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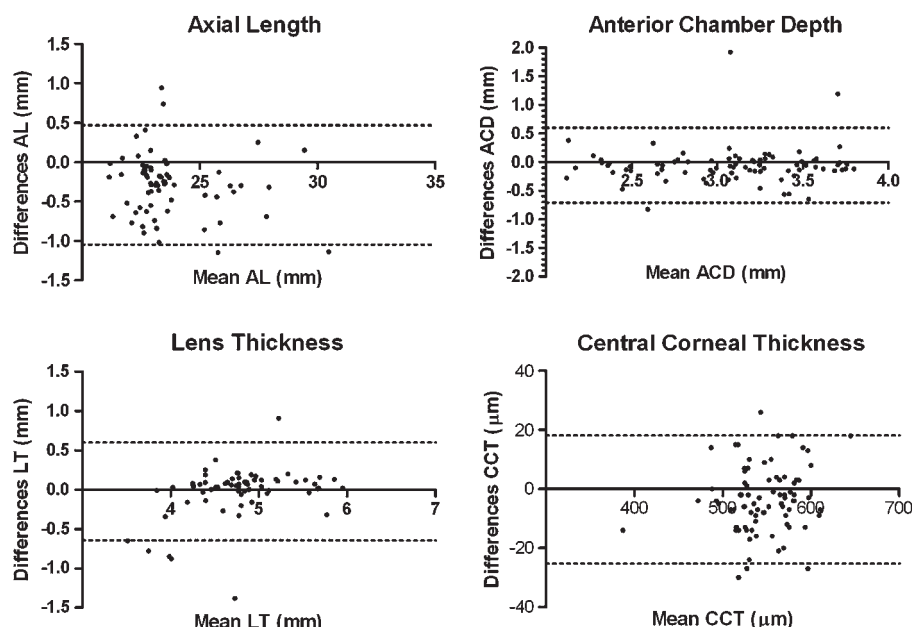
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Figure 1 Bland–Altman plots showing differences against their mean values for axial length (AL) (top left), anterior chamber depth (ACD) (top right), lens thickness (LT) (bottom left) and central corneal thickness (CCT) (bottom right), between the two devices (AL-3000 minus Lenstar values). The 95% limits of agreement are represented by dotted lines.



incorrect probe orientation or refractive indices used. In conclusion, we were able to show a higher reproducibility for all parameters comparing the Lenstar to the AL-3000 in measurable cataract eyes.

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Intravenous immunoglobulin (IVIg) in the management of severe refractory vernal keratoconjunctivitis

INTRODUCTION

Vernal keratoconjunctivitis (VKC) is a chronic, recurrent bilateral inflammatory disorder of the conjunctiva and cornea that affects mostly young males.

Inflammatory control and symptom prevention includes the use of first-line agents—mast cell stabilisers and anti-histamines—and for more severe sight-threatening disease, topical ciclosporin A and systemic steroids are tried, and finally second-line immunosuppressive agents are utilised.

Ethnicity can be an important outcome determinant of atopic disease, independent of the external childhood environment.¹ In our clinical experience, Asian patients, specifically those of Pakistani ethnicity, are less responsive to conventional therapies. The spectrum and phenotype of disease differs in tropical and temperate countries, with a dominance of B lymphocyte infiltration in

tropical patients with VKC.² VKC is potentially sight-threatening, with a risk of visual loss greater than 10% in tropical countries.^{3,4}

We report a case study of successful disease control using intravenous immunoglobulin (IVIg), in a young Pakistani male with severe refractory vernal keratoconjunctivitis, previously unresponsive to both topical and systemic immunosuppression.

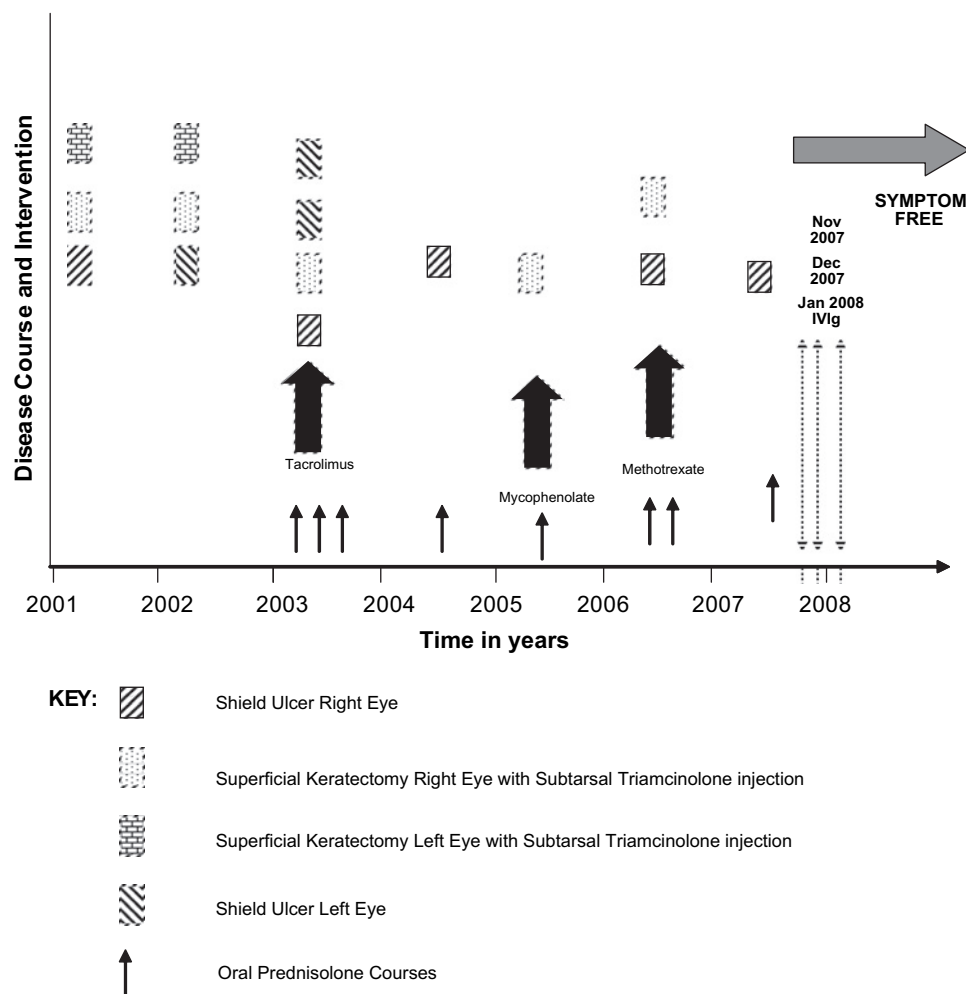
CASE STUDY

A 13-year-old boy was diagnosed at the age of 6 with VKC in 2001. He had a history of atopy from the age of 4 months, with eczema, asthma and frequent episodes of allergic conjunctivitis. Radioallergosorbent testing confirmed multiple environmental triggers such as grass pollen, dust, sunlight, nuts, eggs, melon, beans and chicken.

Clinically there were classical features of VKC such as excessive mucous, giant cobblestone papillae and recurrent shield ulcers complicated by episodes of microbial keratitis. Despite intensive topical treatment including mast cell stabilisers, steroids and ciclosporin 0.2%, his condition remained unstable; exacerbations of his disease quickly responded to 2-week courses of oral prednisolone at 20 mg daily, but intervals of remission lasted no longer than 8 weeks. Second-line therapies such as tacrolimus, mycophenolate mofetil and methotrexate were each trialled for 6-month periods in an attempt to control his disease.

Figure 1 illustrates the patient's disease course over the 7-year postdiagnosis and interventions tried.

All treatments failed to achieve remission, and his severe refractory disease resulted in the development of corneal scarring. Despite maintaining Snellen visual acuity at 6/9 in both eyes, he was developing early steroid induced cataracts.

Figure 1 Time line of disease course versus intervention.

Due to severity of inflammation, resultant morbidity and loss of time from school, alongside development of complications from November 2007, three infusions of intravenous immunoglobulin at a dose of 2 g/kg were commenced at monthly intervals. No side effects were reported. This treatment successfully induced a controlled clinical picture for 8 months; this is the longest period of remission achieved, requiring only simple topical agents for maintenance and not requiring topical ciclosporin A or oral steroids.

DISCUSSION

The severity of disease pattern in allergic eye disease can vary between individuals, with severe cases often associated with widespread atopy, including dermatitis, asthma and food allergies. The mechanism in the pathogenesis of ocular disease is in part IgE-mediated mast cell degranulation, alongside T and B cell activation, eosinophil recruitment, fibroblast activation and scarring. A conjunctival biopsy taken in 2006 of our patient revealed eosinophils, a few CD20 positive cells, yet a near absence of CD4

positive cells. Although profiles of inflammatory infiltrate vary in patient groups, our patient's findings were consistent with chronic allergic inflammatory responses. Therefore, utilising IVIg to modulate antibody-mediated activation of mast cells, B cell activation and suppress T cell activation and consequent cytokine responses⁵ was deemed appropriate.

IVIg therapy is very expensive and has potential side effects including thromboembolism, aseptic meningitis and the risk of transmitting bloodborne infections; however, actual reported side effects are extremely low, and therapy could arguably be cost-effective, as contrasted with the cost of managing a patient with severe refractory disease and potential childhood blind years.

In conclusion, IVIg has potential in the treatment of severe, refractory vernal keratoconjunctivitis. Significant improvement in all signs and symptoms with our patient was observed after treatment with IVIg. This innovative use of an established immunomodulatory therapy to treat VKC requires further long-term follow-up studies, but our early experience is encouraging.

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Scleral autoplasty for the repair of corneal perforations: a case series

We present a case series of patients who underwent a novel technique to reform the integrity of the eye following corneal perforation using autologous partial thickness scleral patch graft, or scleral autoplasty.

The principle was first described by Larsson in 1948, who used a 2.5 mm scleral patch,

from the inferior border from the ipsilateral lateral rectus, to seal a persistent corneal defect.¹

Despite this early promise, scleral autoplasty has subsequently been overlooked until a case which we reported in the BJO in 2006, which is the first case in the series presented here.²

METHODS

Nine autologous lamellar scleral patch grafts were performed in eight eyes of eight patients for corneal perforation in two surgical centres (table 1). A fornix-based conjunctival flap was formed in the same eye, and a 3 or 4 mm trephine used to harvest a sliver of sclera. The same trephine was then used to cut an indent around the perforation, and a superficial keratectomy performed in this region (inlay technique). The donor sclera was sutured with interrupted 10/0 nylon and covered with a therapeutic contact lens. In some cases, a freehand donor patch was fashioned, and if the corneal tissue consistency surrounding the perforation did

not allow trephination, the patch was sutured directly (overlay technique).

RESULTS

In six of eight patients, scleral autoplasty proved to be the definitive procedure for the repair of corneal perforation, when use of cyanoacrylate glue had failed. In the other two, it temporarily sealed the perforation and allowed time to control underlying inflammatory eye disease prior to penetrating keratoplasty.

DISCUSSION

This technique has several significant advantages over penetrating keratoplasty in the treatment of corneal perforation. Tissue is immediately available if other measures fail or are unsuitable. Importantly, there is no risk of rejection. As sclera has a high tensile strength, small, thin discs can be used. For non-axial defects, this has a lesser effect on central astigmatism, and in three cases, this was the reason for selecting this

Figure 1 Case 1 (top), case 2 (middle), case 4 (bottom) at 1 week (left) and 1 year (right).

