Case report

Anterior lens capsule rupture following minor blunt trauma with a past history of minimal change glomerulopathy

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Abstract

Introduction: Few cases have been reported describing anterior lens capsule ruptures secondary to blunt trauma. Case: We present an unusual case of anterior lens capsule ruptures following minor trauma from a human elbow with a two-year history of minimal-change glomerulopathy in a 17-year-old male. In addition to having bilateral posterior subcapsular cataracts, he developed an anterior subcapsular cataract underlying the anterior lens rupture in the right eye. He was managed conservatively. Observations: The current case differs from those of the existing literature in terms of the characteristics of the physical insult and the eye on presentation. We suggest there could be a predisposing anterior capsule weakness secondary to the minimal-change glomerulopathy or the associated drug therapy, but there needs to be more evidence to confirm or refute our claim. Conclusions: Anterior lens capsule may rupture following minor trauma on a background of minimal-change glomerulopathy.

Keywords: Anterior lens capsule, rupture, blunt trauma, glomerulopathy

Introduction

Blunt trauma can cause a range of ocular disturbances, but there are only a few reports describing a resultant anterior capsule rupture. To date, there are five reports, describing seven patients with anterior capsule ruptures secondary to blunt trauma (Zabriskie et al, 1997; Banitt et al, 2009; Dezhagah, 2010; Gremida et al, 2011; Kuan-Jen Chen et al, 2012). All cases describe artificial projectiles, including an airbag, injuring the eye. We present a case of anterior capsule rupture with cataract following minor blunt trauma from a human elbow on a background of minimal-change glomerulopathy. Patient consent to describe the following case and publish related images was gained.

Case Report

A 17-year-old male presented to the Tilganga Institute of Ophthalmology (TIO) outpatient department with a one-week history of blurry vision in his right eye following blunt trauma to the right side of the head from his friend’s elbow eight days ago. He denied any flashes of light, floaters or eye pain and there was no subsequent bruising, bleeding or fracture after the injury. His past medical history was significant for minimal-change glomerulopathy diagnosed two years ago. His drug history included Enalapril 2.5 mg PO HS, Atorvastatin 10 mg PO HS and Cortisone 20 mg PO OD for the past two years to treat his kidney disease.

His uncorrected visual acuity was 6/18 in both eyes and his best corrected visual acuity with pinhole was 6/12 and 6/9 in his right and left eye respectively with no further improvement with refraction.
Intraocular pressure by Goldman applanation tonometry was 16 mmHg in the right eye and 14 mmHg in the left eye. The lids, adnexa, conjunctiva, sclera, cornea and iris examination revealed no signs of a penetrating eye injury. The anterior chambers were of normal depth and quiet bilaterally. Dilated pupil examination of the right eye revealed a paracentral oval defect on the anterior lens capsule measuring 2.2 cm x 1.2 cm with an underlying anterior subcapsular cataract (Figure 1 and 3). There were also posterior subcapsular cataracts (PSCC) bilaterally (Figure 3 and 4). The zonules were intact and there was no phacodonesis. Fundus examination revealed no abnormalities and the cup-to-disc ratio was 0.2:1 in both eyes with a healthy foveal reflex. There were no signs of Alport’s syndrome bilaterally, such as anterior lenticonus or posterior polymorphous corneal dystrophy. This was confirmed by normal anterior segment tomography (Figure 5 and 6) and normal corneal endothelial cell counts.

A conservative management approach was adopted by abstaining from immediate intervention and following-up the patient regularly in the outpatient setting. The patient was offered cataract surgery for the right eye, but he declined. The patient will be monitored for worsening of visual acuity from cataract progression as well as signs of secondary glaucoma or anaphylactic uveitis and treatment would be offered if indicated.

Figure 1: The patient’s right eye under slit-lamp examination. There is a 2.2 x 1.2 cm paracentral oval defect in the anterior capsule with an underlying anterior subcapsular cataract. There is no evidence of a penetrating eye injury.

Figure 2: The patient’s left eye under slit-lamp examination. There are no abnormalities detected.

Figure 3: The patient’s right eye under retroillumination. There is a 2.2 x 1.2 cm paracentral oval defect in the anterior capsule with an underlying anterior subcapsular cataract.

Figure 4: The patient’s left eye under retroillumination. There are posterior subcapsular cataracts.
Discussion

It should be noted that the history of trauma given by the boy was not clear, as he did not remember the details well. Nevertheless, the current theory suggest that the anterior capsule rupture may be due to a coup injury, where the cornea directly indents onto the lens following the traumatic insult, or a contrecoup injury, where the anterior recoil of the vitreous causes the anterior lens capsule to burst open due to fluid-mechanical forces (Banitt et al, 2009).

In contrast to the cases described in the literature, this case differs in the characteristics of the physical insult and the eye on presentation. As opposed to sustaining trauma directly to the eye as described in previous case reports, the boy in our case was struck at the side of the head. In addition, he presented to the outpatient department after a week of blurry vision with few anterior segment abnormalities. In the existing literature, patients would present with lid edema, conjunctival injection, corneal abrasions and red blood cells in the anterior chamber or a hyphema for example (Dezhagah, 2010; Gremsida et al, 2011). Although we did not meet the boy at the time of injury, we can speculate that the magnitude of force was minimal due to a lack of residual external eye and anterior segment abnormalities as well as intact zonules and a lack of phacodonesis. Interestingly, one would expect the anterior chamber in the affected eye to demonstrate a reaction to the antigenic lens cortex, but the absence of any changes suggests that the anterior lens capsule ruptured with preserved lens cortical integrity.

Given the low magnitude of force sustained by the boy, there could be an element of lens capsule weakness. We suggest a cause for this could be his complex history of minimal-change glomerulopathy and subsequent drug therapy. However, although it is known that long-term steroid (Jobling & Augusteyn, 2009) and atorvastatin (Hippisley-Cox, 2010) therapy is associated with PSCCs, there is no literature describing the effects of minimal-change glomerulopathy or enalapril on the lens. Further, there is no reliable evidence linking glucocorticoids or atorvastatin use to a weak or ruptured lens capsule. Thus, more evidence is required to confirm or refute our claim.

Conclusion

This is the first case to describe an anterior lens capsule rupture following minor trauma on a background of minimal-change glomerulopathy.

References


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