Secondary Glaucoma After Blunt Trauma

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Summary: *Secondary glaucoma after closed globe injury is a frequently encountered condition. The rise in intraocular pressure after blunt trauma has multiple causes which can be classified as early and late onset. We describe two different scenarios highlighting the early and late causes of raised intraocular pressures. The patients should be examined thoroughly for the underlying etiology and managed accordingly.*

The reported incidence in literature varies from 2-10%, the Indian studies reporting a figure of 4.5%[1,2]. Glaucoma following a closed globe injury can present with raised intraocular pressure (IOP) in early or late postoperative period. The various reasons for early onset IOP rise are inflammation, hyphema, damage to trabecular meshwork, secondary to organisation of blood/inflammatory products, alterations in lens position, phacoanaphylactic glaucoma, increased episcleral venous pressure whereas the various causes of late onset IOP rise are steroid induced, secondary angle closure, angle recession, ghost cell, hemolytic, hemosiderotic, lens subluxtion, lens particle, lens induced uveitis etc[3].

The early identification of underlying etiology is important for appropriate management of raised intraocular pressures and delay/prevent the irreversible glaucomatous damage. We present 2 cases projecting different causes of post-traumatic raised intraocular pressure.

CASE PROFILE

Case 1

A 30 year old male presented with history of trauma to right eye 14 days back followed by acute, painful, diminution of vision. His best corrected visual acuity (BCVA) was hand motion close to face in right eye and Snellen 6/6 in left eye. The IOP was 40 mmHg and 14 mmHg in right and left eye respectively on Tab.Acetazolamide 250 mg, G.Brimonidine 0.2% and G.Timolol Maleate 0.5% in right eye. Slit lamp examination revealed subconjunctival haemorrhage in nasal, temporal and inferior quadrants (Figure 1a). The cornea was clear, with presence of 2 + pigmented cells in anterior chamber (Figure 1b). The pupil was 'D shaped' with multiple sphincter tears (Figure 1a). There was presence of post- traumatic posterior subcapsular cataract (Figure 1c and d).

Gonioscopy revealed angle recession in nasal quadrant and iridodialysis extending about 2 clock hours (11-1 o' clock) in superior angle. The ciliary processes were visible through iridodialysis (Figure 2).

The media was hazy due to cataract but the disc was healthy with cup disc ratio of 0.3. The left eye examination was within normal limits. The patient was given injection Mannitol 2g/kg b.w i/v over 30 minutes. He was started oral steroids (1 mg/kg body weight) with slow tapering every 3 days. He was also given topical steroids, cycloplegic (G. Atropine sulphate) and continued oral and topical antiglaucoma medication. The IOP lowered down in susbsequent visits as the inflammation settled down and the antiglaucoma drugs were gradually tapered over a period of 3 months.

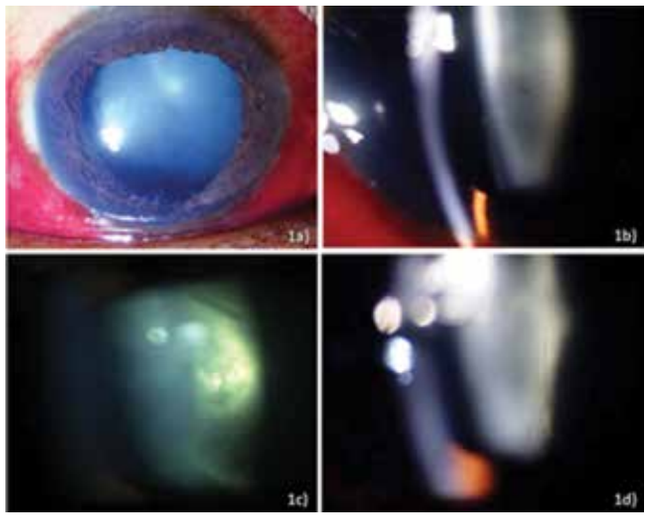

Figure 1

Figure 2

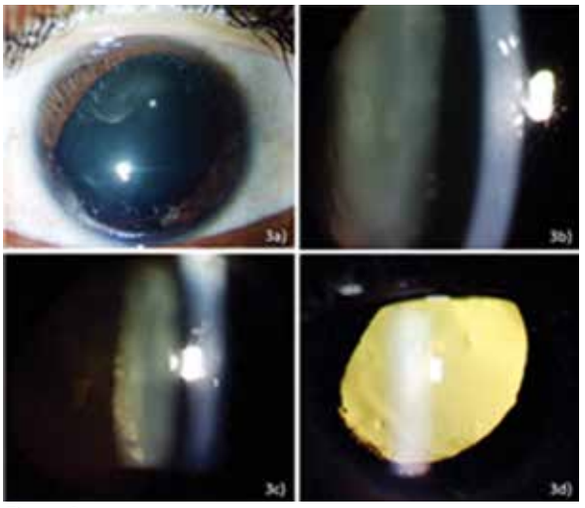

Figure 3

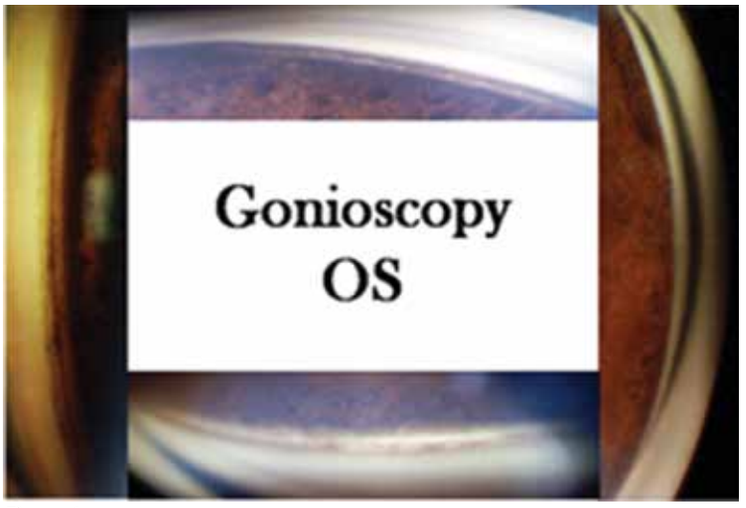

Figure 4

Case 2

A 23 year old male was referred three months after blunt trauma with raised intraocular pressures. His BCVA was Snellen 6/6 in right eye and hand motion close to face in left eye. The IOP was 14 and 50 mmHg in right and left eye respectively on Tablet Tab.Acetazolamide 250 mg, G.Brimonidine 0.2% and G.Timolol Maleate 0.5% in right eye. On slit lamp examination, the conjunctiva was normal. The cornea was clear. The pupil was dilated, non reactive, irregular with presence of multiple sphincter tears and phacododensis (Figure 3a). The anterior chamber depth was non uniform with 3 plus pigmented cells in anterior chamber (Figure 3b). Posterior subcapsular cataract was seen (Figure 3c). On retroillumination, yellow glow was appreciated (Figure 3d).

Gonioscopy revealed pigmented angles and angle recession in 2 quadrants (superior and inferior) (Figure 4).

Ultrasound examination showed the presence of pin point and dense hypereflective echoes suggestive of vitreous hemmorhage. A provisional diagnosis of Khaki cell glaucoma (ghost cell glaucoma) was made. The patient was taken up for L/e Pars plana lensectomy and vitrectomy. The IOP lowered down and the antiglaucoma medication was weaned off in next two months.

COMMENT

Secondary Glaucoma following closed globe injury occurs due to coup-countercoup injury. Thorough clinical examination in case of trauma includes evaluation of 'seven rings of trauma': pupillary tears, iridodialysis, angle recession, cyclodialysis, meshwork tears, ruptured zonules and retinal dialysis. Increased pigmentation of the angle, elevated baseline IOP, hyphema, lens displacement and angle recession of more than 180 degrees are some of the early predictors which are significantly associated with the occurrence of chronic glaucoma after closed globe injury[4]. In a study comparing the outcome of glaucoma after penetrating and closed globe injury, 58% of the eyes with history of penetrating trauma required glaucoma surgery, whereas only 12% of the eyes in the group with blunt trauma in history underwent a glaucoma surgery during follow up. The time of surgery in the eyes with a penetrating trauma in history was after sixth month of follow-up. But in 64% of eyes in the group with blunt trauma in history, early surgical intervention were needed[5]. Early onset raised IOP is more commonly due to trabeculitis and need to be managed with steroids and cycloplegic agents. For late onset IOP rise, patient should be thoroughly examined for underlying etiology and managed accordingly.

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