

weekly dose increases, she was able to return to cattle shows several days per week due to rapid clearance of her facial rash. She required no further medications while on injections. Her only breakthrough symptom was during a scheduled intensive cleaning of cattle pens, which caused some trivial eyelid pruritus lasting an hour. Upon reaching the full dose, her Skindex-30 regressed to 32. She had no anaphylactic reactions to any injections and is now receiving the maintenance dose monthly, with a goal of 3 to 5 years' total to attain long-lasting remission, per routine guidelines for aeroallergen immunotherapy.<sup>4</sup>

This case illustrates, to my knowledge, the first successful treatment of allergic contact dermatitis using rapid desensitization to a commercial epithelial cattle hair extract. Immunotherapy potentially offers a specific, highly efficacious treatment for this frequently severe, debilitating disorder.

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## Posttraumatic Eczema: A Manifestation of the Atopic Diathesis?

To the Editor:

Posttraumatic eczema has been categorized as either an isomorphic response to trauma in patients with endogenous eczema or idiopathic in cases without atopic dermatitis.<sup>1,2</sup> We report a case of idiopathic posttraumatic eczema in a patient

with asthma and allergic rhinitis but no history of atopic dermatitis, and we speculate that the barrier disruption caused by trauma provided the “second hit” needed for an eczema phenotype in this atopy-prone individual.

A 25-year-old woman was referred for a 3-year history of recurring pruritic rashes after venipuncture performed for laboratory monitoring, 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 (Fig. 1A–C). Similar to prior reactions after venipuncture (which had not been performed within the preceding month), 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. Scratches in the popliteal fossa also

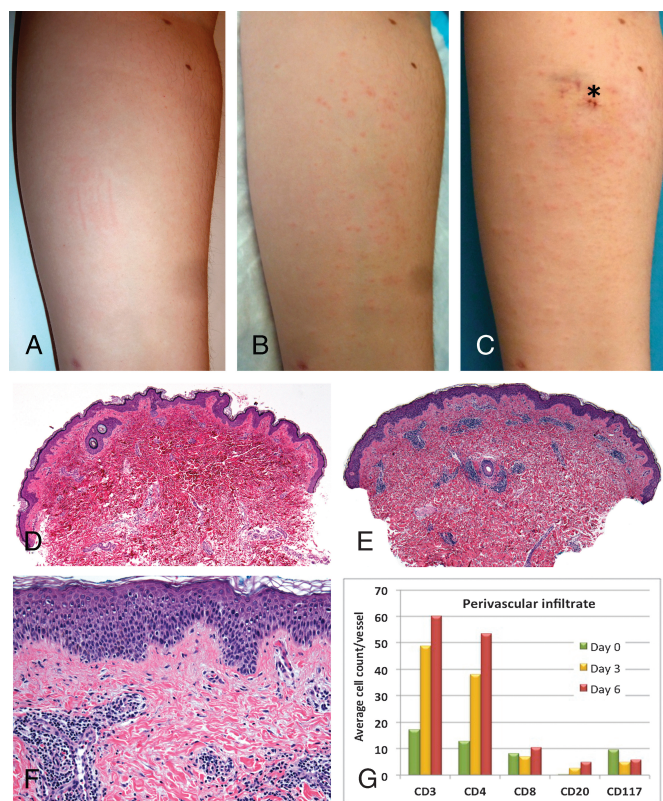


**FIGURE 1.** A, Day 0, 3 parallel scratches on the ventral forearm. B, Day 3, papular eczematous eruption. C, Day 6, worsened eruption with biopsy sites visible on the proximal forearm (asterisk). D, Day 0, biopsy of normal skin of ventral forearm. E, Day 6, perivascular infiltrate. F, Day 6, higher power view. G, Characterization of the perivascular infiltrate using cell counts of immunohistochemical stains.

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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 (Fig. 1D–F). Immunohistochemical stains with antibodies to CD3, CD4, CD8, CD20, and CD117 (Dako, Carpinteria, Calif) were used to perform manual cell counts (Fig. 1G). The majority of the perivascular infiltrate was composed of CD3+ and CD4+ T-cells that increased by 3-fold to 4-fold as would be expected in an eczematous reaction. 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 that was unlikely allergic in nature. Intriguingly, she was most sensitive to trauma in areas classically involved in atopic dermatitis, that is, the antecubital and popliteal fossae. 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 (eg, filaggrin defect) until a defect is created by trauma, leading to an eczematous eruption.

Barrier disruption in mice increased Th2 cytokines and thymic stromal lymphopoietin levels, which are important mediators of atopic dermatitis.<sup>3</sup> In humans, levels of IL-33 were significantly correlated with excoriation score.<sup>4</sup> 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 posttraumatic eczema may

provide unique insights into the early pathomechanism of atopic dermatitis.

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