SKIN

SHORT COMMUNICATION

A Case of Tumid Lupus Erythematosus Highlighting the Importance of Clinicopathologic Correlation

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INTRODUCTION

A 44-year-old male presented with, edematous, erythematous plaques on the back and arms, present for over a decade (**Figure 1**). Focal atrophic scars were noted within several lesions, and the patient attributed these to prior biopsies performed in his home country (Russia) with "normal" results.

Initial punch biopsy of the back showed focal mucinosis with folliculitis. Two subsequent punch biopsies of the back, read as asteatotic dermatitis, showed sparse perivascular dermal infiltrates with small amounts of spongiosis and parakeratosis. Given the lack of consistency between clinical and pathologic findings, a final incisional biopsy of the right arm was obtained (**Figure 2**).

DISCUSSION

Tumid lupus erythematosus (TLE), a subtype of cutaneous lupus, is characterized by firm, edematous, erythematous plaques, predominantly on sun-exposed areas of the body (e.g., face, neck, upper trunk, and arms).[1] Histopathologic findings include a primarily lymphocytic perivascular and periadnexal infiltrate in the papillary and reticular dermis with interstitial mucin.[1] Histiocytes and neutrophils may also be present.[1] In contrast to subacute cutaneous and discoid lupus erythematosus (DLE), epidermal (e.g., follicular plugging, atrophy, loss of rete ridges) and dermoepidermal junction (e.g., vacuolar degeneration, basement membrane thickening) changes are uncommonly seen in TLE.[1]

Our initial clinical differential included lupus panniculitis, cutaneous sarcoidosis, and mycosis fungoides (MF). Unlike TLE, lupus panniculitis exhibits inflammatory changes, such as hyaline fat necrosis and lymphoid bodies, at the level of the subcutaneous fat.[2] Overlying features of DLE are possible, and the coexistence of DLE and lupus panniculitis is termed lupus profundus.[3] Given its varied morphologies, cutaneous sarcoidosis was included in the differential but ruled out by the absence of non-caseating epithelioid granulomas.[4] MF is characterized by atypical lymphocytes (sometimes with "cerebriform" nuclei), Pautrier microabscesses, and epidermotropism. However, these features are often absent in early stages of MF, which may mimic eczematous dermatitis.[5] Repeat biopsy was warranted, not only because lesions did not resemble asteatotic eczema

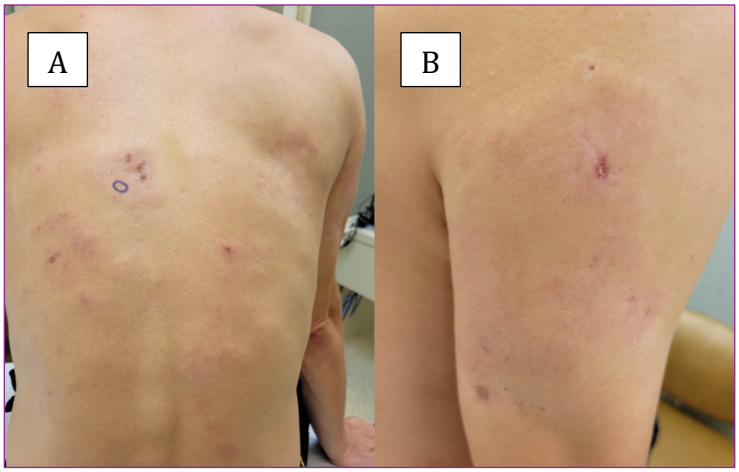

Figure 1. Erythematous plaques on the (**A**) back and (**B**) arms with focal scars.

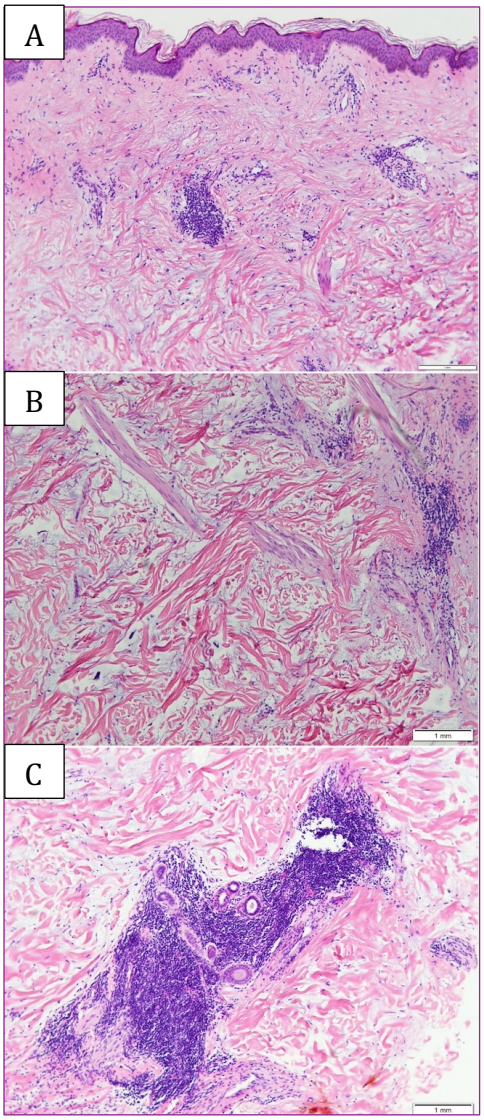

Figure 2. (**A**) Superficial and (**B**) deep perivascular and periadnexal dermal lymphocytic infiltrate with scattered mucin (**C**) between collagen bundles (hematoxylin-eosin x100).

but also, because mild spongiosis and follicular mucinosis may be seen in MF.[5]

CONCLUSION

Altogether, three punch and one incisional biopsy (not to mention multiple outside "normal" biopsies) were obtained to diagnose TLE, thus highlighting the importance of clinicopathologic correlation. Unlike DLE, TLE lacks surface change and is characteristically nonscarring, yet focal scarring was noted in several of the patient's plaques. Scarring as a result of concurrent DLE cannot be ruled out.

In addition to sun avoidance and photoprotection, first-line treatment of TLE consists of topical steroids and/or systemic antimalarials. Refractory cases have been successfully treated with methotrexate or systemic corticosteroids.[2] Our patient declined hydroxychloroquine and opted for topical clobetasol 0.05% ointment.

TLE is infrequently associated with systemic lupus erythematosus (SLE), and significant elevations in ANA and anti-dsDNA antibodies are rare.[2] Our patient's ANA titer was low-positive (1:80). Given accompanying joint pain, he was referred to rheumatology, where he was found to meet criteria for SLE with the following additional findings: anti-dsDNA (1:80,reference (ref) <1:20), anti-Sm/RNP (3.5,ref <1), anti-cardiolipin IgG (87.3 µg,ref <20.0), complements (C3 62 mg/dL,ref 82-193; C4 3 mg/dL,ref 15-57), and fever with negative infectious and malignancy workup to date.

Overall, biopsy was poorly sensitive for TLE as multiple biopsies were required to achieve a diagnosis. It is always important to correlate clinically, as some dermatologic conditions may have ambiguous or subtle pathologic findings.

Conflict of Interest Disclosures: None

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