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Case Presentation

Palmoplantar lichen planus

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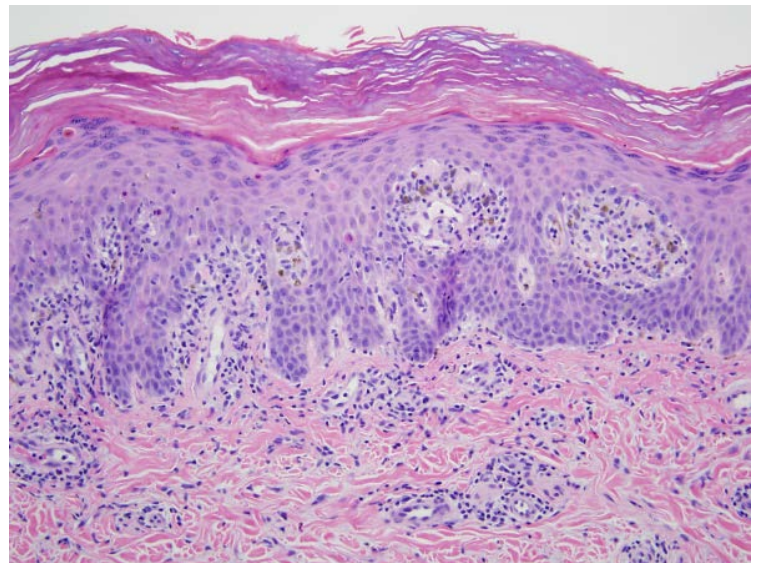
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Abstract

Palmoplantar lichen planus (PPLP) is an uncommon variant of lichen planus that affects the palms and soles. Clinical findings are varied although they have been conceptualized into two large groupings, an erythematous scaly pattern and a hyperkeratotic pattern. Histopathologic features are those of classic LP. We present a case of PPLP that improved with methotrexate after failing treatment with acitretin.



Case synopsis

History: A 51-year-old woman presented to the Skin and Cancer Unit in June, 2013, with a two-month history of painful, scaly plaques of the palms and soles. She had no systemic symptoms. She had been seen by an outside dermatologist who treated her with topical glucocorticoids for presumed psoriasis. Family history included psoriasis in her mother. She worked in design and marketing and denied occupational contact allergens and irritants. She denied fevers, chills, diaphoresis, sun sensitivity, joint pain, antecedent infections, and any medications, which included prescription and over-the-counter pharmaceuticals.

Physical examination: Well-demarcated, erythematous and scaly plaques were present on the palms, soles, and elbows. The left great toenail and right fingernails were dystrophic. The oral mucosa was normal.

Laboratory data: A complete blood count, fasting lipid panel, and comprehensive metabolic panels were normal. Purified protein derivative and hepatitis B and C serologies were non-reactive.

Histopathology: There is a superficial, mid perivascular, patchy, band-like lymphocytic infiltrate. Lymphocytes are present at the dermoepidermal junction where there are dyskeratotic keratinocytes, which also are present within the spinous layer. There is accentuation of the granular in foci and predominantly hyperkeratotic orthokeratosis.

Discussion

Diagnosis: Palmoplantar lichen planus

Comment: Lichen planus (LP) is an idiopathic inflammatory dermatosis that affects the skin, hair, nails, and mucous membranes [1]. LP is seen in 0.2 to 1% of the population, most commonly presenting in the fifth or sixth decade [2]. Classical features include flat-topped, polygonal, purple-to-violaceous plaques that involve the forearms, aspects of the volar wrists, distal aspects of the lower extremities, and genitalia. Lesions typically are covered by a mesh of fine white lines known as Wickham's striae; an isomorphic response, (Koebner reaction) commonly is seen. In nearly 75% of patients with cutaneous LP, the mucous membranes, particularly the oral mucosa, may be affected. Histopathologic features of LP include a band-like, lymphocytic infiltrate underlying an acanthotic epidermis with saw-toothed rete ridges, hypergranulosis, apoptosis, and basal layer destruction. Although the pathogenesis of LP remains unclear, growing evidence suggests that LP represents a T-cell-mediated autoimmune disease with direct damage to basal keratinocytes that express altered self-antigens on their surface [1]. Such antigens include viruses (e.g. hepatitis C), contactants, and exogenous drugs (e.g. angiotensin converting enzyme inhibitors, antimalarials, and diuretics). Although the aforementioned clinical features describe the classical presentation of LP, several clinical variants exist, which include, but are not limited to, annular, bullous, hypertrophic, inverse, linear, and drug-induced LP.

A less common variant, palmoplantar LP (PPLP) rarely has been described in the medical literature. This entity does not share the morphology of classic LP, which makes the diagnosis difficult [3]. PPLP often may be mistaken for other diagnostic entities, which commonly present on the palms and soles. Clinically, PPLP is localized to the palms, soles, and malleoli, with plantar more common than palmar involvement. In addition, PPLP does not demonstrate Wickham's striae or polygonal morphology [4]. The largest case series has separated cases into two morphologic groupings, an erythematous scaly pattern and a hyperkeratotic pattern, although a literature review indicates that all cases do not neatly fit into this scheme [5]. Thus to date, there exists no classic or unifying description of PPLP. Many phenotypes have been described, which include vesicle-like papules, pustule-like papules, umbilicated papules, pitted plaques, and ulcerative, perforating, and petechial lesions [6-11]. Although our patient clinically was presumed to have psoriasis with a consideration of pityriasis rubra pilaris, other case reports have indicated PPLP being mistaken for entities such as tinea nigra, arsenical keratosis, eczematous dermatitis, and secondary syphilis [3, 12]. The differential diagnosis also would include verrucae vulgaris, acrokeratosis paraneoplastica, and punctate prokeratosis. Of note, another important entity to be considered is the LP-lupus erythematosus (LE) overlap syndrome. Such patients have features of both LP and LE with LP lesions occurring preferentially in acral sites. Though clinically similar, patients with LP-LE should demonstrate histopathological findings of both LP and LE [1]. Owing to the histology of our patient's biopsy, the LP-LE syndrome was ruled out.

Owing to the dearth of literature, treatment for PPLP remains experimental. Case reports have suggested rapid response with superpotent topical glucocorticoids, systemic medications such as cyclosporine and acitretin, and combination of topical glucocorticoids with the aforementioned systemics [4, 11, 13, 14]. Our patient was treated with a combination of clobetasol under occlusion, coal tar soaks, and two months of acitretin, which yielded some improvement but resulted in alopecia, xerosis, and xerophthalmia. Upon cessation of acitretin, cutaneous findings began to progress cranially with development of erythematous, scaly plaques on the lower legs. The patient then was treated with methotrexate titrated up to 25mg weekly, folic acid supplementation daily, and urea 40% cream twice daily with robust improvement over two months. These results are consistent with the published literature, which indicates that PPLP lesions usually resolve within a few months, although relapses may occur in 10 to 29% of patients [5].

Our patient adds to a very small literature base that describes the presentation and treatment of PPLP. This case is notable in that it may lead dermatologists to expand their differential diagnosis when treating palmoplantar lesions that seem clinically consistent with psoriasis but do not respond to typical psoriasis regimens. In addition we report that methotrexate is a medication to which PPLP may respond.

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