M031

PHOTOALLERGY TO SUNSCREEN: AN UNEXPECTED TRIGGER OF ANAPHYLAXIS

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Introduction: Photoallergy is a reaction caused by an allergen that requires UV radiation to trigger immune responses mediated by T cells through the interaction of Langerhans cells. Sunscreens (SS) are a group of contact photoallergens due to their potential photosensitizing properties and increased use over the last decades. Manifestations include acute eczema, lichenoid, or urticarial lesions on the sun-exposed areas of the skin where contact with the substance occurred. There is no epidemiological information in the literature about systemic manifestations and anaphylaxis due to photoallergy.
Case Description: A 25-year-old woman with a history of contact urticaria following the application of various SS and makeup products. Her condition began 6 hours after applying SS, presenting with localized urticaria on her forearm. Upon exposure to sunlight, she developed facial erythema, dyspnea, palpitations, and wheezing, prompting her to seek emergency care. Eight weeks later, photopatch tests were conducted, and the results were positive for octocrylene.
Discussion: Octocrylene is a UV absorber used in personal care products. The incidence of photoallergy to octocrylene has increased in recent years. Photopatch testing is required for a definitive diagnosis. The main management strategy aims to identify the causative photoallergen to avoid future exposures. SS contains potential contact allergens that can induce contact photodermatitis and even anaphylaxis. Identifying these allergens through photopatch testing allowed us to provide the patient with safe photoprotection options.

M032

DASATINIB INDUCED HYPEREOSINOPHILIC SKIN ERUPTION AND RESPIRATORY FAILURE

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Introduction: Dasatinib, a second-generation tyrosine kinase inhibitor (TKI), is used for treating chronic myeloid leukemia (CML). There are reported cases of pulmonary toxicity as a possible side effect of Dasatinib but hypereosinophilic syndrome with cutaneous and pulmonary involvement is under-recognized.
Case Description: A 39-year-old male with a recent CML diagnosis status post bone marrow biopsy, was receiving dasatinib for rapidly progressive CML for one month before presenting to the hospital with fever, dyspnea, and lower extremity morbilliform eruption. He required oxygen therapy for acute respiratory failure and was noted to have an eosinophil count of 1,430 cells/microliter. CT chest revealed bilateral lower lobe ground glass/consolidative opacities. Despite receiving antibiotics for a possible infectious etiology, his respiratory status worsened, and his eosinophil count continued to rise. Infectious work-up was non-revealing. Steroid therapy was initiated and within two days eosinophil counts normalized, and respiratory symptoms resolved. Repeat imaging showed resolution of the previous pulmonary findings. Dasatinib was deemed to be the culprit and was discontinued. Risk versus benefit for initiating other TKI was thence discussed with the patient. Steroid taper was started and bosutinib was initiated. Eleven days later, patient was re-admitted for rash recurrence and eosinophilia. Bosutinib was discontinued and asciminib, an emerging TKI, was initiated, which the patient tolerated.
Discussion: Dasatinib-induced hypereosinophilic syndrome is under-reported. This case demonstrates the importance of maintaining high clinical suspicion for this association and timely initiation of steroid therapy. Further research on cross reactivity between TKIs is needed to inform treatment in such clinical scenarios.

M033

A CASE OF ANAPHYLAXIS TO CARDIAC ULTRASOUND ENHANCING AGENT

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Introduction: Ultrasound enhancing agents (UEAs) improve diagnostic echocardiograpic accuracy in assessment of myocardial structure and function. UEAs (e.g. sulfur hexafluoride lipid-type A microspheres and perflutren lipid microspheres) which contain polyethylene glycol (PEG), are gas-filled microspheres encapsulated in a phospholipid or albumin shell that undergo resonant oscillation in size in response to ultrasound waves thus enhancing echogenicity and echocardiographic visualization. In 2021 the FDA reported 11 cases of anaphylaxis and two deaths, following administration of the above UAEs. All 11 cases reported allergy to PEG. Since then, there have been additional reports of anaphylaxis to UEAs. We present the first case of a patient with anaphylactic reaction to Lumason with hereditary alpha-tryptasemia (HaT) and without allergy to PEG.
Case Description: A 67-year-old female with moderate aortic stenosis, naïve to UAEs and with a history of tolerance to PEG 3350 on multiple occasions, experienced itching, urticaria, shortness of breath, and LOC, immediately after injection of sulfur hexafluoride lipid-type A microspheres. She was treated with IM Epinepherine, IV Diphenhydramine and Methylprednisolone. An event tryptase was not drawn. Skin prick testing to both PEG 3350 and UAE was negative with appropriate controls. Baseline tryptase was persistently elevated (12-13ng/mL) and HaT testing revealed an extra-allelic copy of TBSB1.
Discussion: Risk of hypersensitivity reaction with UAEs should be considered in selection of cardiac imaging modalities. Anaphylactic/anaphylactoid reactions can occur with administration of UAEs in patients without allergy to PEG and should prompt evaluation for conditions predisposing to anaphylaxis, including HaT and systemic mastocytosis.

M034

WHEN A FRIEND BECOMES A FOE – CIRCUMVENTING IVIG REACTIONS

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Introduction: Cutaneous adverse reactions to intravenous Ig replacement therapy (IVIG) are rare. We present a severe case of psoriatic dermatitis following IVIG, highlighting potential side effects and therapeutic challenges.
Case Description: A 72-year-old Caucasian male with neuromyelitis optica presented with suspected IVIG reaction used for secondary hypogammaglobulinemia due to rituximab. The patient received Gammagard infusion and cefdinir and developed minimally pruritic skin rash with generalized peeling/scaling within 2 weeks. He was treated with steroid taper and cetirizine. Cefdinir and home allopurinol were discontinued. At the 4-week interval, he received methylprednisolone premedication and another Gammagard dose with worsening rash. Ig replacement therapy was discontinued and he was diagnosed with drug-induced psoriasis requiring 6-month treatment of apremilast and phototherapy with subsequent resolution. Given persistent hypogammaglobulinemia and sinopulmonary infections, patient was initiated on subcutaneous therapy with premedication.
Discussion: IVIG is generally well tolerated, with flu-like illness being the most common side effect. Adverse dermatological reactions are rare with reported incidence of 6%, including urticaria, maculopapular rash, lichenoid eruption, eczema (dyshidrotic), desquamation, and rarely erythema multiforme. Psoriasiform dermatitis has been reported 1-2 weeks post-infusion with earlier onset with subsequent doses. These typically resolved with or without topical/systemic steroids within 1-4 weeks. Suggested mechanisms include immune complex phenomena from interaction with microbial antigens, effects from plasma-derived vasoactive/hemodynamically active