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Conjunctival Lichen Planus in a Patient With Herpes Simplex Virus Keratitis

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Abstract

**Purpose**—To report a case of lichen planus in a patient with a history of herpes simplex virus keratitis.

**Methods**—Case report.

**Results**—A 60-year-old woman with chronic conjunctivitis and a history of herpes simplex virus keratitis was evaluated for irritation and a plaque on her right upper and lower eyelid palpebral conjunctivae. A surgical excision showed acanthosis and an underlying lichenoid infiltrate with a thickened basement membrane.

**Conclusion**—Lichen planus of the conjunctiva can be present in the absence of cicatrization in a patient with chronic irritation of the conjunctiva.

Keywords

conjunctiva; lichen planus; herpes simplex keratitis

Lichen planus is a chronic inflammatory disease that can affect both the skin and mucosa. Although more commonly affecting the oral and genital mucosa, it can also affect the conjunctiva, causing cicatrization in rare cases. It has been reported previously to be associated with hepatitis C infection, paraneoplastic conditions,¹ systemic lupus erythematosus, Sjogren syndrome,² and ocular cicatricial pemphigoid.³ It can be present in the mucosa as a reticulated network of white streaks or an erosive plaque-like process with multiple, recurrent, discrete lesions.⁴ We present a case of conjunctival lichen planus in a patient with a history of herpes simplex virus keratitis without cicatrization.

CASE REPORT

A 60-year-old white woman had a history of a conjunctival plaque that had developed on her right upper and lower eyelids in July 2005. She had an episode of herpes simplex keratitis in 1998. Before referral, she felt that her right eyelid was “enlarged” and she experienced increased irritation and mucous discharge. She had been treated with topical artificial tears and loteprednol 0.2%.

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Her medical history was significant for irritable bowel syndrome, seasonal allergies, and hypertension. Her medication list included acyclovir, estrogen, nifedipine, cetirizine, and mometasone nasal spray. Her best-corrected visual acuity upon presentation was 20/40 OD and 20/20 OS. Intraocular pressures were 18 and 16 mm Hg, right and left eyes, respectively. External examination showed keratin-like white plaques on her right upper (Fig. 1) and lower eyelid conjunctivae with bilateral dermatochalasis and right-sided ptosis. The right cornea had mild anterior stromal scarring in the visual axis with overlying punctate epithelial keratopathy. The remainder of her anterior and posterior segment examination was unremarkable with the exception of a mild nuclear sclerotic cataract. There were no symblepharon or ankyloblepharon. A biopsy of the right lower eyelid showed squamous metaplasia of the conjunctiva with a prominent granular cell layer and stratum corneum. There were focal areas of acanthosis and an underlying chronic inflammatory infiltrate composed of lymphocytes and histiocytes. The epithelial basement membrane was also thickened (Figs. 2A, B). A portion of the biopsy specimen was sent for direct immunofluorescence, which showed extensive fibrin deposition and no C3, immunoglobulin (Ig) G, IgM, or IgA staining that would indicate ocular cicatricial pemphigoid (Fig. 2C). A diagnosis of conjunctival lichen planus was made.

She was continued on the loteprednol twice daily, topical cyclosporine 0.05% 4 times a day, and artificial tears as needed. A dermatology consult revealed no cutaneous or oral mucosal involvement. Two months after starting topical cyclosporine, the patient’s symptoms were improved. Within 7 months of the initiation of therapy, the patient had complete resolution of her upper eyelid plaques (Fig. 3). However, after stopping topical cyclosporine for several months, her symptoms worsened. Approximately 1 year later, the plaques returned in her upper and lower eyelids. Topical cyclosporine therapy was restarted. Her symptoms again improved; however, the plaques did not completely resolve. Over the next 6 months, she developed trichiasis and dystichiasis requiring regular epilation. Two years after her initial presentation, the patient continues to be treated with topical cyclosporine and lubrication. Although her symptoms are well controlled, she continues to have keratinized plaques with early a symblepharon.

**COMMENT**

Lichen planus most commonly affects the skin, oral mucosa, and genital mucosa. Conjunctival involvement has rarely been reported, and in all of those cases, it has been associated with cicatization. The pathogenesis of lichen planus is unknown but likely results from a T cell–mediated auto-immune response to altered self-antigens on keratinocytes, leading to chronic inflammation and damage of the affected tissues. In other reported cases of conjunctival lichen planus, cicatization had already developed after long-term irritation. Cicatization is the end result of long-term conjunctival irritation whether because of an autoimmune process or chronic inflammation. Immunosuppressive agents are the mainstay of therapy. Limited involvement is treated with topical steroids or cyclosporine alone or in combination. In more severe cases, with diffuse involvement, systemic therapy may be warranted. Cyclosporine is a calcineurin inhibitor that blocks the production of interleukin-2. This decreases the activation and proliferation of helper T (CD4) and cytotoxic T cells (CD8), thereby decreasing the inflammatory damage caused in lichen planus. Our patient responded well to topical cyclosporine and corticosteroids and only flared when she discontinued the topical cyclosporine. Restarting her therapy allowed most symptoms to abate, but further scarring occurred. This case illustrates that early identification by clinical examination and histopathology, along with aggressive therapy, can prevent or delay cicatization in cases of conjunctival lichen planus.
Acknowledgments

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References

FIGURE 1.
There is a white plaque present on the tarsal conjunctiva of the right upper eyelid.
FIGURE 2.
A. There is a well-circumscribed area of acanthotic epithelium present corresponding to the lesions shown in Figure 1 (hematoxylin and eosin, ×25). B. Higher magnification shows a prominent granular cell layer and stratum corneum with a thickened basement membrane of the basilar epithelium and an underlying focal chronic inflammatory cell infiltrate (hematoxylin and eosin, ×100). C. Direct immunofluorescence shows fibrin deposition in thickened basement membrane area (fluorescent-labeled anti-fibrin, ×100).
FIGURE 3.
The right, upper eyelid, palpebral, conjunctival plaque has resolved after treatment.