Traumatic Hyphema: A Teaching Case Report

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Abstract

Hyphema is the presence of blood in the anterior chamber of the eye and is most often caused by blunt ocular injury. Hyphema, its complications and associated ocular injuries can pose a serious threat to vision and therefore require appropriate medical management and careful examination and follow-up. This teaching case report reviews the management of traumatic hyphema and discusses treatment options, potential complications and visual prognosis.

Key Words: traumatic hyphema, closed-globe injury, glaucoma medications, traumatic glaucoma

Introduction

The following case report is to be used as a teaching guide appropriate for third- and fourth-year optometry students as well as optometry residents. The case report describes a patient with hyphema as a result of closed-globe injury who develops a number of associated sequelae of blunt force trauma to include increased intraocular pressure, vitreous hemorrhage and angle recession with iridodialysis. The paper discusses appropriate management and treatment options as well as short- and long-term complications of traumatic hyphema. This case demonstrates the importance of identifying clinical findings related to poor visual outcome and how to manage them appropriately.

Learning Objectives

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

1. Discuss signs and symptoms of initial presentation of traumatic hyphema
2. Discuss appropriate treatment for the initial presentation of traumatic hyphema
3. Recognize short- and long-term complications associated with traumatic hyphema
4. Recognize initial and long-term clinical findings related to poor visual outcome as a result of traumatic hyphema
5. Discuss appropriate treatment options for management of increased intraocular pressure (IOP) or glaucoma that results from hyphema or associated complications
6. Provide proper patient education on self-care needed to avoid complications.

Key Concepts

1. Pathophysiology and natural history of traumatic hyphema and its complications
2. Risk factors associated with poor visual prognosis
3. The role of medication in reducing the risk of developing complications
Case Description

Patient RM, a 60-year-old Caucasian male, presented to the eye clinic at the White River Junction Veterans Affairs Medical Center on Aug. 21, 2012, complaining of severe pain, blurred vision and sensitivity to light in the left eye. The patient reported someone threw a rock at him that hit his left eye the previous evening around 7 p.m. The patient had glasses, but was not wearing them at the time of the injury. RM denied flashes and floaters.

RM was an established patient at the eye clinic. His ocular history included a branch retinal vein occlusion in the right eye diagnosed in 2010, early bilateral cataracts and mild non-proliferative diabetic retinopathy without clinically significant macular edema OU. His medical history was significant for diabetes mellitus type 2, hypertension, mixed hyperlipidemia, obstructive sleep apnea, insomnia, osteoarthritis, post-traumatic stress disorder and adjustment disorder with mixed anxiety. His list of medications included aspirin, ibuprofen, bupropion, tramadol, sertraline, glyburide, metformin, lisinopril, metoprolol, simvastatin, nifedipine and prazosin.

Entering visual acuities without correction were 20/20 OD and 20/150 OS. The right pupil was round and responsive to light and measured 2.5 mm. The left pupil was fixed, mid-dilated, and measured 4.0 mm. Extraocular muscles (EOMS) were smooth, accurate, full and extensive (SAFE) OU. There was no irregularity of the left orbital bone or sinus crepitus on palpation of the orbit and adnexal structures. Slit lamp exam revealed clear lids and lashes OD and mild edema and hematoma of the upper and lower left lids without lacerations. The sclera and conjunctiva were clear of defect or hemorrhage OD. The left eye showed a subconjunctival hemorrhage involving the entire bulbar conjunctiva. The corneas were clear of defect with fluorescein staining and there was negative Seidel sign. Anterior chamber angles were open to grade III Van Herick OU. The anterior chamber of the right eye was well-formed, clear and quiet. The left anterior chamber was well-formed but 2+ red blood cells and a hyphema that measured a vertical height of 2.2 mm were noted (Figure 1). The right iris was normal and without rubeosis; the left iris was dyscoric (irregularly shaped) and free of rubeosis. Goldmann tonometry revealed pressures of 14 mmHg OD and 18 mmHg OS. The patient was dilated with two drops of 1.0% tropicamide and two drops of 2.5% phenylephrine OS. No clear view of the left fundus was observed with either binocular indirect ophthalmoscopy (BIO) or 90D lens due to the debris in the anterior chamber. A B-scan ultrasonography was performed OS, which revealed no retinal detachment, vitreous hemorrhage or subluxated lens.

The patient was diagnosed with traumatic hyphema OS and was prescribed prednisolone acetate 1% ophthalmic solution QID OS and...
atropine sulphate 1% ophthalmic solution BID OS. RM was released from the clinic and given instructions for self-care at home. He was educated to shake the bottle of topical steroid before instillation. He was also given a non-pressure eye patch to reduce photophobia when outdoors or in bright light only. In addition, the patient was given a pair of clear protective goggles to be worn during sleep. He was instructed to rest as much as possible, and to avoid any strenuous activity. A follow-up appointment was scheduled for 24 hours, and the patient was instructed to return to clinic sooner if he developed visual changes or increased pain.

The following day, RM reported the pain in the left eye had improved. He was using his drops as instructed and had worn the protective goggles at night. RM denied flashes but noted that vision in the left eye fluctuated. Visual acuity was measured at 20/80-2 OS; EOMS were SAFE OU. There was no pain or crepitus on palpation of the left orbit and adnexa. Anterior segment exam was the same with the exception of the hyphema which was measured at 2.1 mm. IOPs were measured to be 14 mmHg OD and 26 mmHg OS. The lens was well-positioned with mild nuclear sclerotic cataracts in both eyes. BIO of the posterior segment OS revealed diffuse vitreous hemorrhage with coagulation noted to be greatest in the inferior view. The optic nerve had a cup/disc ratio of 0.20 with healthy and distinct rims. The macula was clear and the periphery was flat and intact 360 degrees. RM was instructed to continue with atropine sulphate 1% ophthalmic solution BID OS and prednisolone acetate 1% ophthalmic solution QID OS. Timolol 0.5% ophthalmic solution BID OS was added to treat the ocular hypertension. The patient was instructed to discontinue oral aspirin after consultation with his primary care physician. The patient was instructed to return to clinic again the following day.

The next day, in the left eye, vision measured 20/50-, the hyphema was measured to be 2.0 mm, and intraocular pressure was measured at 34 mmHg OS despite the use of Cosopt TID. Acetazolamide 500 mg PO BID was added to the patient’s medication regimen, which otherwise remained the same. Patient medical history, including allergies and kidney function, was carefully reviewed prior to initiating oral acetazolamide. The patient was also educated on the side effects of acetazolamide to include increased urination, metallic taste and tingling in the extremities. IOP remained elevated at 36 mmHg OS the following day. Pachymetry revealed thick and asymmetric corneas (607 um OD, 662 um OS); thickness was significantly greater in the left eye due to corneal edema that had developed. The patient’s medication was kept the same, and the following day his IOP dropped to 21 mmHg OS. Over the next several visits, vision continued to improve, anterior segment inflammation and corneal edema slowly resolved, and intraocular pressure dropped and stabilized. Prednisolone acetate 1%, Cosopt and acetazolamide were correspondingly tapered and discontinued. See Appendix A for a detailed account of the patient’s pertinent exam findings. Final best corrected visual acuity was 20/20- in the left eye.

Gonioscopy was performed 12 weeks after the initial trauma.

**Summary of Visits and Clinical Outcomes**

The traumatic hyphema slowly resolved after nine days. The anterior segment, level of hyphema and IOP were monitored at each visit. Retinal examinations through direct fundoscopy and B-scan (Figure 2) continued throughout the course of follow-up as well to monitor for cystoid macular edema and to ensure the vitreous hemorrhage was resolving without additional complications such as retinal tear, retinal detachment or vitreoretinal traction. A complication encountered during resolution of the hyphema was a rise in IOP. One week after the initial trauma, intraocular pressure increased to 34 mmHg OS despite the use of Cosopt TID. Acetazolamide 500 mg PO BID was added to the patient’s medication regimen, which otherwise remained the same. Patient medical history, including allergies and kidney function, was carefully reviewed prior to initiating oral acetazolamide. The patient was also educated on the side effects of acetazolamide to include increased urination, metallic taste and tingling in the extremities. IOP remained elevated at 36 mmHg OS the following day. Pachymetry revealed thick and asymmetric corneas (607 um OD, 662 um OS); thickness was significantly greater in the left eye due to corneal edema that had developed. The patient’s medication was kept the same, and the following day his IOP dropped to 21 mmHg OS. Over the next several visits, vision continued to improve, anterior segment inflammation and corneal edema slowly resolved, and intraocular pressure dropped and stabilized. Prednisolone acetate 1%, Cosopt and acetazolamide were correspondingly tapered and discontinued. See Appendix A for a detailed account of the patient’s pertinent exam findings. Final best corrected visual acuity was 20/20- in the left eye.

Gonioscopy was performed 12 weeks after the initial trauma.
after the initial trauma with a three-mirror lens. The right eye was open to ciliary body in all quadrants with 2+ pigmentation of the trabeculum. The left eye was open to ciliary body temporally and nasally. The inferior angle was open to scleral spur with 4+ pigmentation and pigment clumping on the iris (Figure 3). The superior angle revealed four clock hours of angle recession with iridodialysis (Figure 4).

Literature Review

Hyphema is defined as a collection of blood in the anterior chamber. The severity of hyphema can vary from diffuse red blood cells circulating in the aqueous humor to a hemorrhage that fills the entire anterior chamber. Most often hyphema is caused by trauma or intraocular surgery, but may also occur spontaneously in patients with ruberosis iridis, vascular tufts at the pupillary margin, juvenile xanthogranuloma, iris melanoma, myotonic dystrophy, keratouveitis, leukemia, hemophilia, thrombocytopenia or Von Willebrand disease. Hyphema may also be associated with drugs that alter platelet or thrombin function, such as aspirin or warfarin.1

Hyphemas are graded according to examination features as noted in Table 1. The grading system is helpful for predicting clinical outcomes, which assists in educating the patient about short- and long-term prognosis.

Epidemiology

The mean annual incidence of hyphema from all causes is approximately 17 per 100,000.1 The majority of hyphemas occur in males (75%-78%) with a median age of 15.5 to 18.2 years.2 A study of 238 patients with traumatic hyphema showed that the leading cause of trauma was projectile stones, and the majority of the trauma occurred as a result of street violence (43%) and accidents in the home (33%). In children, siblings and friends were responsible for most of the trauma, and in adults the main cause of trauma was accidents.3 Another significant source of injury is sports, which accounted for 60% of traumatic hyphemas in a different study.3 High-risk sports in which the ball hits the eye include baseball, softball, basketball, soccer and paint-
The stick or racquet is more likely to be the source of injury in other high-risk sports such as hockey, racquetball and squash.6

**Pathophysiology**

Hyphema that occurs as a result of trauma is typically caused by damage to the major arterial circle and its branches as a result of a tear in the iris or ciliary body.6 Blunt trauma causes antero-posterior compression of the globe and simultaneous equatorial expansion. This expansion creates stress on the structures of the anterior chamber angle, causing a tear of the ciliary body or iris stromal vessels.1

Patients with hyphema may initially present with low or high IOP. A low IOP may be the result of an accompanying iritis causing reduction in aqueous production or due to temporary increase in outflow from the disruption of structures in the anterior chamber angle.6 More commonly, IOP rises acutely because red blood cells and immune-inflammatory cells block the trabecular meshwork.7 Fresh red blood cells are able to pass through the trabecular meshwork without much difficulty; however, the presence of an overwhelming number of cells in addition to plasma, fibrin and other cellular debris can lead to a transient obstruction of outflow.6 Swelling of the trabecular meshwork (trabeculitis) may also be a contributing factor in limiting outflow.6 In severe cases, acute elevation of IOP may occur secondary to pupillary block, due to a collar button-shaped clot involving both the anterior and posterior chambers.1 The clot prevents the normal flow of aqueous from the posterior chamber, through the space between the iris and lens, and into the anterior chamber. As a result, pressure builds in the posterior chamber, pushing the peripheral iris anteriorly which then closes part or all of the trabecular meshwork via apposition.

Aqueous outflow may be further obstructed in patients with sickle cell hemoglobinopathies. Erythrocytes in these patients become elongated and rigid (sickled) in the aqueous humor, making passage through the trabecular meshwork difficult.6 As a result of an increase in IOP, the anterior and posterior segments of the eye become increasingly hypoperfused and hypoxic, thereby perpetuating a cycle in which further sickling and sludging of erythrocytes occurs.9 Therefore, the incidence of elevated IOP in the presence of hyphema is higher in patients with these disorders, and may occur even in cases of small hyphemas.6

**Associated Trauma**

The pattern of injury from blunt trauma is due to the equatorial expansion of the globe. These injuries have been described as seven rings and include: radial tears of the pupillary sphincter, iridodialysis, angle recession, cyclodialysis, trabecular meshwork tear, zonular dehiscence and retinal dialysis. The presence of these clinical findings, even years after the event, provides evidence of previous blunt trauma.8 Blunt ocular trauma may also cause iridoschisis (separation of the layers of the iris stroma) iritis, cataracts or chorioretinal injury.6

**Treatment and Management**

Management of hyphema from all causes is aimed at preventing secondary hemorrhage, preventing further trauma to the eye, promoting the settling of blood to the bottom of the anterior chamber and controlling traumatic uveitis.4 Close monitoring is essential so that treatment for associated complications is initiated promptly if they occur.

Hospitalization or outpatient care with daily follow-up is recommended. Hospitalization should be considered for patients with severe injuries or blood disorders and those who are not capable of self-care or may be non-compliant with the treatment regimen. Additionally, hospitalization should be considered for children at risk for amblyopia or if child abuse is suspected. Management consists of eye protection with plastic or metal shields, limited physical activity, elevation of head posture, and avoidance of aspirin and other non-steroidal anti-inflammatory agents.10 Having patients sleep at a 30-45-degree angle promotes more rapid blood resorption and lowers venous pressure to the globe, helping to reduce IOP and to allow for clot formation and resolution.7 Normal activities may resume one week after the initial injury or rebleed. However, if blood remains in the anterior chamber after one week, activities should remain restricted until blood resorption occurs.10

Iritis is common in patients presenting with traumatic hyphema. Corticosteroids are prescribed to reduce the inflammation and cycloplegic drugs are used to improve patient comfort and prevent the formation of posterior synechiae.7 Cycloplegics are anticholinergic drugs. They temporarily inhibit acetylcholine receptors in the iris sphincter muscle and ciliary body. This results in pupillary mydriasis, which helps reduce the risk of posterior synechia by minimizing contact between the posterior iris and the anterior lens capsule. Inhibition of acetylcholine receptors in the ciliary body paralyzes the muscle, which relaxes ciliary spasm and reduces pain.11 In addition, both cycloplegics and corticosteroids may reduce the risk of secondary hemorrhage. Steroids stabilize the blood-ocular barrier and directly inhibit fibrinolysis.12 Cycloplegics minimize iris movement and stress on the original ruptured vessels.12

In patients with elevated IOP higher than 25 mmHg, beta blockers and carbonic anhydrase inhibitors (CAIs) are usually first-line treatment.8 Topical CAIs must be used cautiously in patients with sickle cell hemoglobinopathies because these medications may lower the aqueous pH and promote further sickling of the blood cells.10 If topical medication is not adequate in managing IOP, an oral CAI, such as acetazolamide and methazolamide, can be prescribed. The ocular hypotensive effect of acetazolamide in tablet form peaks in two hours and lasts for six hours, whereas in capsule form it peaks in eight hours and persists beyond 12 hours. Acetazolamide is generally dosed as 500 mg PO twice a day for adults. For children, the recommended dose is 5-10 mg/kg of body weight every four to six hours. Methazolamide dosing can begin with 25 mg twice a day and be increased to 50 mg twice a day or up to 100 mg three times a day if needed. Oral CAIs are effective in lowering IOP; however, they have many side effects. Common systemic side effects include increased urinary frequency and paresthesia of the fingers, toes and around the mouth. Other side effects include abdominal discomfort, metallic taste, nausea and diarrhea. Higher
doses of oral CAIs may cause metabolic acidosis and should be avoided in patients with hepatic insufficiency, renal failure, adrenocortical insufficiency, hyperchloremic acidosis, depressed sodium or potassium levels or severe pulmonary obstruction. Oral CAIs are contraindicated in patients with sulfonamide class of drugs. If a systemic CAI is necessary in patients with sickle cell, methazolamide is used because it creates less systemic acidosis, and therefore promotes less erythrocyte sickling, than acetazolamide. Intravenous mannitol, a diuretic, may also be given in cases of uncontrolled IOP; however, extreme caution must be used in patients with sickle cell, as diuretics induce acidosis and volume contraction.

Prostaglandins and pilocarpine are generally avoided in the treatment of elevated IOP because of inflammation associated with the traumatic hyphema. Some early studies showed that large doses of topical prostaglandins resulted in inflammation and breakdown of the blood-aqueous barrier. In more recent reports, a few patients developed anterior uveitis while on latanoprost. Other studies, however, have not shown intraocular inflammatory effects from prostaglandins. An increase in blood-aqueous barrier permeability to plasma proteins has been shown clinically after instillation of pilocarpine. Additionally, miosis induced by pilocarpine increases the zone of contact between the iris and lens, and therefore increases the risk of posterior synechia.

Surgical intervention is required in up to 5% of hyphemas in cases of increased IOP; corneal blood staining, or total hyphemas lasting more than 10 days. If the IOP remains greater than 50 mmHg for five days, or greater than 35 mmHg for seven days, despite medical management, surgery is indicated. Anterior chamber paracentesis is effective for lowering IOP; however it is often only a temporary measure and additional surgical intervention is anticipated. Irrigation of the anterior chamber can be performed to remove dispersed red blood cells, and a formed blood clot can be manually extracted through clear corneal incisions. If necessary, trabeculectomy is performed to manage IOP by creating a new opening for aqueous outflow. Verma reported that the combination of trabeculectomy, peripheral iridectomy and manual extraction of blood clots was satisfactory in lowering IOP. All patients in the study had light projection akyus, corneal blood staining and an average IOP of 45 mmHg before surgery. Average IOP at the last follow-up visit after surgery was 18.4 mmHg, but visual prognosis was still poor. Laser trabeculoplasty is usually ineffective in cases of ocular trauma, due to the damage to the trabecular meshwork.

Additional medications may be used in the treatment of hyphema as blood in the anterior chamber begins to form a clot. Blood clots are cleared from the body through processes known as fibrinolysis. It is during this process that the risk of rebleed is the highest. Antifibrinolytic agents, including ε-aminocaproic acid (ACA) and tranexamic acid, are used to reduce the risk of secondary hemorrhage by slowing or inhibiting the resorption of the blood clot within the traumatized blood vessel. ACA acts as a competitive inhibitor to lysine for binding sites on tissue plasminogen activator, thereby inhibiting the conversion of plasminogen to plasmin. Plasmin is the enzyme involved in the breakdown of the fibrin clot. In addition to preventing the formation of plasmin, ACA also competitively inhibits the binding of plasmin to the fibrin clot itself. These actions stabilize the fibrin clot, thereby preventing rebleeding while permanent vessel repair occurs. Side effects of systemic ACA occur in up to 50% of patients and include nausea, vomiting, systemic hypotension, tinnitus (less commonly), numbness, skin rash, myalgia and hematuria. It is contraindicated in patients with coagulopathies, renal disease, and in patients who are pregnant, and should be used cautiously in patients with hepatic, cardiovascular or cerebrovascular diseases. ACA in a topical gel form has comparable effectiveness as the oral form, but with few side effects. Karkhaneh et al all found topical ACA did not affect the rate of rebleeding, but was associated with a longer time for clot absorption in the anterior chamber. Tranexamic acid, another antifibrinolytic agent, has a similar mechanism of action as ACA. Tranexamic acid also has similar side effects, but less gastric side effects. Although data suggest that antifibrinolytic agents reduce the risk of secondary hemorrhage, they do not have a significant effect on visual acuity, and their use is controversial. Therefore, several authors recommend reserving antifibrinolytics only for those patients at higher risk for secondary hemorrhage based on individual patient characteristics, including race and the presence of sickle cell hemoglobinopathy.

Complications

Elevated IOP

Elevated IOP is the most serious complication of traumatic hyphema as it may result in optic atrophy and corneal blood staining. Elevated IOP occurs in approximately one-third of all hyphema patients. In general, the larger the hyphema, the higher the risk of developing increased IOP. In a study of 162 patients with microhyphema, IOP was elevated (above 22 mmHg) in only 8.6%, and in those patients with elevated IOP there was a significantly higher incidence of angle recession. Patients with sickle cell hemoglobinopathies require aggressive IOP lowering measures, as these patients are at higher risk for central retinal artery occlusion and optic nerve damage, even with only marginal increases in IOP.

Recurrent hemorrhage

Recurrent, or secondary, hemorrhage occurs if the size of the hyphema increases, if fresh blood is seen over the older and darker clot, or if dispersed red blood cells appear over the clot after the initial blood has settled. Lysis and contraction of the fibrin plug in the injured vessels is responsible for the rebleed, which usually occurs two to five days after the initial injury. Recurrent hemorrhage is associated with complications including elevated IOP; corneal blood staining, optic atrophy and peripheral anterior synechia. While these complications can result in permanent loss of vision, some studies have shown that secondary hemorrhage is associated with a worsening of visual prognosis, while others have not. The highest prevalence of hyphema rebleed in the United States is in the nonwhite population. Patients with an initial higher grade hyphema (grade 3-4)
are also at higher risk. In a study of 40 children with traumatic hyphema, the overall rate of secondary hemorrhage was 10.3% and was significantly higher in African American patients. Rahmani et al found a greater chance of rebleeding in patients with poor vision or elevated IOP at the time of presentation. Aspirin has been shown to increase the risk of rebleeding. Therefore, it, along with other non-steroidal anti-inflammatory drugs, should be avoided for two weeks after traumatic hyphema due to their antithrombotic effect. Acetaminophen or codeine, however, are safe to use as an analgesic if not otherwise contraindicated for other medical reasons.

The incidence of rebleed in patients with microhyphema is lower. Recchia et al found that only three out of 162 patients (1.9%) with microhyphema developed rebleed, and all three occurred on post trauma day three. ε-aminocaproic acid is not used in patients with traumatic microhyphema because the risk of significant rebleeding is much lower in these patients, and the benefits of ε-aminocaproic acid do not outweigh the risks of the medication.

**Corneal blood staining**

Corneal blood staining occurs when hemoglobin and hemosiderin enter the corneal stroma. Corneal blood staining is more common with larger hyphemas, rebleeding, prolonged clot duration, sustained elevated IOP and corneal endothelial cell dysfunction. Slit lamp examination reveals straw yellow discoloration of the deep stroma greater centrally than peripherally in the early stages. Corneal blood staining remains for years and does not respond to medical treatment. In young children, staining may cause amblyopia. For these reasons, surgical intervention is required when the presence of microscopic blood staining is noted.

**Ghost cell glaucoma**

Ghost cells are degenerated erythrocytes that form within the vitreous cavity over the course of several weeks after a vitreous hemorrhage. If the anterior hyaloid face is ruptured by trauma, surgery or spontaneously, the ghost cells can freely move into the anterior chamber. These rigid, khaki-colored ghost cells are less pliable than fresh erythrocytes and cause approximately three times the obstruction to trabecular outflow than an equal number of fresh erythrocytes. Ghost cell glaucoma can also occur with large traumatic hyphemas that extend into the vitreous cavity. The ghost cells that form in the vitreous cavity migrate back into the anterior chamber weeks to months after the initial injury, creating another spike in IOP. Slit lamp examination will reveal khaki-colored cells freely floating in the anterior chamber, or a tan stripe in a background of red cells, creating the candy-stripe sign. Medical therapy is frequently sufficient to manage IOP until the supply of ghost cells is exhausted, but surgery may be necessary.

**Traumatic glaucoma**

The most common site of damage in blunt ocular trauma is the anterior segment. The most common of these injuries is angle recession, which is a tear between the longitudinal and circular muscles of the ciliary body. More than 50% of patients with traumatic hyphema will have some degree of angle recession. Greater amounts of angle recession, typically 180 degrees or more, are associated with higher risk for developing glaucoma. Angle recession itself is not the cause of chronically elevated IOP, but rather it provides evidence of previous trauma and represents permanent damage to the trabecular meshwork. Another mechanism of chronically elevated IOP is the extension of an endothelial layer from the cornea over the structures in the anterior chamber angle. The presence of heavy trabecular pigmentation, elevated baseline IOP, hyphema, angle recession and lens displacement with a cataract are significant predictors of chronic traumatic glaucoma. In patients with angle recession, 5-20% will develop glaucoma in the injured eye, and of those who do develop glaucoma, up to 50% will develop glaucoma in the fellow uninjured eye, which suggests these patients may have a predisposition to this condition.

**Discussion**

A patient with traumatic hyphema may present with variable symptoms of blurred vision, pain and photophobia. Vision typically follows the severity of the hyphema itself. Vision may be entirely normal to no light perception with hyphema ranging from microhyphema to “eight ball” hyphema. The degree of pain and photophobia is related to the severity of associated uveitis and the level of IOP.

A ruptured globe must be ruled out at the initial examination. The cornea should be carefully evaluated for positive Seidel sign to rule out full-thickness laceration. Other signs of ruptured globe include a deep or shallow anterior chamber compared to the fellow eye, peaked or irregular pupil, iris transillumination defects, and low IOP. Additionally, orbital fracture must be ruled out. Signs and symptoms of orbital fracture include pain and restriction on eye movement, local tenderness, binocular diplopia, crepitus after nose blowing or on palpation, and hypoesthesia along the ipsilateral upper lip, cheek and forehead. Orbital imaging (X-ray or CT scan) should be ordered if an orbital fracture is suspected.

Patients must be seen daily during the first five to seven days post-hyphema. This exam frequency is critical because recurrent hemorrhage almost always occurs before the seventh day. Visual acuity, slit lamp exam with careful monitoring of corneal clarity and size of hyphema, tonometry, and assessment of patient compliance with medication and self-care are required. The vertical height of the hyphema should be measured at the initial examination because an increase in size at a subsequent visit is an indication that a recurrent hemorrhage has occurred. A careful evaluation of the anterior chamber is needed to identify the presence of, and to distinguish between, white blood cells, red blood cells and ghost cells. The clarity of the cornea must be closely monitored to assess for corneal blood staining.

Dilated fundus exam, without scleral indentation, is necessary to assess the posterior segment for complications secondary to the initial trauma. Posterior segment injuries are significant predictors of poor visual outcome; however, the detection of these findings can be challenging during initial examination due to poor visualization through the hyphema and/or vitreous hemorrhage. Ultrasound can be helpful in evaluating...
the posterior segment if it cannot be examined initially. Care must be taken to avoid too much pressure on the globe due to the risk for rebleed. Virtually no pressure needs to be applied over closed lids to obtain useful echograms. On B-scan, a fresh, mild hemorrhage shows dots and short lines in the vitreous cavity. Denser hemorrhages show higher reflectivity and a greater number of opacities. The vitreous hemorrhage may also organize into layers in the lower periphery of the globe, forming highly reflective pseudomembranes. B-scan of a retinal tear will show a small, focal, echo-dense membrane of the posterior surface attaching to the posterior hyaloid. A retinal detachment appears as a bright, continuous, somewhat folded membrane that may insert at the ora serrata, optic disc or elsewhere in the fundus. Ultrasound may also be used to assess the anterior segment in cases of corneal opacification or large hyphema.24 High frequency ultrasound biomicroscopy provides high resolution images and can be useful in detecting areas of angle recession, cyclodialysis or weak zonules.6

Gonioscopy is performed three to six weeks after the injury to assess for angle recession. Gonioscopy should not be performed before this time because the procedure may increase the risk of rebleed.8 Signs of angle recession include a posteriorly recessed iris revealing an irregular widening of the ciliary body band, an uneven iris insertion, and an area of torn or absent iris processes.19 In addition, the presence of pigment balls or clumps on the trabecular meshwork may be seen, which is highly associated with previous traumatic hyphema.6

Traumatic hyphema is usually a self-limiting condition that typically has good visual prognosis. Approximately 75% of patients with traumatic hyphema have a final visual acuity of 20/50 or better.2 Vision gradually improves as blood settles to the bottom of the anterior chamber and is eventually resorbed. Most uncomplicated hyphemas will resolve within approximately one week.1 Certain characteristics of hyphemas such as height and blood color relate to the prognosis. Hyphemas measuring 3-4 mm or less typically have a favorable course, as do hyphemas with light red color. Hyphemas that are 5 mm or more and dark red or black in color have a more guarded prognosis. The dark color of the blood indicates poor circulation of the aqueous humor and lack of oxygen supply in the anterior chamber.17

The major determining factor in the final visual outcome in traumatic hyphema is usually an associated ocular injury, not the hyphema itself.1 Factors associated with a poor final visual outcome include the presence of posterior segment injuries such as macular edema, retinal hemorrhage, epiretinal membrane or choroidal rupture. Anterior segment findings including corneal blood staining, traumatic mydriasis, iridodialysis, cataract and lens subluxation are also significant predictive factors of a poor final visual outcome. While the presence of traumatic mydriasis and iridodialysis may not significantly compromise the visual function, they likely reflect the severity of the initial trauma.19 In a study of 425 individuals with traumatic hyphema, a higher grade of hyphema on presentation and the presence of retinal damage were each associated with a poorer final visual outcome.20

Conclusion

The most common mode of clinical presentation in blunt ocular trauma is hyphema.6 Hyphema does not typically cause permanent loss of vision; however, its presence signifies considerable insult to the globe and therefore requires careful follow-up and management. Patient education is essential to minimize complications in the first several days after the injury as well as for the long-term ocular health of the patient. The risk of glaucoma remains years after injury and should always be considered in cases of unilateral glaucoma at any time in life.7 The patient in our case description had a good visual outcome. However, due to the area of angle recession and iridodialysis, he is now at higher risk for developing glaucoma and will therefore continue to require careful monitoring throughout his life.

Disclosures

Dr. Dorothy Hitchmoch is a consultant for Annidis Health Systems Corporation and is on the speakers bureau for Zeavision.

References

14. Verma N. Trabeculectomy and manual clot evacuation in traumatic hyphema with corneal blood

Appendix A:
Summary of Examination Findings for the Left Eye

<table>
<thead>
<tr>
<th>Date</th>
<th>Visual Acuity</th>
<th>Hyphema Vertical Height (mm)</th>
<th>Anterior Chamber</th>
<th>IOP mmHg</th>
<th>Diagnostic Testing</th>
<th>Medications</th>
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<td>8/21/12</td>
<td>20/150</td>
<td>2.2</td>
<td>2+RBC</td>
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<td>B-scan</td>
<td>Pred QID, Atropine BID</td>
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<td>20/50-</td>
<td>2.0</td>
<td>2+RBC</td>
<td>25</td>
<td>Orbit X-ray</td>
<td>Pred QID, Atropine BID, Cosopt BID, D/C aspirin</td>
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<td>8/24/12</td>
<td>20/50+</td>
<td>2.0</td>
<td>3++RBC</td>
<td>25</td>
<td>Anterior segment photos</td>
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<td>20/80+</td>
<td>&lt;1.0</td>
<td>2-3+RBC, Flare</td>
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<td>Pred 6x/day, Atropine BID, Cosopt TID, Acetazolamide</td>
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<td>20/100</td>
<td>Trace</td>
<td>3+RBC, Flare</td>
<td>36</td>
<td>Pachymetry; 607 um OD, 662 um OS</td>
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<td>3+RBC, Flare</td>
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<td>8/30/12</td>
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<td>20/50</td>
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<td>2-RBC, Flare</td>
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<td>20/40-</td>
<td>Resolved</td>
<td>Trace RBC</td>
<td>17</td>
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<td>Pred QID, Atropine QD, Acetazolamide, D/C Atropine</td>
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<td>Pred TID, Cosopt BID, D/C Atropine</td>
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Pred = prednisolone acetate 1% ophthalmic solution; Atropine = atropine sulfate 1% ophthalmic solution; Timolol = timolol ophthalmic solution 0.5%; Cosopt = dorzolamide 2%/timolol 0.5%; Acetazolamide = acetazolamide 500 mg PO BID.