occlusion, bandage contact lenses, tarsorrhaphy, botulinum toxin ptosis, conjunctival flaps, and amniotic membrane transplants.

In our patient, as conventional treatment failed to restore ocular surface integrity, he underwent an amniotic membrane transplant (AMT). AMT treats persistent epithelial defects by acting as a biological contact lens, preventing lid trauma and corneal exposure.[8] It also has unique biological properties including antibacterial, epithelialisation-promoting, and fibrous-supporting effects.[9] However, in our case AMT made no difference to the epithelial defect, so autologous serum tears were started.

Autologous serum tears support corneal epithelial cell migration and differentiation, and have been found to be superior to either preserved or unpreserved pharmaceutical preparations in maintaining keratocyte morphology and function.[10] Patients have reported that the duration of symptomatic relief from serum tears is longer than artificial tears.[11] The rationale for treatment is based on the fact that vitamins and growth factors found in tears are also found in serum. Autologous serum tears have been successfully used in the treatment of dry eye secondary to Sjogren's syndrome, cicatricial pemphigoid, Stevens-Johnson syndrome, and superior limbic keratitis.[12] Local variations in strength (20–100%)[10,12] and preparation of autologous serum tears exist. In our patient, a strength of 100% was used, made from 60 ml clotted blood centrifuged at 2000 revs/min for 5–10 min. Aliquots of serum were transferred into several sterile 5 ml glass bottles that were each used for 1 week. Microbial contamination of the autologous serum drops could theoretically lead to further corneal infection, but this does not appear to be a common problem.[12]

Owing to the increasing survival rates of patients following BMT, it is important for both ophthalmologists and haematologists to be aware of the ocular complications associated with cGVHD. Early use of autologous serum drops in such patients who develop epithelial breakdown postpenetrating keratoplasty may have a role in preventing sight-threatening complications.

References

1 Poon AC, Geerling G, Dart JKG, Franenkel GE, Daniels JT. Autologous serum eye drops for dry eyes and epithelial defects: clinical and *in vitro* toxicity studies. *Br J Ophthalmol* 2001; **85**: 1188–1197.
2 Heath JD, Acheson JF, Schulenburg WE. Penetrating keratoplasty in severe ocular graft vs host disease. *Br J Ophthalmol* 1993; **77**: 525–526.
3 Tranos PG, Forbes J, Jagger J. Corneal perforation in chronic graft vs host disease. *Eye* 2001; **15**: 111–113.
4 Trimble RB. Ocular complications of graft vs host disease. *Res Clin Forums* 1982; **4**: 93–95.
5 Peris-Martínez C, Menezo JL, Díaz-Llopis M, Aviñó-Martínez JA, Navea- Tejerina A, Risueño-Reguillo P. Multilayered amniotic membrane transplantation in severe ocular graft vs host disease. *Eur J Ophthalmol* 2001; **11**: 183–186.
6 Jack MK, Jack GM, Sale GE, Shulman HM, Sullivan KM. Ocular manifestations of graft-v-host disease. *Arch Ophthalmol* 1983; **101**: 1080–1084.
7 Killingsworth DW, Stern GA, Driebe WT, Knapp A, Dragon DM. Results of therapeutic penetrating keratoplasty. *Ophthalmology* 1993; **100**: 535–541.
8 Kruse FE, Rohrschneider K, Volcker HE. Multilayered amniotic membrane for reconstruction of deep corneal ulcers. *Ophthalmology* 1999; **106**: 1504–1511.
9 Khodadoust AA, Silverstein AM, Kenyon KR, Dowling JE. Adhesion of regenerating corneal epithelium: the role of basement membrane. *Am J Ophthalmol* 1968; **65**(3): 339–348.
10 Rocha EM, Pelegrino FSA, de Paiva CS, Vigorito AC, de Souza CA. GVHD dry eyes treated with autologous serum tears. *Bone Marrow Transplant* 2000; **25**: 1101–1103.
11 Tsubota K, Goto E, Shimmura S, Shimazaki J. Treatment of persistent corneal epithelial defect by autologous serum application. *Ophthalmology* 1999; **106**: 1984–1989.
12 Ogawa Y, Okamoto S, Mori T, Yamada M, Mashima Y, Wantanabe R *et al*. Autologous serum eye drops for the treatment of severe dry eye in patients with chronic graft vs host disease. *Bone Marrow Transplant* 2003; **31**: 579–583.

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Sir,

Choroidal folds after 25 gauge transconjunctival sutureless vitrectomy

Pursuit for smaller wound incision, expedited postoperative recovery are always the core issues in ophthalmic surgical development. Transconjunctival sutureless vitrectomy (TSV) with 25 gauge (G) is a new approach in vitreoretinal surgery without the need of preparing conjunctival and scleral openings or closure.[1,2] The mean operative time had been shortened by 33.7% with this new vitrectomy system.[1,2] It was demonstrated

to be safe in a retrospective series with only one eye had postoperative retinal detachment. [2] We hereby present a case of choroidal fold after 25 G TSV secondary to ocular hypotony. Surgeons who want to introduce 25 G vitrectomy system into their practice should be aware of the possible complications.

Case

A 53-year-old man received 25 G TSV for a recurrent epiretinal membrane of his left eye (Figure 1a,b). The preoperative best-corrected visual acuity (BCVA) was 20/40. On postoperative day 1, intraocular pressure of 5 mmHg, diffuse conjunctival chemosis, shallow anterior chamber coupled with VA of hand movement were noted in the left eye. Fundal examination revealed 360° choroidal detachment. No cyclodialysis or clefting could be seen in ultrasonic biomicroscopy. Conventional treatment modalities including reformation of anterior chamber with viscoelastic, pressure patching, topical 1% atropine, and oral prednisolone (1 mg/kg/day) were tried. Intraocular pressure began to rise on day 6 and the choroidal detachment resolved subsequently. At 3 months after TSV, marks of choroidal fold persisted with residual metamorphosia and the BCVA was 20/40 (Figure 1c,d).

In contrast to conventional 20 G cannula-entry pars plana vitrectomy, surgical as well as visual merits of 25 G TSV are attributed to smaller conjunctival and scleral incisions, less tissue manipulation, and obviated need for wound suturing.[1,2] Possible complications such as wound leakage and endophthalmitis, however, should be addressed.[3] In fact, nearly 11.4% (four out of 35 eyes) of post 25 G TSV patients experienced unexplained low intraocular pressure ($\leq$7 mmHg) on postoperative day 1.[1] Gupta *et al* reported transient hypotony on the day after 25 G TSV and 14 of their 100 eyes required supplemental intraocular gas, air, or saline injection (Gupta A. ARVO Meeting, 2003, Abstract). Similar complication of choroidal detachment has also been reported with 23 G TSV in 2 out of 225 eyes of a series.[4]

Size of sclerotomy and presence of vitreous tuft at sclerotomy sites have been proposed to be factors related to the extent of self-sealing.[2] Nevertheless, we believe that other conditions like thinning of sclera in pathologic myopia, scarring, or necrosis of previous wound entry sites, excessive intraoperative manipulation are also important determinants in the 25 G TSV system.

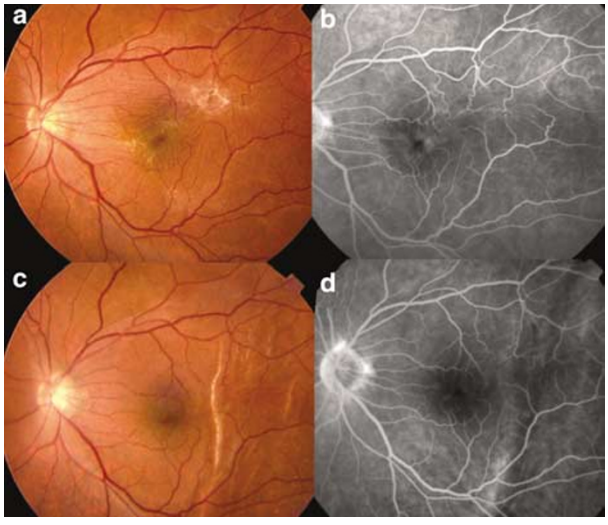

Figure 1 (a) Colour fundus photography showing recurrent epiretinal membrane in the left eye of Case 1. (b) Fluorescein angiography demonstrating the tortuosity of the paramacular vessels and the cystoid macular oedema. (c) Colour fundus photography of the right eye showing choroidal folds temporal to the fovea. (d) Fluorescein angiography showing the typical hypofluorescence and hyperfluoresence bands suggestive of choroidal folds and settled choroidal effusion.

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References

1 Fujii GY, de Juan Jr E, Humayun MS, Chang TS, Pieramici DJ, Barnes A *et al*. A new 25-gauge instrument system for transconjunctival sutureless vitrectomy surgery. *Ophthalmology* 2002; **109**: 1807–1813.
2 Fujii GY, de Juan Jr E, Humayun MS *et al*. Initial experience using the transconjunctival sutureless vitrectomy system for vitreoretinal surgery. *Ophthalmology* 2002; **109**: 1814–1820.
3 Lam DS, Yuen CY, Tam BS *et al*. Sutureless vitrectomy surgery. *Ophthalmology* 2003; **110**: 2428–2429.
4 Hilton GF, Josephberg RG, Halperin LS *et al*. Office-based sutureless transconjunctival pars plana vitrectomy. *Retina* 2002; **22**: 725–732.

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Sir,

Total hyphema following postoperative enoxaparin (Clexane)

Postoperative hyphema is a well-known complication of intraocular surgery. Internists and ophthalmologists are uncertain of the significance of antiplatelet drugs and systemic anticoagulation therapy side effects in these patients. Controversies exist regarding the safety of these drugs with regard to risks of ocular haemorrhage. We report what we believe to be the first case of total hyphema in a patient prescribed enoxaparin (Clexane Aventis Pharma Pty Ltd) in the postoperative period who had undergone a redo trabeculectomy.

Case report

A 73-year-old man underwent a redo trabeculectomy enhanced with 5-fluorouracil (5FU). The surgery was uncomplicated. The bleb was massaged on postoperative day 2 with no hyphaema seen following this. There had been no intraocular bleeding with previous surgery. The patient's general medical history included aortic and mitral valve replacement with long-term warfarin therapy. The haematocrit was 0.425 (normal 0.40–0.55) and platelet count being $2.03 \times 10^5/cm^{-3}$ (normal 1.5–4.5×10^5). Warfarin was not stopped prior to surgery. The INR on the day of the surgery was 1.6. At 2 days following the surgery INR was found to be 1.3. Enoxaparin 1 mg/kg (80 mg) to cover the risk of emboli from the prosthetic valves was recommended by the physician. On day 4 postoperatively the IOP was 12 mmHg with a functioning filtration bleb, clear cornea and noninflamed anterior chamber. However, that night he suffered a total hyphaema with pressures rising to 64 mmHg. The patient denied rubbing his eyes. Medical therapy partially controlled the pressure. A paracentesis was only partially successful and aspiration of the clot with vitrectomy cutter was performed. This controlled the IOP. The residual blood behind the iris and around the intraocular lens gradually resorbed over the next 3 months. The final outcome of the surgery was an IOP of 12 mmHg, with cornea stained and decompensated and a vision of counting fingers at 12 months.

Comment

Most hyphaemas following intraocular surgery occur at the time or in the immediate postoperative period.[1] The source of the bleeding appears to be the iris root or scleral incision.

Crystalline warfarin sodium is an anticoagulant that acts by inhibiting vitamin K-dependant coagulation factors including Factors II (prothrombin), VII, IX and X (intrinsic pathway). Enoxaparin, a low molecular weight heparin (MW 4500 Da), not only inhibits thrombin directly, but also acts on prothrombinase complex, that is, Factors Xa and Va calcium and phospholipid (extrinsic pathway) and by releasing an endothelium-bound pool of tissue factor inhibitor (TFPI). The drugs act on the different limbs of the Coagulation Cascade and intersect at Factor X. Warfarin takes 5–7 days for peak activity. Enoxaparin takes 3–12 h for peak activity to start, has greater bioavailability, and a longer, often unpredictable half-life.[2,3]

In hyphaemas a fibrin–platelet clot forms that reaches maximum stabilisation by day 5–7. These clots do not show any fibroblastic activity unlike other parts of the body and finally break down via the fibrinolytic system.[4]

We propose that enoxaparin with its unpredictable and prolonged half-life acting via the extrinsic arm of the coagulation cascade tipped the balance in favour of haemorrhage when the clot was remodelling at day 4. The fall in INR in 2 days after surgery we feel is probably due to the preoperative antibiotic cover with systemic ampicillin/gentamicin and the midazolam used as part of anaesthetic.[2]

The physicians were concerned with the inadequate anticoagulation in a patient with two prosthetic heart valves. As the effects of warfarin may take up to 4 days to occur, a decision was made to add the low molecular weight heparin similar to what is done when initiating warfarin therapy although this is usually with normal intravenous heparin.

The incidence of sight-threatening complications in patients who have undergone intraocular surgery while receiving long-term anticoagulation therapy with warfarin is not significantly different from patients who were not on such treatment.[5,6] Furthermore, no differences in major haemorrhagic events are noted among patients who had their anticoagulation treatment stopped in the perioperative period for ocular surgery when compared to patients who did not stop therapy.[7,8]