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1. Vernal keratoconjunctivitis with shield ulcers

Vernal keratoconjunctivitis (VKC) is a bilateral, seasonal, external ocular inflammatory disease of unknown cause. Vernal keratoconjunctivitis primarily affects young boys in their first decade of life.¹ Some of the characteristics of VKC include bilateral giant papillae on the tarsal conjunctiva of the upper eyelids, photophobia, mucous discharge, keratopathy, and itching.^{2,3} Patients might present with intense itching, sensitivity to light, and "ropy" discharge from the eye. Patients with VKC frequently have a family or medical history of atopic diseases, such as asthma, rhinitis, and eczema.

Although VKC is self-limiting, it can cause severe corneal complications. Shield ulcer (or *Togby ulcer*) is a serious vision-compromising complication of VKC. It occurs in approximately 3% to 11% of cases.¹ Shield ulcers form when superficial punctate keratitis-associated VKC leads to a break in the corneal epithelium. Mechanical and toxin hypotheses have been proposed to explain the pathogenesis of VKC-associated shield ulcers. The mechanical hypothesis suggests that the giant papillae on the upper tarsal conjunctiva are responsible for corneal abrasion, and this explains the more common superior shield ulcers.⁴ The toxin hypothesis suggests that eosinophil granule major basic protein found in the inflammatory debris covering VKC shield ulcers is cytotoxic and inhibits wound healing of the corneal epithelium.⁵ This might explain ulcer re-epithelialization after the inflammatory debris is removed.⁶

In addition to scarring and vascularization, patients with shield ulcers are at an increased risk of sterile ulceration, amblyopia, bacterial keratitis, and fungal keratitis.⁶⁻⁹ Although VKC is a bilateral disease process, it is not very

common to encounter simultaneous shield ulcers in both eyes.

Differential diagnosis

Atopic keratoconjunctivitis. Atopic keratoconjunctivitis is the most common differential diagnosis of VKC. Atopic keratoconjunctivitis is common among individuals with atopic dermatitis and it is associated with eczema and asthma. It is more prevalent in men than in women aged 30 to 50 years. Atopic keratoconjunctivitis might lead to reduced vision or blindness from corneal complications, such as chronic superficial punctate keratitis, persistent epithelial defects, corneal scarring or thinning, keratoconus, cataracts, and symblepharon formation.

Fibrosing conjunctivitis. Fibrosing conjunctivitis is encountered in patients with chronic conjunctivitis. Features such as foreshortening of the conjunctival fornices and symblephara are clearly visible, especially in long-standing disease. Slit lamp characteristics include the presence of fine, stellar, or white linear lines on the palpebral conjunctiva. Fibrosing conjunctivitis has been associated with trachoma, Stevens-Johnson syndrome, and toxic epidermal necrosis.

Discussion

It has been suggested that shield ulcers increase the risk of bacterial keratitis⁷ and amblyopia.⁶ Both medical¹⁰⁻¹² and surgical¹³⁻¹⁶ management have been successful in treating shield ulcers. In general, medical therapy is the first-line treatment of VKC-associated shield ulcers. These treatments include topical antihistamines, mast cell stabilizers, topical corticosteroids, or artificial tears. Topical antihistamines have been shown to reduce the signs and symptoms of

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ocular allergy.¹⁷ Mast cell stabilizers (such as cromolyn sodium, nedocromil sodium, pemirolast potassium, and lodoxamide tromethamine) are thought to prevent degranulation and release of vasoactive substances by limiting the flux of calcium across the mast cell membrane. Corticosteroids are thought to limit the signs and symptoms associated with ocular allergy by stabilizing capillary permeability, decreasing the influx of inflammatory cells, and inhibiting the activation and degranulation of inflammatory cells.¹⁸ Artificial tears could dilute allergens and mediators in the tears.¹⁹

In the case presented, medical treatment was commenced with topical loteprednol (0.2%) eye drops 4 times a day, levofloxacin (0.5%) eye drops 4 times a day, and preservative-free artificial tears 6 times a day in both eyes. Over the next 4 weeks the patient responded well to the medical treatment. The shield ulcers in both eyes had healed completely, leaving mild corneal haze away from the visual axes. The topical corticosteroids were tapered off and the patient was prescribed olopatadine (0.1%) eye drops twice per day and preservative-free artificial tears 6 times per day. Visual acuity was 20/20 at the last follow-up.

Surgical treatment is usually reserved for cases not responding to medical management. Surgical treatment could include surgical debridement, amniotic membrane transplantation, superficial keratectomy, or excimer laser phototherapeutic keratectomy with or without bandage contact lens.^{16,20,21} The purpose of surgical debridement is to remove the inflammatory material at the base of the ulcer, which consists of cationic proteins secreted from activated eosinophils.²² The inflammatory material is cytotoxic and prevents the wound from healing.²² The amniotic membrane has anti-inflammatory and antiscarring properties and it is found to enhance epithelialization.²³

Conclusion

In a previous report by Sridhar et al,¹⁶ shield ulcers were bilateral in 3 of 4 cases of VKC. These cases did not respond to medical therapy and ultimately required amniotic membrane transplantation. In our case, bilateral shield ulcers were mild; this might be attributable to early presentation or mild grade of the disease. Both shield ulcers re-epithelialized rapidly with a standard combined therapy of topical corticosteroids, antibiotics, and artificial tears. The possibility of bilateral shield ulcers should be suspected in cases of VKC. Timely

management with topical corticosteroids and artificial tears can result in a good outcome.

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Competing interests

None declared

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