

stings, he developed full body urticaria, emesis, pallor, and lethargy. Mom called 911.

At the scene, he had hypotension, tachycardia, perioral cyanosis, upper extremity urticaria and mild abdominal discomfort. Intramuscular epinephrine was administered with improvement in symptoms. In the Emergency Department, he received intravenous fluids, methylprednisolone, dexamethasone, diphenhydramine, cetirizine and ondansetron as adjunctive treatments. He was discharged home after observation with a prescription for epinephrine autoinjectors and to complete one week of cetirizine.

Total tryptase drawn 6 hours after epinephrine administration was 4.2 mcg/L, and total IgE was 336 unit/mL. Venom serum-specific IgEs were positive: honeybee 14.70 kU/L, wasp 3.12 kU/L, white-faced hornet 23.00 kU/L, yellow-faced hornet 26.40 kU/L, and yellow jacket 24.70 kU/L. Skin testing to honeybee and wasp is pending. Physical examination at follow-up in the Allergy Clinic was unremarkable, with no skin lesions concerning for mastocytosis.

Discussion: This patient's symptoms were consistent with anaphylaxis, with no likely alternative cause besides a Hymenoptera venom allergy. Identifying a sting days preceding anaphylaxis may identify candidates for venom immunotherapy, ultimately providing a life-saving therapy.

M018

A CASE OF RECURRENT ANAPHYLAXIS DURING METRONIDAZOLE DESENSITIZATION

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Introduction: *Trichomonas vaginalis* is one of the most common sexually transmitted infections. Nitroimidazoles (metronidazole and tinidazole) are the only class of medications used for treatment. If the patient has immediate IgE-mediated reactions to either medication, they should undergo desensitization.

Case Description: A 32-year-old female with a history of anaphylaxis to metronidazole presented to clinic for desensitization. She had undergone desensitization three times prior to this presentation, with an episode of anaphylaxis each time. She was always able to complete the desensitization after treatment with intramuscular epinephrine. She had previously tolerated 10mg without symptoms but had pruritus with subsequent doses. Premedication with cetirizine was done for this desensitization. We created a 10mg/mL solution from 250mg tablets. She tolerated 10mg and 25mg, developed mild GI symptoms with 50mg, which did not recur with repeating 50mg but did cause mild pruritus. 100mg triggered diffuse, intense pruritus and acute intestinal symptoms. Intramuscular epinephrine led to rapid resolution of symptoms. 100mg was repeated, followed by 150. A half tablet (125mg) was given and tolerated twice, with five final doses given as a full 250mg tablet, all tolerated, to achieve the target total dose for treatment.

Discussion: Despite reported success with currently published metronidazole desensitization protocols, there remains a lot of variation in patient tolerability during desensitization. For patients with known systemic reactions, a more gradual increase in medication dosing with longer intervals between doses should be considered. Although there is variable data, another consideration is to do pre-treatment with antihistamines and oral systemic steroids prior to desensitization.

M019

COVID-19 VACCINE-ASSOCIATED ANAPHYLAXIS: BASOPHIL ACTIVATION IN A SPECIFIC IGE AND IGG NEGATIVE PATIENT

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Introduction: The burgeoning investigation of COVID-19 vaccine anaphylaxis has demonstrated different potential immune reaction

profiles. We discuss an in depth *in vitro* and *in vivo* analysis of a case of non-IgE mediated hypersensitivity to the Pfizer-BioNTech COVID-19 vaccine excipient polyethylene glycol (PEG).

Case Description: A middle-aged woman received her first dose of the Pfizer-BioNTech mRNA vaccine (BNT162b2), and within five minutes developed signs and symptoms of anaphylaxis, including hypotension, wheezing, throat tightness, and gastrointestinal upset. She was administered intramuscular epinephrine with refractory symptoms ultimately requiring an epinephrine infusion and ICU level of care. Three weeks following the index event, follow up studies were conducted, which were significant for negative skin prick tests to BNT162b2 and DMG-PEG 2000 as well as negative serum titers for anti-PEG IgG and IgE. There was, however, a detectable increase in the patient's serum basophil activation via CD63 expression when exposed to BNT162b2 and DMG-PEG 2000 compared to control.

Discussion: Activation of basophils following exposure to the vaccine excipient DMG-PEG 2000 suggests that PEG is the likely culprit allergen. However, this reaction appears to be a non-IgE and non-IgG mediated anaphylaxis. PEG may activate the immune system in a non-classic type I hypersensitivity manner, which can include other pathways involving direct mast cell and basophil activation or complement activation-related pseudoallergy (CARPA). Distinguishing such reactions from classic IgE-mediated allergy may be important because they do not reinforce with repeated exposure.

M020

STATIN INDUCED DRUG REACTION WITH EOSINOPHILIA AND SYSTEMIC SYMPTOMS (DRESS) SYNDROME

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Introduction: Statin drugs are widely prescribed in clinical practice. Common side effects experienced by patients include elevated liver enzymes and muscle pain. Severe multisystemic hypersensitivity drug reactions to statins are very uncommon.

Case Description: A 63-year-old woman presented with fever, eyelid edema and a diffuse maculopapular skin rash and bilateral axillary lymphadenopathy. Five weeks prior, she was prescribed 10-day course of clindamycin for right foot cellulitis and coincidentally begun on 20 mg daily rosuvastatin for primary prevention and hyperlipidemia. Evaluation revealed AEC of 1402 cells/uL, creatinine 5.2 mg/dL, ALT 444 U/L, AST 353 U/L and CK of 8066 U/L. The urinalysis showed 5-6 white blood cells and 8-10 red blood cells. Renal biopsy was consistent with AIN and work up for other possible causes was unrevealing. Clindamycin was suspected as the cause of DRESS and the drug was discontinued immediately. Rosuvastatin was also held on discharge due to elevated transaminases and CK. Patient recovered completely on a prolonged prednisone taper. The patient was restarted on rosuvastatin and, three weeks later, she developed a recurrent maculopapular rash, facial edema, lymphadenopathy and fever. Her labs were remarkable for AEC of 1203, creatinine of 3.75 mg/dL as well as elevated liver function tests and CK. Patient was diagnosed with DRESS syndrome due to rosuvastatin. Dress resolved after discontinuation of the drug and treatment with prednisone.

Conclusion: It is important for clinicians to recognize that in addition to rhabdomyolysis and myositis, statin drugs can also present with DRESS.

M021

MULTIPLE CHEMOTHERAPY MEDICATION ALLERGY IN A PATIENT WITH RECURRENT OVARIAN CANCER

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Introduction: Drug allergy is an adverse immunological response to medications that cause symptoms ranging from mild cutaneous