pigment epithelial mottling and retinal depigmentation and deposits are well described.[12,13] Retinal involvement has been seen as early 3 years of age but is a constant feature from 7 years onwards.[8] Reduced a- and b-wave amplitudes on scotopic and photopic electroretinograms are described in some patients[8,13] and may progress with age.

In this patient, the corneal crystals were initially deemed to be the cause of his visual problems. The cystine stores in non-renal tissues are said to be of little consequence until about the age of 10 years,[12] but this child, although only 5 years of age, has severe visual loss due to retinopathy from cystine deposition. In the series published by Yamamoto *et al.*,[12] all 27 cases with nephropathic cystinosis had vision better than 6/12. The age of the cases ranged from 2 to 19 years. There is no known therapy for the retinopathy and, despite renal transplant, retinopathy is known to progress.[9,12] This case serves to remind us that patients with cystinosis and visual failure should be investigated for posterior segment involvement at all ages. It is particularly important to ascertain the cause of poor vision before embarking on corneal surgery.

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Sir,

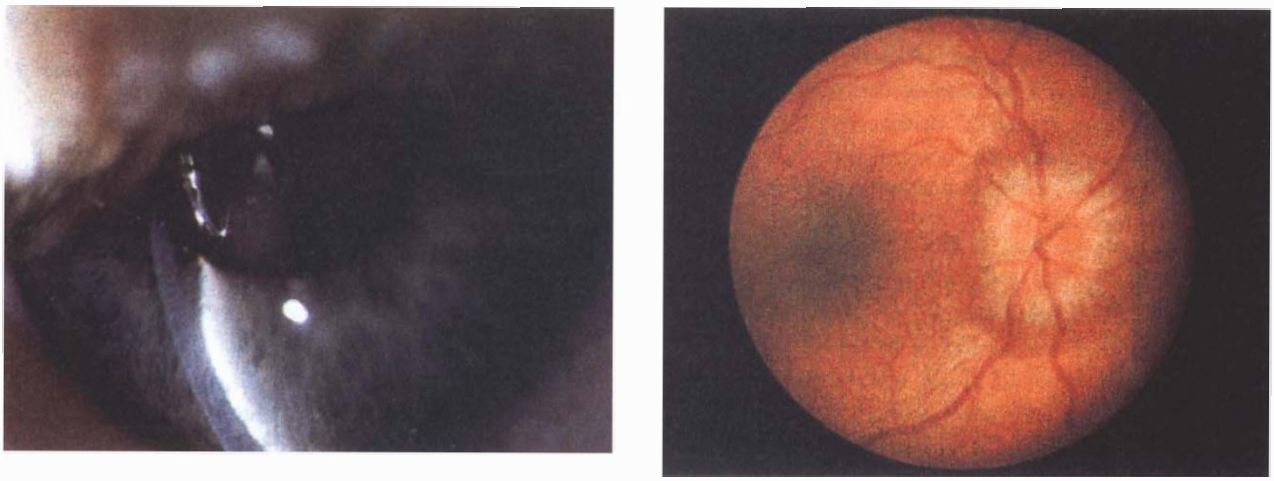
The application of ultrasonic biomicroscopy in the management of traumatic hypotony

Early diagnosis and prompt management of hypotony are important. We report a case of persistent hypotony after primary repair of scleral perforation. The management was guided by the ultrasonic biomicroscopy (UBM) findings.

Case report

A 13-year-old boy presented to another unit with a ruler injury resulting in a temporal scleral perforation, vitreous prolapse, vitreous haemorrhage and hyphaema. After a primary repair, he developed persistent hypotony and presented to our unit 1 month later. His best corrected visual acuity (VA) was 6/24. The anterior segment findings included a mild relative afferent pupillary defect, a shallow anterior chamber (AC) associated with angle closure of the inferotemporal quadrant without any evidence of cyclodialysis on gonioscopy, a vitreous prolapse, a temporally subluxated lens, an intraocular pressure (IOP) of 4 mmHg and mild posterior subcapsular lens opacification (Fig. 1a). The fundus showed a swollen optic disc, maculopathy and tortuous vessels (Fig. 1b). UBM imaging demonstrated 360° of supraciliary effusion (Fig. 2a).

He underwent drainage of suprachoroidal fluid and resuturing of a ciliary body with 6/0 polyglactin sutures through full-thickness sclerotomies (two per quadrant) 3. 5 mm from the limbus. Sulphur hexafluoride (SF_6) gas (20%) was injected into the AC as an internal tamponade. Post-operatively, the IOP was transiently maintained for 1 week only. He had a further operation 2 months later, which involved direct cyclopexy[1] of two main supraciliary effusion areas (as demonstrated by UBM pre-operatively) and injection of 20% SF_6 gas into the AC. A transient IOP rise was treated medically in the first 2 weeks post-operatively. He subsequently underwent anterior vitrectomy, phacoemulsification with a Morcher ring and silicone lens implantation 10 months after the original injury. His latest best corrected (VA) was 6/6



(a) (b)

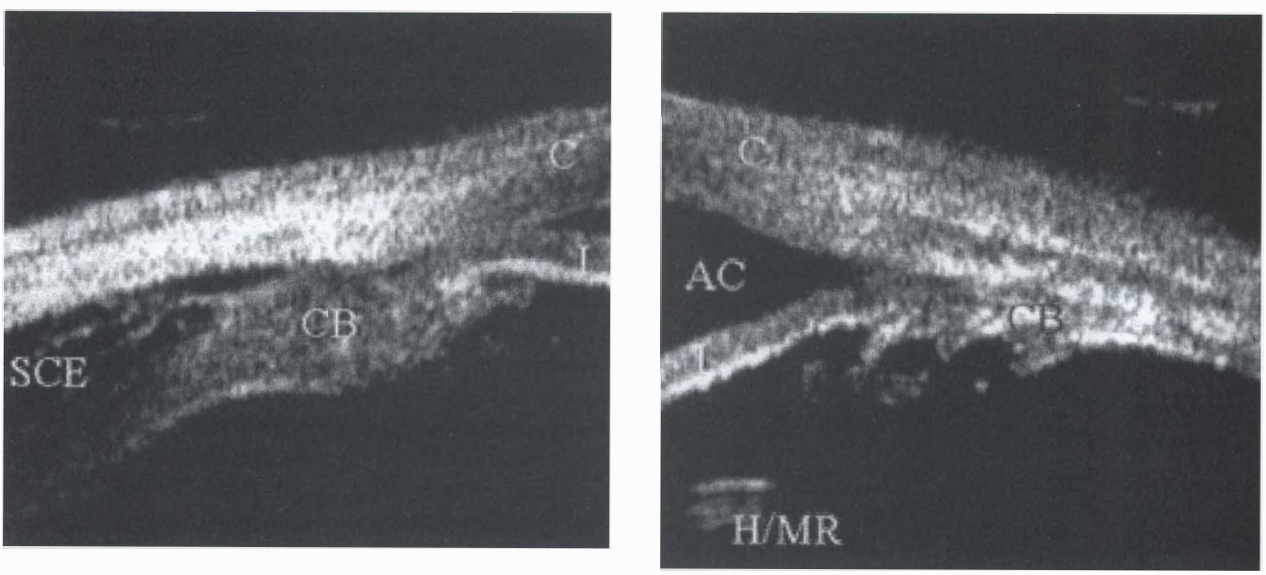
Fig. 1. *(a) The anterior segment photograph demonstrated the shallow anterior chamber in the inferotemporal quadrant, vitreous prolapse and subluxated lens temporally. (b) The fundus photograph showed the swollen optic dis, maculopathy and tortuous vessels.*

part (refraction: –2.0 DS/+2.5 × 97°), and follow-up UBM imaging performed 18 months later (Fig. 2b) showed complete reattachment of the ciliary body.

Comment

Hypotony is due to a combination of increased uveoscleral outflow and decreased aqueous production by the ciliary body detachment.[1–4] Diagnosis of cyclodialysis by gonioscopy can be difficult and misleading, especially in recently traumatised or operated eyes with opaque media secondary to corneal oedema or hyphaema. Gentile *et al.*[5] reported the application of UBM in the diagnosis of traumatic cyclodialysis in six patients. Two patients with hyphaema and four with abnormal iris architecture failed to have a cyclodialysis demonstrated by gonioscopy technique. Conventional B-scan ultrasonography failed to demonstrate any collections of ciliochoroidal fluid. One patient's cyclodialysis was not detectable by immersion B-scan ultrasonography.

The management of persistent cyclodialysis is time and size dependent. Trial of atropine[6] was suggested as initial medical treatment; if this fails, argon laser treatment to the cyclodialysis[6,8] or external YAG cyclophotocoagulation[9] may be attempted. Surgical options include cyclopexy by directly suturing the ciliary body to the scleral spur under a scleral flap, as recommended by Kuchle *et al.*[7] Other techniques reported include cryocoagulation,[10] pars plana vitrectomy with gas tamponade and cryotherapy,[11] but the majority of these reports involved small series of anecdotal case reports.



(a) (b)

Fig. 2 *(a) UBM imaging demonstrated 360° of extensive supraciliary effusion. C, cornea; I, peripheral iris; CB, ciliary body; SCE, supraciliary effusion. (b) Follow-up UBM imaging showed complete reattachment of the ciliary body. C, cornea; I, peripheral iris; CB, ciliary body; AC, anterior chamber; H/MR, haptic of intraocular lens and Morcher ring complex.*

In our patient, the initial surgical attempt by transscleral ciliary body sutures and SF_6 gas treatment managed to close the cyclodialysis only partially. The true extent of the residual cyclodialysis was only revealed by UBM. The remaining areas of cyclodialysis were subsequently closed by direct cyclopexy guided by UBM, and this maintained long-term IOP control.

The application of gonioscopy is helpful when the media are clear, and with the use of viscoelastic to deepen the anterior chamber and repeat gonioscopy perioperatively, may be useful for more accurate localisation of cyclodialysis. UBM technique would complement gonioscopy examination and is of particular value in opaque media. It would also help to identify the posterior extension of the cyclodialysis.

In vitro assessment of capscular tension rings[12] in monkeys confirmed maintenance of the circular contour of the capsular bag and centration of the intraocular lens. The application of corneal phacoemulsification surgery and a capsular tension ring for the subluxated cataract resulted in an excellent visual outcome in our patient.

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Sir

Bilateral pre-retinal macular haemorrhage following blunt head injury

Pre-retinal macular haemorrhage (PRMH) is a cause of severe visual loss most commonly seen in association with proliferative diabetic retinopathy. Other causes of retinal haemorrhage associated with a rise in intraocular hydrostatic pressure include Terson's syndrome and Valsalva retinopathy. We report a patient in whom bilateral PRMH was caused by a blunt head injury.

Case report

A 34-year-old man presented with acute loss of vision after landing on the vertex of his head, having been thrown from a horse. Although suffering a loss of consciousness for several minutes, he had no external injury and a normal brain scan. He described a lower paracentral scotoma on the right and a central 'red' scotoma on the left. Visual acuities were 6/4 right and 6/24 left. The only ocular abnormalities were bilateral PRMH with the right lesion being eccentric superiorly, allowing foveal sparing initially (Fig. 1). Over the next 2 weeks the right PRMH moved down over the fovea and both became more fluid, with red blood cell (RBC) sedimentation (Fig. 2). The variable RBC levels caused subjective visual fluctuation and at 1 month the visual acuity was 6/60 both eyes. Extensive spread of RBCs into the right vitreous was noted at this stage and corresponded with a more rapid absorption than on the left at 2 months (Fig. 3). The vision had recovered to 6/5 bilaterally at 3 months and 6/4 bilaterally at 5 months.

Comment

The head-first deceleration in this case would have caused a sudden increase in retinal venous pressure. This is the presumed mechanism of haemorrhage in other causes of bilateral PRMH due to rupture of retinal capillaries. These capillaries must be superficially placed, as there is often no associated intra-retinal haemorrhage visible, as in this case. Bilateral PRMH has been described in both battered-baby syndrome[1] and Purtscher's retinopathy,[2] although retinal haemorrhages and cotton wool spots around the optic nerve are the more common finding in both. A vascular abnormality in the typical Purtscher's retinopathy has been shown in the choroid,[3] while retinal venous beading has been described in association with PRMH.[4]

Duane described PRMH following the Valsalva manoeuvre.[5] This typically takes several weeks to months to make what is usually a full visual recovery, often with development of a fluid level,[6] as in this case. Gabel *et al.*[7] showed that YAG laser photodisruption of