Verrucous Plaques on Sun-Exposed Areas

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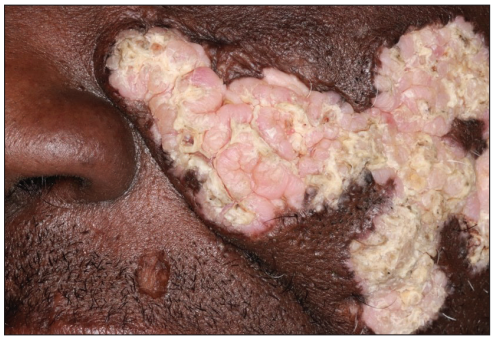

A 54-year-old man with no notable medical history presented to an outpatient dermatology clinic with multiple skin lesions on sun-exposed areas including the face, chest, scalp, and bilateral upper extremities. The patient reported that he had not seen a doctor for 26 years. He noted that the lesions had been present for many years but was unsure of the exact timeframe. Physical examination revealed verrucous plaques with a violaceous rim and central hypopigmentation on the chest, scalp, face, and arms. Scarring alopecia also was noted on the scalp with no associated pain or pruritus. Antinuclear antibody and extractable nuclear antigen tests were negative, and urine analysis was normal. A shave biopsy of the chest was performed for histopathologic evaluation. Rapid plasma reagin tests and HIV antibody tests also were performed.

WHAT'S YOUR **DIAGNOSIS?**

a. blastomycosis
b. hypertrophic lichen planus
c. hypertrophic lupus erythematosus
d. rupioid psoriasis
e. secondary syphilis

PLEASE TURN TO PAGE E14 FOR THE DIAGNOSIS

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THE **DIAGNOSIS:** Hypertrophic Lupus Erythematosus

The biopsy of the face collected at the initial appointment revealed interface dermatitis with epidermal hyperplasia with no parakeratosis or eosinophils (Figure 1). Microscopic findings were suggestive of hypertrophic lupus (HLE) or hypertrophic lichen planus. The rapid plasma reagin and HIV labs collected at the initial appointment were negative, and a review of systems was negative for systemic symptoms. Considering these results and the clinical distribution of the lesions primarily affecting sun-exposed areas of the upper body, a final diagnosis of HLE was made. The patient was counseled on the importance of photoprotection and was started on hydroxychloroquine.

Hypertrophic lupus erythematosus, a rare variant of chronic cutaneous lupus erythematosus (CCLE), typically manifests as verrucous plaques or nodules commonly found on sun-exposed areas of the body, as was observed in our patient on the face, scalp (Figues 2 and 3), chest, and upper extremities.[1] Lesions can have a variable appearance, from hyperkeratotic ulcers to depigmented plaques and keratoacanthomalike lesions.[2] On histopathology, HLE falls into the category of lichenoid interface dermatitis and commonly demonstrates hyperkeratosis, acanthosis, follicular plugging, superficial and deep infiltrate, and increased mucin deposition in the dermis.[3]

Although rare, it is critical to remain vigilant for the development of squamous cell carcinoma in patients with chronic untreated CCLE. Hypertrophic lupus erythematosus, specifically, is the most likely variant to give rise to invasive squamous cell carcinoma and can be more aggressive as a result of this malignant transformation.[3,4] Ruling out squamous cell carcinoma in the setting of HLE can be achieved by staining for CD123, as HLE commonly is associated with many CD123+ plasmacytoid dendritic cells adjacent to the epithelium, unlike squamous cell carcinoma.[3] Fortunately no evidence of invasive squamous cell carcinoma, including cellular atypia or increased mitotic figures, was seen on histology in our patient.

A thorough history and physical examination are essential for screening for HLE, as positive antinuclear antibodies are observed only in half of the patients diagnosed with CCLE.[5] Furthermore, antinuclear antibodies sometimes can be negative in patients with HLE who have end-stage organ involvement.

Hypertrophic lupus erythematosus can be challenging to treat. First-line therapies include antimalarials, topical steroids, and sun-protective measures. Intralesional triamcinolone injection also can be used as an adjunctive therapy to expedite the treatment response.[6] Evidence supports good response following treatment with acitretin or a combination of isotretinoin and hydroxychloroquine.[2] Another therapeutic strategy is implementing immunosuppressants such as methotrexate, mycophenolate mofetil, and azathioprine for persistent disease. Immunomodulators such as thalidomide historically have been shown to treat severe recalcitrant cases of HLE but typically are reserved for extreme cases due to adverse effects. Biologic agents such as intravenous immunoglobulins and rituximab have been shown to treat CCLE

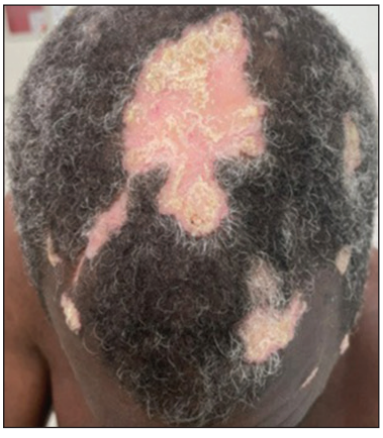

FIGURE 2. Scaly pink patches and plaques with associated scarring alopecia on the scalp.

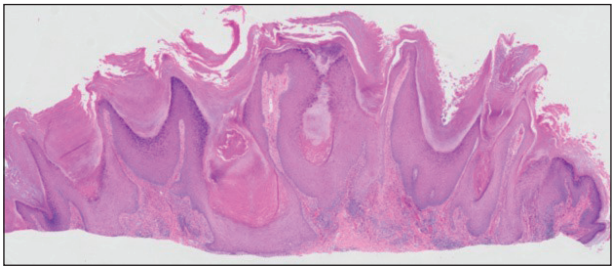

FIGURE 1. Hypertrophic glassy epithelium with follicular plugging and dense lichenoid interface dermatitis.

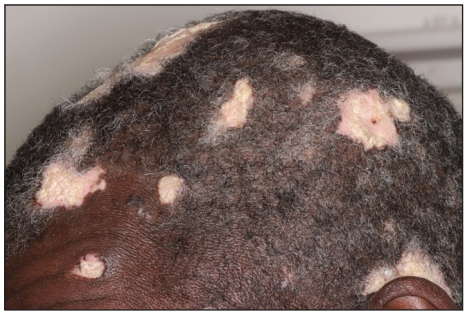

FIGURE 3. Hyperkeratotic pink plaques scattered on the scalp.

successfully, but routine use is limited due to high cost and lack of strong clinical trials.[7]

There have been reports of experimental therapies such as monoclonal antibodies (eg, anifrolumab and tocilizumab therapy) providing remission for patients with refractory CCLE, but information on their efficacy—specifically in patients with HLE—is lacking.[8] Chronic cutaneous lupus erythematosus and its variants require further investigation regarding which treatment options provide the greatest benefit while minimizing adverse effects.

It is important to distinguish HLE from other potential diagnoses. Features of HLE can mimic hypertrophic lichen planus; however, the latter typically appears on the legs while HLE appears more commonly on the upper extremities and face in a photodistributed pattern.[9] Since HLE has a lichenoid appearance histologically, it may appear clinically similar to hypertrophic lichen planus. Although not performed in our patient due to cost, direct immunofluorescence can aid in distinguishing HLE from hypertrophic lichen planus. Chronic cutaneous lupus erythematosus shows a granular pattern of deposition of IgM (primarily), IgG, IgA, and C3. In contrast, hypertrophic lichen planus exhibits cytoid bodies that stain positive for IgM as well as linear deposition of fibrinogen along the basement membrane.[3,10]

Blastomycosis also can lead to development of verrucous plaques in sun-exposed areas, but the lesions typically originate as pustules that ulcerate over time. Lesions also can manifest with central scarring and a heaped edge.[3] Unlike HLE, pseudoepitheliomatous hyperplasia with mixed infiltrate and intradermal pustules are seen in blastomycosis.[3] Fungal organisms often are seen on pathology and are relatively large and uniform in size and shape, are found within giant cells, and have a thick refractile asymmetrical wall.[11] In rupioid psoriasis, skin lesions mostly are widespread and are not limited to sun-exposed areas. Additionally, biopsies from active rupioid lesions typically show psoriasiform epidermal hyperplasia with parakeratosis with no interface inflammation—a key differentiator.[12] In secondary syphilis, chancres often are missed and are not reported by patients. Clinically, secondary syphilis often manifests as scaly patches and plaques with palmar involvement and positive rapid plasma reagin, which was negative in our patient.[13] Histologically, secondary syphilis can exhibit a vacuolar or lichenoid interface dermatitis; however, it typically exhibits slender acanthosis with long rete ridges and neutrophils in the stratum corneum.[3] Furthermore, plasma cells are present in about two-thirds of cases in the United States, with obliteration of the lumen of small vessels and perivascular histiocytes and lymphocytes with apparent cytoplasm commonly seen on pathology. Silver staining or immunostaining for *Treponema pallidum* may reveal the spirochetes that cause this condition.[3]

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