

Acute angle closure glaucoma following head and orbital trauma

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The case is reported of a 52-year-old woman who suffered a minor head injury and orbital trauma and returned 2 days later with a unilateral headache, vomiting and photophobia. This was initially thought to be secondary to her head injury but, once severe visual impairment and a dilated unreactive pupil developed, the true diagnosis became obvious. A diagnosis of acute angle closure glaucoma was made and she was treated with no complications. This case highlights acute angle closure glaucoma as an important diagnosis to consider in patients who present with unilateral headache and dilated pupil after head injury.

A 52-year-old woman presented to the emergency department after falling 4 ft from a ladder to the floor. Her injuries included a periorbital haematoma surrounding the left eye and a laceration of the left upper eyelid which required closure with sutures. At the initial presentation the left pupil was noted to be 1 mm larger than the right pupil, although both were reactive. Fundoscopy was normal bilaterally and visual acuity was 6/9 on the left compared with 6/6 on the right, although there had been a history of astigmatism affecting her left eye. The remainder of her assessment revealed no features of an important brain injury. Plain radiographs of the facial bones revealed no bony injury and the patient was discharged home with head injury advice.

Two days later the patient returned with worsening left-sided periorbital headache, vomiting and photophobia. On examination the left pupil was noted to be 6 mm and unreactive while the right pupil was 4 mm and reactive. With the history of recent head injury, an unenhanced CT head scan was performed revealing a fracture of the medial wall of the left orbit with a fragment in the ethmoid, and normal intracranial appearances. Maxillofacial surgical follow-up was therefore arranged. However, as the patient continued to have nausea and vomiting, she was admitted for overnight neurological observation, antiemetic and intravenous fluid supplementation. While her symptoms improved slightly, she continued to have unilateral headache and vomiting the following day requiring further observation.

On reassessment next morning, 4 days after the initial injury, the patient reported obvious worsening of vision in the left eye. Examination at this point revealed only light perception in the left eye, a dilated and unreactive left pupil with normal extraocular movements.

Urgent ophthalmology review was arranged and intraocular pressure was measured to be 46 mm Hg in the left eye compared with 16 mm Hg in the right eye. Gonioscopy showed that the angle was closed in the left eye and closable in the right eye. A diagnosis of acute angle closure glaucoma was reached and the patient was admitted for intensive medical therapy followed by bilateral YAG peripheral iridotomy with successful reduction in intraocular pressure and improvement in vision.

DISCUSSION

We present an unusual case of acutely raised intraocular pressure due to angle closure glaucoma following blunt trauma to the orbit. Secondary causes of glaucoma constitute approximately 21% of all cases of glaucoma. Of these, studies have attributed 13% to traumatic glaucoma, which represents a heterogeneous group of conditions presenting in the early and late phase following either blunt or penetrating trauma.¹

One mechanism for the development of acutely raised intraocular pressure following blunt trauma is by traumatic hyphaema. Blood, either in the form of macroscopic or microscopic hyphaema, can block the trabecular network and the canal of Schlemm causing an obstruction to the drainage of aqueous humour. Although in this case hyphaema was not seen on repeated examinations, microscopic hyphaema is particularly difficult to detect and therefore a high index of clinical suspicion is required.²

Subsequent follow-up of our patient revealed bilateral shallow anterior chamber depth suggesting that she was already at risk of developing glaucoma. One likely explanation for this patient's presentation is therefore that orbital trauma precipitated acute glaucoma by causing pupil dilatation and subsequent angle closure.

Pupil dilatation could be secondary to sympathetic stimulation as part of the stress response to trauma, or from the effects of sympathomimetic drugs, although in this case none were prescribed. Another mechanism for traumatic mydriasis is iris sphincter rupture, which is mostly seen following blunt trauma to the anterior segment.³ The small radial tears of the iris sphincter may be seen on slit lamp examination and lead to a dilated or irregular pupil, and are often associated with a hyphaema.

CONCLUSION

Angle closure glaucoma is an ophthalmological emergency and is an important diagnosis to consider in patients who present with acute unilateral headache, particularly when accompanied by vomiting and visual symptoms. Even though an acute brain injury may be likely in the presence of recent head trauma, other important differential diagnoses including acute glaucoma should also be considered and excluded. Furthermore, CT scanning has a sensitivity and specificity approaching 100% for detecting a significant intracranial lesion.⁴ In the presence of a negative CT scan, efforts must be made to identify an alternative diagnosis, especially if the patient's symptoms worsen, as seen in our case. Both an awareness of the differential diagnosis and a high index of suspicion are required for the early diagnosis and effective management of this serious condition.

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