JAMA Dermatology Clinicopathological Challenge

Erythematous Plaque on the Inferior Eyelid

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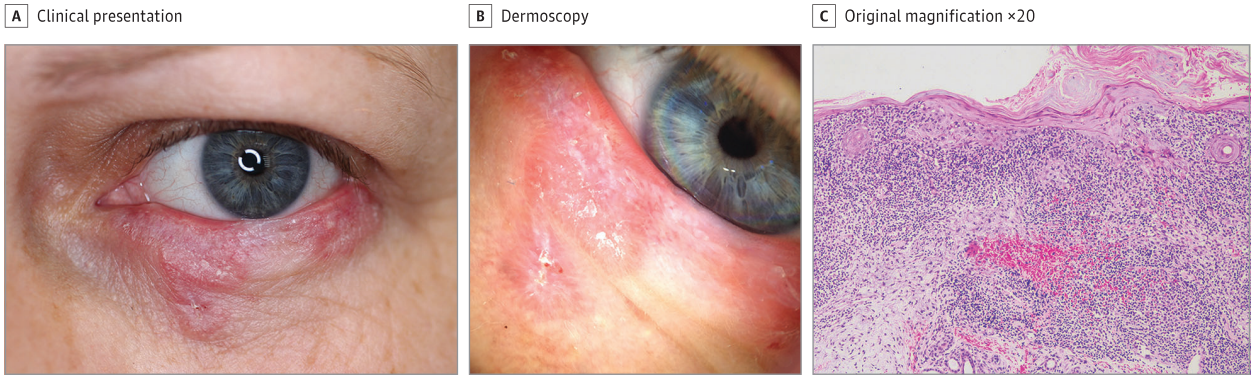

Figure 1. A, Erythematous scaling plaque involving the left lower eyelid. B, Dermoscopy shows erythematous background, telangiectatic vessels, structureless whitish areas, and white scales. C, Histopathological analysis (hematoxylin-eosin).

A 40-year-old healthy woman presented with an 8-year history of a slowly growing erythematous plaque on the left lower eyelid, with occasional discomfort. Physical examination revealed a single erythematous scaling plaque involving the left lower eyelid, including its border. Eyelashes were totally absent (**Figure 1**A). No other lesions were found after complete physical examination. Dermoscopy showed an erythematous background, telangiectatic vessels, whitish structureless areas, and whitish scales (Figure 1B). A 4-mm punch biopsy specimen was obtained (Figure 1C).

WHAT IS YOUR DIAGNOSIS?

- **A.** Sarcoidosis
- **B.** Discoid lupus erythematosus
- **C.** Basal cell carcinoma
- **D.** Bowen disease

Diagnosis

B. Discoid lupus erythematosus

Microscopic Findings and Clinical Course

Histopathologic examination demonstrated an atrophic epidermis with parakeratosis, vacuolar alteration of the basal layer, and dense inflammatory infiltrate both in papillary and reticulate dermis, consistent with discoid lupus erythematosus (DLE) (Figure 1C). Antinuclear antibodies and double-stranded DNA serologic testing were negative. The patient was treated with high-potency topical corticosteroid for 10 days, followed by tacrolimus, combined with oral hydroxychloroquine 400 mg daily. At the 2-month follow-up visit, lesions had completely resolved (**Figure 2**).

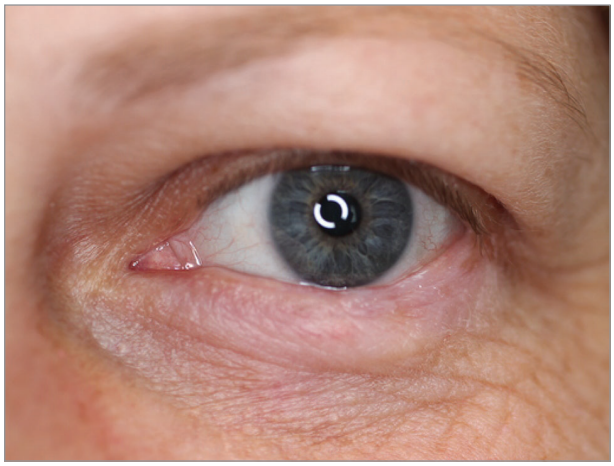

Figure 2. Two-month follow-up.

Discussion

Discoid lupus erythematosus is the most common clinical subtype of cutaneous lupus erythematosus. Clinically, it is characterized by erythematous and hyperchromic plaques with follicular plugging, which may lead to atrophy, pigmentary changes, and scarring. It primarily affects the ears, face, scalp, and neck.[1] Eyelid involvement occurs in only 6% of cases, and DLE solely affecting the eyelids is exceptionally rare.[2,3] Lesions involving areas below the neck, the disseminated form of DLE, occur in less than 20% of cases.[4] The risk of DLE progressing to systemic lupus erythematosus is relatively low. Nonetheless, widespread DLE lesions, arthralgia and/or arthritis, nail changes, anemia, leucopenia, high erythrocyte sedimentation rates, and high titers of antinuclear antibodies increase this risk.[5]

Dermoscopy has been described as an additional tool for the diagnosis of DLE, and its findings correlate with histopathology and disease duration. Perifollicular whitish halos and follicular plugs are seen in recently developed lesions, whereas structureless whitish areas, telangiectatic vessels, and honeycomb pigment network are findings of longer-standing DLE. These findings correlate with the scarring process seen in DLE, initially affecting the hair follicle with destruction of the perifollicular elastic sheath followed by dermal involvement. Fibrosis throughout the dermis, the end-stage of DLE lesions, corresponds to the whitish structureless areas on dermoscopy.[6]

Eyelid involvement as the only manifestation of DLE can mimic contact dermatitis, atopic dermatitis, seborrheic dermatitis, psoriasis, sarcoidosis, basal cell or squamous cell carcinoma, and actinic keratosis.[7,8] Whenever these conditions are suspected or unresponsiveness despite appropriate treatment, DLE should be considered as a potential differential diagnosis.[1,3] All efforts must attempt to diagnosis and treat eyelid DLE early to prevent permanent scarring with eyelash loss, entropion, ectropion, adhesion, trichiasis, and visual dysfunction.[9,10]

ARTICLE INFORMATION

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