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Eye injuries present a significant challenge to emergency personnel. Patient stress and coexisting periorbital findings can complicate any evaluation, and many of the signs of serious injury may be quite subtle. Because the majority of eye injuries present between 10 p.m. and 4 a.m.,[1] when ophthalmology consultation is not available immediately in most hospitals, a tremendous burden is placed on the emergency health care provider to identify and manage potential vision-threatening disorders. The following is a review of ocular trauma with a focus on clinical findings, their implications, and management.

— The Editor

Initial Evaluation of the Traumatized Eye

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Epidemiology and Introduction

More than 2.5 million eye injuries occur in the United States annually.[2] From 1988- 2000, the United States Eye Injury Registry (USEIR) reported more than 10,000 major eye injuries,[3] an annual hospitalized incidence of 13.2 per 100,000.[4,5] Eighty percent of those injured were male, with a mean age of 29 years. Traditionally, the workplace presented the most common site of ocular injuries, but the USEIR reports 40% of these injuries occurring in the home, 13% in the workplace, and another 13% during recreation. Of these injuries, 31% were caused by a blunt object (e.g., rock, fist, baseball, or lumber), 18% by a sharp object, and 12 % were attributed to projectiles such as firearms and pellet (BB) guns.[6-8] Alcohol consumption was involved in 10% of cases, and 57% of all eye injuries occurred duirng warm weather months (spring and summer).[9] In the United States, 1.7% of all eye injuries will progress to permanent visual loss,[9] resulting in 60,000 new cases of monocular blindness related to trauma annually.[10]

Among the pediatric population (birth–16 years of age), eye injuries occur at an annual rate of 15.2 per 100,000 with males outnumbering females four to one. Males, ages 11-15, have the highest incidence of eye injuries at 23.7 per 100,000 annually, and 40% of these injuries are attributed to sports.[11] In victims of child abuse, an ocular injury will be the presenting injury in up to 40% of all cases[12] and in 95% of cases of shaken baby syndrome.[13]

Physical Exam

Dannenberg et al reported that 33% of all registered penetrating eye injuries were in the setting of multisystem trauma.[6] Thus, Advanced Trauma Life Support (ATLS) guidelines and a thorough general examination must precede any ocular evaluation. Secondly, any eye with a potential exposure to hazardous materials (e.g., acids, alkali, particulate matter, or heat) should be irri-

gated prior to evaluation. Whenever possible, a thorough history should be obtained detailing the context, mechanism, time of injury, and use of eye protection. Past medical history should include previous ophthalmologic conditions, surgeries and trauma, current ocular medications, and pre-injury vision status (e.g., glasses/ contact lenses wearer, and visual acuity).

Figure 1 demonstrates a simple guide for the trauma examination of the eye. The first step is an assessment of visual acuity, which can be accomplished with a Snellen Eye Chart or near card. For pre-literate children, Allen cards or HOTV vision test letters may be used. For infants, fixation and smooth pursuit can be assessed using a hand light or colorful target. In many cases, the visual acuity may be too poor to assess by standard charting, and thus a gross assessment of visual acuity should be obtained using finger counting, motion perception, and light perception.

Most patients, regardless of mechanism, will complain of some degree of eye pain, but care must be taken to differentiate between globular and periorbital sources of pain. On inspection, examine the orbit for any step-off deformities or crepitus that may reveal an orbital wall fracture. Although there may be evidence of significant periorbital ecchymosis and edema, the lids still should be examined closely for lacerations and inverted when possible to remove retained foreign bodies. In the setting of prominent periorbital edema, a speculum or retractor may be employed to visualize the globe; however, if there is any evidence of a globe rupture (e.g., prolapse of intraocular contents, hemorrhagic chemosis, or enophthalmos), then this step should be foregone in favor of imaging studies and immediate ophthalmologic consultation. When possible, examine the anterior chamber for hyphema or pupil irregularities, findings that require further examination by slit lamp. Globe pain or foreign body sensation imply, at the minimum, corneal irritation and can be evaluated using fluorescein dye with a Wood's lamp to reveal corneal abrasions, ulcerations, or lacerations. All corneal exams that require fluorescein should be followed by a slit-lamp examination for occult foreign bodies.

Ocular motility should be assessed in both the vertical and horizontal planes. Classically, orbital wall fractures will present with deficits of ocular motility, but a proptotic eye with any motion deficit is the hallmark of a retrobulbar hemorrhage. For this reason, any defects in ocular motion should be evaluated further with a computerized tomography (CT) scan.

Finally, the posterior segment should be assessed by pupil reactivity to direct light and accommodation. Even in cases of prominent trauma to the anterior segment, the retina and optic nerve can be assessed by direct light to the affected eye and monitoring accommodation in the unaffected eye, known as the swinging flashlight test. A pupil that fails to constrict with direct light stimulation, but responds to stimulation of the other eye implies a defect in the posterior segment. Children may have a corresponding lack of a red reflex (Bruckner's test) in the affected eye. Both should be investigated further with an ophthalmoscope, when possible, or imaging studies such as ultrasound or CT scan.

In theory, any patient with a painless eye with intact visual acuity, a normally reactive pupil, and intact ocular motility in the absence of any anatomic deformities following a traumatic event may be discharged without further study and appropriate follow up. Nevertheless, an examining physician should have a low threshold for advanced study in the setting of high energy trauma or any projectiles where these findings may be masked by either trauma elsewhere or the delicate size of the particulate matter. Any new visual deficits or any impairment in mental status that complicates the examination should prompt a full evaluation.[10,14-17]

Classification of Eye Trauma

In 1993, a group of ophthalmologists from Birmingham, Ala., developed a standardized classification of eye trauma that would ease communication between peers and provide prognostic significance. In 1995, the International Society of Ocular Trauma, The USEIR, and the American Academy of Ophthalmology endorsed this new system. Ocular Trauma Classification (Tables 1 and 2) involves identifying the type of injury (open vs closed globe), the grade of the injury (visual acuity), pupil response, and

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This CME/CE activity is intended for emergency, family, osteopathic, trauma, surgical, and general practice physicians and nurses who have contact with trauma patients.

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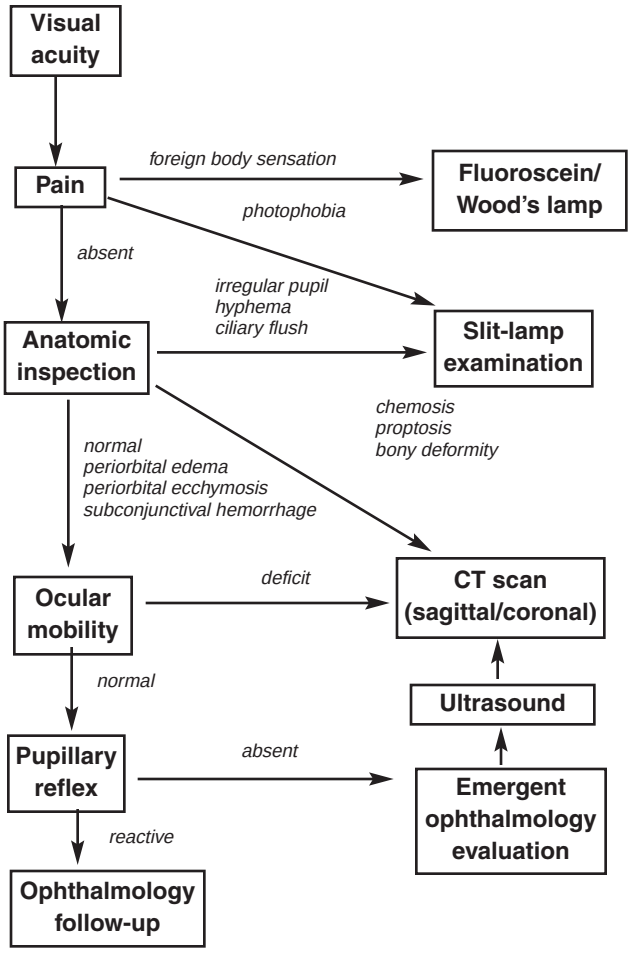
Figure 1. Trauma Examination of the Eye



the zone of the injury. Zone defines the anterior-posterior relationship of the injury on the globe.

An *open-globe injury* contains a full thickness wound of the eyewall (sclera or cornea). Open-globe injuries are then divided into lacerations (sharp mechanism) and ruptures (blunt mechanism). Lacerations are further divided into penetrating injuries (i.e., one entrance wound only), perforating injuries (i.e., two full thickness entrance and exit wounds), and intraocular foreign bodies. A *closed-globe injury* has no full thickness wound of the eyewall, and these injuries are divided into contusion (blunt mechanism), lamellar laceration (sharp mechanism), and superficial foreign body categories.

The Ocular Trauma Classification System provides a universal means of communicating the type and severity of an eye injury to an ophthalmology consultant or between emergency personal. In addition to quantifying an injury, this system, within the ophthalmology community, provides a foundation for management requirements as well as prognostic value for regaining vision.[18-21]

Superficial Injuries of the Eye

Superficial injuries of the eye, such as periorbital edema and ecchymosis, are very common sequelae of eye trauma, and often are quite dramatic on initial presentation. Despite the stress they may cause patients, these injuries involving the eyelids are quite benign in the absence of any globe injury and can be managed conservatively. For this reason, evaluation of lid injuries, regardless of mechanism, should be delayed for the evaluation and management of intraocular injuries.

Eyelid lacerations are divided into three types: vertical, horizontal, and canilicular (involving the medial lacrimal duct). Essentially, all eyelid laceration repairs may be delayed safely for 24-48 hours.[22-23] Thus, it is recommended that these injuries be irrigated to remove any foreign bodies, covered with a semi-moist pressure dressing, and ophthalmology evaluation arranged within one day. This is acceptable management for all pediatric and uncooperative patients as well.

Partial thickness injuries may be closed by emergency personnel, especially if ophthalmology evaluation will be delayed more than two days. However, all canalicular lacerations should be left to a specialist; the most superficial lacerations require proper exploration prior to closure.

Horizontal lacerations are more serious than vertical lacerations due to potentially significant impairment of the levator muscle. Evidence of ptosis or prolapsing fat indicates violation of the orbital septum, and, due to the close proximity of the levator aponeurosis, is highly suggestive of a levator injury. These repairs also should be delayed for a specialist.

Partial or full thickness vertical lacerations can be repaired by first approximating the tarsoconjunctival edge with simple, interrupted polyglycolic acid suture material with a D-1 needle.[22,24] The muscle layer can be sutured with 6-0 catgut, and the external eyelid can be closed with 6-0 silk. Once sutured, a pressure patch should be placed to reduce eye swelling and horizontal tension on the eyelid.[17] Although suture removal is recommended in 7-10 days, an ophthalmology follow-up evaluation should be scheduled in 2-3 days to ensure adequate wound healing.

Injury by Presentation: Pain

Pain is the hallmark of a corneal injury. Corneal abrasions are the single most common ocular condition evaluated in the ED,[25] as well as the most common eye injury related to airbag deployment.[26] For injuries confined to the superficial corneal epithelium, patients will present with a painful eye with conjunctival injection, ciliary flush, and tearing. Prior to examination, the eye should be anesthetized with tetracaine or proparacaine. Findings on fluorescein dye examination of the eye under a cobalt blue light or Wood's lamp will vary depending on the mechanism of the injury. Large abrasions with sharp borders are most often due to blunt trauma. Diffuse, punctuate lesions represent mild burn injuries from chemical exposure or ultraviolet light. Foreign bodies under the eyelid will produce multiple linear defects. Treatment includes removal of any foreign bodies, broad-spectrum antibiotics for the eye (e.g., ciprofloxacin/polymyxin B-trimethoprim), oral analgesia such as non-steriodal anti-inflammatory drugs (NSAIDS) or acetaminophen, tetanus prophylaxis as needed, and ophthalmology follow-up in 24-48 hours.[20]

Burns. Corneal burns account for up to 18% of all ocular trauma, with 68% of these injuries occurring in an occupational setting.[27] Burning agents can be divided into three broad cate-

gories (alkaline chemical agents, acidic chemical agents, and thermal agents), and the severity of the injury is related to the composition of the offending agent, volume, pH, and duration of exposure. Alkaline agents, accounting for 58% of all burn injuries, damage the cornea by saponifying the fatty acid components of the corneal cell membranes. This induces a liquefactive necrosis and allows deeper penetration into the globe. Ammonia is the most common alkaline agent causing eye injuries, but others include lye, potassium hydroxide, and lime.

Acids damage the cornea by inducing protein precipitation and denaturation into the epithelium and superficial stroma. The resultant coagulative necrosis tends to protect deeper intraocular structures. The most common acid-burning agents include sulfuric, sulfurous, hydrofluoric, and hydrochloric acids.[22]

Thermal injuries are usually the result of splash injuries from hot liquids or molten metal. A temperature greater than 100° C is required to injure the corneal epithelium. Cigarette burns to the cornea are a common injury in children ages 2-4 years. Usually the result of a child running into a cigarette at eye level; this burn pattern is rarely a manifestation of abuse.[10]

Eye burns are classified into four grades. A Grade I injury presents with hyperemia, conjuntival ecchymosis, and a cornea with a ground-glass appearance. A cigarette burn will produce this pattern of injury. In addition, a Grade II injury will have conjunctival chemosis and minor eschar formation. Grade III burns are deeper injuries characterized by conjunctival ischemia, thrombosed blood vessels, and reduced corneal clarity with preservation of the remaining anterior segment. Complete opacification of the cornea and lens, in addition to diffuse conjunctival ischemia and a grey iris in mydriasis, typify a Grade IV injury. Necrosis of the conjunctiva is accompanied by a dramatic inflammatory response that will induce further corneal ulceration within 4-6 weeks.

The mainstay of burn therapy is irrigation, preferably with a solution that is isotonic to the corneal stroma. Ideal solutions include lactated Ringers, balanced saline solution, or diphoterine; however, any irrigation (water, or normal saline) is far better than none at all. Upon presentation, patients should be anesthetized topically and then irrigated promptly for 15 minutes or 1L of fluid using a Morgan lens. Irrigation should not stop until normalization of the corneal pH (7.4). Lime and cement products will react with water to produce calcium hydroxide (pH 12.4), exacerbating their effect. These patients will require aggressive cleaning of the cul-de-sac with a cotton-tipped applicator dipped in 1% ethylenedinitrilotetraacetic acid (EDTA).[22]

Grade I and II burns may be discharged home on topical antibiotics and oral analgesics with ophthalmology follow-up within 24 hours. Grade III burns require admission for operative microscopy to determine the depth of tissue destruction. Grade IV lesions require steroids (oral or IV), as well as topical antibiotics prior to admission. Grade III lesions may be admitted to a local hospital with ophthalmology services; however, a Grade IV burn will most likely require a tertiary center with expertise in plastic reconstruction of the eye.[22]

Table 1. Open-Globe Injury Classification

TYPE

- Rupture
- Penetrating
- Intraocular foreign body
- Perforating
- Mixed

GRADE (VISUAL ACUITY)

- > 20/40
- 20/50 to 20/100
- 19/100 to 5/200
- 4/200 to light perception (LP)
- No light perception (NLP)

PUPIL

- *Negative*: Relative afferent pupillary defect absent in affected eye
- *Positive*: Relative afferent pupillary defect present in affected eye

ZONE

- I: Isolated to cornea (including corneoscleral limbus)
- II: Corneoscleral limbus to 5 mm posterior into the sclera
- III: Posterior to the anterior 5 mm of sclera

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Ultraviolet Keratitis. Ultraviolet keratitis is another form of exposure injury to the cornea classically known as snow blindness. Caused by the cumulative effect of ultraviolet light from electric arcs (welding) or tanning lamps, this disorder initially will present as a bilateral foreign body sensation and photophobia. Symptoms progress to severe bilateral eye pain with conjunctival erythema and tearing. Fluorescein staining reveals diffuse punctuate lesions on the cornea. Symptoms usually will resolve in 24-36 hours once removed from the inciting agent. Management includes topical antibiotics and narcotic analgesia.[20]

Lacerations. A corneal laceration is defined as a traumatic disruption of the cornea involving all three layers (epithelium, stroma, and endothelium) due to a penetrating mechanism; 75% of these injuries in children were caused by a sharp object.[28] Like other corneal injuries, pain is the most common presenting complaint. An examination of the eye may reveal a shallow anterior chamber or teardrop-shaped pupil directed toward the laceration. Prolapse of intraocular contents is a confirmatory finding; however, in many cases, the eye will appear normal due to the self-sealing properties of the elastic corneal stroma. Fluorescein evaluation of the cornea under blue light will reveal a bright green stream (Seidel test) caused by egress of fluid from the anterior chamber. A slit-lamp examination on oblique illumination may reveal the length and depth of a laceration. Nevertheless, a normal fluorescein study does not completely eliminate the possibil-

Table 2. Closed-Globe Injury Classification

TYPE

- Contusion
- Lamellar laceration
- Superficial foreign body
- Mixed

GRADE (VISUAL ACUITY)

- > 20/40
- 20/50 to 20/100
- 19/100 to 5/200
- 4/200 to light perception (LP)
- No light perception (NLP)

PUPIL

- *Negative:* Relative afferent pupillary defect absent in affected eye
- *Positive:* Relative afferent pupillary defect present in affected eye

ZONE

- I: External (limited to bulbar conjunctiva, sclera, and cornea)
- II: Anterior segment
- III: Posterior segment

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ity of a corneal laceration. Partial-thickness lacerations can be managed like corneal abrasions.[20,29]

Foreign Bodies. Forty-one percent of all open-globe injuries involve an intraocular foreign body.[30] The majority of these projectiles (80%) are generated in the setting of hammering either a chisel, nail, or stone.[30,31] Other causes of ocular foreign bodies include motor vehicle collisions, explosions, projectile weapons, and activities involving power tools such as drilling. Magnetic metals (iron, lead, and copper) account for the majority of intraocular foreign bodies (IOFB); however, nonmetallic items such as glass, wood, and especially organic materials in pastoral settings, may be a nidus for a disastrous Bacillus infection. More than 80% of IOFBs will penetrate the cornea and settle within the vitreous.[32] Other common sites for foreign bodies include the underside of the upper lid, the infracorneal recess within the lid fissure, and the inferior angle of the anterior chamber.[33]

Missed foreign bodies constitute 56% of all eye trauma-related legal claims, and therefore, IOFBs should be excluded in any patient with a suspicious mechanism. One-fifth of all patients with ocular foreign bodies will have no pain and intact visual acuity.[30] Many patients, especially children, will be unaware of the exposure, resulting in a significant delay to presentation. Like other corneal injuries, pain or foreign body sensation will be the most common complaint, but visual deficits will vary depending on the size of the projectile, extent of the injury, and delay in presentation. Fluorescein examination of the cornea should be followed by a slit lamp examination of the cornea, anterior chamber, and the area under the lids. Any superficial foreign body may be removed with either a cotton-tipped applicator (lid) or a 27-gauge needle.[34]

Assault-related Injuries. Dannenberg et al reported that one-third of all occupational eye injuries[11] and 56% of all assault-related injuries involve the sclera.[5] Scleral rupture is a traumatic disruption of the sclera commonly in the supranasal quadrant at the insertions of the rectus muscles on the globe. Most often this injury is the result of a blunt mechanism and has a prevalence of 3.5% in blunt trauma.[35] In the absence of obvious prolapse of intraocular contents, evidence of a scleral rupture may be quite subtle. Low intraocular pressures (< 6 mmHg), reduced visual acuity on initial presentation, an afferent pupillary defect, and a shallow anterior chamber are clinical findings highly specific to scleral ruptures, and thus, warrant further investigation. In addition, one-quarter of hyphemas and 22% of bloody chemosis cases are associated with a full-thickness scleral injury. [12,30,36-38]

Because full thickness injuries of the eyewall (e.g., corneal lacerations, scleral rupture, and intraocular foreign bodies) are open-globe injuries, any suspicion of such requires prompt ophthalmology evaluation. CT imaging (axial and coronal views) may be diagnostic (sensitivity, 93%; specificity, 75%; PPV 95%)[39,40] of scleral injuries, but is the modality of choice to detect intraocular foreign bodies (sensitivity 100% for objects more than 0.06 mm).[3,41] Magnetic resonance imaging (MRI) is contraindicated for IOFB due to migration of metallic fragments.

The examiner should avoid placing any pressure on a suspected globe rupture to limit further prolapse of intraocular content; therefore, tonometry should be deferred despite the value of confirming low intraocular pressures. An eye shield should be placed promptly pending further evaluation. Tetanus vaccinations should be updated, and prophylactic antibiotics should be started in the ED. Up to 16% of open-globe injuries may progress to bacterial endophthalmitis caused by Staphylococcus, Streptococcus, or Bacillus species, which are particularly aggressive. Current antibiotic recommendations include ceftazidime (1 g every 8 hours) and vancomycin (1 g every 12 hours). Ciprofloxacin (400 mg every 12 hours) and vancomycin may be used in cases of penicillin allergy.[42]

Metallic intraocular foreign bodies may induce similar localized inflammatory reactions known as metallosis (chalcosis for copper IOFB; siderosis for iron IOFB), which can progress to hypopyon, retinal detachment, and irreversible visual deficits within hours. Therefore, prompt surgical evaluation is the definitive management for open-globe injuries, ideally performed by an ophthalmology specialist familiar with anatomical reconstruction to optimize visual outcomes.

Injury by Presentation: Diplopia

The two major components of the external eye are the extraocular muscles and the bones of the orbit. Post-traumatic deficits in ocular motility are suggestive of extraocular injuries leading to the common complaint of binocular diplopia.

The conical orbit comprises six bones that integrate to create a floor (maxilla), roof (frontal bone), lateral wall (sphenoid and zygoma), and medial wall (maxilla, lacrimal bone, ethmoid, and sphenoid). Orbital wall fractures are largely the result of blunt trauma from an object greater in diameter than the orbital rim such as a fist, ball, or dashboard. The energy from the trauma is dispersed across the orbital rim and elastic globe, which will transmit this force into the orbit. The evolutionary shape of the orbit minimizes the effect of these sudden elevations in intraocular pressure by collapsing, like a safety valve, at its weakest points – the floor and ethmoid aspect of medial wall (lamina papyracea). Sports-related injuries (e.g., baseball, softball, and soccer) are the most common causes of orbital wall fractures in children.[43] In adults, these injuries often are attributed to motor vehicle collisions, assault with a blunt object, and falls.[44]

Blow-out Fractures. Impairment of upward gaze due to entrapment of the inferior rectus muscle is the classic presentation of a blow-out fracture, a fracture of the floor or medial wall of the orbit. Patients commonly will complain of binocular diplopia with periorbital edema, ecchymosis, and an acutely proptotic eye. This can be associated with periorbital crepitus due to subcutaneous emphysema,[45] as well as hypoesthesias of the upper lip and maxillary teeth secondary to infraorbital nerve injury. Enophthalmos, usually a delayed finding, may develop proportional to the size of the maxillary fracture.

Blow-in Fractures. A blow-in fracture is a fracture of the orbital roof (frontal bone) caused by high-velocity blunt trauma directed at the superior orbital rim. A rare fracture (5% of all facial fractures), orbital roof fractures have a strong association with other facial fractures (73% of cases) and intracranial injuries (44% of cases).[46] Because this fracture reduces orbital space, exophthalmos will persist after the periorbital edema has regressed. As in a blow-out fracture, patients will complain of diplopia, but this may be associated with supraorbital hypoesthesia and ptosis secondary to entrapment of the levator palpabrae. In rare cases, intracranial contents may herniate inferiorly, and cerebrospinal fluid (CSF) may leak from the eye.

A CT scan with fine-cut axial and coronal images of the face and orbit is the modality of choice for the diagnosis of orbital bone fractures. Due to the bony immaturity, children will have a higher rate of false negatives despite obvious clinical signs, and management should proceed as if a fracture is present.[47] Surgery is the definitive treatment, and indications include fractures greater than 50% of the orbital floor, extraocular muscle entrapment, and enophthalmos greater than 2 mm. In most cases, intervention will be delayed 7-10 days to allow reduction of post-traumatic edema; thus, emergent management is conservative. Ice packs with head elevation are primary, and the patient should be instructed to avoid nose blowing. Nasal decongestants and a 10-day course of antibiotic prophylaxis against sinusitis may be initiated. Because practice varies, ophthalmology should be notified, but does not require urgent consultation unless the patient demonstrates evidence of an orbital roof fracture, elevated orbital pressures, or stimulation of the oculocardiac reflex (bradycardia/hypotension).[17,22,33,40,43-45,48-52]

Extraocular Muscular Avulsion. Another cause of post-traumatic diplopia is extraocular muscle avulsion. The superior oblique most often is affected due to the close proximity of the trochlea to the superior orbital rim. Penetrating mechanisms are almost always the cause including projectiles (e.g., pellets [BBs], bullets), bone fragments from orbital wall injuries, and dog bites in children. Presentation will vary depending upon the degree of transection. Patients will complain of diplopia, and examination will reveal a motion deficit or, in cases of complete transection, deviation of the globe. CT imaging may confirm a muscle injury, and isolated injuries may be managed conservatively with ophthalmology follow-up within 24-48 hours.[33,44,52]

Retrobulbar Hemorrhage. Because the orbit is a rigid, enclosed space, it is susceptible to a compartment syndrome. Retrobulbar hemorrhage is a very rare injury with serious consequences. Unfortunately studies in the United Kingdom have suggested that an overwhelming majority (73%) of emergency personnel are not familiar with the presentation and management of this disorder.[54] Post-traumatic retrobulbar hemorrhage will occur in the setting of either penetrating or blunt mechanism. Like compartment syndrome elsewhere, pain is the hallmark of this injury with rigid, rock-hard proptosis, which may be accompanied by painful ocular motility progressing to diplopia. Increasing intraocular pressures will reduce perfusion pressures to the optic nerve causing a progressive visual deficit and an afferent pupillary defect that is irreversible within 1-2 hours of onset.[55]

Management begins with recognition of a painful proptotic eye. CT imaging will confirm the presence of an intraorbital hematoma, but if the patient has any visual or pupillary deficits on presentation, immediate intervention is necessary. Concerning presentations with negative CT scans can be assessed with tonometry to confirm normal ocular pressures prior to discharge.

An ophthalmologist should be notified. The patient's head should be elevated, and he should be instructed to avoid any valsalva maneuvers (e.g., coughing, sneezing, and straining). Mannitol, acetazolamide, and steroids should be given to reduce intraocular pressures and protect the optic nerve. If symptoms continue to progress, a lateral canthotomy should be performed with lateral cantholysis of the inferior crus. If intraocular pressures are normalized within a timely manner, symptoms typically resolve within hours, including a return to baseline vision.[44-46,56-58]

Injury by Presentation: Photophobia

The uvea is composed of the iris, ciliary body, and choroids. Because its primary role is pupillary regulation, injuries usually will present as pain with pupillary constriction, and patients will complain of photophobia.

Traumatic Iritis. Traumatic iritis is acute inflammation of the anterior uvea, secondary to blunt or penetrating trauma. The chief complaint will be photophobia, but patients also may complain of headache, blurred vision, or light sensitivity due to traumatic mydriasis. They also will have pain in the affected eye when a light is shone in the unaffected eye, termed consensual photophobia. Slit-lamp examination will reveal ciliary flush with characteristic cell and flare. Treatment consists of symptomatic

relief with a cycloplegic agent such as 5% homatropine and ophthalmology follow-up in 2-3 days.[16,33]

Sympathetic Ophthalmia. Sympathetic ophthalmia is a bilateral, autoimmune, granulomatous uveitis incited by penetrating injuries to the eye. The incidence ranges from 1% in open-globe injuries[59] to 10 cases per 100,000 penetrating eye wounds.[61] Onset can range from five days to 66 years, but the average is 4-8 weeks following trauma.[61] Patients will complain of photophobia with tearing and visual deficits, but the key element in the history is the bilateral presentation (consensual photophobia) despite sustaining an injury to only one eye. If left untreated, the inflammatory conditions related to sympathetic ophthalmia can progress rapidly to profound visual impairment from secondary cataract, glaucoma, or retinal detachment. Slit-lamp examination will reveal cell and flare, and the standard of care in the ED is steroid therapy with prompt ophthalmology follow-up.[62]

Blurred Vision with Intact Afferent Pupillary Reflex

A normal pupillary response is indicative of an intact posterior segment, mainly the retina and optic nerve. Visual deficits with normal accommodation reflect injury anterior to the retina. These injuries obscure the visual axis to the posterior eye; therefore, patients will complain of aberrant vision without a complete deficit.

Traumatic Hyphema. Traumatic hyphema is the presence of blood in the anterior chamber secondary to trauma. The annual incidence is 17 to 20 per 100,000 with a peak age range of 10-20.[63,64] Two-thirds of all hyphemas are due to blunt mechanisms with 44% percent due to assaults,[5,62] 44% sports-related,[59,65] and the remainder associated with motor vehicle collisions and the workplace.[1] Hyphemas are also the most common eye injury associated with paintball trauma.[66] In the pediatric population, 65% of these injuries are sports-related.

A hyphema results from shearing forces on the uvea, leading to disruption of the iris, ciliary body, or choroid with subsequent hemorrhage into the anterior chamber. It is the hallmark of a severe ocular injury, and 25% are associated with a scleral rupture.[35] Most patients will present with eye pain and visual deficits, but the degree of visual impairment will be proportional to the percentage of the anterior chamber occupied by blood. For that reason, hyphemas are graded:

- A Grade I lesion occupies less than one-third of the anterior chamber;
- a Grade II lesion occupies between one-third and one-half of the anterior chamber;
- a Grade III lesion occupies more than half of the anterior chamber; and
- a Grade IV lesion is a total hyphema (black ball or eight ball).[63]

The severity of hyphemas is related to the corresponding complications. One-third of hyphemas are associated with elevated intraocular pressures, which may progress to acute angle-closure glaucoma secondary to obstruction of the trebecular meshwork by blood products or direct compression of outflow channels. Sickle cell disease or trait may exacerbate this effect, progressing to dramatic increases in intraocular pressure even in low volume hyphemas. Persistently elevated intraocular pressures can lead to optic nerve atrophy, and irreversible visual deficits. Finally, large hyphemas may cause corneal blood staining, a yellow discoloration of the cornea contributing to permanent visual deficits.

The first step in examining a hyphema is to rule out an open-globe injury. In the absence of any signs or symptoms of a full-thickness eyewall injury, a thorough history should be obtained, focusing on a medical history of blood disorders such as sickle cell disease, leukemia, Von Willebrand disease, or hemophilia. The history also should note any use of anticoagulant or antiplatelet medications such as aspirin, NSAIDS, clopidogrel, warfarin, or enoxaparin. In children, any delay in presentation or historical inconsistency warrants concern of child abuse.

A CT scan may be necessary to rule out associated orbital fractures or open-globe injury. All hyphemas require slit-lamp examination to evaluate the anterior chamber, and tonometry once an open-globe injury is ruled out.

Twenty-five percent (Grade I) to 67% (Grade III) of hyphemas will rebleed, usually 2-5 days post-trauma.[63] For this reason, management is directed at limiting rebleeding and subsequent elevations in intraocular pressure. This management may vary among specialists; ophthalmology should be notified prior to initiating treatment. The patient should be positioned with the head of the bed elevated to promote settling of the hyphema, and a rigid shield should be placed over the eye to prevent further injury. Topical cycloplegics, such as 1% atropine and topical steroids, may be given to relieve photophobia and intraocular inflammation. To limit rebleeding, recommendations include a five-day regimen of either aminocaproic acid or prednisone. Aminocaproic acid is favored in the setting of sickle cell disease, and may require concomitant, antiemetic therapy. For elevated intraocular pressures (> 25 mmHg), topical beta-adrenergic antagonists, alpha-adrenergic agonists, and carbonic anhydrase inhibitors are recommended. Table 3 outlines indications for outpatient management of hyphemas. If a patient is discharged, he should be instructed to discontinue all anticoagulant/antiplatelet medication, avoid any rigorous activity, and follow up with ophthalmology within 24 hours.[12,62,63,67]

Cyclodialysis. Cyclodialysis is a disruption of the ciliary muscle attachment at the sclera that results in a cleft allowing extravasation of aqueous humor into a potential space within the choroids. It will present in an estimated 4% of blunt eye traumas.[67] Patients will complain of poor vision with possible pain, erythema, and tearing. Because of the loss of volume, the anterior chamber will appear shallow on slit-lamp examination, and intraocular pressures will be low. This hypotony can lead to corneal edema, choroidal detachment, and optic disc edema. All cases of cyclodialysis will require surgical correction, but most of the sequelae are chronic manifestations. Normal vision still can be restored within eight weeks of the injury. Thus, patients may be discharged home with topical cycloplegics, and follow-up with an ophthalmologist within 3-4 days.[58,68]

Lens Subluxation. Lens subluxation is a partial displacement of the lens off the visual axis due to disruption of the lens zonule fibers that anchor it to the ciliary body. Blunt trauma is the most common mechanism. A complete dissociation of the lens from the ciliary body is known as a lens dislocation.

Patients may present with reduced visual acuity, monocular diplopia, or a visual glare, but symptoms may be delayed until months after the initial trauma. Conditions that predispose to lens dislocation with minimal trauma include Marfan's syndrome, homocystinuria, syphilis, Weill-Marchesani syndrome, and retinitis pigmentosa. A slit-lamp examination with pupillary dilation will reveal a displaced lens, prolapse of vitreous into the anterior chamber, or iridodonesis (a trembling of the iris with rapid eye movements). In children, an asymmetric red reflex will characterize lens abnormalities. Subluxation and posterior dislocation are benign conditions that can be managed with a corrective lens. Anterior displacement of a dislocated lens into the anterior chamber is an ocular emergency leading to pupillary obstruction and elevated intraocular pressures (pupillary block glaucoma). Any concern for anterior dislocation requires immediate ophthalmology evaluation for surgical correction, assessment of intraocular pressures, and management with topical beta-adrenergic antagonists, alpha-adrenergic agonists, or carbonic anhydrase inhibitors.[16,17,69]

Traumatic Cataracts. A traumatic cataract results from swelling and opacification of the lens secondary to disruption of the external capsule. This is the most common lens injury in trauma occurring in 39% of open-globe injuries and 11% of closed-globe injuries.[69] In addition, traumatic cataracts accounts for 10% of eye injuries secondary to assault,[6] 32% of workplace-related injuries,[1] and 12% of sports-related injuries.[2] Symptoms tend to be delayed in onset (weeks to months) with a common complaint of a unilateral, progressive blurring of the vision. Because the majority are associated with open-globe injuries and associated vitreoretinal injuries, many traumatic cataracts are diagnosed and managed in the operating room. Chronic, isolated injuries will be apparent on slit-lamp examination and are managed conservatively with ophthalmology follow-up in 2-3 days. Because a swollen, deformed lens may obstruct the pupil lending to pupillary block glaucoma, intraocular pressures should be assessed prior to discharge.[16,70]

Vitreous Hemorrhage. Vitreous hemorrhage is the extravasation of blood into the vitreous space. It is one of the most common post-traumatic ocular injuries, accounting for 40% of assault-related eye injuries,[6] 42% of work-related eye injuries,[1] and more than 60% of paintball-related injuries.[65] Vitreous hemorrhage also has a strong association with shaken baby syndrome.[17] The sources of bleeding include the iris, ciliary body, choroids, and retina. Although the mechanism may be blunt or penetrating, vitreous hemorrhage most often is associated with a closed-globe injury. Visual deficits represent the most common presentation, with patients complaining of a haze, floaters, cobwebs, smoke signals, or simply a shadowy appearance to their vision. This may be associated with pain or photophobia, depending on the site of disruption. Work-up is directed at excluding retinal detachment. Direct ophthalmoscopy should be employed, but may be obscured by blood. Ultrasound is useful in diagnosing a corresponding retinal detachment, but CT imaging will provide additional information on the integrity of the choroids, sclera, or any evidence of intraocular foreign body. Treatment of an isolated vitreous hemorrhage is conservative with symptomatic relief of pain as needed. Patients can be discharged home with instructions for bed rest with head elevated and ophthalmology follow-up in 2-3 days.

Complications related to vitreous hemorrhage include retinal detachment secondary to traction from the hemorrhage and ghost cell glaucoma, elevated intraocular pressures secondary to obstruction of aqueous humor outflow tracts by the byproducts of hemoglobin degradation. Onset is usually 1-3 weeks post-trauma, and patients will present with a painful eye with a beige collection of cells within the anterior chamber that often is confused for a hypopion. The presence of ghost-cells, elevated intraocular pressures, and impending retinal detachment are all indications for immediate surgical evacuation of the vitreous.[33,71,72]

Table 3. Traumatic Hyphema: Indications for Outpatient Management

- No associated ocular injury mandating hospitalization
- Hyphema less than Grade II
- Intraocular pressure < 35 mmHg
- No history of sickle cell disease, blood dyscrasias, or coagulopathic disorders
- No concern regarding the safety of home environment (including child abuse), ability to comply with limited activity, ability to comply with medication regimen, or ability to follow up with ophthalmology within 24 hours

Vision Loss with an Afferent Pupillary Defect

Trauma to the posterior segment of the eye (retina/optic nerve) may impair the pupillary light reflex, causing a pupil that will fail to constrict with direct light stimulation, known as a relative afferent pupillary defect.[73] In addition, posterior segment injuries also may have profound visual deficits, including complete loss of vision.

Retinal Detachment. Retinal detachment is a separation of the superficial neurosensory layer of the retina from the underlying pigmented layer. Fifteen percent of all retinal detachments are caused by trauma with blunt mechanisms (70-85% of cases), the most common etiology.[74] The development of a detached retina is a three-step process. The first is a break in the retinal layer generated during the initial trauma. Retinal dialysis (detachment from ciliary body) is the most common of these precipitants, but others include operculated holes, horseshoe flaps, or areas of necrosis. Secondly, fluid will seep into this defect and weaken the bond between the two superficial layers. Finally, traction must be provided to separate the two layers. During the initial trauma, this is provided by a blunt mechanism and the subsequent elastic recoil of the globe that places traction at the vitreous base. In many instances, this traction is a delayed process caused by the contraction of the vitreous secondary to healing.

Due to the separation from its blood supply in the choroids, the separated layer becomes ischemic and irreversibly atrophies.

In many cases, a retinal detachment will be delayed months to years after the initial injury. Patients will complain of seeing light flashes progressing to a "falling curtain of darkness." This may lead to profound visual deficits with a relative afferent pupillary defect, an unreactive iris to direct stimulation that constricts with stimulation of the opposite eye (consensual response). Examination in children may reveal a diminished red reflex. On direct ophthalmoscopy, detachments appear as grayish billowing of the retina; however, many smaller or peripheral detachments may not be seen with simple fundoscopy (even after dilating the pupil). Ultrasound will reveal a smooth membrane within the vitreous cavity or a triangular shape extending from the optic disc in cases of total detachment. Immediate surgical intervention is indicated, thus, any pupillary deficit or suspicion of retinal detachment requires immediate ophthalmology evaluation.[16,71,72,75-77]

Because of the high incidence of ocular findings in child abuse, retinal detachment in children should raise suspicion, but retinal hemorrhage in children younger than 3 years is pathognomonic for shaken baby syndrome.

Optic Nerve Injuries. Optic nerve injuries have a 7% incidence in major trauma[9] and will result in permanent visual loss in 50% of cases.[78] The optic nerve can be injured by two major mechanisms. Direct injuries cause trauma to the nerve usually by penetrating, open-globe mechanisms such as bone fragments, foreign bodies, or projectiles (e.g., bullets, BBs). Walsh and Hoyt described indirect injuries as a "traumatic loss of vision which occurs without external or initial ophthalmoscopic evidence of injury to the eye or the nerve."[79] These injuries are caused by transmitted forces upon the optic nerve due to bony apposition or globe mobility. Indirect injuries are the most common cause of traumatic optic neuropathy usually attributed to blunt, deceleration injuries with impact at the supraorbital or frontal regions of the head. Such forces are encountered in motor vehicle collisions, bicycle accidents, assaults, and falls, but optic nerve injuries also have been described in lower energy mechanisms such as falling debris and skateboard accidents.[77,80,81]

The optic nerve can be divided into four anatomic sections: the intraocular portion that we recognize as the optic disc; a mobile intraorbital section; a fixed intracanalicular section that is accompanied by the ophthalmic artery as it runs through the optic canal; and an intracranial section that joins the complimentary optic nerve to form the optic chiasm. The intracanalicular portion is the most common site of injury and follows a dual insult paradigm. The primary insult, at the time of initial trauma, results from shearing forces due to a mobile globe and intraorbital nerve section placing stress on an immobile intracanalicular section and vessels. This results in permanent axonal injury, as well as, disruption of the vasculature with possible hemorrhage. This loss of blood supply produces a secondary ischemia and circulation of toxic metabolites such as free radicals, bradykinin, and calcium. Any axons that survive the initial insult may succumb to the secondary effects of ischemia, contributing to the delayed presentation of optic nerve injury.[75,77,79,82,83]

The most common presentation for traumatic optic neuropathy is a visual deficit with an afferent pupillary defect (Marcus-Gunn pupil) on exam. Typically, patients present with vision of 20/400 or less, with 10% having a delayed-onset of symptoms. Visual fields deficits are indicative of intracranial injury, but in most instances will be difficult to elicit; more than 50% of optic nerve injuries are associated with a loss of consciousness.[50,77]

Any deficits in vision should raise suspicion of an optic nerve injury. Again, an afferent pupillary defect is diagnostic of a posterior segment injury and requires immediate ophthalmologic evaluation. Delayed loss of vision can be described as a lucid interval before normal vision rapidly fades. Often it is associated with compression from an expanding hematoma; in most cases, full visual acuity is returned with prompt surgical decompression. This rapid deterioration of vision has a good prognosis, while delayed regressions during a 1-2 week period are associated with axonal atrophy and suggest more permanent visual deficits.

The physical exam should focus on reversible causes of optic nerve injury—specifically signs of retrobulbar hemorrhage such as a rigid globe and diplopia. Ophthalmoscopic examination will vary depending upon the location of the injury. Anterior injuries involving the intraocular portion of the nerve will demonstrate an edematous retina with a pale optic disc if the central retinal artery is involved. Otherwise, an avulsed intraocular optic nerve will produce a hemorrhagic ring on the fundus, with the optic disc appearing as a deep round pit. Posterior injuries involving the intracanalicular portion of the nerve will appear normal on initial presentation. Only after 3-6 weeks will the disc appear pale and atrophic. In more subtle cases, be aware of the close association of optic nerve injury with midface fractures (2.5% incidence). All patients with suspected ocular nerve injuries should receive a CT scan to evaluate for fractures, as well as, pathologic nerve sheath hematomas or retrobulbar hemorrhages.[16,77,84,85]

In 1998, The International Optic Nerve Trauma Study[86] attempted to evaluate the current management recommendations for optic nerve trauma, high-dose steroids vs. surgical decompression. Researchers concluded that neither improved visual outcomes, but this study was retrospective and grossly lacked power (n=133). There has never been a prospective, randomized trial to determine efficacy in the management of these injuries; one of the reasons is that the incidence of the disorder is too low to generate statistical significance within a given community. Thus, high-dose steroids commonly are used in some centers. Surgical decompression also is employed, but due to a lack of a universal protocol, the use of these therapies will vary by region, therefore, a consulting ophthalmologist should be notified prior to initiating any treatments.[77,79,81]

Prognosis and Conclusion

In general, post-traumatic eye injuries with poor visual acuity on initial presentation have a lower probability of regaining baseline vision. Pieramici et al demonstrated that 88% of patients with post-traumatic visual acuity of 20/40 or better retained that degree of acuity on follow-up visits. In contrast, 79% of patients with a post-traumatic inability to perceive light on initial evalua-

tion required enucleation of the affected eye.[87] Much of this prognostic data related to eye trauma focused on open-globe injuries in recent years; other initial indicators of poor visual outcome in the setting of these injuries include the length of the eye-wall defect, presence of an afferent pupillary defect, prolapse of intraocular contents, and finally, the presence of a hyphema.[88,89]

Pitfalls in assessing and managing eye trauma are related to the rarity of true vision-threatening injuries. The majority of the eye injuries evaluated by emergency health care providers will be benign, but complacency can be overcome by maintaining a systematic approach to all eye injuries, while being mindful of the hallmarks of severe injuries such as rigid proptosis and the absence of a pupillary response. As a final precaution, all eye injuries, regardless of their triviality, should be referred for follow-up ophthalmology evaluation.

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CE/CME Objectives

Upon completing this program, the participants will be able to:

a.) recognize or increase suspicion for traumatic injuries that present to the emergency department;

b) describe the various modalities used to identify different traumatic conditions covered in the newsletter;

c) describe how to correctly and quickly stabilize, and then to manage patients with the particular condition covered in the newsletter; and

d) identify both likely and rare complications that may occur with traumatic injuries.

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

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CE/CME Questions

1. Which of the following conditions is *not* characterized as an open-globe injury?
   A. Intraocular foreign body
   B. Corneal abrasion
   C. Corneal laceration
   D. Scleral rupture

2. Which of the following conditions is *not* associated with diplopia?
   A. Orbital wall fractures
   B. Lens subluxation
   C. Retrobulbar hemorrhage
   D. Retinal detachment

3. A rigid rock-hard proptotic eye is characteristic of what post-traumatic eye injury?
   A. Retrobulbar hemorrhage
   B. Traumatic hyphema
   C. Scleral rupture
   D. Orbital wall fracture

4. Which of the following conditions is *not* associated with elevated intraocular pressures?
   A. Traumatic hyphema
   B. Vitreous hemorrhage
   C. Anterior lens dislocation
   D. Posterior lens dislocation

5. Which of the following eye injuries is the most specific for shaken baby syndrome?
   A. Retinal hemorrhage
   B. Retinal detachment
   C. Traumatic optic neuropathy
   D. Vitreous hemorrhage

6. Which of the following is *not* part of the anterior segment of the eye?
   A. Lens
   B. Iris
   C. Retina
   D. Cornea

7. Which of the following groupings has the highest incidence of eye trauma?
   A. Male, ages 10-15
   B. Female, ages 10-15
   C. Male, ages 25-30
   D. Female, ages 25 -30

8. Which of the following agents account for the majority of corneal burns?
   A. Acidic chemical agents
   B. Alkali chemical agents
   C. Thermal agents
   D. Ultraviolet light

9. What are the two most common bones injured in orbital wall fractures?
   A. Maxilla and sphenoid
   B. Maxilla and frontal
   C. Maxilla and ethmoid
   D. Maxilla and zygoma

10. Where is the most common anatomic site of optic nerve injury from blunt trauma?
    A. Intraocular (optic disc)
    B. Intraorbital
    C. Intracanalicular
    D. Intracranial (optic chiasm)

Answer Key: 1.B; 2.D; 3.A; 4.D; 5.A; 6.C; 7. A; 8.B; 9.C; 10.C

In Future Issues:

Management of the Burned Patient