Discoid lupus erythematosus of the eyelid

Sir,

Discoid lupus erythematosus (DLE) is the most common form of chronic cutaneous lupus erythematosus (CCLE). Discoid lesions often present as well-defined erythematous plaques showing thick adherent scale and follicular plugging. In old lesions, atrophic scarring with verrucous hyperkeratosis may be seen. DLE usually affects photoexposed skin, most often the face. Lower eyelid involvement occurs in 6% of patients: however, eyelid lesions are rarely the sole manifestation of DLE.[1,2]

A 56-year-old woman presented in June 2006 due to redness on the left lower eyelid persisting for two years. She had been treated for blepharitis and eczema with topical antibiotic and corticosteroid ointments. The patient is a grinder by profession and she stated that metal dust, with silica as the main component, injured her left eye on several occasions before the onset of eyelid lesion. The dermatological examination revealed an erythematous plaque with atrophy and adherent scales in the center, on the inner third of the left lower eyelid with madarosis [Figure 1]. There was a

pale erythema over the malar area. Inner eye structures were not affected. Biochemical blood and urine tests were within reference values. Immunoserological examinations showed a positive antinuclear antibody (titer 1:80) with a nucleolar pattern but a negative anti-ds DNA. Anti SS-A (Ro) and anti SS-B (La), C3, C4, IgG, IgM, IgA were within reference values or negative. The histopathological analysis of the skin lesion biopsy showed typical features of DLE: follicular hyperkeratosis, vacuolar degeneration of the basal cell layer, and focal lymphocytic infiltrate in dermis [Figure 2]. The treatment included chloroquine 250 mg/day, 1% hydrocortisone ointment and photoprotection topically. The discoid lesion on the eyelid completely resolved after four weeks. Chloroquine therapy was continued for another eight weeks. The patient was examined every six months and no lesions occurred for two years. In September 2008, a mild relapse occurred on the same site. The patient pointed out that during the summer of 2008, metal dust had got into her left eye on two occasions.

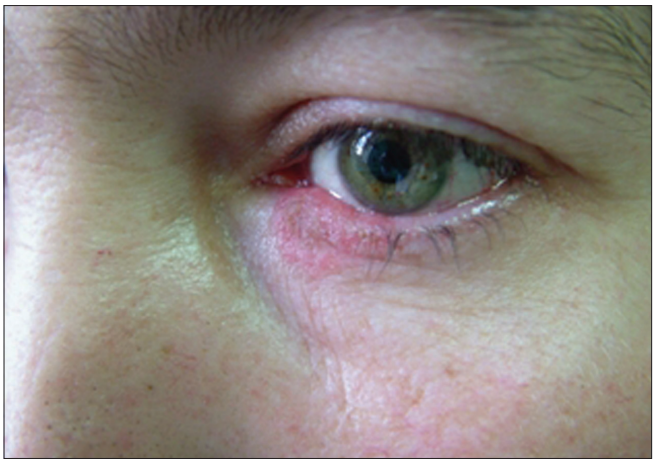

Figure 1: Atrophic erythematous plaque of the lower left eyelid and loss of eyelashes

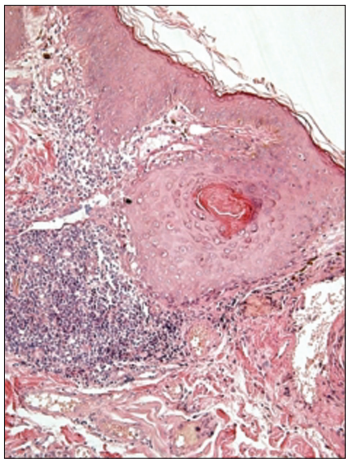

Figure 2: Excisional biopsy was performed and histopathological examination shows follicular hyperkeratosis, vacuolar degeneration of the basal cell layer, and focal lymphocytic infiltrate in the dermis (H&E, ×200)

Involvement of the eyelid only with DLE is uncommon and the diagnosis may be difficult. Donzis and co-workers reported an average delay of two years before the correct diagnosis.[3] Patients with these symptoms have usually been treated for presumed chronic blepharitis or eczema. DLE may be associated with other eyelid diseases, which makes the timely recognition of lupus lesions more difficult. Trindade *et al*. presented the association of DLE and contact dermatitis, which delayed the diagnosis for a period of 10 years, whereas Ena and co-workers reported DLE associated with staphylococcal blepharitis and Meibomian gland dysfunction.[2,4]

The described case represents a typical DLE manifestation with erythema, atrophy, adherent scales and loss of eyelashes. Atypical DLE presentation of the eyelid in the form of madarosis without previous erythema and edema has also been described.[1] Since asymmetrical blepharitis or madarosis raises the suspicion of sebaceous cell carcinoma, full-thickness eyelid biopsy may be considered.[2]

The etiology of cutaneous lupus erythematosus is still unclear. It is suspected that beside ultraviolet (UV) irradiation many exogenous agents like medications, pesticides and insecticides, heavy metals, silica dust and infections may induce lupus-like syndrome in genetically susceptible persons.[5] The interesting aspect of this case is the appearance of DLE eyelid lesion during summertime in association with repeat eye trauma by metal dust. The eyelid DLE is often exacerbated by trauma or sunlight.[1] Heavy metals have been associated with polyclonal activation of the immune system and production of autoantibodies in animals but human evidence is limited to sparse case reports such as one concerning molibden contained in cervical metal plates.[5] Thus, an environmental agent present in metal dust and repeat eye injuries coupled with exposure to UV light may have induced apoptosis of keratinocytes, autoantigen translocation and the release of proinflammatory cytokines and likely triggered the onset of DLE in our patient.

Eyelid DLE should be differentiated from Meibomian

gland dysfunction, sebaceous and basal cell carcinoma, lymphoma, psoriasis, seborrhoeic dermatitis, rosacea, sarcoidosis and contact dermatitis.[2] Early recognition and treatment of the eyelid DLE is important in order to avoid eyelid dysfunction resulting from scarring, synechiae, trichiasis, entropion and ectropion.[1] Furthermore, classic DLE patients in whom eyelids are the only manifestation of disease need to be observed over a long period in order to identify recurrence and to institute treatment.[4] Also, patient should be examined periodically for systemic involvement.

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