Glaucoma in Trauma

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7.1 Background

Glaucoma following trauma is unique as it is caused by a heterogeneous group of challenging and potentially devastating mechanisms like blunt, sharp, or blast injuries. A variety of pathological mechanisms increase the intraocular pressure (IOP) in the early phase or years after the trauma. Ocular trauma can be divided into closed globe injuries (CGI) and open globe injuries (OGI). There are other types of traumatic insults that also lead to an injured globe, causing secondary glaucoma, like chemical injuries, blast injuries, and surgery. Transient or prolonged elevations in IOP occur depending upon the extent of damage to the trabecular meshwork and disorganization and damage to other ocular structures. A long-term increase in IOP can lead to permanent damage to the optic nerve head and visual functions.

7.2 Prevalence and Incidence

The risk of developing glaucoma after a specifically closed globe contusion was found to be 19% in one study [1]. Another study found the corresponding risk of penetrating ocular injury to be about 3% [2]. In the United States trauma registry of 1998, 58% of ocular trauma subjects were less than 30 years of age [3]. Young males are prone to ocular trauma, with male-to-female ratios ranging from 3.4: 1 to 13.2: 1 [3].

The causes and circumstances of ocular injury can vary widely according to geographic, socioeconomic, occupational, and cultural factors. For instance, in Los Angeles County, California, violent assault accounts for 41% [4] of ocular injuries. In the past, a majority of ocular trauma originated in the workplace or in settings of violent conflict, but in recent years, there has been an increase in ocular injuries secondary to leisure activities and motor vehicle accidents [3]. Among 32 patients hospitalized for sport-related ocular injury, ball games were the most common cause, smaller the ball more, the more damage it causes as it fits in the socket [5]. A golf ball or shuttlecock is far more dangerous than football. An increasingly common source of severe blunt ocular trauma is airbag injuries related to motor vehicle accidents [6].

When armed forces are used against civilians in political unrest, actions can result in a dramatic increase in the incidence of eye injuries. Injuries can be specifically brutal from tear-gas canisters and rubber-coated bullets [7]. In armed forces conflicts like insurgency and war, the types of injuries are splinters from mine blast and bomb blasts. These mechanisms may lead to closed or open globe injury and may be further associated

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with the effect of blast making damage more than if one mechanism were responsible. An Andhra Pradesh Eye Study in rural southern India showed a high incidence of injury among workers at the workplace (55.9%), followed by injuries at home (21.7%). Laborers or industrial workers are performing hazardous duties in factories and construction sites without adequate safety standards.

A study by Kamalkar et al. found post-traumatic IOP elevation occurred in 15% of pediatric eyes, more common with closed globe injuries compared to open globe injuries. In a closed-globe injury group, nearly 25% of patients required glaucoma surgery and 63% required medical management. High-risk populations like the military, police, and athletes playing contact games and ball games are more susceptible than the general population [8].

7.3 Mechanisms of Glaucoma Secondary to Closed Globe Injury

7.3.1 Early Onset

Common mechanisms of raised IOP after CGI are angle recession, hyphema, inflammation, and lens-related factors. Another possible mechanism could be uveal effusion leading to shallowing of the anterior chamber and liberation of retinal photoreceptors in **Schwartz–Matsuo syndrome**, typically accompanied by important IOP fluctuations [9].

7.3.2 Hyphema

Hyphema is defined as the presence of blood in the anterior chamber. The annual incidence of traumatic hyphema has been estimated at 12 injuries per 100,000 people, with males being affected three-to-five times more frequently than females [10]. Sports-related injuries account for 60% of ocular trauma resulting in hyphema [11].

CGI causes high-impact compression followed by expansion, leading to rupture of iris stromal or anterior ciliary body blood vessels and bleeding into the anterior chamber. Based on the extent of blood in the anterior chamber and the type of treatment required, hyphema is graded as under (Figs. 7.1 and 7.2).

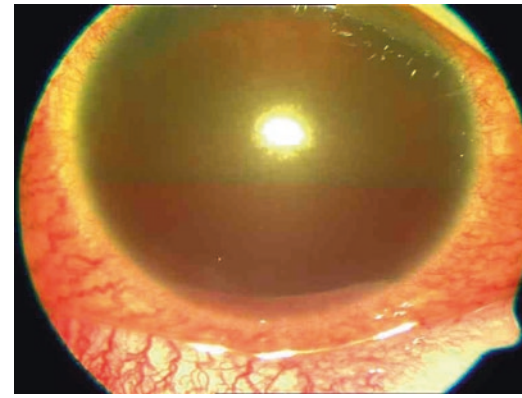

Fig. 7.1 Hyphema half chamber

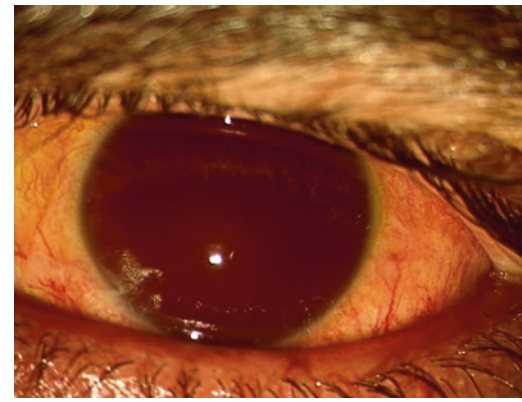

Fig. 7.2 Full chamber hyphema

Hyphema grading	
Grade I	Less than one-third filling of the anterior chamber
Grade II	One-third to one-half of the anterior chamber
Grade III	One-half to near total filling of the anterior chamber
Grade IV	Hyphema corresponds to complete filling of the anterior chamber

The degree of IOP elevation has been shown to be related to the extent of hyphema [11]. In healthy children and adolescents, pressures up to 50 mmHg can be tolerated well for 5 days without permanent optic nerve damage [12].

However, patients with sickle hemoglobinopathy can only withstand an IOP of 24 mmHg for

24 h (**Goldberg's rule**) before permanent damage to the optic nerve occurs [13].

Secondary hemorrhage typically occurs two to five days after the initial injury, as the initial clot begins to retract and lyse [14]. Potential predisposing factors for rebleeding include: those with high IOP at first examination (>22 mmHg) [14], sickle cell disease or trait [15], African-American race without hemoglobinopathy [16], use of antiplatelet and anticoagulant medications, and systemic bleeding dyscrasias [17]. The real threat to glaucoma damage depends on the degree and duration of elevated IOP and rebleeding. In one study of 235 cases, the prevalence of glaucoma was 13.5% in eyes with grade II hyphema, 27% in eyes with grade III hyphema, and 52% of eyes developed glaucoma with total hyphema [18]. About 60–94% of eyes with traumatic hyphema are found to have associated angle recession [19] (Figs. 7.3 and 7.4).

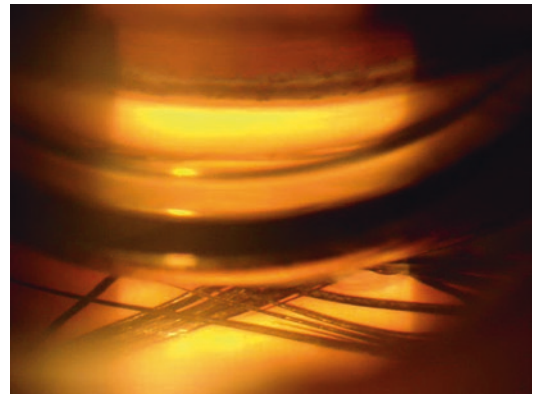

Fig. 7.3 Angle recession

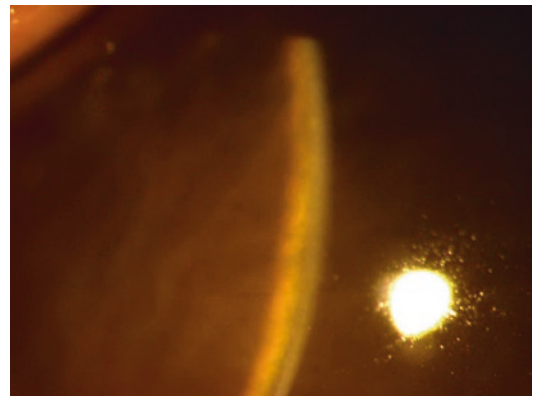

Fig. 7.4 Blood staining of cornea

7.3.3 Management

Immediate patching of the eye with an eye shield, restriction of activity (even reading), and elevation of the head of the bed by 30° are the best ways to start treatment. Before patching, it is important to ascertain that there is no abrasion on the cornea. If no abrasion is seen, the eye is patched with prednisolone and atropine eye drops and lubricant. In the presence of an abrasion, prednisolone eye drops are withheld until the abrasion heals. The Patient is supported by adequate medicine for pain and vomiting. Acetazolamide is a standard drug that is given for IOP control in all the hyphema in cases of AC > 1/3 filled with blood. However, it is extremely important to ascertain the history of sickle cell disease and sulpha allergy due to a cross-drug reaction with acetazolamide. H/O drug intake like aspirin or antiplatelets should also be asked in elderly patients. Hospitalization is suggested for high intraocular pressure on initial examination, delayed presentation, hyperactive children, or large hyphema (filling >50% or more of the anterior chamber). Systemic diseases like hemophilia and associated body trauma [17].

Medical management focuses on the prevention of secondary hemorrhage and raising IOP. Cycloplegia with cyclopentolate or homatropine provides pain relief and prevents the development of posterior synechiae [17]. The prednisolone acetate may lower the risk of rebleeding and reduce inflammation. Oral antifibrinolytics, including aminocaproic acid and tranexamic acid, stabilize anterior chamber blood clots and delay clot retraction so that rebleeding is reduced [20]. Once patching is removed, topical antiglaucoma medications like beta blockers or Alpha 2 agonists can be used. In sickle cell patients, IOP has to be aggressively controlled in the first 24 hours. A carbonic anhydrase inhibitor or mannitol is typically avoided in patients with sickle cell disease because of the risk of sickling caused by metabolic acidosis or volume depletion.

Surgical interventions are aimed at anterior chamber clot evacuation in patients with large persistent hyphema (≥Grade III for >10 days), early corneal bloodstaining, and uncontrolled IOP despite maximal medical therapy. Uncontrolled intraocular hypertension is defined as >60 mmHg for 2 days, >50 mmHg for 5 days, >35 mmHg for 7 days, or >25 mmHg for >24 h in patients with sickle hemoglobinopathy [17, 20].

7.4 Delayed Onset Glaucoma

7.4.1 Angle Recession

Angle recession occurs due to separation between the longitudinal and circular muscle fibers of the ciliary body. Intractable secondary open-angle glaucoma may develop even many years later, up to 15 years following trauma, and raised IOP may be seen even in the contralateral eye (Figs. 7.5 and 7.6). In the presence of a hyphema, an angle recession should be suspected in the majority of eyes [19]. The angle recession itself does not cause outflow obstruction, but represents a visible marker of invisible degenerative damage. However, the total number of patients who develop glaucoma following an angle recession is only 7–9% [21, 22]. The risk of glaucoma appears to be greater if more than 180° or 240° of angle recession is present. Raised IOP in the contralateral eye of the patient's angle recession may be up to 50% [23, 24]. However even 360 degree angle recession in the absence of raised IOP does not require treatment.

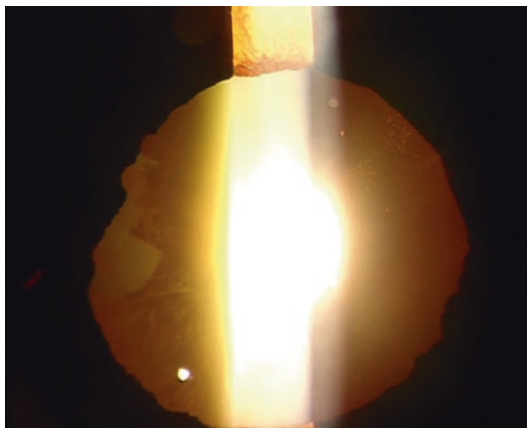

Fig. 7.5 Traumatic sphincter rupture

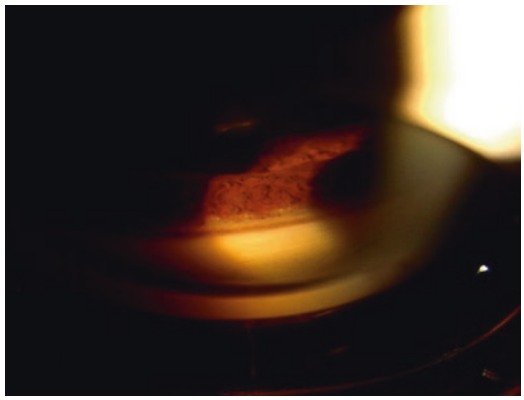

Fig. 7.6 Angle recession

7.4.2 Management

Antiglaucoma medicines that increase outflow and/or laser trabeculoplasty usually have a poor response due to damaged trabecular meshwork [25, 26]. Aqueous suppressants are generally used to lower elevated IOP. Trabeculectomy augmented with antimetabolites is the first-line surgical therapy and is associated with the greatest reduction of IOP and the fewest postoperative glaucoma medications [27, 28] (Figs. 7.7 and 7.8).

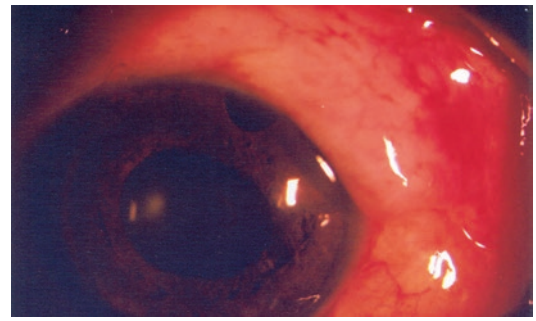

Fig. 7.7 Diffuse bleb with shallow AC

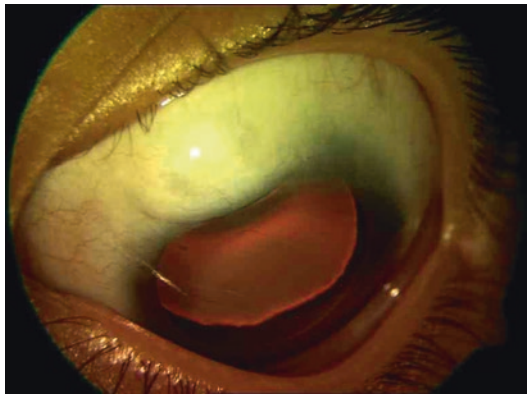

Fig. 7.8 Small localized bleb with subluxation of lens

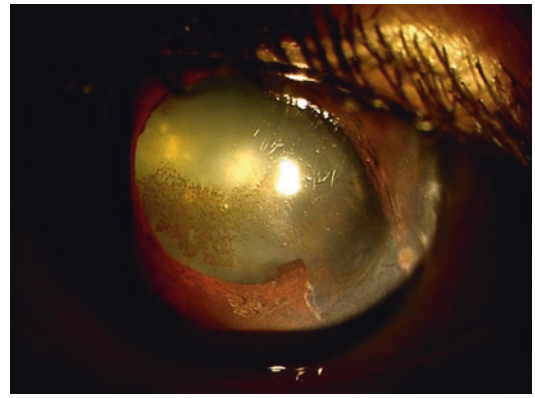

Fig. 7.9 Post penetrating Injury loss of Iris pigment on lens, shallow AC

7.5 Ghost Cell Glaucoma

First described by Campbell [29], this type of glaucoma occurs in patients who have had a vitreous hemorrhage. The hemoglobin is absorbed, and the rigid shells of RBCs travel to the anterior chamber and block the trabecular meshwork, leading to a raised IOP. A tan-colored pseudohypopyon forms once these cells precipitate. A **'candy-striped' sign** is present when a mix of fresh red blood cells and ghost cells layers out. The diagnosis is confirmed by demonstrating thin-walled, hollow erythrocytes with a shrunken appearance in an anterior chamber aspirate specimen under phase contrast or light microscopy [30] (Fig. 7.9).

7.5.1 Management

In most cases, raised IOP lasts for several months, and treatment with topical aqueous suppressants is sufficient to control IOP. Cases with dense hemorrhage or pressure not adequately controlled by medical therapy may require an anterior chamber wash or vitrectomy to remove all the remaining ghost cells. In refractory cases, anterior chamber lavage and removal of the blood source (vitrectomy in the presence of vitreous hemorrhage) are indicated. In the event that the lens is ruptured or dislocated, it may need to be removed out simultaneously, as lens proteins or position may also play a part in the IOP rise (Figs. 7.10 and 7.11).

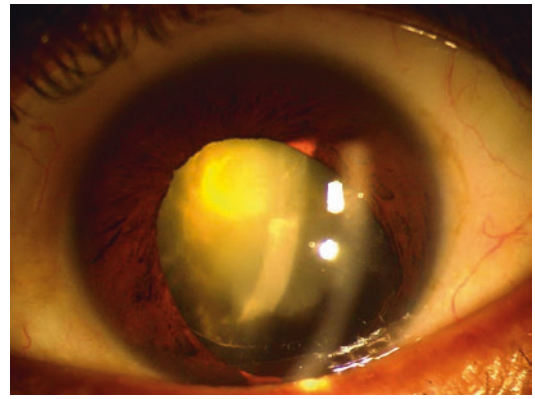

Fig. 7.10 Traumatic subluxation with cataract

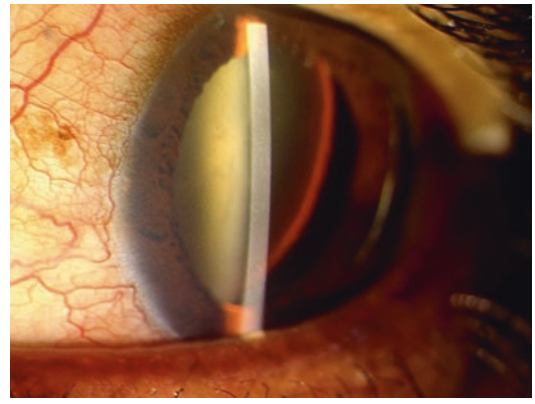

Fig. 7.11 Traumatic posterior subluxation

7.6 Penetrating Trauma

Glaucoma following penetrating injury occurs usually after a few weeks or months. The origin of penetrating injuries can be blunt force, sharp objects, or lacerations of the globe. A gonioscopic evaluation needs to be done to discover an unsuspected foreign body at the angle as and when the eye is safe enough for the procedure.

7.6.1 Mechanisms of Glaucoma Secondary to Penetrating Trauma

Secondary angle closure due to peripheral anterior synechiae is the most common mechanism for the development of glaucoma in penetrating eye injuries.

Epithelialization of the anterior chamber can occur in cases of improper primary surgery and may lead to glaucoma through several mechanisms, including growth over the trabecular meshwork and peripheral anterior synechiae formation [31]. Iron released from intraocularly retained iron objects is toxic to the trabecular meshwork, leading to decreased outflow and elevated IOP (siderosis). Copper may be oxidized within the eye, causing similar trabecular changes as with iron but with less frequency (chalcosis) (Figs. 7.12 and 7.13).

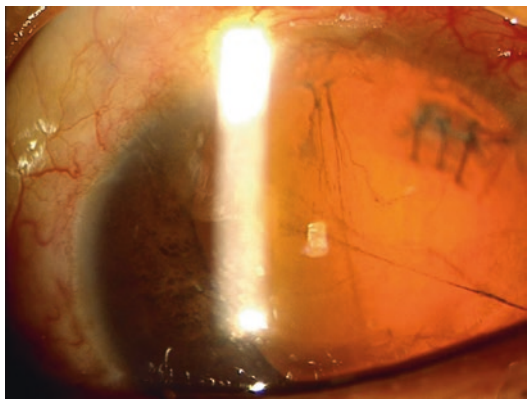

Fig. 7.12 Corneal wound repair with aphakia

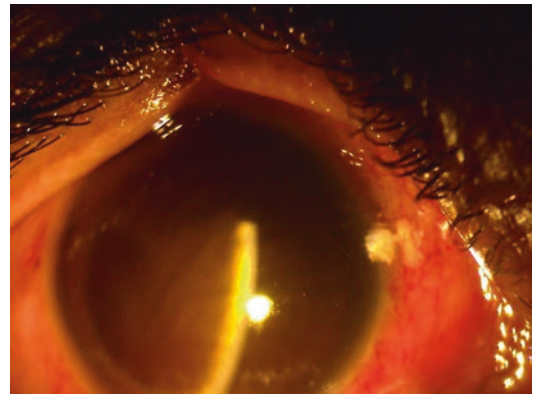

Fig. 7.13 Blood staining cornea

7.6.2 Management

The management consists of globe closure with rapid removal of the foreign objects, if any. Corticosteroids oral as well as topical need be given to avoid severe inflammation, cyclitic membranes and sympathetic ophthalmia. Antibiotics in appropriate dosage are given for endophthalmitis prophylaxis. Raised IOP can be treated with beta-adrenergic antagonists, carbonic anhydrase inhibitors, alpha-2-agonists, and hyperosmotic agents. Filtering surgery is required for uncontrolled IOP. Glaucoma drainage devices are required in eyes that have very badly damaged conjunctiva or a deformed anterior segment.

7.7 Chemical Injuries and Secondary Glaucoma

7.7.1 Alkali Burns

Alkaline substances may penetrate into the eyeball within seconds of contact, causing severe damage to the anterior segment structures. These types of burns may produce characteristic IOP changes with an initial rapid rise, which has been attributed to anterior segment shrinkage and increased uveal blood flow [32, 33]. These effects may be prostaglandin-mediated. Trabecular meshwork may undergo irreversible damage, as a direct consequence of the original alkali burn or

due to secondary angle closure as a result of peripheral anterior synechiae. Pupillary block due to inflammation leading to iris bombe and angle closure may be yet another reason for IOP rise in the late phase.

7.7.2 Management

Raised IOP in the early phase can be controlled with beta-adrenergic antagonists, carbonic anhydrase inhibitors, alpha-2-agonists, and hyperosmotic agents. The damage caused by alkali burns is mostly due to a severe inflammatory reaction. Topical corticosteroids must be used for the first two weeks but are contraindicated beyond that period because of the risk of stromal lysis [34]. Surgical intervention should be the last option in these patients, and if required, a glaucoma drainage device is the best possible option as a primary procedure, as trabeculectomy does not work due to associated damage to the ocular surface. Acid burns cause coagulation of surface tissues, so penetration inside the eye is less, but if high concentrations of acid are the cause, it behaves almost like alkali.

7.7.3 War Time Injuries and Glaucoma

During war, most of the injuries are penetrating or blast, and both are lethal to the eye. Blast injury is associated with contusion, and if impact is less, the globe remains intact, but severe tissue trauma occurs as a result, and glaucoma is seen more often than contusion or closed globe injury in civil set-ups. Another association of blast is the chemical and physical burning of the ocular surface, making the conjunctiva unhealthy for glaucoma surgery. Penetrating injuries are more common and have a higher morbidity due to the severity of the damage and the often-delayed treatment. If these eyes survive with a high IOP, initial treatment is done with antiglaucoma medications like betablockers, alpha-2 agonists, and topical CAI inhibitors. Prostaglandins are withheld until inflammation subsides completely. As and when surgery is needed, the main choice remains between glaucoma drainage devices and cyclodestructive procedures.

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