Pediatric Traumatic Hyphema

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Author's Bio

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Introduction

Globally 1.6 million people suffer from blindness due to eye injuries. ¹ Furthermore, pediatric patients have the highest rate of unilateral non-congenital blindness due to ocular trauma. ^{2,3} Prevention through patient education and protection is certainly of growing importance, but managing the complications associated with ocular trauma, once it occurs, is imperative. Often, trauma to the eye can result in multiple complications such as lid lacerations, corneal abrasions, lens subluxation, commotio retinae, and retinal detachment, to name a few. ⁴ A common complication of ocular trauma is a hyphema where erythrocytes begin to fill the anterior chamber due to the pressure applied to the anterior surface of the eye from the trauma. This leads to distension of the tissues, increased intra ocular pressures, damage to the cilliary body and the major arterial circle.² Additionally, hyphemas can occur from non-traumatic reasons such as rubeosis irides, xanthogranuloma, iris melanoma, myotonic dystrophy, blood dyscrasias or substances that alter platelet or thrombin function.⁵ Treatments remain debated, especially in children, regarding the necessity of hospitalization versus outpatient management and the use of medications, with the ultimate goal being to minimize potential vision threatening sequelae.⁵ These may include re-bleeding, corneal blood staining, secondary glaucoma, and ischemic optic neuropathy.^{2,6}

Case

A nine-year -old Hispanic female, presented to the clinic for a red eye evaluation with her parents after having been hit on the right upper lid with a small round projectile shot from a toy gun while playing with her younger brother at home a half hour prior. The parents described the projectile as a waterbased gelatinous pellet that dissolved upon impact. The patient reported her eye was closed when she was hit at distance of a few feet. She reported soreness of the lid at the area of impact with no changes in vision, flashes of light or floaters and only mild photophobia. Her ocular history was unremarkable with no reported problems with her vision. She had no history of spectacle wear and no previous eye exams. Her medical history was negative for any systemic disease and she was not taking any medication although her mother did administer children's advil (Ibprofen 100mg one capsule PO) to her just before arriving at the clinic. Her family's ocular and medical history was unremarkable. The patient and mother both denied ocular medications and reported no known medication allergies. The patient was fully alert to time and place.

Her uncorrected visual acuities were 20/15 OD, and 20/15 OS. Ocular motility was found to be full and unrestricted OU. Confrontation fields were full to finger counting in all quadrants OD and OS. Pupils were equal in size, round and reactive to light without an afferent pupillary defect. Blood pressure at this visit was measured at 100/60. Intraocular pressures (IOP) were 24 mmHg OD and 17mmHg OS as measured with non-contact tonometry (NCT). Anterior segment evaluation by slit lamp revealed diffuse hyperemia of the right upper lid without ecchymosis, pain to touch or any lacerations. The left eye lid was found to be unremarkable. The lacrimal system in both eyes was normal with open puncta and normal lid-globe apposition OU. The bulbar and palpebral conjunctivas were white and quiet OU with no hemorrhages or foreign bodies OU. Corneal evaluation with white light and cobalt blue, after instillation of Fluress, revealed scattered punctate epithelial erosions (PEE) on the superior cornea with one area of coalesced PEE's superior temporally on the cornea OD. The corneal stroma was free of defects without edema. Seidel's test was negative. The cornea of the left eye was unremarkable. The anterior chamber of the right eye was positive for a grade 1 hyphema that measured approximately 2mm in size with 4+ cells and 1+ flare and no posterior synechia on the lens. Angle estimation with Von Herrick method was greater than 1:1 nasal and temporal OU. The anterior chamber of the left eye was found to be unremarkable. Given the moderately elevated pressure in the affected eye, in office treatment was initiated with 2 drops of apraclonidine 0.5% OD spaced out by 15 minutes between drops, after which IOP measured by NCT remained 24mmHg OD. An additional drop of apraclonidine 0.5% OD and 1 drop of Brimonidine 0.1% OD, again, spaced out by 15 minutes between drops were applied. IOP's were then measured using Goldmann applanation tonometry (GAT) measuring at 20mmHg OD, 17mmHg OS.

The patient was then dilated with 1 drop of proparacaine 0.5% OU, homatropine 5% OD and tropicamide 1% OS. The crystalline lens was clear and centered OU. The vitreous was normal in both eyes with a negative Schaffer's sign OU. The optic nerves in both eyes were normal with distinct margins and well perfused rim tissue. No pallor was noted OU. Cup to disc (C/D) ratios were found to be 0.25 round OD, and 0.30 round OS. Macular anatomy was flat and avascular with a positive foveal reflex OU. Complete evaluation of the retina revealed a two disc diameter area of localized commotio retinae in the temporal periphery or the right eye, without any tears, hemorrhages or detachments. There was no intraocular foreign body OD. The retina of the left eye was found to be intact and unremarkable.

The differentials in this case were few given the very specific history of localized trauma to the eye. Potential differentials included:

- Idiopathic iritis
- Iritis secondary to an infectious agent, genetic predisposition or autoimmune related
- Rubeosis irides
- Hyphema from an associated blood dyscrasia such as sickle cell hemoglobinopathy

- Related to retinoblastoma or melanoma
- Traumatic hyphema

The patient was diagnosed with and acute traumatic hyphema with a secondary iritis OD and commotio retinae OD given the correlation of onset of symptoms directly after the inciting event. Iritis secondary to an infectious agent, genetic predisposition or autoimmune related or hyphema from an associated blood dyscrasia was ruled out given the patient's unremarkable medical record and family history. Both rubeosis irides, retinoblastoma and melanoma were ruled out during clinical exam with no ocular findings indicating their presence.

The patient was treated with predforte 1% every hour OD, homatropine 1% BID OD and Combigan BID OD to control her IOPs. The patient and parents were advised to wear a fox shield, which was applied in office and to continue to maintain at least a 45 degree elevation of her head until her next follow up with strict minimization of any head movements. The patient and parents were also advised to seek immediate care should she note any changes in vision, flashes, floaters or pain.

She was followed daily for the first 3 days after which the hyphema had resolved with only the iritis remaining. The patient continued on the topical steroid and IOP lowering agent in conjunction until complete resolution of the iritis after which the steroid was tapered for 3 weeks and then discontinued along with the Combigan. The patient maintained stable vision throughout and the commotion retinae OD resolved without treatment after the 3rd day. Gonioscopy at her 1 month follow up showed no angle recession with open angles and healthy posterior segment findings OU. Although her IOPs were elevated OD initially they remained controlled after initiation of the Combigan and after its discontinuation at resolution of the iritis.

Discussion

Often with a trauma there will be multiple structures that are affected within the eye. Clinician must comprehensively evaluate the the ocular system in its entirety to rule out associated sequelae that may need to be managed concurrently. For the purposes of this article, traumatic hyphemas will be the majority of the scope of the discussion. Traumatic hyphemas, as the name implies, are caused by blunt force applied to the eye, causing a sudden increase in intraocular pressure followed by displacement of the lens iris diaphragm. ⁷ This ruptures the vascular supply of the iris and cilliary body ^{5,7,8} As the bleeding ceases and begins to clot, due to the tamponade effect of increased intraocular pressure,⁹ plasmin activates the fibrinolytic system that will break down the clot so it can be reabsorbed through the trabecular meshwork. ^{5,7}

During an examination it is important to determine the cause of trauma. Caution should be taken to rule out child abuse in pediatric patients with no reported cause. Patients may present with decreased vision, eye pain or photophobia. One should take care to search for an open globe injury or intraocular foreign body. Intraocular pressure and dilated fundus exam should be performed along with a complete ophthalmic examination avoiding scleral depression or gonioscopy in the acute phase as they may

exacerbate the bleeding. ⁷ Gonioscopy should be performed 1 month out to rule out any trabecular meshwork blockage and angle recession.²

Hyphemas are typically classified using a grading system describing how much of the anterior chamber has been filled with erythrocytes. When the hyphema is less than 1/3 of the anterior chamber it is classified as a grade I, grade II is between 1/3 to 1/2 of the anterior chamber, and a grade IV hyphema, also known as an 8 ball hyphema, is a total hyphema.^{2,6,7,10} Reabsorption rate or clearance time and visual prognosis is correlated to the degree of the hyphema, with more severe ones having a longer clinical course.^{2,5}

When discussing the risks of hyphema there are multiple considerations that must be accounted for. Complications include increased intraocular pressure due to blockage of the trabecular meshwork by erythrocytes or inflammatory cells.⁵ Secondary hemorrhages are of particular concern as they have been associated with permanent vision loss due to secondary glaucoma from angle recession leading to elevated IOPs¹¹, corneal blood staining and optic atrophy.⁸ Re-bleed rates in the literature range largely from 3.5% to 38%. These warrant investigation into any hemoglobinopathies in the patient, especially those of African American decent.^{2,4,6,12} With this in mind, aspirin and other blood thinning agents are contra-indicated throughout the course of treatment due to an increased risk for re-bleed.²

Treatment options have been controversial but should be tailored to each patient depending on the severity at presentation and any associated risk factors for complications such as *8 ball* hyphema and sickle cell disease.^{5,6} Historically, hyphemas were treated with hospitalization in order to ensure minimal head movement, especially in children where the risk of noncompliance is presumably higher.^{7,13} With increasing research and documentation of outpatient treatment success, it has become a more acceptable practice.¹³ Never the less, inpatient treatment is still indicated in patients with large hyphemas, sickle cells disease or a known likelihood of noncompliance.¹³ Antifibrinolytic agents such as aminocaprioic acid and tranexamic acid have been recommended in patients with a higher risk for rebleeding. Studies have shown that they delay clot reabsorption until blood vessels are healed, although systemic side effects including nausea, headaches and vomiting have been documented.^{4-6,8,14}

Topical steroids are utilized to treat any inflammatory uveitic component of the eye that results after trauma.⁷ Studies also suggest topical steroid may reduce the risk of re-bleed by re-establishing the blood ocular barrier although care must be taken with long term use due to the risk of IOP response and cataract formation.^{5,7} A cycloplegic agent is typically used in conjunction to other treatment modalities to decrease inflammation by stabilizing the cilliary body and allowing the vessels to heal and thereby preventing posterior synechiae and reducing photophobia.^{5,7} A protective shield, worn at all times, over the affect eye is also recommended to prevent further trauma to the compromised eye until complete resolution.⁷ If IOPs are elevated treatment with an intraocular pressure lowering agent is indicated. Most commonly this is achieved with a beta adrenergic agonist or alpha adrenergic agonist.⁷

Finally, surgical intervention may be necessary in a small percentage of patients where medical therapy has failed. This includes cases with persistently elevated IOPs at 50mmHg after 5 days or 35mmHg for 7 days or after 24 hours in those with sickle cell disease. ⁷ Methods include manual irrigation and

aspiration of the blood, vitrectomy and/or trabeculectomy with anterior chamber washout and peripheral iridectomy.⁷

Fortunately, the management of this patient was relatively straightforward as she responded well to topical therapies, however she did require daily follow up visits to monitor for any increase in signs and symptoms. The patient did present with an additional ocular sequelae of commotio retinae that resolved on its own, as was expected, without further complication. She was fortunate that the more serious complications of ocular trauma were avoided, such as a penetrating ocular injury, retinal detachment or lens subluxation, which would require surgical intervention.

Conclusion

In the case of pediatric traumatic hyphema, due to the various potential complications, daily follow up is critical in monitoring progress as well as very clear and repeated patient and parent education to ensure compliance with prescribed treatments. As treatment options remain controversial, the clinical course of a hyphema dictates that interventional modification may be necessary to protect the patient's vision and ocular health. Lastly, prevention of ocular trauma with the use of protective eye gear should be emphasized with all children and every patient exposed to high risk situations.

Bibliography

- **1.** Negrel AD, Thylefors B. The global impact of eye injuries. *Ophthalmic epidemiology*. Sep 1998;5(3):143-169.
- 2. Rocha KM, Martins EN, Melo JLAS, Bueno de Moraes NS. Outpatient management of traumatic hyphema in children: Prospective evaluation. *Journal of American Association for Pediatric Ophthalmology and Strabismus.* 8// 2004;8(4):357-361.
- **3.** Agapitos PJ, Noel LP, Clarke WN. Traumatic hyphema in children. *Ophthalmology*. Oct 1987;94(10):1238-1241.
- **4.** Salvin JH. Systematic approach to pediatric ocular trauma. *Current opinion in ophthalmology.* Sep 2007;18(5):366-372.
- **5.** Walton W, Von Hagen S, Grigorian R, Zarbin M. Management of traumatic hyphema. *Survey of ophthalmology*. Jul-Aug 2002;47(4):297-334.
- **6.** Lai Jc FSBYGMF. Traumatic hyphema in children: Risk factors for complications. *Archives of ophthalmology*. 2001;119(1):64-70.
- **7.** Sankar PS, Chen TC, Grosskreutz CL, Pasquale LR. Traumatic hyphema. *International ophthalmology clinics*. Summer 2002;42(3):57-68.
- **8.** Agrawal R, Shah M, Mireskandari K, Yong GK. Controversies in ocular trauma classification and management: review. *International ophthalmology*. Jan 22 2013.
- **9.** Romano PE, Phillips PJ. Traumatic hyphema: a critical review of the scientifically catastrophic history of steroid treatment therefore; and A report of 24 additional cases with no rebleeding after treatment with the Yasuna systemic steroid, no touch PLUS protocol. *Binocular vision & strabismus quarterly.* 2000;15(2):187-196.
- **10.** Kearns P. Traumatic hyphaema: a retrospective study of 314 cases. *British Journal of Ophthalmology.* March 1, 1991 1991;75(3):137-141.

- **11.** Sihota R, Kumar S, Gupta V, et al. Early predictors of traumatic glaucoma after closed globe injury: trabecular pigmentation, widened angle recess, and higher baseline intraocular pressure. *Archives of ophthalmology.* Jul 2008;126(7):921-926.
- **12.** Volpe NJ, Larrison WI, Hersh PS, Kim T, Shingleton BJ. Secondary hemorrhage in traumatic hyphema. *American journal of ophthalmology*. Nov 15 1991;112(5):507-513.
- **13.** Coats DK, Viestenz A, Paysse EA, Plager DA. Outpatient management of traumatic hyphemas in children. *Binocular vision & strabismus quarterly.* 2000;15(2):169-174.
- **14.** Pieramici DJ, Goldberg MF, Melia M, et al. A phase III, multicenter, randomized, placebocontrolled clinical trial of topical aminocaproic acid (Caprogel) in the management of traumatic hyphema. *Ophthalmology*. 11// 2003;110(11):2106-2112.

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- **1.** The clinician must rule out child abuse in cases of pediatric traumatic hyphemas.
 - a. True
 - b. False
- True/ False: Reabsorption rate and visual prognosis is correlated to the degree of the hyphema, with more severe ones having a shorter clinical course.
 - a. True
 - b. False
- **3.** A dilated exam with scleral depression should be done at the first visit to rule out retinal breaks.
 - a. True
 - b. False
- **4.** What medication is contraindicated with hyphema cases:
 - a. Timolol
 - b. Aspirin
 - c. Prednisolone acetate
 - d. Aminocaprioic acid
- **5.** Hospitalization is always required in every hyphema case.
 - a. True
 - b. False

- **6.** Aminocaprioic acid may be indicated in patients that are at a higher risk for re-bleeding.
 - a. True
 - b. False
- **7.** What is a potential secondary complication of a hyphema?
 - a. Corneal blood staining
 - b. Glaucoma
 - c. Optic neuropathy
 - d. All of the above
- **8.** How often should a patient who presents with a hyphema be followed until resolution?
 - a. Once a week
 - b. Only one visit is necessary
 - c. Every day
- **9.** A cycloplegic agent is typically used in a hyphema case to:
 - a. Improve vision
 - b. Stabilize the cilliary body
 - c. Induce posterior synechiae
- **10.** Gonioscopy is performed typically one month after to determine:
 - a. If angle recession has occurred
 - b. If there is blockage of the trabecular meshwork
 - c. Both a and b