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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

Table 1 Summary of each patient and their clinical course (including ocular history) before and after developing postoperative scleral inflammation

Case	Age	Sex	Diagnosis	Surgery	Onset of PKS	Treatment of PKS	Outcome
1	40	M	Left corneal abscess with perforation. Ocular history: recurrent erosion syndrome, blepharitis (acne roseacea)	Left PK, interrupted suture technique, prophylactic systemic prednisolone 80 mg commenced 2 days post surgery	12 days post surgery	Systemic prednisolone continued and supplemented with CSA 5 mg/kg daily Prednisolone 80 mg commenced	Scleritis responded within 2 weeks and absent at 8 weeks post surgery, immunosuppression tapered off. Clear corneal graft
2	23	M	Left blood stained cornea secondary to blunt trauma and rebleeding ×2	Left PK, continuous suture technique, excision cyclitic membrane, pupilloplasty, extracapsular cataract extraction and posterior chamber implant	7 days post surgery	Prednisolone 80 mg commenced	Reduction of inflammation within 7 days. Suture loosening at 3 weeks, suture trimming at 5 weeks, complete suture removal at 10 weeks post surgery Surgical peripheral anterior synechial release at 8 months post surgery—hyphaema + vitreous haemorrhage Recurrent scleritis 7 days post procedure responding to prednisolone 60 mg. Graft failure noted—second graft performed with systemic steroid cover uneventfully Inflammation settled 8 weeks post surgery. Presented 2 months later with inflammation and endothelial rejection. Graft failure at 5 months post surgery
3	57	M	Right corneal graft failure. Ocular history: bilateral congenital cataract extraction, left blunt trauma at 42 years, bilateral anterior chamber intraocular lens at 49 years. Right corneal decompensation with PK at 52 years, graft failure	Repeat right PK, HLA matched donor, interrupted suture technique	7 days post surgery	Prednisolone 60 mg daily, CSA 7.5 mg/kg daily added 9 days later and discontinued within 3 days (adverse effects)	Inflammation settled 8 weeks post surgery. Presented 2 months later with inflammation and endothelial rejection. Graft failure at 5 months post surgery
4	39	M	Left band shaped keratopathy, graft failure, thinning and active herpes simplex keratitis. Ocular history: left pterygium excision (1989), PK for scarring. Infective keratitis and repeat PK (1998)	Left PK, extracapsular cataract extraction, posterior chamber intraocular lens. Systemic prednisolone 40 mg daily + aciclovir	5 weeks post surgery when prednisolone tapered	Prednisolone 80 mg daily, CSA 7.5 mg/kg daily. Surgical release of peripheral anterior synechiae + intraocular TPA (12.5 µg) 7 weeks post surgery	Inflammation settled over 6 months when prednisolone stopped, CSA continued for further 18 months, clear graft
5	54	M	Left bullous keratopathy. Ocular history: high myopia, ankylosing spondylitis associated uveitis, open angle glaucoma. Multiple glaucoma procedures including, trabeculectomy, cyclodiode, Baerveldt procedures; extracapsular cataract extractions and anterior chamber intraocular lens	Left PK, removal of anterior chamber intraocular lens and insertion of a posterior chamber sulcus fixated lens. Interrupted suture closure	2 weeks post surgery	Prednisolone 80 mg daily — little response, supplemented by CSA 7.5 mg/kg 1 week later	Inflammation settled 2 months post surgery. Clear graft
6	67	M	Bilateral scarred corneae of indeterminate aetiology, normal posterior segments	Right PK, extracapsular cataract extraction and posterior chamber intraocular lens	7 days post surgery	Prednisolone 80 mg daily	Inflammation responded within 7 days and prednisolone tapered over 6 weeks. Clear graft

PKS, post-keratoplasty inflammation, CSA, ciclosporin A, PK, penetrating keratoplasty, TPA, tissue plasminogen activator.

Figure 1 Scleral inflammation evident 7 days post penetrating keratoplasty (case 2). **Figure 2** Appearance after resolution of inflammation (case 2).

systemic corticosteroids, the others required supplementation of this with ciclosporin A as an immunosuppressive as well as steroid sparing agent. It has been noted that patients with additional risk factors for inflammation may be refractory to systemic corticosteroids alone.⁹ There are now several newer agents available—rapamycin, tacrolimus, mycophenolate mofetil, and anti-tumour necrosis factor agents—that may have a role in the future. It should be noted that all these agents are associated with a potential element of additional morbidity. At present, treatment choice based on clinical assessment of previous inflammatory episodes or predisposing medical disorders should guide the clinician in judicious choice of therapy for PKS.

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Persistent detachment of the fovea after non-buckling repair of rhegmatogenous retinal detachment

Persistent detachment of the fovea is increasingly recognised as a cause of delayed visual recovery after repair of rhegmatogenous retinal detachment (RRD).^{1–6} Often subfoveal fluid cannot be seen on biomicroscopy or indirect ophthalmoscopy. It has only been the recent introduction of optical coherence tomography (OCT) into clinical practice that has allowed a more detailed study of this fluid. Persistent subfoveal fluid has primarily been reported after scleral buckling (SB) surgery,⁷ though it has been documented after pneumatic retinopexy^{8–9} and pars plana vitrectomy (PPV).¹⁰ The persistent fluid can be very subtle and difficult to detect on fundus examination. We report three cases of delayed visual recovery caused by persistent neurosensory macular detachment after non-scleral buckling procedures. Despite a normal appearing fundus, OCT confirmed the presence of persistent subretinal fluid.

Case report

A 58 year old myopic woman was referred for the treatment of a macula-off retinal detachment in the left eye. Her symptoms had begun 6 weeks earlier. Examination of the right eye was within normal limits. Best corrected visual acuity (BCVA) in the left eye was 20/400. Slit lamp examination (SLE) was

Figure 1 Top: Fundus photograph and OCT (nasal to temporal scan) from the patient in case 1 taken 1 month after the pneumatic retinopexy. The OCT shows the presence of persistent subretinal fluid. Middle: Fundus photograph and OCT (nasal to temporal scan) from the patient in case 2. The OCT shows the presence of persistent subretinal fluid. Note the presence of a fine epiretinal membrane along the temporal macula, but this was not thought to be contributing to the persistence of the subretinal fluid. Bottom: OCT (nasal to temporal scan) from the patient in case 3 confirming the presence of persistent subretinal fluid.

unremarkable. Dilated fundus examination revealed a horseshoe retinal tear at 1 o'clock with macula-off temporal retinal detachment. Cryotherapy and pneumatic retinopexy with C₃F₈ gas were performed the same day without complication. One week after the procedure the BCVA had improved to 20/100. By one month, the BCVA improved to 20/60. Though the retina appeared attached on ophthalmoscopic examination, OCT showed a trace amount of persistent subretinal fluid (fig 1, top). Over the course of the next 3 months the OCT finding of subretinal fluid resolved completely and the BCVA improved to 20/40.

A 54 year old woman was referred for treatment of a macula-off RRD. She had noted a visual acuity decrease in her left eye for the previous 3–4 days. Ophthalmic history was significant for lipid keratopathy secondary to herpetic keratitis, resulting in a baseline BCVA of 20/50 left eye. On examination, the visual acuity in the left eye was hand motions. Dilated fundus examination showed a horseshoe tear at the 2 o'clock position with an associated macula-off RRD. Cryotherapy and pneumatic retinopexy with C₃F₈ gas were performed the same day without complication. One week later the visual acuity improved to 20/70. Additional laser photocoagulation was performed around the tear, as there was only a light cryotherapy scar. Although the fundus view was not clear because of corneal opacification, the retina appeared attached on ophthalmoscopy. OCT imaging, however, revealed a neurosensory detachment with persistent subretinal fluid under the fovea (fig 1, middle). Complete reabsorption of the fluid occurred by 18 months and final BCVA improved to 20/50.

A 65 year old man was referred for treatment of a macula splitting retinal detachment in the left eye. The BCVA in the left eye was 20/20. SLE revealed an intact posterior capsule with a posterior chamber intraocular lens, which was implanted 2.5 years previously. Dilated fundus examination of the left eye revealed a macula splitting detachment caused by a retinal tear at the 2 o'clock position. PPV with fluid-gas exchange (12% C₃F₈) and endolaser was performed. In addition, small retinal breaks at the 11 30, 12 30, 1 30, and 3 00 o'clock positions were treated with transscleral cryotherapy. Postoperatively, after the gas had reabsorbed, the retina appeared attached, but OCT showed multiple, focal detachments involving the central macula (fig 1, bottom). These fluid loculations resolved after 11 months and the patient's final BCVA was 20/15.

Comment

It is not known with certainty why shallow areas of subretinal fluid can persist after retinal detachment repair. Fluorescein and indocyanine green angiography studies have failed to show leakage in the areas of persistent subretinal fluid.^{1–2} One study suggested that the presence of posterior vitreous detachment (PVD) may have a role, since only three out of 10 eyes with PVD had persistent retinal detachment compared to four out of five eyes without PVD.³ Another explanation could be the focal impairment of the retinal pigment epithelium pump affecting its ability to reabsorb the subretinal fluid.⁷ The subretinal fluid itself may also provide an impediment to reabsorption because of the protein rich and highly viscous nature of the fluid in chronic retinal detachment. In previous reports these detachments resolved spontaneously within 4–26 months,^{2–4 8 9} which is similar to our cases.

We observed two types of persistent detachment after RRD surgery based on OCT characteristics. The first is a diffuse shallow retinal detachment, and the second is a circumscribed focal detachment. These two types can co-exist in the same patient, and may represent a spectrum of severity as diffuse detachment might convert to focal detachments as the fluid reabsorbs. On OCT, focal detachments appear to resemble small pigment epithelial detachments, since the outer surface of the detached retina, which faces the subretinal fluid, has an outer edge of higher than normal reflectivity. In all our cases, however, there was a second, normally positioned highly reflective layer underneath the subretinal fluid that corresponded to the actual retinal pigment epithelial layer. The high reflectivity of the neurosensory detachment seen on OCT could be an image artefact resulting from the summation of reflectance of the outer retina that is now being scanned more perpendicularly than normal. Another explanation could be that the outer segments of the photoreceptors are disrupted and have a higher reflectivity compared with their normal parallel arrangement. High resolution OCT might help resolve this discrepancy.

Subclinical areas of retinal detachment may persist after RD surgery, including vitrectomy and pneumatic retinopexy. Clinicians should consider imaging patients with OCT if this phenomenon is suspected, particularly if the visual recovery does not correspond to the biomicroscopic and ophthalmoscopic examination.