Ocular trauma is an event witnessed very frequently by most ophthalmologists with Indian data documenting an incidence of 4.5%, 0.4% being bilateral. Maximum injuries are related to domestic accidents and sporting activities with young males being most afflicted. School going children are the most vulnerable due to unsupervised activities amongst active peers versus younger stay at home children. The usual causative agents are blunt trauma with toys or household appliances, penetrating eye injuries with sharp objects, ball games, boxing, gullidanda, bow and arrow, firecracker related injuries and airbag related motor vehicle accidents. Another type is chemical injury in which category lime (chuna) is the commonest culprit in India. Chemical injuries will however not be detailed in this chapter.

Since trauma usually affects young children/ productive adults the consequent visual disability has a lasting impact on child’s future academic, financial and social prospect with propensity for the traumatized eye developing vision deprivation amblyopia. Ocular trauma is usually classified as post blunt or penetrating injuries with Indian data documenting blunt injuries to be responsible for almost 55% cases. Glaucoma complicates the situation in many such injury events especially for blunt trauma cases. The underlying eye is often totally healthy therefore timely and appropriate management of these injuries may restore vision to a greater extent than in those with underlying primary pathology. Different scenarios of glaucoma depend on type of trauma which is concussion (closed globe) or penetrating (Open globe).

### Concussion/ Blunt Injury

Acute phase glaucoma

Elevated intraocular pressures (IOP) post trauma is mostly transient in early period; sometimes, there may be an initial drop in IOP due to iritis or temporary angle distortion causing an increased outflow facility. The acute phase high IOP usually subsides, on medical treatment, within 4-6 weeks. The various causes of high IOP in acute phase in order of frequency are given below and few are depicted in (Figure 1).

- Hyphaema
- Traumatic iridocyclitis
- Lens dislocation/subluxation/ rupture
- Shallowing of the anterior chamber due to uveal effusion
- Vitreous disruption and presence in deep anterior chamber
- Tears of the non-pigmented epithelium of the ciliary body along with retinal detachment causing IOP fluctuations – Schwartz-Matsuo syndrome

Hyphaema occurs as a result of distortion of anterior chamber angle with subsequent rupture of iris or ciliary body vessels. Approximately one-third of all hyphema patients exhibit increased intraocular pressure in the early period which increases to 65% in cases of rebleed. The reason for glaucoma is occlusion of trabecular meshwork by blood clot, inflammatory cells, erythrocytic debris; or
pupillary block secondary to a collar button-shaped (eight ball) clot involving both anterior and posterior chambers. Larger the hyphaema volume, greater is the likelihood of increased intraocular pressure except in patients with sickle cell anaemia who may exhibit high IOP even with minor bleeds.

Figure 1(a): Hypotony in acute hyphema (b): Acute glaucoma with Hyphema. (c): Acute glaucoma in firecracker injury with corneal tattooing. (d): Firecracker injury with traumatic cataract. (e): Acute glaucoma due to ruptured lens matter in anterior chamber. (f): Acute glaucoma with intumescent cataract and iridodialysis.
Clot retraction starts after 3-5 days and may be associated with re bleeding. Hypotony associated with cyclodialysis cleft or retinal detachment may mask the glaucoma in the early phase of trauma.

Vitreous haemorrhage if small is managed conservatively and dense haemorrhage requires vitrectomy at a later date once posterior vitreous detachment has occurred and ocular inflammation has resolved.

Treatment

The patient of hyphema is advised restricted activity and sleeping in head elevated position. Use of Aspirin or NSAIDs which hinder platelet activity are restricted. Topical corticosteroids are used judiciously; their anti-inflammatory activity needs to be weighed against their propensity to increase IOP in steroid responders. They are prescribed at 4-6 hourly intervals and tapered once inflammation resolves. Use of aminocaproic acid to prevent re-bleeding is controversial. The risk of re-bleeding is variable and the evidence that aminocaproic reduces its incidence anecdotal. This antifibrinolytic agent prevents clot from dissolving early by inhibiting conversion of plasminogen to plasmin. If given the dose is 50mg/kg every four hours with maximum dose of 30 g/day for 5 days.

Topical β blockers, adrenergic agents and carbonic anhydrase inhibitors are the drugs commonly used to control the IOP. Pilocarpine and prostamide anti glaucoma drugs are avoided due to their propensity to increase inflammation. Pilocarpine increases vascular permeability and promotes fibrin deposition in an already inflamed eye; in addition it can cause irido-lenticular adhesion/seclusio-pupillae due to a miosed pupil. Rarely systemic acetazolamide may be required to control IOP. Its use is contraindicated in sickle cell disease patients as this drug is known to cause metabolic acidosis thereby increasing sickling of the RBC in anterior chamber. For specific causes like shallow anterior chamber post uveal effusion judicious use of steroids and mydriatic-cycloplegics is required.

Surgical intervention

- Surgical drainage of the clot is accomplished by simple paracentesis, washout of blood from anterior chamber, visco-expression of clot or automated extraction. Usually a two port entry is performed and a total removal must not be aimed at.

- This intervention is assessed on individual basis depending on IOP and prior optic nerve status.

- For previously healthy eyes the guidelines are IOP of >50 mm Hg for 5 days or >35 mm Hg for 7 days

- In Grade 3 hyphema (>50% AC) or total "eight ball" hyphema remaining for 8 days the risk of synechia formation increases so these patients should be taken up for hyphaema drainage.

- Another indication is appearance of corneal blood staining.

- Lensectomy, vitrectomy should also be individualised keeping in mind that these tend to make glaucoma management, later on, more difficult.

Late onset glaucoma

This occurs days to years after inciting injury and is commonly a consequence of damage to trabecular meshwork (often seen in association with angle recession), descemetization and fibrosis of trabecular meshwork, siderosis of trabecular endothelium, or peripheral anterior synechia formation post inflammation leading to secondary angle closure glaucoma. Incidence of this late-onset glaucoma ranges from 0–20% in eyes with a history of traumatic hyphaema. There is a 4-9% risk in patients with angle recession while the incidence of angle recession after eye trauma ranging from 20-94%. Following is the list of causes of late phase traumatic glaucoma –

- Angle recession (Figure 2)
Peripheral anterior synechiae
• Ghost-cell / khaki cell glaucoma in vitreous haemorrhage cases
• Retained intraocular foreign bodies
• Ongoing inflammation / cyclitic membrane
• Epithelial ingrowth (Figure 3)
• Sympathetic ophthalmia

Angle recession is a tear between circular and longitudinal ciliary muscles which manifests clinically as a widening of ciliary body band. Hydrodynamic forces generated during trauma forcefully push aqueous against ciliary body causing the tear. The tear is peripheral to arterial arcade of ciliary body. It disrupts branches of anterior and posterior ciliary arteries leading to hyphema. Mere presence of angle recession does not preclude glaucoma and tear in ciliary body per se is not the cause for glaucoma. This initial trauma stimulates proliferative or degenerative changes with subsequent scarring of trabecular meshwork leading to ultimate dysfunction of the outflow pathway. Intraocular pressure elevation in angle recession usually presents with two peaks of onset, first around 3 months post injury and second after an interval of 10 years.

In those presenting late, angle recession merely unmasks a predisposition to open angle glaucoma. Various predictors for post-traumatic glaucoma are presence of increased angle pigmentation, elevated baseline IOP, hyphema, lens displacement, angle recession more than 180 degrees, wider angle on UBM, absence of cyclodialysis, phacoanaphylaxis and siderosis. Older adults are more susceptible to late-post contusion pressure elevation. Iris sphincter tears, lens and posterior segment injury are often associated and determine the final visual prognosis.

There is also evidence to suggest an underlying susceptibility/ genetic predisposition for glaucoma in these angle recession eyes as evident by IOP fluctuations in normal fellow eye and a tendency for steroid responsiveness. Clinching evidence comes from a study where lifetime risk of developing glaucoma in fellow non-traumatized eye was 50%.

Ghost cell / khaki cell / haemolytic glaucoma: A condition where distorted, non-pliable, khaki coloured RBC migrate from a resolving vitreous haemorrhage (2-3 week old) and block the trabecular meshwork. Red blood cells trapped in vitreous transform as they age within 1-3 weeks from pliable, biconcave cells to rigid, spherical, khaki coloured cells due to partial loss of haemoglobin. Residual intracellular haemoglobin denatures into Heinz bodies, which characterizes these cells. These rigid khaki coloured RBC lower outflow facility three time more than fresh RBC. The RBC’s layer down in the anterior chamber in tan coloured stripes and can be confused with a hypopyon. If there is concomitant fresh blood then the ghost cells occupy the tan stripe in background of fresh red RBC which presents as “Candy stripe” sign.

Management
• Gonioscopy is the clinching investigation and delineates angle recession from PAS or down growth as the cause of glaucoma. A dilated anterior segment exam must be performed after gonioscopy to rule out subtle evidence of zonular dialysis and indirect ophthalmoscopy with indentation needs to be done to rule out peripheral retinal dialysis or breaks.
• For ghost cell glaucoma medical treatment usually suffices as the condition is usually transient, rarely vitrectomy along with anterior chamber wash out is required to control recalcitrant glaucoma.
Ocular Trauma

- Open angle glaucoma post trabecular meshwork scarring responds to aqueous suppressive drugs. Over last few years we have found pilocarpine to be dramatically effective in few such cases.
- Coexisting ocular problems like cataract and stimulus deprivation amblyopia for children need to be addressed timely to retain functional vision.
- Both eyes of an angle recession patient need to be examined annually, life-long to detect early glaucoma change.

Surgical intervention

- Laser trabeculoplasty can be tried as a supplement if medical therapy before attempting filtering surgery. Fukuchi et al have reported an alternative laser procedure, Nd:YAG laser trabeculopuncture, in which 1.0 to 2.5 mJ energy is delivered to TM like argon laser trabeculoplasty with reported efficacy in angle-recession glaucoma treatment.
- Trabeculectomy if required is usually performed with anti-fibrotics as most cases are young adults. Trabeculectomy should be performed with releasable sutures and supero-nasal quadrant is utilized keeping the supero-temporal area free for a repeat trabeculectomy if required during the remaining life span of these young adults.
- Before attempting heroic surgeries to control the glaucoma it is always wise to evaluate the posterior segment thoroughly, wherever possible. A large central choroidal rupture (Figure 4), maculopathy, optic nerve avulsion would preclude any visual recovery thus necessitating a rethink in glaucoma management strategy.

Penetrating injury / Open Globe Injuries

Initial IOP is invariably low in penetrating injuries due to open wound situation. The pressure can rise post repair due to lens intumescence, inflammation or hyphema. A U.S. Eye Injury Registry notes a 2.67% 6-month incidence of glaucoma after penetrating trauma. The common causes are unremoved lens particles, inflammation, and hyphaema in early stage (within one month), presence of synechial angle closure, ghost cells in intermediate stage (2 to 6 months) and retained lens particles, angle recession, synechial angle closure in late stage (>6 months). Another study reported a 17% incidence of ocular hypertension after open globe injury with risk factors like increased age, hyphema, lens injury, and zone II injury; advancing age, lens injury, poor visual acuity, and intraocular inflammation have also been reported.

Treatment

Medical management can control IOP in 30-74% patients. Prevention is the key strategy and meticulous wound closure with removal of incarcerated uveal tissue, removal of foreign body, lens aspiration if integrity of anterior capsule is doubtful, adequate reformation of anterior chamber minimizes glaucoma propensity in these patients. Surgical interventions needed include glaucoma filtering surgery either trabeculectomy or tube surgery in 12-27%, anterior chamber wash in 4-7%, lens aspiration and cyclophotocoagulation.

Preventive strategies

- Use of eye protective glasses while playing outdoor sports involving high speed projectiles prevent almost 90% of sports related injuries. Unbreakable polycarbonate spectacles should be used by children.
- Age appropriate toys should be given to children with missile firing toys need to be banned.
- All chemicals and sprays should be kept out of reach of children.
- Playing with fireworks is to be totally discouraged and safe Holi practices should be followed.

Conclusion

Regular follow-up is a must for all cases of ocular trauma and evaluation of both eyes should be done, keeping in mind the fact that glaucoma can develop even 15 years post trauma.

References

Ocular Trauma: Post Traumatic Glaucoma


