Acute Ocular Trauma in a Child: A Teaching Case Report

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Abstract

Ocular injuries are common among children, especially boys. Nonpenetrating ocular trauma can cause various types of damage in the eye. The adnexa, anterior segment and posterior segment can all be affected. This damage can be mild to severe and transient or permanent. This case report of a nonpenetrating ocular injury of a young boy reviews the clinical findings and management.

Key Words: *Ocular trauma, closed globe, child, traumatic iritis, commotio retinae*

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Background

Trauma to the eye is a common cause of ocular morbidity encountered by eyecare providers. Nonpenetrating ocular trauma can manifest in many ways in both the anterior and posterior segments depending on the nature and severity of the trauma. The following case report discusses a young boy with ocular trauma and its sequelae. It is most appropriately used as a teaching guide for second-, third- and fourth-year students, as well as early residents, particularly those participating in urgent care clinics. For second-year students, even if they have not begun examining patients, it could be used as a review of ocular anatomy, as all portions of the globe can be affected in blunt trauma. Additionally, the case can be used to reinforce the uses of ocular health examination skills that second-year students learn (slit lamp, gonioscopy, tonometry and fundus evaluation). For upper-level students and residents examining patients, the potential sequelae of a trauma case will allow for the review of damage to nearly all structures of the eye and the discussion of complex management decisions encountered in these cases.

Student Discussion Guide

Case Description

Patient LH, a 10-year-old African American male, presented to our urgent care clinic with his mother on Saturday, Sept. 20, 2008. He reported having been hit in the right eye two days prior with a plastic toy sword. He was complaining of blurry vision, photophobia, redness and mild pain (2 on a scale of 10). He reported that the pain had improved since the incident, but the blur and photophobia had not. His ocular history was significant for vision therapy in our pediatric clinic for saccadic dysfunction and accommodative insufficiency in 2005. He wore no spectacle correction and had never been prescribed one.

Medical history was remarkable for attention deficit hyperactivity disorder, which was treated with dexmethylphenidate (Focalin, Novartis). An allergy to the antibiotic cefuroxime (Ceftin, GlaxoSmithKline) was reported, but no other allergies were known. Family history was positive for hypertension. Examination findings are noted in Table 1.

The patient was prescribed prednisolone acetate (Pred Forte 1%) q.i.d. OD and homatropine 5% b.i.d. OD and scheduled to return in five days.

Follow-up #1: 9/25/08

One week after the trauma, LH reported his eye felt much better. He no longer had blur or photophobia, but he still experienced some mild pain. His mother reported he had been taking the homatropine appropriately since the last visit. However, they were unable to obtain the Pred Forte until 9/23/08, so he had only been taking it for two days. See Table 2 for pertinent exam findings. The patient was told to continue taking the Pred Forte q.i.d. and the homatropine b.i.d. OD. He was scheduled to return in nine days, on a Saturday, so that he did not have to be taken out of school.

Follow-up #2: 10/4/2008

Sixteen days after the initial injury, LH reported that all of his symptoms had subsided. Despite instruction to continue the medication as before, the patient had only been taking the Pred Forte b.i.d. and the homatropine q.d. OD. See Table 3 for pertinent exam findings. The patient was asked to continue the current drop regimen (Pred Forte b.i.d. and homatropine q.d. OD) and to return in one week.

Follow-up #3: 10/11/2008

The patient returned 23 days after the trauma. He again had no complaints regarding his eyes. About four days prior, the patient's mother had decided to discontinue all eye drops, so at this time he was not being treated with any medications. See Table 4 for pertinent exam findings. At this time the medications were not re-initiated. The patient was asked to return in two weeks, but due to a scheduling conflict he was scheduled for a three week follow-up.

Follow-up #4: 11/1/2008

The final examination for this patient was 43 days after the trauma OD. He had no ocular complaints at this time, and he was not taking any ocular medications. See Table 5 for pertinent exam results.

Learning Objectives

At the conclusion of the case discussion, participants should be able to:

Table 1
Initial Presentation 9/20/2008

	OD	OS
Visual Acuity (sc)	20/50, pinhole 20/40	20/25
Pupils	ERRL, -APD	ERRL, -APD
Extraocular muscles	Full range of motion	Full range of motion
Confrontation fields	Full to finger counting	Full to finger counting
External	Ecchymosis of upper lid	Clear
Biomicroscopy: Conj	3+ perilimbal injection	Clear
Cornea	- NaFl stain, + fine keratic precipitates	Clear
Anterior chamber	3+ cells, moderate flare, no blood	Deep and quiet
Iris	Clear	Clear
Lens	Clear	Clear
Intraocular pressures (Goldmann)	17 mmHg	19 mmHg
Dilated fundus exam	0.2 C/D, - cells, pale (flat) area of retina inferior temporal	0.2 C/D, normal findings with all structures

Table 2
Follow-up #1 9/25/2008

	OD	OS
Visual acuity (sc)	20/25	20/25
Pupils	Dilated, nonreactive	RRL
External	Mild ecchymosis, improved	Clear
Conjunctiva	Trace injection	Clear
Cornea	Fewer keratic precipitates than previously	Clear
Anterior chamber	2+ cells, moderate flare	Deep and quiet
Intraocular pressures	13 mmHg	13 mmHg
Dilated fundus exam	White area still present, but improved	Normal findings

Table 3
Follow-up #2 10/4/2008

	OD	OS
Visual acuity (sc)	20/25	20/25
External	Clear, ecchymosis resolved	Clear
Conjunctiva	Clear	Clear
Cornea	Clear, - keratic precipitates	Clear
Anterior chamber	1+ cells, trace flare	Deep and quiet
Intraocular pressures	14 mmHg	14 mmHg
Dilated fundus exam	Clear, whitening resolved	Clear

Table 4
Follow-up #3 10/11/2008

	OD	OS
Visual acuity (sc)	20/25	20/25
Anterior chamber	Trace cells, no flare	Deep and quiet
Intraocular pressure	14 mmHg	14 mmHg

1. Describe potential clinical findings of ocular trauma, using the anatomy of the eye as a guide.
2. Understand the impact each potential complication from ocular trauma may have on the vision and prognosis.
3. Differentiate a ruptured globe from a closed-globe trauma.
4. Understand the short- and long-term management of patients with ocular trauma.
5. Correlate clinical findings with the patient history to determine diagnosis.

Key Concepts

1. Recognition of clinical findings with ocular trauma.
2. Management of clinical findings with ocular trauma.
3. Considerations in treating a potentially self-limiting condition.
4. Education and management of long-term sequelae of trauma.
5. Recognition of concomitant systemic disease implications with trauma.

Discussion Questions

A. Knowledge of Potential Clinical Findings
   1. What are the possible manifestations of closed-globe ocular trauma?
      a. Anterior segment
      b. Posterior segment
      c. Globe and adnexa
   2. Of the above, which are short-term or transient and which are long-term or permanent?
   3. Which potential trauma manifestations were ruled out by normal entrance tests?
      a. Pupils
      b. Extraocular muscle motility
      c. Confrontation visual fields
   4. What potential findings would be the most concerning for the patient in the long term?
   5. How would a ruptured globe from trauma be ruled out?

Table 5
Follow-up #4 11/1/2008

	OD	OS
Best corrected VA	20/20	20/20
Refraction	-0.25 DS	-0.25-0.50x180
Anterior chamber	Deep and quiet, -cell/flare	Deep and quiet
Intraocular pressure	15 mmHg	15 mmHg
Gonioscopy	Ciliary body in all quadrants	Ciliary body in all quadrants
Dilated exam with scleral depression 360°	No retinal breaks	No retinal breaks

B. Comprehension of Clinical Examination Results
   1. What is the differential diagnosis of the cells in the anterior chamber? What about the retinal whitening?
   2. What are the manifestations from trauma this patient exhibits? (What is your assessment?)
   3. Which portions of the case history and exam findings support the diagnoses from question 2?
   4. What should be the treatment plan? Is treatment necessary?
   5. How should the patient be educated regarding the benefits and side effects of the medications prescribed?
   6. Why were gonioscopy and scleral depression done at the last visit? Should they have been done sooner?

C. Consideration of Medical and Social Issues
   1. If this patient had a hyphema, what systemic conditions would need to be ruled out? Why?
   2. This patient had questionable compliance. How could this be dealt with?
   3. How should the patient and parent be educated for the prevention of future ocular injuries?
   4. What are the requirements for reporting suspected physical abuse in children?

Educator's Guide

Case Description

This case demonstrates a young boy with closed-globe trauma due to a plastic toy sword. The examination details are presented in the Student Discussion Guide. Below are the considerations and diagnoses for each visit.

Initial Presentation: 9/20/2008

Visual acuity was decreased OD, most likely due to the inflammation in the anterior chamber and the keratic precipitates. Pupil testing was normal, which rules out acute optic nerve damage. Extraocular muscle movements were normal without diplopia, which rules out a muscle entrapment in a blow-out fracture. The patient had ecchymosis of the upper lid OD, but no lid laceration. The cornea showed no staining, so no abrasion was present at this time. However, there may have been a small abrasion that resolved previous to the exam, as the trauma was two days prior and the pain level had been higher initially.

The keratic precipitates, perilimbal injection, cells and flare are indicative of a traumatic iritis (TI). No blood was noted in the anterior chamber, so there was no hyphema. If there were a hyphema, a diagnosis of sickle cell anemia would need to be ruled out, especially because the patient is African American. The iris showed no tears, and the crystalline lens

had no defects. Intraocular pressures (IOPs) were asymmetric, slightly lower OD than OS. This was likely due to the iritis. A significantly lower pressure OD may indicate a penetrated globe, which would need to be managed very differently from a closed-globe trauma such as this one.

Dilated examination showed only commotio retinae (CR) OD. Gonioscopy and scleral depression were not performed at this visit due to the traumatic iritis. They were deferred for one month after the injury.

The patient was diagnosed with TI and CR. Treatment was initiated for the TI due to the patient's complaints, although many TIs resolve without treatment.[1] The patient and his mother were educated about the side effects of the medications.

Follow-up #1: 9/25/2008

The patient had a slight improvement in the TI. More improvement may have been seen, but he did not take the Pred Forte until two days prior to this examination. The CR was resolving, but still present. Because there had only been a mild improvement in the TI, the current medication schedule was not changed.

Follow-up #2: 10/4/08

At this visit the CR had resolved completely. The TI was still present but improved. The patient was continued on the eye drops to expedite the resolution of the inflammation.

Follow-up #3: 10/11/08

At this time the TI was almost completely resolved. The patient had already discontinued the medications, and they were not reinstated because the inflammation was almost resolved.

Follow-up #4: 11/1/08

The purpose of this visit was to perform gonioscopy to rule out angle recession. In addition, scleral depression was performed to rule out a retinal break. Neither of these conditions was found, so the patient's parent was educated on possible long-term sequelae and encouraged to return annually for comprehensive exams.

Literature Review

Trauma is a leading cause of visual impairment in the United States. An estimated 2.4 million ocular injuries occur in this country every year. The majority of these patients are young, with 57% below age 30, and 80% being male.[2,3]

Ocular trauma can be either closed-globe, as this one, or penetrating. This is an important distinction, and a penetrating injury must be ruled out with an in-depth ocular examination. In penetrating injuries, one might find a full-thickness laceration to the sclera or cornea (positive Seidel's sign), a deep or shallow anterior chamber compared with the other eye, an irregular pupil, iris transillumination defects, low IOP or limited extraocular muscle motilities.[3] Subconjunctival hemorrhages are also a common finding after trauma – with or without a ruptured globe. If the subconjunctival hemorrhage is 360°, then exploratory surgery may be necessary to rule out a ruptured globe that is being masked by the hemorrhage. The diffuse subconjunctival hemorrhage may also be indicative of a retrobulbar hemorrhage, which may also require hospitalization and surgical treatment.[1]

Non-penetrating trauma to the eye can manifest in many ways. A nonexhaustive list of possible sequelae is shown in Table 6. In the cornea, closed-globe trauma can cause abrasions, scarring, persistent edema and endothelial blood staining (residual from hyphema).[2] The anterior chamber may manifest TI or hyphema, which would be of particular concern in a person with sickle cell anemia (more common in those of African descent) due to the increased likelihood for elevated IOPs and rebleeding caused by sickling of the red blood cells.[4] The anterior chamber angle may have recession of varying degrees. Trauma may affect the iris with tears or iridodialysis, and in some cases the patient is left with permanent mydriasis due to the injury. The crystalline lens may be affected with traumatic cataracts (anterior cortical or posterior cortical), which are often rosette-shaped. Lenses may also become partially or completely dislocated.[2]

In the posterior chamber, patients may have vitreous hemorrhage, CR, choroidal rupture, macular holes, optic neuropathies and retinal breaks (tears, detachment, or dialysis).[5] Orbitally, a blow-out fracture must be ruled out. This can be done with extraocular muscle movements, palpation of the orbital rim for fractures, palpation of the eyelids for crepitus, and comparing the sensation of the infraorbital areas.[1] All of these potential sequelae should be ruled out in a patient with a history of nonpenetrating ocular trauma. If iritis or hyphema is present, gonioscopy and scleral depression are typically deferred for two to four weeks after the trauma. However, if the IOP is uncontrollably elevated, then gonioscopy can be performed carefully.[1,3]

According to a retrospective study by Canavan and Archer, the most common initial finding after ocular trauma is hyphema, and the most common long-term consequence is angle recession, which occurred in 80.5% of the cases reviewed. This was followed by iris trauma and lens injury. This retrospective study was performed on patients who were admitted to a hospital, perhaps leading the findings to be skewed more to the severe as compared to a walk-in eye clinic. The same study also found that young males were the most likely group to have ocular injuries, and that they most commonly occurred during play and sport.[2] A small study of the acute injuries associated with "Airsoft" guns, performed by Ramstead et al in an on-call ophthalmology clinic, also found that the most common ocular manifestation was hyphema, followed by traumatic mydriasis, corneal abrasion, and lid contusion.[6]

The long-term concern in these patients is the risk for the development of post-traumatic glaucoma. The rise in IOP after trauma can be unpredictable and may occur immediately or years after the injury.[7] The potential etiologies of the IOP spike, and development of optic nerve damage indicating glaucoma, are many. Secondary glaucoma may develop from angle closure due to pupillary block caused by loosened or broken lens zonules resulting in lens subluxation. Secondary angle closure may also occur either due to 360° of posterior synechiae or due to direct obstruction of the trabecular meshwork from peripheral anterior synechiae. The latter is more often seen with chronic inflammation. Secondary open angle glaucoma may develop due to direct damage and subsequent scarring of the

trabecular meshwork from the injury. Additionally, there may be a reduction or elimination of the tractional effect of the ciliary muscle pulling the scleral spur, which can also reduce trabecular outflow. Finally, obstruction of the trabecular meshwork by inflammatory debris, lens particles or red blood cells from hyphema may occur.[7, 8] A study by Girkin et al. found that within six months of their injuries, 3.4% of patients developed glaucoma. The same study found that the highest risk for glaucoma development was with the presence of a hyphema upon initial presentation. Patients with visual acuity worse than 20/200 at baseline, lens injury and angle recession also showed high conversion to glaucoma within six months.[8] Because many patients develop glaucoma several years after the initial injury, these statistics may look different in a longer-term study. Fortunately, this patient had none of these risk factors, as there was no hyphema, only mildly reduced vision (20/50), no lens injury and no angle recession. Therefore, there is less likelihood of developing glaucoma in the future.

Differential Diagnoses

The two main clinical signs in this case were the anterior chamber inflammation (TI) and the peripheral retinal whitening (CR). Listed below is a differential diagnosis for each of these findings in the presence of trauma.

Differential diagnosis of anterior chamber inflammation:

- Rhegmatogenous retinal detachment
  - A retinal detachment can be caused by trauma. It may be accompanied by an anterior chamber reaction and may show pigment cells in the anterior chamber.
- Traumatic microhyphema
  - This is defined as red blood cells in the anterior chamber.
- Traumatic corneal abrasion
  - This manifests as a sodium fluorescein defect in the cornea. It may be accompanied by an anterior chamber reaction.
- Granulomatous uveitis due to systemic etiology
  - Mutton fat keratic precipitates accompany the anterior chamber reaction. A systemic cause is common, including syphilis, tuberculosis and sarcoidosis.
- Nongranulomatous uveitis due to systemic etiology
  - There are many possible systemic causes of anterior chamber reaction with fine keratic precipitates.
- Traumatic iritis (TI)
  - Defined as nongranulomatous inflammation due to trauma within the preceding few days.

Differential diagnosis of pale area of peripheral retina:

- Retinal detachment
  - Elevation of the retina would accompany a retinal detachment. There also may be a visible retinal break.
- Branch retinal artery occlusion
  - The whitening is along the path of an artery, and edema, cotton wool spots, narrowed arterioles and dilated venules may be seen.
- White without pressure
  - This condition is unrelated to trauma. It may be seen in multiple areas of the peripheral retina, and also may be present in the other eye. Retinal hemorrhages would not be present with this condition.
- Commotio retinae (CR)
  - An area of retinal whitening with a history of recent ocular trauma. Blood vessels are undisturbed, but retinal hemorrhages may be present.

Table 6
Possible Sequelae of Closed-Globe Trauma

Orbit/Adnexa	Anterior Segment	Posterior Segment
Lid laceration	Corneal abrasion	Vitreous hemorrhage
Lid ecchymosis	Corneal scarring	Optic neuropathy
Orbital fracture	Corneal edema	Optic nerve evulsion
Retrobulbar hemorrhage	Endothelial staining	Traumatic glaucoma
	Conjunctival laceration	Commotio retinae
	Subconjunctival hemorrhage	Retinal break
	Hyphema	Retinal detachment
	Iritis	Macular hole
	Angle recession	Choroidal rupture
	Iris sphincter tear	Artery occlusion
	Iridodialysis	
	Cyclodialysis	
	Mydriasis	
	Cataract	
	Dislocated lens	

Discussion

This section discusses the intricacies of this case, questions to promote debate, and other potential findings of ocular trauma that were not found in this case.

This patient was lucky enough to only manifest transient complications from his ocular trauma. He did not present with a hyphema, vision worse than 20/200, lens injury or angle recession. Therefore, according to the study by Girkin et al., his risk of developing post-traumatic glaucoma in the short-term is low[8]. However, due to his young age, he was advised to consistently have annual eye examinations with dilation, and to inform all of his future eye doctors of his history of ocular trauma.

Traumatic Iritis

LH presented with complaints of photophobia, blurred vision and mild pain.

His clinical findings showed perilimbal injection, cells and flare in the anterior chamber and asymmetric IOPs (OD<OS). These are classic symptoms and signs of iritis, and given the history of trauma it is assumed to have a traumatic etiology. In both traumatic and nontraumatic iritis cases, the IOPs tend to be lower in the affected eye due to a reduction in aqueous production from the inflamed ciliary body, but the pressure may elevate over time as inflammatory material obstructs the trabecular meshwork. However, if this occurs in a patient who is being treated with a corticosteroid, steroid response must be ruled out as the cause for the increased pressure.[3]

In both traumatic and nontraumatic iritis, the affected pupil tends to be smaller than the pupil in the nonaffected eye, but in some traumatic cases the pupil may be larger if an iris sphincter tear is present.[1] Unlike a nontraumatic iritis, a TI often resolves on its own if it is mild. In these cases, a topical cycloplegic alone may be used for patient comfort.[3] However, if unresolved, iritis may lead to posterior synechiae, blurred vision from keratic precipitates, corneal edema, increased IOP potentially causing glaucoma and cystoid macular edema.[1] Therefore, some advocate for aggressive treatment of significant inflammation. In this patient's case, the TI was moderately severe, and he was symptomatic at the initial presentation. Therefore, the decision was made to treat him with both a topical cycloplegic and a topical corticosteroid. The schedule of four times a day was selected for the steroid over a more aggressive therapy due to the relatively mild symptoms, as he complained of mild pain accompanying his photophobia and blurred vision.

With his inconsistent use of the medication, the iritis had mostly resolved 23 days after the trauma, and was completely resolved by the 43rd day. Does this information confirm that the patient should have been treated, or does it suggest that treatment was unnecessary? Also, what methods might have been employed to obtain better compliance by the patient and his mother?

Commotio Retinae

The patient also presented with CR. Fortunately, he had peripheral CR only, so it did not affect his visual acuity. This condition presents as a confluent area of whitening of the outer retinal tissue, and it is typically opposite the side of the impact due to the contrecoup effect of the trauma. The retinal blood vessels are unaffected, but intraretinal hemorrhages may accompany the retinal pallor.[1,9,10] When it is in the macular area, CR is referred to as Berlin's edema, and it can cause decreased visual acuity. In such cases, the CR can be measured subjectively with an Amsler grid or objectively with macular optical coherence tomography (OCT). The visual field may also be affected when CR is in the periphery.[10]

The underlying pathogenesis of CR is still debated, and there is no treatment. The patient is monitored for spontaneous resolution, typically occurring in several days. In this case, the retinal whitening resolved completely somewhere between seven and 16 days after the trauma. This coincides with one study that found resolution within 10 days.[11] The lesion typically resolves completely, but in some cases pigmentation remains or macular holes may occur.[11,12] In most cases, the vision returns to normal levels. However, in cases with more severe trauma, the damage to the photoreceptors and RPE is extensive enough to result in permanent vision loss.[11,12] A peripheral visual field may show persistent defects in cases with severe peripheral CR.

Corneal Abrasion

Corneal abrasion is another common transient finding in ocular trauma. It occurs when the corneal epithelium is compromised, exposing its basement membrane and superficial corneal nerves. Therefore, patients with corneal abrasions will complain of significant pain, photophobia and foreign body sensation.[13] They will have an isolated epithelial defect that stains with sodium fluorescein, and they may also have injection to the conjunctiva. The goal of treating corneal abrasions is to promote healing of the corneal epithelium and decrease patient discomfort. To improve comfort, patients are treated with topical cycloplegics to control ciliary spasm. Depending on the size of the abrasion, these eye drops may be used anywhere from just once (in-office) to multiple times daily.[3] Because the corneal epithelium is the main barrier to infection, patients with corneal abrasions are also placed on prophylactic broad-spectrum antibiotics until the lesion is healed. If the abrasion is large or if the patient is significantly uncomfortable, then either a bandage contact lens or pressure patching can be used overnight. This also helps promote healing of the epithelium, as either there is a buffer between the lid and cornea (with a bandage contact lens) or the lid is immobilized preventing re-abrasion from blinking (with patching). However, if the patient is a contact lens wearer or if the eye was scratched by something that may cause infection (e.g., vegetable matter), then neither contact lenses nor patching should be performed.[3] Additionally, topical nonsteroidal anti-inflammatory eye drops can be used to alleviate pain. However, topical corticosteroids and topical anesthetics are contraindicated because they slow the healing process.[3,13] These patients are watched closely until the cornea is re-epithelialized. Any patient with a bandage contact lens or patch should be seen within 24 hours, and other corneal abrasions should be seen within three days. Once the lesion has healed, the practitioner may educate the patient on the possibility of future recurrent corneal erosion, especially in the case of a large abrasion.[3]

Hyphema

As noted previously, hyphema is a common presenting sign of ocular trauma, though this patient was fortunate enough not to have it. Hyphema presents as blood in the anterior chamber, ranging from only a few red blood cells (erythrocytes) seen with a biomicroscope (microhyphema) to a completely blood-filled anterior chamber that can be seen without magnification ("8-ball" hyphema).[1] It is commonly caused by a tear in the ciliary body resulting in damage to the iris vasculature.[14] Patients present complaining of blurred vision and pain. If a TI is also present they may complain of photophobia as well. Treatment of these patients is focused on preventing re-bleeding while the blood reabsorbs and is guided by the amount of the anterior chamber that is filled with blood. It is generally suggested to hospitalize those with an "8-ball" hyphema and noncompliant patients (e.g., small

children).[4] Most other patients can be treated on an out-patient basis with strong topical cycloplegics and topical corticosteroids. The cycloplegic (i.e., atropine 1%) helps to immobilize the iris and ciliary body in an attempt to prevent re-bleeding. The corticosteroid (i.e., prednisolone acetate 1%) will also help prevent re-bleeding by stabilizing the blood-aqueous barrier.[4] Additionally, it will help resolve a concurrent TI. In cases where the IOP is elevated due to aqueous outflow being blocked by red and white blood cells, typically occurring with total hyphemas, topical glaucoma medications can be used, but prostaglandin analogs and miotics should be avoided.[1,3] In the case where IOP is normal or low in the presence of a total hyphema, the practitioner should investigate for a penetrating injury.[3] Patients with hyphema are also asked to limit activity and sleep with their heads elevated to promote reabsorption of the blood. Often a shield is recommended to protect the eye, but the patient should be able to see through it to monitor for a change in vision. They are warned not to take any medications that may decrease clotting of the blood, such as aspirin or other nonsteroidal anti-inflammatory drugs. They are monitored daily initially until improvement is seen. If IOPs cannot be controlled by topical medications, or if significant corneal endothelial blood staining is seen, then surgical evacuation of the hyphema is indicated.[1,4]

As previously mentioned, patients of African descent should be questioned as to their sickle status in the presence of hyphema, as these patients have an increased risk for re-bleeding and elevated IOP. Sickle cell anemia may cause elevated IOP due to the abnormally shaped erythrocytes having a higher likelihood to block the trabecular meshwork.[4] If a patient with sickle cell anemia has elevated IOP, carbonic anhydrase inhibitors and alpha$_2$agonists should be used with caution, due to their effects on erythrocyte sickling and iris vasculature, respectively. If an African American patient is unsure of his/her sickle status, blood work should be done to verify this. Additionally, any patient with a blood dyscrasia, bleeding disorder, or who is on anticoagulant therapy should be watched closely, and the primary care physician should be consulted.[3]

Angle Recession

Angle recession is a permanent sequela of closed-globe trauma, and it has been reported to occur in 70%-100% of eyes with hyphema.[3] It is defined as a rupture between the circular and longitudinal fibers of the ciliary body. This tear is usually symptomless, so it is the examiner's responsibility to identify it upon gonioscopic evaluation. With a gonio lens, the ciliary body will appear widened in one portion or 360° compared to the adjacent angle or the other eye. The exposed ciliary muscle appears light gray or tan as compared to the normal dark ciliary band, so it is typically an obvious distinction.[14] While angle recession does not increase the IOP, it does correlate with an increased risk for post-traumatic glaucoma. A reported 4%-9% of patients with angle recession of 180° or more of the angle will go on to develop glaucoma at some point, typically years, after the trauma.[3,15] Therefore, they should be monitored at minimum yearly.

Post-Traumatic Glaucoma

Post-traumatic glaucoma is the most significant concern after closed-globe trauma. It can be classified as either a rise in IOP soon after the insult, or late-onset increased IOP. These classifications have different etiologies.

As discussed previously, the etiology of an early-onset IOP increase can be pupillary block caused by lens dislocation or disruption of the trabecular meshwork due to injury, inflammation or obstruction. With the former, surgery is indicated to remove the lens. With the latter, the glaucoma is treated similarly to primary open angle glaucoma or secondary closed angle, depending on the mechanism of blockage.[10] The rise in IOP is typically related to the severity of the damage sustained from the trauma. The medications of choice in these situations are those that decrease aqueous production due to the decreased outflow: topical beta-blockers, alpha$_2$agonists, carbonic anhydrase inhibitors, and, possibly, corticosteroids.[14,15]

Even if the patient exhibits no signs of increased IOP initially, the risk for late-onset post-traumatic glaucoma exists. These patients develop glaucoma sometime after the trauma, often many years later. This type of glaucoma is often called "angle-recession glaucoma," as it can be seen among those with significant angle recession (though with low prevalence). However, this may be a misnomer, as the increase in IOP may not be caused by the angle recession itself. Instead, the IOP rises in response to the obstruction of aqueous outflow due to scarring and degeneration of the trabeculum that occurred after the initial trauma, and the recession is just an indicator of previous trauma.[15] This outflow obstruction can also be caused by the growth of a Descemet-like membrane over the anterior chamber angle.[16] There is some thought that these patients have a predisposition to decreased aqueous outflow, causing them to be more susceptible to glaucoma post-trauma.[17] They are also treated with medications to reduce aqueous production, as well as, potentially, prostaglandin analogs to increase uveoscleral outflow.[14] Miotics are avoided, and laser trabeculoplasty has been shown to have little effect on IOP reduction. Filtering surgery can be considered for patients not adequately controlled medically.[14]

It is due to the concern for the development of late-onset post-traumatic glaucoma that patients should be educated to obtain annual comprehensive examinations, and they should know to tell all future eyecare providers of the trauma.

This patient currently has no indications of permanent damage from his ocular trauma. Both his TI and CR resolved completely, and there are no signs of retinal breaks or angle recession. How often should he be monitored? With what tests should he be monitored? What education should be given to the patient and his mother regarding his prognosis?

Prevention of Pediatric Eye Injuries

Eye injury is the main cause of monocular visual disability and blindness in children in the United States. Developmental disabilities may result from such visual impairments which can lead to long-term financial and quality of life concerns.[18] This issue is of such importance that it is included in the

Healthy People 2020 initiatives: "to reduce visual impairment and blindness in children and adolescents age 17 years and under, and increase the use of protective eyewear in recreational activities and hazardous situations around the home."[19]

In one large study, looking at pediatric eye injuries treated in U.S. emergency rooms from 1997-2006, sports was the most common category associated with eye injury, followed by household cleaning chemicals, toys (not including toy guns), furniture and desk supplies.[18] Age-related patterns were also found in this study. Eye injuries related to sports, swimming pools/equipment, toy guns and recreational vehicles increased with age. Injuries due to household cleaning chemicals, furniture and baby items decreased with age.[18]

It is suggested that pediatric eye trauma may be largely prevented by the education of children, use of protective eyewear and increased parental supervision. These tactics may be tailored accordingly.[18] For example, the American Academy of Pediatrics and the American Academy of Ophthalmology strongly recommend protective eyewear for all participants in sports in which there is risk of eye injury. Protective eyewear should be mandatory for athletes who are functionally one-eyed and for athletes whose eyecare providers recommend eye protection after eye surgery or trauma.[20]

What are other examples of patient and guardian education related to ocular injury prevention?

Concern for Child Abuse in Pediatric Ocular Trauma

As primary care providers, optometrists may need to determine whether an ocular injury is the result of abuse. A misdiagnosis of child abuse can have serious consequences for a child and family. However, a child who is a victim of unreported child abuse can suffer further abuse and even death.[21] As optometrists, we have an ethical, moral and legal obligation to report suspected abuse in order to protect the child.

A detailed history is important in trauma cases.[21,22] It may be helpful to allow the parent to lead the history with a narrative of the injury.[22] Use direct, open-ended, non-leading questions when talking with the child.[22] Red flags that may alert the provider to possible abuse include a history that is inconsistent with the injury, no explanation offered for the injury, history that is inconsistent with the child's developmental level, and/or injury blamed on another child or sibling.[21] Additionally, conflicting histories given by caretakers, delay in seeking medical care, and doctor-shopping may be indicators of physical abuse.[22] If abuse is suspected, questioning the child and parent separately, if possible, may be helpful.[21,22] As in any clinical diagnosis, the examination findings should correlate with the history.

Healthcare providers are mandated by law to report suspected abuse. Providers are not required to prove the abuse occurred prior to reporting, and, in fact, they are immune from legal and civil liability when reporting concerns for child abuse. Furthermore, providers who fail to report concerns of child abuse to child protective services can be prosecuted.[22] Optometrists should be aware of their individual state reporting laws and child protective service contacts. In 2008, the U.S. Department of Health & Human Services published a report: Mandatory Reporters of Child Abuse and Neglect: Summary of State Laws. This may be found at: http://www.childwelfare.gov/responding/mandated.cfm. [22] Most states have toll-free numbers for reporting suspected abuse.[23] A list of child abuse reporting numbers may be found at: http://www.childwelfare.gov/responding/reporting.cfm. [24]

Conclusion

This case is representative of the types of trauma cases optometrists frequently encounter. It is important to be cognizant of the possible ocular effects of trauma and the potential long-term effects. Patients with nonpenetrating ocular trauma should be evaluated for the presence of subconjunctival hemorrhage, corneal abrasion, traumatic iritis, hyphema, choroidal rupture, blow-out fracture, traumatic optic neuropathy, retinal hemorrhages, commotio retinae, angle recession and retinal breaks. The patient's risk for the development of post-traumatic secondary glaucoma should be assessed, and the patient should be counseled on the risk for its development in the future, as well as future ocular injury prevention. In cases of pediatric trauma where physical abuse is suspected, optometrists are required to report the case to appropriate authorities for further investigation.

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