CASE REPORT

Bilateral angle recession following occiput injury

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Abstract

Angle recession is a common complication of blunt eye injury, but its occurrence without direct eye injury is rare. We report a case of bilateral angle recession secondary to occiput injury, without direct impact to the eyes. A 56-year-old patient presented to us with bilateral blurring noted after contusion to the occiput, without direct injury to the facial region. Ophthalmic examination revealed bilateral microhyphema with 360-degree angle recession. The findings in this patient highlight the importance of gonioscopic examination in patients with blurring after blunt injury to the head region.

Key words: Craniocerebral trauma; Glaucoma, open-angle; Hyphema

Introduction

Angle recession is a common complication of blunt eye injury.[1] However, its occurrence without direct eye or periorbital trauma is rarely reported. We describe an unusual patient with bilateral angle recession secondary to occiput injury without direct impact to the eyes. To the best of our knowledge, this is the first case report documenting angle recession due to such a mechanism of injury.

Case report

A 56-year-old man was hit over the occiput by a broken cable that swung towards him accidentally, while working on a boat, and immediately noticed bilateral blurring of vision. There was no loss of consciousness, nor did he sustain a fall. Moreover, there was no direct trauma to the eyes or the front part of the head, nor was there any shaking of the head.

Around 14 hours after the injury, he presented to the Accident and Emergency (A&E) department of our hospital and was assessed by our team. On presentation his vision was 0.2 Snellen (20/100) in the right eye and 0.1 Snellen (20/200) in the left eye. Intraocular pressure was 11 mm Hg in the right eye and 18 mm Hg in the left eye. There were mild bilateral periorbital bruises and a subconjunctival hemorrhage was evident in the left eye. Slit-lamp examination showed microhyphema in both eyes. There was an iris sphincter tear at 2 o'clock in the left eye (**Figure 1**). In dim light, the pupil size was 4 mm in the left eye and 3 mm in the right eye. The 1-mm anisocoria (pupil diameter inequality) was also present in bright room light. Both pupils were reactive to light, and there was no relative afferent pupillary defect. Gonioscopy showed 360-degree angle recession with small blood clots in the angle in both eyes (**Figures 2a-b**). Fundal examination showed a small patch of commotio retinae in the right eye, which did not affect foveal region. There was no vitreous hemorrhage or retinal tear. The optic discs were normal with cup-disc ratios of 0.3. On presentation to the A&E department, the patient was conscious and alert. Physical examination by the attending doctor at the A&E department revealed erythema over occipital region, but no scalp wound. The cervical spine was non-tender. Brain computed tomography was unremarkable and did not show any focal lesion, intracranial hemorrhage, or orbital fracture. The patient was prescribed 1% prednisolone acetate eye drop every 2 hours and chloramphenicol eye drop 4 times a day to both eyes and 1% cyclopentolate eye drop 4 times per day to left eye, and instructed to remain propped up at bedtime. On day 9 post-injury, pinhole-assisted visual acuity was 0.6 Snellen (20/32) in both eyes with reduction in the extent of microhyphema. Fundal examination was unremarkable with normal optic discs and resolution of commotio retinae in the right eye. Ultrasonographic biomicroscopy (UBM) and anterior segment optical coherence tomography confirmed 360-degree angle recessions in both eyes (**Figure 3**).

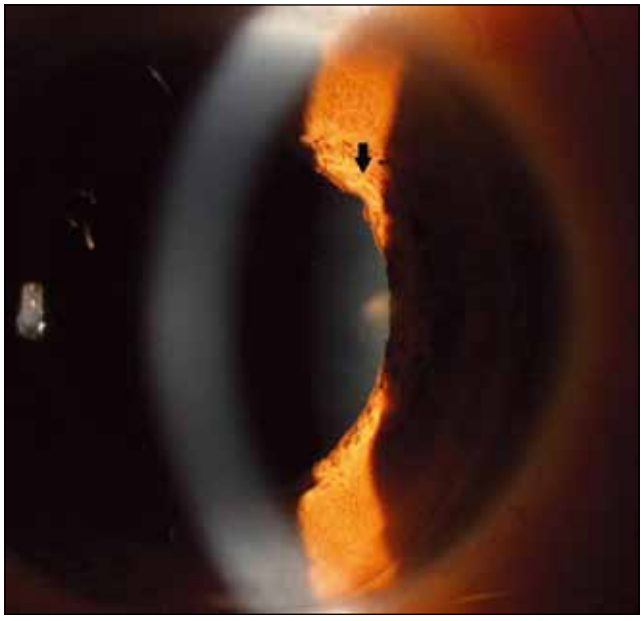

Figure 1. Slit-lamp photograph of left eye showing a sphincter tear at 2 o'clock.

The microhyphema in both eyes had resolved completely 2 months post-injury. The patient was followed up every 4 to 6 months. At the last follow-up 1 year post-injury, bilateral IOPs were normal (ranging from 10 to 14 mm Hg) and no evidence of glaucomatous disc change were noted. Visual acuity at the last visit was 0.7 and 0.6 Snellen for right eye and left eye, respectively.

Discussion

Depending on severity, the gonioscopic appearance of angle recession may vary from darker and wider ciliary bands with whiter scleral spurs in shallow angle tear, to deep fissures extending into the ciliary body in deep angle tears.[2] A retrospective survey by Canavan and Archer[1] in 1982 showed that angle recession was present in 80.5% of patients with eye contusions.

In this case, the angle recession occurred without direct trauma to the ocular or the orbital area, and there was a clear temporal relationship between the occipital head injury and onset of ocular symptoms. In our patient, the diagnosis of angle recession might have been missed, had his vision not been affected by the microhyphema. Comparison of the 2 eyes may not be helpful in diagnosing 360-degree angle recession in this patient because of bilateral involvement. Characteristic gonioscopic appearance and the presence of blood clots in the angles of both eyes suggested bilateral angle recession. While gonioscopy is the reference standard for anterior chamber assessment, UBM using high-frequency (50 to 100 MHz) transducers may also provide more detailed imaging of the anterior segment than traditional B-scan ultrasound.[3]

In blunt eye injury, the line of force traversing the eye may

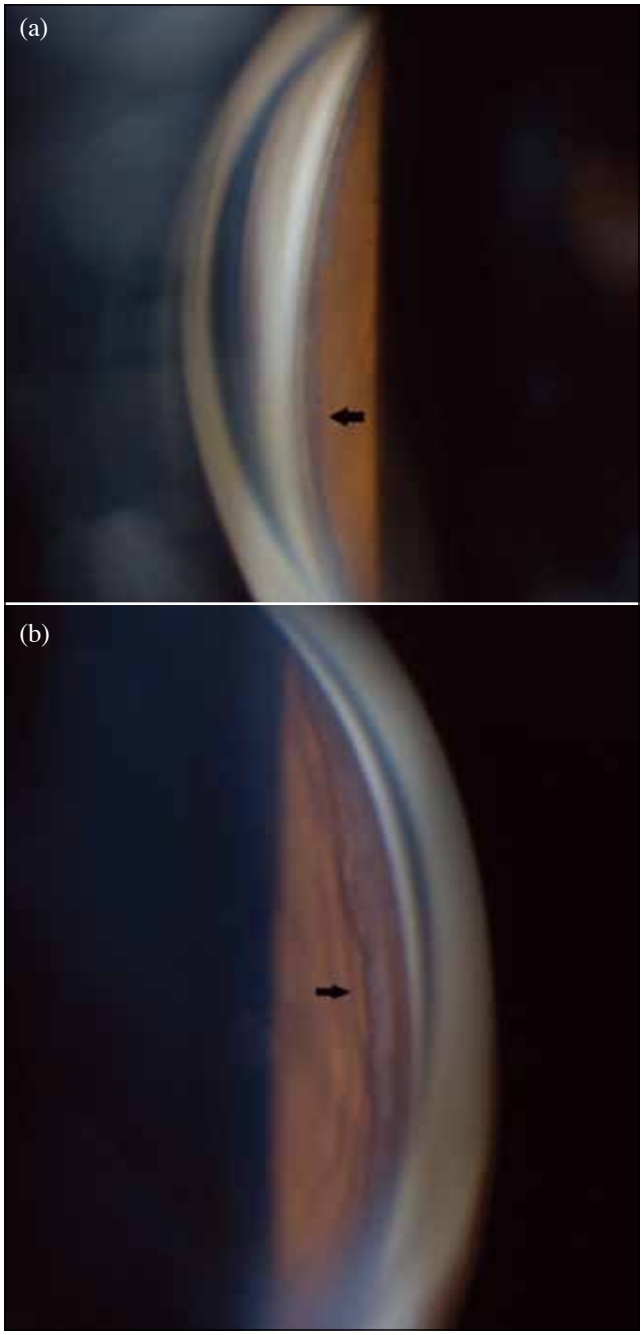

Figure 2. Gonioscopy photographs showing widening of the ciliary band in the nasal angle of (a) right eye and (b) left eye.

Figure 3. Ultrasonographic biomicroscopic image of nasal angle recession in the left eye.

lead to damage at all interfaces, causing contrecoup injury.[1,4] The impact force causes anteroposterior compression of the globe and forces the aqueous backwards and laterally, exerting traction on the relatively unsupported iris diaphragm and the anterior chamber angle, thus resulting in tears in the ciliary body.[1,5] The same mechanism may have applied to our patient, despite the direction of the external force being postero-anterior. The bilateral 360-degree angle recession in this case also suggests that the force was transmitted relatively uniformly to the 2 eyes. We postulate that such transmission of force to the anterior parts of the eye and orbital area may also account for the periorbital ecchymosis in both eyes and the subconjunctival haemorrhage in the left eye. Although uncommon, this postulate regarding the transmission of force to the eyes from a remote skull injury is also consistent with other reported observations. Papageorgiou et al[6] reported delayed optochiasmal transection secondary to minor occiput injury due to transmission of compression waves, in the absence of an external wound at the impact site, orbital fracture, or intracranial hemorrhage. In another report by Brown and Lissner,[7] a presumed lamina papyracea fracture with surgical emphysema was also observed after occiput injury, in the absence of a basal skull or floor fracture. For this phenomenon, a similar 'seismic' mechanism was proposed, whereby the force applied to the occiput was transmitted to the medial orbital wall (the weakest part of the orbit).

In conclusion, extensive bilateral angle recession may occur following occiput injury, without direct ocular trauma. A thorough eye examination including gonioscopy is important in patients with blunt injury to the head region whenever there is a complaint of blurred vision, especially when hyphema or microhyphema is present.

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