Case Report

Discoid Lupus Erythematosus affecting the Eyelid with Dramatic Response to Chloroquine and Intralesional Triamcinolone

Khalid Mahmood
Consultant Dermatologist, PAF Hospital Islamabad, Pakistan.

Summary: The discoid lupus erythematosus (DLE) may present with a wide range of cutaneous lesions. However, the eyelid involvement has been only rarely reported. The involvement of eyelids may lead to potentially serious complications secondary to the scarring like ectropion, entropion, symblepharon and loss of eyelashes. It is therefore important to diagnose and appropriately treat the eyelid lesions of DLE at an early stage. A case of DLE involving the eyelid which responded dramatically well to chloroquine and intralesional triamcinolone, is being reported.

Key Words: Eyelid, Discoid lupus erythematosus, chloroquine, triamcinolone.

Case Report
A 32 year old woman had erythematous annular plaques on both her cheeks, lip, and extensor aspect of the forearms for two years. The lesions showed partial scarring and hyperpigmentation. She developed a semi annular plaque on her left lower eyelid nearly two months prior to her consultation at our hospital. This lesion had a raised erythematous border and central depressed, pigmented and partially scarred area. The eyelid margin was inflamed with loss of eyelashes from the affected part of the eyelid (Fig 1). The adjacent part of the palpebral conjunctiva was inflamed as well (Fig 2). She had mild conjunctival irritation. She did not complain of dryness of the affected eye. Fortunately, there was no deformation of the eyelid margin, contracture, ectropion, entropion or symblepharon. She had a kissing lesion affecting the central portion of both lips. There were whitish plaques on her hard palate. There were neither any active lesions nor scarring alopecia on scalp. The covered skin was spared. Her general health was fine and there was no history of joint pain, fever, malaise or oral ulcers. Her family history was unremarkable. The systemic examination revealed no abnormality.

Fig 1: A Classical Plaque of DLE affecting the Lower Eyelid.

Her blood complete picture, urine examination, liver function tests urea and creatinine were within normal limits. The antinuclear factor and anti double stranded DNA antibodies were negative. A skin biopsy revealed hyperkeratosis, follicular...
plugging, basal vacuolar change and pigmentary incontinence. There was lymphocytic inflammatory infiltrate around the pilosebaceous unit, sweat glands and blood vessels. The blood vessels showed endothelial swelling and extravasation of red blood cells. There was significant damage to the adnexae and sebaceous glands were lost (Fig 3&4). The amount of mucin was increased in the dermis.

**Fig 2:** Inflammed Palpebral Conjunctiva of the Lower Lid. The Hair Loss is also Visible.

**Fig 3:** There is Hyperkeratosis and Periadenexal and Perivascular Lymphocytic Inflammatory Infiltrate (Hematoxylin & Eosin x 10).
A diagnosis of discoid lupus erythematosus was made based upon the clinical picture and histological features. She was started on chloroquine 250 mg twice daily. She was prescribed a potent topical steroid for twice daily application on her skin lesions. However, anticipating the potentially serious complications of eyelid lesion, 2 mg of triamcinolone was injected intralesionally in this lesion once. The response was dramatic. All the lesions healed in eight weeks time. The eyelid lesion healed without appreciable scarring with good re-growth of eyelashes. However, the skin lesions which were not injected with triamcinolone healed with significant scarring (Fig 5).
Discussion

The spectrum of cutaneous lesions and systemic involvement in lupus erythematosus is highly variable and wide ranged. Three major clinical variants of lupus erythematosus include discoid lupus erythematosus, subacute lupus and systemic lupus erythematosus. The classical lesions of Discoid Lupus Erythematosus present as erythematous, slightly scaly, well demarcated lesions on sun exposed areas of the body. The lesions heal by scarring. The adnexal structures, particularly hair, are lost in the process. Other common manifestations of DLE include hypertrophic lesions, annular lesions and papulonodular lesions.

The eyelid lesions have been rarely reported with DLE and just over twenty cases have been reported in the world literature to date. However, in one study, Burge et al, have reported 6% incidence of eyelid involvement in chronic cutaneous lupus erythematosus. The eyelid involvement may be in the form of classical plaques of DLE as in our case, conjunctival erythema and hypertrophy, dry eyes, periorbital erythema and swelling. If the patients present at a late stage, they may well have complications due to scarring like distortion of lid margin, permanent loss of eyelashes, ectropion, entropion and rarely symblepharon. The conjunctival direct immunofluorescence may provide a helpful clue in monitoring disease activity because there are linear immune deposits in uninvolved bulbar conjunctiva in 48% cases compared to very rare positive immunofluorescence in the uninvolved skin. About 5-10% case of DLE may progress to systemic lupus erythematosus. There are few case reports which suggest the eye involvement as a useful marker for progression to systemic disease.

Considering the potentially serious complications resulting from the scarring of the eyelids and conjunctiva due to DLE lesions, it is extremely important to start the appropriate treatment as early as possible. Chloroquine appears to be the most effective treatment option. However, the optimum anti-inflammatory effect of chloroquine does not start until about six weeks after the start of the treatment. By this time significant scarring may have already set in. In order to minimize the scarring, we feel that intrallesional triamcinolone is highly effective. Its anti-inflammatory action starts immediately and the formation of scar tissue is minimized.

References