topical caspofungin was progressively decreased over 3 further weeks. Following the completion of treatment, complete healing of the corneal epithelium and resolution of the corneal infiltrate were observed, although the corneal opacity persisted (Figure, D). There was no evidence of ocular toxic effects and no recurrence of fungal keratitis over a follow-up period of 6 months.

Comment. C. albicans is the most frequent cause of fungal keratitis in temperate regions and is an opportunistic organism that can compromise chronic keratopathy and corneal grafting. Persistent epithelial defects and suture-related problems, along with immunosuppression, have been found to be the major predisposing risk factors.

Caspofungin is a first-in-class echinocandin with potent activity against Candida and Aspergillus, the dominant human fungal pathogens. In contrast to all other antifungal drugs, echinocandins have a selective action on a target present only in fungal cell walls (not in mammalian cells); they inhibit the synthesis of an essential component, (1,3)-β-glucan. Caspofungin is fungicidal in vivo and in vitro against all Candida species, including fluconazole-resistant strains. Its activity differs from that of the azole antifungal group, which is fungistatic. In our case, we think that voriconazole stopped progression of the infiltrate but did not kill the microorganism. The presence of the fungus after 1 month could be due to poor drug penetration, fungal resistance, or both. We therefore suggest that an ideal treatment protocol should include antifungal agents chosen on the basis of in vitro and in vivo monitoring of fungal filament or yeasts.

To our knowledge, the topical ocular use of caspofungin has been reported in rabbits. There has been only 1 report of its use in humans, although it was in association with other antifungal drugs.

In conclusion, topical caspofungin, 0.5%, is a new, promising option in the treatment of refractory fungal-related corneal ulcers with no evidence of ocular toxic effects. However, future studies with larger samples are indicated to further evaluate its efficacy and tolerance.

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observed in the lacrimal gland (Figure 2B). In situ hybridization for immunoglobulin light chains confirmed polytypic infiltrates. Abundant IgG4-positive plasma cells were discovered, with an average of 99 per 4 high-power fields in the submandibular gland (Figure 2C) and 116 in the lacrimal gland (Figure 2D).

Comment. Unilateral, sclerosing, follicular, lymphoid hyperplasia of the submandibular gland (Küttner tumor) is an immunophenotypically polyclonal process with a favorable prognosis. It is typically unassociated with systemic disease and rarely encountered with dacryoadenitis.1-3 The submandibular and lacrimal diseases can be...
linked through the recently characterized spectrum of IgG4-positive sclerosing inflammatory diseases, which we documented in the biopsies of our patient’s 2 lesions. Concomitant serological elevation of IgG4 levels may develop; it was not evaluated in our patient but should probably be sought in orbital pseudotumors. Other IgG4-related diseases include autoimmune pancreatitis, sclerosing orbital inflammatory disease, Riedel struma, and retroperitoneal fibrosis. IgG4-positive disease is highly sensitive to corticosteroid therapy. The role of IgG4-positive plasma cells in fibrosclerosis is unknown.

In SS with dry eye and mouth symptoms, the lacrimal and parotid glands are usually bilaterally enlarged. Primary SS (formerly termed Mikulicz syndrome) lacks a systemic cause, while secondary SS is associated with underlying autoimmune disease and sometimes lymphoma. Serum antibodies against SS type A (Ro) and SS type B (La) antigens and lymphoepithelial lesions (also called epimyoepithelial islands in the parotid gland but rarely seen in the lacrimal gland) are absent in idiopathic dacryoadenitis and chronic sclerosing sialadenitis in a patient also lacking aspect of the solution to disinfect at point of contact.

Figure 2. Immunohistochemical studies (immunoperoxidase method, diaminobenzidine chromogen). A, CD3+ T lymphocytes in the submandibular gland infiltrate. CD20+ B lymphocytes were slightly less represented (not shown) (original magnification ×200). B, CD20+ B lymphocytes in the lacrimal gland infiltrate, which mildly outnumbered T lymphocytes (not shown) (original magnification ×200). C, IgG4-positive plasma cells with cytoplasmic immunostaining are prominently represented in the submandibular gland infiltrate (original magnification ×200). D, IgG4-positive plasma cells in the lacrimal gland (original magnification ×400).

Contact Lens Solutions Revisited

The Fusarium keratitis outbreak of 2004 and 2006 associated with ReNu with MoistureLoc (Bausch & Lomb, Rochester, New York) was reportedly due to failure of the solution to disinfect at point of contact. Cohen cites an article by Bausch & Lomb investigators Levy et al in which they simulated the reuse and evaporation of contact lens solutions, concluding that both situations diminished the effectiveness of ReNu with MoistureLoc against Fusarium, as well as reports that Fusarium survives on drying films of contact lens solutions. While these factors may have contributed to the vulnerability of ReNu with MoistureLoc with regard to Fusarium infection, none would explain an often overlooked aspect of the outbreak; all of the original cases were related only to the ReNu with MoistureLoc produced in Bausch & Lomb’s Greenville, South Carolina, plant (as opposed to their other 3 worldwide manufac-

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COMMENT AND OPINIONS

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