

Glaucoma Secondary to Traumatic Hyphema

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CASE PRESENTATION

A 48-year-old black male was referred to the Glaucoma Service at the Medical College of Georgia in Augusta 8 days after he was hit in the left eye by an unexploded firework. The patient initially presented to an outside ER, where a CT scan showed a fracture of the orbital floor without entrapment of the inferior rectus muscle. The globe was intact, and no intraocular foreign body was present. The patient was initially followed by an outside ophthalmologist and the general eye service at the Medical College of Georgia for hyphema and elevated IOP. He did not have any sickle cell disease. His initial medical regimen included Diamox Sequels 500 mg b.i.d. (acetazolamide; Duramed Pharmaceuticals, Inc., Pomona, NY) for an IOP of 24 mm Hg and three topical agents (atropine b.i.d., moxifloxacin q.i.d., and prednisolone acetate 1% q2h). Prior to his referral to the Glaucoma Service, his treating physician had discontinued the oral acetazolamide, reduced the prednisolone to q.i.d., and started the patient on topical brimonidine t.i.d. and timolol/dorzolamide b.i.d. The patient continued using the moxifloxacin and atropine as previously described. He also slept with his head elevated and avoided strenuous activities, including reading and watching television.

By the time the patient presented to the Glaucoma Service, the BCVA in his left eye had decreased from 20/70 (as measured by the outside ophthalmologist) to light perception only, his IOP had increased from the low-to-mid 20s with medication to 40 mm Hg, and the height of the hyphema in the anterior chamber had increased from 1.1 to 3.6 mm. The hyphema was suspicious for recurrent bleeding.

On external examination, the patient's left eyelids were edematous, and he had 3+ injection of his conjunctiva and chemosis for 360°. A slit-lamp examination showed a hazy cornea with visible Descemet's folds and a large layered hyphema with dispersed blood that obscured the lens, vitreous, and fundus. The hyphema also included a large white component associated with the presence of vitreous and/or fibrin in the superior part of the anterior chamber (Figure 1). Ultrasound biomicroscopy of the left eye

revealed no retinal detachment but was positive for mild vitreous hemorrhage. The patient's symptoms included pain and decreased visual acuity, but he denied experiencing photophobia, discharge, flashes, or floaters.

HOW WOULD YOU PROCEED?

1. Would you continue to manage the patient medically? If so, would you follow or change the current regimen?
2. Perform an anterior vitrectomy?
3. Wash out the anterior chamber through a paracentesis?
4. Combine a washout of the paracentesis/anterior chamber with the implantation of a drainage tube or a trabeculectomy?

SURGICAL COURSE

The patient's examination suggested a differential diagnosis of hyphema-related glaucoma, ghost-cell glaucoma, possible angle recession, and phacomorphic glaucoma. The last two options were unlikely, given the normal depth of the patient's anterior chamber and the tendency for angle recession to emerge as a late sequela to blunt trauma. We could not rule out the possibility of traumatic cataract, a subluxated lens, or iridodialysis, because we could not visu-



Figure 1. Eight days after sustaining blunt trauma to his left eye, the patient presented to the glaucoma service with a hyphema that filled approximately 50% of the anterior chamber.

alize the iris and lens well enough through the hyphema.

We took the patient to the OR 9 days after he sustained ocular trauma to evacuate blood and possibly vitreous from the anterior chamber of his left eye. As we used balanced salt solution to wash loose red blood cells from the anterior chamber through a corneal paracentesis, we discovered the hyphema was partially composed of a trans-pupillary membrane and an organized fibrin clot. At this time, we had difficulty visualizing the crystalline lens and noted a dull red reflex.

We used an anterior vitrector to remove the fibrin clot. During the procedure, we intermittently stripped portions of the clot away from the iris with Utrata forceps to provide an edge on which to engage the vitrector.

The patient also had posterior synechiae over several clock hours. We did not attempt to lyse these synechiae, however, because we did not want to disturb the lens and induce further injury. We did not observe any complications such as iridal bleeding during the procedure.

At the end of the case, the patient received a subconjunctival injection of an antibiotic and methylprednisolone sodium as well as a 20-mg sub-Tenon's injection of triamcinolone. We sent the patient home with prescriptions for Diamox Sequels 500 mg and a tapering dose of oral prednisone. We postulate that the organization of the hyphema was the result of a severe blunt trauma that had not received adequate anti-inflammatory treatment.

OUTCOME

One day postoperatively, the patient's visual acuity was hand motions, and his IOP measured 18 mm Hg OS. At this time, the patient continued to use his oral medications and was restarted on prednisolone acetate 1%, moxifloxacin, and cyclopentolate hydrochloride 2%. The last three medications were tapered over the next 2 months. Over the next week, the patient regained some vision and showed signs of resolving inflammation (ie, reduced redness and fewer cells in the anterior chamber).

Nine days postoperatively and 4 days after the patient finished the course of oral prednisone, his IOP increased to 29 mm Hg OS. We continued treatment with Diamox Sequels 500 mg b.i.d. and restarted the patient on oral prednisone 20 mg b.i.d. Over the next 2 weeks, we tapered the patient's prednisone to 30 mg per day, but the patient stopped the medication between visits and did not complete the prescribed taper. Nevertheless, over the ensuing month, his conjunctival injection improved, the residual fibrin in his anterior chamber dissolved, and his IOP remained below 20 mm Hg.

On follow-up, we saw no iridodialysis, but we observed a nuclear cataract with pigment on the anterior capsule. Approximately 6 weeks after the evacuation of the hyphema,

a dilated fundus examination of the patient's left eye revealed no gross abnormalities and a cup-to-disc ratio of 0.4. The patient's BCVA had stabilized at 20/80, and his Diamox was changed to a topical glaucoma medication.

"The management of elevated IOP secondary to traumatic hyphema remains controversial."

The patient eventually underwent phacoemulsification and the implantation of a posterior chamber IOL in his left eye. Although the procedure was complicated by the presence of zonular dehiscence and postoperative cystoid macular edema, the vision in the patient's left eye improved to 20/50, and his IOP remained controlled in the normal range on a fixed combination of timolol/dorzolamide.

DISCUSSION

Elevated IOP After Ocular Trauma

Blunt trauma is the third most common cause of secondary glaucoma in the United States.¹ A 6-month cohort study (N = 6,021) by the United States Eye Injury Registry found that 3.39% of patients who sustained ocular injuries developed secondary glaucoma. Patients who developed hyphema (odds ratio = 2.23) or presented with a visual acuity of 20/200 (odds ratio = 1.92) had the highest risk of developing elevated IOPs after blunt ocular trauma.²

Crouch and Williams reported that half of all patients who have hyphema exhibit increased IOPs.³ Another study showed that more patients who experience recurrent bleeding after the initial trauma develop elevated IOPs than those who do not (approximately 52% vs 5%).⁴ This sequence of events appeared to be the mechanism behind our patient's high IOP.

The causes of acutely increased IOP after traumatic hyphema include occlusion of the trabecular meshwork by clotted blood, inflammatory cells, or erythrocytic debris as well as pupillary block secondary to a collar-button-shaped clot involving both the anterior and posterior chambers.⁴

Medical Therapy

The management of elevated IOP secondary to traumatic hyphema remains a controversial subject. Because recurrent bleeding puts patients at risk of increased IOP, clinicians should always ask patients if they have coagulopathies or blood dyscrasias or if they are using anticoagulant drugs. As described in a major review in the *Survey of*

Ophthalmology, IOP elevation with traumatic hyphema is initially treated with topical beta-adrenergic antagonists or alpha-2-adrenergic agonists.⁴ If these drugs are inadequate, guidelines suggest switching patients (excluding those with sickle cell disease) to topical or systemic carbonic anhydrase inhibitors. When the IOP remains elevated, patients can be treated with isosorbide, oral glycerin, or intravenous mannitol in the acute setting. Pilocarpine is contraindicated, because it promotes the deposition of fibrin and may increase the likelihood of iridolenticular adhesions and seclusio pupillae. The use of prostaglandin analogues is controversial, because they could increase inflammation. Many physicians avoid these agents, as we did with our patient.

Surgical Therapy

Physicians should consider using irrigation, vitrectomy, or trabeculectomy to evacuate a clot from the anterior chamber, because about 5% of hyphema patients do not respond to medical therapy.⁵

The Indications for surgery described by Wilson include uncontrolled glaucoma, corneal blood staining, the persistence of a large or total hyphema for 9 days, and active bleeding in the anterior chamber.⁶ We decided to take our patient to the OR, because the hyphema filled 50% of the anterior chamber and he had poorly controlled IOP. Medical treatment had failed, and we thought that increasing his anti-inflammatory regimen at this point would not resolve the hyphema quickly enough to avoid problems associated with elevated IOP.

A commonly performed and relatively easy technique for resolving elevated IOP secondary to hyphema involves washing debris from the anterior chamber with a manual I/A system or an irrigation cannula. Because this procedure is usually performed through one or two small corneal incisions, it spares the conjunctiva for possible future filtering procedures. To lower the IOP, this technique does not require removing all of the blood from the anterior chamber.⁷

Belcher et al successfully used simple irrigation of the anterior chamber to lower the IOPs of 13 patients who had traumatic hyphema.⁸ The investigators' indications for surgery included a persistently high IOP without clearing of the hyphema, ghost-cell glaucoma, or evidence of corneal blood staining. Postoperatively, the patients' average IOP dropped dramatically from 47.6 to 15.5 mm Hg. The investigators observed a significant improvement in most patients and did not report any complications.⁸ Unfortunately, this approach alone could not help our patient.

Although some investigators have suggested that a combined trabeculectomy, washout of the anterior chamber,

and peripheral iridectomy should be performed only after the failure of prior medical and surgical therapy,⁷ Graul et al found that this procedure significantly decreased the mean IOP in 10 of 11 consecutive patients whose hyphemas were refractive to medical therapy. The investigators felt that, relative to other surgical procedures, trabeculectomy can keep the IOP low while the remaining blood clears from the anterior chamber.⁹ Because our patient did not have preexisting glaucoma and his IOP was elevated for only a short time due to trauma, we decided to evacuate the hyphema from his anterior chamber without also performing a trabeculectomy. We estimated that the optic nerve was likely still healthy and that its survival did not depend on significantly lowering the IOP.

"We decided to take our patient to the OR, because the hyphema filled 50% of [his left eye's] anterior chamber."

Diddie described how he successfully used vitrectomy instrumentation to remove hyphemas from the anterior chambers of 12 patients during single procedures.¹⁰ Postoperatively, 11 of the 12 patients maintained IOPs of less than 20 mm Hg and experienced improved visual acuity. The investigator could not attribute a permanent loss of vision to the operative technique.¹⁰

Another study found that six of eight patients treated with vitrectomy instrumentation developed no intraoperative complications, and seven of eight achieved a final BCVA of 20/40 or better.¹¹ The latter group achieved controlled IOPs in the early postoperative period and maintained these pressures without glaucoma medication for 1 to 4 years postoperatively. The two patients with complications developed flat anterior chambers and multiple staphylomas associated with uncontrolled glaucoma, respectively. The latter patient eventually had his eye enucleated.¹¹

Ghost-Cell Glaucoma

It is important to note that multiple etiologies can contribute to elevated IOP after traumatic hyphema. Our patient's condition was complicated by recurrent bleeding and possibly concomitant ghost-cell glaucoma, as suggested by the history of blood in the vitreous and the khaki color of his hyphema.

In 1960, Vannas identified hemosiderin and hemosiderin-rich macrophages as the source of elevated IOP in eyes with a vitreous hemorrhage.¹² Fenton and Zimmerman

subsequently determined that macrophages filled with hemoglobin raised the IOP by obstructing the trabecular meshwork after a vitreous hemorrhage.¹³ Finally, Campbell et al described a transient secondary glaucoma resulting from the obstruction of the trabecular meshwork by degenerated erythrocytes (ie, ghost cells).¹⁴⁻¹⁶

The natural history of ghost-cell glaucoma includes three features: vitreous hemorrhage, a disruption of the hyaloid face, and increased IOP. Patients with ghost-cell glaucoma usually develop decreased visual acuity and pain approximately 2 weeks after their ocular trauma (a longer duration than we observed in our case). The diagnosis can be aided by examining unstained aspirate from the anterior chamber.

Normally, erythrocytes are pliable enough to pass easily through the trabecular meshwork. Red blood cells that persist in the posterior chamber for more than 1 week, however, become more rigid and lose some of their intracellular hemoglobin to the vitreous space. The remainder of the intracellular hemoglobin denatures into Heinz bodies. While the clumps of extracellular hemoglobin stay trapped in the vitreous, the ghost cells can migrate to the anterior chamber, obstruct the trabecular meshwork, and decrease the eye's outflow facility by threefold.¹⁴⁻¹⁶

Because our patient's IOP rose to 40 mm Hg 8 days following trauma and was associated with some vitreous hemorrhage, we determined that his condition could possibly be associated with ghost-cell glaucoma.

Maximal medical therapy is often insufficient to lower the IOP in eyes with ghost-cell glaucoma. Effective therapies include systemic carbonic anhydrase inhibitors, topical hypotensive drugs, and if medical therapy fails, anterior chamber washout with a pars plana vitrectomy.¹⁷ Patients who have especially large vitreous hemorrhages may require a total vitrectomy to remove all of the ghost cells from their eyes.¹⁸ We could have referred our patient for a pars plana vitrectomy if we had still had difficulty controlling his IOP after we had removed the blood clot and fibrin from his anterior chamber.¹⁹

CONCLUSION

Physicians should manage elevated IOP secondary to isolated traumatic hyphema first with medical therapy that includes the aggressive use of anti-inflammatory agents. If this approach is inadequate, surgical therapy should be considered. While formulating treatment plans, physicians should remember that the elevated IOP may be due to multiple concomitant etiologies and adjust their strategy accordingly.

Finally, patients should be observed closely with gonioscopy when the eye is stable, because approximately 20% to 94% of those who develop hyphema experience associated angle-recession glaucoma.⁴ Predictors of

glaucoma after blunt trauma include angle recession of 180° or more, the presence of increased pigment in the angle, an elevated IOP at baseline, hyphema, and dislocation of the lens.¹⁹ □

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