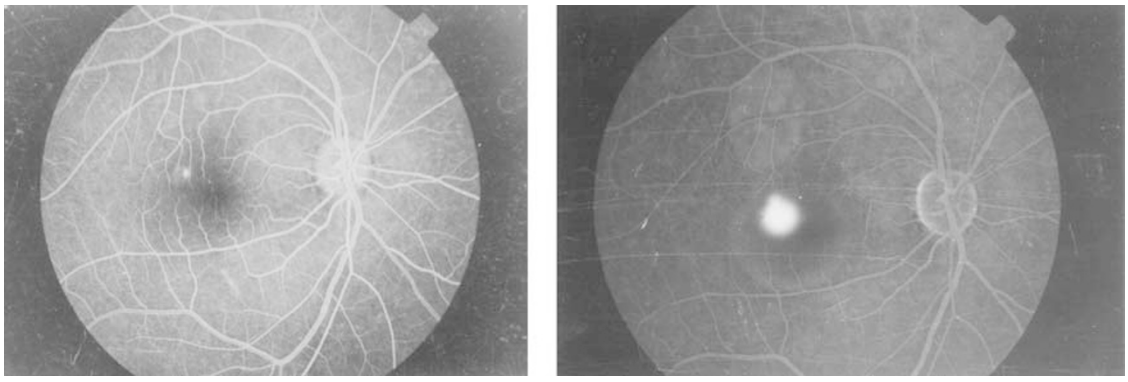

Figure 1 Early (left) and late (right) phases of fundus fluorescein angiogram showing a leak typical of CSR in the right eye at presentation.

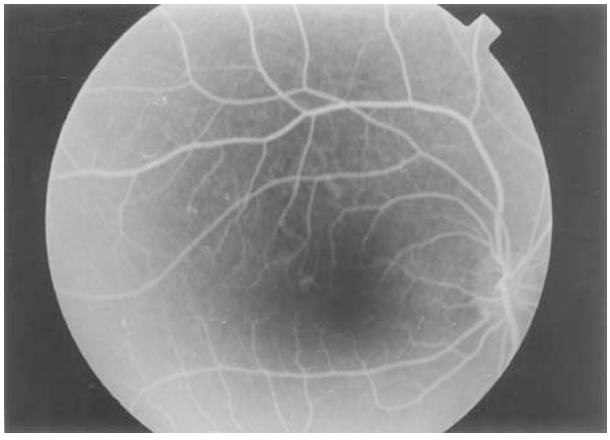

Figure 2 Fluorescein angiogram of right eye 12 weeks after stopping oral steroids showing a small window defect.

ionic pump function or by altering the permeability of the blood aqueous barrier and disrupting the outer blood retinal barrier.[3] Retrolental flare with few cells observed in this case could have been owing to breakdown of the blood aqueous barrier.

This case raises the question of whether prophylactic steroid therapy for sympathetic ophthalmia is justified particularly when it has been reported not to be foolproof.[4] It highlights the importance of considering CSR masquerading as sympathetic ophthalmia in such situations. Clinical improvement following tapering of steroids supports the diagnosis.

We wish to emphasise the importance of fundus FA in investigating patients at risk for sympathetic ophthalmia who present with diminished vision in the fellow eye. Ophthalmologists must be aware of the possibility of CSR precipitated by stress and steroids as in this case, as the management of CSR is completely different from that of sympathetic ophthalmia and the use of corticosteroids in such a case can be detrimental.

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Sir,

Traumatic optic nerve avulsion: role of ultrasonography
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Optic nerve avulsion is a rare presentation of ocular trauma. The diagnosis of this entity is often obscured by the presence of concomitant vitreous haemorrhage, which precludes the visualization of optic head excavation. Various electrodiagnostic and expensive neuroimaging studies have not proved helpful in substantiating the diagnosis of this entity in the early stages and may not be readily available in most of the

centres. We herein present the two cases of traumatic optic nerve avulsion diagnosed on B-scan ultrasonography before the ophthalmoscopic picture became clear. The role of ocular ultrasonography, readily available with ophthalmologists, and relatively inexpensive imaging in the diagnosis of suspected optic nerve head avulsion has been highlighted.

Case report 1

A 27-year-old male presented to us with a sudden loss of vision in the left eye following blunt trauma with the hook of a crane at the construction site. On examination, 6 h after the injury, visual acuity in the left eye was no light perception and in the right eye 20/20 without correction. There was an upper lid laceration of about 6 cm. The left pupil was unresponsive to light and showed an afferent pupillary defect. The right pupil reacted normally to direct light. Intraocular pressures were normal. Examination of the right eye showed a normal anterior segment and fundus. The left eye showed an inferonasal subconjunctival haemorrhage and mild chemosis. The sclera was intact. The cornea and lens were clear and the anterior chamber contained a few pigmented cells. Posterior segment examination revealed the presence of vitreous haemorrhage inferiorly. The optic nerve head was obscured by the overlying haemorrhage. There was extensive retinal opacification in the posterior pole and a cherry red spot was present. The arteries were attenuated, whereas veins contained segmented blood column (Figure 1a). Optic nerve avulsion was suspected.

The patient belonged to poor socioeconomic strata and therefore could not afford expensive investigations like computed tomography (CT) and magnetic resonance imaging (MRI). A greyscale B-scan ultrasound with high-frequency linear probe (9.5 MHz) was performed. It demonstrated vitreous haemorrhage overlying the site of optic nerve insertion. The optic nerve was not seen to reach the optic disc and an area of hypolucency was seen anteriorly, just posterior to the optic nerve head (Figure 2). The diagnosis of optic nerve avulsion was made on the basis of these ultrasonographic findings. X-ray of the left orbit did not show any fracture or foreign body. Visual evoked response (VER) was unrecordable in the left eye but normal in the right eye.

The patient continued to have no light perception in the left eye. At 2 months after the injury, fibrovascular tissue obscured the optic nerve head and extended into the vitreous.

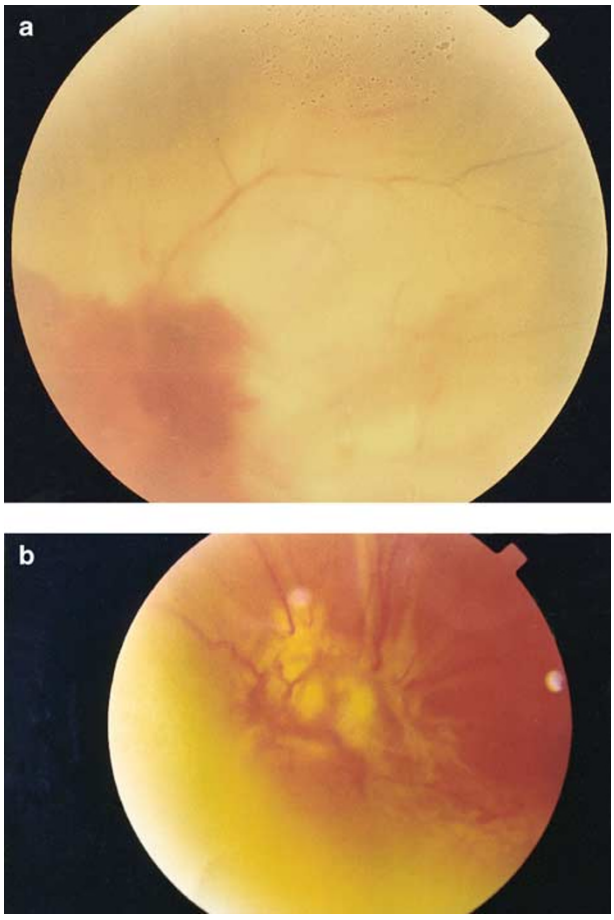

Figure 1 Fundus photograph of: (a) case 1 showing vitreous haemorrhage overlying optic nerve head and central retinal artery occlusion and (b) case 2 showing 360° excavation of optic nerve head and curling of vessels upon themselves failing to reach the optic nerve head.

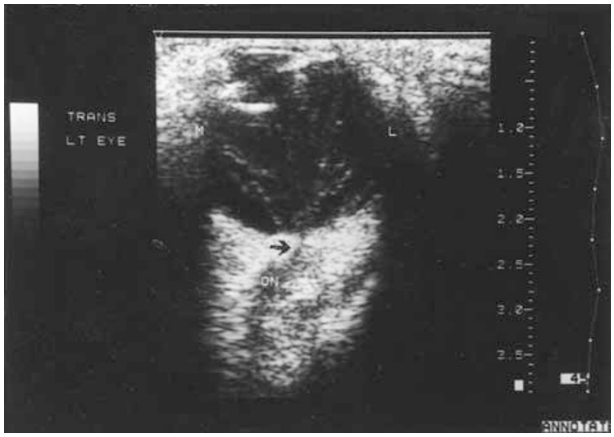

Figure 2 B-scan photograph of case 1 showing hypolucency (small arrow) just posterior to the optic nerve head.

Case report 2

A 17-year-old male presented with light perception only in the left eye a week after being hit by a wooden object accidentally. Ocular examination revealed repaired left lower lid laceration with traumatic ptosis. Extraocular movements were full. Slit-lamp examination showed 4+ anterior chamber reaction with fixed dilated pupil,

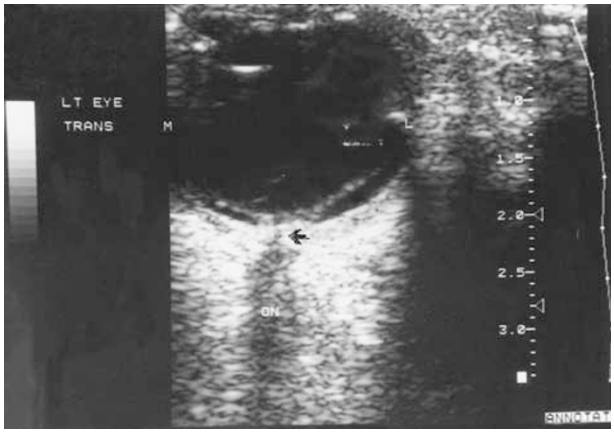

Figure 3 B-scan photograph of case 2 showing hypolucency (small arrow) just posterior to the optic nerve head.

cataractous and subluxated lens with irododonesis. Intraocular pressure was 30 mmHg. Indirect ophthalmoscopy revealed dense vitreous haemorrhage. Relative afferent pupillary defect was present in the left eye with absent consensual light reflex in the right eye. Diagnosis of closed globe injury of the left eye with traumatic indirect optic neuropathy was made. CT scan performed just after the injury and before presentation to us revealed no bony fragment impinging on optic nerve in the optic canal. No other abnormality in optic nerve was noted. X-ray orbit showed intact bony orbit. Patient's vision decreased to no light perception over 6 days. The intraocular pressure during this period was consistently high despite all antiglaucoma medication. VER of the left eye showed no wave formation. Intravenous methylprednisolone was planned as per the recommendations of the Extracranial Optic nerve Decompression meeting, Boston, 1993. Meanwhile, ultrasonography revealed superotemporal retinal detachment, few echogenic shadows just in the front of the optic nerve. Also, a small tear of the optic nerve just posterior after optic nerve head was seen as an area of hypolucency (Figure 3). With the clearance of vitreous haemorrhage, indirect ophthalmology revealed superotemporal retinal detachment with 360° excavation of optic nerve head as classically described in optic nerve avulsion with vessels failing to reach the optic nerve head and curling upon themselves to obscure optic nerve head (Figure 1b). These findings were consistent with ultrasonography findings, and a final diagnosis of traumatic optic nerve avulsion was made and the patient was managed conservatively.

Comment

In optic nerve avulsion, the optic nerve is forcibly disinserted from the retina, choroid, and vitreous, and the lamina cribrosa is retracted from the scleral rim. Both complete and partial avulsions have been described. Optic nerve avulsion usually results when an object intrudes between the globe and the orbit wall and displaces the eye. Several mechanisms have been postulated; sudden extreme rotation of the globe, sudden rise in intraocular pressure leading to the expulsion of nerve out of scleral canal or sudden anterior displacement of the globe.

The diagnosis of optic nerve avulsion is quite apparent if the media is clear. The fundus examination in such cases shows an excavation in the disc area. The diagnosis can only be suspected and not confirmed if disc area is obscured by vitreous haemorrhage. It is essential to confirm the diagnosis so that the patient may not be subjected to unnecessary treatment such as optic nerve decompression or high-dose steroids.

Various diagnostic modalities like electrodiagnostic tests, CT scan, MRI and fluorescein angiography have been used without much help. CT scanning has demonstrated complete or partial optic nerve avulsion in some cases and intact optic nerve sheath without evidence of disruption in others. Roth and Warman[1] have described a patient with optic nerve avulsion after being struck in the eye by a golf club. CT of brain and orbits revealed no abnormalities. Foster *et al*[2] carried a study to characterize the presentation, role of diagnostic imaging in patients with optic nerve avulsion. Six patients underwent neuroimaging including CT and MRI or both. Separation of optic nerve from the globe was suggested in only one case by an area of hypolucency at the junction of the nerve and globe, and a linear hyperlucency a little posteriorly consistent with a retrodisplaced lamina cribrosa. MRI was also used by the authors in two cases that, however, failed to demonstrate optic nerve avulsion. In one case, complete optic nerve avulsion was found when the globe was surgically explored. Fluorescein angiography findings may be useful in eyes with clear media only. Hart and Pilley[3] evaluated a patient with avulsion of superior segment of the optic disc, and found partial occlusion of the superior retinal vein and the absence of capillary filling in the upper half of the optic disc.

Previous reports of ultrasonography in the diagnosis of optic nerve avulsion have shown encouraging results.[4–6] Talwar *et al*[4] reported a posterior ocular wall defect in the region of the optic nerve head characterized by hypoechoic defect. A-scan showed a marked widening of the nerve suggesting haemorrhage and oedema within the nerve sheath in addition to optic nerve avulsion.

In both our cases, ultrasonography of the globe showed the hypoechoic defect in the posterior ocular coat implying complete avulsion. There were, however, no associated findings of swelling of optic nerve sheath with

an increase in the optic nerve diameter[5] or optic nerve sheath haemorrhage[6] that correlated with the anatomic features of an avulsed optic nerve head in the presence of overlying vitreous haemorrhage, as is frequently the case. These two cases amply demonstrate that ultrasonography is a helpful tool in the diagnosis of optic nerve avulsion obscured by overlying vitreous haemorrhage and a reasonably better alternative to expensive and less readily available neuroimaging studies.

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Sir,

Granulomatous anterior uveitis associated with 0.2% topical brimonidine

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Brimonidine tartrate 0.2% is an α_2 adrenergic receptor agonist used in the treatment of open-angle glaucoma and ocular hypertension. Granulomatous anterior uveitis in association with topical brimonidine has been reported in only five patients since its introduction.[1,2] We report another case of bilateral granulomatous anterior uveitis developing as a late side effect of topical brimonidine therapy, providing further evidence of this potentially sight-threatening complication.

Case report

A 79-year-old woman presented in February 2002 with bilateral granulomatous anterior uveitis. She had no prior history of uveitis and was in good general health. Following diagnosis with advanced primary open-angle glaucoma in 1998, she was started on topical timolol 0.25% twice daily to both eyes. Adjunctive treatment with topical latanoprost 0.005% to both eyes at night was initiated 1 year later. In August 2000, this was changed to brimonidine 0.2% instilled twice daily to both eyes, as the intraocular pressures were inadequately controlled. Treatment with timolol continued. The patient subsequently developed bilateral age-related macular disciform degeneration, reducing her vision to hand movements right and 4/60 left.

At presentation, 18 months after starting treatment with brimonidine, the patient gave a 4-week history of increasingly sore and uncomfortable eyes. As her vision was so poor, she was unable to see how injected her eyes were and had continued to instil her medication while awaiting her routine follow-up appointment. She was found to have severe conjunctival injection bilaterally, with florid conjunctival follicles. There were mutton-fat keratic precipitates, 2+ anterior chamber cells, posterior synechiae and a few iris nodules bilaterally (Figure 1). Intraocular pressure was elevated to right 22 mmHg and left 40 mmHg. There was no posterior segment

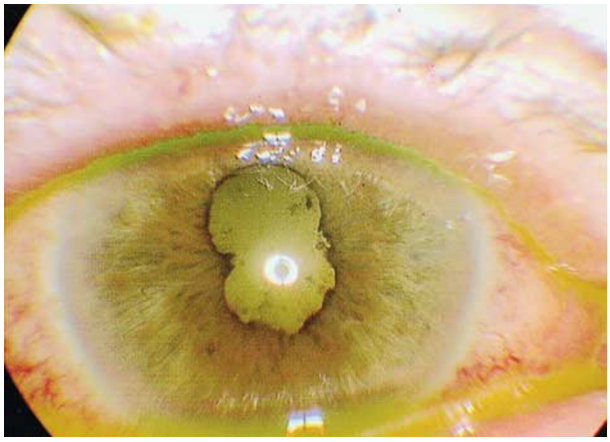

Figure 1 Colour photograph demonstrating granulomatous keratic precipitates and posterior synechiae.