



Figure 2. Allergic contact dermatitis to genipin in jagua gel (approximately 14 days after the first application).

Genipin is currently used in traditional Chinese medicine, industrial processes, and topical medical devices. It may be used in the future in pharmaceuticals. Application in sensitized patients could result in systemic contact dermatitis or allergic exanthems. Dermatologists should be aware of this new contact allergen in temporary jagua tattoos and should discourage the application of such dyes.

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Psoriasis-like Dermatitis Developing in a Patient with Atopic Dermatitis Treated with Dupilumab

To the Editor:

Atopic dermatitis (AD) and psoriasis are chronic, inflammatory skin diseases that may be phenotypes at polar ends of the T-cell inflammatory milieu.¹ Type 2 T helper (T_H2) cytokines interleukin (IL)-4, IL-13, IL-31, and possibly IL-22 are implicated in AD pathogenesis. Type 1 T helper cytokines IL-17 and IL-23 are implicated in psoriasis, without activation of IL-4 or IL-13.¹ Dupilumab is a fully human monoclonal antibody targeting the IL-4 receptor α subunit, thereby blocking IL-4

J.I.S. served as a consultant and/or advisory board member for Abbvie, AnaptysBio, Asana, Arena, Boehringer-Ingelheim, Dermavant, Eli Lilly, Galderma, GlaxoSmithKline, Glenmark, Kiniksa, Leo, Menlo, Novartis, Pfizer, and Regeneron-Sanofi; is receiving honoraria from RealM; is speaker for Regeneron-Sanofi; and received research grants from GlaxoSmithKline. The other authors have no funding or conflicts of interest to declare.

M.S. and J.I.S. had full access to all of the data in the study and take responsibility for the integrity of the data. J.I.S. conceived and designed the study. J.I.S., M.S., J.G., and T.T. performed the acquisition, analysis, and interpretation of data. M.S. and J.I.S. drafted the manuscript. J.I.S., M.S., J.G., and T.T. performed the critical revision of the manuscript for important intellectual content.

DOI: 10.1097/DER.0000000000000509

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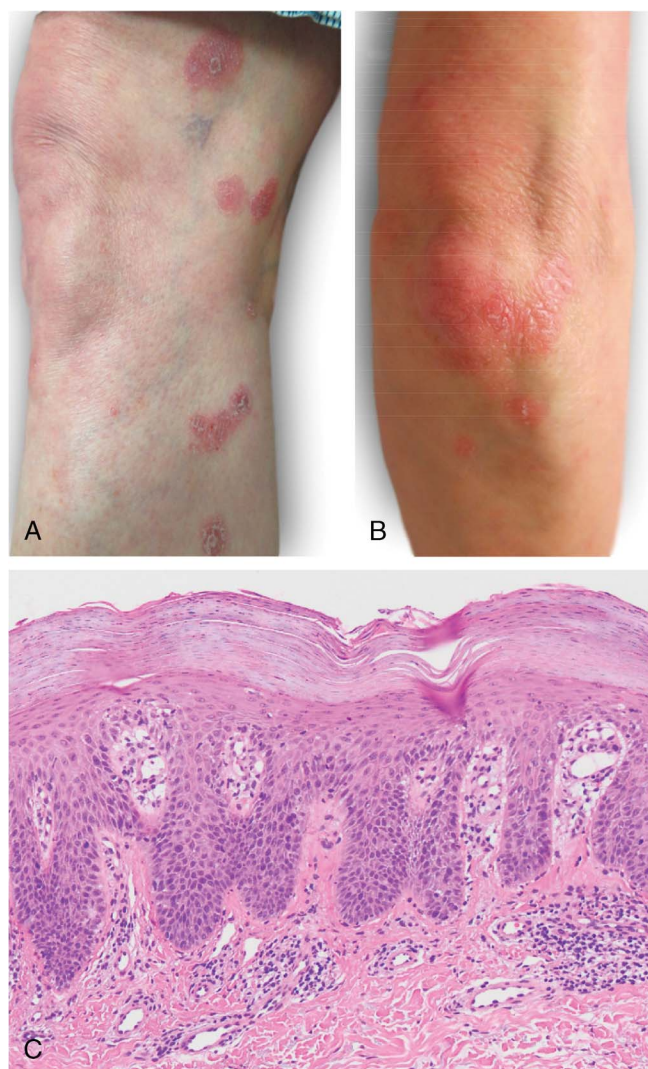


Figure 1. Psoriasiform plaques on the lower (A) and upper (B) extremities. C, Histopathology revealed regular acanthosis with thinning of suprapapillary plates and confluent parakeratosis with absence of the granular cell layer consistent with psoriasis.

and IL-13 signaling. We present a case of psoriasis that developed in an adult with AD who was treated with dupilumab.

A 59-year-old woman was seen at the dermatology practice at Northwestern Medicine for assessment and management of AD. She had childhood AD that remitted in adolescence but recurred at the age of 56 years. Medical history was significant for seasonal allergies diagnosed in adolescence and hypertension, hypercholesterolemia, and depression diagnosed in middle age. She had no prostheses or dental implants. Family history included AD in her mother and father and allergic rhinitis in her brother. Physical examination was notable for generalized, erythematous, ill-demarcated, lichenified plaques and erosions, including the popliteal fossae, ankles, wrists, and anterior neck. Punch biopsy was previously performed by another dermatologist. Histopathology

revealed spongiotic dermatitis with eosinophils. Epicutaneous patch testing was performed to the North American Contact Dermatitis standard and supplemental allergen series. She had numerous irritant reactions and weak positive reactions to 2-hydroxyethyl methacrylate, ethyl acrylate, coconut diethanolamide, and cinnamic aldehyde. Her condition only partially improved with strict allergen avoidance and optimized topical therapy. She was deemed to have AD based on Hanifin-Rajka² criteria (chronic, pruritic, flexural lesions; personal and family atopic history; early-onset xerosis; tendency toward skin infection; hand dermatitis; cheilitis; anterior neck fold lesions; facial erythema; intolerance to wool and solvents; worsening with emotional factors).

Subcutaneous dupilumab was started with 600-mg loading dose, followed by 300 mg every other week. At baseline, she had moderate to severe AD (eczema area and severity index = 12.8, average numerical rating scale itch = 10, Patient-Oriented Eczema Measure = 20, and Dermatology Life Quality Index = 22). After 16 weeks, all signs and symptoms cleared. After 36 weeks, she self-discontinued dupilumab. One year after discontinuation, her AD flared again. She had an inadequate response to triamcinolone 0.1% ointment. Dupilumab was restarted. Within 2 weeks, she noted clinical improvement of her signs and symptoms. However, at 4 weeks, she developed new lesions on the extensor elbows and knees, forearms, lower legs, and thighs. She reported that these lesions looked different and “felt different” than her previous AD lesions (“irritated” but not pruritic). Physical examination revealed psoriasiform plaques on the bilateral extensor surfaces of her upper and lower extremities (Figs. 1A, B). A 3-mm punch biopsy was performed of a lesion on the thigh. Histopathology revealed confluent parakeratosis with absent granular cell layer, regular acanthosis, and thinning of the suprapapillary plates consistent with classic psoriasis vulgaris (Fig. 1C). Dupilumab was discontinued, and lesions improved with topical triamcinolone ointment.

The mechanisms of psoriasis developing in dupilumab-treated patients remain unknown. Targeted blockade of CD4⁺ T_H2 cytokines IL-4 and IL-13 may result in unchecked signaling of other CD4⁺ T_H subsets (eg, T_H1 or T_H17) or CD8⁺ T cells. In particular, CD8⁺ T cells were found to produce IL-22 and IL-17 in psoriatic skin lesions and may contribute to psoriasis in dupilumab-treated patients.³ We were unable to examine the T-cell cytokine and receptor profile in this patient. Although it is possible that she had AD and psoriasis at baseline, there were never any psoriasiform lesions on previous examinations or patient reports. Previous case reports also reported dupilumab-induced psoriasis.^{4,5} However, psoriasis was not a reported adverse event in the clinical trials for dupilumab. Future real-world studies are needed to determine whether psoriasis truly occurs at higher incidence in dupilumab-treated patients.

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