

The Impact of Aeroallergens on Military Readiness: A Case Report

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ABSTRACT Seasonal aeroallergens commonly cause allergic conjunctivitis, rhinorrhea, sinusitis, and cough in sensitized individuals. These clinical symptoms are the result of IgE-mediated type I hypersensitivity reactions, which trigger the degranulation of mast cells and basophils. In contrast, aeroallergens are not common precipitants of urticarial dermatitis, which is driven by a cell-mediated type IV hypersensitivity reaction. In this case report, we describe an Active Duty Air Force member stationed in San Antonio, TX, who presented to the dermatology clinic with a three year history of recalcitrant urticarial dermatitis found to be directly related to exposure to the pollen from the *Juniperus ashei* tree, also referred to as Mountain Cedar. While laboratory findings confirmed a high level of circulating IgE antibody to Mountain Cedar, the patient had no upper respiratory symptoms consistent with a typical allergic reaction. Further, his skin disease rapidly cleared within 24 hours of leaving southern Texas. Because of the recalcitrant nature of his condition upon returning home, he was considered unfit for further military service. This case not only highlights the growing link between IgE and chronic skin disease, but also the impact aeroallergens can have on the medical readiness and world-wide deployability of Airmen, Sailors, Soldiers, and Marines.

INTRODUCTION

Seasonal environmental aeroallergens are common causes of allergy symptoms across the United States. In south-central Texas, one of the most potent allergens is pollen from the *Juniperus ashei* tree, also referred to as Mountain Cedar. The largest concentration of this drought-tolerant evergreen is central Texas, though it ranges from Missouri to northern Mexico.

Mountain Cedar trees typically bloom during the winter months and cause predominantly IgE-mediated Type I hypersensitivity reactions in sensitized patients. Sneezing, rhinorrhea, ocular pruritus and cough often result and can be severe. Some patients experience low grade fevers, a phenomenon locally referred to as Cedar Fever. Subsequent secondary bacterial pneumonia is rare but has been reported. Primary skin disease, including contact urticaria, allergic contact dermatitis, or irritant contact dermatitis, is not commonly associated with exposure to Mountain Cedar allergens in sensitized patients.

In this case report, we describe an Active Duty Air Force Air Traffic Controller with a 3-year history of severe, recalcitrant urticarial dermatitis found to be directly related to exposure to airborne Mountain Cedar allergen. The patient's skin disease was so severe it directly affected his military medical readiness, resulting in a medical board that determined he was unfit for continued military service.

CASE REPORT

A 29-year-old Caucasian male presented to the Wilford Hall Ambulatory Surgical Center Dermatology department for

evaluation of dermatitis. The rash was intensely pruritic, involving >50% of his total body surface area. He denied sneezing, rhinorrhea, and ocular pruritus. Symptoms began three years prior, soon after a permanent change of station to southern Texas, and reportedly flared during the winter months. He had no other significant medical history, including seasonal allergic rhinitis, asthma, or previously diagnosed atopic dermatitis. He took no medications other than prescribed oral Cetirizine at up to 40 mg per day. Interestingly, the patient insisted that his skin condition would rapidly resolve upon leaving southern Texas.

On clinical examination, numerous pink to red, edematous, sometimes scaling plaques covering >60% of the patient's face, scalp, neck, upper trunk, upper extremities and proximal lower extremities were noted. Additional eczematous appearing plaques were also seen in the involved areas. Some facial lesions showed signs of impetiginization, and significant lichenification secondary to chronic excoriation was noted on the bilateral dorsal forearms and proximal thighs. Individual lesions were clinically confirmed to persist longer than 24 hours. The differential diagnosis included *Pityrosporum*-driven atopic dermatitis, sebo-psoriasis, severe seborrheic dermatitis, allergic contact dermatitis and urticarial dermatitis. The contribution of HIV-related immunosuppression was considered a possibility as well.

An unremarkable cell blood count and comprehensive metabolic panel were noted, and the patient was HIV negative. A punch biopsy of a representative lesion demonstrated non-specific psoriasiform spongiotic dermatitis, and a superficial perivascular lymphocytic infiltrate with rare eosinophils. Patch testing against the North American Standard and Medicament panels was negative.

Initial treatment included oral cephalexin 500 mg QID for 7 days, oral fluconazole 200 mg per week for 4 weeks, triamcinolone 0.1% ointment from the neck down twice daily for

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doi: 10.1093/milmed/usz106

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2 weeks, and tacrolimus 0.1% ointment for head/neck lesions. Hypoallergenic soaps, shampoos, laundry detergents, and topical emollients were all recommended and/or prescribed. Upon re-examination one month later, the impetiginized facial plaques had resolved, but the remainder of the examination was only moderately improved. Continued attempts to control the skin eruption with only topical medications proved unsuccessful, as rapid recurrence was noted. Additional labs were drawn and showed a total IgE level of 2218 IU/mL (normal range 0–100). Elevated IgE titers against dog dander (47.10 U/mL), cat dander (27.30 U/mL) and Mountain Cedar (> 100 U/mL) were specifically noted. Interestingly, the patient subsequently traveled out of the state and reported a marked decrease in both generalized pruritus and urticarial skin lesions within 24 hours (Fig. 1).

A diagnosis of chronic urticarial dermatitis secondary to Mountain Cedar allergens was made based off the morphology and clinical course of skin lesions, the histological examination of the biopsy specimen, immunologic laboratory findings, and the resolution of skin symptoms upon leaving South Texas. Upon return to San Antonio, TX, the patient's symptoms rapidly returned. Due to his job as an air traffic controller, systemic treatment options were very limited. Ultimately, a medical board deemed the patient unfit for further military service. One year after leaving San Antonio, the patient reported continued control of symptoms without topical or oral medications.

DISCUSSION

The case described here is significant in that sensitivity to a prominent south Texas aeroallergen resulted in a severe, recalcitrant urticarial dermatitis in the absence of any other allergy symptoms. Laboratory testing confirmed the presence of elevated IgE titers specifically against Mountain Cedar. Unfortunately, trending IgE titers in relation to the patient's

skin disease after permanently moving from the local area was not accomplished. However, the noted rapid clinical resolution of symptoms upon leaving south Texas strongly supported this aeroallergen as the driving force behind the clinical findings.

Multiple diagnoses were entertained, including seborrheic dermatitis, adult atopic dermatitis (AD), and chronic allergic contact dermatitis (ACD), when the patient first presented to the Dermatology clinic. Sensitivity to aeroallergens was not initially thought likely, as the patient denied any upper respiratory symptoms consistent with an IgE-mediated immune reaction. The level of pruritus did not readily support a diagnosis of seborrheic dermatitis, and the paucity of eosinophils noted on evaluation of the skin biopsy argued against allergic contact dermatitis. The diagnosis of urticarial dermatitis was rendered, though its underlying etiology was not known until further laboratory testing was done and the patient traveled outside the local area.

Urticarial dermatitis (UD) is a non-specific diagnosis, however, and is described clinically as multiple urticarial and eczematous plaques that can become scaly and impetiginized, especially in chronic cases.¹ Because the underlying pathophysiologic process of UD- a type IV hypersensitivity reaction- is the same as that for both ACD and flares of AD, it makes sense that these three conditions produce skin lesions that are morphologically similar. Histologic similarities are noted as well, which include non-specific epidermal spongiosis and variable degrees of a psoriasiform reaction pattern.

On the other hand, etiologically UD, ACD and AD differ significantly, particularly for the latter two. First, ACD is not commonly caused by aeroallergens. Metals, fragrances, and Balsam of Peru are its most common triggers, with topical antibiotics, chemicals, adhesives, and preservatives listed as other typical offending agents.² In contrast, atopic dermatitis is considered primarily a compromised skin barrier issue with a large genetic component. It is thought that allergens penetrate more readily through the epidermis of AD patients, resulting in the generation of cell-mediated immune reactions to specific antigens over time. The genetic component of AD also explains why most of these patients present during early childhood.

An essential point in this discussion, though, is that UD, ACD and flares of AD are all mediated by the same immunological process- a cell-mediated type IV hypersensitivity reaction. This is in contrast to type I hypersensitivity reactions- generated through the humoral arm of the immune system- which are dependent on IgE antibodies. In type I reactions, mast cell and basophil degranulation follow IgE binding and cause the symptoms typically associated with seasonal allergic rhinitis (SAR), asthma, true urticaria and anaphylaxis. Our patient had none of these acute symptoms and had no history of SAR or asthma. Rather, he developed a chronic, type IV hypersensitivity mediated dermatitis following repeat exposure to an allergen against which he had markedly elevated IgE titers.



FIGURE 1. Clinical presentation in San Antonio (left) and 24 hours after leaving local area (right).

Historically, IgE-mediated immune reactions have not been considered significant contributors to the development or exacerbation of skin conditions that result from type IV reactions. However, there is mounting evidence that elevated IgE against common allergens can play a role. For example, as many as 80% of children with atopic dermatitis will develop asthma and/or allergic rhinitis as they age,³ conditions known to be mediated by IgE. Up to 63% of infants with AD have concomitant IgE-mediated food allergies.^{4,5} Total serum IgE levels have also been found to be strong predictors of developing AD as children age.⁶ Finally, IgE-mediated food allergy and aeroallergen exacerbations of atopic dermatitis are being confirmed with increasing frequency in the literature.⁷

Though this is only one case report, the clinical findings and course of disease further supports the growing link between IgE and the development of chronic skin diseases otherwise caused by cell-mediated mechanisms. It also suggests there is more overlap between type I and type IV hypersensitivity reactions from a pathophysiologic perspective than previously thought. As in the case presented here, patients with recalcitrant UD, ACD or AD may benefit from a referral to an allergist to facilitate more collaborative care.

From a military perspective, this case highlights the impact aeroallergens can have on military readiness and retention. While most sensitized patients can be managed using over-the-counter antihistamines, others may experience severe disease that is not readily attributed to local allergens. With the growing military presence in south Texas, local allergens have the potential to drastically impact the world-

wide deployability and medical readiness of soldiers, airmen, sailors and Marines.

ACKNOWLEDGMENTS

Dr. Fajardo and Dr. Wohltmann both work for the Department of Defense. However, their opinions are personal and do not reflect the opinions of the United States Air Force, the Military Health System, or the Department of Defense.

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