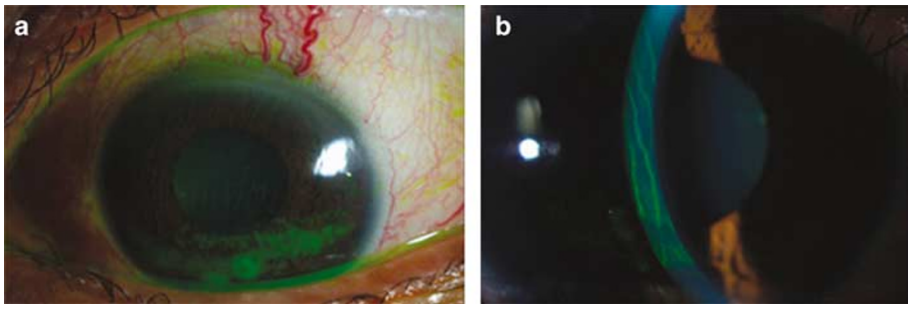

Figure 1 Corneal striae in both left (a) and right eye (b).

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 choridal 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

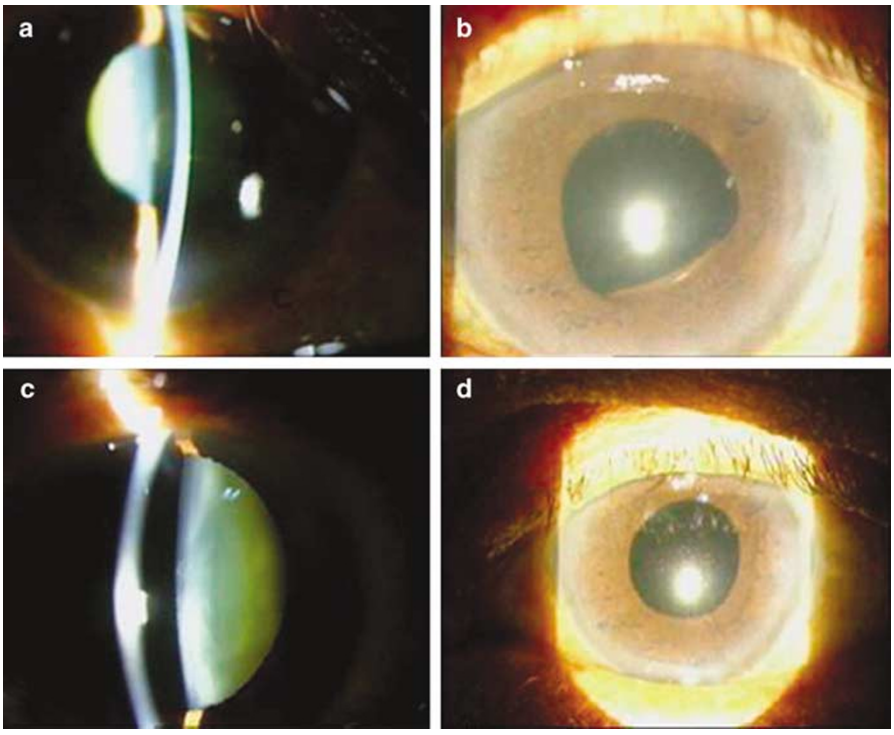

Figure 1 (a) RE first postoperative day showing corneal oedema and shallow AC. (b) LE first postoperative day showing corneal oedema and pupillary capture. (c) RE fifth postoperative day showing clear cornea and normal AC depth. (d) LE fifth postoperative day showing clear cornea, resolved pupillary capture, and deep AC.

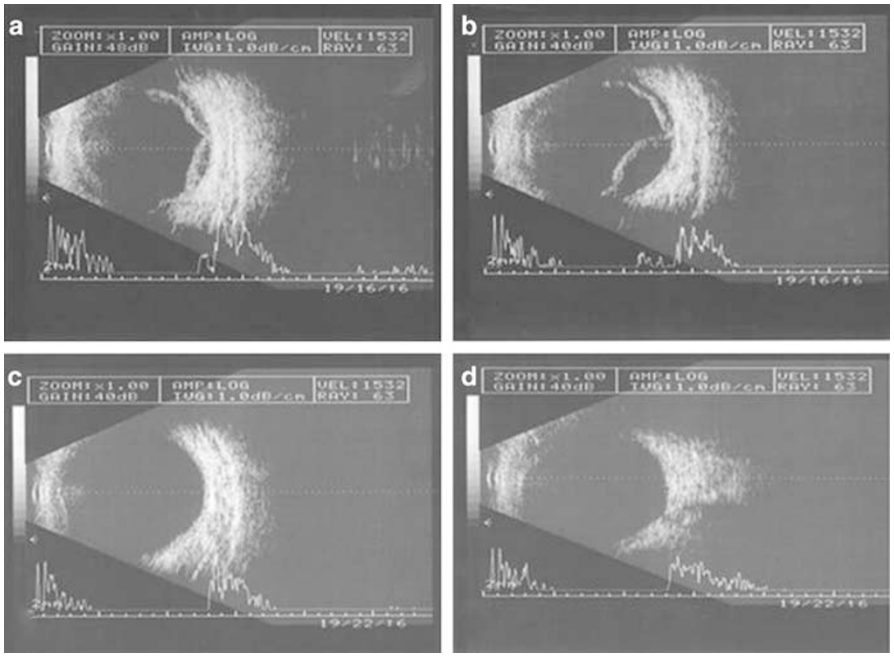

Figure 2 (a) First postoperative day showing peripheral choroidal effusions in the superiotemporal and nasal quadrants. (b) LE 270° effusions extending anteriorly to the equator. (c and d) Fifth postoperative day showing resolved choroidal effusion in both eyes.

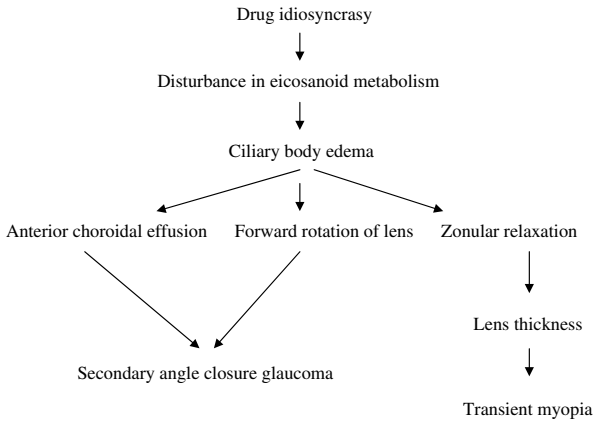

Figure 3 Flow chart for the mechanism of drug-induced secondary angle closure glaucoma.

and d) with clear cornea, normal AC depth and IOP of 18 mm Hg in each eye (Figure 1c and d). He achieved final best-corrected visual acuities of 6/6.

Comment

Acetazolamide-induced transient myopia and angle closure glaucoma has been reported in nonglaucomatous patient.[1,2,6] Forward shift of crystalline lens caused by ciliary body oedema has been postulated as the mechanism.[1,2,5] Ciliary body oedema has been based on an imbalance in eicosanoid (prostaglandin–thromboxane) metabolism (Figure 3).[5]

In this patient, the cloudy cornea precluded laser iridotomy and cycloplegia was avoided as angle closure was a differential diagnosis. Dexamethasone was used empirically as an inflammatory aetiology was suspected.

The failure of laser iridotomy and cycloplegia was reported as the mechanical effect following forward rotation of ciliary body and displacement of iris–lens diaphragm secondary to choroidal effusion.[6] Only when the latter resolved could the angle be opened. Corticosteroids have been shown to cause choroidal effusion[3,4] and are ineffective in managing this complication. Other methods of controlling IOP might have been considered to avoid this unusual complication.

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Sir,

Angle-closure triggered by orgasm: a new provocative test?

Case

Sexual arousal has been reported to lead to angle-closure glaucoma[1] and to blurred vision in women with narrow angles.[2] We treated a 34-year-old Caucasian woman with uniocular pain, blurred vision, and haloes since 2 days. She had a past history of recurrent episodes occurring immediately after orgasm, resolving spontaneously and that occurred only when she was having sex in the dark while in the top position, with her upper torso prone. Her mother and maternal grandmother had had angle-closure.

On examination, best-corrected acuity was 20/25 OU with +5.00 sph +1.00 cyl × 90 OD and +5.25 sph +1.75 cyl × 85 OS. Intraocular pressure was 12 mm Hg OD and 17 mm Hg OS. Gonioscopy revealed plateau iris configuration OU and appositional angle-closure OD with scattered peripheral anterior synechiae and a slit angle OS. Axial lengths were 20.72 mm OD and 20.38 mm