Airborne Contact Dermatitis
Margaret L. Soderberg, MD
Independent Contractor
Allergy & Immunology
Studio City, CA, USA
April 2015

Airborne contact dermatitis is most frequent in the occupational setting, e.g. as caused by methacrylate resins in the automobile industry or Pinus roxburghii in the woodworking industry. The inhalation of pollen can cause allergic rhinitis and allergic asthma. The latter are typical of an IgE mediated immediate hypersensitivity. What is less known is that airborne pollen may act as a trigger for a delayed hypersensitivity reaction or Type IV cellular response manifested as a form of contact dermatitis.(1)

In my allergy medical practice in Southern California a 58 year old Caucasian male presented to my office for evaluation of what seemed to be an atypical form of atopic dermatitis presented as an erythematous maculopapular rash over the sun exposed areas of his body, e.g. his face and the extensor surfaces of his arms.

Allergic rhinitis was a minor problem for this patient. Allergy skin testing revealed sensitivities to the grass, weed, and tree pollens of Southern California. An IgE level was only moderately elevated. The patient began allergy immunotherapy. Despite adequate medical management, he continued to have eczematous flares on a monthly basis. A crucial historical point was that the patient had a second home in the Palm Springs (desert) area of Southern California where he would spend at least one weekend a month. After 24 hours there, he would develop the same rash in the same atypical locations, which became
florid after 48 hours. He had shrubs of greasewood (Sarcobatus vermiculatus) forming a hedge around the perimeter of the house. Although he was not in physical contact with these shrubs, he wondered if the greasewood was the source of his problem. Sarcobatus vermiculatus is in the Chenopodiaceae family; Genus: Sarcobatus. This plant is native to California, as flora of the desert regions. It is considered to be only a mild allergen which pollinates from spring to summer.

A prick test with freshly crushed fruiting bodies from this shrub was negative. The material was then applied as a patch test to the patient’s upper arm. After 48 hours he developed a 4+ (maximum) reaction to the material. All of the greasewood bushes were removed from the property around his house. His eczematous flares ceased, and he was then able to enjoy his weekends in Palm Springs in good health. Thus, although this aeroallergen is considered a minor trigger for allergic rhinitis, it seems that an intensive/concentrated exposure was instrumental in triggering his eczematous response.

Adinoff A, Tellez P, et al, described a subset of patients with atopic dermatitis who demonstrated both IgE and delayed hypersensitivity mediated immune responses. In this paper aeroallergens eliciting eczematous reactions to patch tests were grasses, trees, weeds, and house dust mites. The corresponding prick tests were positive as well. A control group with only symptoms of rhinitis did not have positive patch tests.(2) Thus in warmer climates, especially in patients with refractory or atypical presentations of atopic dermatitis, an airborne contact dermatitis should be considered in the differential diagnosis. In such instances it would be appropriate to perform patch testing.

References: