Lichen Planus Mimicking Recalcitrant Ulcerative Psoriasis: When Is It Time to Biopsy?

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Practice Points

- Hypertrophic lichen planus (LP) is a rare variant of LP that can mimic psoriasis.
- Hypertrophic LP also can overlap with hypertrophic lupus erythematosus; treatment regimens also overlap.
- Before escalating treatment, consider biopsying any scaly plaques that are not responding to standard therapy.

Hypertrophic lichen planus (LP), also known as LP verrucosus, is a rare disorder that presents as verrucous plaques, typically on the lower extremities and ankles. This variant differs from the common presentation of LP, which appears as flat, polygonal, pink-purple papules spread diffusely on the flexor wrists, trunk, shins, and dorsal aspects of the feet, frequently involving the oral mucosa. Clinically, hypertrophic LP can be confused with psoriasis and usually does not respond to therapy with biologics. We present a case of hypertrophic LP in a 42-year-old woman who had been treated extensively for psoriasis. Although the morphology and location of the hyperkeratotic plaques mimicked psoriasis, biopsy results exhibited characteristic features of hypertrophic LP, and the lesions responded to treatment with acitretin, clobetasol propionate ointment, hydroxychloroquine, and simple wound care. The hypertrophic variant of LP can be extremely challenging to differentiate from psoriasis. Physicians who treat patients with scaly plaques should think beyond psoriasis and consider the hypertrophic variant of LP as a potential diagnosis.

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Lichen planus (LP) is an idiopathic, T-cell mediated inflammatory disease of the skin, nails, hair, and mucous membranes that is characterized by flat-topped violaceous papules and plaques. Clinical variants of LP include annular, bullous, hypertrophic, linear, ulcerative, and lichen planopilaris. Hypertrophic LP lesions tend to be symmetric and chronic, manifesting as thick, pruritic, hyperkeratotic plaques that primarily are seen on the shins or dorsal aspects of the feet. Histologically, a dense bandlike lymphocytic infiltrate with destruction of the epidermal basal cell layer usually is observed.

Clinically, hypertrophic LP often is mistaken for psoriasis and usually does not respond to therapy with biologics. Topical corticosteroids are a mainstay in the management of LP; however, treatment with other modalities such as calcineurin inhibitors,

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retinoids, dapsone, hydroxychloroquine, and mycophenolate mofetil also has been successful.[1]

Case Report

A 42-year-old woman was diagnosed at an outside facility with plaque-type psoriasis approximately 20 years prior to presentation and had been treated with various topical agents (eg, corticosteroids, vitamin D analogues) with minimal to moderate success. At that facility she initially was started on etanercept due to increasing body surface area of involvement, but treatment was discontinued after 18 months due to lack of response. She then was switched to adalimumab, which also was discontinued after 9 months due to lack of improvement. The patient subsequently underwent 3 cycles (4 months) of ustekinumab but stopped taking the medication due to reported slurred speech and dizziness. Over the last 12 months while on adalimumab and then ustekinumab, the patient developed painful ulcerated plaques and fungating nodules on the upper and lower legs. A swab culture of the wounds grew *Pseudomonas aeruginosa* and *Candida albicans*; subsequently, she was treated with multiple drugs including ciprofloxacin, tobramycin, doxycycline, and fluconazole, none of which improved the leg wounds. Simultaneous to the antibiotics, she received treatment at a wound care center. After approximately 4 months of wound care, she presented to our clinic for refractory psoriasis.

On presentation, hyperkeratotic plaques were noted on the elbows (Figure 1), arms, trunk, and knees. The patient had painful, ulcerated, nearly confluent plaques on the lower legs (Figure 2), as well as scattered ulcerated nodules on the anterior and posterior thighs. There was no facial, scalp, or nail involvement.

The patient's medical history was remarkable for nonspecific arthritis, hypertension, migraine headaches, and meningitis (unspecified type) approximately 1 year prior to presentation. Current medications included lisinopril, migraine medication (eg, aspirin, butalbital, caffeine), and hydrocodone. She did not smoke, denied use of illicit drugs, and reported no recent travel. The patient also reported a history of recurrent oral ulcers; however, none were present on initial examination.

Laboratory studies were negative for hepatitis B, hepatitis C, human immunodeficiency virus, and rapid plasma reagin. The patient had an elevated rheumatoid factor of 458 IU/mL (reference range, <14 IU/mL), elevated antinuclear antibodies (ANAs)(speckled, 1:640; nucleolar, 1:160), positive SS-A and SS-B (Sjögren syndrome antibodies), and mildly elevated alanine aminotransferase. The patient was evaluated by the rheumatology department and was diagnosed with Sjögren syndrome; she was started on hydroxychloroquine 100 mg twice daily parallel to our workup. There were insufficient criteria to make a diagnosis of systemic lupus erythematous (LE), rheumatoid arthritis, or psoriatic arthritis.

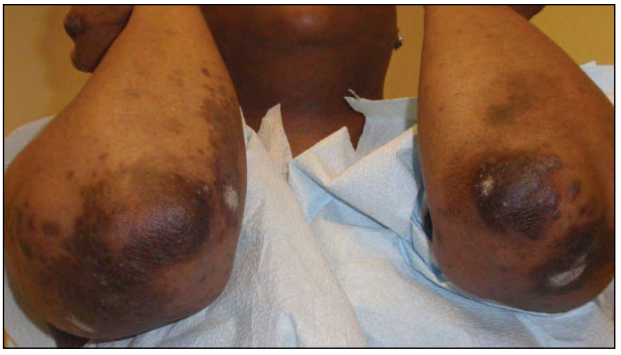

Figure 1. Hyperpigmented scaly plaques on the elbows.

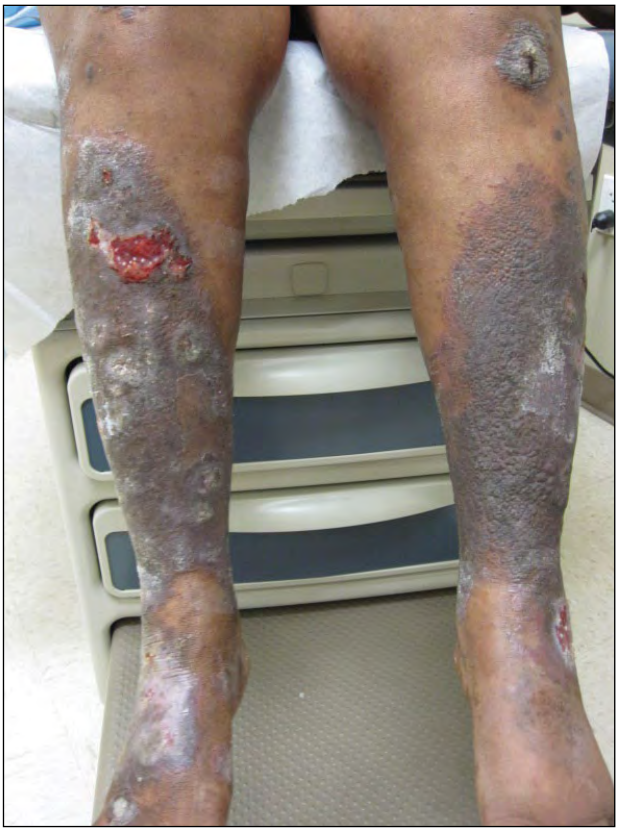

Figure 2. Scaly, hyperpigmented, erythematous plaques with focal ulceration on the lower legs.

Three 4-mm punch biopsies were taken from 3 sample lesions: the edge of an ulcerated plaque, an ulcerated nodule, and a hyperkeratotic plaque. Results of all 3 biopsies revealed a psoriasiform

lichenoid dermatitis with the presence of a bandlike infiltrate involving the upper dermis associated with epidermal hyperplasia (Figure 3). The biopsies from the 2 plaques showed a thickened cornified layer with compact orthokeratosis. Immunohistochemical stains were negative for cutaneous B-cell lymphoma or cutaneous T-cell lymphoma (CTCL), and T-cell receptor gene rearrangement studies were negative for evidence of a clonal T-cell proliferation. Spirochete staining also was negative. Based on the clinical findings, laboratory test results, and histology, the new differential diagnosis was ulcerating hypertrophic LP versus hypertrophic LE, as CTCL and psoriasis were eliminated. We started the patient on acitretin 25 mg daily at this point.

Given the patient's elevated ANA levels and Sjögren syndrome, hypertrophic LE seemed a reasonable diagnosis; however, after several months on acitretin, the patient revealed she had an outbreak of oral ulcers. On examination, white reticulated patches with shallow ulcerations were noted on the bilateral posterior buccal mucosa that were consistent with oral ulcerative LP. The patient declined oral biopsy. In light of the classic oral LP lesions, the diagnosis of hypertrophic LP was made.

Comment

Our patient represents a case of hypertrophic LP mimicking psoriasis. Hypertrophic LP usually presents with papules and symmetric plaques with a lichenified and highly hyperkeratotic surface and a predilection for the pretibial region.[2] Hypertrophic LP may be confused with psoriasis, which is characterized by well-demarcated plaques with loosely adherent, silvery white scales; however, psoriasis does not ulcerate or cause marked pain. Although some of the lesions in our patient had the right morphology and the right location for psoriasis, the progression of the lesions into nodules and ulcerations and the failure to respond to treatment were evidence against it.

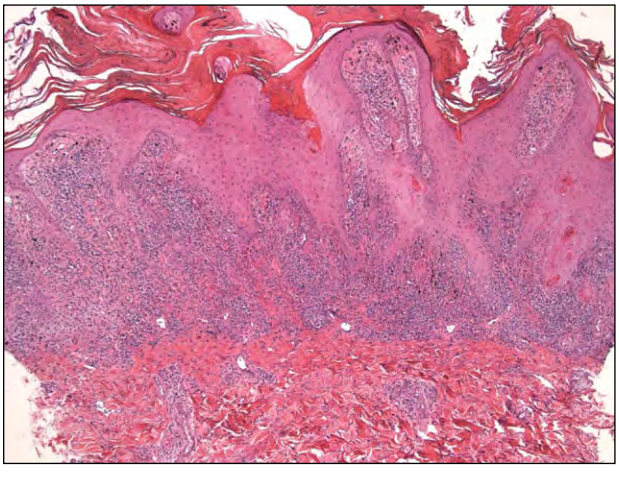

Figure 3. Marked irregular epidermal hyperplasia with hyperkeratosis and a dense bandlike infiltrate of lymphocytes. Scattered melanin-laden macrophages were seen, particularly at the base of the infiltrate (H&E, original magnification ×40).

Cutaneous T-cell lymphoma may manifest as persistent and progressive, pruritic, hyperpigmented patches or plaques that can progress to tumors. It is histologically characterized by the infiltration of small- to medium-sized lymphocytes with cerebriform nuclei in the upper dermis and epidermis.[3] The morphology of our patient's lesions fit with a suspicion for plaque- and tumor-stage CTCL, but histology did not support this diagnosis.

Rarely, the development of squamous cell carcinoma has been reported to arise in lesions of chronic LP.[4] In the setting of chronic wounds, the development of squamous cell carcinoma should be considered. However, our patient's lesions were widespread and rapidly evolved; a diagnosis of squamous cell carcinoma was not supported by histology.

Lichen planus classically has been considered a dermatosis without ANAs or other specific autoantibodies; however, ANAs are more frequently observed in patients with erosive LP,[5] which was the case in our patient. Lichen planus is associated with other diseases that have an autoimmune basis, such as diabetes mellitus, chronic persistent hepatitis, and Sjögren syndrome[6] that also was diagnosed in our patient. There have been cases of LP that demonstrate the clinical findings of LE, and some authors have suggested that ulcerative LP might overlap with unusual variants of LE.[7]

The etiology of hypertrophic LP is unknown; however, many studies support an immunologic pathogenesis.[8] For this reason, many of the treatments of hypertrophic LP overlap with psoriasis (eg, potent topical or intralesional corticosteroids, systemic retinoids, psoralen plus UVA phototherapy, cyclosporine).[1] Hydroxychloroquine also has been reported as an effective treatment of LP. Unfortunately, in the case of hypertrophic LP, larger randomized trials to support the effectiveness of most of these treatments are lacking, and many treatments are recommended based on anecdotal evidence.[9] After 1 year of titrating doses of hydroxychloroquine and acitretin, our patient continues to have some spontaneous ulcerations; however, lesions typically resolve fairly quickly with clobetasol propionate ointment and wound care with silver sulfadiazine cream, petroleum jelly, and nonstick dressings.

Combining her other clinical history with the biopsy proved to be the key to arriving at the correct

diagnosis in this patient. Scaly plaques on elbows and knees certainly suggest psoriasis to even the most inexperienced practitioner. However, progression to nodules and ulcers combined with the fact that her disease was refractory to escalating psoriasis treatments made us most concerned about CTCL. Although psoriasis frequently is a clinical diagnosis, anytime the question of CTCL is raised or one encounters a papulosquamous eruption not responding to standard treatments, do not hesitate to reach for a sharp instrument and specimen cup. Ultimately, CTCL was not her diagnosis either, but this case illustrates the importance of histology in determining a diagnosis and begs the point, do not forget one of our most basic tools as dermatologists: the biopsy.

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