely documented. We present a case of Rowell syndrome induced by Lamotrigine, emphasizing its potential as a drug trigger for LE-like eruptions with EM features.

Materials & Methods:

A 50-year-old female with epilepsy (Grand mal seizures), treated with lamotrigine and valproate for one year was admitted in our dermatology department. The patient developed on trunk and upper extremities disseminated, painful, annular targeted lesions, resembling erythema multiforme or subacute cutaneous lupus erythematosus (SCLE), along with fatigue, chills, and fever. Due to suspicion of drug-induced reaction, lamotrigine was discontinued one week before hospitalization. Laboratory investigations included ANA screening and extended autoantibody testing at two time points. Skin biopsy and direct immunofluorescence (DIF) were performed for histopathologic and immunologic evaluation. Screening for other causes of erythema multiforme proved negative.

Results:

Initial ANA screening showed a titer of 1:320, which increased to 1:1280 after two months. Autoantibody testing revealed elevated anti-SS-A (Ro) (139 U/ml) and anti-Ro 52 (113 U/ml), with normal anti-Scl-70 and anti-SS-B levels. Histopathology confirmed features of erythema multiforme. Direct immunofluorescence (DIF) from lesional skin demonstrated annular nuclear pattern fluorescence in the epidermis with IgG deposition, consistent with lupus erythematosus. DIF from non-lesional, non-irradiated skin was negative. The patient was treated with systemic corticosteroid (40mg Methylprednisolone), with slight improvement.

Conclusion:

This case highlights the role of antiepileptic drugs as potential triggers for autoimmune dermatoses. The absence of mucosal involvement, presence of lymphocytopenia and anemia, and poor response to systemic steroids add to the complexity of the disease management. The progressive elevation of ANA titers and anti-Ro antibodies, combined with histopathological and immunofluorescence findings, emphasize the importance of early recognition and drug discontinuation to prevent disease progression and improve clinical outcomes.

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Abstract N°: 4711

Profile of drugs involved in symmetrical drug-related intertriginous and flexural exanthema (sdrife): a 10-year retrospective study

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Introduction & Objectives:

SDRIFE (Symmetrical Drug-Related Intertriginous and Flexural Exanthema), also known as “baboon syndrome,” is a rare drug reaction characterized by a symmetric erythematous rash predominantly affecting intertriginous areas and pressure points. Initially described in 1984, the syndrome is commonly associated with the systemic administration of various drugs. However, its pathophysiological mechanisms and drug profile remain incompletely understood.

Materials & Methods:

This retrospective, descriptive, monocentric study was conducted at a university hospital over a 10-year period (2014-2024). All patients diagnosed with SDRIFE based on clinical and chronological criteria, as well as reintroduction of the drug when possible, were included.

Results:

A total of 34 cases of SDRIFE were diagnosed. The mean age of patients was 46.8 ± 18.7 years, with a range from 19 to 72 years. A slight female predominance was observed, with a female-to-male ratio of 1.2. The implicated drugs varied, with antibiotics representing the most frequently involved class (56% of cases). Among them, beta-lactams were predominant, particularly amoxicillin (10 cases, 29.4%) and amoxicillin/clavulanic acid (6 cases, 17.6%). Macrolides, such as clarithromycin, were involved in 3 cases (8.8%). Nonsteroidal anti-inflammatory drugs (NSAIDs) accounted for 18% of cases, with ibuprofen (4 cases, 11.8%) and diclofenac (2 cases, 5.8%) as the most common. Anticonvulsants, particularly carbamazepine, were implicated in 12% of cases, followed by other drugs such as barbiturates (phenobarbital), proton pump inhibitors, and beta-blockers, each in smaller proportions. The median time between drug intake and the appearance of lesions was 24 hours, with variations ranging from a few hours to three days. The skin eruptions were symmetric and predominantly localized to intertriginous regions (axillae, inguinal folds, submammary folds) in 100% of cases, as well as the gluteal regions in 85%. The lesions were erythematous, occasionally pruritic, and purpuric in 6 cases (17.6%). No mucosal involvement or systemic manifestations were observed. Discontinuation of the offending drug led to improvement in all cases, with complete resolution of lesions within 3 to 7 days. Topical corticosteroid treatment was required in 90% of cases, while no patients required prolonged hospitalization or intensive care. Drug reintroduction tests, conducted in a hospital setting in 9 patients (26.5%), confirmed the diagnosis in all cases, resulting in reappearance of lesions at the same sites within a few hours of drug administration. Skin biopsies were performed in 12 patients (35.3%), revealing a superficial mixed perivascular infiltrate consisting of lymphocytes, neutrophils, and eosinophils. In 3 cases (8.8%), subcorneal pustules were observed. These features were consistent with a delayed hypersensitivity reaction.

Conclusion:

Although rare, SDRIFE is a benign drug reaction most commonly associated with antibiotics, particularly beta-lactams. Prompt recognition of clinical signs and immediate discontinuation of the offending drug allows for complete and rapid resolution of lesions, minimizing complications and recurrence risk.

Abstract N°: 4848

Case-based evaluation of a digital timeline tool for drug causality assessment in skin eruptions

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Introduction & Objectives: Drug-induced skin reactions are frequent but challenging to assess, especially in polymedicated patients. Establishing causality requires a clear chronology of drug exposures, which is often difficult to reconstruct. We report a case illustrating how a digital timeline tool facilitated the evaluation of suspected drug causality by providing a structured and standardized visualization of the patient's medication history.

Materials & Methods: A 74-year-old woman with no known allergies developed a maculopapular exanthem during hospitalization for an acute ischemic event. Initial reconstruction of her medication history relied on a hand-drawn timeline, which proved difficult to interpret due to imprecise treatment dates and inconsistent time scaling. A digital tool specifically designed for visualizing drug exposure timelines was subsequently used. Treatment periods, concomitant medications, and the date of rash onset were inputted, producing a chronological, color-coded visualization linking drug exposures to symptom onset.

Results: The digital timeline identified five drugs with temporally compatible exposure: rilmenidine, diosmectite, enoxaparin, lercanidipine, and the combination perindopril/indapamide. The structured visualization facilitated the identification of suspect drugs. Alternative causes were excluded, and the patient was referred for further allergological investigations. The tool allowed standardization of the data presentation, improved clarity for the clinical team, and supported decision-making regarding further diagnostic steps.

Conclusion: This case highlights the potential benefit of digital tools in standardizing the visualization of medication histories for suspected drug-induced skin reactions. Digitalization of causality assessment processes may enhance diagnostic accuracy and workflow efficiency in complex clinical scenarios.

Abstract N°: 4908

Methotrexate-induced acneiform eruption: A rare adverse reaction

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Introduction & Objectives:

Methotrexate is a folate antagonist widely used as a chemotherapeutic and immunosuppressive agent, commonly prescribed for autoimmune diseases and malignancies such as leukemias. While its well-documented side effects include hepatotoxicity, myelosuppression, and mucositis, it can also induce a range of dermatological reactions. However, cases of methotrexate-induced acneiform eruptions remain rare in the literature.

Materials & Methods:

We report a case of an acneiform eruption induced by Methotrexate in a 16-year-old male patient.

Results:

A 16-year-old patient undergoing treatment for acute lymphoblastic leukemia with a regimen including high-dose Methotrexate and Purinethol, developed a pruritic eruption involving the face, trunk, back, thighs, and both legs following the first Methotrexate injection. The lesions resolved spontaneously but recurred upon each reintroduction of the treatment. On physical examination, the patient presented monomorphic acne lesions made of inflammatory papules with post inflammatory pigmentation, without pustules or comedones. Given the clinical overlap between acneiform eruption and lichenoid drug reaction, a skin biopsy was performed to clarify the diagnosis. Histologically, the hair follicle is destroyed and filled with an inflammatory infiltrate mainly composed of neutrophils, often degenerated and mixed with fibrin. This infiltrate extends into the surrounding dermis. Notably, there is an absence of a lichenoid infiltrate, which supports the diagnosis of acneiform eruption. Following remission of his leukemia and discontinuation of treatment, the patient showed no recurrence of cutaneous lesions. These findings, along with the clinical course and histopathological confirmation, supported the diagnosis of methotrexate-induced acneiform eruption.

Conclusion:

Methotrexate-induced acneiform eruptions, though rare, should be considered in patients undergoing treatment with this drug, especially when typical acne triggers are absent. The distinct temporal relationship between the initiation of methotrexate therapy and the onset of acneiform lesions, coupled with the resolution of symptoms following drug discontinuation, underscores the importance of recognizing this adverse effect. Clinicians should be mindful of this uncommon dermatological reaction, as early diagnosis and management can improve patient outcomes and prevent unnecessary treatment interruptions.

Abstract N°: 4917

Drug-Induced Subacute Cutaneous Lupus Erythematosus in a Patient with Sjögren's Syndrome: A Diagnostic Challenge

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Introduction & Objectives:

Drug-induced cutaneous reactions in patients with pre-existing autoimmune diseases, such as Sjögren's syndrome, pose significant diagnostic challenges. We present a case highlighting the importance of thorough drug history analysis and the utility of dermoscopy in differential diagnosis.

Materials & Methods:

A 79-year-old woman with Sjögren's syndrome in long-term remission presented with pruritic eruptions on the upper trunk and extremities. Medical history revealed self-escalation of an anxiolytic agent (mebicar) without medical supervision. Dermatological examination and dermoscopic evaluation were performed. A repeat skin biopsy was taken for histopathological analysis, and differential diagnoses were considered.

Results:

Clinical examination revealed erythematous papules on the chest, shoulders, and forearms, with a tendency to coalesce. Dermoscopy demonstrated linear and arborizing vessels, white structureless areas, scaling, and erosions. Histopathology showed a lichenoid, erythematous inflammatory infiltrate. Cutaneous lymphoproliferative disease was excluded. Treatment involved increasing systemic glucocorticosteroid therapy to 20 mg daily and discontinuing the anxiolytic agent, resulting in near-complete resolution of skin lesions and pruritus. Follow-up showed only a few residual erythematous papules, with linear vessels observed on dermoscopy.

Conclusion: **

The final diagnosis was drug-induced subacute cutaneous lupus erythematosus (DI-SCLE). This case emphasizes the need for meticulous drug history assessment in patients with autoimmune backgrounds and highlights the role of dermoscopy in identifying characteristic features that aid in accurate clinical evaluation and management.

Abstract N°: 4922

Cutaneous Adverse Reactions under the Anti-CCR4 Antibody Mogamulizumab in a Patient with Stage IIIA Erythrodermic Mycosis Fungoides: A Case Report

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Introduction & Objectives:

Mogamulizumab is an anti-CCR4 monoclonal antibody approved for the treatment of relapsed or refractory Mycosis fungoides (MF) and Sézary syndrome. Although effective, its administration is associated with specific cutaneous adverse events. This case report is about a patient with erythrodermic MF, focusing on the therapeutic response to Mogamulizumab and associated cutaneous reactions, aiming to underline the importance of recognizing and managing these side effects.

Materials & Methods:

We present the case of a 53-year-old patient diagnosed in March 2023 with Stage IIIA erythrodermic MF (T4, N0, M0, B0), initially resistant to phototherapy, topical therapy, and systemic treatments (Brentuximab, Methotrexate). Due to disease progression, Mogamulizumab 80 mg was initiated in August 2023, administered weekly for 4 weeks, followed by biweekly maintenance. Diagnostic follow-up included clinical assessments, peripheral blood immunophenotyping, CT imaging, Dermatology Life Quality Index (DLQI), and pruritus Numeric Rating Scale (NRS) scoring.

Results:

During treatment, significant clinical improvement was achieved: near-complete remission of initially diffuse alopecia, substantial reduction of cutaneous erythema and recalcitrant pruritus, documented by clinical examination and a DLQI score of 9, pruritus NRS score of 2–1. Peripheral blood immunophenotyping revealed dynamic changes: initial Sezary cell count of 10.7% (May 2023) decreased to 0.6% (September 2024). CT scans revealed stable bilateral inguinal lymphadenopathy (histopathologically confirmed as reactive lymph nodes), with no evidence of systemic progression.

However, the patient developed cutaneous side effects attributed to Mogamulizumab: a transient, moderate erythemato-squamous rash, predominantly located on the trunk and extremities, and non-cicatriceal circumscribed alopecic plaques. The rash, initially disseminated and responsible only to oral corticosteroids, evolved into smaller plaques resolving spontaneously within 4–5 days, while the developed alopecia areata, considerably improved under topical corticosteroids.

Given the favorable disease control and manageable adverse effects, Mogamulizumab therapy was continued alongside symptomatic treatment (emollients, topical corticosteroids, antihistamines, intermitent low-dose prednisone).

Conclusion:

Mogamulizumab induced significant clinical improvement in a patient with refractory Stage IIIA erythrodermic MF. Although cutaneous adverse reactions such as transient rash and alopecia areata occurred, they were manageable and did not require treatment discontinuation. Recognizing and addressing these adverse events is crucial to maintaining patient adherence and optimizing therapeutic outcomes in advanced MF.

EA DV CONGRESS
PARIS 17-20 SEPTEMBER 2025
EUROPEAN ACADEMY OF DERMATOLOGY & VENEREOLOGY

Abstract N°: 4932

One man's meat is another man's poison: High incidence ofsevere cutaneous drug reactions induced by Enfortumab Vedotin

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Introduction & Objectives:

Enfortumab vedotin (EV), an antibody-drug conjugate targeting nectin-4 and linked to monomethyl auristatin E, has shown efficacy in metastatic urothelial carcinoma. However, emerging evidence has identified severe cutaneous adverse events (cAEs), particularly Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN). To date, no comprehensive analysis of cAEs associated with EV has been reported. The objective of this study is to investigate the incidence, onset timing, and risk factors associated with cAEs, particularly SJS/TEN, in patients treated with EV, using data from the U.S. Food and Drug Administration's Adverse Event Reporting System (FAERS).

Materials & Methods:

This retrospective study analyzed data from January 1, 2020, to November 1, 2024, covering 2,736 patients. Disproportionality analysis and positive signal detection were used to assess the correlation between EV and AEs. Logistic regression was employed to evaluate risk factors associated with the occurrence of AEs.

Results:

Among the 2,736 patients analyzed, a total of 6,520 AEs were reported. Skin-related AEs were the most frequent, accounting for 1,027 cases (37.54%), with SJS/TEN occurring in 8.59% of cases (Figure 1). Our analysis shows most cAEs occurred within 30 days of EV treatment start, with SJS/TEN onset peaking at 14 days (Figure 2). Disproportionality analysis showed that severe cAEs had higher positive signal values, with the ROR of 41.76, PRR of 39.65, MHRA of 5232.16, BCPNN of 5.28, EBGM of 39.02 in SJS/TEN (Figure 3). Risk factors for skin-limited AEs included prior immunotherapy (adjusted odds ratio [aOR] 1.90, 95% confidence interval [CI] 1.15-3.15; p=0.013), male gender (aOR 1.51, 95% CI 1.19-1.91; p<0.001), and chemotherapy (aOR 1.51, 95% CI 1.19-1.91; p<0.001). Increased risks of SJS/TEN were associated with older age (65-80 years: aOR 2.06, 95% CI 1.15-3.68; p=0.015; >80 years: aOR 2.17, 95% CI 1.04-4.53; p=0.038) and hypertension (aOR 1.65, 95% CI 1.03-2.65; p=0.038).

Conclusion:

Our study highlights a higher incidence of cAEs during EV treatment, particularly severe cAEs such as SJS/TEN. These findings underscore the need for vigilant monitoring and proactive management of these AEs in clinical practice, especially in high-risk populations.

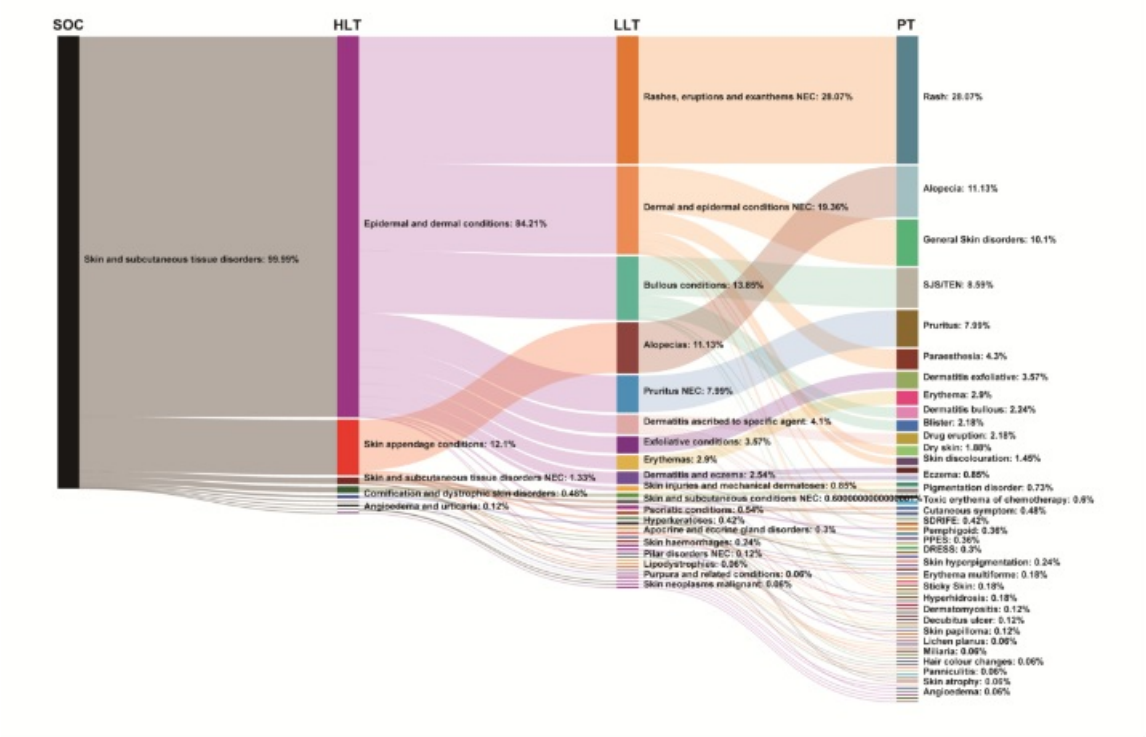

Figure 1 Types of cutaneous adverse events by MedDRA classification

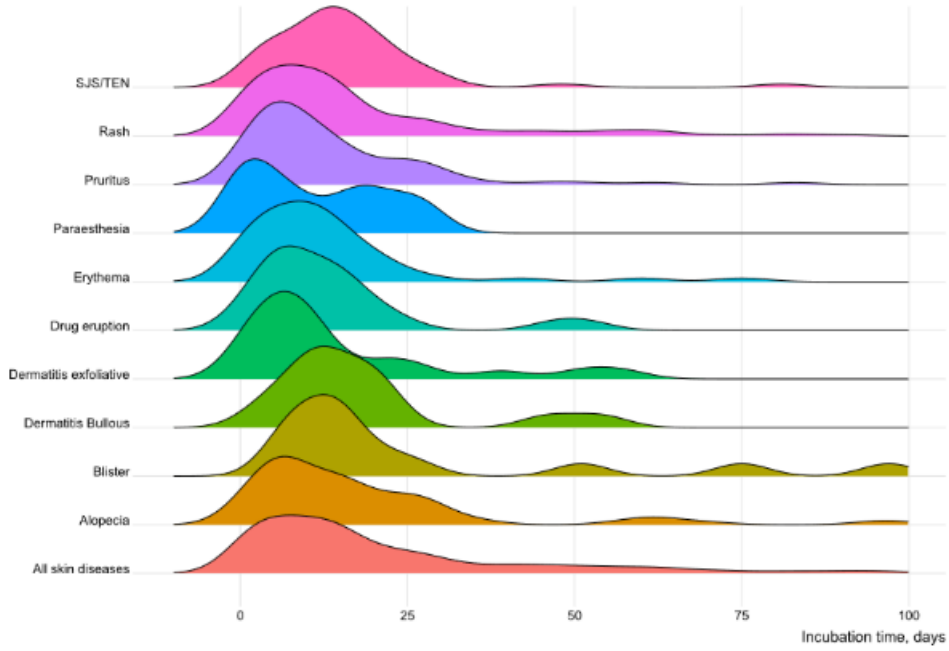

Figure 2 Time-to-onsets of top 10 cutaneous adverse events

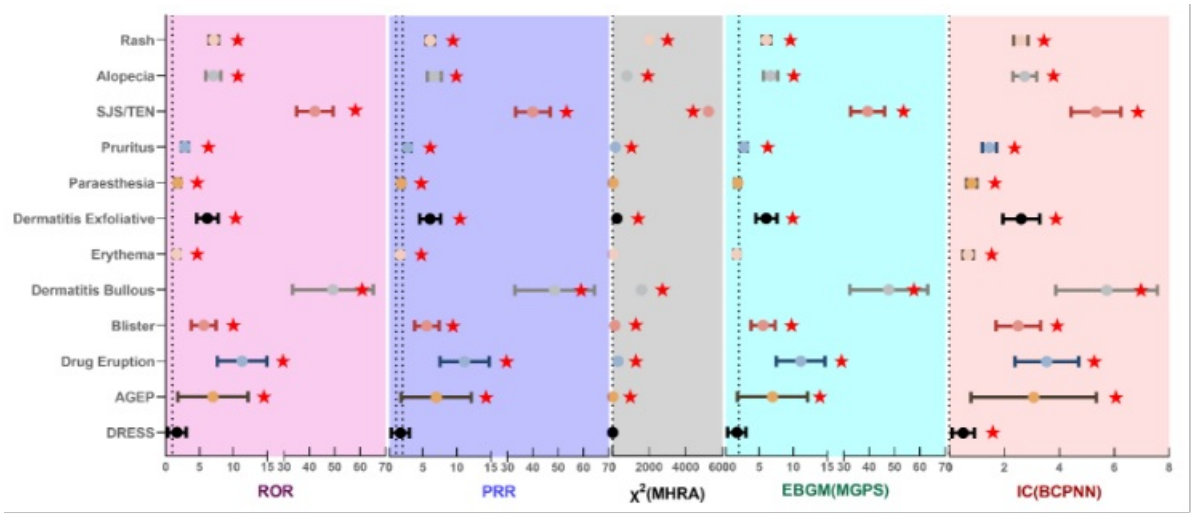

Figure 3 Signal strength of top 10 cutaneous adverse events by different methods. The stars mean the positive signals. ROR, reporting odds ratio; PRR, proportional reporting ration; MHRA, Method used by Medicines and Healthcare Products Regulatory Agency; MGPS, multi-item gamma Poisson shrinker; EBGM, empirical Bayesian geometric mean; BCPNN, Bayesian confidence propagation neural network; IC, information component.

EADV CONGRESS
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Abstract N°: 4953

First Report of Cabozantinib-Associated Toxic Epidermal Necrolysis

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Introduction & Objectives:

Toxic epidermal necrolysis (TEN) is a rare, life-threatening mucocutaneous reaction, most commonly drug-induced. Cases of TEN have been reported in patients treated with immune checkpoint inhibitors such as nivolumab and ipilimumab in the context of advanced malignancies. However, TEN solely attributed to cabozantinib monotherapy has not been previously described. We report the first documented case of TEN directly associated with cabozantinib in the absence of concomitant immunotherapy.

Materials & Methods:

A 54-year-old male diagnosed with stage IV renal carcinoma, previously treated with nivolumab and ipilimumab—discontinued due to immune-mediated pancreatitis and splanchnic thrombosis—subsequently initiated cabozantinib monotherapy, developing painful gluteal ulcers. Skin biopsy was consistent with drug-induced vasculopathy, and lesions resolved after treatment discontinuation. Following disease progression, cabozantinib was reintroduced at a reduced dose. A few days later, the patient developed fever, diffuse erythematous rash, mucosal involvement, and rapid progression to epidermal detachment affecting over 30% of the body surface area.

Results:

Skin biopsy revealed full-thickness epidermal necrosis, minimal dermal inflammation, no acantholysis, and negative direct immunofluorescence, findings consistent with drug-induced TEN. The ALDEN algorithm indicated probable causality for cabozantinib. The patient had a SCORTEN score of 4, with an estimated mortality of 58%. Treatment with intravenous methylprednisolone pulses and immunoglobulins was initiated. Additional immunosuppressants were avoided due to the advanced oncologic disease and prior severe immune-related toxicity. ICU admission was ruled out based on clinical criteria. The patient ultimately died due to multiorgan failure.

Conclusion:

Although cases of TEN have been reported in patients receiving nivolumab plus cabozantinib, immunotherapy has been identified as the causative agent in all such instances. This case strengthens the evidence for cabozantinib as a direct cause of TEN. The clear temporal sequence, histopathologic findings, and absence of alternative drugs support this association. Early recognition of cutaneous toxicity signs related to cabozantinib is crucial, and re-exposure should be avoided in sensitized patients. We contribute novel evidence of a severe cutaneous adverse reaction associated with cabozantinib monotherapy.

Abstract N°: 4968

Paradoxical lichenoid reaction during dupilumab treatment- A case report and literature review

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Introduction & Objectives:

Paradoxical reactions (PRs) are defined as the onset of new or the exacerbation of existing immune-mediated disorders following the initiation of targeted biologic therapies. Dupilumab, an IL-4Rα inhibitor, is effective for atopic dermatitis (AD) but may trigger PRs. While paradoxical psoriasiform or head and neck dermatitis are common, lichenoid reactions remain rare.

Materials & Methods:

We present a case of dupilumab-induced paradoxical lichenoid reaction with erythroderma and review existing literature to identify clinical patterns.

Results:

We report a 64-year-old male with refractory AD who developed erythroderma and lichenoid reaction four months after dupilumab initiation. Histopathology revealed lichenoid interface dermatitis with parakeratosis and infiltration of lymphocytes, histiocytes and eosinophils. Immunohistochemistry confirmed T-cell and histiocytic proliferation. Symptoms resolved after dupilumab cessation and systemic therapy. A literature review of seven cases identified three histopathologic subtypes: classic lichen planus (LP), lichenoid drug eruption (LDE), and lichenoid granulomatous reaction (LGR), with a median onset of 29.7 weeks. Management universally included dupilumab discontinuation (85.7%), with systemic therapies such as glucocorticoids (57.1%), JAK inhibitors (28.6%), and cyclosporine or methotrexate (14.3%) employed.

Conclusion:

These findings emphasize the need for heightened vigilance regarding paradoxical reactions during biologic therapies. In addition, AD is associated with higher risks of cutaneous T-cell lymphoma (CTCL), which could also mimic eczematous skin lesions or erythroderma, requiring skin biopsy for accurate diagnosis.

Abstract N°: 4987

Polymorphic Eruption and Lichen Planopilaris-Like Hair Loss Induced by Ixekizumab

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Introduction & Objectives:

Ixekizumab, an IL-17A inhibitor, is used for psoriasis, psoriatic arthritis, ankylosing spondylitis, and axial spondyloarthritis. Common cutaneous side effects include injection site reactions and tinea infections; paradoxical eczema is less frequently reported. We present a case of polymorphic eruptions and alopecia associated with ixekizumab in a psoriasis patient.

Case Report:

A 57-year-old woman with palmoplantar and large plaque psoriasis (PASI 15.1, DLQI 24) involving feet, groin, axillae, and scalp, was started on ixekizumab in February 2024 after secondary failure on adalimumab. Her background included hypertension and depression (on sertraline). Within two weeks she noticed clearance of palmoplantar and scalp psoriasis.

However, a month after starting ixekizumab, she developed a sore throat with periorbital swelling, ocular discharge, crusting, and weeping around the ears and cervical lymphadenopathy with new rashes. Clinical examination revealed dermatitis with greasy, adherent scale affecting the face, neck, annular erythema with advancing scaly edge on the chest, upper back, and forearms, as well as crusting and weeping behind the ears and along the posterior scalp line, and a draining axillary boil which grew a doxycycline sensitive staphylococcus aureus. Clinical findings were interpreted as severe seborrhoeic dermatitis with possible tinea corporis and secondary bacterial infection and was treated with two-week delay in ixekizumab injections, oral itraconazole, doxycycline and antimicrobial washes. Mycology was negative.

Despite partial improvement, two months later she developed extensive scaly alopecia on the posterior, vertex and parietal scalp with dermoscopic signs of scarring and dermatitis on the neck, upper chest, and back, along with eruptive seborrhoeic keratoses. Ixekizumab was stopped.

Investigations ruled out underlying malignancy and dermatomyositis. Myositis antibodies, CT chest/abdomen/pelvis, breast screening, and cervical smear were all negative. Scalp biopsy showed features of evolving lichen planopilaris (LPP). Skin biopsies revealed eczematous changes and seborrhoeic keratosis.

Neck, chest, and forearm dermatitis resolved with topical betamethasone dipropionate/clotrimazole. Due to rapid hair loss, she was started on hydroxychloroquine, doxycycline, diprosalic, and clobetasol scalp applications.

After one month, dermatitis settled, keratoses resolved, and hair loss stabilized. Psoriasis recurred, prompting initiation of risankizumab (IL-23A inhibitor), which led to psoriasis clearance, rash resolution, and sustained hair regrowth - even after stopping hydroxychloroquine and doxycycline.

Conclusion:

Ixekizumab, while effective for clearing psoriasis, may trigger severe dermatologic side effects. Our patient developed polymorphic rashes, seborrhoeic keratoses, and LPP-like alopecia. The Naranjo score of 6 suggests a probable drug reaction. Although ixekizumab has not been previously linked to LPP, it has been associated with

lichenoid drug reactions, alopecia areata, folliculitis decalvans, and psoriasiform alopecia. Eruptive seborrhoeic keratoses have only been reported once before with ixekizumab and resolved upon discontinuation in our case. Given the absence of biomarkers to predict these rare adverse events, this case highlights the role of personalised empirical class switching, based on clinical response, to optimise outcomes.

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Abstract N°: 5092

Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis: A Series of 11 Cases

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Introduction & Objectives: Epidermal necrolysis is a rare, severe cutaneous adverse drug reaction that encompasses Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN).

The objective of our study was to describe the epidemiological, clinical, and prognostic characteristics of this pathology, as well as to identify the most frequently implicated drugs

Materials & Methods: We conducted a retrospective descriptive study of SJS and TEN cases hospitalized in our department between March 2019 and January 2024.

All patients who met international diagnostic criteria for SJS/TEN were included in the study.

Results: A total of 11 cases were identified, including 4 cases of SJS, 6 cases of TEN, and 1 overlap syndrome, accounting for 17% of all drug-induced skin reactions.

The mean age was 45 years (range: 7–80 years), with a clear female predominance (80%).

Among the patients, 54.5% were hypertensive and 18% had pre-existing dermatological conditions, such as Rowell syndrome or bullous pemphigoid.

The most frequently implicated drugs were allopurinol, hydroxychloroquine, and lamotrigine, each accounting for 18% of cases, followed by antibiotics (including sulfonamides, secnidazole, cephalexin, cefotaxime, and amoxicillin), each responsible for 9.1% of cases.

The mean latency period between drug intake and symptom onset was 9 days (range: 1–21 days).

Mucosal involvement was present in 63.6% of patients, and cutaneous lesions were pruritic in 45.5% of cases.

Biological abnormalities were noted in the form of hepatic cytolysis (27.27%), functional acute kidney injury (18.2%), electrolyte disturbances (18.2%), and hypoproteinemia (9.1%).

In 36.4% of patients, progression to the more severe TEN form was observed.

Management consisted of immediate withdrawal of the suspected drug, local wound care (cutaneous and mucosal), correction of fluid and electrolyte imbalances, and systemic corticosteroid therapy in 80% of cases. Intravenous immunoglobulins (IVIG) were administered in a single TEN case at a dosage of 2 g/kg over 5 days.

The mortality rate in this series was 18.2%.

Conclusion: SJS and TEN are rare but severe cutaneous drug reactions that can be life-threatening. Further studies are needed to clarify prognostic factors and optimize therapeutic strategies.

Abstract N°: 5096

Acute Generalized Exanthematous Pustulosis (AGEP): A Series of 22 Cases

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Introduction & Objectives: Acute Generalized Exanthematous Pustulosis (AGEP) is a rare but severe cutaneous drug reaction, characterized by an abrupt onset typically following an infectious episode or drug exposure.

The main causative agents are now well established, including pristinamycin, ampicillin, quinolones, hydroxychloroquine, and antibacterial sulfonamides.

The aim of this study was to identify the most implicated drugs and to describe the clinical and evolutionary profile of patients hospitalized for AGEP

Materials & Methods: We conducted a descriptive retrospective study of cases of drug-induced AGEP hospitalized in our department between March 2019 and October 2023.

Results: A total of 22 patients were enrolled, with a male-to-female sex ratio of 0.17 (14% male, 86% female). The mean age was 42.45 years (range: 8–72 years), and 80% of patients were under 50 years of age. One pediatric case was identified.

AGEP was attributed to amoxicillin in 31.8% of cases, hydroxychloroquine in 27.3%, terbinafine in 13.6%, ciprofloxacin in 9.1%, and to metronidazole, cefixime, cephalexin, and diltiazem in 4.5% of cases each. Drug causality was considered highly suggestive in 84% of cases, according to the French causality assessment method.

The mean latency period between drug introduction and symptom onset was ≤ 10 days in 17 patients, with a median delay of 6 days. Analytical evaluation demonstrated a statistically significant difference in the onset of symptoms between drug groups, with a shorter latency period for amoxicillin (1–7 days).

Clinically, patients presented with a generalized edematous erythema covered by non-follicular pustules. Lesions involved the trunk in 93.7% of cases, the limbs in 81.2%, and the face in 25%. Flexural areas (axillary, inguinal, and inframammary folds) were most frequently affected. Fever was present in 90% of cases. Laboratory investigations consistently revealed elevated C-reactive protein (CRP) and neutrophilic leukocytosis.

Skin biopsy was performed in 10 patients, revealing spongiform infundibular pustules, necrotic keratinocytes, and superficial dermal edema. Mucosal involvement was documented in 10% of cases. Pruritus was reported in 50% of cases.

Systemic corticosteroid therapy (0.5–1 mg/kg/day), combined with topical corticosteroids and adequate hydration, resulted in favorable outcomes in the majority of cases. Two patients developed acute renal failure, three had electrolyte disturbances, and cutaneous worsening was observed in seven patients.

Complete clinico-biological resolution was achieved in an average of 15 days (range: 4–30 days), with no fatalities reported.

Conclusion: AGEP is drug-induced in more than 90% of cases, most frequently following antibiotic exposure. In this series, hydroxychloroquine- and terbinafine-induced AGEP were rare but noteworthy.

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Abstract N°: 5097

Drug Profile of Severe Toxicodermias in the Dermatology Department, Bab El Oued University Hospital, Algiers

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Introduction & Objectives: Toxicodermias encompass a wide range of clinical presentations; some are severe, posing a risk to life, and represent true dermatological emergencies. These include acute generalized exanthematic pustulosis (AGEP), drug reaction with eosinophilia and systemic symptoms (DRESS), and toxic epidermal necrolysis (TEN), which includes Stevens-Johnson syndrome (SJS) and Lyell's syndrome.

The objective of this study was to investigate the drug profile of patients with severe toxicodermia.

Materials & Methods: We conducted a retrospective, descriptive, monocentric study in the Dermatology Department of Bab El Oued University Hospital, spanning a 6-year period from January 2019 to January 2025, including all cases of severe toxicodermia

Results:

We included 51 patients with severe toxicodermia. Of these, 22 patients (43%) had acute generalized exanthematic pustulosis (AGEP); 18 patients (35%) had drug reaction with eosinophilia and systemic symptoms (DRESS); and 11 patients (21.5%) had Stevens-Johnson syndrome or Lyell's syndrome, or a combination of both.

The average age was 45.29 years, with a range from 7 to 86 years, with a female predominance (female-to-male ratio of 1.48). Amoxicillin was the most frequently implicated drug (31.8% of cases) in patients with AGEP, followed by hydroxychloroquine (27.3%), terbinafine (13.6%), and various other medications in 27.3% of patients. The most commonly implicated drugs in DRESS patients were antibiotics (38.88%) (amoxicillin +/− clavulanic acid, cefotaxime, ciprofloxacin, vancomycin, Bactrim, and azithromycin), followed by aromatic antiepileptics (33.33%) (carbamazepine, phenobarbital, lamotrigine, sodium valproate), and allopurinol (3 cases, 16.7%), with sulfasalazine and acetazolamide.

In patients with Stevens-Johnson syndrome/Lyell's syndrome or a combination, the most implicated drugs were allopurinol, hydroxychloroquine, and lamotrigine (18% for each), followed by antibiotics (sulfamide, secnidazole, cefalexin, cefotaxime, amoxicillin) (9.1% each).

In AGEP, the clinical-biological resolution occurred in an average of 15 days (range: 4-30 days), and no deaths were recorded. In DRESS, clinical-biological resolution occurred in an average of 11.06 days (range: 3-20 days), with no deaths reported. In toxic epidermal necrolysis, the mortality rate was 18.2%.

Conclusion: Antiepileptics and antibiotics remain the most frequently implicated drugs. The hierarchy of inducing drugs varies depending on the type of toxicodermia, which may be explained by the genetic predisposition of patients, exogenous factors, and the pharmacodynamics of the drugs

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Abstract N°: 5103

Finding the Incriminated Drug in a Case of SDRIFE Syndrome: A Challenge Still to Date

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Introduction & Objectives:

SDRIFE (Symmetrical Drug-Related Intertriginous and Flexural Exanthema), previously called Baboon Syndrome, is a rare, benign drug reaction. It typically occurs within a few hours to a few days after exposure to a drug, usually for the first time, although recurrence is possible with subsequent drug exposures. Several risk factors are known, including a medical history of drug allergies, heart, kidney, and thyroid diseases, and finally age

Materials & Methods: A case report

We report the case of an 83-year-old patient recently diagnosed with type 2 diabetes, who was treated with Metformin. He was admitted for an extensive rash affecting the buttocks, inguinal, cervical, and palpebral areas. The symptoms initially started a month prior when he presented with an inguinal rash, which was initially diagnosed as infected eczema. He was treated with Fluconazole, Clav-amoxicillin, and steroids, leading to partial regression of the symptoms over the following weeks. However, a few days prior to his admission, he noted the rash extended to the aforementioned areas. Further investigation by the pharmacovigilance department revealed that the patient also reported occasional stomach pain, which he treated on his own with Omeprazole. Additionally, he suffers from intermittent gout attacks for which he takes Colchicine or Allopurinol, although he does not have a clear timeline for each intake.

Results:

The eruption meets all SDRIFE diagnostic criteria, and the biopsy showed the typical histopathological findings usually seen in this syndrome. Considering the drugs previously reported in the literature as potential causes of SDRIFE, Fluconazole, Clav-amoxicillin, and possibly Omeprazole can be ruled out since these medications were previously taken with a favorable outcome; however, colchicine was only recently introduced in an intermittent manner, and as such, it is the most likely culprit. To our best knowledge this might be the first reported case of SDRIFE syndrome induced by Colchicine.

Identifying the incriminated drug in a polymedicated patient proved to be a real challenge. Many elderly patients today take more than two or three prescribed medications, along with occasional over-the-counter drugs such as painkillers, proton pump inhibitors, benzodiazepines, and even traditional medicine preparations. Additionally, if they develop a bacterial infection, they are likely to take one or more antibiotics. As a result, we are increasingly encountering situations where determining the culprit drug becomes a significant challenge.

Patch tests are useful in identifying the offending drug in SDRIFE. In Häusermann et al.'s series, patch tests were performed in 23 of 42 SDRIFE cases, with half (12) showing positive results. Other investigations, such as prick tests, intradermal tests, human basophil degranulation tests, and lymphocyte activation tests, have yielded unsatisfactory results in other cases. Oral provocation tests, when feasible, remain the gold standard, but caution must be exercised, as some patients may not consent to them.

Conclusion: A better understanding of the pathology of this drug reaction could help optimize diagnostic tools

and ADR (Adverse Drug Reaction) probability scales tailored to this specific syndrome in the future.

Abstract N°: 5202

„Reccurent erythema fixum bullosum with acral involvment : a case report''

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Introduction :

Erythema fixum bulosum is a rare subtype of fixed drug eruption characterized by recurring, localized erythematous and bullous lesions, commonly triggered by medications. Acral involvement is uncommon, and recurrence in the same regions poses diagnostic and therapeutic challenges.

Case Presentation:

We report the case of a 50-year-old male with a 4–5-day history of painful erythematous and bullous lesions on the dorsal aspects of both feet and hands. The lesions recurred in the same locations, with a documented history of similar episodes triggered by medications such as paracetamol and possibly other agents. Histopathological examination of a fresh lesion confirmed the diagnosis of erythema fixum bulosum. Treatment with systemic corticosteroids, loratadine, and topical agents resulted in clinical improvement.

Discussion:

Recurrent erythema fixum bulosum with acral distribution is extremely rare. Recognition of the triggering medication is essential to prevent further episodes. The histological findings supported the diagnosis and excluded differential diagnoses such as bullous pemphigoid or erythema multiforme. The case underscores the importance of accurate anamnesis, histopathological correlation, and medication avoidance

Conclusion:

This case highlights a rare recurrent presentation of erythema fixum bulosum localized to the hands and feet, with histological confirmation. Early diagnosis, drug withdrawal, and appropriate therapy led to resolution of symptoms and prevention of complications.

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Abstract N°: 5240

Misleading Blisters: 2 Cases of Generalized Bullous Fixed Drug Eruption Mimicking TEN

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Introduction & Objectives:

Generalized bullous fixed drug eruption (GBFDE) is a rare and severe form of fixed drug eruption. It is the only dermatosis with an exclusively drug-induced cause. Common medications such as non-steroidal anti-inflammatory drugs, paracetamol and antibiotics are the most common inductors. The delay varies from 1 to 4 days, and is increasingly reduced in recurrent forms. The main differential diagnosis clinically and histologically is toxic epidermal necrolysis. Through these two challenging cases, we will highlight key clinical and histopathological features that help differentiate GBFDE from TEN, given the implications for prognosis and management.

Materials & Methods:

We describe two patients who presented with acute-onset, widespread bullous eruptions clinically suggestive initially of TEN. Detailed clinical evaluations were performed, including lesion mapping and mucosal involvement assessment. A thorough medication history was obtained to identify the causative agents and prior similar episodes and drug exposure. Histopathological examination was conducted. The diagnosis of GBFDE was established based on the combination of recurrent fixed lesion sites, histological features, and rapid resolution following drug withdrawal.

Results:

Patient 1: 76-year-old man with diabetes and ischemic heart disease. Hospitalized for painful oozing lesions that appeared 24 hours after being started on Amoxicillin-clavulanic acid and Ciprofloxacin for pneumonia. Clinical examination revealed a patient in good overall general condition, with multiple well-limited erosive plaques of variable size, genital erosions and cheilitis, with no involvement of oral mucosa. Paraclinical examinations initially performed in search of systemic involvement didn't reveal any abnormality. The course was marked by the onset of renal failure 2 days after the patient was put on Aciclovir for a herpetic superinfection, with a good evolution.

Patient 2: 51-year-old woman with a history of 2 episodes of skin eruption several years ago following undocumented medication for back pain. She was admitted to our department with oozing skin lesions that appeared few hours after self-medication with a non-steroidal anti-inflammatory drug for dental pain. On clinical examination, the patient was in good general condition, with multiple erosive plaques with wet linen detachment at the edges and aphthoid erosions on the tongue. Nikolsky sign was negative in perilesional skin. Standardized workup protocol and thorough investigations were performed and didn't reveal any abnormality. Histology showed junctional detachment, keratinocytic necrosis and pigment incontinence in both patients.

Conclusion:

GBFDE clinically manifests as well-limited, asymmetrical erosive plaques separated by wide intervals of healthy skin, leaving a residual pigmentation that is unavoidable and difficult to treat. The average skin surface affected is 20%. Mucosal involvement is unipolar and mild. Good general condition is maintained. There is no associated systemic involvement. Hospitalization is required when the skin surface exceeds 10%. The prognosis is generally

good, except in the elderly, fragile population, and in recurrent forms. A better understanding of this type of drug eruption prevents the reintroduction of causing drugs, which are responsible for more severe, more extensive forms, occurring in increasingly shorter periods of time, sometimes just in few hours.

Abstract N°: 5268

Nail Toxicity Induced by Anticancer Treatments

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Introduction & Objectives:

Adverse effects caused by anticancer drugs are frequently encountered in dermatological practice, particularly changes in the nail unit, which can sometimes be associated with pain and functional impairment. Through this prospective study, we aimed to analyze these manifestations to improve understanding and enable early diagnosis.

Materials & Methods:

This is a prospective, all patients followed for cancer and undergoing anticancer treatments presenting with a nail toxicity were included in this study.

Results:

46 patients, with a clear female predominance and a male-to-female ratio of 0.28. The average age was 54 years. The average time to symptom onset after drug administration was one month. The most frequently implicated drugs were taxanes (Docetaxel and Paclitaxel) in 41% of cases. Among patients receiving anticancer treatments, 35% developed nail alterations, with melanonychia being the most common (50% of cases). Melanonychia was mainly associated with taxanes (68% of cases) and anthracyclines (32% of cases). Onycholysis was observed in 37% of cases, and nail fragility was noted in one-quarter of patients, particularly those treated with taxanes. Pyogenic granulomas were observed in 22% of cases, mainly in patients receiving targeted therapies and immunotherapy. Beau’s lines, onychomadesis, Mees’ lines, trachyonychia, and other nail abnormalities were also identified.

Conclusion:

Nail changes induced by anticancer treatments are well-recognized side effects that can lead to significant morbidity and impact patients’ quality of life. Currently, data on the management of nail toxicity remain scarce, highlighting the need for further research to better define the underlying mechanisms and develop effective prevention and treatment strategies.

Abstract N°: 5354

NUDT15 and TPMT Pharmacogenomic Testing for Azathioprine-Induced Adverse Reactions in Immune-mediated Skin Diseases

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Introduction & Objectives: While thiopurine S-methyltransferase (TPMT) variants are known to correlate with azathioprine (AZA)-induced adverse reactions (AEs), nucleoside diphosphate-linked moiety X motif 15 (NUDT15) variants have been reported to be more prevalent and strongly associated with AZA toxicity in Asians. However, the association between these genetic variants in immune-mediated skin diseases (IMSDs) remains limited. We aim to evaluate the prevalence of NUDT15 and TMPT genetic variants and their association with AZA-induced AEs in Thai patients with IMSDs.

Materials & Methods: A cross-sectional study was conducted in patients with IMSDs (e.g., autoimmune bullous diseases, vitiligo) who had received AZA for > 4 weeks. Data on clinical features, laboratory findings, AZA treatment regimens, and AZA-associated AEs were reviewed. Pharmacogenomic testing of NUDT15 and TPMT was performed.

Results: Among 103 patients, the mean AZA dose was 1.02 mg/kg/day. Eighteen AEs were observed, including myelotoxicity (n = 13) and hepatotoxicity (n = 5). The prevalence of NUDT15 and TPMT mutations was 16.5% and 2.9%, respectively. In multivariate analysis, both NUDT15 and TPMT variants were significantly associated with AZA-induced myelotoxicity (OR = 13.56, 95%CI = 2.43-75.64, P = 0.003, and OR = 42.65, 95%CI = 1.40-1300.13, P = 0.031, respectively). However, neither was significantly related to hepatotoxicity. For AZA-induced myelotoxicity screening, isolated NUDT15 and TPMT tests showed limited sensitivity, with NUDT15 demonstrating higher sensitivity (50%) and good specificity (88.8%) compared to TPMT (sensitivity 14.3%, specificity 98.9%). Combining both tests improved screening performance (LR+ = 5.72, 95% CI = 2.83–11.50; sensitivity 64.3%, specificity 88.8%).

Conclusion: NUDT15 variants are relatively more common than TPMT variants in Thais. In addition to TPMT, NUDT15 testing should be performed before initiating AZA to reduce the risk of AZA-induced myelotoxicity in patients with IMSDs.

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Abstract N°: 5737

Pseudo-thrombocytopenia associated with glucantime

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Introduction & Objectives:

Cutaneous leishmaniasis is a parasitic disease caused by a flagellated protozoan, which typically presents as an ulcerated and crusted nodule. The standard treatment remains N-methylglucamine antimonates [meglumine antimoniate, e.g., Glucantime], which can induce several side effects, including hematological ones. We report a case of cutaneous leishmaniasis , treated with Glucantime, which induced pseudo-thrombocytopenia due to platelet aggregation.

Observation :

This concerns a 57-year-old patient presenting with two ulcerated and crusted nodular lesions on the left hand, which had been developing for two months, associated with a raised, violaceous plaque with an angiolupoid appearance on the dorsal aspect of the same hand. The rest of the clinical examination and paraclinical tests were unremarkable.A parasitological sample was taken to search for Leishmania bodies, which came back positive.The diagnosis of cutaneous leishmaniasis was confirmed. The patient was treated with intralesional Glucantime twice a week, combined with systemic Glucantime treatment intravenously (IV) at a dose of 1 ampoule per 10 kg per day for 15 days, with a favorable outcome. After initiating intravenous (IV) Glucantime at 50 mg/kg/day (= 4000 mg/day), pseudo-thrombocytopenia due to platelet aggregation was noted (53,000 platelets/mm^3), with levels returning to normal after stopping the treatment.

The pseudo-thrombocytopenia reappeared upon reintroduction of the same treatment (TTT), thus confirming the latter’s causality. This led to its discontinuation and the introduction of fluconazole, with a favorable outcome.

Discussion

Cutaneous leishmaniasis is a common condition in dermatology. It can manifest in various forms (polymorphic aspects): vegetating, impetiginoid, erysipeloid. . In our patient, 2 forms are present together: an ulcerated and crusted nodule and a lupoid plaque on the calf. The diagnosis is generally straight forward, based on clinical findings and parasitological examination.Histology is sometimes necessary for atypical forms.

Glucantime, the first-line treatment (TRT), has several side effects. Depending on the time of onset, a distinction is made between: stibio-intolerance (intolerance to antimony) and stibio-intoxication (antimony toxicity), which include, among others, hematological disorders, especially agranulocytosis. Pseudo-thrombocytopenia is not common; it is probably related to platelet aggregation caused by the formation of anti-EDTA antibodies.

Conclusion:

Despite the usually good response of cutaneous leishmaniasis to Glucantime, there are still cases that are difficult to manage and significant side effects.

Abstract N°: 5745

Benralizumab in Corticosteroid-Refractory DRESS Syndrome: A Case Report

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Introduction:

Drug rash with eosinophilia and systemic symptoms (DRESS) is a severe delayed hypersensitivity reaction triggered by medication exposure that involves multiple organ systems. First-line therapy after discontinuation of the offending drug is high-dose systemic glucocorticoids. While the majority of cases respond to this treatment, treatment failure may occur in a subset of patients. Effective second-line options are limited. Herein, we describe a severe case of steroid-refractory DRESS with acute fulminant hepatic failure remarkably responsive to benralizumab, an interleukin (IL)-5 receptor monoclonal antibody, sparing a liver transplant.

Case Description:

A 20-year-old male with epilepsy, started on lamotrigine three weeks prior, presented with a diffuse morbilliform rash, fevers, elevated liver enzymes, and eosinophilia. He developed sharp chest pain with electrocardiogram findings consistent with acute pericarditis. Skin biopsy showed interface dermatitis suggestive of DRESS syndrome. Lamotrigine was discontinued and he was started on oral prednisone (1 mg/kg/d) and colchicine. After clinical improvement and normalization of labs over several days, he was discharged on a slow prednisone taper with recommended outpatient follow-up. Five days later, he was readmitted with worsening rash, fevers, facial edema, and lymphadenopathy. Prednisone was increased to 1.5-2 mg/kg/d without improvement. He was transferred to the ICU within 72 hours of admission with steeply rising liver enzymes (AST/ALT >7000), encephalopathy, and concern for acute fulminant hepatic failure necessitating transplant workup and authorization. His RegiSCAR score was 8/8. He was given a one-time subcutaneous injection of benralizumab (30 mg), after which liver enzymes rapidly improved, eosinophilia returned to baseline, and dermatitis resolved. His preparation for liver transplant was thus canceled.

Discussion:

Treating refractory or relapsing cases of DRESS syndrome can be challenging. Immunosuppressants such as cyclosporine, cyclophosphamide, intravenous immunoglobulin, and Janus kinase inhibitors are often used as second-line therapy, though they are not always effective and can cause significant side effects. IL-5 is thought to induce the overactivation of eosinophils in DRESS, leading to severe inflammation and organ damage classically seen in the syndrome. Benralizumab, a biologic approved for treatment of severe asthma, is an IL-5 monoclonal antibody targeting the IL-5 receptor alpha and offers an effective second-line treatment in steroid-recalcitrant DRESS. To our knowledge, seven cases of benralizumab use in DRESS have been described, showing rapid eosinophil suppression and clinical improvement, often after a single 30 mg dose. These findings suggest anti-IL-5 therapies are an emerging, safe, and effective treatment in managing DRESS.

Conclusion:

This case report highlights the successful use of anti-IL-5 therapy in the management of severe, corticosteroid-refractory DRESS syndrome with fulminant liver failure, allowing for sparing of an impending liver transplant. These findings support the potential role of IL-5 axis blockade as a promising strategy. Further research is warranted to validate its efficacy and establish standardized treatment protocols.

Abstract N°: 5778

“When Treatment Heals… Then Betrays: Morphea and DRESS under Hydroxychloroquine”

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Introduction & Objectives:

Morphea (localized scleroderma) is a rare fibrosing condition of the skin and underlying tissues. The treatment of morphea is not standardized; several therapeutic options are indicated according to the physician’s judgment and the patient’s context. Few studies exist to support the treatment of morphea (localized scleroderma) with hydroxychloroquine. Here, we report the evolution of morphea under hydroxychloroquine, between a therapeutic miracle and the threat of immunoallergic drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome.

Cas report:

The patient is T.N., a 70-year-old with a history of diabetes; asthma, and penicillin allergy, admitted to our department for the management of superficial cigarette paper-like scleroatrophic lesions surrounded by a whitish halo extending to the entire chest, the upper thorax; axillary and inguinal folds, as well as the back with a burning sensation. The clinical presentation was resistant to multiple antifungal treatments. A skin biopsy with histopathological study confirmed the diagnosis of superficial morphea with deposits of collagen bundles. The patient was started on HCQ 400 mg for 2 months with spectacular improvement: disinfiltration, softening; significant desquamation, and reduction of erythema. During her treatment, she developed an evolving polymorphous eruption consisting of annular lesions in pseudo-cockades, papular lesions; urticarial lesions, diffuse erythema; facial edema; fever; and intense pruritus: a laboratory workup revealed hyperleukocytosis with a high percentage of atypical lymphocytes, a Régiscare score of 4 points indicating a highly probable DRESS. HCQ was discontinued and systemic corticosteroid therapy was initiated, with good clinical outcome.

Discussion:

A 2011 systematic review described the treatments and therapeutic algorithm for morphea. Hydroxychloroquine (HCQ) is a unique immunomodulatory drug that inhibits the action of Toll receptors involved in the activation of B2 lymphocytes, used to treat various dermatological conditions. However, to date, little evidence supports its use in morphea. The German guidelines (Association of Scientific Medical Societies in Germany) for the diagnosis and treatment of localized scleroderma (morphea) support the use of HCQ on a case-by-case basis with careful consideration, although HCQ is not included in the treatment algorithm[cite: 87]. In a retrospective study by Kumar et al., 81.0% of patients with morphea showed a complete response (CR) or partial response (PR) > 50%. While HCQ is associated with a myriad of adverse dermatological effects, most of which are poorly characterized by the literature, with unknown frequencies and risk factors. DRESS syndrome has been known for many decades as a severe adverse drug reaction, particularly to allopurinol, anticonvulsants, and sulfonamide-containing antibiotics. Few cases have been reported with HCQ, mainly in the field of rheumatology. Our observation describes HCQ as both a remedy and a risk, which complicated the patient’s management. Close monitoring of treatment is necessary, especially in patients with a history of atopy and drug allergies.

Conclusion:

Although HCQ appears to be an effective treatment in the management of morphea, it is considered a double-edged sword, due to its serious side effects requiring rigorous monitoring and reporting to pharmacovigilance.

Abstract N°: 5815

A Case Report of Hydroxychloroquine-Induced Acute Generalized Exanthematous Pustulosis with Prolonged Latency

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Introduction & Objectives:

Acute Generalized Exanthematous Pustulosis (AGEP) is a rare, acute dermatologic adverse reaction, most often induced by medications, although cases related to infections and vaccinations have also been reported. Its estimated incidence is a few cases per million annually. The drugs most commonly triggering AGEP include amoxicillin, fluoroquinolones, hydroxychloroquine, terbinafine, and sulfonamides. Skin lesions typically develop within 24–48 hours following exposure to the causative agent. The clinical picture is marked by high fever (exceeding 38°C) and the sudden onset of erythematous and edematous skin lesions with numerous, sterile, pruritic pustules, predominantly in intertriginous areas, followed by superficial desquamation. Involvement of mucous membranes is uncommon. Laboratory tests often reveal leukocytosis with neutrophilia, and, less commonly, hepatic or renal dysfunction. To aid diagnosis, the EuroSCAR study group established standardized diagnostic criteria based on the characteristic clinical course and laboratory abnormalities.

Materials & Methods:

A 71-year-old woman presented with a disseminated, confluent eruption of erythematous and edematous lesions, accompanied by numerous pustules on the trunk and extremities. The lesions were associated with marked pruritus and fever up to 39°C. Mucous membranes were unaffected. The initial skin changes appeared 24 days after the introduction of hydroxychloroquine, which had been prescribed for a suspected cutaneous sarcoidosis. Laboratory tests showed elevated CRP (252 mg/L), increased AST (106 IU/L) and ALT (62 IU/L), mild renal dysfunction (creatinine 125 µmol/L), and neutrophilia (23.3 × 10^3/µL). Histopathological examination of a skin biopsy demonstrated intraepidermal spongiform pustules, consistent with a pustular dermatosis.

Results:

Hydroxychloroquine was immediately discontinued upon clinical suspicion of AGEP. The patient was subsequently treated with intravenous dexamethasone and ceftriaxone over a period of 12 days, which resulted in a significant reduction of systemic inflammatory parameters and marked improvement of dermatological symptoms. Based on the clinical course, histopathological findings, and laboratory results, the patient achieved a score of 5 points according to the EuroSCAR diagnostic criteria, classifying the case as a *probable AGEP*. During a one-year follow-up period, no recurrence of the disease was observed.

Conclusion:

The classic course of AGEP involves rapid onset of skin lesions shortly after the introduction of the triggering medication, with spontaneous resolution usually within two weeks of drug withdrawal. The EuroSCAR scoring system reduces the score if resolution exceeds 14 days, underscoring the diagnostic importance of this clinical feature. Clinicians should be alert for atypical disease courses, particularly with drugs of prolonged half-life. Hydroxychloroquine, with a half-life of approximately 50 days, may lead to a delayed onset of symptoms, developing several weeks after initiation of therapy, and a prolonged resolution phase lasting several weeks following drug withdrawal. In clinical practice, it is crucial to consider the pharmacokinetics of the suspected

causative agent, as it may significantly alter the expected presentation and evolution of AGEP, potentially complicating timely diagnosis and management.

Abstract N°: 5842

Diffuse Pseudoangiomatous Skin Lesions in a Patient Under Anti-TNFα Therapy: A Challenging Differential Diagnosis

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Diffuse Pseudoangiomatous Skin Lesions in a Patient Under Anti-TNFα Therapy: A Challenging Differential Diagnosis

Introduction & Objectives:

Pseudoangiomatous skin lesions represent a diagnostic challenge in immunosuppressed patients, particularly those receiving biologic therapies. While infectious etiologies such as bacillary angiomatosis or Kaposi sarcoma are typically prioritized, drug-induced vascular proliferations or paraneoplastic manifestations must also be considered. We report a rare case of diffuse angiomatous-like lesions in a patient treated with anti-TNFα therapy, initially mimicking an opportunistic infection or vascular tumor.

Materials & Methods:

We present the case of a 63-year-old woman followed for severe psoriatic arthritis treated with anti-TNFα (adalimumab) for 5 years. She developed multiple erythematous-violaceous papules and nodules on the trunk and limbs, along with a tumor syndrome characterized by generalized lymphadenopathy. A thorough clinical, histopathological and microbiological workup was performed, including skin biopsy, HHV-8 immunohistochemistry, Bartonella PCR, infectious screening, and imaging studies.

Results:

Histological examination revealed a well-circumscribed lobular proliferation of small capillaries in the superficial dermis, consistent with benign capillary hemangiomas. No cellular atypia or mitotic activity was observed. Immunohistochemistry was negative for HHV-8, effectively ruling out Kaposi sarcoma. PCR and special stains for *Bartonella* were also negative, excluding bacillary angiomatosis. Imaging revealed multiple small reactive lymph nodes without signs of malignancy.

Given the exclusion of infectious and neoplastic causes, and in light of the patient's prolonged exposure to anti-TNFα therapy, the lesions were interpreted as **pseudoangiomatous**, likely **drug-induced**. Although rare, vascular proliferations have been reported in patients on TNF inhibitors, possibly linked to altered angiogenic signaling or immune dysregulation. A paraneoplastic origin could not be entirely excluded, but no underlying malignancy was detected over a six-month follow-up period.

Conclusion:

This case highlights an uncommon but significant presentation of pseudoangiomatous skin lesions in a patient under anti-TNFα therapy. The exclusion of infectious and neoplastic causes is essential before considering a drug-induced or paraneoplastic mechanism. Clinicians should be aware of such vascular skin reactions in the context of biologic therapy, to avoid misdiagnosis and inappropriate treatment.

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Abstract N°: 5859

Interstitial Granulomatous Dermatitis Induced by Dulaglutide

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Introduction & Objectives: Reactive granulomatous dermatitis (RGD) encompasses a spectrum of histologically and clinically overlapping entities, including interstitial granulomatous dermatitis (IGD). IGD is often seen in association with systemic diseases but it can also be drug-induced. We report the first case of IGD associated with dulaglutide, a glucagon-like peptide-1 (GLP-1) receptor agonist.

Materials & Methods:

Results: A 75-year-old man presented with a persistent, asymptomatic, erythematous truncal eruption for two weeks. His medical history included type 2 diabetes, hypertension, dyslipidemia, and atrial fibrillation. Recent medication changes included the addition of dulaglutide one month prior to symptom onset. Physical examination revealed bilateral, well-demarcated, erythematous patches (6 × 9 cm) on the flanks. Histopathological analysis demonstrated a dermal interstitial and perivascular lymphohistiocytic infiltrate with small interstitial granulomas, consistent with IGD (figure 1). There was no mucin deposition, eosinophilia, interface dermatitis, or lymphoid atypia. Discontinuation of dulaglutide led to complete resolution of lesions within 10 days.

Conclusion: IGD is a subtype of the broader category of RGD, which also includes palisaded neutrophilic and granulomatous dermatitis (PNGD) and interstitial granulomatous drug reaction (IGDR). While its classic “rope sign” was described in early IGD cases, newer reports highlight a broader clinical spectrum, including erythematous patches and plaques on the trunk. Histological findings typically include interstitial histiocytic infiltrates without prominent mucin or vasculitis, helping differentiate it from PNGD and IGDR. Drug-induced IGD has been associated with multiple cardiovascular and immunomodulatory agents; however, to our knowledge, this is the first reported case implicating a GLP-1 receptor agonist. The temporal relationship between drug initiation and rash onset, as well as prompt resolution following discontinuation, strongly supports a causal link. Clinicians should be aware of the potential for GLP-1 receptor agonists to induce IGD, especially given their increasing use in diabetes and weight management. Recognition of this association is critical to avoid misdiagnosis and unnecessary interventions. A comprehensive medication history is essential in evaluating patients with granulomatous skin eruptions.

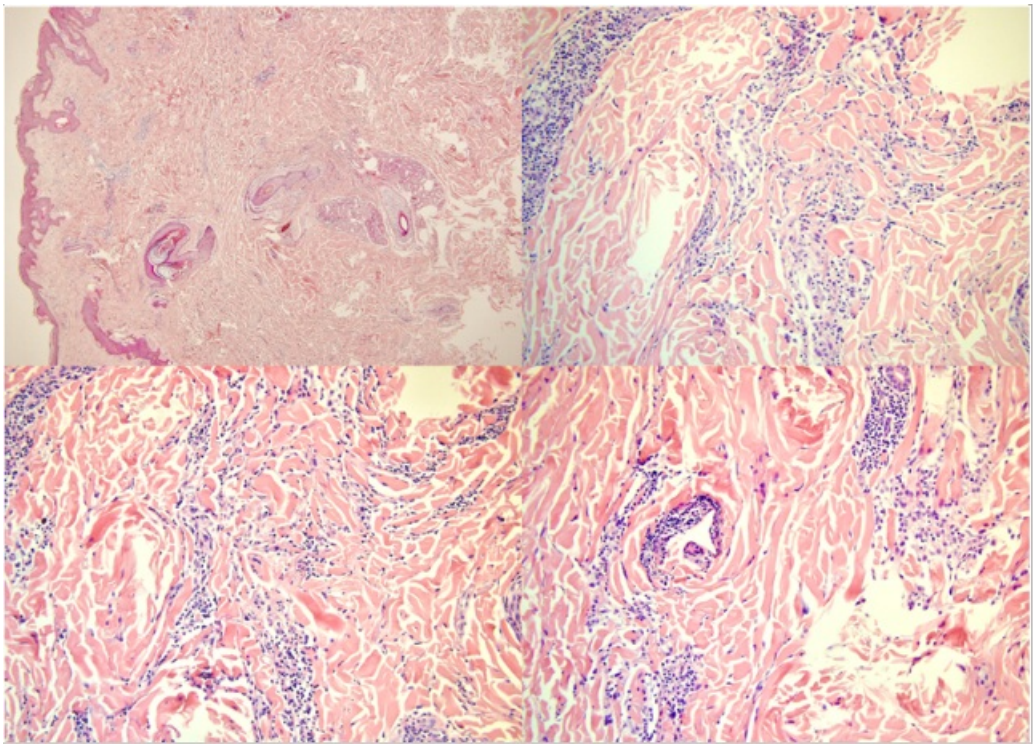

Figure 1

Abstract N°: 5881

Symmetrical Drug-Related Intertriginous and Flexural Exanthema (SDRIFE) Induced by Topical Diclofenac Sodium: A Case Report

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Introduction & Objectives: Symmetrical Drug-Related Intertriginous and Flexural Exanthema (SDRIFE), also known as baboon syndrome, is a rare, well-defined cutaneous adverse drug reaction characterized by symmetric erythematous eruptions predominantly affecting intertriginous and flexural areas, without systemic involvement. While systemic drugs are the usual triggers, topical agents have been rarely implicated.

Materials & Methods:

This case report was conducted in accordance with the principles outlined in the Declaration of Helsinki. The patient provided written informed consent for the use of clinical information and photographs for academic and publication purposes.

Clinical data were obtained through direct patient examination, medical history, and photographic documentation of skin lesions

A drug causality assessment was performed using the Naranjo Adverse Drug Reaction Probability Scale.

The diagnosis of SDRIFE was made based on clinical presentation, history of systemic drug exposure, and absence of systemic symptoms consistent with a drug-induced exanthem.

Results:

A 90-year-old female patient, presented with well-demarcated, non-pruritic erythematous eruption, initially localized to the left wrist and progressively extending in a symmetrical pattern to the forearms, abdomen, inframammary folds, axillae, lower back, gluteal region, inguinal folds, thighs, and, to a lesser extent, popliteal fossae. Areas of superficial desquamation were also noted within the erythematous plaques. No mucosal involvement or systemic symptoms were present

Three weeks prior, following a wrist injury due to a fall, the patient had begun daily application of a topical diclofenac sodium gel for pain management.

The clinical features, symmetrical distribution, predominant involvement of flexural and intertriginous areas, temporal association with topical diclofenac application, and absence of systemic manifestations, were strongly suggestive of SDRIFE.

A treatment regimen consisting of systemic and topical corticosteroids was initiated, leading to complete clinical resolution. The patient was advised to avoid further exposure to diclofenac and related compounds.

Given the patient’s advanced age, the excellent clinical response to treatment, and the typical presentation, it was decided not to perform a skin biopsy or drug provocation testing.

Conclusion: This case highlights the potential of topical non-steroidal anti-inflammatory drugs (NSAIDs) to trigger SDRIFE, a diagnosis that should be considered even in the absence of systemic drug administration. Prompt recognition and withdrawal of the offending agent, along with appropriate therapeutic intervention, are

crucial for resolution and prevention of recurrence.

No recent introduction of systemic medications was reported, but we can not exclude a recall bias because the patient was 90.

Abstract N°: 5893

Diagnostic challenge of erythroderma in a psoriatic and psychotic patient

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Introduction & Objectives:

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) is a rare, severe, and potentially life-threatening adverse reaction to medication. Early identification of the triggering drug is crucial to improving prognosis. In this case, we present a patient with a background of psoriasis and chronic psychosis who developed erythroderma, posing a diagnostic dilemma between a psoriatic flare and DRESS.

Materials & Methods:

A 50-year-old man with a lifelong history of psoriasis and chronic psychosis (marked by delusional jealousy, depression, and anxiety) was admitted with erythroderma that had evolved over the course of 7 days. He had been on carbamazepine (Tegretol®) since November 15, 2024. The skin symptoms started on December 26, 2024. Clinical examination revealed generalized erythroderma with both fine and thick scaling, mucosal involvement (including anal erosion, scaling of the glans penis, and bilateral conjunctivitis), and fever of 38.8°C. Lab tests showed marked leukocytosis (36,890/mm^3) with pronounced eosinophilia (13,740/mm^3), elevated CRP (68.6 mg/L), and liver enzyme abnormalities (ALT 1.7× normal). A skin biopsy was not performed due to patient refusal. Parasitic testing for scabies returned negative.

Results:

While erythrodermic psoriasis could have been suspected based on the patient's history, several key findings pointed toward DRESS: febrile erythroderma, significant eosinophilia, hepatic involvement, and a recent drug exposure (the timing of symptoms—onset approximately 6 weeks after starting carbamazepine). The RegiSCAR scoring system supported a probable diagnosis of DRESS. This case also highlights the known association between psoriasis and psychotic disorders.**1** Management included the immediate withdrawal of carbamazepine, initiation of systemic corticosteroids (prednisolone 0.5 mg/kg/day), topical skin care, empiric antibiotics (gentamicin and cefotaxime), and IV fluids. The patient showed rapid clinical improvement with complete resolution of cutaneous, mucosal, and lab abnormalities.

Conclusion:

This case illustrates the diagnostic complexity of erythroderma in patients with a coexisting psoriatic and psychiatric background treated with anticonvulsants. DRESS should always be considered when febrile erythroderma with eosinophilia appears after recent initiation of high-risk medications such as carbamazepine. Early recognition and management are crucial for improving both survival and functional outcomes.

1. ***https://doi.org/10.1016/j.jad.2023.05.059***

https://pmc.ncbi.nlm.nih.gov/articles/PMC4928455/

Abstract N°: 5939

Rhabdomyolysis Induced by Low-Dose Oral Isotretinoin: Case Report

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Introduction & Objectives:

Oral isotretinoin is the most effective treatment modality for severe and refractory cases of acne vulgaris. The occurrence of benign and self-limiting muscle damage associated with its use, with consequent elevation of creatine kinase (CPK) levels, is well described. However, serious musculoskeletal adverse effects, such as rhabdomyolysis, are rare and often dose-dependent. It is believed that a synergy between the drug and physical exercise may occur in the development of this complication.This report describesthe case of a young, previously healthy patient taking low doses of isotretinoin who developed severe rhabdomyolysis and acute renal failure (AKI).

Materials & Methods:

The information was obtained through the analysis of medical records and bibliographic review in the main databases (Pubmed, Scielo, Lilacs).

Results:

This is a 30-year-old female patient with no known comorbidities, using oral contraceptives, no history of other medication use or substance abuse, and physically active. She presented with refractory adult female acne. She had undergone two previous cycles of isotretinoin treatment, the last of which began due to acne conglobata and ended last year. After approximately six months of using topical and oral alternatives for maintenance treatment, it was decided to reintroduce low-dose isotretinoin (20 mg every other day)to control the condition. Laboratory tests prior to starting treatment revealed a CPK of 185 U/L, with a baseline creatinine (Cr) of 1.0 mg/dL. After a few weeks of use, the patient developed fatigue and myalgia in the lower limbs disproportionate to the physical activity performed. She went to the emergency room, where a diagnosis of rhabdomyolysis was raised and tests were ordered (CPK 65,200 U/L, Cr 1.38 mg/dL), which confirmed the suspected diagnosis and also revealed the presence of AKI. The patient was hospitalized and received supportive treatment, being discharged after clinical stabilization and improvement in laboratory parameters. Following discharge, she underwent an extensive workupto rule out other etiologies, such as inflammatory myopathies,supporting the hypothesis thatthese events occurred as a result of synergism between isotretinoin and moderate-intensity physical activity.

Conclusion:

Because it is a widely used medication with potentially serious side effects, a narrow segment of patients using isotretinoin is recommended, even at low doses.

Abstract N°: 6095

Targeting JAK-STAT signaling attenuates cytotoxic T lymphocyte activations in Stevens-Johnson syndrome and toxic epidermal necrolysis

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Introduction & Objectives: Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are life-threatening severe cutaneous adverse reactions primarily mediated by activated cytotoxic T lymphocytes (CTLs). The JAK-STAT signaling pathway has been implicated in driving CTL activation and epidermal necrolysis. This study aims to elucidate the role of JAK1/3-STAT1 signaling in SJS/TEN pathogenesis and evaluate the therapeutic efficacy of the JAK1/3 inhibitor tofacitinib.

Materials & Methods: Patients diagnosed with SJS/TEN were enrolled and categorized into discovery and clinical trial cohorts. Biological samples, including lesional blister cells, peripheral blood mononuclear cells (PBMC), and skin biopsies, underwent comprehensive analysis using single-cell RNA sequencing (scRNA-seq) and spatial transcriptomics (ST). Additional validation was conducted via immunostaining, flow cytometry, cytokine profiling, enzyme-linked immunosorbent assays (ELISA), and ex vivo blocking assays with specific JAK/STAT pathway inhibitors. Flow cytometry specifically evaluated phosphorylation states of STAT1 and STAT3 proteins within CD3+CD8+ cytotoxic T cells to assess cellular activation profiles. A single-arm clinical trial (NCT06474078) involving 20 patients with SJS/TEN was conducted, where participants received tofacitinib treatment, with clinical outcomes and safety profiles rigorously monitored.

Results: Transcriptomic analyses highlighted significant activation of IFN-γ, JAK-STAT, and apoptotic signaling pathways, prominently involving clonally expanded CD8+ tissue-resident memory T cells characterized by high expression of cytotoxic proteins (GNLY, GZMB), inflammatory cytokines, immune checkpoint molecules, and critical JAK-STAT pathway components. Immunohistochemistry confirmed markedly elevated phosphorylation levels of JAK1, JAK3, and STAT1 within the epidermis and dermal inflammatory infiltrates. Flow cytometry demonstrated significantly increased phosphorylation of STAT1 and STAT3 in blister-derived CD3+CD8+ T cells compared to PBMCs and healthy controls, corroborating transcriptomic findings. Ex vivo blocking assays revealed substantial reductions in cytotoxic proteins (GNLY, GZMB) secretion upon targeted JAK1/3 inhibition. Clinical trial data showed tofacitinib treatment notably reduced skin healing time (median 12.5 days), prevented further blister progression, and markedly improved patient outcomes with zero observed mortality.

Conclusion: Our findings establish JAK1/3-STAT1 signaling as a pivotal mediator in the immunopathogenesis of SJS/TEN, underscoring targeted pathway inhibition as a promising therapeutic strategy. Clinical data strongly support tofacitinib's efficacy in reducing disease severity and accelerating skin recovery, advocating further investigation of JAK1/3 inhibitors as viable treatments for severe cutaneous adverse drug reactions.

Abstract N°: 6141

Exploring Late Toxicity: Late-Onset Raynaud's Phenomenon After Vinblastine Therapy in Pediatric Langerhans Cell Histiocytosis Survivor

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Introduction & Objectives:

Chemotherapy is associated with early and late side effects, impacting the quality of life (QOL) of cancer survivors, especially in pediatric populations. One such late effect is Raynaud's phenomenon (RP), characterized by intermittent ischemia in the fingers and toes due to vasospasm, and often occurring within a year after antineoplastic treatment. RP has been reported with Vinblastine, commonly used for treatment of Langerhans cell histiocytosis (LCH).

This case report describes secondary RP in a 12-year-old patient who completed Vinblastine therapy for multisystemic LCH. The study aims to raise awareness of this late-onset side effect and discuss the challenges of managing such cases in pediatric patients, where treatment protocols are often adapted from adult recommendations.

Materials & Methods:

A 12-year-old female patient diagnosed with multisystemic LCH at 9 years old completed 12 months of Vinblastine and corticosteroids, achieving complete remission. Twelve months after treatment, she developed debilitating RP, with episodes triggered by cold or hand manipulation, occurring up to five times daily, impairing daily activities.

Physical examination revealed no signs of connective tissue disease or hematological disorder. There was no personal or family history of autoimmune disease or prior RP. The patient's Dermatology Life Quality Index (DLQI) score was 18, reflecting a significant impact on QOL.

Capillaroscopy showed disorganized microvascular architecture, mega-capillaries, ectatic capillary dystrophies, and micro-hemorrhages. The autoimmune panel was positive for antinuclear antibodies (ANA) with a speckled pattern but negative for other markers (anti-SSA, anti-SSB, anti-DNA, anti-SM, anti-RNP, anti-Scl70, anti-Jo1). Blood count, complement levels, inflammatory markers (CRP, ESR), and 24-hour proteinuria were normal.

Results:

Secondary RP was diagnosed, likely triggered by Vinblastine therapy. The case was reported to pharmacovigilance with an imputability score of C2S3B3. Symptomatic treatments were ineffective, prompting Nifedipine initiation with gradual dose escalation due to the lack of pediatric dosing guidelines. A significant reduction in episodes was observed, from five to one per day or every other day. The patient's DLQI score improved to 10 after three months, reflecting an improvement in QOL.

Conclusion:

Raynaud's phenomenon (RP) is a well-documented side effect of chemotherapy, particularly following Vinblastine

therapy. The mechanism remains unclear, but vascular toxicity and endothelial damage are thought to contribute. This case highlights the importance of long-term monitoring for late toxicities in pediatric cancer survivors, as chemotherapy-induced RP can develop months or years after treatment. It also underscores the challenges of managing such conditions in pediatric patients, where treatment protocols are often adapted from adult regimens.

Abstract N°: 6224

Drug-Induced Erythema Multiforme Associated with Semaglutide: A Case Report

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Introduction & Objectives:

Semaglutide, a GLP-1 receptor agonist, is widely used for type 2 diabetes and weight management. While gastrointestinal side effects are common, cutaneous adverse reactions are rare. Erythema multiforme (EM) is an acute, immune-mediated skin condition that can be triggered by infections or medications. We present a rare case of drug-induced EM associated with Semaglutide.

Materials & Methods:

We report the case of a 30-year-old woman who developed vesiculobullous targetoid lesions on the upper back, neck, and scalp one week after increasing her Semaglutide dose to 0.5 mg. Her clinical history included mild hypersensitivity reactions to fresh fruits, cosmetic products, and a previous reaction to another GLP-1 receptor agonist, Liraglutide. Diagnostic evaluation included skin swabs for viral screening, lesional and perilesional punch biopsies for histopathology and direct immunofluorescence.

Results:

Histopathological examination showed orthokeratotic hyperkeratosis, mild spongiosis, and superficial perivascular lymphohistiocytic infiltrates with intraluminal neutrophils, consistent with a drug-induced reaction. Direct immunofluorescence was negative for IgA, IgG, IgM, and C3. The patient responded well to topical corticosteroid treatment (diflucortolone valerate), and Semaglutide therapy was continued with no further recurrence after symptomatic control.

Conclusion:

This case highlights Semaglutide as a potential but rare trigger of erythema multiforme, particularly in patients with prior hypersensitivity to GLP-1 receptor agonists. Early recognition and appropriate management are essential. In selected cases, continuation of Semaglutide with concurrent topical treatment may be considered when clinically appropriate. Further research is warranted to understand the immunologic mechanisms and potential class effects associated with GLP-1 receptor agonists.

Abstract N°: 6254

A Prospective Cohort Study of Serum Sickness-Like Reactions in the Pediatric Population

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Introduction & Objectives: Serum Sickness-Like Reaction (SSLR) is a hypersensitivity reaction primarily linked to antibiotics, especially beta-lactams. However, other drugs or infectious agents have also been implicated in triggering this condition. SSLR is characterized by a skin rash and distinct cutaneous morphologies, such as annular, urticarial, or maculopapular eruptions, often accompanied by joint pain or inflammation, with or without a fever. Unfortunately, SSLR is often misdiagnosed due to a lack of standardized diagnostic criteria and overlapping features with other pediatric dermatoses. This study aims to identify clinical and laboratory features that may aid in the improved diagnosis of this condition.

Materials & Methods: A prospective cohort study was conducted at a tertiary children's hospital from October 1, 2022, to September 30, 2024. Children aged 0-18 years presenting with a skin rash and joint edema or pain, with or without fever, were included. Data collected included clinical presentation, laboratory findings, joint ultrasound results, and treatment responses.

Results: Among 27 patients (59.26% female, 40.74% male; mean age: 4 years), amoxicillin was the primary trigger (85.19%), followed by a combination of amoxicillin/cephalosporin (11.11%), and nitrofurantoin (3.70%). Prior drug exposure was noted in 92.5% of cases, with 12% reporting prior adverse reactions. Rash morphologies included annular (52%), urticarial (33%), and maculopapular (15%). Associated purplish discoloration was present in 67%, and targetoid lesions were seen in 22%. Joint pain was universal, but edema overlying the joints was only present in 96.3%. Joint ultrasound revealed mild-to-moderate joint effusion (42%) and extra-articular edema (53%), indicating peri-articular inflammation, but not true arthritis. Treatments included antihistamines (100%), NSAIDs (74%), systemic steroids (37%), and topical steroids (22%). The mean recovery times were 5 days for rash, 3.8 days for joint pain, and 3.9 days for malaise. Most patients fully recovered within two weeks with no long-term complications. To confirm drug causality, lymphocyte toxicity assay (LTA) was performed in 88.89% of cases, (24/27) with 58.33% testing positive. Among the 10 patients with negative LTAs, six had a negative oral challenge and four declined the test. Patients presenting with annular rash and peri-articular inflammation had a strong association with LTA positivity. Flow cytometry in patients with annular morphology showed a higher percentage of T-cell activation markers. In all patients, levels of CH50, C3 and C4 were normal. IgE levels were elevated in 48% of the patients, but there was no significant difference observed between annular and urticarial morphology.

Conclusion: SSLR most commonly presents with skin lesions of annular or urticarial-like morphology, associated with arthralgias, but not true arthritis. Pruritus and facial edema are common, but systemic involvement or anaphylaxis is not observed. Clinical and laboratory findings in SSLR patients overlapped with those seen in urticaria multiforme, suggesting the possibility that these conditions are part of the same continuum. This study highlights the need for increased clinical recognition, standardized diagnostic criteria, and further research into SSLR pathophysiology and treatment.

Abstract N°: 6255

A Case of Early-Onset, Steroid-Resistant DRESS (Drug Reaction with Eosinophilia and Systemic Symptoms) Following Antiepileptic Drug Use

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Introduction & Objectives:

DRESS (Drug Reaction with Eosinophilia and Systemic Symptoms) syndrome is a rare, potentially life-threatening drug reaction characterized by skin rashes, eosinophilia, fever, lymphadenopathy, and internal organ involvement. Symptoms typically appear 2–8 weeks after starting the causative drug. The objective of this case report is to present a case of early-onset, steroid-resistant DRESS that developed 1 week after the initiation of an antiepileptic drug and was successfully treated with cyclosporine.

Materials & Methods:

A 44-year-old woman presented with widespread erythematous rashes, fever, and shortness of breath. One month prior, she had been treated for subarachnoid hemorrhage and started on Epantoin. One week after the initiation of Epantoin, she developed rashes, and the dosage was reduced, but the rashes progressively worsened over the next 2 weeks. She was started on oral methylprednisolone (48 mg/day for 3 days, reduced to 32 mg/day), along with cetirizine, but her symptoms did not improve. Two days before admission, the Epantoin was discontinued. On admission, laboratory tests revealed eosinophilia and elevated liver function tests (LFTs). A skin biopsy was performed to aid diagnosis.

Results:

Histopathological examination revealed interface dermatitis with a perivascular mixed inflammatory infiltrate, including eosinophils and leukocytes, consistent with DRESS. Despite systemic corticosteroid therapy, LFTs and C-reactive protein (CRP) levels remained elevated, and fever persisted. Therefore, cyclosporine (400 mg/day) was initiated. Within a short period, the patient showed dramatic clinical improvement with resolution of the rashes and fever. The steroid dose was reduced to 40 mg/day, and the patient was discharged with oral methylprednisolone and cyclosporine. However, after discontinuing her medications post-discharge, she returned with high fever and worsened symptoms. Laboratory tests confirmed elevated LFTs and CRP again, and the patient was re-admitted and restarted on systemic corticosteroids and cyclosporine. The patient’s symptoms improved rapidly upon re-initiation of therapy.

Conclusion:

DRESS syndrome symptoms typically appear 2-8 weeks after starting the causative drug. However, in cases triggered by antiepileptic drugs, the onset may occur earlier. While systemic corticosteroids are the first-line treatment, they may be insufficient in severe or resistant cases. Cyclosporine has been proposed as an alternative treatment option and may better target the pathogenesis of DRESS syndrome. Early initiation of cyclosporine, in conjunction with careful medication management, is essential for controlling disease progression and preventing relapse.

Abstract N°: 6339

Panitumumab and Periorbital Dermatitis: Expanding the Spectrum of EGFR Inhibitor-Related Skin Toxicities

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Introduction & Objectives: Epidermal growth factor receptor (EGFR) inhibitors are known to cause a variety of cutaneous adverse effects, including acneiform eruptions, xerosis, paronychia, and less commonly, periorbital dermatitis. Among EGFR inhibitors, cetuximab and erlotinib have been frequently associated with dermatologic toxicities. Panitumumab, a fully human monoclonal antibody targeting EGFR, is generally associated with similar adverse skin reactions, though periorbital involvement has been rarely documented.

Materials & Methods: The case was a clinical observation.

Results: We present the case of a 64-year-old female with post-operative metastatic rectal adenocarcinoma who was treated with a biweekly chemotherapy regimen consisting of irinotecan, folinic acid, 5-fluorouracil (FOLFIRI), and panitumumab. Approximately 50 days after initiation of therapy, the patient developed bilateral periorbital erythema with pruritus and desquamation, extending to the cheeks. Ectropion was observed on the lower right eyelid. The patient attempted to alleviate the symptoms using a commercially available moisturizing cream with known ingredients, but without improvement. She had no previous history of allergic contact dermatitis or atopy.

Differential diagnoses included drug-induced dermatitis secondary to chemotherapy agents or allergic contact dermatitis to the applied topical product. A short course of topical hydrocortisone acetate ointment resulted in almost complete resolution of the lesions. To further investigate the possibility of allergic contact dermatitis, a repeat open application test (ROAT) was performed using the same moisturizing cream, applied twice daily to the inner elbow crease for one week. No local reaction was observed. Following the completion of the chemotherapy regimen, the periorbital dermatitis did not recur.

Conclusion: This case emphasizes the importance of recognizing uncommon presentations of EGFR inhibitor–related skin toxicities. While periorbital dermatitis is a well-known cutaneous side effect associated with certain EGFR inhibitors, it has been infrequently reported in patients receiving panitumumab. Accurate diagnosis relies on clinical history, therapeutic response, and, when necessary, allergology testing. Awareness of such rare dermatologic reactions can aid clinicians in timely identification and management, preventing unnecessary discontinuation of effective oncologic treatments.

Abstract N°: 6342

When chemotherapy overflows: progression to extensive skin necrosis

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Introduction & Objectives:

Extravasation is the accidental leakage of chemotherapy into surrounding tissues and can lead to severe complications, depending on the drug involved. Prompt and appropriate management is essential to minimize local damage.

Cas report :

We report the case of an 83-year-old man treated for Hodgkin's lymphoma, who was in complete remission after nine cycles of EBVD chemotherapy. During one session, improper catheter placement resulted in extravasation of the chemotherapeutic agents into the left forearm. Initial management included corticosteroids; however, the patient developed extensive skin necrosis requiring surgical debridement. The resulting ulcer (20 × 11 cm) was further complicated by superficial thrombophlebitis and secondary infection with *Escherichia coli*. Wound care, antibiotic therapy, and multidisciplinary follow-up were initiated.

Discussion :

The severity of extravasation depends on the nature of the drug involved. Vesicant agents, such as anthracyclines and vinca alkaloids, can cause severe tissue necrosis, whereas irritants typically result in less severe inflammation. Management strategies vary by drug and may include localized cold or heat application, hyaluronidase, or dexrazoxane. Notably, corticosteroids are contraindicated in cases of vesicant extravasation.

Conclusion:

This case illustrates the potentially severe consequences of chemotherapy extravasation. Early recognition and appropriate intervention are critical to minimize complications. Prevention remains paramount and requires meticulous infusion techniques and adequate staff training.

Abstract N°: 6527

Phototoxic Reaction During Combined BRAF/MEK Inhibition: A Case-Based Clinical Insight

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Introduction & Objectives:

The combination of Vemurafenib and Cobimetinib, selective BRAF and MEK1/2 inhibitors respectively, has demonstrated superior overall survival when compared to monotherapy for advanced melanoma cases. The photosensitivity reactions are among the most frequently reported cutaneous toxicities associated with Vemurafenib, attributed to its strong UVA absorption spectrum. In contrast, there is absence of documented cases in the literature of photosensitivity reactions solely to Cobimetinib. Although current evidence is limited to regulatory data, emerging clinical observations suggest a potential additive or synergistic effect of Cobimetinib when combined with Vemurafenib. We present a case of a phototoxic drug reaction in a melanoma patient undergoing dual BRAF/MEK inhibition, with the aim to highlight the underexplored risk of combined-agent photosensitivity and the need for early dermatologic care.

Materials & Methods:

We report a case of a 37-year-old female who presented to our clinic with a skin reaction following initiation of combined therapy with Vemurafenib and Cobimetinib for the treatment of metastatic melanoma. Two weeks into therapy, following incidental UVA exposure, the patient developed an acute, well-demarcated erythema with subtle vesiculation and edema in the facial and anterior cervical (V-line) region. The diagnosis of acute phototoxic reaction was made clinically.

Results:

Treatment commenced with intravenous corticosteroids and antihistamines, followed by oral tapering of prednisolone and nonsedating H1-antihistamines. Strict UVA avoidance and broad-spectrum photoprotection measures were instituted. Lesions regressed significantly within one week after starting the therapy. The oncologic treatment was temporarily withheld and continued after complete resolution of the cutaneous changes, with reinforced photoprophylaxis and dermatologic surveillance. The patient remained recurrence-free for cutaneous symptoms during follow-up.

Conclusion:

This case shows the importance of recognizing and managing phototoxic reactions induced by BRAF and MEK inhibitor combination therapy. Given the predictable UVA-dependent nature of Vemurafenib toxicity and the contributory potential of Cobimetinib, clinicians should maintain high clinical vigilance, especially in the early phases of treatment. All patients should be informed in advance about immediate and delayed reactions potentially caused even by low doses of UVA highlighting the importance of sun avoidance and photoprotective measures. Prompt patient-centered education and preventive strategies are crucial to ensure patient adherence to treatment, minimize morbidity and maintain continuity of oncologic care in metastatic melanoma. Further studies are needed to characterize the cumulative phototoxic risk of dual BRAF/MEK inhibition.

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Abstract N°: 6548

Skin related side effects in children treated with clofazimine for rifampicin resistant tuberculosis

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Introduction & Objectives:

Clofazimine (CFZ) is routinely used to treat rifampicin-resistant tuberculosis (RR-TB) in children, including in new shorter, all-oral regimens. While its cutaneous adverse effects, most notably skin discolouration and xerosis, are well documented in adults, these effects are understudied in children. Visible dermatological changes may have psychosocial consequences in children and adolescents, including stigmatization and caregiver concerns about the acceptability of treatment. We characterise the dermatological effects of clofazimine in children treated for RR-TB.

Materials & Methods:

This prospective observational study is part of a Phase I/II study (Clofazimine PK2; SAPHRA 20200110) evaluating the pharmacokinetics, safety and tolerability of novel clofazimine formulations and doses in children with RR-TB. Children weighing less than 30kg and receiving <12 weeks of a clofazimine-containing regimen for RR-TB were consecutively enrolled in Cape Town, South Africa. Skin assessments were performed at screening and on Day 1, and repeated at 4, 8 and 12 weeks. Data collection included caregiver-reported skin symptoms, systematic dermatological examination and skin colorimetry. Severity of skin discoloration and xerosis was assessed using a standardized, study-specific grading scale. Skin colorimetry was performed in both sun-exposed (face) and unexposed (inner upper arm) areas using a Konica Minolta CM700-d photospectrometer. Colorimetry measurements were represented using the CIELab color space (L*a*b*-values). Colour difference (ΔE) was calculated using the ΔE 1976 formula.

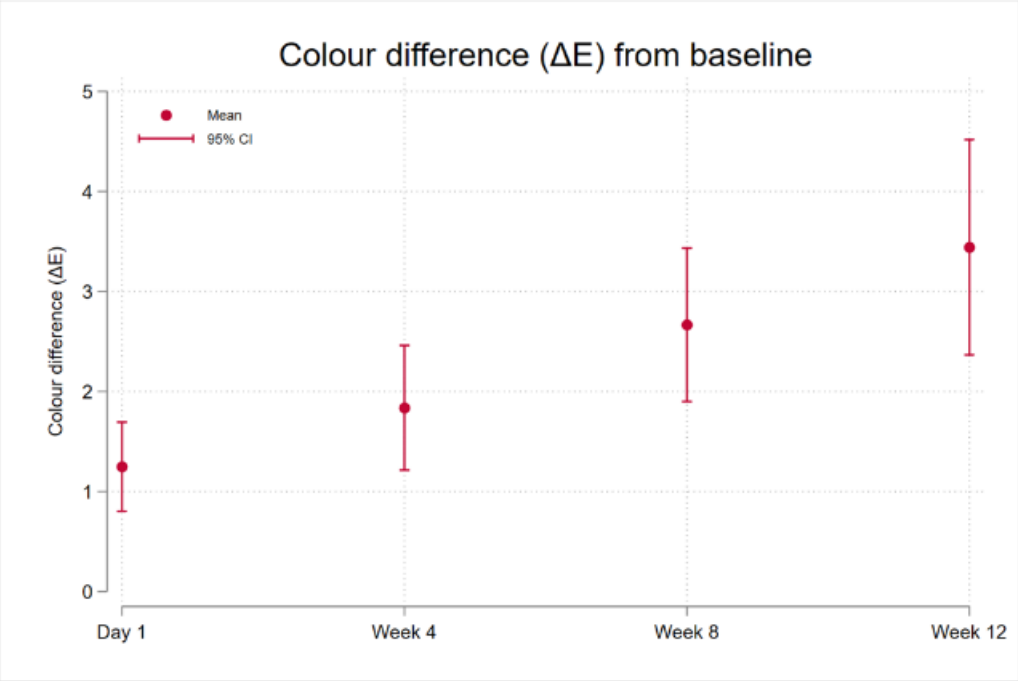
Results:

Twelve children (median age of 2.8 years, 64% female) were enrolled, none were living with HIV. All children were Fitzpatrick skin phototypes V or VI at baseline. At enrolment, the median time on clofazimine was 44.5 days (IQR 21.0, 60.0 days). Caregivers reported skin discoloration in 42 % of children at baseline, increasing to 72% by week 8 on treatment. Study physicians documented skin discoloration in 100% of participants from week 8 onwards, with progressive worsening. One participant (9%) experienced severe skin discoloration at week 12, as defined by a study-specific grading system indicating a marked change in pigmentation. Xerosis was reported by 46% of the caregivers at week 12 on treatment, consistent with investigator assessments (56%). Colorimetry measurements showed a mean ΔE of 3.44 (95% CI: 2.4–4.5) by week 12, indicating progressive discoloration while on clofazimine (Figure 1). Skin discoloration was characterised by increasing a*-values (reddening), decreasing L*-values (darkening), and decreasing b*-values (less yellow).

Conclusion:

In this study, all children receiving clofazimine-containing RR-TB treatment developed subtle, yet progressive skin discolouration of varying severity, as characterised using caregiver reports, clinical assessments and objective colorimetry measures. Xerosis was present in more than half of children after 12 weeks. These findings highlight the importance of monitoring dermatological side effects of antituberculosis treatment in children and their importance in assessing the safety and tolerability of novel antituberculosis regimens.

Figure 1: Colour difference over time during clofazimine exposure, as measured by skin colorimetry.



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Abstract N°: 6733

Apalutamide - Induced Acquired Ichthyosis in an elderly patient: A case report

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Introduction & Objectives:

Ichthyosis is a condition characterized by generalized scaling of the skin, which can be either congenital or acquired. Acquired ichthyosis may be associated with inflammatory, endocrinologic, and neoplastic processes, as well as certain medications. New drugs used in cancer treatments often have cutaneous side effects, which tend to emerge over time. Apalutamide, a second-generation antiandrogen used in prostate cancer treatment, has been reported to cause dermatologic adverse effects.

Case Report:

A 73-year-old man presented to our clinic with a 45-day history of pruritus and severe xerosis affecting his trunk and all extremities. His medical history included prostate cancer (Gleason score: 4+4) with metastasis to the lymph nodes, lungs, and lumbar vertebrae for the past six years. He had no other chronic disease and was not on any medication aside from chemotherapy for prostate cancer.

Three months prior, he had started treatment with the gonadotropin-releasing hormone antagonist degarelix, followed by the selective androgen receptor inhibitor apalutamide (240 mg daily). Approximately 45 days after initiating chemotherapy, he developed a pruritic rash and xerosis over his trunk and extremities. Physical examination revealed diffuse dry, rough skin with polygonal scaling across the body, predominantly affecting the trunk and the right lower leg. There was no personal or family history of ichthyosis or atopy. No new medications had been introduced other than apalutamide. The Naranjo Adverse Drug Reaction Probability Scale score for apalutamide was 4, whereas it was 0 for the other chemotherapy agents the patient had been receiving for years. Based on these findings, apalutamide-induced acquired ichthyosis was suspected and a skin biopsy was performed on a polygonal scale on the right tibia.

Results:

Histopathologic examination revealed hyper-orthokeratosis on the surface, hydropic degeneration in the basal layer of the epidermis, and melanophage-containing infiltration in the papillary dermis. Clinical and histopathological findings were consistent with acquired ichthyosis. The patient was prescribed topical emollients and keratolytic agents.

Two weeks after the biopsy, apalutamide was discontinued by the oncology department due to skin toxicity. With topical treatment and cessation of the suspected drug, the patient showed significant improvement in the skin lesions.

Conclusion:

Recently, in 2024,, Ciolfi et al. reported the first case of ichthyosiform eruption in a patient receiving apalutamide. To the best of our knowledge, our case represents the second reported instance of ichthyosiform eruption with apalutamide treatment. It should be considered that ichthyosis may develop secondary to malignancy, as well drug-induced ichthyosis. However, the occurrence of the adverse event occurred following apalutamide administration and its regression upon discontinuation of the drug supports the diagnosis of drug-induced

ichthyosis.

With the increasing use of nonsteroidal, second-generation antiandrogens, recognizing their potential dermatologic side effects is crucial. By presenting this case, we aim to raise awareness of apalutamide-induced ichthyosis as a possible adverse reaction. Further studies and case series are needed to better understand the underlying pathogenetic mechanisms.

Abstract N°: 6766

Rituximab in Dermatology: Adverse Reactions and Desensitization Protocols

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Introduction & Objectives:

Rituximab is a chimeric monoclonal antibody (mAb) targeting the CD20 molecule found on human B cells. It is commonly used to treat a range of lymphoid malignancies, lymphoproliferative disorders, autoimmune diseases such as bullous dermatoses. While rituximab is typically well tolerated, its increased use has been linked to hypersensitivity reactions. We report a serie of cases of reaction to rituximab observed in our dermatology department.

Materials & Methods:

We performed a 2-year prospective study, from January 2023 to February 2025, of all reactions to rituximab in the dermatology department. Patient demographics, clinical symptoms and management of reactions were examined. All cases were reported to pharmacovigilance to guide our management. We classified the grade of the reaction using a modified oncologic version of the National Cancer Institute Common Terminology Criteria for Adverse Events Scale, which scores a reaction from 1 (mild reaction) to 4 (severe reaction) similar to our previous work. Grade 1A is defined by cutaneous symptoms only (rash, itching, flushing). Grade 1B includes skin manifestations plus either back pain or hypertension. Grade 2 includes urticaria, nausea, vomiting, throat tightness, asymptomatic bronchospasm, and/or chest tightness. Grade 3 is defined by symptomatic bronchospasm, dyspnea, hypoxia, and/or wheezing. Grade 4 includes anaphylaxis or hypotension.

Results:

A total of 11 patients (mean age 45.18 years; 8 females and 3 males) were identified. Of these, 8 were receiving treatment for pemphigus vulgaris and 1 for pemphigus vegetans. One patient had marginal zone B-cell lymphoma, and another had mycosis fungoides. Hypertension and diabetes were the most frequent comorbid conditions (n=5). None of the patients had any other known drug allergy. A minority of patients (n=3) had a history of atopy (eczema or seasonal allergies). Most reactions occurred during the first exposure to rituximab 1000 mg (n=7), and during the second exposure to rituximab (n=4) 5-15 min after perfusion. Patients reported systemic symptoms such as hypotension, oxygen desaturation, and respiratory discomfort. On the cutaneous level, pruritus, urticaria, and skin rash were observed. All our patients were treated with H1 antihistamines, steroids and resumption of infusion with a low flow rate of 50mg/hour. All reaction (n =11) were grade 1 or 2 and were rechallenged with rituximab on the same day as the initial reaction. Most patients with a grade 1 or 2 reaction tolerated rechallenge.

On the other hand, patients with a grade 2 reaction (n=2) experienced a reaction during rechallenge, which led to the decision to place them on a desensitization protocol (n=1), this protocol involved giving the patient increasing doses of the medication at a slow rate until the goal dose was reached, a decision also supported by pharmacovigilance results, with good tolerance.

Conclusion:

In line with the literature, our observations confirm that the majority of secondary reactions to rituximab occur

during the first infusion, typically due to cytokine release syndrome. We also suggest that patients with mild Grade 1 reactions to rituximab can safely be re-administered the same day, unlike patients with Grade 2 reactions, who may require desensitization.

Abstract N°: 6849

Spatial Transcriptomic Profiling Reveals Antiviral NK and Cytotoxic T Cell Responses Associated with Increased Severity in DRESS Syndrome

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Introduction & Objectives:

Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) syndrome is a life-threatening drug-induced hypersensitivity syndrome. Molecular mechanisms underlying its severity remain poorly understood. This study aimed to investigate the pathogenic mechanisms associated with increased disease severity in DRESS syndrome.

Materials & Methods:

Six lesional skin tissues were collected across three patients with DRESS syndrome. Disease severity was classified as mild, moderate, and severe based on the DDS severity scoring system. Spatial transcriptomic analysis was performed separately for epidermal and dermal regions of interest (ROIs) to compare transcriptomic profiles across different severity groups.

Results:

A total of 15 epidermal ROIs and 20 dermal ROIs were included in the analysis. In CD45+ immune cell–rich dermal areas, we identified 45 upregulated and 11 downregulated differentially expressed genes (DEGs) in the severe group compared to the mild group. Protein–protein interaction (PPI) analysis revealed activation of interferon-responsive signatures associated with natural killer (NK) cells and cytotoxic T cells in the severe group. Conversely, pathways related to skin barrier function and epithelial differentiation were downregulated in the severe group. In pancytokeratin+ epidermal areas, comparison between severe and mild groups revealed 6 upregulated and 38 downregulated DEGs. PPI analysis showed enrichment of antiviral response pathways and decreased expression of genes involved in skin barrier function and epithelial differentiation in the severe group.

Conclusion:

Our data suggest that increased cytotoxic immune responses and impaired skin homeostasis are key features of severe DRESS syndrome. Enhanced antiviral cytotoxic activity, involving NK cells and cytotoxic T cells, may play a pivotal role in exacerbating disease severity in DRESS syndrome.

Abstract N°: 6867

first documented case of dapagliflozin-induced acute generalized exanthematous pustulosis :an unusual adverse reaction

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First documented case of *Dapagliflozin*-induced acute generalized exanthematous pustulosis: an unusual adverse reaction

Introduction & Objectives:

Acute Generalized Exanthematous Pustulosis (AGEP) is a rare but potentially severe drug-induced hypersensitivity reaction. It requires careful clinical differentiation from other serious eruptive dermatoses, such as toxic epidermal necrolysis (Lyell syndrome), Stevens-Johnson syndrome, and drug-induced hypersensitivity syndrome. We present here the first reported case of AGEP induced by *Dapagliflozin*.

Materials & Methods:

NA

Results:

We describe the case of a 78-year-old male patient with a history of non-stented coronary artery disease and heart failure, who was being treated with spironolactone. Following a cardiac decompensation, *Dapagliflozin*, an SGLT2 inhibitor, was added to his treatment regimen. Three days after starting the medication, the patient developed a generalized skin rash characterized by confluent erythematous plaques with multiple non-follicular pustules. The lesions were primarily located on the trunk, face, and in the axillary and inguinal folds, accompanied by fever and a general decline in health. A diagnosis of AGEP was made after ruling out other potential causes and was confirmed by a EuroSCAR score of 9 and histopathological findings compatible with AGEP. The discontinuation of this medication was followed by an improvement and complete resolution of the lesions within 4 days.

Conclusion:

AGEP is a rare skin eruption, often induced by drugs. Commonly implicated drugs include pristinamycin, amoxicillin, quinolones, antibacterial sulfonamides, terbinafine, and diltiazem. The eruption typically occurs within 2 to 5 days after drug exposure. In this case, *Dapagliflozin*, an SGLT2 inhibitor widely used for treating type 2 diabetes and heart failure, was identified as the causative agent. Although generally well-tolerated, this medication can, in rare instances, cause severe adverse reactions, including hypoglycemia, euglycemic diabetic ketoacidosis, acute kidney injury, and Fournier’s gangrene. This case represents the first reported instance of AGEP induced by *Dapagliflozin*, emphasizing the need for heightened awareness when initiating this treatment. Management involves the immediate discontinuation of the offending drug and appropriate symptomatic treatment. This case highlights the importance of closely monitoring patients, particularly elderly individuals, when introducing new medications. The rarity of AGEP associated with *Dapagliflozin*, coupled with the lack of such reports in the

literature, underscores the need for thorough documentation of such cases to better understand the risks associated with this drug class. Increased vigilance and early recognition of this reaction are essential for optimizing patient management.

Abstract N°: 7003

Delayed Inflammatory Reaction to BDDE-Stabilized Hyaluronic Acid Fillers Triggered by Hordeolum in a Diabetic Patient

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Introduction & Objectives:

Hyaluronic acid (HA) fillers are among the most frequently used injectable materials for facial aesthetic enhancement due to their favorable safety profile and reversibility. However, delayed inflammatory reactions (DIRs), manifesting weeks to months after injection, have been increasingly recognized. These immune-mediated reactions can be triggered by systemic events such as infections, vaccinations, and dental procedures, and are often linked to hypersensitivity to filler components, particularly the cross-linking agent BDDE. We present a case of a delayed inflammatory reaction to BDDE-stabilized HA filler in a diabetic patient, likely triggered by a periorbital infection.

Materials & Methods:

A 61-year-old female patient with type 2 diabetes mellitus (treated with metformin 1000 mg/day) received HA filler injections (Croma®) on October 2, 2024, in the zygomatic area, tear troughs, nasolabial folds, chin, and lips. The immediate and intermediate post-procedure course was uneventful. On April 2, 2025—six months after the procedure—she developed right eye pruritus, erythema, and a medial hordeolum with mild suborbital edema. She was treated with oral amoxicillin-clavulanate (1000 mg BID), ofloxacin eye drops, and topical oxytetracycline. Despite improvement in ocular symptoms, she developed a firm subcutaneous nodule in the right zygoma within one week, followed by similar nodules in the left zygoma and chin. On April 12, she presented with prominent right-sided facial swelling, periorbital erythema, edema, and multiple firm nodules. MRI revealed residual filler deposits with surrounding inflammation and lymphatic obstruction. Ultrasonography confirmed localized HA-related nodules. A diagnosis of delayed-type inflammatory reaction to HA fillers was made.

Results:

The patient was started on systemic clindamycin (300 mg TID) and ciprofloxacin (500 mg BID). Intramuscular dexamethasone 8 mg was administered once daily for three consecutive days, followed by a tapering course of oral prednisolone: 48 mg/day (5 days), 32 mg/day (5 days), 16 mg/day (5 days), and 8 mg/day (5 days). A maintenance dose of prednisolone 4 mg/day was continued for an additional 5 days. Notable clinical improvement was observed with reduction in both edema and nodule size. No hyaluronidase was required.

Conclusion:

Delayed inflammatory reactions to HA fillers are likely immune-mediated and may be triggered by systemic events, even months after injection. Although a hordeolum is typically a localized eyelid infection, it may serve as an immune trigger in susceptible individuals. Diabetic status may predispose to altered immune responses and impaired lymphatic drainage, increasing the risk of filler-related complications. MRI and ultrasonography were instrumental in identifying filler remnants and guiding management. While no standardized treatment protocol exists for DIRs, combination therapy with systemic corticosteroids and antibiotics proved effective in this case.

Clinicians should maintain a high index of suspicion for DIRs in patients presenting with late-onset nodules and swelling after HA filler injections, particularly those with recent infections or comorbidities.

Abstract N°: 7052

Pembrolizumab associated acquired generalised lipodystrophy: a case report and literature review

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Introduction & Objectives:

Immune checkpoint inhibitors are fast becoming the mainstay of therapy for various malignancies enabling the immune system to recognise and target malignant cells. With their ever-increasing use, immune related adverse events (irAE) are becoming more common representing treatment challenges for physicians. Dermatological immune related adverse events (dirAE) present in up to 40 % of patients treated with immune checkpoint inhibitors and can range from psoriasiform to more severe cutaneous eruptions such as TEN/SJS.

Materials & Methods:

We would like to present the case of a 44 year old female patient with triple negative, node positive breast cancer who first presented to services in 2023 and was treated with the Keynote 222 regime from July 2023 to July 2024 including pembrolizumab.

Results:

In May of 2024 the patient notices significant lipoatrophy affecting her right upper arm. This rapidly progressed to include her bilateral upper limbs, back, gluteal area and thighs. Investigations including lipid profile were normal. Diffuse fatty infiltration was noted on liver ultrasound. Histology revealed foamy histiocytes and lymphocytes with normal epidermis in keeping with lipodystrophy.

Conclusion:

Acquired generalise lipodystrophy is a rare complication of immune checkpoint inhibition. To date only there are only 8 case reports of acquired lipodystrophy in the literature. We believe our case highlights an unusual presentation of dirAE's associated with immune checkpoint inhibition promoting early recognition amongst dermatologists resulting in effective intervention.

Abstract N°: 7098

Cutaneous Reaction at the Azacitidine Injection Site

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Introduction & Objectives:

Azacitidine is a DNA methyltransferase inhibitor and represents a key therapeutic option in the management of **myelodysplastic syndromes (MDS)**. It improves overall patient survival and prognosis. Azacitidine is administered subcutaneously (SC), a route that can lead to **severe localized reactions**, sometimes requiring treatment discontinuation. Such an interruption may worsen the course of MDS. Therefore, a careful **benefit-risk assessment** is crucial before deciding to stop the medication in the face of adverse effects.

We describe the case of a patient presenting with **recurrent cutaneous reactions** at the subcutaneous azacitidine injection site.

Materials & Methods:

We report the case of a **68-year-old woman** being treated for MDS with subcutaneous azacitidine injections.

Clinical history revealed the occurrence of **well-demarcated, oval erythematous plaques**, each surrounding the previous injection site of azacitidine, appearing within 24 hours of the injection, without any associated systemic symptoms. The lesions spontaneously regressed within a week, leaving behind **post-inflammatory hyperpigmentation**.

Clinical examination revealed a **well-circumscribed, erythematous plaque** at the current injection site, which was **very painful on palpation**, gradually increasing in size, with the presence of a **central blister** that appeared the same day as the injection. Post-inflammatory hyperpigmentation was also noted at the sites of previous lesions, without signs of active inflammation.

A diagnosis of **benign cutaneous reaction** at the azacitidine injection site was made. The patient was treated with **topical corticosteroids** at the injection site, leading to **marked improvement**: pain and erythema resolved by the **third day** of treatment, with signs of healing in the blistered area.

A **systematic application of topical corticosteroids** following subsequent injections was recommended, along with **oral antihistamines** and a **slow injection rate** for subcutaneous administration.

Results:

Cutaneous reactions at the azacitidine injection site are common. They are often **benign**, presenting as localized erythema that **resolves spontaneously**, even with continued treatment. These reactions are typically **managed symptomatically** and do not require discontinuation of the medication.

However, **more severe skin reactions**, such as **neutrophilic dermatoses** at the injection site, require a different approach, including **systemic corticosteroids**, akin to the management of systemic inflammatory reactions. **Histological examination** can be helpful in distinguishing **neutrophilic variants** from **erythematous types**.

A **thorough benefit-risk evaluation** is essential before considering discontinuation of azacitidine in cases of severe or systemic cutaneous reactions. **Enhanced collaboration with hematologists** can improve the

understanding and management of these **under-characterized dermatological manifestations**.

Conclusion:

This case highlights the importance of recognizing and accurately characterizing cutaneous reactions associated with azacitidine, particularly when they occur repeatedly at the injection site. Although such reactions may appear alarming, they are often benign and do not necessarily warrant discontinuation of treatment.

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Abstract N°: 7107

Dermatological adverse events to immune-checkpoint inhibitors in cancer patients: Impacton antineoplasic treatment adherence

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Introduction & Objectives:

Dermatological adverse drug events (DAEs) are among the most common complications associated with oncological therapies, presenting a broad spectrum of potential mucocutaneous reactions that significantly impact patients' quality of life and adherence to antineoplastic regimens. Immune checkpoint inhibitors present a peculiar safety profile, with immune-mediated DAEs generally well-tolerated. However, the growing number of patients undergoing these treatments requires a thorough understanding of the most frequent and clinically significant DAEs.

Materials & Methods:

This observational, retrospective, single-center study aims to assess the immune-mediated DAEs and the demographic and oncological characteristics of patients receiving immune checkpoint inhibitors. Additionally, it seeks to identify the drugs most frequently involved and evaluate their impact on oncological therapy, including whether these adverse effects lead to alterations or discontinuation of treatment.

Results:

Dermatological adverse effects were identified in a cohort of 160 patients treated with immunotherapy over a 7-year period at a tertiary care center. Overall, 26.3% of them required a change or suspension of oncological treatment due to DAEs. Eczematous dermatitis, maculopapular rash, and lichenoid eruption were the most prevalent manifestations, with the majority occurring during active treatment, at a median onset of 209 (SD 279.57) days post-initiation. Multivariate analysis revealed that liver, melanoma, and lung cancers were the most frequently associated with DAEs. Urological cancer presented the most severe toxicities (50% grade 3-4, p=0.025), followed by gynecological neoplasms with 45.5%. Urological cancers demonstrated the strongest correlation with treatment discontinuation. Moreover, anti-PD1 agents were most commonly implicated, with these drugs showing significant associations with treatment modifications. The factors significantly associated with ICI modification were the type and severity

of the immune-mediated DAEs (p = 0.002). Twenty percent of the total cohort required specific systemic

treatment options in addition to systemic steroids.

Conclusion:

Dermatological adverse effects are common in oncologic patients treated with ICI, frequently leading to treatment modification. Urological malignancies may be associated with a higher risk of severe DAEs and treatment discontinuation. Understanding the factors contributing to these adverse effects is crucial for optimizing oncologic therapy.

Abstract N°: 7125

Treatment of Toxic Epidermal Necrolysis by Plasmapheresis: A Prospective Study

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Introduction & Objectives:

Toxic epidermal necrolysis (TEN) is a severe drug-induced reaction characterized by mucocutaneous involvement and widespread epidermal detachment. Currently, there is insufficient evidence to support the routine use of any specific systemic therapy.

The objective of our study was to evaluate the effectiveness of early treatment with plasmapheresis combined with corticosteroid therapy in the management of TEN.

Materials & Methods:

We conducted a prospective study over a one-year period, from January 2023 to January 2024, in the Dermatology Department of Mohammed VI University Hospital in Oujda. The study included all patients diagnosed with TEN who received early plasmapheresis treatment, with or without concomitant corticosteroid therapy.

Results:

We included 6 patients with TEN. The mean age was 59 ± 14 years, with a female predominance (female-to-male ratio of 2:1). Allopurinol was the most frequently implicated drug (50% of cases), followed by flucloxacillin–amoxicillin combination (16.7%), fenoprofen (16.7%), and spiramycin combined with metronidazole (16.7%). The mean latency period between drug intake and onset of symptoms was 14 ± 10 days. The mean body surface area affected was 46% ± 15, with mucosal involvement observed in 83.3% of patients.

Corticosteroid therapy at a dose of 1 mg/kg/day was administered to 83.3% (5 out of 6) of patients. One patient did not receive corticosteroids due to sepsis. All patients underwent plasmapheresis using fresh frozen plasma, with sessions performed every two days, and an average of 4 ± 2 sessions per patient. A very good clinical outcome was observed in 4 out of 6 patients, with complete re-epithelialization achieved within 15 days of treatment. The mortality rate was 33.3%, with deaths attributed to cardiogenic pulmonary edema in patients with a history of ischemic heart disease.

Conclusion:

Plasmapheresis appears to be a promising therapeutic option in the management of TEN, although current evidence for its efficacy remains limited to isolated case reports and small case series. This technique enables the removal of the offending drug, its metabolites, and immune mediators involved in the inflammatory cascade of TEN. Further studies are needed to confirm its efficacy and establish standardized protocols in the treatment of toxic epidermal necrolysis.

Abstract N°: 7144

Fixed Drug Eruption in Diabetes: The Intersection of Drug Hypersensitivity and Metabolic Disease

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Introduction & Objectives:

characteristically recurs in the same anatomical sites upon re-exposure to the causative agent. White less common than exanthematous eruptions, PDE is frequently associated with antibiotics such as cotrimoxazole. Although the clinical and histopathological features of FDE are well described, its occurrence in patients with type 2 diabetes mellitus remains underreported. Given the altered immune response and microvascular changes in diabetes, the presentation or resolution of drug-induced skin reactions might differ. We present a case of cotrimoxazole-induced FDE in a patient with type 2 diabetes, highlighting a rarely discussed clinical context.

Case report:

A 56-year-old female patient with type 2 diabetes, treated with Diamicron for two years, presented with pruritic skin lesions accompanied by a few rapidly ruptured blisters, evolving over one day. These symptoms appeared one week after taking cotrimoxazole for a gastrointestinal condition, all occurring in an afebrile context and with preserved general condition. The patient was conscious, hemodynamically stable, and afebrile. Cutaneous examination revealed multiple well-defined, round to oval macular lesions of varying sizes, ranging from erythematous-purplish to brownish, scattered over healthy skin. These lesions were localized on the back, abdomen, lower limbs, and face—particularly in the fronto-mental region. Additionally, post-bullous erosions (1–3 cm in diameter) with central erythematous lesions and crusting were observed in the subumbilical area, right perinasal region, and the anterior aspect of the left leg. The examination also showed telangiectasias on the lower limbs, while mucous membranes and skin appendages were unremarkable.

A skin biopsy was performed, revealing focal epidermal atrophy, hyperorthokeratosis, and parakeratosis, with moderate spongiosis and keratinocyte necrosis. The dermis exhibited edema with a lymphoplasmocytic and eosinophilic inflammatory infiltrate, without signs of vasculitis or fungal spores (PAS-negative). These findings were consistent with a drug-induced toxidermia.

The final diagnosis was FDE , with cotrimoxazole identified as the causative agent. Management included immediate discontinuation of the offending drug, antihistamines, and high-potency topical corticosteroids. Follow-up showed resolution with residual hyperpigmented macules.

Conclusion:

This case illustrates a rarely reported association between fixed drug eruption and type 2 diabetes mellitus. While cotrimoxazole is a recognized cause of FDE, little is known about its behavior in diabetic patients. Diabetes may alter inflammatory responses and delay healing, potentially influencing the clinical course and sequelae of FDE. This underlines the importance of considering diabetes as a possible modifying factor in adverse cutaneous drug reactions. Further studies are needed to better understand the interaction between diabetes and drug hypersensitivity.

Abstract N°: 7247

When Treatment Turns Fatal: Lyell's Syndrome Induced by Sulfasalazine

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When Treatment Turns Fatal: Lyell's Syndrome Induced by Sulfasalazine

Introduction & Objectives:

Sulfasalazine has been used for over 40 years for the treatment of ulcerative colitis (UC). Despite its proven benefits in UC, the usefulness of sulfasalazine is limited by side effects. Exceptionally, adverse skin reactions to sulfasalazine can manifest as Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN). The incidence of SJS and TEN ranges from 0.4 to 1.2, and 1.2 to 6 per million person-years respectively. We report a new case.

Materials & Methods:

A case observational approach was used involving a 66-year-old male patient with confirmed diagnosis of Lyell's syndrome.

Results:

67-year-old woman admitted for a cutaneous-mucosal reaction suspected of being SJS evolving for 5 days. She had been treated for ulcerative colitis with salazopyrine. Physical examination revealed a diffuse rash over the whole body, purpura on the lower limbs, a 25% detached surface, an erosive patch on the genital mucosa, conjunctival hyperemia and erosive cheilitis.Biological workup showed normocytic normochromic anemia, hyperleukocytosis, C-reactive protein 54, hyperkalemia, correct natremia, impaired renal function, D-dimer 8680, troponin negative, PT and APTT normal, ECBU negative, rhabdomyolysis 5N.

The patient benefited from discontinuation of salazopyrin, reporting to pharmacovigilance, monitoring, bed bathing and application of a healing cream to the erosive plaques. Biopsy showed epidermal keratinocyte necrosis with subepidermal detachment, and a perivascular inflammatory infiltrate of lymphocytes in the dermis. The diagnosis of Lyell was accepted, and the evolution was marked by an alteration in general condition and an extension of the erosive placards, with a CS of 40%.

Conclusion:

Lyell's syndrome, is a rare but life-threatening dermatologic emergency, with sulfasalazine being an infrequent trigger. Due to its low incidence, its association with this drug may be easily unknown. This case highlights the necessity of recognizing TEN as a potential adverse reaction to sulfasalazine.

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Abstract N°: 7494

Pembrolizumab-Induced Bullous Pemphigoid: First Case Report from Algeria

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Introduction & Objectives: Pembrolizumab, a programmed death-1 (PD-1) immune checkpoint inhibitor, has significantly improved outcomes in patients with metastatic melanoma. However, its use is associated with a spectrum of immune-related adverse events (irAEs), particularly involving the skin. Among these, bullous pemphigoid (BP) is a rare but increasingly recognized cutaneous irAE. We present the first documented case of pembrolizumab-induced BP in Algeria.

Materials & Methods: A 69-year-old male with a history of type 2 diabetes mellitus, managed with metformin, was diagnosed with stage T4bN1b melanoma with lymph node, hepatic, splenic, and pulmonary metastases. He was initiated on pembrolizumab therapy in September 2023.

After approximately 12 months of treatment, the patient developed pruritic, ulcerated papules on the trunk and forearms. A presumptive diagnosis of pembrolizumab-induced prurigo was made. Topical clobetasol therapy resulted in partial improvement, allowing continuation of immunotherapy.

Subsequently, the patient experienced progressive worsening with each immunotherapy cycle, developing tense, clear or hemorrhagic bullae involving the trunk, extensor limbs, and inner thighs. Nikolsky's sign was negative, consistent with grade 4 toxicity. Skin biopsy with direct immunofluorescence demonstrated linear deposition of IgG and C3 along the basement membrane. ELISA testing revealed elevated anti-BP180 antibodies, confirming the diagnosis of bullous pemphigoid.

Systemic corticosteroid therapy (1 mg/kg/day of prednisone) was initiated, resulting in rapid clinical improvement. Immunotherapy was temporarily withheld for one cycle and subsequently resumed with close dermatologic monitoring.

Results: Bullous pemphigoid induced by immune checkpoint inhibitors results from the loss of immune tolerance, triggering autoantibody production against the dermo-epidermal junction. The latency between initiation of immunotherapy and BP onset typically ranges from several months to one year.

Diagnosis is based on clinical presentation, histopathological features, direct immunofluorescence, and serological markers. Management is guided by the severity of the cutaneous involvement. In moderate to severe cases, systemic corticosteroids are indicated. Decisions regarding continuation of immunotherapy should be individualized and require multidisciplinary coordination.

Conclusion: This case underscores the importance of early recognition and appropriate management of immune-related bullous dermatoses in patients receiving checkpoint inhibitors. Prompt diagnosis and intervention can enable the continuation of life-saving oncologic therapy while minimizing cutaneous morbidity

Abstract N°: 7587

Interest of dermocosmetics as supportive care for skin disorders associated with Graft-Versus-Host Disease

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Introduction & Objectives:

Graft-versus-host disease (GVHD) occurs when the healthy stem cells of the graft (the donor) attack the cells of the host (the recipient). Acute GvHD is a fairly common complication (30% of cases) of hematopoietic stem cell transplantation. Symptoms of GVHD can affect any organ, especially the skin, causing dryness, rash, itching or scaling. No study has looked at the benefits of emollients to relief clinical signs and symptoms of GVHD

Materials & Methods:

The aim was to evaluate the 7-day efficacy of a dermocosmetic (Emollient 'plus' containing microbiome-rebalancing ingredients Vitreoscilla filiformis microlysyl) with proven efficacy in atopic dermatitis (ref published ncbi.nlm.nih.gov/38553277). Patients with cutaneous manifestations of graft-versus-host disease (GVHD) were recruited from 2 centers in Paris: Hôpital Saint Antoine with 32 patients (76.2%) and Hôpital Saint Louis with 10 patients (23.8%). Patients were included upon the onset of cutaneous symptoms (Day 0), and their relatives and health professionals were invited to fill in a questionnaire at baseline (Day 0) and 7 days after symptom onset (Day 7). The product was made available to them

Results:

Of the 42 patients recruited, 41 completed the 7-day questionnaire for a response rate of 97.6%. All patients presented cutaneous symptoms of GVHD at inclusion (Day 0). The mean age at enrolment was 53.02 years. The gender distribution was comparable (47.6% males and 50% females). With regard to dry body parts, the feet (38.1%), head (19%), and hands (14.3%) were the most frequently affected areas. Other sites included forearms (7.1%), pelvis (4.8%), nose (4.8%), neck (9.5%), lips (4.8%), elbows (7.1%), arms (11.9%), chest (4.8%), and stomach (7.1%). Unpleasant sensations (tightness, pain, burning, pruritus) showed variation with specific percentages of improvement for each symptom. After 7 days, tightness had decreased by 10.1% and pain by 17.0%. Burning improved slightly by 2.4%, while pruritus decreased by 68.75%. With regard to desquamation, the results were as follows: improvement in 14 patients (33.3%), status quo in 22 patients (52.4%), and worsening in 5 patients (11.9%). Evaluation of erythema symptoms on day 7 showed various percentages of improvement: improvement in 9 patients (21.4%), status quo in 26 patients (61.9%), and deterioration in 7 patients (16.7%).

Use of the product was considered very easy by 51.35% of patients and fairly easy by 45.95%. The majority also reported that the product was absorbed quickly (81.08%) and did not stick (86.49%). In addition, 59.46% of patients reported that their skin was very soothed after application of the product and 40.54% described their skin as soothed.

Conclusion:

The results of our evaluation show a marked improvement in cutaneous symptoms of GVHD, including a significant reduction in pruritus (68.75%) and tightness (10.1%) after only 7 days of application. Tolerance and satisfaction among healthcare professionals and patients were high, with the application perceived as easy and with a marked soothing effect. These data confirm the efficacy of this Emollient 'plus' dermocosmetic as a supportive care product with the potential to improve the quality of life of patients with cutaneous GVHD. This study, which should be considered a feasibility study, has highlighted the opportunity to set up a more robust study in the future with a greater number of patients and centers to avoid center effects.

Abstract N°: 7628

Cyclosporine for the Treatment of Stevens - Johnson syndrome (SJS) and Toxic Epidermal Necrolysis (TEN): A Systematic Review of Observational Studies and Clinical Trials Focusing on Single Therapy, Combination Therapy, and Comparative Assessments

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Introduction & Objectives:

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are rare, severe, and potentially life-threatening skin and mucous membrane disorders. They are characterized by widespread skin and mucosal detachment and necrosis, and are classified based on the percentage of total body surface area (TBSA) affected. Given the severe and often life-threatening nature of these conditions, the identification and implementation of effective treatments is crucial. Notably, cyclosporin has demonstrated efficacy in managing these challenging conditions.

Materials & Methods:

A systematic search was carried out through the PubMed, Scopus, Embase, Web of Science, and Cochrane Library databases until May 2024. Additionally, a manual search was conducted through the reference lists of the included studies to minimize the risk of missing reports.

Results:

Overall, 17 studies involving 4761 patients were included in our analysis. The majority of the included studies suggested favorable outcomes for the use of cyclosporin in SJS/TEN patients. The use of cyclosporin was associated with improved survival rates, early arrest of disease progression, faster re-epithelialization, reduced length of hospital stays, and lower rates of multi-organ failure. However, a few studies did not find a survival advantage with cyclosporin and even reported an increased risk of mortality, as well as an increased TBSA detachment and risk of infection.

Conclusion:

Most studies indicate positive outcomes with cyclosporin treatment in SJS/TEN patients. This is likely due to cyclosporin’s immunomodulatory properties, which may help attenuate the severe inflammatory response associated with these conditions.

Abstract N°: 7674

Histopathological Features Distinguish Enfortumab Vedotin–Induced Skin Reactions from Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis: A Multicenter Observational Study

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Introduction & Objectives:

Enfortumab vedotin (EV) is an antibody-drug conjugate targeting Nectin-4, used in the treatment of advanced urothelial carcinoma. While EV has demonstrated therapeutic benefits, nearly half of treated patients develop skin disorders, likely due to Nectin-4 expression in epidermal keratinocytes. In severe cases, patients may present with erythema, blistering, and erosion, mimicking Stevens-Johnson syndrome/toxic epidermal necrolysis (SJS/TEN). As a result, EV-induced skin reactions are sometimes misdiagnosed as SJS/TEN, potentially leading to inappropriate management and premature discontinuation of EV therapy. This study aims to clarify the histopathological differences between EV-induced skin disorders and SJS/TEN, to improve diagnostic accuracy and avoid misclassification.

Materials & Methods:

In this retrospective observational study, we analyzed 23 patients who developed skin rashes following EV treatment at multiple institutions between November 2021 and April 2024. Clinical data—including dermatologic findings, systemic symptoms, laboratory values, and histopathological features—were collected and reviewed. Histological assessment focused on keratinocyte necrosis, atypical mitotic figures, blister formation, and inflammatory cell infiltration. All slides were independently reviewed and confirmed by a third-party dermatopathologist.

Results:

Based on clinical presentation, cases were categorized into two groups: those with intertriginous erythema without epidermal detachment (non-detachment group, n=13), and those with erythema accompanied by epidermal detachment (detachment group, n=5). The detachment group frequently presented with fever and widespread erosions, clinically resembling SJS/TEN. However, histopathological analysis revealed features distinct from SJS/TEN in EV-induced cases. These included keratinocyte necrosis with ring-shaped or starburst-shaped atypical mitotic figures, as well as involvement of eccrine sweat glands—findings that are typically absent in classic SJS/TEN. In addition, five cases presented with fine papules or vesicles resembling miliaria, representing one of the milder clinical presentations observed during EV treatment.

Conclusion:

EV-induced skin disorders demonstrate clinical overlap with SJS/TEN but are histopathologically distinct. Recognizing these differences is critical to avoid misdiagnosis and unnecessary discontinuation of EV therapy. Histopathological evaluation plays a pivotal role in distinguishing EV-associated reactions from SJS/TEN, and may help support continued EV use with appropriate monitoring and dose modifications.

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Abstract N°: 7736

A severe dermatosis of unusual etiology : Stevens-Johnson Syndrome following traditional Henna application

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Introduction:

Stevens-Johnson Syndrome (SJS) is a severe, potentially life-threatening dermatosis characterized by acute epidermal necrolysis. It is most often triggered by medications, but non-drug-related agents such as certain infections or cosmetic products can also be involved. We report here an atypical case of SJS likely triggered by traditional henna application, with possible secondary drug involvement.

Observation:

We present the case of a 43-year-old woman with undocumented medical history of a recovered stroke and childhood rheumatic fever, admitted for a generalized exanthem evolving over 10 days. The symptoms began 10 days after the application of traditional henna and worsened 4 days prior to admission following the use of a medication regimen including amoxicillin-clavulanic acid, flucloxacillin, antihistamines, montelukast, topical antiseptics, and topical antifungals. The patient's history also revealed a similar episode in her sister after applying the same henna, suggesting exposure to a common agent. Dermatological examination revealed widespread erythematous lesions with epidermal detachment on the feet, legs, and back. Flaccid bullae, as well as target and pseudo-target lesions, were noted on the forearms and inner thighs. The trunk showed confluent erythematous macules. Isolated conjunctival hyperemia was present, without significant mucosal involvement.

Discussion:

This case raises the possibility of Stevens-Johnson Syndrome being triggered by henna use. Although rare, this association has been reported in the literature, particularly in connection with para-phenylenediamine (PPD), a common additive in black henna known for its high allergenic potential. The clinical worsening after the administration of beta-lactam antibiotics, themselves known triggers of epidermal necrolysis, suggests a possible role for drug co-factors. This case highlights the importance of a detailed history that includes cosmetic or traditional products, which can be the source of severe reactions. The occurrence of a similar episode in a family member supports the hypothesis of sensitization to a shared component in the henna used.

Conclusion:

Stevens-Johnson Syndrome is a dermatological emergency primarily diagnosed based on clinical findings. This case underlines the importance of considering, beyond medications, less conventional triggers such as artisanal cosmetic products like henna. Medical and public awareness of this risk is essential, particularly in regions where henna use is widespread, in order to prevent serious complications.

Abstract N°: 7796

Severe Toxidermias Induced by Sulfasalazine: A Report of 9 Cases

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Introduction & Objectives:

Sulfasalazine, commonly prescribed for rheumatic and inflammatory bowel diseases, is generally well tolerated but can rarely cause severe toxidermias, which can be life-threatening. This study aims to describe the clinical and epidemiological characteristics of patients who developed severe toxidermia associated with sulfasalazine use.

Materials & Methods:

This is a retrospective and prospective descriptive study of severe sulfasalazine-induced toxidermias in patients hospitalized in the dermatology department at CHU Hassan II in Fez, between April 2018 and August 2024.

Results:

The study included 9 cases, with a mean age of 49 years and a female predominance (88.9%). One patient had a history of atopy, while diabetes and hypertension were present in another patient. Two patients were also taking NSAIDs concurrently. The majority of sulfasalazine prescriptions were for rheumatic conditions (88.9%), with a mean latency period of 34 days before symptom onset. General symptoms included fever (88.9%) and a deterioration in general health (66.7%). Cutaneous manifestations included erythroderma (66.7%), maculopapular rash (55.6%), skin detachment (22.2%), and purpura (55.6%). Facial edema was present in 88.9% of patients, while mucosal involvement was observed in 55.6% of cases. Systemic involvement primarily affected the liver (88.9%), with one case of acute renal failure and another with cerebral vasculitis. Biologically, 66.7% of patients had eosinophilia, 22.2% had lymphopenia, and 33.3% had hyponatremia. The most common diagnosis was DRESS syndrome (88.9%), with a single case of Lyell syndrome (TEN). The overall outcome was fatal in two patients, both of whom died from sepsis. Treatment included topical corticosteroids (66.7%) and systemic corticosteroids for severe cases. Recovery was observed in 66.7% of patients, with 44.4% retaining cutaneous and nail sequelae.

Conclusion:

This study underscores the importance of careful monitoring when prescribing sulfasalazine and highlights the need for close follow-up to minimize complications and improve patient outcomes.

Abstract N°: 7915

Bleomycin-induced Flagellate Erythema in a Patient with Mediastinal Seminoma: A Case Report

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Introduction: Flagellate erythema or flagellate dermatitis is characterized by pruritic linear streaks of erythematous or brown patches and plaques, presenting with a whip-like appearance. The lesions are associated with various etiologies like consumption of shiitake mushrooms, rheumatologic disorders, and drugs, among others. Its association with bleomycin as an adverse reaction was first reported in \1970. It is a rare phenomenon with a reported incidence of only 8 to 20%.

Case Report: This is a case of a 29-year-old male with mediastinal seminoma who was started on bleomycin, etoposide, and cisplatin (BEP) regimen. He developed pruritic erythematous papules and linear streaks of erythematous and brown patches and plaques, most prominent on the trunk and extremities ten days after starting the chemotherapy regimen cycle. The initial presentation was subtle, with worsening after continued administration of bleomycin. A skin punch biopsy showed spongiotic interface dermatitis with eosinophils, and the patient was managed symptomatically.

Conclusion: Flagellate erythema is a rare form of cutaneous adverse reaction to bleomycin. It may initially present with subtle skin changes but the temporality of bleomycin administration and the evolution of the lesions, becoming whip-like linear streaks, may guide clinical diagnosis. The disease course is varied and histopathologic findings are nonspecific. Treatment involves discontinuation of bleomycin and supportive management of symptoms.

Abstract N°: 7959

Generalized Bullous Fixed Drug Eruption Mimicking Toxic Epidermal Necrolysis Caused by Griseofulvin

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Introduction & Objectives:

Fixed drug eruption (FDE) is a cutaneous adverse drug reaction presenting as recurrent, round, erythematous to violaceous patches that often recur at the same site upon re-exposure to the culprit drug. Repeated exposure can lead to increased number and size of lesions, and rarely, a generalized distribution with extensive skin detachment. Generalized bullous FDE (GBFDE) is a rare and severe variant of FDE, characterized by widespread bullae and erosions mimicking Stevens–Johnson syndrome (SJS) or toxic epidermal necrolysis (TEN). Common causative agents include paracetamol, NSAIDs, trimethoprim-sulfamethoxazole, systemic antifungals, antibiotics, and psychotropic medications. Differentiating GBFDE from SJS/TEN is critical due to overlapping features but differing management and prognosis.

Materials & Methods:

A 47-year-old male without prior comorbidities presented with a 3-day history of fever followed by generalized erythematous to dusky patches, and subsequent blister formation with spontaneous rupture. He reported ocular irritation and oral erosions. The patient had been self-medicating for tinea corporis with weekly fluconazole and, on the day of symptom onset, had taken a dose of griseofulvin. On detailed history, he recalled a similar self-limiting reaction after griseofulvin intake 6 months earlier. On examination, multiple flaccid bullae and erythematous patches were noted over the trunk and extremities with positive Nikolsky sign and skin tenderness. No target lesions were seen. Mucosal involvement was limited to oral erosions; ocular and genital mucosa were normal.

Results:

Laboratory tests revealed neutrophilic leukocytosis and elevated C-reactive protein. Liver and renal function tests were within normal limits. Skin swab culture was positive for methicillin-sensitive *Staphylococcus aureus*. Histopathology from lesional skin showed necrotic keratinocytes, interface dermatitis, and upper dermal inflammatory infiltrate with eosinophils, neutrophils, and lymphocytes. The patient was diagnosed with GBFDE. Treatment included intravenous dexamethasone 6 mg every 6 hours which was given for 48 hours, oral co-amoxiclav, and topical steroids and antibiotic ointments. Rapid clinical improvement was noted within 36 hours with subsiding skin tenderness and arrest in lesion progression. The patient was discharged after five days of hospitalization.

Conclusion:

GBFDE is a rare but potentially severe drug reaction that may closely mimic SJS/TEN. A detailed history of prior drug exposure and lesion recurrence at the same sites is crucial for diagnosis. Histopathology plays an important role in distinguishing GBFDE from SJS/TEN. Prompt withdrawal of the offending agent and initiation of systemic corticosteroids are vital for management. In severe cases, treatment may need to align with SJS/TEN protocols using systemic immunosuppressants like cyclosporine. Awareness and early recognition of GBFDE can significantly improve patient outcomes.

Abstract N°: 8015

A Rare Adverse Effect of Ribociclib: Vitiligo-like Lesions in a Patient with Metastatic Breast Cancer

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Introduction & Objectives:

Ribociclib, a cyclin-dependent kinase 4 and 6 inhibitor, is an antineoplastic agent used in the first-line treatment of hormone receptor-positive, human epidermal growth factor receptor 2 (HER2)-negative metastatic breast cancer in combination with an aromatase inhibitor. Hematologic side effects are common during treatment, but cutaneous adverse effects such as alopecia, pruritus, skin rash, and, rarely, vitiligo-like lesions have also been reported.

Materials & Methods:

Herein, we present a female patient with metastatic hormone receptor-positive, HER2-negative breast cancer who developed vitiligo-like skin lesions during ribociclib therapy, diagnosed based on the clinical findings.

Results:

A 69-year-old female patient presented to our clinic with a 5-month history of pruritus over the legs, followed by vitiligo-like lesions on the elbows and legs. The patient had previously undergone modified radical mastectomy, followed by adjuvant chemotherapy with cyclophosphamide, doxorubicin, and paclitaxel with a diagnosis of hormone receptor-positive, HER2-negative breast cancer. After lung and bone metastases were detected, she received ribociclib in combination with letrozole for 8 months. She had no personal or family history of autoimmune disease. She did not described trauma. A dermatological examination revealed symmetrical hypopigmented macules and patches with erythematous borders on the arms, forearms, hands, thighs, legs, and feet. Wood's light examination showed sharply demarcated depigmented lesions. Based on the history and clinical findings, the diagnosis of ribociclib-induced vitiligo-like lesions was made. According to the World Health Organization Causality Assessment Scale and the Naranjo Adverse Drug Reaction Probability Scale, the case was classified as a "probable" adverse drug reaction. Treatment included topical corticosteroids and topical tacrolimus, along with photoprotection. However, we did not observe a significant response to the treatment, likely due to the continuation of ribociclib therapy.

Conclusion:

Vitiligo-like lesions, unlike vitiligo, are characterized by Koebner-negative, depigmented macules on sun-exposed areas. Although the pathogenesis is not fully understood, they have been reported as side effect during oncological treatment, including tyrosine kinase inhibitors, and PD1 inhibitors.

Ribociclib acts as a cyclin-dependent kinase 4 and 6 inhibitor, thereby blocking the progression from the G1 to the S phase of the cell cycle. Ribociclib-induced cell cycle arrest may culminate in the premature death of melanocytes, manifesting as depigmented lesions.

Depigmented lesions typically follow pruritus and erythema in both sun-exposed and non-sun-exposed regions, and involving more than 25% of the body surface area. Topical corticosteroids and calcineurin inhibitors remain the prevailing treatment modalities, though ribociclib-induced vitiligo-like lesions have been reported to be more resistant to treatment than vitiligo.

In a previously reported case series, tumor regression coincided with the development of vitiligo-like lesions in 12 out of 14 patients, suggesting a potential favorable prognostic indicator. Further research is needed to better understand the implications of ribociclib-induced vitiligo-like lesions. Nonetheless, recognizing and managing this adverse effect is essential due to its potential impact on patients' quality of life.

Table 1. Naranjo Adverse Drug Reaction Probability Scale for our case

Question	Yes	No	Do Not Know	Answer	Score
1. Are there previous conclusive reports on this reaction?	+1	0	0	Yes	+1
2. Did the adverse event appear after the suspected drug was administered?	+2	-1	0	Yes	+2
3. Did the adverse event improve when the drug was discontinued or a specific antagonist was administered?	+1	0	0	Do Not Know	0
4. Did the adverse event reappear when the drug was readministered?	+2	-1	0	Do Not Know	0
5. Are there alternative causes that could on their own have caused the reaction?	-1	+2	0	No	+2
6. Did the reaction reappear when a placebo was given?	-1	+1	0	Do Not Know	0
7. Was the drug detected in blood or other fluids in concentrations known to be toxic?	+1	0	0	Do Not Know	0
8. Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	+1	0	0	Do Not Know	0
9. Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	+1	0	0	No	0
10. Was the adverse event confirmed by any objective evidence?	+1	0	0	Yes	+1
Total Score ≥9 : Definite. **Total Score 5 to 8: Probable.** **Total Score 1 to 4: Possible.** **Total Score ≤0: Doubtful.**				**Total Score: 6 Probable**	

Abstract N°: 8033

Metronidazole Induced Photo-Distributed Cutaneous Vasculitis: A Case Report

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Introduction & Objectives:

Metronidazole, a nitroimidazole, is used for treating conditions like gastrointestinal infections, bacterial vaginosis, rosacea, and lichen planus. Side effects are mainly neurological and digestive, with rare cutaneous reactions. We report a case of photo-distributed vasculitis induced by metronidazole, where the timeline of events suggests its causality.

Materials & Methods:

We present a 28-year-old female patient with no significant medical history, consulting for the appearance of purpuric lesions on photo-exposed areas, which occurred two days after starting metronidazole treatment at a dose of 1000 mg/day for suspected bacterial vaginosis. Clinical examination revealed petechial, ecchymotic, sometimes infiltrated purpura, located around the face, neck, nape, as well as on the dorsal surfaces of the hands and feet, with no pruritus or mucosal involvement, and no associated symptoms, including no arthralgia or abdominal pain.

Systemic vasculitis immunological tests and viral serologies were negative. Histology of a skin biopsy showed keratinocyte necrosis, a perivascular infiltrate rich in lymphocytes, eosinophils, and neutrophils, penetrating the vessel walls without wall necrosis. Direct immunofluorescence was negative. Management consisted of discontinuing all non-essential medications, administering an antihistamine, topical corticosteroid, and an emollient. The condition improved after two weeks.

The diagnosis of metronidazole-induced vasculitis was confirmed based on chronological, clinical, histological, and evolutionary evidence, as well as a likely imputability score.

Results:

Drug-induced vasculitis represents 10-20% of cutaneous vasculitis cases. Its mechanism is multifactorial, involving immune complexes, ANCA, and/or cellular mechanisms, as well as genetic factors. Symptoms typically appear three weeks after the start of treatment, but may occur anywhere from two days to ten years. Metronidazole-induced toxidermias are mainly described in the literature as fixed pigmented erythema, contact dermatitis, Quincke’s edema, anaphylaxis, serum sickness-like reactions, Stevens-Johnson syndrome/toxic epidermal necrolysis, acute generalized exanthematous pustulosis, a case of DRESS, and photosensitivity. The case of photo-distributed vasculitis observed in our patient has, to our knowledge, never been reported in the literature. The drug imputability score (I3 B1) indicates a likely causality. Further investigations are needed to better understand the diagnostic.

Conclusion:

This observation contributes to expanding the current knowledge on the cutaneous side effects of metronidazole, with the aim of preventing potentially severe complications. Management appears to be effective when it involves the immediate discontinuation of the suspected treatment and appropriate symptomatic therapy.

Abstract N°: 8037

Drug reaction with eosinophilia and systemic symptoms triggered bycarbamazepine : Cross-reactivity to lamotrigine confirmed by rechallenge

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Introduction & Objectives:

Drug reaction with eosinophilia and systemic symptoms (DRESS syndrome) is a serious idiosyncratic drug reaction, associated with a high mortality rate and triggered commonly by antiepileptic drugs (AEDs). The frequency of cross- reactivity among anticonvulsants (carbamazepine, phenytoin, phenobarbital, and primidone) is as high as 40%—80% among aromatic AEDs explained by their common aromatic ring. Nevertheless, cross-reactivity between aromatic and non-aromatic AEDs (valproic acid, lamotrigine, and levetiracetam.) is less commonly reported. We report a case of carbamazepine (CMZ) induced DRESS with a cross-reactivity to a non-aromatic AED (lamotrigine).

Materials & Methods:

NA

Results:

A 35-year old female was newly diagnosed with bipolar disorder and treated with CMZ, risperidone and biperiden. Twenty-five days later, she presented to the emergency room with a four-day history of fever (39.8°C), itchy diffuse maculopapular exanthema, a facial oedema. two palpable lymphadenopathy (Figure 3), with no mucosal involvement. Laboratory investigations revealed prominent eosinophilia (5600 /µL), atypical lymphocytes and sever hepatic cytolysis (AST of 1230 IU/l, ALT of 1910 IU/l) and prothrombin time of 40%.Serological tests for viral and bacterial infections, including hepatitis A, B, C, CMV, EBV, Chlamydia and Mycoplasma, were all negative assessed by polymerase chain reaction. With a score of 7 points, DRESS syndrome is considered to be « definite », according to the RegiSCAR group and CMZ, risperidone and biperiden were withdrawn. Supportive treatment was started for symptomatic relief. The clinical and biological symptoms regressed and disappeared approximately four weeks later. A patch test to CBZ (20% petrolatum), carried out two months after total recovery, yielded positive result. To evaluate cross-reactivity to other AEDs, we performed patch tests to phenobarbital test (10% saline solution) and lamotrigine (10% petrolatum) with negative results. Eight months later, lamotrigine was started. However the patient re-experienced on the 20 th day a morbilliform eruption with an increased eosinophilia count of 530/µL) with no systemic organ involvement. The clinical and biological picture resolved after lamotrigine discontinuation, with 2 weeks.

Conclusion:

According to the Naranjo Causality Scale, the accountability of both drugs was found to be

probable (score of ‘6’). Through this report, we add to the medical literature a new case of DRESS syndrome induced by CMZ, cross-reacting to lamotrigine, anon aromatic AED. This was confirmed by a positive rechallenge to lamotrigine, with a negative result on PT.

Abstract N°: 8038

Paradoxical Psoriasiform and Hidradenitis Suppurativa-like Reactions Induced by Anti-TNF Therapy in a Patient with Ulcerative Colitis: Diagnostic and Therapeutic Challenges

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Introduction & Objectives: Paradoxical skin reactions such as psoriasis and hidradenitis suppurativa (HS)-like lesions are increasingly recognized as adverse events associated with anti-TNF therapy. These manifestations pose significant diagnostic and therapeutic dilemmas, particularly in patients with inflammatory bowel disease (IBD), where biologics are central to disease control.

Materials & Methods: We report the case of a 43-year-old female with a 3-year history of ulcerative colitis (UC), initially managed with mesalazine and corticosteroids, followed by escalation to adalimumab in 2023 due to disease progression. After one year of apparent disease control, the patient developed painful inflammatory nodules in the axillary and inframammary regions, with progressive extension to the thighs—clinically consistent with hidradenitis suppurativa. Despite antibiotic treatment and a switch to infliximab, she subsequently developed erythematous, scaly psoriasiform plaques on the scalp and lumbar area. These skin findings, atypical for her personal history and temporally associated with anti-TNF therapy, were interpreted as paradoxical cutaneous reactions. Infliximab was discontinued, and systemic corticosteroids were initiated, leading to temporary improvement. Due to recurrence after tapering, a transition to ustekinumab was proposed, targeting IL-12/23 pathways implicated in both psoriasis and HS, as well as UC.

Results: This case underscores the paradoxical emergence of psoriasiform and HS-like lesions under anti-TNF agents, an increasingly reported phenomenon with unclear pathogenesis. These reactions often mimic idiopathic dermatoses but require recognition as drug-induced to avoid misdiagnosis and therapeutic delay. The case illustrates the need for interdisciplinary collaboration in managing overlapping autoimmune and autoinflammatory conditions and supports a personalized treatment strategy based on cytokine profiling and disease burden.

Conclusion: Paradoxical dermatologic reactions during anti-TNF therapy represent a significant therapeutic challenge in IBD patients. Switching to an alternative biologic class, such as IL-12/23 inhibitors like ustekinumab, may provide effective disease control across mucocutaneous systems, particularly when paradoxical reactions compromise treatment efficacy and quality of life.

Abstract N°: 8053

Ichthyosiform Erythroderma in a 3-Month-Old Infant Following Topical Argan Oil Application: A Culturally Rooted Allergen?

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Introduction & Objectives:

In Morocco, the topical use of natural oils—particularly Argan oil—is a deeply embedded cultural practice. Argan oil, extracted from the nuts of *Argania spinosa*, is rich in tocopherols, phenols, fatty acids, and other bioactive compounds. It is widely used for skin and hair care, even in infants, and is traditionally viewed as safe. However, this presumed innocuity is occasionnaly challenged by reports of adverse effects, including two published cases of erythroderma and contact dermatitis, and three cases of Argan-related occupational asthma. We report what appears to be the first case of ichthyosiform erythroderma in an infant triggered by topical Argan oil.

Materials & Methods:

A 3-month-old infant with no prior medical history was admitted for generalized erythema with overlying ichthyosiform scaling, which developed within 24 hours of Argan oil application. The infant was apyretic, with no hepatosplenomegaly or lymphadenopathy. There was no consanguinity and no personal or family history of allergic or dermatological conditions. Laboratory workup—including CBC, liver and renal function tests, and viral serologies—was normal. Given the patient's age, no skin biopsy or allergology patch testing was performed.

Results:

Symptomatic treatment with intensive emollients and encouragement of frequent breastfeeding to prevent dehydration led to rapid improvement. Complete regression of the erythroderma and scaling was noted within three days. The absence of systemic signs, normal investigations, and favorable evolution under supportive care argued against differential diagnoses such as Leiner-Moussous disease, Psoriatic erythroderma, or viral exanthema. An acute cutaneous reaction to Argan oil was retained as the most likely diagnosis. Eviction of Argan oil was advised; no broader dietary or allergen restrictions were deemed necessary.

Conclusion:

This case illustrates a rare but relevant example of Argan oil-induced ichthyosiform erythroderma in infancy. Despite its cultural ubiquity and perceived harmlessness, Argan oil can act as a potential contact allergen. Physicians should remain aware of such reactions, particularly in regions where traditional skin remedies are commonly used.

Abstract N°: 8065

Bullous Generalized Fixed Drug Eruption in a Child: A Rare Clinical Entity

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Introduction & Objectives:

Bullous fixed drug eruption (FDE) is a severe form of toxidermia and a rare variant of fixed drug eruption. It can be localized or generalized, often mimicking Lyell's syndrome. As a result, it is frequently underdiagnosed or misdiagnosed as other bullous dermatoses. We report a case of bullous generalized fixed drug eruption induced by ibuprofen in a child.

Materials & Methods:

Results:

The patient, an eight-month-old child, with no significant medical history, presented with diffuse skin lesions that had evolved over three days, along with a mild fever. The history revealed ibuprofen intake 48 hours before the eruption started. The infant's general condition remained stable. Dermatological examination revealed four well-demarcated, round post-bullous erosive lesions, each measuring 12 cm in diameter, with hemorrhagic centers and hyperpigmented borders. These lesions were asymmetrically distributed on the face, trunk, left arm, and scrotum, with large areas of healthy skin in between. Examination of the mucous membranes and appendages revealed no abnormalities, and the rest of the physical exam was unremarkable.

At this point, the differential diagnosis included bullous generalized fixed drug eruption, toxic epidermal necrolysis (TEN), bullous erythema multiforme, and bullous impetigo. Pharmacological review identified ibuprofen as the triggering agent. The blood count was normal, C-reactive protein (CRP) was slightly elevated at 20 mg/L, and skin swabs were sterile. A skin biopsy was not performed. Given the highly suggestive clinical presentation and anamnesis, a diagnosis of bullous generalized fixed drug eruption was confirmed. Ibuprofen was discontinued, and treatment with emollient and reparative creams was initiated. The lesions healed within seven days after stopping the implicated drug, leaving behind residual hyperpigmentation.

Conclusion:

Fixed drug eruption is generally a benign form of toxidermia, but bullous forms are rare and represent severe cases of bullous generalized fixed drug eruption. Distinguishing this condition from toxic epidermal necrolysis is challenging, underscoring the importance of recognizing the specific clinical features of each entity.

Abstract N°: 8089

Unmasking the Offending Agent: A Prospective Study of Cutaneous Adverse Drug Reactions to Anti-Tubercular Therapy Using Rechallenge and Skin Tests

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Introduction & Objectives:

Tuberculosis treatment frequently results in cutaneous adverse drug reactions (CADRs), which can significantly impact patient adherence and treatment success. Identifying the specific drug responsible remains a challenge due to polypharmacy and the lack of standardized diagnostic tools. Patch and prick testing are potential methods for evaluating delayed hypersensitivity reactions, and supervised drug rechallenge remains a valuable tool for confirming causality.

This study aimed to evaluate the demographic profile, clinical patterns, and implicated drugs in CADRs due to anti-tubercular therapy (ATT), and to assess the role of patch/prick testing and rechallenge in identifying the offending agent.

Materials & Methods:

A prospective study was conducted over two years at a tertiary care centre in Mumbai. Patients aged >18 years with CADRs attributed to anti-tubercular therapy (ATT) were enrolled based on defined inclusion criteria. A supervised drug rechallenge with individual ATT agents was performed in a stepwise manner under close clinical observation, with the aim of identifying the causative drug and confirming the diagnosis. Patch testing was performed in consenting patients between 4 weeks and 1 year after resolution of skin lesions, with standard readings taken at 20 minutes, Day 2–4, and Day 7. Prick testing was also conducted in selected cases, with readings at 20 minutes.

Results:

CADRs to ATT were evaluated in 43 patients. The most common pattern was drug-induced hyperpigmentation (21.4%), followed by acneiform eruptions (19%), DRESS and lichenoid dermatitis (14.3% each), bullous fixed drug eruption (14%), prurigo and maculopapular rash (4.7% each), and drug-induced pellagra and erythema multiforme (2.4% each). Rifampicin (23.3%) was the most frequently implicated drug, followed by clofazimine (20.9%) and isoniazid (11.6%). Levofloxacin accounted for 4.7%, while ethionamide, moxifloxacin, and pyrazinamide were responsible in 2.3% each. In 32.6% of cases, the offending drug could not be identified. A female predominance (66.6%) was observed, with the highest incidence in the 25–45 year age group. Acneiform eruptions were more common among unemployed individuals. Patch testing was performed in 10 patients, and prick testing in 4; all results were negative and failed to confirm the causative agents. In all other cases where a specific drug was suspected, supervised drug rechallenge was performed to establish causality.

Conclusion:

This study highlights the diverse clinical spectrum of CADRs due to anti-tubercular therapy, with rifampicin, clofazimine, and isoniazid being the most frequently implicated agents. Patch and prick testing failed to confirm drug causality, reflecting limited utility in this setting. Rechallenge, although requiring careful supervision, proved to be the most reliable method for establishing drug causality in CADRs to ATT.

Abstract N°: 8152

Symmetrical drug-related intetriginous and flexural exanthema is considered a benign rash due to its self limited course and complications are exceptional

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Introduction & Objectives: Symmetrical drug-related intertriginous and flexural exanthema (SDRIFE), is a rare form of drug eruption which is caused by delayed Type-IV immune reaction. SDRIFE has a short latency of 2-5 days due to drug directly binding to T-cell receptors. SDRIFE is clinically defined by five criteria: (i) onset after initial or repeated exposure to a systemically administered drug (contact allergens excluded) , (ii) sharply demarcated erythema in the gluteal/perianal area and/or V-shaped erythema in the inguinal/perigenital region, (iii) involvement of at least one other interginious/flexural fold (e.g. axillae, antecubital fossae), (ıv) symmetrical distrubiton, and (v) absence of systemic involvement. Antibiotics, in particular beta-lactams , comprise the majority of causes of SDRIFE. The clinical course of SDRIFE is usually benign and self-limited.

Materials & Methods: We present a patient presented with symmetrical drug-related intertriginous and flexural exanthema due to intravenous asiclovir use.

Results: A 51 year-old female patient was admitted to the emergency department with complaints of neurologic deterioration and was hospitalized with a diagnosis of viral encephalitis. Intravenous acyclovir treatment was started from the day of admission and the patient was evaluated by the dermatology department on the 5th day of treatment because of generalized rashes on the body. Patient had no systemic symptoms. On examination, erythematous plaques were seen in bilateral axillary area, bilateral inguinal fold area, bilateral gluteal and lumbosacral areas, all symetrically.However, there was no involvement of the oral mucosa. Blood results showed mild eosinophilia and mildly elevated white blood cells. The patient refused a biopsy for histopathologic diagnosis so the patient was clinically diagnosed with SDRIFE. Despite this patient continued to receive acyclovir 14 days as viral encephalitis because of treatment could not be interrupted. Systemic antihistaminic and topical corticosteroid were given as treatment. The lesions completely resolved with symptomatic treatment.

Conclusion: In the literature ,only a few cases of SDRIFE due to antiviral agents have been reported. Two of these were due to valacyclovir,one was due to acyclovir and one was due to remdesivir. Clinical signs regressed in all patients with drug discontinuation. SDRIFE is considered a benign rash due to its self limited course and complications are exceptional.

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Abstract N°: 8199

Upadacitinib Induced Facial Erythema and Edema in Atopic Dermatitis: A Paradoxical Adverse Event

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Introduction & Objectives:

Upadacitinib is a selective Janus kinase 1 (JAK1) inhibitor approved for moderate to severe atopic dermatitis (AD). JAK inhibitors offer effective symptom control by suppressing Th2-mediated inflammation; however, paradoxical cutaneous adverse effects are increasingly recognized. While dupilumab is commonly associated with facial and neck erythema, JAK inhibitors are often considered alternatives in such cases. Here, we present a case in which upadacitinib, paradoxically, induced severe facial erythema and edema.

Materials & Methods:

Results:

A 29-year-old female with long-standing moderate-to-severe AD was started on upadacitinib 15 mg/day following insufficient response and accessibility to prior treatments (topical agents, cyclosporine, phototherapy, baricitinib). After three months of upadacitinib, she developed sharply demarcated facial and neck erythema, edema, and worsening pruritus. Physical examination showed widespread erythematous plaques with edema and desquamation confined to the face and neck. Laboratory tests and skin evaluations ruled out infections, contact allergens, and Demodex. Antifungal therapy was ineffective. Upadacitinib was discontinued and pimecrolimus cream was initiated; subsequent clinical evaluation revealed partial regression of facial erythema following drug discontinuation. No clear exogenous triggers were identified. The reaction was classified as probable (Naranjo score = 6), suggesting a drug induced mechanism.

Conclusion:

Facial dermatitis is a well-documented adverse event during dupilumab therapy, potentially linked to Malassezia hypersensitivity, rosacea, or allergic contact dermatitis. While JAK inhibitors are typically used to manage such cases, we report a rare paradoxical reaction where facial dermatitis was induced by JAK inhibition with upadacitinib. The exact mechanism remains unclear, but one plausible explanation involves the disinhibition of Th1 and Th17 pathways secondary to the suppression of Th2 cytokines such as IL-4 and IL-13. This immunological shift may lead to an overexpression of pro-inflammatory cytokines like IL-17 and IL-22, promoting localized inflammation, epidermal barrier disruption, and microbial dysbiosis, particularly involving facial skin, which is rich in sebaceous glands and microbial colonization. This case is notable in reversing the expected pattern where a JAK inhibitor induced, rather than resolved, facial dermatitis. Such paradoxical effects underscore the complex immunologic balance in AD and highlight the need for close monitoring of cutaneous responses, even with targeted therapies.

In conclusion, while JAK inhibitors like upadacitinib provide substantial benefits, they may also cause paradoxical reactions that necessitate careful monitoring and management. Further research is needed to clarify the mechanisms of these events to refine treatment approaches.

Abstract N°: 8204

Impact of dermatological toxicities on quality of life in breast cancer patients exposed to adjuvant endocrine therapy

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Introduction & Objectives:

The BCARE (Breast Cancer Adjuvant Real-world Evaluation of dermatological adverse events) study aims to evaluate the impact of dermatological toxicities on the quality of life (QoL) in patients with early-stage breast cancer (EBC) treated with adjuvant endocrine therapy. The development of dermatological toxicities, such as skin inflammation, hair/nail changes, and dry skin, and their potential impact on patients' physical, functional, emotional, and social well-being has not been studied until now. Despite the improved efficacy of anticancer therapies, managing these dermatological toxicities remains a challenge. This study aims to fill the gap in literature regarding the dermatological adverse effects experienced by these patients and their significant impact on physical, emotional, and social well-being.

Materials & Methods:

This cross-sectional, non-interventional study will use the Dermatology Life Quality Index (DLQI) questionnaire as primary outcome to measure Quality of Life related to dermatological toxicities in female patients aged 18 and above with histologically confirmed EBC undergoing adjuvant endocrine monotherapy initiated 2 to 3 years ago.

It is being conducted across nine centers in four countries (France, Greece, Italy, and Spain; FPI Nov 22, 2024). Patients who have received concomitant targeted therapies and/or have had relevant or severe comorbidities are excluded. Secondary outcomes include dermatological QoL assessed using Skindex-16, Hairdex, and ItchyQoL questionnaires, and the description of dermatological toxicities at inclusion.

Results:

The study will collect and analyze demographics, clinical characteristics and data collected during a single visit on Quality of Life through questionnaires completed by patients, and dermatological toxicities from medical charts. The primary endpoint is the rate of patients with a DLQI score ≥ 6, indicating moderate to severe impact on QoL. All the dermatological toxicities will be assessed by type and by grade and classified according to the final impact on QoL

50 patients have been included to date and all the centres are actively recruiting patients.

Conclusion:

The BCARE study highlights the significant impact of dermatological toxicities on the quality of life in breast cancer patients undergoing endocrine therapy. By providing comprehensive data on these adverse effects, the study aims to inform better management strategies and improve patient care. The findings underscore the need for targeted interventions to mitigate dermatological side effects and enhance treatment adherence and overall well-being.

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Abstract N°: 8247

A Rare Lingual Manifestation: Black Hairy Tongue Associated with Linezolid Use

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A Rare Lingual Manifestation: Black Hairy Tongue Associated with Linezolid Use

Introduction & Objectives:

Black hairy tongue (BHT) is a benign but visually concerning condition caused by delayed desquamation of the filiform papillae, leading to an accumulation of keratin creating a characteristic “hairy” appearance, with pigmentation typically caused by bacteria, fungi, food, or medications. Common predispositions could be poor oral hygiene, smoking, certain medications, and xerostomia, but BHT induced by linezolid remains rare. The pathophysiology of BHT in this context likely involves the disruption of the oral microbiome, resulting in an overgrowth of keratinized papillae. By altering the balance of protective microbial flora, linezolid may impede the normal desquamation of keratin, allowing for the proliferation of bacteria or fungi that contribute to the discolouration.

Materials & Methods: Not Applicable

Results: Case Synopsis:

A 34-year-old female with ecthyma, a bacterial skin infection often caused by*Staphylococcus aureus* or *Streptococcus pyogenes*, was initially treated with standard antibiotics. Due to no significant effect was then started on oral linezolid 600 mg twice daily. She had no significant medical history, no tobacco or alcohol use, and her oral hygiene was suboptimal but otherwise unremarkable. After seven days of treatment, she presented with black discolouration and a rough texture on her tongue. On examination blackish pigmentation and elongation of the filiform papillae on the dorsal surface of the tongue, a hallmark feature of black hairy tongue (BHT), also known as lingua villosa nigra were seen. She is vitally normal and no other systemic symptoms. Given the temporal association between the start of linezolid and the onset of symptoms, the drug was identified as the most likely cause of BHT. The Naranjo score for this case was 7, indicating a probable adverse drug reaction. Linezolid was discontinued, and the patient was instructed to maintain good oral hygiene, including gentle tongue brushing and the use of a non-alcoholic mouthwash. Within two weeks, the discolouration and rough texture resolved completely, with no recurrence on follow-up.

Conclusion:

This case represents a rare instance of linezolid-induced BHT, an unusual yet benign side effect. The temporal relationship between drug administration and symptom onset, along with resolution on drug discontinuation, supports linezolid as the causative factor. This case reinforces the importance of clinician awareness regarding rare but benign side effects of medications like linezolid. In patients presenting with BHT, discontinuation of the causative drug, where possible, and a focus on improving oral hygiene are usually sufficient for resolution.

Abstract N°: 8284

From Atopic Dermatitis to Psoriasis: A Complication of Dupilumab Therapy

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Introduction & Objectives: Dupilumab, a monoclonal antibody targeting IL-4 and IL-13 pathways, is approved for the treatment of moderate-to-severe atopic dermatitis (AD). Historically, the coexistence of psoriasis and AD was considered rare due to their distinct immunopathogenic mechanisms. However, emerging reports suggest that psoriasis may either coexist with AD or arise as a paradoxical reaction to AD therapies, including dupilumab. We present a case illustrating the onset of psoriasis in a patient undergoing long-term dupilumab treatment for AD.

Materials & Methods: We report the case of a 21-year-old Caucasian male with a longstanding history of AD, who had been treated successfully with dupilumab for three years. The patient achieved complete remission of AD lesions within six months of therapy initiation. However, he developed new psoriasiform lesions during ongoing treatment.

Results: The patient presented with erythematous, scaly, well-demarcated plaques localized to the scalp and macular lesions on the upper body. His medical history included infancy-onset AD treated initially with topical corticosteroids, systemic steroids, and subsequently dupilumab. After 2.5 years of stable disease control, the patient developed new scalp lesions resembling psoriasis. A scalp biopsy confirmed the histopathological diagnosis of psoriasis vulgaris. This was interpreted as a paradoxical phenomenon secondary to dupilumab and therefore the administration was discontinued. The patient was transitioned to topical antipsoriatic therapy and a JAK inhibitor for the management of AD, resulting in complete clearance of the psoriatic lesions within one month.

Conclusion: Paradoxical development of psoriasis during dupilumab therapy represents a therapeutic challenge. Prompt recognition and management are essential to minimize morbidity and optimize long-term outcomes. Clinicians should be aware of this potential adverse event when treating patients with biologics and should individualize treatment adjustments as necessary.

Abstract N°: 8288

Hydroxyurea-Induced Pseudo-Dermatomyositis: A Mimic of Antisynthetase Syndrome

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Introduction & Objectives:

Hydroxyurea (HU) is a cytoreductive agent widely used in myeloproliferative neoplasms (MPNs). While cutaneous adverse effects such as hyperpigmentation and ulcers are well-documented, rare dermatological manifestations can mimic autoimmune skin diseases, posing diagnostic challenges. We present a case of HU-induced pseudo-dermatomyositis resembling antisynthetase syndrome, emphasizing the importance of distinguishing drug-induced reactions from true autoimmune disorders.

Materials & Methods:

NA

Results:

A 65-year-old woman with a 12-year history of essential thrombocythemia (ET), managed with long-term hydroxyurea, developed progressive erythematous, band-like lesions on the dorsal aspects of her fingers, predominantly over the proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints. Clinical evaluation revealed no muscular weakness, arthralgia, or respiratory symptoms. Laboratory investigations, including creatine kinase (CK), aldolase, and inflammatory markers (CRP, ESR), were within normal limits. Autoimmune serology, including antisynthetase antibodies (anti-Jo1, PL-7, PL-12), was negative. Histopathological examination of the skin demonstrated hyperkeratosis, acanthosis, and a mild nonspecific dermal lymphocytic infiltrate, with no evidence of interface dermatitis or muscle involvement. A reduction in HU dosage resulted in gradual resolution of the cutaneous lesions, supporting a drug-induced etiology.

Conclusion:

This case illustrates HU-induced pseudo-dermatomyositis, a rare cutaneous reaction that clinically mimics antisynthetase syndrome or idiopathic dermatomyositis. The patient’s presentation lacked systemic features such as myositis, interstitial lung disease, or arthritis, distinguishing it from classic antisynthetase syndrome. The absence of antisynthetase antibodies and normal muscle enzyme levels further reinforced a drug-related cause rather than a primary autoimmune process. Histopathological findings of nonspecific inflammation, without interface changes or myopathic involvement, corroborated the diagnosis of pseudo-dermatomyositis. The temporal correlation with HU therapy and clinical improvement upon dose reduction confirmed the causative role of the drug. HU-associated cutaneous reactions range from benign hyperpigmentation to severe ulcerations. Dermatomyositis-like eruptions, though rare, are clinically significant due to their potential misdiagnosis as autoimmune disease. The underlying mechanism remains unclear but may involve direct keratinocyte toxicity, immune dysregulation, or interferon pathway activation. Given the expanding use of HU in MPNs, clinicians must recognize this entity to avoid inappropriate immunosuppressive therapy. Management primarily involves dose adjustment or drug cessation, with close monitoring for lesion recurrence. This case highlights the necessity of considering iatrogenic causes in patients on long-term HU presenting with dermatomyositis-like features,

particularly in the absence of systemic or serological autoimmune evidence.

Abstract N°: 8333

Acneiform Drug Eruption: Retrospective Evaluation Of The Cases From a Tertiary Health-care Center

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Introduction & Objectives:

Acneiform drug eruptions (ADEs) are inflammatory papulopustular reactions triggered by medications such as corticosteroids, antiepileptics, biologics and chemotherapeutics agents. Their pathogenesis involves sebocyte dysfunction, immune dysregulation and individual drug metabolism variations.

This retrospective study analyzed 80 patients with ADEs, focusing on demographics, underlying diseases, causative drugs, and clinical course. The findings aim to enhance the understanding of the etiopathogenesis and guide clinical management.

Materials & Methods:

In this study, demographic, clinical, laboratory data of cases diagnosed with ADE between 2021-2024 in our hospital were evaluated.

Results:

In this study, data of 80 patients diagnosed with ADEs were analyzed. The study population included 62.5% male and 37.5% female, with a mean age of 40.93±16.73 years.

A statistically significant relationship was found between gender and eruption severity (p=0.001). The mean duration from symptom onset to diagnosis was 6.89 ± 13.19 weeks, with EGFR inhibitor-induced eruptions lasting longer than those caused by other drugs.

The most frequently implicated drugs were EGFR inhibitors (26.25%), systemic corticosteroids (21.25%) and conventional chemotherapeutics (10%). However, no significant association was found between the causative drug and eruption severity (p> 0.05). No significant correlation was found between the severity of eruption and the underlying diseases requiring the use of these drugs (p> 0.05).

The trunk and face were the most frequently affected areas. According to the CTCAE, 63.75% of patients had Grade 1, 28.75% had Grade 2, and 7.5% had Grade 3 eruptions, with malignancy detected in 83.3% of Grade 3 patients.

No statistically significant correlation was observed between laboratory parameters (LDH, albumin, creatinine, WBC) and eruption severity. A weak positive correlation was found between albumin and severity (r=0.041), while LDH showed minimal correlation (r=0.000), but neither was statistically significant.

Regarding treatment, 62.5% of patients received topical therapy alone, 32.5% received combined systemic and topical therapy, and 5% were treated with systemic therapy alone. Treatment modality was not significantly associated with eruption severity (p=0.647).

Conclusion:

This retrospective study evaluated the etiological factors, clinical course, and treatment responses of ADEs. The

findings indicate that gender significantly influences eruption severity, with male patients experiencing more severe reactions, possibly due to hormonal and sebaceous gland differences.

Although no significant difference was found in eruption duration among drug groups, lesions caused by EGFR inhibitors lasted longer, supporting their role in prolonged inflammation. Neither the causative drug nor underlying diseases were significantly associated with severity. Similarly, metastatic disease did not influence severity.

There was no significant correlation between laboratory parameters (LDH, albumin, creatinine, WBC) and eruption severity. Treatment modality was not statistically linked to severity, though systemic therapy was more common in severe cases, reflecting clinical variability in treatment approaches.

These findings emphasize gender as a key determinant of acneiform eruption severity, while drug type, underlying diseases, and laboratory parameters do not play a significant role.

Abstract N°: 8435

A Rare Case of SDRIFE (Symmetrical Drug-Related Intertriginous and Flexural Exanthema) Induced by Systemic Acyclovir Use

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Introduction & Objectives: Symmetrical drug-related intertriginous and flexural exanthema (SDRIFE), is a rare form of drug eruption which is caused by delayed Type-IV immune reaction. SDRIFE has a short latency of 2-5 days due to drug directly binding to T-cell receptors. SDRIFE is clinically defined by five criteria: (i) onset after initial or repeated exposure to a systemically administered drug (contact allergens excluded) , (ii) sharply demarcated erythema in the gluteal/perianal area and/or V-shaped erythema in the inguinal/perigenital region, (iii) involvement of at least one other interginious/flexural fold (e.g. axillae, antecubital fossae), (ıv) symmetrical distrubiton, and (v) absence of systemic involvement. Antibiotics, in particular beta-lactams , comprise the majority of causes of SDRIFE. The clinical course of SDRIFE is usually benign and self-limited.

Materials & Methods: We present a patient presented with symmetrical drug-related intertriginous and flexural exanthema due to intravenous acyclovir use.

Results: A 51 year-old female patient was admitted to the emergency department with complaints of neurologic deterioration and was hospitalized with a diagnosis of viral encephalitis. Intravenous acyclovir treatment was started from the day of admission and the patient was evaluated by the dermatology department on the 5th day of treatment because of generalized rashes on the body. Patient had no systemic symptoms. On examination, erythematous plaques were seen in bilateral axillary area, bilateral inguinal fold area, bilateral gluteal and lumbosacral areas, all symetrically.However, there was no involvement of the oral mucosa. Blood results showed mild eosinophilia and mildly elevated white blood cells. The patient refused a biopsy for histopathologic diagnosis so the patient was clinically diagnosed with SDRIFE. Despite this patient continued to receive acyclovir 14 days as viral encephalitis because of treatment could not be interrupted. Systemic antihistaminic and topical corticosteroid were given as treatment. The lesions completely resolved with symptomatic treatment.

Conclusion: In the literature ,only a few cases of SDRIFE due to antiviral agents have been reported. Two of these were due to valacyclovir,one was due to acyclovir and one was due to remdesivir. Clinical signs regressed in all patients with drug discontinuation. SDRIFE is considered a benign rash due to its self limited course and complications are exceptional.

Abstract N°: 8451

When common antibiotics cause uncommon reactions: Bullous SDRIFE induced by doxycycline

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Introduction & Objectives:

Symmetrical drug-related intertriginous and flexural exanthema (SDRIFE) is a rare type IV hypersensitivity reaction, considered a variant of systemic allergic contact dermatitis. It typically presents as symmetrical erythema with flexural prominence, most commonly triggered by antibiotics such as beta-lactams, and less commonly by other drugs. However, we report an exceptionally rare blistering variant of SDRIFE following the administration of doxycycline.

Materials & Methods:

Case report

Results:

A 61-year-old male presented with sharply demarcated, symmetrical erythema localized to the gluteal, inguinal, perianal, and axillary regions, with extension to the flexural aspect of the upper arm, lateral thorax, and submammary areas. The presence of bullae and desquamation further characterized the affected regions. Symmetrical erythema also affected the dorsal hands and feet, with bullae on the plantar surfaces. Symptoms developed abruptly following a single oral dose of doxycycline, prescribed for persistent cough after initial influenza-like illness treated with azithromycin and paracetamol 12 days prior to the appearance of the rash. A thorough patient history revealed a prior hospitalization 20 years earlier for a similar reaction to Hiramicin® (doxycycline), presenting with generalized erythema, vesiculation, and desquamation. Laboratory tests, including complete blood count and liver and renal function, were within normal limits, and systemic involvement was excluded. A skin biopsy of the rash was performed, revealing a histopathological pattern of pronounced vacuolar interface dermatitis, which aligns with the clinical diagnosis of a drug eruption. Based on the clinical presentation and lack of systemic and mucosal involvement, the differential diagnosis included bullous SDRIFE, fixed drug eruption, and erythema multiforme exudativum (EEM). Autoimmune bullous diseases were ruled out based on negative direct immunofluorescence and the absence of anti-desmoglein 1/3 and anti-envoplakin antibodies. Treatment consisted of systemic corticosteroids, initiated at a high dose that was gradually tapered and discontinued over 30 days. Topical management included high-potency corticosteroids, antibiotic creams, and Vaseline gauze applied to the groin. Doxycycline was immediately discontinued. The patient responded well to the treatment, with complete resolution of symptoms and no recurrence of the rash or systemic involvement during follow-up. Patch testing was deferred due to clinical clarity and patient preference.

Conclusion:

This case represents an extremely rare bullous variant of SDRIFE and highlights the importance of distinguishing blistering cutaneous adverse reactions with flexural distribution. Although SDRIFE is typically self-limiting and managed by discontinuation of the causative agent, future avoidance, and supportive therapy such as corticosteroids, accurate diagnosis is essential to distinguish it from other severe cutaneous adverse reactions (SCARs). Key distinguishing factors include latency period, histopathologic features, and absence of mucosal and

systemic involvement. Unlike typical SDRIFE, the bullous form may result in blistering and rupture, increasing the risk of secondary complications and morbidity.

Abstract N°: 8512

dress syndrome complicated by kaposi-juliusberg syndrome: case report

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Introduction & Objectives:

Kaposi-Juliusberg syndrome and DRESS syndrome are two very serious dermatological emergencies. Their rare, even exceptional, association is morbid and requires urgent, adequate, and especially proactive management. We present an original case of the association between DRESS syndrome and Kaposi-Juliusberg syndrome.

Materials & Methods:

A 38-year-old patient, followed in nephrology for ANCA vascularitis at an early stage of renal failure, was hospitalized in our department for management of a DRESS syndrome due to use of allopurinol, treated by systemic corticosteroid with a favorable evolution. Three weeks later, the patient presented with a generalized, febrile erythrodermia (40°C), with the appearance of smallpox-like erosive vesicles on the face, neck, trunk, and scalp. The diagnosis of Kaposi-Juliusberg syndrome was clinically suggested and confirmed by Tzanck cytodiagnosis and viral PCR. An intraveinous acyclovir treatment was administred urgently. The progression was characterized by the reduction in Kaposi-Juliusberg syndrome DRESS syndrome persisted (subintrant seizures), warranting the use of Valaciclovir as a prophylactic treatment. This approach led to a total remission over a follow-up period of 6 months.

Results:

Although the association between Kaposi-Juliusberg syndrome and DRESS syndrome is well established due to their shared viral origin, it remains an exceptionally rare occurrence and, to our knowledge, has never been previously reported, emphasizing the uniqueness of our case. The prognosis can be severe, as the complications of both syndromes do not simply add up but rather amplify each other, leading to an intensified immune response and a significantly higher risk to the patient’s life. This risk becomes even greater when the syndromes occur in a fragile or unstable condition, as seen in our patient. Fatal outcomes often result from organ failure, affecting the liver, heart, kidneys, or nervous system. Management requires intensive care, including targeted treatment for organ failure, discontinuation of the triggering drug, intravenous Acyclovir, general corticosteroid therapy, and polyvalent immunoglobulins. Given the potential for a prolonged disease course with recurrent flare-ups, extended corticosteroid therapy with gradual tapering, alongside prophylactic antiviral treatment, is essential.

Conclusion:

This association, although rare, should be considered when encountering any smallpox-like eruption that may aggravate or perpetuate the progression of DRESS syndrome.

Abstract N°: 8529

Amivantamab-associated erosive pustular dermatosis of the scalp in a patient with non-small cell lung cancer

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Introduction & Objectives:

Materials & Methods:

Results:

A 79-year-old patient developed erosive crusty lesions of the hairless scalp in the parietal region within 3 months of starting immunotherapy with amivantamab (EGFR-MET-bispecific antibody) due to non-small cell lung cancer with bone metastases. Previous therapies with disinfectants, topical antibiotics and steroids had not brought any improvement.

On physical examination, the scalp showed confluent partly hyperkeratotic, partly erosive plaques of 1-2 cm in diameter with purulent secretion in places. In addition, perifollicular monomorphic erythematous papulopustules were noted on the chest and back, which had preceded the development of the scalp lesions.

The clinical chemistry and blood count did not reveal any pathologic findings. A smear showed colonization with Klebsiella pneumoniae and a biopsy of the scalp showed erosions of the epidermis with alternating cell- and vessel-rich granulation tissue and a mixed-cell, inflammatory infiltrate in the corium.

Based on the patient’s medical history, clinic and histopathology, we diagnosed amivantamab-associated erosive pustular dermatosis of the scalp (EPDK) and acneiform exanthema on the trunk. Disinfection with octenidine, topical steroids and immunomodulatory systemic therapy with dapsone 100mg/d led to a rapid improvement in the findings. However, with low to normal glucose-6-phosphate dehydrogenase activity, methemoglobin increased from 2.8% to 7%, necessitating a dose reduction of dapsone to 50mg/d. Here, too, the findings stabilized and cancer therapy with the bispecific antibody could be continued.

As a bispecific antibody, amivantamab inhibits the EGFR-dependent cell proliferation of tumour cells and keratinocytes, resulting in apoptosis and the release of pro-inflammatory cytokines. Inhibition of the mesenchymal-epithelial transition factor (MET) further increases the inhibition of cell proliferation. The biologic, which has been approved 3 years ago for non-small cell lung cancer, exhibits considerable cutaneous toxicity. Acneiform exanthema and paronychia occur most frequently, hypertrichosis or EPDK less frequently.

Topical corticosteroids, oral retinoids, antibiotics and dapsone are therapeutic options for this chronic dermatosis, which is usually observed in elderly patients with severe actinic damage.

In summary, this case shows that early interdisciplinary cooperation is essential in the case of side effects of oncological therapies and that dapsone with its anti-inflammatory and neutrophil-inhibiting effect can enable oncological therapy to be continued.

Conclusion:

Abstract N°: 8565

Levamisole-Associated Cutaneous Reaction with Atypical Presentation

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[3]Ribeirão Preto Medical School - University of São Paulo, Emergency Medicine, Internal Medicine, Ribeirão Preto, Brazil

Introduction & Objectives:

Levamisole, a veterinary anti-helminthic drug with immunomodulatory effects, has emerged as a common adulterant in illicit cocaine. Its use has been linked to serious complications including agranulocytosis and various forms of vasculitis. Cutaneous findings classically include retiform purpura and necrosis, particularly on the ears and extremities. However, recent evidence suggests a broader spectrum of skin manifestations. This study aims to report an atypical case of presumed levamisole-related toxicity with unusual dermatological presentation and confirmed exposure by Gas Chromatography–Mass Spectrometry (GC-MS) analysis.

Materials & Methods:

A 57-year-old male presented with skin lesions following suspected involuntary exposure to adulterated cocaine inadvertently added to his beer. A few days after the incident, the patient developed paresthesia and edema in his hands and feet, followed by asymptomatic purpuric lesions. These evolved into violaceous plaques with fissures and crusts. About a week later, similar lesions appeared in the genital area, with edema, pustules, and mild necrosis of the penis, scrotum, and buttocks. As soon as the skin lesions began, the patient took nimesulide for one day (two tablets). Biopsy showed superficial perivascular dermatitis with eosinophils and red blood cell extravasation with no vasculitis. Direct immunofluorescence showed weak positivity for fibrinogen in capillary walls. For levamisole detection, a 5 mL urine aliquot was processed with NaOH and hexane/isoamyl alcohol extraction, centrifuged, and the organic layer was evaporated under nitrogen. The residue was reconstituted in methanol and analyzed by GC-MS using an Agilent 7890A system coupled to a 7975C mass spectrometer. Levamisole was identified by its characteristic mass ions (148, 204, 205) and retention time, which confirms the exposure with a high degree of certainty.

Results:

The clinical distribution of lesions, including intertriginous areas, suggested a broader drug reaction possibly resembling symmetric drug-related intertriginous and flexural exanthema (SDRIFE). Histopathology was not consistent with classic leukocytoclastic vasculitis. GC-MS confirmed the presence of levamisole in urine, supporting the hypothesis of levamisole-induced toxicity. Nonetheless, the concurrent use of nimesulide raised the possibility of a drug-induced hypersensitivity reaction. The combination of clinical, histological, and toxicological data supports a cutaneous drug reaction possibly involving both agents.

Conclusion:

This case expands the clinical spectrum of cutaneous reactions associated with levamisole exposure. While vasculitic patterns remain typical, alternative presentations such as SDRIFE-like eruptions should be considered.

Confirmatory toxicology through GC-MS is essential to establish exposure. Clinicians should maintain a high index of suspicion in patients with unexplained purpuric or intertriginous lesions and a history of cocaine use.

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Abstract N°: 8569

Drug-Induced Cutaneous Vasculitis: A Report of Three Cases

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Introduction & Objectives:

Drug-induced cutaneous vasculitis is a hypersensitivity reaction affecting small dermal vessels. Many drugs have been implicated. We report three cases triggered by different pharmacological agents..

Materials & Methods:

Case1 An 84-year-old woman with diabetes, hypothyroidism, and hypertension developed palpable purpura and necrotic ulceration on the lower limbs 10 days after starting lercanidipine. She had no fever or infectious signs. Labs showed eosinophilia (1300/mm^3), elevated ESR, negative CRP, and acute renal failure. Skin biopsy from an older lesion showed dermal lymphohistiocytic infiltrates. Discontinuation of lercanidipine led to near-complete lesion regression and rapid renal function improvement within two weeks.

Case2 A 44-year-old man, hospitalized for an ankle fracture, developed palpable purpura on his upper and lower limbs seven days after starting cefotaxime and ciprofloxacin. He had no systemic symptoms. Lab tests revealed neutrophilic leukocytosis and elevated CRP; ANCA, ANA, and serologies were negative. Skin biopsy showed dermal fibrosis with a lymphohistiocytic infiltrate. Stopping the antibiotics led to rapid clinical resolution.

Case3 A 23-year-old woman with Crohn’s disease developed palpable purpura on her limbs, with arthralgia, one week after her first adalimumab injection. No renal or visceral involvement was found. Lesions regressed within a week after stopping the medication.

Results:

Drug-induced cutaneous vasculitis is typically a leukocytoclastic small-vessel vasculitis caused by delayed hypersensitivity to medications. Onset varies from days to weeks after exposure. Clinically, it presents mainly as palpable purpura, sometimes with systemic symptoms, but can mimic primary vasculitis or infections, complicating diagnosis.

In our cases, the temporal relationship with drug exposure, absence of major systemic involvement, and rapid resolution after drug withdrawal supported a drug-induced etiology. The implicated agents—lercanidipine, cephalosporins, fluoroquinolones, adalimumab—illustrate the wide pharmacologic range of potential triggers.

Histopathology can assist diagnosis but is ideally performed on fresh lesions (<48h), as older lesions may lose characteristic vascular changes, limiting diagnostic value.

Conclusion:

In any recent-onset cutaneous vasculitis, drug-induced causes should be systematically considered and assessed through clinical evolution after withdrawal of the suspected agent.

Abstract N°: 8575

Atypical and Giant Striae Induced by Potent Topical Corticosteroids: A Case Report

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Introduction

Striae distensae (stretch marks) are a well-known side effect of topical corticosteroids, but their occurrence in an extensive and severe form is rare. We report a case of atypical and giant striae following excessive use of clobetasol propionate.

Case presentation:

A 27-year-old woman with no significant medical history presented with skin lesions that had appeared three months earlier. These lesions were characteristic of atypical and giant striae, consisting of linear, atrophic, and depressed areas measuring several centimeters. The striae were pink to hypopigmented with a slightly pearly sheen. One month prior to the onset of these lesions, the patient had applied topical clobetasol propionate to treat axillary hyperpigmentation, using large quantities (30 g every 2–3 days). No other treatments were used during this period.

Discussion:

Striae are a well-recognized complication of prolonged topical corticosteroid use, but such severe and extensive presentations are exceptional. In this case, the lesions were notable for their unusual size, deep depressed appearance, pearly coloration, and widespread linear distribution, reflecting severe cutaneous atrophy. The severity can be explained by the high potency of clobetasol propionate, its application to a fragile area such as the axillae, and the excessive amount used over a short period. This case highlights an atypical form of steroid-induced striae, underscoring the need for careful differential diagnosis and greater patient education regarding self-medication.

Conclusion:

This case illustrates a rare and severe presentation of striae induced by clobetasol overuse, emphasizing the importance of strict medical supervision to prevent such complications.

Abstract N°: 8594

A Case of Bullous Fixed Pigmented Erythema with Mucosal Involvement in a Child

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Introduction

Skin reactions in children, in the context of drug administration, are a very common reason for consultation. The hypothesis of a drug eruption is often raised. Here, we present a drug eruption, specifically a bullous fixed pigmented erythema with mucosal involvement, in a child.

Case presentation:

A 10-year-old child, with no significant medical history, presented with a skin rash evolving for 5 days, following an infectious episode the day before, and had taken amoxicillin-clavulanate and ibuprofen. Upon clinical examination, the child was conscious, afebrile, hemodynamically and respiratorily stable, with erythematous rounded macules with central blistering on the limbs, trunk, and back. The examination also revealed fissured and hemorrhagic cheilitis, along with non-purulent bilateral conjunctivitis with palpebral erosions. Histological analysis showed a normoacanthotic epidermis with liquefactive necrosis of the stratum corneum, associated with keratinocytic necrosis without inflammatory exocytosis. The basal layer was vacuolated without blistering detachment, and the dermis displayed marked edema with inflammatory cells, including lymphocytes and histiocytes. After a pharmacological investigation, ibuprofen was identified as the most likely culprit. The child showed significant improvement after discontinuation of all implicated treatments.

Discussion:

Fixed pigmented erythema is a benign drug eruption, and its clinical characteristics in children are similar to those in adults. It initially presents with one or several well-defined, rounded or oval macules that are erythematous or purplish, occasionally evolving into bullae. The lesions can be singular or multiple, affecting the skin or, more rarely, the mucous membranes. The lesions undergo a cyclical evolution. The prognosis is favorable within a few days, with residual pigmentation. The main drugs implicated are analgesics (pyrazoles, paracetamol, aspirin), antibiotics (sulfamides, tetracyclines), antiepileptics (phenytoin, barbiturates, carbamazepine), and NSAIDs. The distinctive feature in our patient was the blistering aspect of the lesions and mucosal involvement in a child.

Conclusion :

The primary challenge in pediatric drug eruptions lies fundamentally in distinguishing them from infectious diseases, particularly viral infections, which are much more common at this age.**

Abstract N°: 8595

Anti-TNF-Induced Lupus in a Patient with Hidradenitis Suppurativa: A Case Report

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Introduction & Objectives:

Hidradenitis suppurativa (HS) is a chronic, relapsing inflammatory skin disorder characterised by painful nodules, abscesses with sinus tract formation, primarily affecting intertriginous areas. It is frequently recalcitrant to conventional therapies and significantly impacts quality of life. Psychiatric comorbidities are common, with depression affecting approximately 20% of HS patients. The advent of anti-tumour necrosis factor (anti-TNF) agents has provided valuable therapeutic options for moderate-to-severe HS. However, drug-induced lupus erythematosus (DILE), albeit rare, is a recognised complication of anti-TNF therapy, with an estimated prevalence of 0.5%–1% in this patient cohort. Reports of anti-TNF-induced lupus (ATIL) in HS are limited to a small number of case studies. This case report describes a 33-year-old female with HS who developed ATIL following adalimumab therapy.

Materials & Methods:

We report a case of a 33-year-old female with Hurley stage II HS and associated factors, including elevated BMI, smoking history, first-degree family history of HS, polycystic ovary syndrome, anxiety, and depression. She had failed standard therapies including doxycycline, metformin, topical agents, and lifestyle modification. A modest response to clindamycin and rifampicin prompted escalation to adalimumab. The patient experienced two temporary interruptions in treatment due to intercurrent infections

Results:

After six months, she developed systemic symptoms including fatigue, arthralgia and hand joint swelling. Immunological testing revealed rising ANA and anti-dsDNA titres, consistent with ATIL. Notably, her HS showed clinical improvement on adalimumab, with corresponding improvement in quality of life and validated mental health scores. Adalimumab was promptly discontinued. HS management was resumed with clindamycin and rifampicin, followed by a switch to secukinumab. Lupus management involved rheumatology specialists in a multi-disciplinary care approach.

Conclusion:

Drug induced lupus should be considered in HS patients who develop new systemic symptoms following anti-TNF therapy. The distinction between true drug-induced lupus and unmasked systemic lupus erythematous remains challenging. This case highlights the importance of baseline immunological screening, close clinical monitoring, and robust pharmacovigilance in biologic-treated HS patients to mitigate risks and optimise outcomes.

Abstract N°: 8597

A Diagnostic Challenge: Methotrexate-Induced Toxicity Mimicking Stevens–Johnson Syndrome in a Patient with Psoriasis

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Introduction & Objectives: Stevens–Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are severe mucocutaneous reactions often triggered by medications. However, methotrexate (MTX)-induced toxicity may mimic these entities, especially in elderly patients with long-term MTX use and without folic acid supplementation. This case aims to highlight the importance of considering MTX toxicity in the differential diagnosis of SJS/TEN-like presentations, particularly in patients with psoriasis, and to discuss the systemic complications associated with this toxicity.

Materials & Methods: We present a 71-year-old male patient with a 20-year history of psoriasis, referred with a preliminary diagnosis of pemphigus vulgaris and later considered SJS/TEN due to rapidly spreading erythematous, crusted, erosive skin lesions and mucosal involvement. The patient had been using low-dose MTX (7.5 mg/week) intermittently over the past two years, with consecutive daily dosing in the preceding three weeks, and without folic acid supplementation. A detailed clinical, laboratory, histopathological, and radiological evaluation was performed, including skin and esophageal biopsies, hematological monitoring, and infectious work-up.

Results: Despite initial SJS/TEN-oriented management with IVIG and systemic corticosteroids, skin biopsy results were non-specific and not supportive of SJS/TEN. The patient developed profound pancytopenia (WBC: 0.43×10^9/L, PLT: 9×10^9/L, Hb: 8.6 g/dL), hepatocellular injury, gastrointestinal bleeding (melena), and mucosal ulceration. CMV esophagitis was suspected but not confirmed histologically. MTX blood level was undetectable, likely due to delayed measurement. Folate deficiency (1.1 ng/mL) was noted. Opportunistic infections including Klebsiella pneumoniae and ESBL-positive Enterobacter were isolated. Clinical and hematological improvement occurred after G-CSF support, folate supplementation, and broad-spectrum antibiotics.

Conclusion: This case illustrates that low-dose MTX toxicity, particularly in the setting of folate deficiency and advanced age, can closely mimic SJS/TEN, but requires a fundamentally different therapeutic approach. Differentiation is essential as prompt recognition and cessation of MTX, along with supportive measures (folic acid, G-CSF, antimicrobial therapy), can reverse life-threatening complications. Histopathological evaluation and systemic findings should guide the diagnosis rather than cutaneous appearance alone.

Abstract N°: 8634

ALEP Beyond Drugs: Clinical and Histological Insights from a 7-Case Series

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Introduction & Objectives:

Acute localized exanthematous pustulosis (ALEP) is a rare dermatologic entity related to acute generalized exanthematous pustulosis (AGEP). Although most cases are attributed to medications, non-drug-related exogenous factors have also been implicated in certain cases.

Materials & Methods:

We conducted a retrospective study including all cases of ALEP diagnosed in our department between 2021 and 2025. The EuroSCAR diagnostic criteria were applied to confirm the cases.

Results:

We collected 7 cases of ALEP: 60% were male and 40% female, with a mean age of 40 years (range: 14–76). Lesions were primarily located on the face (45%) and upper limbs (35%). A skin biopsy was performed in 50% of cases, confirming the histological diagnosis of ALEP with subcorneal pustules composed of neutrophils and eosinophils, epidermal spongiosis, scattered keratinocyte necrosis, vacuolization of the basal layer, and a perivascular lymphohistiocytic dermal infiltrate. Identified etiologies included exposure to plants (3 cases), cosmetic products (2 cases), and tattoos (1 case). All patients were treated with topical corticosteroids, resulting in favorable outcomes in 100% of cases.

Conclusion:

Our study confirms that ALEP can be induced by non-drug-related agents. The predominance of facial lesions and the diversity of causes suggest sensitization to airborne or contact substances. These findings support the notion that contact-induced ALEP is underdiagnosed and highlight the need for thorough etiological investigation.

Abstract N°: 8652

Improving Eye Assessment in Acute SJS/TEN and RIME/MIRM : A Practical Toolf for Dermatologists

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[2]Hospices Civils de Lyon - HCL, Lyon, France

Introduction & Objectives:

There is currently no standardised approach for assessing acute eye involvement in Stevens-Johnson Syndrome (SJS), Toxic Epidermal Necrolysis (TEN), or Reactive Infectious Mucocutaneous Eruption (RIME), also referred to as Mycoplasma-Induced Rash and Mucositis (MIRM), by non-eye specialists such as dermatologists, acute physicians, or general practitioners. Yet, these clinicians are often the first to assess the patient. Existing ophthalmic grading systems (e.g. Power et al., 1995; Sotozono et al., 2015) rely on slit-lamp examination and fluorescein staining, making them unsuitable for use outside specialist settings. Early recognition of eye involvement and timely referral to ophthalmology significantly influence patient outcomes, but delays in referral are common, leading to preventable complications including dryness, scarring, and visual loss. The objective of this work was to identify or adapt a clinically relevant, simplified staging system for use by non-eye specialists at the point of initial presentation, with endorsement from ophthalmology colleagues.

Materials & Methods:

A literature review covering the period from January 1990 to January 2025 was conducted to examine current guidance and clinical practice regarding the assessment of eye involvement in acute SJS/TEN by non-ophthalmologists. The review focused on identifying practical clinical signs that could support meaningful communication with ophthalmology and prompt appropriate early intervention. The findings were used to inform the development of a simplified staging tool.

Results:

A practical, simplified staging system for acute eye involvement—ranging from grade 0 to grade 3—was developed by dermatologists in collaboration with ophthalmology specialists. It includes clear, accessible terminology and a side-by-side comparison of initial assessment findings suitable for non-eye specialists (in green) with corresponding ophthalmological findings expected at each grade (in blue). The tool is designed for ease of use in acute settings where specialist equipment is not available. The proposed tool is presented in Table 1.

Conclusion:

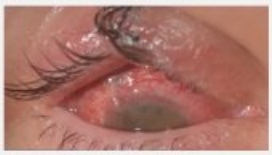
This simplified staging system aims to support earlier recognition of eye involvement, improve communication between non-eye specialists and ophthalmologists, and promote timely treatment of acute ocular disease in SJS/TEN and related conditions. By serving as a standardised assessment tool, it has potential utility in both clinical practice and research settings to reduce variation in care and improve patient outcomes.

Table 1

Acute SJS/TEN - Eye assessment & management

Anna B Chapman (ABC) Sotozone grading adaptation for Dermatologists, May 2025

Sotozono, C. et al. Predictive factors associated with acute ocular involvement in Stevens–Johnson syndrome and toxic epidermal necrolysis. Am. J. Ophthalmol. 160, 228–237 (2015).

Patient details						
Date of admission	**Dermatology assessment**			**Ophthalmology assessment**		
	Both eyes Conjunctival swabs	Right eye Y/N	Left eye Y/N		Right eye Y/N	Left eye Y/N
Grade 0 No eye involvement						
Grade 1 Conjunctival hyperaemia	**Date**			**Date**		
 **Hyperaemia - red eye** Conjunctival hyperaemia is noted in both eyes. Even when hyperaemia looks mild, epithelial defect may be present concomitantly. Patient should be referred to ophthalmologist for further assessment and management of potential presence of punctate keratitis, pseudomembranes, corneal erosions, conjunctival adhesions	**Impaired vision : patient report**			Impaired vision		
	Red Eye - hyperaemia			Eyelid oedema		
	Eyelid oedema			Mild conjunctival injection		
	Purulent discharge			Chemosis		
	Eyelid closure					
Grade 2 Conjunctival hyperaemia Either ocular surface epithelial defect * Pseudomembrane						
 **Pseudomembrane** A pseudomembrane is composed of fibrin, necrotised epithelial cells and infiltrating cells (mainly neutrophils), indicates severe inflammation on the ocular surface. If treatment works, pseudomembrane will spontaneously become milder. Increased pseudomembrane formation noted during treatment suggests aggravation of inflammation on the surface of the eye.	**Impaired vision : patient report**			Impaired vision		
	Red eye- hyperaemia			Punctate keratitis		
	Eyelid oedema			Either epithelial defect		
	Purulent discharge			Corneal erosions		
	Eyelid closure			Corneal infiltrate		
	Pseudomembrane					
Grade 3 Conjunctival hyperaemia Both ocular surface epithelial defect & Pseudomembrane + Symblepharon						
 **Symblepharon** Severe inflammation affects conjunctiva over sclera as well as inner eyelids and can be clinically appreciated by redness and swelling of the eyelids. It may be accompanied by acute loss of eyelashes in severe cases. Symblepharon (adhesion of the eyelid conjunctiva and the eyeball/ scleral conjunctiva) conjunctiva) rapidly progresses to irreversible changes.	**Impaired vision : patient report**			Impaired vision		
	Red eye- hyperaemia			Both epithelial defects		
	Eyelid oedema			Conjunctival fornix foreshortening		
	Purulent discharge			Symblepharon formation		
	Eyelid closure					

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Abstract N°: 8677

A Simplified Skin Assessment Tool for SJS/TEN: Toward a Practical Electronic Application

Anna Chapman*[1], BEN SAID Benoit[2]

[1]Homerton University Hospital , London, United Kingdom
[2]Hospices Civils de Lyon - HCL, Lyon, France

Introduction & Objectives:

A skin involvement tool has been developed by dermatologists to support efficient and user-friendly assessment of acute **Stevens-Johnson Syndrome (SJS)** and **Toxic Epidermal Necrolysis (TEN)**. It enables clinicians to estimate:

1. **%TBS (Erythema)** – percentage of total body surface involved with erythema

2. **%BSA (Epidermal Detachment)** – percentage of body surface area affected by epidermal detachment (sum of blistered and eroded skin)

This tool adapts the **Wallace Rule of Nines**, widely used in burn assessment, into a simplified format appropriate for SJS/TEN. The first objective was to provide a clear diagrammatic interface for marking erythema and detachment separately, allowing easy calculation of %TBS, %BSA, and SCORTEN. The second objective was its use in routine clinical and research settings to monitor disease progression over time. Ultimately, the tool is intended as the basis for developing an electronic application—similar to the **E-Burn app** used in burn care—to further streamline and standardise skin assessment in SJS/TEN.

Materials & Methods:

A literature review (January 1990–January 2025) was conducted to evaluate current clinical practices for skin assessment in SJS/TEN. The focus was on identifying practical, accessible tools suitable for bedside use. The E-Burn app, which incorporates the Wallace Rule of Nines for burns, served as a conceptual model for the SJS/TEN-specific adaptation. A draft version of the tool is illustrated in **Figure 1**.

Results:

The simplified skin involvement tool is now available for pilot testing. It enables dermatologists and other clinicians to visually and numerically record the extent of skin involvement and monitor disease progression. It is also designed to form the foundation for the development of a digital version, allowing wider accessibility, data tracking, and integration with SCORTEN and clinical documentation systems.

Conclusion:

SJS/TEN are rare but severe drug-induced conditions requiring prolonged hospitalisation and intensive multidisciplinary care. The estimated annual incidence is approximately 1–6 cases per million for SJS and 0.4–1.2 per million for TEN. Mortality rates range from 10% to 40%, with many survivors experiencing long-term disability and reduced life expectancy.

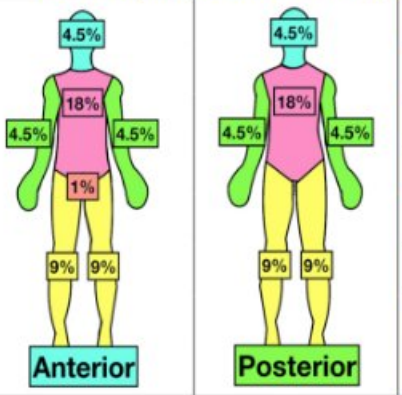
In the absence of reliable biomarkers of disease activity, a clear and standardised method of assessing and tracking skin involvement during hospitalisation can support timely and appropriate management decisions. Progressive erythema and epidermal detachment may indicate worsening disease and prompt escalation of care,

while stabilisation can reinforce current management strategies. This tool offers a practical, visual method to assist clinicians in daily decision-making and future research applications

Acute SJS/TEN - Skin involvement assessment

Modified Wallace Rule of Nines for Dermatology SJS/TEN assessment purpose

TBSA erythema and %BSA by epidermal detachment calculation



Body Part	Body Surface Area	Erythema TBSA	%Blisters + Erosions
Entire Head & Neck	9%		
Entire Right Arm	9%		
Entire Left Arm	9%		
Entire Trunk	36%		
Groin	1%		
Entire Right Leg	18%		
Entire Left Leg	18%		

Patient details	Admission date Admission place	Any other relevant information	Sum TBSA % Erythema	Sum BSA % Epidermal Detachment
Assessor name: Date:				

SCORTEN score calculation

Parameter	Threshold	Points
Age	≥ 40 years	1
Heart rate	≥ 120 beats per minute	1
Presence of malignancy	Yes	1
Percentage of epidermal detachment (blisters + erosions)	> 10% of body surface area (BSA)	1
Serum urea level (UK) / BUN (USA)	> 10 mmol/L (UK) /28 mg/dL (USA)	1
Serum glucose level	> 14 mmol/L UK) /252 mg/dL(USA)	1
Serum bicarbonate level	< 20 mmol/L	1
SCORTEN Assessment date	SCORTEN score:	

Mortality risk by SCORTEN score

Total Score	Predicted Mortality Rate
0-1	3.2%
2	12.1%
3	35.3%
4	58.3%
≥5	>90%

Table 1

Abstract N°: 8756

Recognising Eye Involvement in Acute SJS/TEN and RIME/MIRM: A practical approch for Dermatologists

Anna Chapman*[1], BEN SAID Benoit[2]

[1]Homerton University Hospital , Dermatology, London, United Kingdom
[2]Hospices Civils de Lyon - HCL, Lyon, France

Introduction & Objectives:

There is currently no standardised approach for assessing acute eye involvement in Stevens-Johnson Syndrome (SJS), Toxic Epidermal Necrolysis (TEN), or Reactive Infectious Mucocutaneous Eruption (RIME), also referred to as Mycoplasma-Induced Rash and Mucositis (MIRM), by non-eye specialists such as dermatologists, acute physicians, or general practitioners. Yet, these clinicians are often the first to assess the patient. Existing ophthalmic grading systems (e.g. Power et al., 1995; Sotozono et al., 2015) rely on slit-lamp examination and fluorescein staining, making them unsuitable for use outside specialist settings. Early recognition of eye involvement and timely referral to ophthalmology significantly influence patient outcomes, but delays in referral are common, leading to preventable complications including dryness, scarring, and visual loss. The objective of this work was to identify or adapt a clinically relevant, simplified staging system for use by non-eye specialists at the point of initial presentation, with endorsement from ophthalmology colleagues.

Materials & Methods:

A literature review covering the period from January 1990 to January 2025 was conducted to examine current guidance and clinical practice regarding the assessment of eye involvement in acute SJS/TEN by non-ophthalmologists. The review focused on identifying practical clinical signs that could support meaningful communication with ophthalmology and prompt appropriate early intervention. The findings were used to inform the development of a simplified staging tool.

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A practical, simplified staging system for acute eye involvement—ranging from grade 0 to grade 3—was developed by dermatologists in collaboration with ophthalmology specialists. It includes clear, accessible terminology and a side-by-side comparison of initial assessment findings suitable for non-eye specialists (in green) with corresponding ophthalmological findings expected at each grade (in blue). The tool is designed for ease of use in acute settings where specialist equipment is not available. The proposed tool is presented in Table 1.

Conclusion:

This simplified staging system aims to support earlier recognition of eye involvement, improve communication between non-eye specialists and ophthalmologists, and promote timely treatment of acute ocular disease in SJS/TEN and related conditions. By serving as a standardised assessment tool, it has potential utility in both clinical practice and research settings to reduce variation in care and improve patient outcomes.

Table 1

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Sotozono grading adaptation for Dermatologists; May 2025

Sotozono, C. et al. Predictive factors associated with acute ocular involvement in Stevens-Johnson syndrome and toxic epidermal necrolysis. Am. J. Ophthalmol. 160, 228–237 (2015).

Patient details						
Date of admission	**Dermatology assessment**			**Ophthalmology assessment**		
	Ask if vision impaired **Examine the eyes & take photos**	Right eye Y/N	Left eye Y/N		Right eye Y/N	Left eye Y/N
Grade 0 No eye involvement						
Grade 1 Conjunctival hyperaemia	**Date** **Refer to Ophthalmology**	**Date** **Free text**				
Hyperaemia - red eye Conjunctival hyperaemia is noted in both eyes. Even when hyperaemia looks mild, epithelial defect may be present concomitantly. Patient should be referred to ophthalmologist for further assessment and management of potential presence of punctate keratitis, pseudomembranes, corneal erosions, conjunctival adhesions	**Impaired vision : patient report**			Impaired vision		
	Red Eye - hyperaemia			Eyelid oedema		
	Eyelid oedema			Mild conjunctival injection		
	Purulent discharge			Chemosis		
	Eyelid closure					
Grade 2 Conjunctival hyperaemia Either ocular surface epithelial defect * Pseudomembrane	**Date** **Refer to Ophthalmology**	**Date** **Free text**				
Pseudomembrane A pseudomembrane is composed of fibrin, necrotised epithelial cells and infiltrating cells (mainly neutrophils), indicates severe inflammation on the ocular surface. If treatment works, pseudomembranes will spontaneously become milder. Increased pseudomembrane formation noted during treatment suggests aggravation of inflammation on the surface of the eye.	**Impaired vision : patient report**			Impaired vision		
	Red eye- hyperaemia			Punctate keratitis		
	Eyelid oedema			Either epithelial defect		
	Purulent discharge			Corneal erosions		
	Eyelid closure			Corneal infiltrate		
	Pseudomembrane					
Grade 3 Conjunctival hyperaemia Both ocular surface epithelial defect & Pseudomembrane + Symblepharon	**Date** **Refer to Ophthalmology**	**Date** **Free text**				
Symblepharon Severe inflammation affects conjunctiva over sclera as well as inner eyelids and can be clinically appreciated by redness and swelling of the eyelids. It may be accompanied by acute loss of eyelashes in severe cases. Symblepharon (adhesion of the eyelid conjunctiva and the eyeball/ scleral conjunctiva) conjunctival rapidly progresses to irreversible changes.	**Impaired vision : patient report**			Impaired vision		
	Red eye- hyperaemia			Both epithelial defects		
	Eyelid oedema			Conjunctival fornix foreshortening		
	Purulent discharge			Symblepharon formation		
	Eyelid closure					

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Abstract N°: 8770

Toxic epidermal necrolysis: clinical aspects, treatment, complications and follow-up

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Introduction & Objectives: Toxic epidermal necrolysis (TEN) is an acute and serious adverse drug reaction with mucocutaneous involvement in more than 30% of the body surface. It has a mortality rate of approximately 25-30% in addition to the high risk of chronic multi-systemic sequelae. The diagnosis is clinical, and treatment includes discontinuation of the causative drug and interdisciplinary support in an intensive care unit (ICU). Effective specific therapies are being researched for better management of the patients.

Materials & Methods: Retrospective, observational, descriptive, cross-sectional, case series study of patients diagnosed with toxic epidermal necrolysis, treated from 2020 to 2025. All patients were evaluated according to the protocol established by Paradisi *et al.,* 2014 (table 1) and staged the possibility of systemic therapy within 6 hours of their arrival at the hospital or request for evaluation, by the same dermatologist.

Results: Five patients were diagnosed with TEN. Four male patients were diagnosed with TEN and were able to receive etanercept 50 mg, subcutaneously, in a single dose, in addition all patients were treated at ICU and with immediate suspension of the causative drug (figure 1 and table1). One female patient received only support therapy due to sespsis at the admission.

Case 1: A 57-year-old male patient, after 4 days of exposure to lamotrigine, diagnosed with NET SCORTEN 4. He didn't have any complications after 22 days of treatment.

Case 2: A 44-year-old male patient, 5 days after the start of the rash, NET SCORTEN 3, due to Coronavac. During hospitalization, he had sepsis due to *Staphylococcus aureus*, tuberculosis and chronic obstructive pulmonary disease. He had, after 33 days, visual sequelae.

Case 3: An 88-year-old male patient treated at another service was being investigated for dengue, hemolytic-uremic syndrome, endocarditis and meningococcemia. After 4 days of hospitalization, he was transferred and diagnosed with NET SCORTEN 4 due to beta-lactam. He developed acute kidney injury and refractory septic shock caused by *Candida albicans*.

Case 4: A 32-year-old female patient, 17 days after starting lamotrigine, presented a rash treat like allergy in another service. She received azathioprine, prednisone, cyclosporine, immunoglobulin. She remained hospitalized for 11 days, when she was transferred to our service and diagnosed with NET SCORTEN 4. In addition, she was diagnosed with abscesses in iliac vessels and septic shock. She developed pulmonary thromboembolism, hemophagocytic syndrome, and refractory septic shock due to *Pseudomonas aeruginosa*. Case 5: A 41-year-old male patient had NET SCORTEN 3, 7 days after starting allopurinol. Admitted to another service, he used methylprednisolone and immunoglobulins. The patient was discharged 14 days after admission with global onycholysis.

Conclusion: TEN is a rare and serious drug-induced disease with a high rate of mortality and complications. Although it is a medical emergency, there is still no effective and well-defined specific treatment. Lack of knowledge and delayed diagnosis are the main reasons for poor outcomes. It is necessary to define the causative drug and exclude its use. Studies show that etanercept may be a new approach for TEN.

Abstract N°: 8839

A Menstrual-Linked Rash That Wasn't: Etoricoxib-Induced Fixed Drug Eruption Confirmed by Lesional Patch Testing

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Introduction & Objectives:

Fixed Drug Eruption (FDE) is a delayed-type hypersensitivity reaction characterised by recurring lesions at the same site following drug re-exposure. Lesional patch testing may be necessary to confirm the diagnosis, especially when standard testing is inconclusive. We describe a rare case of FDE due to Etoricoxib that mimicked erythema multiforme and was only diagnosed after targeted lesional patch testing.

Materials & Methods:

A 24-year-old woman from Hong Kong presented with a 4-month history of recurrent vesicular eruptions affecting her lips, periorbital area, oral mucosa, neck, arms, and legs. The eruptions occurred monthly, typically the day after the onset of menstruation. She was previously diagnosed with erythema multiforme or recurrent herpes simplex and treated empirically with prophylactic acyclovir without benefit. Viral PCR was negative.

A second-opinion consultation revealed concurrent menorrhagia and use of over-the-counter**Etoricoxib 60 mg**, which she took at the start of each menstrual cycle. This was not recorded in her UK medical notes. Patch testing was performed using the standard baseline, facial, and photopatch series including NSAIDs, and Etoricoxib (crushed in paraffin) applied under occlusion to **non-lesional skin on the back** and **lesional skin on previously affected body sites**.

Results:

At 48 hours, all patch tests were negative. At 72 hours, vesicular reactions appeared*only* at the lesional sites, clinically replicating her prior flares. No reaction was noted at the non-lesional back test sites. The patient discontinued Etoricoxib and has since had complete resolution of symptoms, confirming the diagnosis of FDE.

Conclusion:

This case highlights the importance of thorough medication history-taking—including over-the-counter and international drug use—in patients with atypical rashes. The cyclical timing of the eruptions in this patient was a misleading association, delaying the correct diagnosis. Lesional patch testing proved essential in uncovering FDE when standard testing was inconclusive. Although rare, Etoricoxib-induced FDE should be considered in EM-like presentations with fixed localisation. This case reinforces the diagnostic value of targeted patch testing and clinician awareness of non-documented drug exposures.