- 4 Binder MI, Chua J, Kaiser PK, *et al.* Endogenous endophthalmitis: an 18-year review of culture-positive cases at a tertiary care center. *Medicine (Baltimore)* 2003;**82**:97–105.
- 5 Trindade PA, McCulloch JA, Oliveira GA, *et al.* Molecular techniques for MRSA typing: current issues and perspectives. *Braz J Infect Dis* 2003;**7**:32–43.
- 6 Tenover FC, Arbeit RD, Goering RV. How to select and interpret molecular strain typing methods for epidemiological studies of bacterial infections: a review for healthcare epidemiologists. Molecular Typing Working Group of the Society for Healthcare Epidemiology of America. *Infect Control Hosp Epidemiol* 1997;**18**:426–39.
- 7 Beadle J, Wright M, McNeely L, *et al.* Electrophoretic karyotype analysis in fungi. *Adv Appl Microbiol* 2003;**53**:243–70.
- 8 Fiorito SM, Galarza PG, Sparo M, *et al.* An unusual transmission of *Neisseria meningitidis*: neonatal conjunctivitis acquired at delivery from the mother's endocervical infection. *Sex Transm Dis* 2001;**28**:29–32.
- 9 Chandra NS, Torres MF, Winthrop KL, *et al.* Cluster of *Mycobacterium chelonae* keratitis cases following laser in-situ keratomileusis. *Am J Ophthalmol* 2001;**132**:819–30.

Figure 1 A two port pars plana vitrectomy was performed using an anterior chamber maintainer with limbal infusion to free the intraocular lenses from surrounding vitreous so they could gently float to the posterior pole.

The second lens was then removed in the same fashion and a new IOL was placed by transscleral fixation.³

All three patients had pseudoexfoliation syndrome but no complications were noted during the initial cataract surgeries. The initial dislocation in all cases followed minimal blunt ocular trauma. The second dislocation occurred spontaneously 7–15 days following placement of the secondary sulcus lens. All procedures were performed as described above within 2 days of presentation. Follow up ranged from 6 months to 3 years; postoperative visual acuities ranged from 20/30 to 20/50. No complications developed in the postoperative period.

Comment

Postoperative complications of dislocated IOLs include retinal detachment, cystoid macular oedema, and vitreous haemorrhage.⁴ The behaviour and risk of having multiple dislocated IOLs within the vitreous cavity is not well known and, to our knowledge, there has been only one case report of multiple dislocated IOLs.⁵

Treatment options include observation, medical therapy (pharmacological miosis), repositioning, removal, IOL exchange, or placement of a second IOL into the eye.^{6–8} If dislocation is noted to occur intraoperatively, attempts to retrieve the PCIOI without the use of posterior vitrectomy techniques may be associated with severe posterior segment complications and should be avoided. The temptation to place an anterior chamber IOL (ACIOI) at this time also should be resisted since the potential for complications is difficult to predict, and the presence of an ACIOI may render subsequent management more complex.⁹

Although the initial dislocation in all cases followed minimal blunt ocular trauma, the presence of two dislocated PCIOIs within the vitreous cavity, and the fact that dislocations occurred within 10–20 days, both for the first and second PCIOI likely indicate that residual capsule support was inadequate to place a sulcus lens. Vitrectomy and repositioning with scleral fixation or intraocular lens exchange should be considered first at the time of primary PCIOI dislocation, rather than secondary sulcus fixation of a second PCIOI in pseudoexfoliation syndrome.

In conclusion, pseudoexfoliation syndrome seems to be a risk factor for dislocated foldable IOLs in the vitreous cavity after cataract surgery.¹⁰ Eyes with two foldable IOLs in the vitreous cavity after cataract surgery are at risk of developing posterior segment complications such as retinal detachment, vitreous haemorrhage, and macular oedema. However, performing two port pars plana vitrectomy, with limbal extraction and transscleral fixation of a new IOL results in improved visual acuity and rapid rehabilitation without further complications.

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References

- 1 Mathai A, Thomas R. Incidence and management of posteriorly dislocated nuclear fragments following phacoemulsification. *Indian J Ophthalmol* 1999;**47**:173–6.

Table Clinical findings in patients with multiple dislocated intraocular lenses

Patient (eye)	Age (years)	Sex	Pre-phaco VA	Post-phaco VA	Time of first dislocation	Cause of dislocation	Time of placement of second lens	Time of second dislocation	Pre-vitrectomy VA	Post-vitrectomy VA
1	75	F	20/300	20/40	POD #17	Zonular laxity and poor capsular support	Sulcus IOL at POD # 28	POD2 #15	20/400	20/30
2	80	F	20/400	20/40	POD #15	Zonular laxity and poor capsular support	Sulcus IOL at POD # 22	POD2 #20	20/300	20/50
3	60	M	20/200	20/20	POD #10	Zonular laxity and poor capsular support	Sulcus IOL at POD # 25	POD2 #14	20/300	20/50

Phaco, phacoemulsification and intraocular lens (IOL) implantation; VA, Snellen visual acuity; POD, postoperative day; POD2, postoperative day after sulcus IOL.

- 2 Tappin MJ, Larkin DF. Factors leading to lens implant decentration and exchange. *Eye* 2000;14:773-6.
- 3 Malbran ES, Malbran E, Negri Aranguren A. Scleral-fixated intraocular lenses. *Arch Ophthalmol* 1988;106:1347-8.
- 4 Mello MO Jr, Scott IU, Smiddy WE, et al. Surgical management and outcomes of dislocated intraocular lenses. *Ophthalmology* 2000;107:62-7.
- 5 Cavallini GM, Pagliani L, Campi L, et al. Scleral fixation of a posterior chamber intraocular lens in a patient with two dislocated lens implants. *Eur J Ophthalmol* 2004;14:149-52.
- 6 Mitra RA, Connor TB, Han DP, et al. Removal of dislocated intraocular lenses using pars plana vitrectomy with placement of an open-loop, flexible anterior chamber lens. *Ophthalmology* 1998;105:1011-14.
- 7 Parmar DN, Kychenthal A, Ezra E, et al. Vitreoretinal management of posteriorly dislocated plate haptic silicone intraocular lenses. *Retina* 2002;22:169-75.
- 8 Sarrafzadeh R, Ruby AJ, Hassan TS, et al. A comparison of visual results and complications in eyes with posterior chamber intraocular lens dislocation treated with pars plana vitrectomy and lens repositioning or lens exchange. *Ophthalmology* 2001;108:82-9.
- 9 Williams DF, Del Piero EJ, Ferrone PJ, et al. Management of complications in eyes containing two intraocular lenses. *Ophthalmology* 1998;105:2017-22.
- 10 Gross JG, Kokame GT, Weinberg DV. Dislocated In-The-Bag Intraocular Lens Study Group. In-the-bag intraocular lens dislocation. *Am J Ophthalmol* 2004;137:630-5.

The management of severe sclerokeratitis following corneal transplantation surgery

There is a paucity of information in the scientific literature regarding post keratoplasty inflammation and the management of this rare but serious problem. Although corneal graft survival is known to be reduced when keratoplasty is undertaken in inflamed eyes,¹ and published data are available for patients with known risk factors of atopy² and *acanthamoeba* keratitis,³ little is known in this regard for grafted eyes in individuals without discernable risk factors who develop severe (non-rejection) inflammation following surgery.

We describe a series of six disparate patients who developed severe scleral inflammation in the postoperative period and outline our management strategy in each case. Scleritis was diagnosed using the following criteria: severe diffuse scleral inflammation associated with pain and photosensitivity. All the cases manifested diffuse rather than nodular scleral involvement without involvement of the residual host cornea. The corneal grafts were secured using 10/0 nylon monofilament suture mounted on a 3/8 circle side-cutting stainless steel needle (Catalogue No 8065-208001 Alcon Laboratories, Hemel Hempstead, UK). Two cases are described in detail and a comparison in all the cases of the salient features is made in a summary table 1.

Case reports

A 40 year old man presented with a left central corneal abscess. He had been treated for a presumed episode of herpetic epithelial keratitis and conjunctivitis several years before this but had had documented recurrent corneal erosion syndrome and blepharitis for the previous 2 years. On examination his left visual acuity was hand motions. He was found to have a central left corneal abscess 2.3 mm in width and 2.6 mm in

height; this was covered by a mucous plaque but was noted to be Seidel positive and he manifested a flat anterior chamber. He also had acne roseacea and mild meibomianitis but no clinical evidence of scleritis. He had been treated with intensive fortified topical antibiotics (cefuroxime 5% and gentamicin forte) by the referring unit.

A putative diagnosis of phlyctenular keratitis secondary to ocular roseacea was made and cyanoacrylate glue was applied to the perforation and kept in place with a therapeutic contact lens; treatment was commenced with prophylactic topical antibiotics (cefuroxime drops 5% four times daily), doxycycline 100 mg daily, and aciclovir 400 mg three times daily orally. His anterior chamber remained extremely shallow despite these measures and he underwent a left penetrating keratoplasty, using interrupted suture technique (donor graft 7.25 mm, host trephination 7.00 mm) 2 days after presentation. Prednisolone 80 mg daily was commenced, this was reduced to 60 mg at review 5 days after the operation. Twelve days post surgery the graft was clear; however, he manifested a profound circumcorneal scleral inflammation associated with pain and photophobia. Oral ciclosporin 5 mg/kg was commenced in addition to the oral prednisolone and within 2 weeks the inflammation was settling. Snellen acuity noted to be 6/24 at this stage. At review 8 weeks post surgery the inflammation had settled and he was able to discontinue systemic prednisolone and reduce the oral ciclosporin therapy. At last review 1 year post grafting he was free of inflammation.

A 23 year old man presented with a history of blunt trauma to the left eye 11 months previously. This had resulted in a total hyphaema and vitreous haemorrhage (but no retinal detachment) and elevated intraocular pressure. Over the 2 weeks following the injury he was documented to have rebled twice. On examination his acuity was perception of light with good projection and he had an opaque blood stained cornea of 730 µm central thickness and intraocular pressure of 22 mm Hg.

He underwent a left penetrating keratoplasty with a continuous suture closure technique (donor 8.25 mm, host 8.00 mm), combined with excision of cyclitic membrane, pupilloplasty, extracapsular cataract extraction, and posterior chamber implant. A week after surgery he manifested severe scleral inflammation, pain, and photosensitivity and a brisk anterior chamber cellular response (fig 1). Systemic prednisolone 80 mg daily was commenced. Within a week the inflammation was settling and the prednisolone was reduced to 60 mg daily. Two weeks later, however, he had evidence of suture loosening without hypotony (Seidel negative) and a therapeutic contact lens was applied. Snellen acuity at this time was 6/60 improving to 6/12 with pinhole. Two weeks later further suture loosening was noted and the running suture was trimmed; complete suture removal was required a month later (10 weeks post keratoplasty). He was noted to have developed 90° of inferior peripheral anterior synechiae at next review and underwent surgical release of these 8 months post keratoplasty, he sustained a one third depth hyphaema and vitreous haemorrhage post-operatively. He once again developed a severe scleral inflammation within a week of surgery and was commenced on prednisolone 60 mg daily. The inflammation settled on a

reducing regimen of this; however, the corneal graft failed because of delayed clearing of the hyphaema and a tractional retinal detachment was noted 4 months later. A combined repeat penetrating keratoplasty (interrupted suture closure) with detachment repair was undertaken under systemic prednisolone (80 mg) cover to prevent scleral inflammation successfully. A year later he still has a clear functioning graft (fig 2).

Comment

The series of patients here represent a disparate group with varying indications for keratoplasty; four of the six patients had undergone previous ocular surgical procedures.

Comparing this group to the unit's patient profile for an entire calendar year revealed no discernable trend in susceptibility to post keratoplasty scleral inflammation (PKS) making it difficult to identify potential cases in advance and hence plan prophylactic measures.

Five of the presented cases developed PKS within 2 weeks of operation with the sixth—who had had prophylactic systemic immunosuppression—manifesting PKS as the prophylaxis was reduced, arguably too rapidly over a 5 week period. It is difficult to justify using potentially toxic immunosuppressive agents for longer than this in prophylaxis.

The aetiology of PKS is uncertain, it has been noted that post surgical scleritis develops more frequently following multiple surgical procedures,⁴ implying a sensitisation of the ocular immune system. Surgical technique and suture material have also been implicated in the aetiology of surgically induced necrotising scleritis (SINS), the majority of these cases occurring after cataract surgery and it has been rarely described in keratoplasty. We may speculate that PKS is a distinct entity but there is little evidence for this either way. Other authorities have suggested that surgical trauma or temporary localised ischaemia⁵ may be a factor in triggering scleritis. In patients undergoing their first ocular procedure post surgical scleritis also occurs more frequently in patients with a predisposing medical disorder⁵ (this group having a fivefold higher incidence of collagen vascular disease) and vasculitis secondary to deposition of circulating immune complexes in deep episcleral vasculature maybe one patho-aetiological mechanism at hand. Only one of our patients has a history an associated systemic condition—having previously manifested ankylosing spondylitis related uveitis. Molecular mimicry or cross reactivity between an ocular antigen and other tissue antigens or microbial antigens has also been suggested as an inflammatory trigger.^{5,6}

It has also been noted that corneal grafts devoid of donor epithelium reject more rapidly than full thickness grafts.⁷ This has led to speculation as to the role of donor epithelium—as a barrier to proinflammatory agents from the tear film or ocular surface; as a source of anti-inflammatory factors itself; or a sink inactivating proinflammatory factors released within the graft or surrounding host tissue.⁸ The majority of our cases had an intact epithelium in the immediate post-operative period.

The optimum treatment strategy for patients with PKS remains to be fully elucidated, the inflammation in half of our patients settled on a short course of high dose