weakness, and extensive vitreous degeneration, all of which are associated with RP.^{3,4} In this case, vitreous degeneration is also likely to have facilitated migration of the IOL from the vitreous cavity to the AC following pharmacological mydriasis. Following 1 year of conservative management he presented with pupil block glaucoma. Although this resolving following laser iridotomy a combination of the acute attack and IOL-cornea touch has resulted in corneal decompensation.

This case serves to highlight the problems that can arise owing to the lens and vitreous abnormalities which occur in RP. Although similar cases are likely to be rare we would recommend prophylactic laser iridotomy in any case where the IOL has migrated into the AC.

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Sir,
Corneal striae in thyroid eye disease

We report a patient with thyroid eye disease (TED) in whom corneal striae were a sign of increased orbital pressure.

Case report

A 48-year-old Oriental lady with TED was referred urgently to the orbital clinic. She had had previous radioactive iodine treatment and was euthyroid. She was a smoker.

Although her best-corrected visual acuities were 6/12 bilaterally, examination of the anterior segments revealed bilateral corneal stromal vertical striae (Figure 1a and b) and shallow anterior chambers. Hertel exophthalmometry was 21 mm bilaterally. Ishihara colour vision and visual fields to confrontation were normal.

Gonioscopy confirmed the angle grade as 0-I in all quadrants using Schaffer's method. Her intraocular pressures (IOPs) in primary position were right 22 mm Hg and left 25 mmHg with further increase in upgaze (right 28, left 31 mmHg). She was started on topical latanoprost to both eyes.

A few months before her referral she had undergone bilateral 11/2 wall endoscopic orbital decompression for bilateral optic nerve compression. The corneal striae had also been noted at that point.

Orbital computed topography was performed revealing adequate bony decompression but increased rectus muscle bulk on both sides. In view of increased muscle bulk and risk of further compressive optic neuropathy from increased orbital pressure (as evidenced by anterior segment findings), she underwent a three-wall external orbital decompression followed by orbital radiotherapy (20 G in 12 fractions).

After her surgery, visual acuities were 6/9 (pinhole 6/6) bilaterally and anterior segment examination revealed minimal corneal punctate epitheliopathy with reversal of all corneal striae. The anterior chamber also deepened (grade II–III in all quadrants). The IOPs were controlled on topical latanoprost (right 18, left 18) and dilated funduscopy confirmed normal discs and retina. There was no change in her refraction at any point.

Comment

Corneal disease in TED has been described in the context of tear film instability, lagophthalmos, exposure keratitis, and corneal astigmatism.¹

The Oriental upper eyelid has subcutaneous, suborbicularis, and pretarsal fat components. The orbital septum is tighter compared to the Caucasian orbits with the orbital septum fusing with the levator aponeurosis below the superior tarsal border.²

Our patient had enlarged extraocular muscles, mild proptosis, a tight orbital septum and we feel that the

Figure 1 Corneal striae in both left (a) and right eye (b).

resultant high orbital pressure induced globe compression. In the presence of a tight orbital septum, the increased orbital pressure may manifest as corneal striae.

Striae results from the whole cornea swelling and buckling back upon itself causing vertical folds or lines in the Descemet's membrane.³ In this case the corneal changes reversed with further orbital decompression, which opened the septum and enlarged the orbital bony cavity thus reducing the orbital pressure. Clinicians should be aware of this sign in TED as an indicator of a tense orbit in the absence of marked proptosis.

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Sir, Bilateral acetazolamide-induced choroidal effusion following cataract surgery

Idiosyncratic reactions to drugs including sulfonamide^{1,2,6} and corticosteroids^{3,4} have been reported to cause choroidal detachment and secondary angle closure glaucoma. We report a case of bilateral angle closure glaucoma and extensive choroidal effusion following administration of oral acetazolamide unresponsive to intravenous (i.v.) dexamethasone immediate postcataract surgery.

Case report

A 66-year-old man with chronic open angle glaucoma underwent left routine cataract surgery and intraocular lens implant under local anaesthesia. He received oral acetazolamide 250 mg overnight and immediately following the surgery. Three hours after surgery, he complained of severe pain in the operated eye. On examination the left eye (LE) showed corneal oedema shallow anterior chamber (AC), pupillary capture from 4 to 7 O' clock hours (Figure 1b) and an intraocular pressure (IOP) of 52 mm Hg. The right eye (RE) showed circumcorneal congestion, corneal oedema shallow AC with an immature cataract (Figure 1a), and IOP of 40 mm Hg. Gonioscopy showed 360° appositional angle closure with a convex iris configuration. He was treated with i.v. mannitol 2 mg/kg and oral acetazolamide 250 mg t.i.d., G.Timolol 0.5% bd, G Ofloxacin 0.3%, and G dexamethasone 1% q.i.d. The next day, the corneal oedema and shallow AC persisted with an IOP of 32 mm Hg in each eye. Ultrasound B-scan showed bilateral choroidal effusion (Figure 2a and b). Acetazolamide was thought to be the cause and was discontinued. i.v. dexamethasone 8 mg daily was started in an attempt to reduce inflammation related to the choroidal effusion but no positive effect therefore was discontinued. On day 5, the choroidal effusion showed resolution (Figure 2c