Chapter 8

BLUNT TRAUMA AND NONPENETRATING INJURIES OF THE ANTERIOR SEGMENT

GLENN C. COCKERHAM, MD*

Colonel, US Air Force (Ret); Cornea Service, Allegheny General Hospital, 420 East North Avenue, Suite 116, Pittsburgh, Pennsylvania 15212; formerly, Chief, Ophthalmology Service, Malcolm Grow Air Force Medical Center, Andrews Air Force Base, Maryland

INTRODUCTION

The anterior segment of the eye consists of the conjunctiva, cornea, anterior chamber, trabecular meshwork, iris, and crystalline lens. Despite the surrounding facial bones, which protect the orbital contents from lateral or tangential blows, the anterior segment is vulnerable to direct trauma. Blunt ocular trauma may occur on the battlefield from missile injury, concussive effects from high explosives, and physical blows. It may also occur in industrial accidents, sports injuries, domestic assault, violent crime, motor vehicle accidents, fireworks, and in other military and civilian settings.

The numerous sequelae of blunt anterior segment trauma (Exhibit 8-1) can cause transient or permanent visual loss and disability. These injuries may be associated with globe rupture and damage to the orbital bones; eyelids and ocular adnexa; and posterior ocular structures, including the retina, choroid, and optic nerve.

EXHIBIT 8-1

SEQUELAE OF ANTERIOR SEGMENT TRAUMA

- Conjunctiva
  - Conjunctival abrasion
  - Foreign body
  - Conjunctival laceration
  - Subconjunctival hemorrhage
- Cornea
  - Abrasion
  - Foreign body
  - Traumatic keratopathy
  - Corneal laceration/rupture
  - Ultraviolet radiation injury
- Iris
  - Traumatic iritis
  - Traumatic mydriasis
  - Iridodialysis
  - Traumatic hyphema
- Ciliary Body
  - Cyclodialysis
  - Angle recession
- Lens
  - Subluxation/Dislocation
  - Traumatic cataract
- Traumatic Glaucoma

Incidence of Ocular Trauma

From World War II to the Persian Gulf War, ocular injuries have accounted for 2% to 13% of wartime injuries.[1] Ocular injuries are also seen frequently in civilian trauma. Of 727 patients with maxillofacial trauma evaluated in a large urban hospital, 90 (1.24%) patients had a serious eye injury, including hyphema in 38 (0.52%) patients.[2] Of 6,254 eye admissions at a large, metropolitan eye hospital, 7.5% of the patients were admitted with ocular trauma; hyphema was the most common injury, present in 25% of the ocular trauma cases.[3] Severe injury (defined as hyphema, open globe, intraocular foreign body [IOFB], or orbital/facial fracture) was present in 5% of 3,184 patients with ocular trauma who were evaluated in an urban tertiary care hospital over a 6-month period.[4]

Prevention of Injuries

A polycarbonate protective lens will stop small-caliber gunshot, low-velocity projectiles (eg, blast shrapnel), and windblown dust and sand. It affords some protection against a direct blow to the eye. In the Arab–Israeli Yom Kippur War (6–24 October 1973), combatants who wore protective lenses sustained fewer ocular injuries than those who did not wear them.[1] Military ophthalmologists must emphasize that soldiers in the theater of operations must continuously wear their protective eyewear.

CONJUNCTIVA

The conjunctiva is a smooth, thin, transparent mucus membrane that covers the globe and lines the posterior eyelid surfaces. It consists of nonkeratinizing, stratified, columnar epithelium and an underlying stroma composed of connective tissue with blood vessels and lymphatic vessels. The portion overlying the eye—the bulbar conjunctiva—is loosely attached to the underlying episclera and sclera, whereas the portion lining the eyelids—the palpebral conjunctiva—is more firmly attached.

Blunt trauma may damage the conjunctival layer by means of scrapes and tears to the conjunctiva itself, and by allowing blood to pool in and trapped air to enter the subconjunctival layers.

Conjunctival Abrasions and Lacerations

Conjunctival abrasions and lacerations are caused by a direct or shearing force, such as may be applied by a fist, finger, or other object. The patient can usually recall the time and type of injury. Symptoms of a conjunctival abrasion include a foreign body (FB) sensation and discomfort. Localized redness is noted at the area of injury. Subconjunctival hemorrhage is possible. Fluorescein staining reveals a discontinuity in the conjunctival surface.

Treatment consists of the use of a broad-spectrum ophthalmic ointment (eg, bacitracin or erythromycin) several times per day in the affected eye. Physicians can reassure their patients by telling them that conjunctival abrasions typically heal within several days.

Patients with conjunctival lacerations may present with the same history and symptoms as those with conjunctival abrasion. Inspection with anesthetic eyedrops reveals a full-thickness tear in the conjunctival epithelium, possibly with hemorrhage. A thorough examination, including dilation, is necessary to exclude an associated globe injury in that area or a retained FB. Small lacerations do not require closure and may be managed with antibiotic ointment and, if the patient is uncomfortable, application of a pressure patch for 1 or 2 days. Larger lacerations, usually more than 1 cm long, should be closed with an absorbable suture, such as 7-0 or 8-0 polyglactin (Vicryl, mfg by Johnson & Johnson, Summerville, NJ), on either a tapered or a spatulated needle. Tenon's fascia should be excluded from the wound to avoid an unsightly scar.[5]

Subconjunctival Hemorrhage and Emphysema

The subepithelial connective tissue underneath the bulbar conjunctiva, known as the lamina or substantia propria, contains numerous blood vessels derived from the anterior ciliary artery. These vessels are mobile and are vulnerable to rupture or tear in blunt trauma (Figure 8-1). Consequently, subconjunctival hemorrhage is common. Isolated subconjunctival hemorrhages require no specific therapy and will resorb within several weeks.

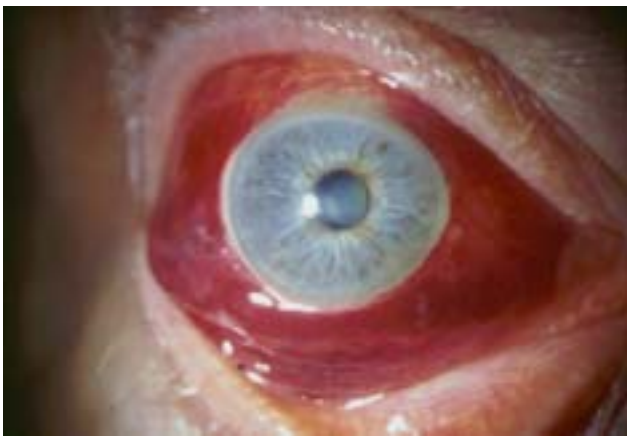


Fig. 8-1. In this patient with subconjunctival hemorrhage, diffuse hemorrhage can be observed underneath the conjunctiva following blunt trauma. There was no evidence of globe rupture.

Subconjunctival emphysema is loculated air in the lamina propria and is distinguished by a cystic appearance of the conjunctiva and by crepitus on palpation. These findings signify fracture of the periorbital sinuses with leakage of free air under the conjunctiva. Most commonly, the orbital fracture involves the lamina papyracea of the ethmoid sinus or the orbital floor overlying the maxillary sinus.[5]

The presence of subconjunctival hemorrhage, edema, or air may be associated with other eye injuries. Conjunctival edema, or chemosis, may accompany blunt trauma of the anterior segment. Specifically, localized hemorrhagic chemosis may overlie the site of a globe rupture. A complete ocular examination with imaging of the orbital bones is indicated in these cases.

CORNEA

The cornea is a specialized, transparent area of the outer coat of the eye. Its anterior location and aspheric conic shape make it susceptible to trauma. The cornea is most thin centrally, where it averages 0.52 mm in thickness, and increases to 0.65 mm peripherally.[6] Superficially, it consists of nonkeratinizing stratified squamous epithelium. The stroma, which constitutes 90% of the corneal thickness, consists of avascular collagen. Underlying the stroma is a thick basement membrane known as Descemet's membrane. Nonreplicating endothelial cells line Descemet's membrane and are responsible for maintaining corneal clarity by removing water from the corneal stroma.

Abrasion

Blunt or sharp trauma may disrupt the surface epithelium, producing a corneal abrasion. Because of the rich innervation by the corneal nerves, such injuries are very symptomatic and are accompanied by tearing, pain, photophobia, and protective closure of the eyelids. The visual acuity is usually decreased, from both the abrasion and profuse tearing. Topical anesthetic drops may be necessary for adequate anterior segment examination. Fluorescein, a dye applied either as a 2% solution or with a paper strip wetted with a sterile solution, will stain areas of epithelial discontinuity. Use of a cobalt filter with the slitlamp or of a hand-held Wood's ultraviolet (UV) lamp stimulates a green fluorescence by fluorescein and facilitates the diagnosis. Magnification of the cornea may be obtained with a slitlamp, a Wood's lamp, or a direct ophthalmoscope set at high-plus diopters (black).

Traumatic corneal abrasions have traditionally been treated with an antibiotic ointment, a mild cycloplegic agent, and a pressure patch. Patched patients require daily examinations to determine if the abrasion has healed and to verify that there is no corneal infection. Pressure patching has disadvantages, including occlusion of vision in the injured eye, discomfort, and an increased risk of infection, especially in contact lens-associated corneal abrasions.[7] Small- to moderate-sized traumatic abrasions have been shown[8] to heal faster with less discomfort, compared with pressure patching, if treated only with a broad-spectrum antibiotic (polymyxin [Polysporin Ophthalmic Ointment, mfg by Burroughs Wellcome Co, Research Triangle Park, NC] or erythromycin) and a mild cycloplegic (1% tropicamide). Topical nonsteroidal drops (0.5% ketorolac tromethamine) used four times daily for 3 days in conjunction with the no-pressure patch regimen described above can reduce the pain associated with traumatic corneal abrasion.[9] However, institution of topical nonsteroidal drops requires medical supervision and discontinuation after 3 days, as prolonged use may cause persistent epithelial defects.

A traumatic corneal abrasion may also be treated successfully with a bandage contact lens, topical antibiotic drops instilled four times daily, and topical nonsteroidal drops also instilled four times daily, which allow quicker visual rehabilitation.[10] For active duty soldiers, the increased risk of corneal infection and the expense associated with use of a bandage contact lens and nonsteroidal eyedrops must be weighed against the possible benefit of a quicker return to duty.

Uncomplicated, small corneal abrasions usually heal within a day or two. Superficial abrasions involving the epithelium heal without scarring, although corneal irregularity may impair vision for days to weeks. If a corneal wound breaches the underlying Bowman's layer and involves the stroma, scarring is usual. Topical anesthetics are *never* prescribed for pain management of corneal disorders, because their continued use may cause persistent epithelial defects and stromal scarring.

Corneal abrasions breach a protective layer of the eye and may become secondarily infected with bacterial, fungal, or parasitic agents. A white or gray infiltrate, best seen under magnification, is an indicator of corneal infection. Contact lenses are a predisposing factor for bacterial infection, especially Gram-negative bacteria such as *Pseudomonas* species. Inoculation of a corneal abrasion with vegetable matter is a risk factor for fungal infection. If microbiological laboratory support is available, diagnostic scraping with topical anesthesia and either a platinum spatula or a surgical blade is indicated. Appropriate studies include microscopic examination with Gram's or Giemsa stains, as well as microbial culture on solid media, such as blood agar, chocolate agar, and Sabouraud's agar (fungal). Until the results of the cultures are available, the initial treatment can be based on the results of the Gram's stain.

Fortified topical antibiotics should be used for infiltrates that (1) are associated with contact lens wear and (2) involve the visual axis or (3) are unresponsive to antibiotic monotherapy. Commonly used regimens include fortified cefazolin or vancomycin for coverage against Gram-positive bacteria and fortified gentamycin or tobramycin for coverage of Gram-negative organisms. Monotherapy with a single, broad-spectrum, fluoroquinolone antibiotic (eg, ciprofloxacin or ofloxacin) can be used for small or peripheral infiltrates or ulcers. However, fluoroquinolone antibiotics may not eradicate some Gram-positive bacteria, including *Staphylococcus* and *Streptococcus* species.

Patients with corneal infiltrates or ulcers should be followed closely. As previously noted, pressure patching of abrasions or ulcers associated with contact lens use is contraindicated. Topical steroids should not be used in the initial management of corneal infections.

Traumatic Keratopathy

Concussive force may directly damage endothelial cells with sector corneal edema. Damaged en-

dothelial cells may regain function, or undamaged adjacent cells may slide over and cover the injured area, leading to eventual clearing.[11] Sufficient concussive force may cause a single or multiple breaks in Descemet's membrane and severe corneal edema due to acute hydrops.[5] Endothelial cells will slide over the gap and eventually regenerate a new basement membrane. However, a tear in the original Descemet's membrane will remain visible, owing to scrolling or curling of the edges.

Corneal edema will often clear in weeks to months. The efficacy of topical steroids in the treatment of traumatic corneal edema remains unproven. Persistent sector edema, especially if localized inferiorly, can indicate an IOFB in the chamber angle. Gonioscopy is necessary to rule out this possibility.

Corneal Foreign Bodies

Foreign objects in the cornea may occur in a variety of industrial or military settings. The mechanism of injury is an important clue to possible occult ocular damage. High-velocity FBs (eg, particles generated by a blast, power tools, or metal striking metal) can lodge within any level of the cornea or even penetrate the ocular coat and locate anywhere within the eye. The presence of a conjunctival or corneal FB should prompt a thorough examination of the entire external area, including eversion of the upper and lower eyelids.

Like corneal abrasions, corneal FBs may be very painful, with blurred vision, photophobia, and tearing. Slitlamp examination will reveal single or multiple imbedded FBs at any level of the cornea. Superficial objects may be irrigated or removed with a moistened sterile swab under topical anesthesia. Deeper objects may be removed with a spud or a sterile hypodermic needle (22-gauge or smaller) mounted on a tuberculin syringe. This is best done at the slitlamp with firm support for the hand or wrist. A rust ring, common with iron FBs, may be removed with a hypodermic needle, dental burr, or spud. Residual rust may be left in the cornea, especially if the object was located away from the central cornea. Topical antibiotics should be applied until the epithelium has healed and the danger of infection has passed.

Corneal Lacerations

Any sufficient force can disrupt the ocular coat with a partial or complete tear through the cornea and sclera. Rupture is more likely to occur in areas of thin sclera, such as the insertions of the rectus muscles. It is also more likely to occur in areas of previous surgery or injury; rupture is especially problematic with avascular, clear corneal wounds (eg, cataract, transplantation, refractive incisions), owing to prolonged and incomplete wound healing.

If globe rupture is suspected, a protective metal shield should be placed over the patient's eye pending definitive diagnosis and management. Topical medications are to be avoided if rupture is suspected. It is important to determine if Descemet's membrane has been breached; careful slitlamp examination may reveal this. A Seidel test with 2% fluorescein is performed to detect leakage of the aqueous. Full-thickness lacerations may seal themselves because the corneal stroma swells on contact with aqueous fluid; in this case, the Seidel test may be negative initially. A provocative test with gentle pressure on the upper or lower lid may reveal fluid leakage.[12]

Partial-thickness wounds are treated with antibiotic prophylaxis, such as a broad-spectrum ophthalmic ointment or solution instilled three or four times daily until epithelial healing occurs. A bandage soft–contact lens may also be used to splint the wound. These wounds heal with scar formation, which may impair vision owing to the ensuing opacity or irregularity of the corneal surface. Partial-thickness avulsions or wounds with gaps may require suture closure, as will almost all full-thickness lacerations. Repair of corneal lacerations is addressed in Chapter 9, Sharp Trauma of the Anterior Segment.

Ultraviolet Radiation Keratitis

The cornea transmits most of the visible wavelengths of light between 400 and 800 nm. As the wavelength decreases into the invisible UV portion of the electromagnetic spectrum, more and more of the energy is absorbed by corneal tissue. The wavelengths between 200 and 300 nm are strongly absorbed by corneal cellular elements.[13] UV radiation keratitis occurs with exposure to sunlight, especially in situations with high reflectivity (snow or desert). High-altitude activities and exposure to arc welding or tanning lamps are also common causes of UV radiation keratitis. The corneal epithelium incurs the most damage from UV radiation. The damage typically becomes apparent within 8 to 12 hours after exposure.[13] Symptoms include an FB sensation, pain, and photophobia. Epithelial stippling and epithelial defects seen with fluorescein

staining occur with moderate to severe exposure. Resolution occurs in 1 to 2 days.

Protective lenses that absorb UV radiation prevent this injury. Military personnel should be alerted to the increased risk at high altitude and in snow or desert conditions. Treatment of UV injury is similar to that of corneal abrasion: antibiotic ointment, mild cycloplegic agents, and oral analgesics. Pressure patching should be considered on a case-by-case basis.

IRIS

The iris forms a diaphragm that separates the anterior and the posterior chambers of the eye. It consists of an anterior stroma with connective tissue, blood vessels, nerves, and melanocytes; the posterior iris contains a pigment epithelial layer. A sphincter muscle encircles the pupil, controlling light entry into the posterior chamber and maintaining the pupillary contour. A dilator muscle extends from the sphincter muscle to the base of the iris.[14] The iris is diaphanous and easily injured.

Traumatic Iritis

An inflammatory response to blunt trauma begins within hours. Typically, patients present with tearing, photophobia, and pain. Conjunctival injection and reduced vision may be noted. White blood cells are visible in the aqueous, with flare. Medical management of traumatic iritis consists of the application of topical steroid drops until resolution occurs, usually within several days. Prednisolone acetate, 1%, is commonly used three or four times daily. Cyclopentolate, 1%, administered three or four times daily, is sufficient for cycloplegia and offers the advantage of a shorter duration of action than atropine, homatropine, or scopolamine.

Traumatic Mydriasis

Concussion of the anterior segment may damage the iris sphincter muscle, resulting in anisocoria and iris deformity, including a sector defect or an oval pupil. A transient or permanently dilated, nonreactive pupil may be the result. Liberated pigment from the iris pigment epithelium may be deposited on the endothelium, trabecular meshwork, or lens. A circular pigment deposit on the anterior lens capsule due to trauma is known as a Vossius ring.

Iridodialysis

Iridodialysis, separation of the iris root from its scleral attachment (Figure 8-2), may result in bleeding with hyphema. Surgical repair is indicated for a displeasing appearance or polyopia and glare. A small-incision repair is possible with use of a 10-0 polypropylene (Prolene) suture on a double-armed CIF-4 needle (Ethicon, mfg by Johnson & Johnson, Summerville, NJ).[15] The technique is illustrated in Figure 8-3.

HYPHEMA

Characteristics

The presence of precipitated blood cells in the anterior chamber is called *hyphema* (Figures 8-4 and 8-5). Cell suspension in the aqueous is termed microscopic hyphema. Hyphemas may occur for a variety of reasons, including trauma; surgery; bleeding diathesis; neovascularization of the iris or chamber angle; or neoplasia, including juvenile xanthogranuloma.

A direct blow to the anterior globe can indent and shorten the anterior–posterior axis, with an attendant expansion equatorially. The iris–lens diaphragm may be retrodisplaced by the pressure wave. Stretching or shearing of tissues may lead to a tear of the ciliary body or the iris root. A tear in the anterior face of the ciliary body, or traumatic angle recession, is the most common cause of bleeding.[16] In this injury, the circular and oblique muscle fibers of the ciliary body are torn away from the longitudinal fibers, which remain attached to the scleral spur. A tear in this location may disrupt the major arterial circle of the iris or other arteries or veins in the area.[16] Traumatic cyclodialysis with separation of all ciliary body attachments, including the longitudinal fibers, from the scleral spur may also lead to hyphema. Iridodialysis and tears in the iris stroma or sphincter muscle uncommonly produce bleeding.

Small amounts of hemorrhage may circulate as suspended erythrocytes in the aqueous humor or collect as clumps on the iris, lens surface, or corneal endothelium. As the density of red blood cells in the anterior chamber increases, blood settles de-

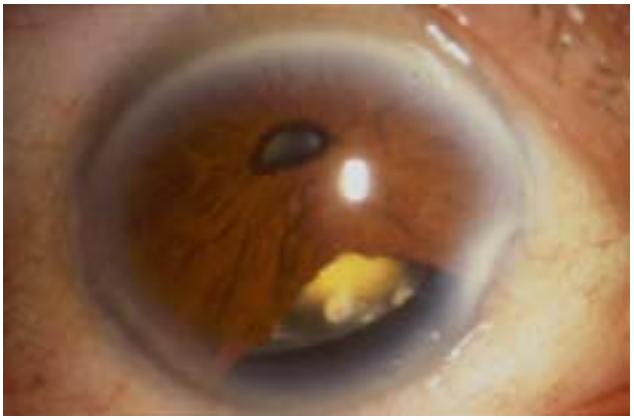


Fig. 8-2. A traumatic iridodialysis is present inferotemporally. A traumatic cataract is evident in the area of iris dehiscence. Photograph: Ms Ellen Foer, Ophthalmic Photographer, Walter Reed Army Medical Center, Washington, DC.

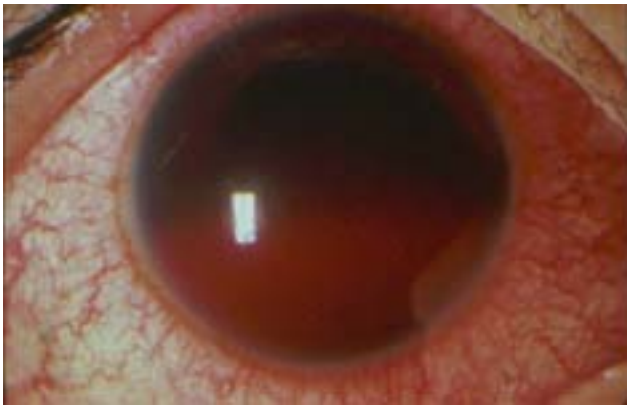


Fig. 8-4. In this hyphema, blood occupies approximately 40% of the anterior chamber following blunt trauma. Photograph: Ms Ellen Foer, Ophthalmic Photographer, Walter Reed Army Medical Center, Washington, DC.

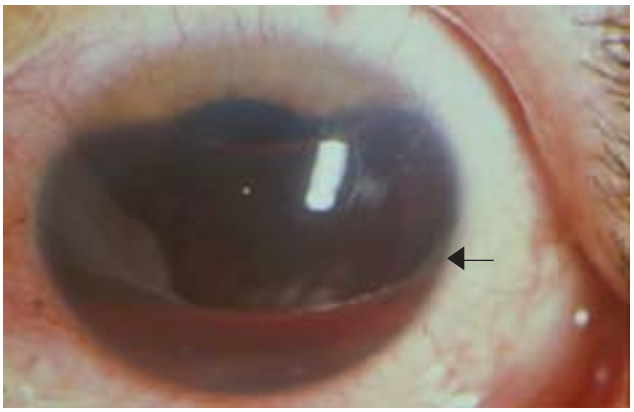
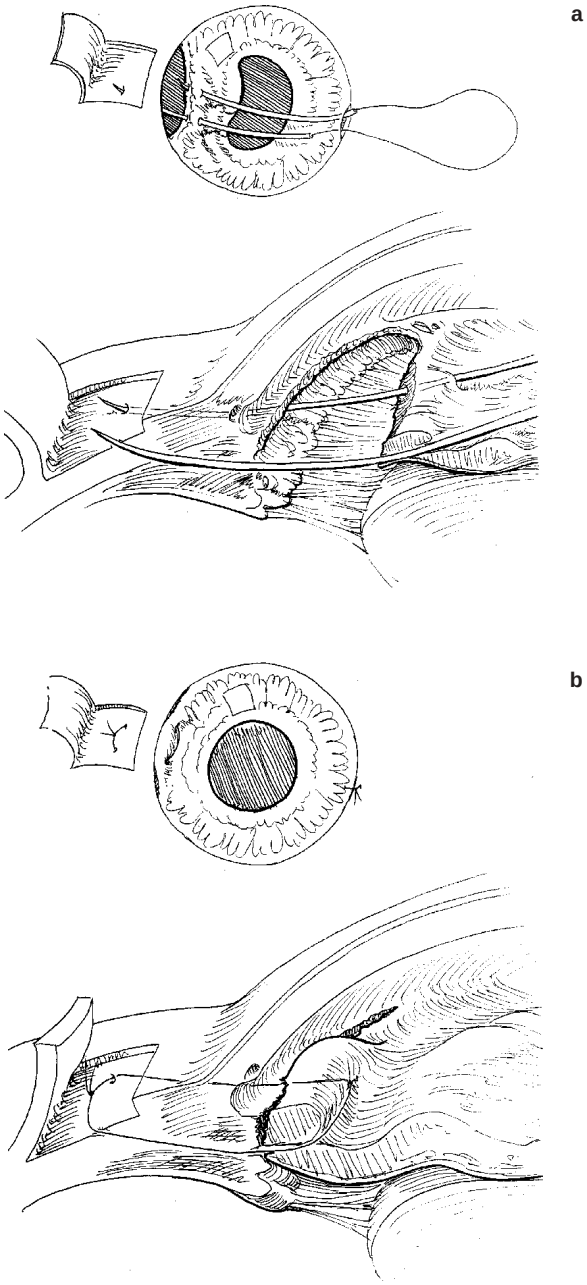


Fig. 8-5. A traumatic hyphema occupies 80% of the anterior chamber. Note the separation of clot and liquid blood (arrow). Photograph: Ms Ellen Foer, Ophthalmic Photographer, Walter Reed Army Medical Center, Washington, DC.

Fig. 8-3. Iridodialysis repair is depicted in the drawings. (**a**) A limbal peritomy and underlying scleral flap are created adjacent to the iridodialysis, and a limbal stab incision is made 180° away. Both needles of double-armed 10-0 polypropylene (CIF-4 needle, Ethicon, mfg by Johnson & Johnson, Summerville, NJ) cross the anterior chamber, engage the edge of the torn iris tissue, and exit through the scleral flap 0.5 mm behind the surgical limbus. (**b**) The suture material is tied with a triple knot and rotated. The scleral flap is closed with 10-0 nylon monofilament suture and the conjunctiva is replaced. Drawings prepared for this textbook by Gary Wind, MD, Uniformed Services University of the Health Sciences, Bethesda, Md.



pendently into the inferior chamber angle. Initially, this blood separates into a layered hyphema consisting of solid and plasmoid phases. A fibrin clot eventually forms. Hyphemas are generally graded by the percentage of the anterior chamber volume that they occupy.[17]

Complications

Visual loss may occur secondary to the trauma associated with hyphema. Visually significant complication from hyphema may occur days or weeks after the initial injury and require careful observation.

Rebleeding

Secondary hemorrhage (rebleeding) may complicate hyphema, occurring in 3.5% to 38% of patients, depending on the population studied.[17–21] Rebleeding has been associated with initial hyphema grade II or higher (occupying more than one third of the anterior chamber area),[19,20] although other studies[18,22,23] did not correlate rebleeding with initial hyphema size. Several studies[24–26] suggest that black patients of African descent are at higher risk of secondary hemorrhage. Fresh bleeding, which usually happens 2 to 5 days after the injury, may be caused by clot lysis.[16] Eyes that have rebled have generally been associated with poorer prognoses, although other studies[18,22] suggest that associated ocular injuries, especially retinal abnormalities, may account for this observed tendency.

Elevated Intraocular Pressure

Elevated intraocular pressure (IOP) can occur with hyphema. An acute rise in pressure may follow (1) concussive damage to the trabecular meshwork, with edema or collapse; or (2) obstruction of the outflow channels by formed blood elements and fibrin, or, later, with erythroclasis. Pupillary block by the clot can also occur.[27] A rise in IOP above 25 mm Hg occurs in 25% of patients with hyphema.[26]

Patients with hyphema who are young and whose optic nerves are healthy can withstand moderate rises in IOP without glaucomatous damage. However, patients with sickle cell disease (homozygous SS) or sickle cell trait (heterozygous SC or SA) are at risk of developing high IOP with hyphema. The rigid sickle cells are unable to deform themselves to exit through the trabecular meshwork and therefore clog the pathways, with a consequent pressure spike. Elevated IOP may cause anterior segment ischemia due to hypoperfusion, with acidosis and hypoxia, thereby exacerbating the sickling process.[17] Patients with sickle cell disease or trait are at higher risk for glaucomatous damage, possibly due to vascular occlusion by sickled erythrocytes with hypoperfusion of the optic nerve.[28]

Corneal Bloodstaining

Corneal bloodstaining occurs in 5%[16,26] of hyphema patients. It is associated with larger hyphemas, elevated IOP, prolonged clot duration, corneal endothelium dysfunction, and rebleeding.[16] Hemosiderin and other products of red blood cell degradation are present in the stromal keratocytes. Early signs include a yellowish discoloration in the posterior stroma and reduced definition of the posterior stromal fibrillar structure.[17] Corneal bloodstaining may clear over months to years, beginning peripherally.

Management

Management goals include detection and treatment of other injuries; resolution of the hyphema; and prevention of complications. The risk of deprivation amblyopia must be considered in the pediatric age group. Documentation of vision, IOP, extent of hyphema, and results of anterior and posterior segment examination at presentation is essential. Gonioscopy is not performed as part of the initial examination because manipulation may provoke further bleeding. A metal shield may be used to prevent accidental contact with the injured eye. Because sickling may occur in a patient with either the sickle cell disease or trait, any black patients of African descent with hyphema should undergo a blood test for sickle cell disease or trait and hemoglobin electrophoresis.[17]

Pharmacological Therapy

Inpatient observation has advantages in compliance and follow-up but has not been shown to improve the prognosis of hyphema, compared with outpatients maintained on moderate activity.[29] Discontinuing aspirin and nonsteroidal antiinflammatory oral agents is prudent because of their antiplatelet effects. Acetaminophen is preferred for pain management. Cycloplegic agents, such as a 1% atropine solution, are usually prescribed for twice-daily instillation for patient comfort, prevention of posterior synechiae, and facilitation of posterior pole examination. Cycloplegic agents do not appear

to have a beneficial effect on clot resolution or frequency of rebleeding.[18] Topical steroids can reduce associated inflammation and reduce the incidence of rebleeding, possibly through clot stabilization.[18] Prednisolone acetate, 1%, may be administered four times daily to the affected eye. Systemic prednisone has been shown[30] to have an equivalent rate of secondary hemorrhage, compared with oral antifibrinolytic agents, whereas other studies[23] have shown no effect. No study has compared the efficacy of topical versus systemic steroids. The side effects of systemic prednisone must be borne in mind.

Elevated IOP is initially treated medically. Topical timolol maleate, apraclonidine hydrochloride, and oral methazolamide or acetozolamide are used as indicated. Acetozolamide (Diamox, mfg by Lederle Laboratories, Wayne, NJ), which leads to systemic acidosis, hemoconcentration, and elevated ascorbic acid, is contraindicated in patients with sickle cell disease or trait because it causes increased sickling activity. Methazolamide is theoretically preferable in this situation because it produces less acidosis.[17] Miotics are not used because they may increase inflammation.

Systemic antifibrinolytic agents, such as ε-aminocaproic acid and tranexamic acid, have been reported to reduce the incidence of secondary hemorrhage in traumatic hyphema.[23,31–33] Two categories of patients are most likely to benefit: black patients of African descent and those who have been taking aspirin products.[34,35] These agents competitively inhibit the activation of plasminogen (profibrinolysin) to plasmin (fibrinolysin) and prevent early dissolution of clots, presumably allowing healing of injured blood vessels.[33] The usual dosage is 50 to 100 mg/kg orally every 4 hours, up to 30 g/d, for 5 days.[35] Side effects include nausea, vomiting, and postural hypotension.[36] Recently, the use of 30% ε-aminocaproic acid in a topical gel, instilled in the eye four times a day for 5 days, has been reported[37] to be effective without the systemic side effects of oral therapy.

Surgical Intervention

Surgical intervention to remove the clot and the free blood may be indicated in certain patients. Elevated IOP above 50 mm Hg for 5 days or above 35 mm Hg for 7 days, despite use of pressure-lowering agents, has been suggested[17] as an indication for surgery to avoid optic nerve damage. However, a lower threshold is required for patients with sickle cell trait or sickle cell disease or preexisting glaucomatous optic atrophy.[17] Surgery has been recommended[38] if IOP remains higher than 24 mm Hg for 24 hours in a patient with sickle cell disease.

Intervention is warranted if corneal bloodstaining occurs. In patients with total or near-total hyphema and IOP above 25 mm Hg that persists for 5 days, clot evacuation may prevent corneal bloodstaining. Prevention of peripheral anterior synechiae is another consideration: large clots persisting more than several days or total hyphemas lasting more than 5 days may be evacuated.[17] Clot removal in hyphema is not innocuous, however. Risks include damage to the cornea, iris, or lens; inadvertent extraction of the iris; prolapse of intraocular contents; renewed bleeding; formation of synechiae; and postoperative glaucoma.[16]

The simplest and safest technique for hyphema evacuation is an anterior chamber washout through one or two clear corneal limbal paracentesis wounds (Figure 8-6). This procedure allows removal of free blood and fluid and lowers IOP. If bleeding or in-

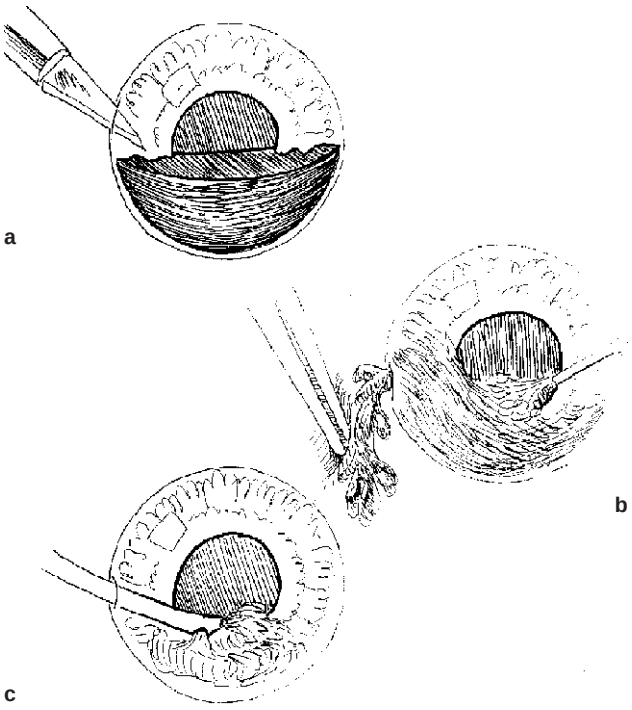


Fig. 8-6. Methods of hyphema evacuation. (**a**) Limbal stab incisions approximately 2 mm wide are made at 10 o'clock and 2 o'clock with a 15° surgical knife. (**b**) Hyphema washout is achieved by irrigation of balanced salt solution into the anterior chamber, while a closed forceps depresses the opposite paracentisis site to allow egress of the fluid and the clot. (**c**) If the washout is insufficient to remove the hyphema, a manual or automated irrigating/aspiration unit is introduced to remove the blood and the clot. Drawings prepared for this textbook by Gary Wind, MD, Uniformed Services University of the Health Sciences, Bethesda, Md.

creased IOP recur, the paracentesis sites may be reopened at the slitlamp with sterile jeweler's or 0.12-mm forceps to "burp" out additional fluid. This procedure leaves the conjunctiva intact in case filtering surgery is required.[17] Large clots may be removed through a limbal incision combined with anterior chamber washout and a peripheral iridectomy, or with trabeculectomy.[27,39]

Disadvantages of surgical intervention include inadvertent delivery of the iris, lens, or vitreous. Automated irrigation/aspiration or automated vitrectomy instruments may be used initially or after conversion from an unsuccessful anterior chamber washout. Tamponade of active bleeding may be obtained by raising the height of the irrigating solution bottle.[17]

SUMMARY

Most blunt trauma and UV radiation injuries are preventable with appropriate protective eyewear, which is available to and should be worn continuously by all military personnel in a theater of action. Blunt trauma to the eye may cause incapacitation through visual loss and pain, adversely affecting the military mission by the loss of a combatant, the use of medical resources, and, in more severe cases, transport of the patient to a rear-echelon facility for surgical management.

In all cases of ocular trauma, a thorough examination is essential to detect occult injury, establish a prognosis, and to formulate a management plan. Most nonperforating injuries to the anterior segment may be treated conservatively, with the goals of therapy to restore vision and ameliorate pain and to prevent secondary complications.

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