

Confluent Erythematous Plaques on the Palm

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A 45-year-old woman was referred to dermatology by her general internist for the management of a pruritic rash on the hands and feet that was unresponsive to topical steroid creams. The pruritus also was unresponsive to hydroxyzine and aspirin. Erythematous plaques were present on the palms and soles. Physical examination revealed thickened volar skin with a yellowish surface. There were individual papules with atrophic tops at the edge of the plaques on the inner wrists. The patient's medical history was otherwise unremarkable. Blood tests for glucose and liver function did not reveal any abnormalities.

What's the diagnosis?

- a. hereditary palmoplantar keratoderma
- b. hyperkeratotic eczema
- c. palmoplantar lichen planus
- d. psoriasis
- e. secondary syphilis

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Palmoplantar Lichen Planus

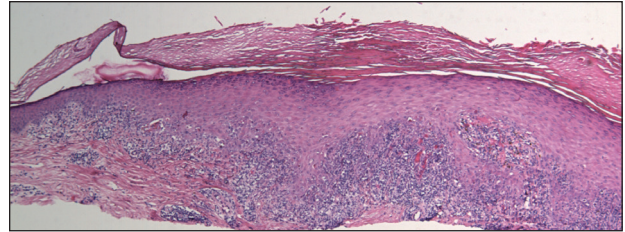
A skin biopsy from a lesion on the inner wrist showed an interface pattern with a dense bandlike infiltrate obscuring the dermoepidermal junction coupled with a superficial perivascular infiltrate (Figure). At higher magnification ($\times 10$), the histologic features included compact orthokeratosis, wedge-shaped hypergranulosis, vacuolar degeneration of the basal layer, basal dyskeratosis, a dense lymphohistiocytic infiltrate obscuring the basement membrane, and melanophages in the papillary dermis.

Lichen planus (LP) is a common inflammatory disease of the skin presenting with flat-topped, violaceous, polygonal papules with fine white lines (Wickham striae) on the surface. It is recognized and diagnosed clinically by its characteristic appearance. Common areas of LP presentation include the shins, inner thighs, genitalia, trunk, volar aspect of the wrists, and oral mucosa.

Palmoplantar LP can present as erythematous plaques, punctuate keratosis, diffuse keratoderma, or ulcerated lesions. The most common concern among patients with LP is pruritus. One-fourth of patients with LP may present with lesions on the palms and soles, but diffuse palmoplantar hyperkeratosis is rare.¹ Lesions typically heal in 1 to 8 months, with an average of 3 months. Palmoplantar LP recurs within 1 year after stopping treatment in one-third of patients.¹

The cause of LP is unknown, but the pathophysiology is beginning to be understood. Cytotoxic CD8⁺ T cells stimulate apoptosis of the keratinocytes. The induction of this mechanism may be due to a self-antigen in a genetically predisposed patient. The evidence for LP being an autoimmune disease is supported by the high female predominance and the association of LP with other autoimmune diseases.² Patients with LP have an increased chance of coexisting hepatitis C virus. In a cross-sectional study of 303 patients, Lodi et al³ found that approximately 20% of LP patients were hepatitis C virus seropositive.

Treatment options for LP include topical and systemic steroids, tazarotene, acitretin, and immunosuppressive agents.⁴ Our patient initially was treated with oral cyclosporine 100 mg every morning and



Wrist biopsy of lichen planus (H&E, original magnification $\times 2$).

oral methotrexate at a dose of 7.5 mg weekly. She also was treated with clobetasol ointment 0.05%. After 3 months, cyclosporine was discontinued. Methotrexate was maintained. At 5 months' follow-up there was marked improvement of both clinical and symptomatic concerns with only residual palmoplantar erythema.

The differential diagnosis for pruritic palmoplantar hyperkeratosis is large. The most common differential diagnoses include hyperkeratotic eczema, psoriasis, secondary syphilis, and hereditary palmoplantar keratoderma. Lichen planus should be considered in the differential diagnosis of palmoplantar hyperkeratosis. A skin biopsy may be needed, as palmoplantar LP often has an atypical presentation.⁵

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