Chapter 11

GLAUCOMA ASSOCIATED WITH OCULAR TRAUMA

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INTRODUCTION

The definition of glaucoma has undergone considerable change in the last 20 years. As commonly used today, *glaucoma* refers to a diverse group of eye disorders characterized by progressive loss of axons from the optic nerve, resulting in loss of visual function as manifested in the visual field.[1] The structural changes in the optic nerve head are recognizable, and the patterns of visual field damage are characteristic but not specific. Glaucoma may thus be considered to be an optic neuropathy. For purposes of this discussion, the term *glaucoma* is used to describe conditions resulting in high intraocular pressure (IOP); eventual optic neuropathy is presumed to be the long-term result if the elevated IOP is not adequately lowered.

In the population without glaucoma, approximately 95% of people have IOPs between 11 and 21 mm Hg. An IOP above 24 mm Hg is considered elevated. Although the exact etiology of glaucomatous optic neuropathy is not known, many risk factors have been identified, with elevated IOP considered to be one of the most important. The risk of glaucoma increases with the level of IOP, and the risk of disease progression decreases as the pressure is lowered. Nevertheless, not all patients with elevated IOP develop glaucoma, and not all patients with glaucoma have elevated IOP. In the context of ocular trauma, patients with glaucoma *may* present with optic neuropathy but *almost assuredly will* present with elevated IOP. Because changes in the eye following trauma can result in significant elevations of IOP, it is presumed that optic nerve damage will occur if the pressure remains high enough long enough, even if the optic nerve is normal at the time of presentation.

Although glaucomatous optic neuropathy may take some time to develop, elevated IOP after ocular trauma can occur immediately after the injury or at any time in the future, even years later. Conditions associated with elevated IOP at the time of injury include the following:

- alterations in outflow from inflammation and inflammatory byproducts,
- hyphema with or without pupillary block,
- subluxation of the lens, and
- increased episcleral venous pressure.

Elevated IOP may also be a consequence of changes in ocular tissues due to chemical injuries or from damage to nonocular structures. Weeks, months, or years following injury, IOP may become elevated due to chronic use of corticosteroids for control of inflammation, secondary angle closure, ghost cells, scar tissue formation, effects of retained foreign bodies, or damage to the drainage structures (angle recession). For purposes of this discussion, *early* refers to the period from the time of injury through the first 2 weeks following injury, whereas *late* refers to any time after the first 2 weeks.

GLAUCOMA OCCURRING EARLY FOLLOWING OCULAR TRAUMA

Inflammation

Inflammation of varying degrees invariably follows any injury to the eye. Although iritis and iridocyclitis usually cause lower IOP because of decreased aqueous formation, elevated IOP may occur through a variety of mechanisms.[2] Leakage of proteins into the anterior chamber from the increased vascular permeability that accompanies inflammation may result in elevated IOP through the osmotic influx of water. Inflammatory cells and other particulate debris (eg, blood, fibrin, iris pigment, lens material, vitreous) can mechanically block the outflow pathways. Figure 11-1 shows a typical inflammatory reaction in the anterior chamber with fibrin formation, which may lead to pupillary block and secondary angle closure, or an open-angle glaucoma due to outflow pathway obstruction. Blunt trauma can also cause direct damage to trabecular endothelial cells or to meshwork extracellular components, resulting in elevated IOP.[3]

Alterations in Lens Position

Blunt trauma may cause zonular disruption, resulting in dislocation of the lens. Partial subluxation may allow prolapse of vitreous into the anterior chamber. This process may, in itself, result in elevated IOP, probably by an osmotic mechanism. Anterior movement of vitreous may also cause a pupillary block with secondary angle closure. Partial subluxation of the lens is illustrated in Figure 11-2. Complete dislocation of the lens into the anterior chamber may result in pupillary block and angle closure as shown in Figure 11-3.

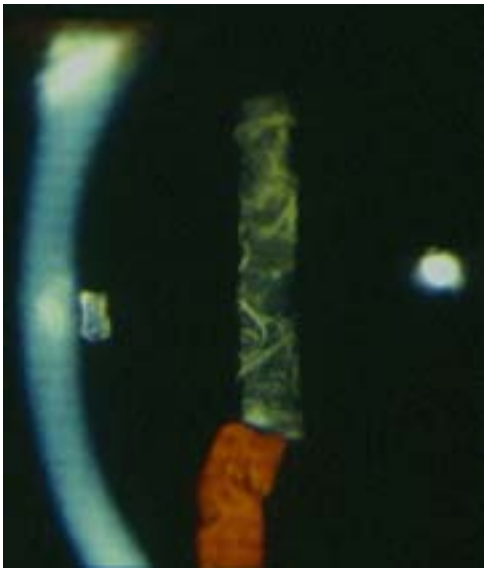

Fig. 11-1. Intraocular inflammation. Severe intraocular inflammation, as can occur following trauma, may result in the formation of a fibrinous exudate, seen here. Fibrin, inflammatory debris, and cells may obstruct the outflow pathways and cause elevated intraocular pressure. Reproduced with permission from Meyer J, Katz L. Secondary open-angle glaucomas. In: Choplin NT, Lundy DC, eds. *Atlas of Glaucoma.* London, England: Martin Dunitz, Ltd; 1998: Figure 9.14(a).

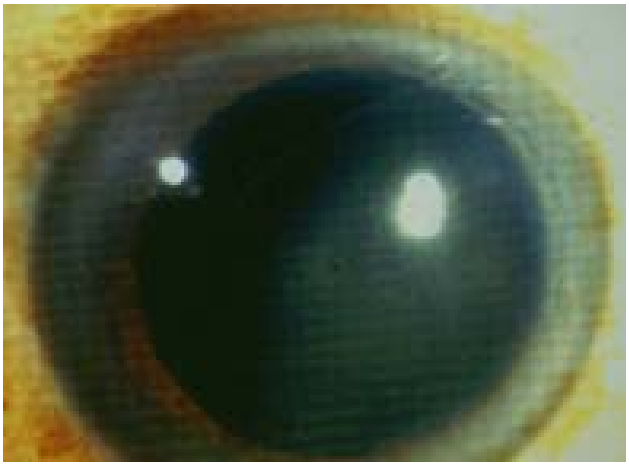

Fig. 11-2. Blunt trauma may rupture zonules and cause total or partial dislocation of the crystalline lens. This lens is partially dislocated inferiorly and temporally. Vitreous may come around the edge of the lens and cause a pupillary block or direct elevation of intraocular pressure. Reproduced with permission from Meyer J, Katz L. Secondary open-angle glaucomas. In: Choplin NT, Lundy DC, eds. *Atlas of Glaucoma.* London, England: Martin Dunitz, Ltd; 1998: Figure 9.13.

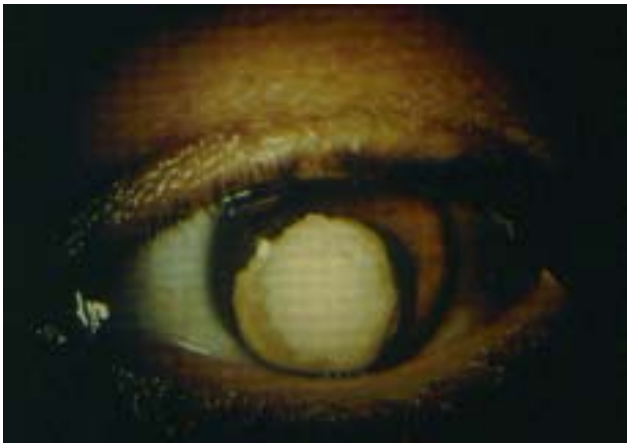

Fig. 11-3. This photograph shows subluxation of the lens into the anterior chamber, causing pupillary block and secondary angle closure glaucoma. Reproduced with permission from Liebmann JM, Ritch R, Greenfield DS. The angle-closure glaucomas. In: Choplin NT, Lundy DC, eds. *Atlas of Glaucoma.* London, England: Martin Dunitz, Ltd; 1998: Figure 10.6.

Hyphema

Bleeding into the anterior chamber, or hyphema (Figure 11-4), is discussed in detail in Chapter 8, Blunt Trauma and Nonpenetrating Injuries of the Anterior Segment. Blood in the anterior chamber may cause elevated IOP by mechanically blocking outflow channels. A total, or "eight-ball," hyphema

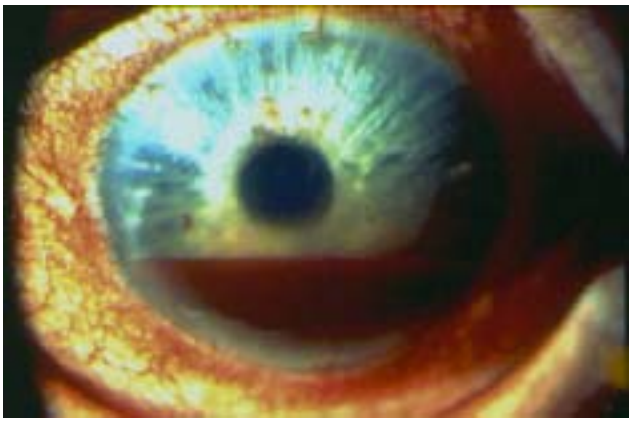

Fig. 11-4. Hyphema. Blood in the anterior chamber from trauma may elevate intraocular pressure by blocking outflow pathways or by causing pupillary block and secondary angle closure. Reproduced with permission from American Academy of Ophthalmology. *Ophthalmology Study Guide.* San Francisco, Calif: American Academy of Ophthalmology; 1987: Figure 42.

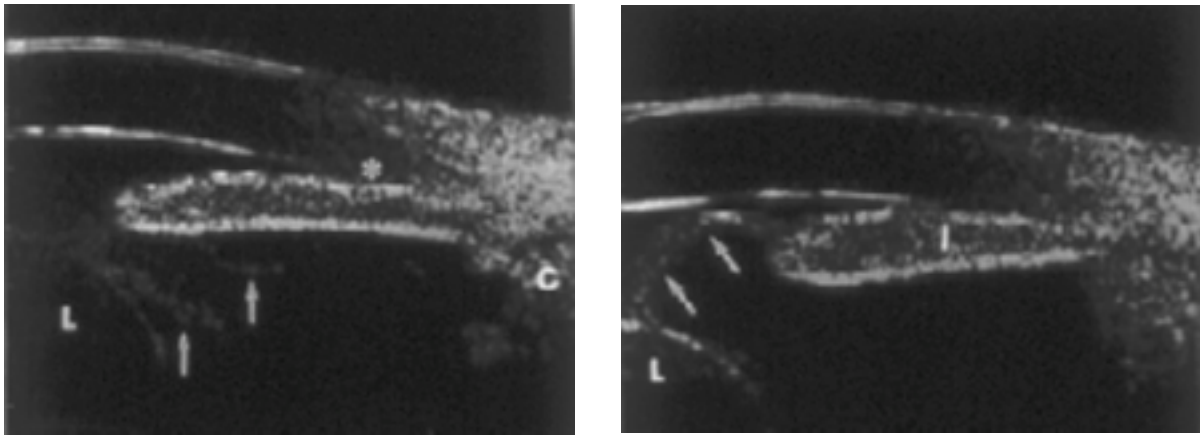

Fig. 11-5. Ultrasound biomicroscope image of an eye following blunt trauma. (**a**) This image shows a subluxed lens (L) and broken zonules (arrows), disinserted from the ciliary body (C). The angle is closed (indicated with an asterisk) due to pupillary block caused by the clotted blood and (**b**) anteriorly displaced vitreous (arrows). The iris (I) is pushed up against the cornea. Reproduced with permission from Berinstein DM, Gentile RC, Sidoti PA, et al. Ultrasound biomicroscopy in anterior ocular trauma. *Ophthalmic Surg Lasers.* 1997;28:201–207.

or an organized clot of sufficient size can cause pupillary block. Figure 11-5[4] is an ultrasound biomicroscope image from an eye with a subluxed lens with clotted blood and anterior movement of vitreous, resulting in pupillary block. The iris is apposed to the trabecular meshwork, resulting in secondary angle closure.

Phacoanaphylactic Glaucoma

Lens protein is normally sequestered within the capsular bag from the time of embryonic development. If the lens is ruptured by trauma (usually penetrating trauma), the immune system reacts to the released lens protein with a granulomatous inflammatory response, often associated with elevated IOP. This phenomenon is illustrated in Figure 11-6.

Increased Episcleral Venous Pressure

Orbital hemorrhage from trauma may result in proptosis and marked congestion within the orbit, compressing the orbital veins and preventing drainage from the outflow channels. The resulting increase in episcleral venous pressure may cause a significant rise in IOP.

Glaucoma Following Chemical Injuries

Alkali burns can cause extensive damage to the anterior segment; glaucoma may result acutely from the inflammatory response accompanying the injury. Alkali can also cause direct damage to the outflow pathways, leading to elevated IOP.[5] However, elevation of IOP may occur almost immediately following the injury, due to scleral shrinkage and pros-

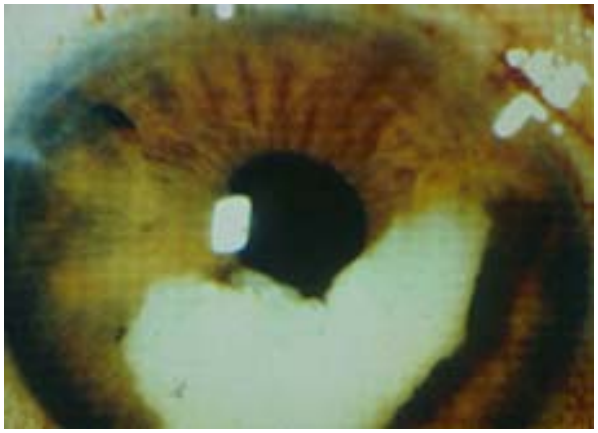

Fig. 11-6. Phacoanaphylactic uveitis and glaucoma. The lens in this eye has been ruptured; cortical material has been released into the anterior chamber, resulting in a massive inflammatory response. The inflammation, along with the osmotic effect of the lens protein's pulling water into the anterior chamber, may cause marked increases in intraocular pressure. Reproduced with permission from Meyer J, Katz L. Secondary open-angle glaucomas. In: Choplin NT, Lundy DC, eds. *Atlas of Glaucoma.* London, England: Martin Dunitz, Ltd; 1998: Figure 9.26(d).

taglandin-mediated alterations in ocular blood flow.[6] See Chapter 7, Chemical Injuries of the Eye, for a more extensive discussion of chemical injuries of the eye.

Glaucoma Due to Trauma to Nonocular Structures

Anything that impedes venous drainage from the head can cause increased episcleral venous pressure and elevated IOP. Theoretically, a chest injury causing a superior vena caval syndrome or a neck injury with jugular vein obstruction may increase IOP. A more common occurrence is secondary glaucoma due to development of a carotid-cavernous sinus fistula following head trauma. Figure 11-7 demonstrates the vascular congestion that may be seen with the high-flow type of arteriovenous shunt that occurs with a carotid-cavernous sinus fistula.[7] The arterialization of the venous drainage increases resistance to drainage of blood from the eye, and, as the "sewer" backs up, the drainage of aqueous humor is also impeded. This can result in very high IOP.

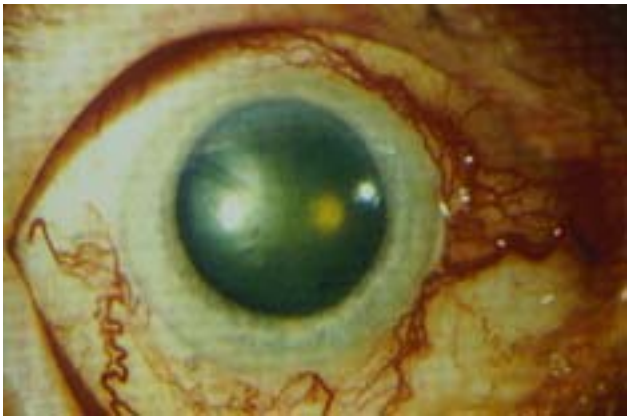

Fig. 11-7. Carotid–cavernous sinus fistula. Intense vascular congestion with engorgement of episcleral veins is seen in this external view. The resulting increase in episcleral venous pressure may cause marked increases in intraocular pressure. Patients with this condition are also at risk for ocular ischemia, possibly leading to neovascular glaucoma. Reproduced with permission from Meyer J, Katz L. Secondary open-angle glaucomas. In: Choplin NT, Lundy DC, eds. *Atlas of Glaucoma.* London, England: Martin Dunitz, Ltd; 1998: Figure 9.38.

GLAUCOMA OCCURRING LATE FOLLOWING OCULAR TRAUMA

Steroid-Induced Glaucoma

Glaucoma following trauma may occur as a result of the treatment as well as from the injuries to the eye. Prolonged use of corticosteroids may elevate IOP and lead to glaucomatous optic neuropathy in susceptible individuals, even in the absence of trauma.[8] Figure 11-8 shows the appearance of the visual field in one eye from a patient with steroid-induced glaucoma. The patient was a physician who self-medicated with steroid drops for ocular allergy. Elevated IOP from chronic steroid use may occur from the accumulation of fibrillar material within the intertrabecular and intratrabecular spaces. Enzymes that would normally clear this material are thought to be inhibited by the corticosteroids.

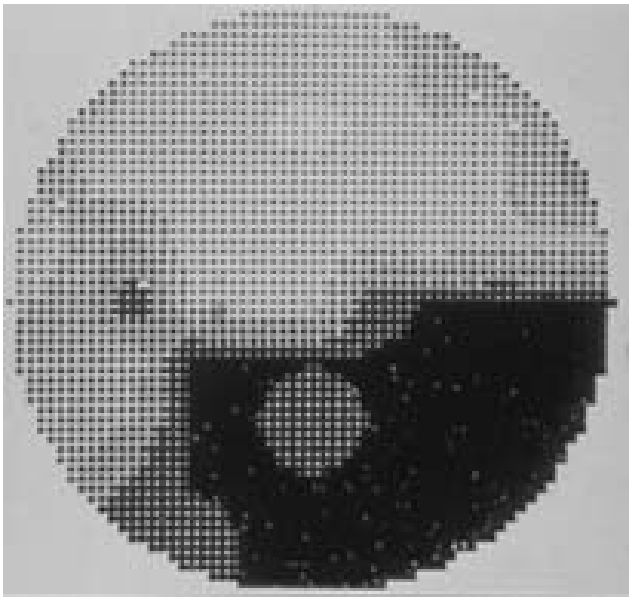

Fig. 11-8. Visual field from the left eye of a physician who self-prescribed topical steroids for allergic eye disease for many years, resulting in steroid glaucoma. Reproduced with permission from Meyer J, Katz L. Secondary open-angle glaucomas. In: Choplin NT, Lundy DC, eds. *Atlas of Glaucoma.* London, England: Martin Dunitz, Ltd; 1998: Figure 9.17.

Secondary Angle Closure

Organization of anterior chamber blood, inflammatory products, or both, may result in the formation of both anterior (between the iris and angle structures) and posterior (between the pupillary border and the lens) synechiae. Three hundred sixty degrees of posterior synechiae, resulting in seclusion of the pupil, will cause pupillary block. These conditions may lead to secondary permanent clo-

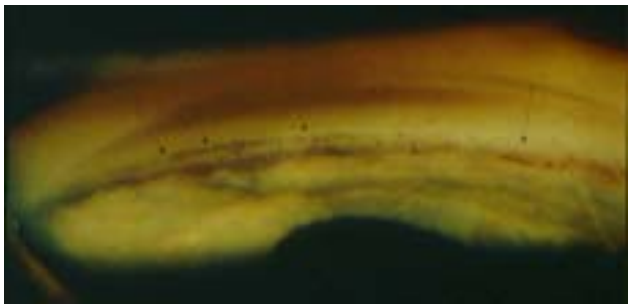

Fig. 11-9. Peripheral anterior synechiae, showing the types of adhesions (arrows) that can occur between the peripheral iris and the anterior chamber angle structures following chronic inflammation in the eye. When synechiae extend around the angle for 360°, outflow is extremely compromised and secondary angle closure glaucoma develops. Reproduced with permission from Fellman RL. Gonioscopy. In: Choplin NT, Lundy DC, eds. *Atlas of Glaucoma.* London, England: Martin Dunitz, Ltd; 1998: Figure 5.12(c).

sure of the anterior chamber angle, resulting in late glaucoma following trauma. Untreated pupillary block from subluxation or dislocation of the lens can also result in permanent angle closure. Peripheral anterior synechiae with partial secondary angle closure is demonstrated in Figure 11-9.

Ghost Cell (Hemolytic) Glaucoma

Following long-standing vitreous hemorrhage or hyphema, red blood cells, having undergone normal senescence, lose their hemoglobin and their normal cell membrane malleability. The resulting "ghost" cells lose the ability to flex and move through the intertrabecular spaces of the trabecular meshwork, becoming trapped within the meshwork and reducing aqueous outflow. This phenomenon increases IOP and leads to a secondary type of open-angle glaucoma known as ghost cell glaucoma.[9] The trapped ghost cells and the inflammatory response to them may be seen in Figure 11-10.

Posttraumatic Angle Deformity (Angle Recession)

The transient rise in IOP that occurs at the time of a blunt injury may abruptly force aqueous into the anterior chamber angle and cause tears in the face of the ciliary body at its insertion into the scleral spur. The amount of tearing and the percentage of the angle involved is highly variable and not necessarily correlated to the specific type of injury. The gonioscopic appearance of this type of angle damage is that of an apparent widening of the ciliary body band in the involved area, usually with a noticeable drop-off between the normal and recessed areas. Variable

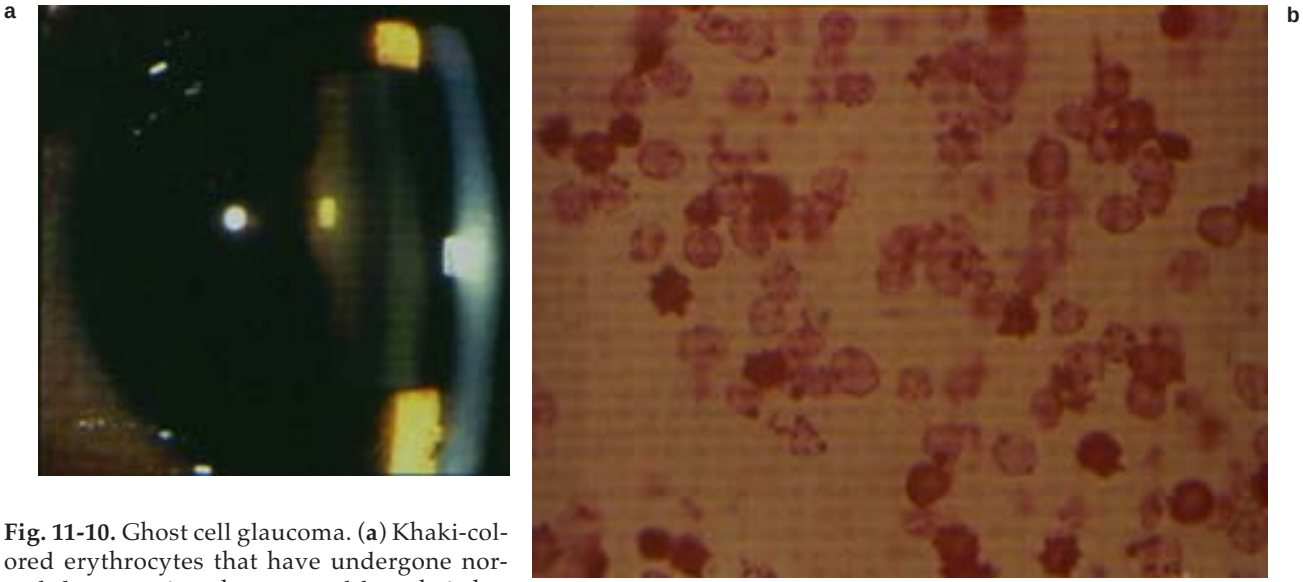

Fig. 11-10. Ghost cell glaucoma. (**a**) Khaki-colored erythrocytes that have undergone normal degenerative changes and lost their hemoglobin may be seen in the anterior chamber and anterior vitreous on slitlamp examination. (**b**) Ghost cells, along with the macrophage response, are seen in this vitrectomy specimen. Reproduced with permission from Meyer J, Katz L. Secondary open-angle glaucomas. In: Choplin NT, Lundy DC, eds. *Atlas of Glaucoma.* London, England: Martin Dunitz, Ltd; 1998: Figure 9.22.

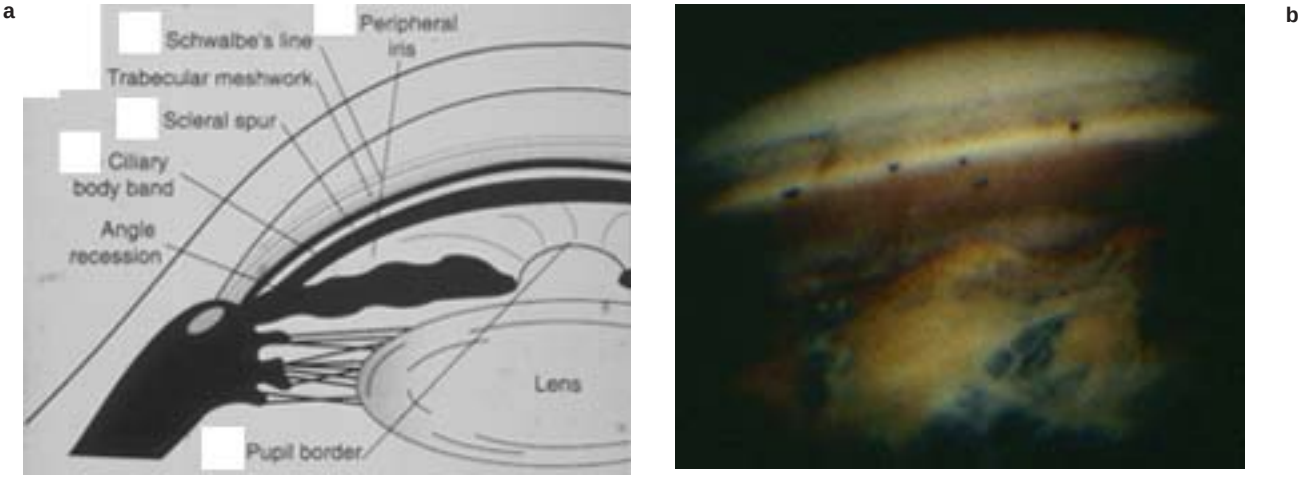

Fig. 11-11. (**a**) Posttraumatic angle deformity (angle recession). (**b**) In this photomicrograph, the widened appearance of the recessed angle is evident by the visible sclera anterior to the iris root. Scattered pigmentation is also seen in the angle. Reproduced with permission from Fellman RL. Gonioscopy. In: Choplin NT, Lundy DC, eds. *Atlas of Glaucoma.* London, England: Martin Dunitz, Ltd; 1998: Figure 5.17.

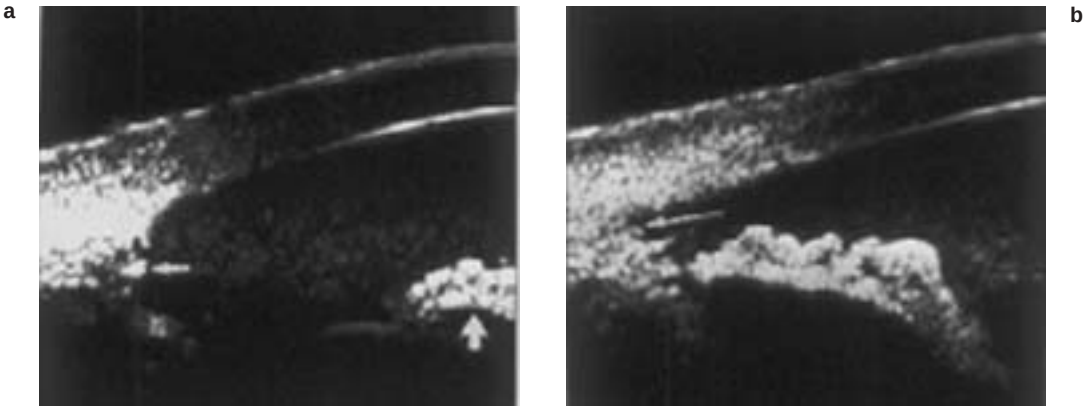

Fig. 11-12. Ultrasound biomicroscope image of angle recession. (**a**) The large arrow (right) points to an area of iridodialysis following blunt trauma, with a small remnant of iris attached to the ciliary body indicated by the small arrow (left). (**b**) The arrow (left) points to an area of angle recession in the same eye. Reproduced with permission from Berinstein DM, Gentile RC, Sidoti PA, et al. Ultrasound biomicroscopy in anterior ocular trauma. *Ophthalmic Surg Lasers.* 1997;28:201–207.

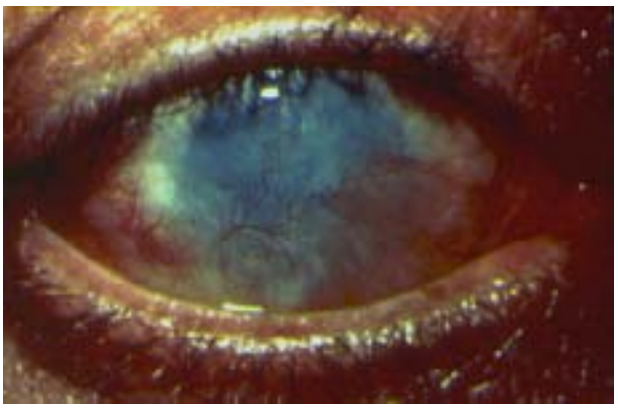

Fig. 11-13. Scarring following an alkali injury. This photograph shows the typical appearance of an eye following an alkali burn after the healing process is completed, with vascularization of the cornea and marked disruption of the anterior segment. Glaucoma may result from scleral shrinkage. Reproduced with permission from American Academy of Ophthalmology. *Ophthalmology Study Guide.* San Francisco, Calif: American Academy of Ophthalmology; 1987: Figure 45.

Fig. 11-14. Heterochromia from siderosis bulbi. The iris of this patient's affected right eye shows darkening compared with the normal (left) eye because of the deposition of iron from a retained intraocular foreign body. Reproduced with permission from Kanski JJ, Nischal KK. *Ophthalmology: Clinical Signs and Differential Diagnosis.* London, England: Harcourt Publishers Ltd; 2000: 189.

changes in pigmentation in the angle are also seen.

The mechanism by which IOP becomes elevated in eyes with angle recession is not clear, and which eyes with angle recession will later develop increased IOP with glaucoma cannot be predicted. It is possible that the increase in IOP is due in part to sclerosis within the trabecular meshwork, or it may be due to the change in forces exerted on the meshwork by the ciliary muscle that has been damaged. One study[10] detected glaucoma in only 8% of eyes with 360° of recession. In addition, patients who develop angle recession glaucoma may have an underlying predisposition to glaucoma; one study[11] showed that 50% of patients with angle recession glaucoma had evidence of glaucoma in the fellow (nontraumatized) eye.

Glaucoma has been reported[12] to occur up to 20 years after injury, and it is therefore necessary for patients who have had blunt eye trauma to have periodic eye examinations for the remainder of their lives. Population-based studies[13] suggest that the risk of developing glaucoma in an eye with some degree of angle recession is up to 6%, which may be lower than that observed following perforating injuries. Figure 11-11 shows an area of posttraumatic angle deformity in a traumatized eye and a schematic drawing of the angle illustrating the concept of angle recession. Figure 11-12 demonstrates the findings of an ultrasound biomicroscopy study in an eye with angle recession.

Chemical Injuries

Elevated IOP and glaucoma may occur because of inflammatory changes and scarring that follow a chemical injury. Alkali burns can cause scleral shrinkage. Figure 11-13 shows the extensive type of anterior segment disruption that may occur after such an injury.

Glaucoma Following Penetrating and Perforating Injuries

All mechanisms that can elevate IOP after blunt trauma can also lead to increased IOP in eyes that have had penetrating or perforating injuries. In addition, eyes that have been ruptured may have fibrous ingrowth, epithelial downgrowth, or retained foreign bodies that may cause elevated IOP by different mechanisms.[14]

Siderosis Bulbi

Iron from retained metallic foreign bodies may deposit in the anterior segment, causing siderosis bulbi and retinal dysfunction, as well. The iris may become darker in color (Figure 11-14), and the IOP may become elevated from deposition of iron in the outflow pathways, resulting in inflammation and sclerosis.

TREATMENT

The general principles for the treatment of secondary glaucomas also apply to glaucomas associated with ocular trauma. Therapy should be directed first at the underlying cause of the glaucoma and second at lowering IOP. Thus, if inflammation is the primary problem, topical (and systemic, if necessary) corticosteroids should be used judiciously and then tapered to discontinuance as the conditions allow. Strong consideration should be given to cycloplegia as well, which not only may alleviate discomfort by relieving ciliary spasm but may also increase uveoscleral outflow and lower IOP. The guidelines for the treatment of hyphema (see Chapter 8, Blunt Trauma and Nonpenetrating Injuries of the Anterior Segment) should be followed as to the timing of evacuation of the blood. Pupillary block should be treated with iridotomy, and dislocated lenses should be removed.

The mainstay of medical therapy for secondary glaucoma is the use of aqueous suppressants such as β-adrenergic antagonists, carbonic anhydrase inhibitors (topical or systemic, as appropriate), and α_2 agonists. Adrenergic agonists (eg, dipivefrin and epinephrine) may also prove useful in the management of glaucoma associated with inflammation owing to their vasoconstrictive properties. Miotics

should be avoided, as they tend to further disrupt the blood–aqueous barrier, may shallow the anterior chamber, and can hasten the formation of synechiae. Prostaglandin analogues should probably be avoided as well, at least in the early glaucomas, because the effect of these agents on conditions associated with inflammation are not yet well established.

Should medical therapy fail, consideration should be given to surgery, depending on the level of IOP and the condition of the optic nerve. In general, argon laser trabeculoplasty does *not* work in secondary glaucoma and may even make the IOP worse. Increased failure rates from standard filtering procedures should be anticipated, and filtering surgery should be undertaken with the concomitant use of an antimetabolite, such as mitomycin-C or 5-fluorouracil. Consideration should also be given to the use of an aqueous shunt, especially if the eye is pseudophakic.

SUMMARY

Glaucoma must be considered as a possibility with every ocular injury, and it can occur by a variety of mechanisms at the time of the injury or anytime thereafter. Treatment must be directed at the underlying injuries, with the suppression of inflammation, removal of foreign material, and restoration of normal anatomy being the mainstays. Treatment of elevated IOP should rely on aqueous suppressants (β-adrenoreceptor antagonists, α_2 agonists, and carbonic anhydrase inhibitors); miotics and prostaglandin analogues, which have the potential for exacerbating inflammation in the early postinjury period, should be avoided. Surgery may be necessary for control of IOP if medical therapy fails, depending on the level of IOP and the assessed risk for glaucomatous optic neuropathy.

Decisions regarding return to duty must be individualized and based on the degree of injury, visual function, and the need (and availability) for continued medical treatment. Most likely it is the nature of the injury and not the IOP that determines if the service member is removed from the theater of operations or if he or she may be treated in theater. Each military service has its own guidelines regarding medical evacuation.

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