

Letters to the Editor

Parthenium dermatitis caused by immediate and delayed hypersensitivity

Dear Sir,

This is with reference to the publication by Mahajan and Sharma on parthenium dermatitis (1).

The authors experimentally proved that the dermatitis can be caused by inhalation of the allergen. They have hypothesized based on the results of (i) positive patch test to parthenium, (ii) negative immediate reaction of the prick test, (iii) positive delayed or late-phase reaction (LPR) at the prick test site on D1, and (iv) histopathological features of vasculitis on biopsy of the LPR that parthenium causes an overlap of type IV hypersensitivity (systemic contact dermatitis), which was evident clinically, and type III hypersensitivity (vasculitis), which was evident on histopathological examination.

We have been intrigued by the dermatitis. Recently, we have postulated the possible role of a combined type I and type IV hypersensitivity in parthenium dermatitis (2).

We were able to demonstrate a raised serum immunoglobulin E (IgE), positive patch test and positive prick test (both immediate reactions at 15 min characterized by the weal and flare and the LPR at 2 days). Recently, we also biopsied the prick test and the patch test sites after 2 days. Both biopsy samples showed identical features but with more severe changes in the prick-tested site.

The patch test site showed spongiosis and mild lymphocytic exocytosis suggestive of delayed hypersensitivity (type IV). The upper and mid dermis showed nuclear dust, lymphocytes, eosinophils, and neutrophils. These cells also surrounded the blood vessels suggestive of vasculitis. Occasional mast cells were present. The prick test site showed more severe changes with epidermal vesicles, bulky dermal infiltrates with histiocytes, and occasional mast cells. As the histopathology of the LPR is suggestive of vasculitis (3), we propose that parthenium causes systemic contact dermatitis by type I and type IV hypersensitivity and possibly not by type III mechanism.

Most drugs that inhibit the immediate hypersensitivity response do not inhibit the LPR and

vice versa. Typical H1 antagonists significantly attenuate the immediate weal and flare but have minimal effect on the LPR (4). The LPR can also be triggered by various other factors [e.g. compound 48/80, anaphylatoxin and complement-fixing immune complexes, mast cell granules, or purified granule proteins (3)]. This could probably explain the negative immediate reaction and positive delayed reaction (LPR) observed by the authors.

Type I hypersensitivity is mediated by allergen cross-linking of IgE bound to IgE Fc receptors on mast cells. Generation of IgE antibodies in response to inhaled or ingested allergens is the hallmark of the atopic immune system with aggravation of dermatitis on inhalation or ingestion of allergen. This could also explain the aggravation of dermatitis following inhalation of fresh parthenium plants reported by the authors.

The Arthus reaction (type III hypersensitivity) occurs in and around small blood vessels and is frequently demonstrated in skin. Following subcutaneous or intradermal injection of the sensitized antigen, a reaction develops which peaks at 4–10 hr, wanes, and is minimal within 2 days. A classic example is the erythema nodosum leprosum or type 2 Lepra reaction, which is evanescent. It is mediated by IgG precipitating antibodies and is complement dependent. The LPR was initially thought to be an immune-complex-mediated (type III hypersensitivity) reaction; however, precipitating antibodies have not been found and is now known to be IgE dependent (5). The LPR is mediated by newly formed mast cell mediators in concert with inflammatory cells. They generate a second wave of smooth muscle contraction with sustained oedema. This response occurs within 2 days and may persist for several days. It is thought to be responsible for the chronic skin and bronchial hyperreactivity in atopic patients with dermatitis or bronchial asthma, respectively (6).

Thus, we would like to conclude that parthenium dermatitis is a combination of type I and type IV hypersensitivity and possibly not of type I and type III hypersensitivity. In addition, this combined immediate–delayed reaction may

induce exacerbation of lesions in sensitized subjects with atopic dermatitis.

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