Traumatic Glaucoma

The etiology and management of glaucoma after ocular trauma.

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In the United States, the average annual rate of hospitalization for ocular trauma as a principal diagnosis is 13.2 cases per 100,000 hospital admissions. Ocular trauma occurs more often in the young than the old and among men versus women. The type and severity of ocular trauma generally depend on the age group and the circumstances surrounding the injury. Children are mostly injured while at play; young adults frequently sustain the trauma while involved in sports, assaults, and car accidents; and older adults are typically injured at work or as a result of domestic violence.

Multiple factors determine if an injury will produce blunt, nonpenetrating/nonperforating trauma with or without hyphema, or penetrating/perforating trauma with or without the presence of an intraocular foreign body. Other frequent causes of ocular injury include chemical burns, radiation, and electrical accidents. This article discusses the various etiologies of traumatic glaucoma and its management.

INTRAOCULAR PRESSURE AFTER OCULAR TRAUMA

During the early posttraumatic period, the IOP may be low or high. Excluding rupture of the globe, relative hypotony may result from retinal detachment, a reduction in aqueous formation due to uveitis or uveal effusion, or an increase in outflow secondary to disruption of angle architecture or cyclodialysis. Elevated IOP may result from associated uveitis, trabecular dysfunction, or physical obstruction of the angle by hyphema, a dislocated lens, a shallow anterior chamber due to posterior segment effusion or hemorrhage, disorganization of the intraocular structures, or angle closure owing to diverse mechanisms such as pupillary block and more posterior mechanisms.

After appropriate intervention for the acute cause of elevated IOP without penetrating injury, the IOP of most patients with mild or moderate trauma and common disorders such as hyphema or uveitis often normalizes days or weeks after surgery. Some of these patients, particularly those with a hyphema or traumatic injury to angle structures, may develop persistently elevated IOP and glaucomatous damage weeks, months, years, or even decades after the initial injury. For this reason, all individuals with a history of significant ocular trauma require lifelong surveillance for the onset and progression of ocular hypertension (OHT) and/or glaucoma.

GLAUCOMA ASSOCIATED WITH HYPHEMA

Hyphema is a very common sequela of ocular trauma and is characterized by the presence of erythrocytes within the anterior chamber. The most common source of bleeding is trauma to the anterior face of the ciliary body, which disrupts the major arterial circle of iris.

“Rebleeding” can occur, most often in the first week. Patients with acute hyphema should be counseled to avoid significant exertion or strain until healed. The frequency of rebleeding is influenced by the presence of hypotony or hypertension, the patient’s use of anti-coagulants, and African ancestry.

The management of uncomplicated hyphema should be conservative, with topical steroids, cycloplegia, protective eyewear to prevent further trauma, and eleva-
tion of the head. The OHT associated with hyphema also calls for conservative treatment, with or without oral carbonic anhydrase inhibitors (CAIs), as needed and as tolerated. Elevated IOP in the setting of hyphema usually responds well to topical suppressants of aqueous humor formation (often in the form of beta-adrenergic antagonists, alpha 2-adrenergic agonists, and CAIs, although prostaglandin analogues can also be tried). Systemic CAIs in patients with sickle cell hemoglobinopathies should be avoided when possible, because acidosis increases the propensity for sickling. If the patient’s elevated IOP cannot be controlled medically, and it threatens corneal staining or the function of the optic nerve, surgical intervention is appropriate. Treatment can take the form of paracentesis (once or more than once), anterior chamber washout, or more invasive glaucoma surgery such as trabeculectomy.²⁴

GLAUCOMAS ASSOCIATED WITH DEGENERATED ERYTHROCYTES

Ghost cell glaucoma results from IOP raised by degenerated erythrocytes (ghost cells), which lose their hemoglobin in the vitreous cavity and subsequently migrate forward to the anterior chamber via a disrupted anterior hyaloid face. Ghost cells are spherical and more rigid than normal erythrocytes and have a tendency to obstruct the trabecular meshwork. Although the diagnosis is made clinically, an anterior chamber paracentesis can confirm the cells’ presence cytologically. Ghost cell glaucoma will persist until the supply of ghost cells is depleted. The management is standard glaucoma therapy, but vitrectomy is effective for eliminating the remaining reservoir of erythrocyte ghosts.

Hemolytic glaucoma is a rare form of open-angle glaucoma that can occur days to weeks after an intraocular hemorrhage. Gonioscopy reveals an open angle, but the trabecular meshwork is covered with reddish-brown pigment. The diagnosis is confirmed cytologically: characteristically, testing reveals macrophages containing golden-brown pigment. The elevated IOP is secondary to the obstruction of the trabecular meshwork by these hemoglobin-laden macrophages. Recalcitrant cases may need anterior chamber washout and/or pars plana vitrectomy.

Hemosiderotic glaucoma is a rare condition that occurs when hemoglobin becomes phagocytosed by endothelial cells of the trabecular meshwork. The iron liberated from hemoglobin may cause siderosis of the trabecular meshwork, which can lead to increased outflow resistance.

TRAUMATIC ANGLE DEFORMITY

Nonpenetrating ocular trauma often leads to recession of the anterior chamber angle, which represents a
The diagnosis of angle recession is made based on the history of blunt trauma that led to unilateral glaucoma or a history of traumatic hyphema. Gonioscopy is the gold standard for the diagnosis of angle recession, and it is often aided by a comparison of the two eyes (Figures 1 and 2). Angle recession may encompass small or large areas of the angle. The clinical appearance is that of a widened ciliary body band during gonioscopy.

"Angle recession should be viewed as ... a risk factor for the future development of ocular hypertension or glaucoma."

The pathophysiology of angle-recession glaucoma consists of mechanical disruption from physical force to the angle structures and an elevation in IOP. Blunt trauma pushes the iris against the lens, creating reverse pupillary block and forcing aqueous into the angle recess. This hydrodynamic pressure splits the circular and longitudinal muscle fibers of the ciliary body. An acute rupture of the angle vasculature and permanent damage to the angle ensue. Splitting the ciliary body muscle may lead to hyphema secondary to disruption of the major arterial circle of the iris located in the ciliary body. Loss tension from the ciliary muscle on the scleral spur can further compromise outflow. In general, the presence of angle recession should be viewed as a marker of injury to the trabecular meshwork and a risk factor for the future development of OHT or glaucoma. The frequency of periodic reassessment should be determined by the health of the eye, age of the patient, level of IOP, and other patient-related and ocular factors.

GLAUCOMA AFTER PENETRATING TRAUMA

Penetrating ocular trauma can result from blunt, sharp, or missile injury. Initially, the IOP tends to be low because of uveitis and a penetrating injury. An intraocular foreign body is reportedly present in 6% of assault cases and 35% of occupational injuries.

Glaucoma secondary to an unsuspected remaining fragment may develop years after the injury. Iron and copper are known to cause toxicity and damage to intraocular tissues. The former is toxic to both the retina and the trabecular meshwork. Glaucoma is much more common with siderosis than chalcosis.

OTHER CAUSES OF GLAUCOMA AFTER TRAUMA

Permanent ocular injury can result from alkali, acid, or thermal injury. Alkali burns are more severe than acid burns and may lead to an early, rapid increase in IOP as a result of initial corneal and scleral shrinkage. Vascular remodeling and venous congestion after radiation may cause the IOP to rise. Electrical injury only leads to transient increases in IOP and may be associated with the release of iris pigment.

CONCLUSION

Caused either by particulate obstruction or frank damage to the outflow structures, glaucoma associated with trauma can be multifactorial in etiology. The treating clinician must be vigilant or risk missing early-, intermediate-, or late-onset glaucoma that, if not treated, could lead to a devastating loss of vision.

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