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Post-traumatic eczema: a manifestation of the atopic diathesis?

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LETTER

Post-traumatic eczema has been categorized as either an isomorphic responses to trauma in patients with endogenous eczema, or idiopathic in cases without atopic dermatitis^{1,2}. We report a case of idiopathic post-traumatic eczema in a patient with asthma and allergic rhinitis but no history of atopic dermatitis, and speculate that the barrier disruption caused by trauma may provide the “second hit” needed for an atopic dermatitis phenotype in this atopy-prone individual.

A 25-year-old woman was referred for a 3-year history of recurring pruritic skin eruptions after venipuncture, attributed to a metal allergy from the hypodermic needle. She denied applying any topical preparations and had no reactions to alcohol, adhesives, or costume jewelry. Her only medical history was hypothyroidism controlled by levothyroxine. Patch testing with an expanded North American Contact Dermatitis Research Group Screening Series and a Metals Series (Chemotechnique) was negative at 2 and 4 days, suggesting that a metal allergy was not the inciting factor.

A glass slide was used to lightly scratch her volar forearm (figure 1A-C). Similar to venipuncture, she developed pink papules and pruritus around the scratches after 48 hours. The eruption worsened until day 6, and then resolved over the following week. Similar scratches in the popliteal fossa also exhibited surrounding eczema but the flank and buttock showed no reaction.

A biopsy from the ventral forearm on day 0 showed normal skin. Biopsies from days 3 and 6 showed increasing spongiosis and a mixed perivascular infiltrate composed primarily of lymphocytes and eosinophils (figure 1D-F). Immunohistochemical stains with antibodies to CD3, CD4, CD8, CD20, and CD117 (Dako, Capintaria, CA) were used to perform manual cell counts (figure 1G). The majority of the perivascular infiltrate was composed of CD3+ and CD4+ T-cells that increased by 3 – 4-fold as would be expected in an eczematous reaction. The number of CD8+ T-cells remained constant. The number of CD20+ B-cells increased, although this comprised a small subset of the infiltrate. A decreasing number of CD117+ mast cells suggested that the response was not mast cell mediated.

In summary, the patient developed an eczematous eruption after trauma, which was unlikely to be allergic in nature. Intriguingly, she was most sensitive to trauma in areas classically involved in atopic dermatitis, i.e. the antecubital and popliteal fossae, and resistant to this

phenomenon in other areas. Given her history of asthma and allergic rhinitis, we postulate that she may have an immune system prone to atopy but lacking the “second hit” of a skin barrier defect (e.g., filaggrin defect) until a barrier defect is created by trauma, thus leading to an eczematous eruption.

Barrier disruption in mice increased Th2 cytokines and thymic stromal lymphopoietin levels, which are important mediators of atopic dermatitis³. In humans, levels of IL-33 were significantly correlated with excoriation score⁴. Atopic dermatitis has been described as the “itch without a rash,” implying that trauma from scratching may incite the eruption seen in atopic patients. Thus, patients with post-traumatic eczema may provide unique insights into the early patho mechanism of atopic dermatitis.

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Figure.

(A) Day 0, three parallel scratches on the ventral forearm. (B) Day 3, papular eczematous eruption. (C) Day 6, worsened eruption with biopsy sites visible on the proximal forearm (star). (D) Day 0, biopsy of normal skin of ventral forearm. (E) Day 6 perivascular infiltrate. (F) Day 6 higher power view. (G) Characterization of the perivascular infiltrate using cell counts of immunohistochemical stains.