Introduction

Trauma related glaucomas are a mixed bag of conditions that raise the intraocular pressure (IOP) and hence compromise the optic nerve function. When we talk about trauma we tend to think about blunt trauma and penetrating trauma (as well as chemical, electrical and radiation traumas). Most of the secondary glaucomas are a result of blunt trauma. It can occur acutely or more often delayed as in angle recession.

Blunt Trauma and Glaucoma

Blunt frontal trauma causes the corneal apex to indent and consequently, the limbal ring gets stretched. The peripheral cornea is pushed outward and the iris root rotates backwards. The zonules are stretched pushing the lens back. When this happens violently enough that would tear tissues to detach from their attachment at the limbus – leading to iridodialysis, trabecular meshwork tears, angle recession, cyclodialysis, and zonulolysis. This would first open up blood vessels and cause a hyphaema.

Figure 1: Blunt Trauma to cornea: Corneal apex is indented. Limbal ring stretched. Peripheral cornea pushed out. Iris root rotates backwards. Zonules stretch pushing lens back. Causing iridodialysis, trabecular meshwork tears, angle recession, cyclodialysis and zonulolysis.

Hyphaema

Hyphaema is presence of blood in the anterior chamber. It occurs after trauma due to a tear into a blood vessel bounding the anterior chamber – most often in the angle recess. Hence one should always suspect angle recession in any case of traumatic hyphaema.

Hyphaema causes a rise in intraocular pressure via a few mechanisms. Primarily it causes an increase in resistance to aqueous outflow by the blood cells (predominantly RBCs) blocking the trabecular meshwork. Viscosity of aqueous is also marginally increased by the blood proteins. To add to this there is inflammation due to the trauma itself causing a trabeculitis and swelling of the trabecular meshwork reducing the pore spaces in the same. If the clot covers the pupil then it will occlude the same and cause pupillary block glaucoma as well. If the situation continues long enough then peripheral anterior synchiae can form due to pupillary block over a wide area or due to the inflammation per se in limited areas. Both can compromise aqueous outflow over long term.

At the initial setting the IOP may be normal, low or high. This is due to associated changes. There maybe a cyclodialysis cleft draining out the aqueous. Alternatively the ciliary body may temporarily shut down in the context of inflammation. There may be a retinal detachment. (Of course a globe rupture or penetration has to be ruled out and managed). The IOP is the net effect of inflow vs. outflow. Hence when the inflow exceeds out flow the IOP will rise. So IOP should be monitored at least daily in the acute setting.

A specific situation warrants mention. Normally the RBCs would wriggle out through the trabecular meshwork. If they become stiff (not pliable) they get stuck in the meshwork. This happens in sickle cell disease. When deoxygenation occurs, the RBCs sickle because the PH decreases. The sickled cells clog the meshwork more as they are stiff and the cells are now more tapered and can wedge better. This causes the IOP to rise fast and thereby reduce the retinal blood flow. Slowing of blood in the retinal circulation causes intravascular sickling, as the cells get deoxygenated more, leading to obstruction of small vessels and even a central retinal artery occlusion. Thus in sickle cell disease and trait patients hyphaema causes a rise in IOP more often and the damage to retina and optic nerve are more profound. Additionally acetazolamide which is the first choice drug to be used in hyphaema related IOP rise worsens sickling and is contraindicated in these patients. Hyperosmotics like mannitol cause haemoconcentration and this causes sickling as well. (So even though sickle cell disease is rare in our patients it is still prudent to order a thick peripheral blood smear or a sickle preparation with your clinical pathologist in all cases of hyphaema.)
Rebleeds

By the 2nd to 4th day the clot retracts and this can reopen the bleeding site causing an increase in the hyphaema. Thus these patients need at least daily examinations to look for the same. Earlier management of hyphaemas included epsilon aminocaproic acid as an antifibrinolytic agent to combat this. The side effect and cost profile did not match the risk involved and not many favour this anymore.

Management of IOP in Hyphaema

All hyphaema patients need treatment of the inflammation with topical steroids and cycloplegics (Earlier pilocarpine was advocated to increase surface area of iris for absorption of blood. But the added inflammation due to pilocarpine has prompted its withdrawal from treatment regimes). The IOP is to be treated when elevated medically. One prefers aqueous humor suppressants in this situation viz., acetazolamide oral, topical β-blockers and topical dorzolamide. In sickle cell disease methazolamide systemically and dorzolamide topically are probably safer than acetazolamide which is contraindicated. Hyperosmotics like mannitol are also effective. Prostaglandin analogues and α-agonists are not well tolerated by inflamed eyes (though not absolutely contraindicated).

Surgical intervention is required in hyphaema in certain situations. If there is an eight ball hyphaema or a total blood clot filling the anterior chamber or if the clot is causing an obvious pupillary block, then the glaucoma is an angle closure glaucoma. If the pupil has been given enough time and cycloplegic medications to dilate and this situation persists, then it is unlikely to respond only to medical management. A peripheral iridectomy is warranted. Laser iridotomies are too small and a surgical iridectomy is easier in the presence of a blood clot and can be done to relieve the pupillary block.

Surgical intervention is usually indicated on or after the fourth day. Overall, indications for surgical intervention are outlined below:

- Microscopic corneal blood staining (at any time)
- Total hyphaema with intraocular pressures of 50 mm Hg or more for 4 days (to prevent optic atrophy)
- Total hyphaemmas or hyphaemas filling greater than 75% of the anterior chamber present for 6 days with pressures of 25 mm Hg or more (to prevent corneal blood staining)
- Hyphaemas filling greater than 50% of the anterior chamber retained longer than 8-9 days (to prevent peripheral anterior synechiae)
- In patients with sickle cell trait or sickle cell disease who have hyphaemmas of any size that are associated with intraocular pressures of greater than 35 mm Hg for more than 24 hours

A host of surgical options are described. The ideal day for surgery is probably day 4 when the clot retracts. The intervention can be anterior chamber washout with BSS, mechanised hyphaemectomy with a vitrectomy probe, a routine trabeculectomy, etc. Blind pulling on the clot is to be discouraged as one may cause a rebleed from the injured vessel in the least and may cause inadvertent tissue injury by pulling off iris, descemets, lens capsule, etc.

Trabecular meshwork tears

If one does a gentle gonioscopy in the first few days after trauma one may see a trabecular meshwork tear (Figure 2). This is a disinsertion of the trabeculum from the Schwalbe’s line. Initially this leads to a lowering of IOP as it works like a trabeculotomy – opening up the Schlem’s canal directly to the anterior chamber. Later on a glass membrane forms over it and the tear gets obscured. Now the IOP rises as that part of the angle is non-functional. If the initial tear was not deep enough to cause a recession picture the late picture could be akin to a POAG. Medical control of IOP is done and trabeculectomy is an option only if medical management fails.

Figure 2: Trabecular tears (black arrow in Photograph and white arrow in the UBM picture) are disinsertion of trabecular meshwork from Schwalbe’s line. Can initially work like trabeculotomy with low IOP but later glass membrane grows over and IOP rise.

Lens related glaucomas in Trauma

Trauma can cause subluxation or dislocation of the lens. It can disrupt the capsule ad allow lens matter to travel into the anterior chamber. The contusion itself can cause lens swelling or intumescence of the lens. All these can affect the IOP.

A subluxated or dislocated lens can cause pupillary block glaucoma. This can either be by the lens moving forward or by a knuckle of vitreous blocking the pupil. If the lens is clear and reasonably supported by zonules a laser PI can be attempted as a first option. If there is significant cataract or if lens edge is in the pupillary area lens extraction is indicated.

A subluxated lens can move forward and physically push the peripheral iris forward and close the angle. Lens intumescence also does the same. These situations cause phacomorphic glaucoma like picture. Again lens extraction would cure the glaucoma.

If the lens capsule be torn, then the lens matter can move...
into the anterior chamber. The fragments of lens fibres would block the trabecular meshwork causing a lens particle glaucoma\(^1\).

All these situations warrant lens extraction – preferably extra capsular, if not possible then intra capsular. Preoperatively an indentation gonioscopy is needed as there could be a compromised angle contraindicating an anterior chamber IOL (in case the lens is removed intra capsularly). In general, we prefer an in the capsular bag IOL or a scleral fixated IOL over the use of ACIOL in cases with significant trauma.

**Angle recession glaucoma**

Angle recession glaucoma is a post traumatic secondary open angle glaucoma\(^2\). This is often under diagnosed as the rise in IOP is often delayed and by then history of injury is often forgotten.

Angle recession is sequelae of blunt trauma and is characterised by cleavage between the circular and longitudinal muscle planes of the ciliary body (Fig 3)\(^1\)\(^-\)\(^4\). For this to occur, the cleavage has to go through the aqueous drainage channels in the angle causing variable changes in outflow resistance. The histologic basis for this theory has been described by Wolf and Zimmerman\(^9\).

The initial IOP after trauma may be low, normal or high. This could be due to ciliary body shut down as part of inflammation following the trauma. Later on this aqueous inflow picks up over a few weeks and IOP may rise\(^1\)\(^-\)\(^7\). Healing can cause a glass membrane to form across the angle as an extension of the descemet’s membrane and cause an increase in outflow resistance\(^1\). Trabecular fibrosis also contributes to rise in IOP\(^1\). Hence the rise in IOP can be delayed by weeks to years in these patients.

On gonioscopy one sees a widened angle recess with an irregularly widened ciliary body band. Scleral spur appears abnormally white\(^1\). One may also see torn iris processes on gonioscopy. Even in the absence of a history of trauma one may see telltale signs of an old hyphema in the form of black pigment balls (hemosiderin). Iris pigment looks more brown and not black in the angle (Fig 4). These last for very long time – years in fact.

**Closure of a Cyclodialysis cleft**

A cyclodialysis cleft causes a hypotony. But over a period of time these can spontaneously close (or need surgical closure to improve vision). At this point the outflow resistance goes up and the IOP rises\(^1\). Essentially this is also due to angle damage from trauma (recession) and needs management like recession. The cleft closure only affects the timing of the rise in IOP.

Identification of cyclodialysis is by indentation during gonioscopy. In Figure 5 the first gonioscopic picture looks normal and on indentation we see the cleft open up.
Ghost Cell Glaucoma
Trauma can cause a vitreous hemorrhage. The blood degrades over 3 weeks or so in the vitreous. The blood cells lose hemoglobin and become stiff spherical skeletons of the cells called Heinz bodies or ghost cells. These cells if they migrate into the anterior chamber would block the pores of the trabecular meshwork. Unlike RBCs in a hyphaema, which are pliable and can wriggle out of the trabecular meshwork, these stiff cells get stuck. This increases the outflow resistance and the IOP rises with even a few cells.

For migration of ghost cells often there has to be a breach in the anterior vitreous face and the zonular diaphragm (often present in the setting of trauma). Clinically Ghost cells appear as tan (light brown or khaki) coloured cells in the anterior chamber. These can settle down as a tan coloured hypopyon. If blood cells in different stages of degeneration are there they get layered giving us the candy stripe sign in this hypopyon. The setting looks like a uveitis but keratic precipitates are conspicuous by their absence. In the vitreous we see old vitreous hemorrhage.

Management would be by anterior chamber washout. The diagnosis can be confirmed by centrifuging the washout and looking for Heinz bodies under phase contrast microscopy or using a wet mount preparation with methyl violet stain (Fig 6). Invariably this washout will need to be repeated in a few weeks, if the reservoir of altered blood in the vitreous is not removed. So a pars plana vitrectomy is also indicated. Alternatively anterior chamber washout with trabeculectomy can be done in phakic eyes.

Figure 6: Altered blood cells (Heinz bodies) seen with wet mount preparation with methyl violet stain

Penetrating Trauma
Penetrating trauma causes the anterior chamber to stay shallow in the setting of an inflamed eye. This causes peripheral anterior synechiae to form. This leads to an angle closure glaucoma. Medical management is opted for first but often a trabeculectomy is required.

Lens trauma can occur as well and is dealt with as described earlier in this treatise.

Fibrous ingrowth and epithelial downgrowth (Fig 7) can occur. These cause intractable glaucoma and aggressive surgery to remove the offending cell layers are described. A mitomycin augmented trabeculectomy or a seton is also indicated in this setting.
Retained metallic foreign bodies can cause siderotic glaucoma. Elemental iron causes degeneration of the trabecular meshwork. Localization and removal of the foreign body solves the problem in its initial stages. Sympathetic ophthalmia can also be associated with glaucoma.

Schwartz syndrome
Retinal detachment can cause glaucoma if the rod outer segments migrate into the anterior chamber and block the trabecular meshwork. The anterior chamber seems to have cells (actually the photoceptor outer segments) and can resemble a uveitic glaucoma. On careful dilated indirect ophthalmoscopy, one may find a shallow peripheral rhegmatogenous retinal detachment. Surgical closure of the offending retinal break would relieve this glaucoma.

Occasionally one may get a curious iris retraction syndrome. Here there is a seclutio pupillae due to 360 degree posterior synechiae. The anterior chamber is shallow when the IOP is high and on administering an aqueous suppressant (acetazolamide) the anterior chamber dramatically deepens to a concave iris and hypotony. Here again it is a combination of retinal detachment and secondary angle closure that is the culprit. The carbonic anhydrase inhibitors stimulate the retinal pigment epithelial pump and hence reverse the aqueous flow from anterior to posterior via the retinal break. Here both the angle closure and the retinal detachment need to be addressed to resolve the situation.

Chemical Injury
Chemical injury causes a bimodal increase in IOP. Initial insult causes the sclera to shrink causing a transient elevation of IOP in about 10 minutes to pressures in the range of 50mmHg. Later the ciliary body shutdown would lower the IOP. In about 1-2 hours the trabecular inflammation causes a more sustained rise in IOP. In this acute setting the status of the cornea does not allow objective IOP measurement and we are often left with presumptive treatment (aided by finger tonometry only). The treatment is only medical with aqueous suppressants alone.

In the intermediate and late stages of evolution of the chemical injury, IOP rises due to presence of peripheral anterior synechiae and trabecular fibrosis due to sustained inflammation. The lens may also become intumescent and worsen the situation. Again treatment is medical and if inadequate one can opt for graded cyclodestruction (trabeculectomy is not an option as almost all conjunctiva would be scarred down). By graded cyclodestruction we mean 90 degrees at a time, either cyclophotocoagulation or cyclocryotherapy. Further cyclodestruction is embarked on only after a period of at least 3 weeks, to assess the full effect of previous therapy. This is usually done prior to keratoplasty in these patients.

Miscellaneous
Exposure to penetrating radiation as part of treatment or otherwise leads to radiation retinopathy and as a consequence neovascular glaucoma can occur.

Electrical injury or lightning strike can cause iris pigment dispersion causing a transient rise in IOP that needs no treatment.

Periocular trauma can cause a caroticocavernous fistula, leading to elevated episcleral venous pressure and hence a secondary glaucoma.

Technically rise in IOP due to a retro bulbar hemorrhage and orbital emphysema also is secondary glucomas. These often need only conservative management but if there is spontaneous central retinal artery pulsation or a central retinal artery occlusion, we need to decompress the orbit. This again needs a graded approach monitoring the fundus till retinal blood flow is established (without spontaneous arterial pulsations). A lateral canthotomy → cantholysis → 360 degree peritomy → Bony orbitotomy (as described in good old Duke Elder) can be attempted. These procedures allow for propostosis of the eye to decompress the orbit and hence post operatively one should look out for and manage the consequent exposure keratopathy.

Epilogue
Significant blunt trauma usually causes a hyphaema. Glaucoma can appear immediately, weeks later or even years later. So every patient with hyphaema needs IOP monitoring and at least one gonioscopy to rule in/out angle recession. In this setting the patient needs to be monitored for life.

References
Glaucoma, Angle Recession. Sullivan BR.