Destructive Eyelid Lesions in Sarcoidosis

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Purpose: To report the clinical and histopathologic findings of a patient with sarcoidosis causing bilateral destruction of the lower eyelids.

Methods: Case report.

Results: Bilateral destructive lower eyelid lesions and cicatricial entropion developed in a 43-year-old man with systemic sarcoidosis. Histopathology was consistent with sarcoid granulomas. Disease progression was arrested with systemic prednisone and methotrexate before eyelid reconstruction was performed.

Conclusions: Sarcoidosis very rarely can cause destruction of full-thickness eyelid architecture. Active inflammation should be controlled before reconstruction.

Sarcoidosis is a multisystem disorder of unknown cause characterized by noncaseating inflammatory granulomas. Ocular involvement occurs in 25% to 50% of persons affected with sarcoidosis.1,2 Although skin may be involved in up to 22%3 of cases, eyelid skin involvement is very rare. There have been only ten previously reported cases, mostly in the form of skin nodules.

To our knowledge, this is the first reported case of full-thickness destruction and necrosis involving both lower eyelids resulting from sarcoidosis.

CASE REPORT

A 43-year-old black man was examined in July 1998 with a 2.5-year history of intermittent redness, swelling, itching, and irritation of both eyes. After a 5-year history of an unknown inflammatory condition, he was diagnosed with sarcoidosis in May 1998 after a biopsy of throat and nasal polyps. The patient also had erythematous maculopapular lesions of the skin over the bridge of the nose, chin, and neck. There were several 2- to 3-cm subcutaneous nodules on his left upper arm and both legs. A chest radiograph demonstrated hilar adenopathy consistent with pulmonary sarcoidosis. The remainder of the physical examination and medical history was noncontributory. Previous blood tests included an erythrocyte sedimentation rate of 15 mm and an elevated serum angiotensin-converting enzyme of 102 U/L.

The pertinent findings on ophthalmic examination were marked conjunctival hyperemia, left greater than right, with an apparent inflammatory nodule (Fig. 1) arising from bulbar conjunctiva adjacent to the limbus in the left eye. There was bilateral extensive destruction of lower eyelid architecture with indurated thickening, madarosis, and obliteration of the eyelid margin (Fig. 2), and marked fornical foreshortening and symblepharon formation. The lateral 60% of the right lower eyelid showed full-thickness inflammation and destruction of normal landmarks. Slit-lamp and fundus examinations showed no evidence of anterior or posterior segment inflammation.

Biopsies of the eyelid, conjunctiva, and facial skin nodules all revealed noncaseating granulomatous inflammation consistent with sarcoidosis. The histopathology of the eyelid showed unremarkable
epithelium with focal granulomas consisting of epithelioid histiocytes, few multinucleated giant cells, and a peripheral rim of lymphocytes and plasma cells in the subepithelial connective tissue (Fig. 3). Acid fast bacilli and fungal stains were negative.

Prednisone had been prescribed by the patient’s internist to control his systemic sarcoidosis coincident with his first visit to our office in July 1998. He was lost to our follow-up for 6 months, during which time methotrexate had been added and the prednisone dosage had been reduced. Reevaluation at that time revealed the destructive inflammatory process involving the eyelids to have resolved dramatically; the ulcerated areas had reepithelialized and the indurated thickening of the eyelid margin had softened. Full-thickness loss of the eyelid over the lateral 60% of the right lower eyelid was apparent (Fig. 4), and the left lower eyelid had extensive symblepharon formation and cicatricial entropion with trichiasis.

Reconstruction of the left inferior fornix with a mucous membrane graft and repositioning of the entropic left lower eyelid was performed. The right lower eyelid was reconstructed with a free tarsal graft from the left upper eyelid and transpositional skin and muscle flap from the right upper eyelid for replacement of the anterior lamella (Fig. 5). This resulted in a satisfactory outcome with resolution of the trichiasis and restoration of eyelid architecture. At the last visit 2 months postoperatively, the patient still was taking methotrexate for his systemic sarcoidosis, and there was no recurrence of ocular sarcoidosis.
Ocular involvement has been reported to occur in 25% to 50% of patients with sarcoidosis. This wide variation is because of the patient population studied, definitions of ophthalmic involvement, and the nature of evaluations conducted. Sarcoidosis can affect the eye as anterior or posterior uveitis, secondary glaucoma, cataracts, lacrimal gland involvement, conjunctival involvement, band keratopathy, scleral plaque, optic nerve involvement, orbital involvement, and eyelid nodules. Cutaneous sarcoidosis has been reported in 12% to 27% of patients with systemic disease, but eyelid involvement is very rare. Descriptions of the eyelid lesions include “millet seed” nodules, papular eruptions, larger nodules, and rarely, lupus pernio plaques or swollen eyelids.

The skin lesions in sarcoidosis characteristically manifest as elevated nodules or papules that only very rarely ulcerate or become necrotic. Most prior reported cases of ulcerative cutaneous sarcoidosis involved the lower extremities. There has been one report of ulcerative nodules and plaques affecting the face and eyelids. The case reported herein is the first to develop destructive sarcoidosis affecting the anterior and posterior lamella of the eyelid leading to full-thickness loss and cicatricial entropion.

Eyelid involvement in sarcoidosis may be treated with systemic or intralesional corticosteroids, immunosuppressants, or antimalarials during the active phase. Once the active inflammation has been controlled, surgical reconstruction may be successfully performed.

REFERENCES