Management of the Patient with Hyphema

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Abstract

Bleeding into the anterior chamber (hyphema) occurs most commonly after blunt or penetrating injuries, but also in a number of other settings. Complications include elevation of intraocular pressure and its consequences as well as corneal blood staining, and vision loss from associated injuries. The risk of complications is believed to be higher in patients with sickle cell disease. Although there is no consensus on the use of various medications in patients with hyphema, most ophthalmologists agree that cycloplegics, topical steroids, and anti-glaucoma medications are important and prevent complications such as elevated intraocular pressure, posterior synechiae, and possibility of rebleeds. Management of pediatric patients as out or inpatients and the restriction of their level of activity continue to be debated, as is the use of systemic anti-fibrinolytics and oral steroids. It is critical to identify associated ocular, orbital, or other accompanying injuries. The surgical evacuation of blood from the anterior chamber is considered and performed in cases in which the hyphema does not resolve and intraocular pressure is significantly elevated.

Keywords

Hyphema • Trauma • Glaucoma • Injury • Sickle cell • Therapy

Introduction

A hyphema, or blood in the anterior chamber, commonly occurs following blunt or penetrating ocular injuries [1]. While trauma is the most common cause of hyphema, other conditions associated with spontaneous bleeding into the anterior chamber have been identified such as juvenile xanthogranuloma, uveitis [2], leukemia [3], retinoblastoma [4], and Swan syndrome, a condition defined as the development of hyphema months to years after cataract surgery due to an abnormal proliferation of blood vessels at the cataract wound site [5]. Children comprise about 70–75 % of patients who present with traumatic hyphemas [6]. Complications of traumatic hyphemas can lead to permanent vision loss, and include elevated intraocular pressure (IOP) leading to optic nerve damage, secondary hemorrhage, corneal blood staining, and, in children, deprivation amblyopia [1]. It is important to evaluate and to recognize penetrating injuries to the globe in patients with traumatic hyphema so that the appropriate surgical interventions are performed. For details regarding the management of penetrating globe injuries please refer to Chaps. 17 and 30, as the remainder of this chapter addresses the diagnosis and management of traumatic hyphema from non-penetrating ocular trauma.

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Pathophysiology

The impact of a blunt object hitting the eyeball results in distortion of the globe, a decreased antero-posterior diameter with stretching and posterior displacement of the lens-iris diaphragm and scleral expansion in the equatorial zone. This distortion leads to disruption of the arterioles of the ciliary body and iris vessels that results in bleeding into the anterior chamber. The formation of a fibrin clot and the rise in intraocular pressure (IOP) tamponade the bleeding vessel, leading to a cessation of bleeding [1, 6].

One of the most common acute complications of traumatic hyphema is elevation of IOP. The fibrin clot that forms at the site of a vascular tear breaks down into its degradation products, which are cleared from the eye through the trabecular meshwork. Obstruction of aqueous fluid outflow at the site of the trabecular meshwork with the fibrin degradation products and red blood cell components often leads to a rise in intraocular pressure if not counterbalanced by decreased aqueous production from inflammation. If the dissolution of the fibrin clot occurs prior to the healing of the injured blood vessel a secondary hemorrhage may result.

Clinical Features

The timing and mechanism of injury should be recorded. Lacerating and penetrating injuries must be ruled out, and if head trauma or orbital wall fractures are suspected, appropriate imaging studies such as computed tomography (CT) scans or ultrasonography should be obtained. Ocular trauma in the course of children's play activities is the most common cause of hyphema [7], but other situations include sports injuries, airbag, finger or fist to the eye, paintball and with increasing incidence, air-soft pellet injuries [8]. In children, non-accidental trauma should also be considered as a cause of hyphema, and social services alerted in case it is suspected.

Clinical symptoms and signs associated with traumatic hyphema include: decreased vision, photophobia, pain, nausea and vomiting, elevated IOP and injury to adjacent structures including the eyelids, iris, and cornea. Iridodialysis is common. Table 15.1 provides a scheme for grading macrohyphemas [1], microhyphema may be graded using the Standardization of Uveitis criteria scale for grading cells [9].

Table 15.1 Grade of hyphema according to amount of blood in anterior chamber

Grade	Percentage of anterior chamber involvement
Grade 4	Fills the whole anterior chamber. "Eight ball hyphema"
Grade 3	1/2 to nearly entire anterior chamber
Grade 2	Between 1/3 and 1/2 of anterior chamber
Grade 1	Less than 1/3 of anterior chamber
Microhyphema	Circulating red blood cells

All patients of African descent should be screened for sickle cell disease using a sickle prep, followed by a hemoglobin electrophoresis [1, 10]. Patients with sickle cell anemia can have a more complicated course, with increased risk for elevated intraocular pressure, central retinal artery occlusion, optic nerve compromise from mildly elevated IOP, visual impairment, and rebleed [6, 9, 11]. This greater propensity for complications is due to the particular environment of the anterior chamber and the sickled red blood cells. The red blood cells have a greater propensity to sickle in aqueous humor than in venous blood [11]. Sickled red blood cells go on to obstruct the trabecular meshwork, which increases intraocular pressure and also results in an acidic, hypoxic environment that perpetuates additional sickling [11].

Medical Management

Treating pediatric patients in an inpatient versus outpatient setting continues to be a source of debate. While inpatient management of children with traumatic hyphema allows a restriction of the child's activities and a more reliable administration of eye drops, studies have not shown a statistically significant benefit of inpatient vs. outpatient treatment with regard to the rate of secondary hemorrhage (rebleed) or visual acuity outcomes [12].

Regardless of the setting, initial management requires a protective shield to be placed over the eye, restricted activities to prevent secondary hemorrhage, and elevation of the head of the bed to allow settling of the hyphema to the inferior part of the anterior chamber, facilitating a view of the retina and optic nerve. In order to prevent pain associated with ciliary spasm and to reduce posterior synechiae formation, a cycloplegic agent, such as atropine sulfate 1 %, homatropine 2 % or 5 %, or even cyclopentolate 1 % or 2 % is given. Topical corticosteroids are administered to reduce inflammation, and may play a role in the reduction of secondary hemorrhage. Oral corticosteroids have also been used with the thought that they also help to reduce inflammation, stabilize the blood ocular barrier, and slow clot dissolution [6]. Another medication that helps to prevent the dissolution of the clot is aminocaproic acid. Aminocaproic acid is an anti-fibrinolytic that is administered intravenously or topically. It has been shown to reduce the rate of rebleed but can significantly increase the time it takes for the clot to dissolve [13]. Because of the increased time to clot dissolution, this medication is not recommended in hyphemas that fill >50 % of the anterior chamber as a large clot will obstruct the vision [6]. The use of systemic aminocaproic acid has not been shown to improve visual outcomes. It is also possible that the cessation of this medication can result in a higher risk of

Table 15.2 Commonly used medication in the treatment of traumatic hyphema

Medication	Dosage	Reason
Atropine sulfate 1% or other cycloplegics	Daily or as necessary for the individual medication	Cycloplegic to help relieve ciliary spasm and pain
Prednisolone acetate 1 %	Four times daily, but can vary based on grade of hyphema[a]	Reduce inflammation; possibly reduce secondary hemorrhage
Treatment of elevated IOP	Medication and dosage	Cautions/side effects
Beta-blocker	Twice daily	Caution in asthma or heart disease
Carbonic anhydrase inhibitor	Dorzolamide or Brinzolamide 3 times daily topically or Acetazolamide or Methazolamide oral or IV	Can precipitate sickling
Combination medications	Dorzolamide/Timolol (Cosopt) twice daily	See above
Hyperosmotic agents	Latanoprost qHS	Can lead to increased inflammation

[a]If more extensive hyphema or corneal edema from the blunt trauma, may require more frequent dosing. In the setting of concomitant large corneal epithelial defect, judicious utilization as may delay corneal re-epithelialization

secondary hemorrhage [14]. At the time of writing of this chapter we do not believe that anti-fibrinolytics are widely and routinely administered.

In the pediatric population, treatment of IOP requires special consideration. Elevated IOP is often initially treated with an aqueous suppressant, such as a topical β-blocker or carbonic anhydrase inhibitor (CAI), or a combination thereof [1]. It should be mentioned that in sickle cell trait or disease, CAIs, topical or oral, should not be used as they cause a metabolic acidosis, which may worsen sickling. In addition, in very young children, alpha-adrenergic agonists are avoided as they can cause respiratory depression especially in those patients less than 12 years of age. In cases where IOP remains unacceptably elevated, oral or intravenous CAIs or hyperosmotic agents may be required. The following table (Table 15.2) can help to guide initial medical management of patients with traumatic hyphema.

Surgical Management

Evacuation of blood clots or washout of hyphema from the anterior chamber is rarely necessary, but there are specific indications for these procedures. Reasons to consider surgery include corneal blood staining and uncontrolled IOP. Corneal blood staining is most common in patients with rebleeds and results from the combination of elevated IOP, endothelial dysfunction, and anterior chamber hemorrhage. Red blood cells release hemoglobin that is absorbed by the corneal stroma and by keratocytes leading to keratocyte death. Corneal blood staining may be difficult to detect because of apposition of the hemorrhage to the corneal endothelium. Slit-lamp examination is significant for yellow granular changes or haze of the posterior corneal stroma. Blood staining leads to a reduction in corneal transparency that may be permanent. Surgical evacuation is recommended at the earliest detection of blood staining. Empirical guidelines have been suggested for performing anterior chamber washout of hyphemas associated with elevated intraocular pressure and include: (1) an intraocular pressure of 25 mmHG or more for 5 days with a total hyphema; or (2) an intraocular pressure of 60 mmHG or more for 2 days. The surgical guidelines in patients with sickle cell disease differ, as the optic nerves of patients are more susceptible to damage from modest elevations of pressure. The guidelines are: (1) an intraocular pressure above 25 mmHg for greater than 24 h; or (2) repeated spiking of intraocular pressure to above 30 mmHg for 2–4 days [1].

Complications

One of the more common acute complications of traumatic hyphema is an elevation of IOP. However, elevated IOP and glaucoma can also manifest as a late complication due to angle recession, peripheral anterior synechiae formation, or posterior synechiae with iris bombe. Therefore, gonioscopy and a description of angle findings are recommended as soon as the hyphema resolves and 3–6 months after initial injury [1].

In addition to an elevation of IOP, traumatic hyphema can cause corneal blood staining. In the pediatric population this is especially concerning, as it can result in deprivation amblyopia. Corneal blood staining can be minimized by judicious daily follow-up and evacuation of the hemorrhage if IOP consistently elevated as above or at the earliest detection of corneal blood staining. Another serious complication is secondary hemorrhage or rebleed. In such patients, visual prognosis appears to be worse than those without rebleed [6]. The most likely time for secondary hemorrhage are the first 4–7 days after trauma. A higher rate of secondary hemorrhage is more likely in the following populations: African Americans, patients with sickle cell disease or trait, and younger patients, possibly due to the difficulty in maintaining limited activity [6]. Furthermore it has also been reported that high IOP and worse vision at time of initial exam may be associated with an increased probability of secondary hemorrhage [15].

Outcomes and Conclusions

Hyphemas resolve without consequence in the majority of patients who do not have other associated ocular injuries and who do not experience any rebleeds [13]. Visual outcomes in the absence of commotio retinae are good. Patients with sickle cell trait/disease require judicious observation, avoidance of medications that may contribute to sickling, and evacuation of hemorrhage if IOP remains elevated. The use of corticosteroids, cycloplegics, and interventions such as binocular patching, bed rest, or head elevation are done at the discretion of the treating physician and are possibly of benefit to the individual patient [16].

Cases

Clinical Case 1

A 10-year-old Caucasian male presents to the emergency department with left eye pain 1 day after an altercation at school which resulted in a fist to the eye. He reports that vision was blurry when he awoke this morning. Light made his pain worse.

Examination was significant for a visual acuity of 20/20 in the right eye and 20/40 in the left eye. Extraocular movements were full. IOP was OD = 12 mmHg, and OS = 34 mmHg. On slit-lamp examination there was a grade I hyphema in the left eye and dispersed microhyphema in the anterior chamber (Fig. 15.1). The rest of the eye examination was within normal limits.

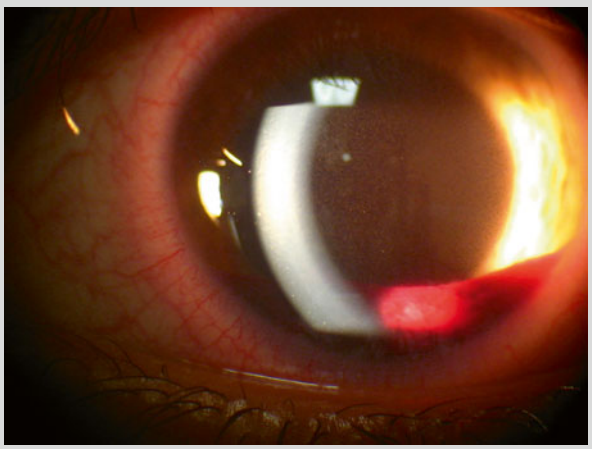

Fig. 15.1 Grade I hyphema of the left eye (occupied <1/3 of the anterior chamber) and dispersed (4+ RBCs) present in the anterior chamber. Note the absence of corneal blood staining. Image courtesy of Daniele Saltarelli, OD, Cincinnati, OH

Management: The patient was started on atropine sulfate 1 % drops twice daily and prednisolone acetate 1 % drops four times daily, along with timolol maleate/dorzolamide combination drops twice daily for the elevated IOP. He was instructed to limit activities, sit whenever possible, and to keep the head of the bed elevated. The risks and consequences of secondary hemorrhage were explained to the parents. The patient was followed daily until resolution of the hyphema. The drops were discontinued over the course of 3 weeks and gonioscopy 3 months post trauma showed no angle recession or peripheral anterior synechiae.

Clinical Case 2

An 8-year-old African American male with sickle cell trait presented to the emergency room 2 days after his brother hit his right eye with an air soft bullet. His mother states that he has had two episodes of emesis upon waking that morning, along with complaints of headache. Examination showed a visual acuity of hand motions in the right eye and 20/20 in the left eye. The pupil could not be visualized OD and was normal OS. The IOP was 62 mmHg in the right eye and 15 mmHg in the left eye. Anterior segment examination was significant for diffuse corneal edema and a Grade 4 hyphema. The left eye was normal. There was no view of the iris or the fundus on the right. A B-scan was obtained OD and showed no evidence of retinal detachment or vitreous hemorrhage.

Management: This patient with sickle cell trait presents with a total, hyphema, significant symptoms, and elevated and severely elevated intraocular pressure. Because of the severely elevated intraocular pressure and contraindications to both systemic agents to rapidly reduce pressure, the patient was immediately taken to the operating room for surgical removal of hyphema. At the conclusion of the case, atropine sulfate 1 %, prednisolone acetate 1 %, and antibiotic drops were placed in the eye; and a patch and shield were applied. The head of the bed was elevated and the patient was placed on a limited activity regimen. The following day visual acuity in the right eye had improved to 20/50 and the anterior chamber was deep with a 3+ microhyphema. Intraocular pressure was 18 mmHg. The patient was started on topical timolol,

(continued)

prednisolone acetate eye drops and cycloplegics, and followed daily. Six days after the initial event the patient returned with an acute decrease in visual acuity to 20/100 and intraocular pressure elevation to 26 mmHg. A rebleed was diagnosed and the patient was maintained on the eye drops with strict adherence to head of bed elevation and limited activity. Over the next couple of days, vision continued to improve and the intraocular pressure continued to decrease. At 3 months post trauma, visual acuity was 20/20, intraocular pressure was 18 in both eyes and the anterior chamber was quiet. Gonioscopy was performed and there was no evidence of angle recession.

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