ACUTE RUPTURE OF THE ANTERIOR CAPSULE

Options for managing a patient whose vision decreased after trabeculectomy.

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CASE PRESENTATION

A 45-year-old woman with diabetes underwent an uncomplicated vitrectomy for a nonclearing vitreous hemorrhage associated with diabetic retinopathy in the left eye. When medical therapy failed to control a postoperative IOP elevation, a trabeculectomy was performed. The patient's vision decreased precipitously within a week of glaucoma surgery, and she was referred for an evaluation.

On presentation 2 weeks after the trabeculectomy, the bleb was flat, and the IOP was 38 mm Hg despite treatment with three topical hypotensive medications and atropine. Vision in the left eye was hand motions with projection.

Testing found no relative afferent pupillary defect. The anterior chamber of the left eye was deep. Flocculant cortical material was detected in the anterior chamber and obscured the view of the posterior segment. Light projection was accurate. The patient's UCVA was 20/20 OD, and the IOP in the right eye was normal. Mild nonproliferative diabetic retinopathy was evident.

What do you suspect is the etiology of the problem? How would you proceed?

–Case prepared by Lisa Brothers Arbisser, MD

BEN LAHOOD, MD, MBCHB(DIST), PGDIPOPHTH(DIST), PHD, FRANZCO

The source of the patient's problem appears to be an iatrogenic capsular rupture that is releasing lens material into the anterior chamber. The priority now is to remove the leaking lens material and retain as much of the intact capsule as possible. The IOP remains elevated because swollen lens material is physically blocking aqueous outflow and the released lens fibers have inflamed the trabecular meshwork. The flat bleb is likely caused by physical obstruction at the drainage ostium and aqueous carrying inflammatory mediators, which seal tissue layers of the bleb. The only option for management is lens removal.

The absence of a relative afferent pupillary defect and the presence of good light projection indicate intact optic nerve function. Significant asymmetry in the severity of diabetic eye disease raises concern about other etiologies responsible for hemorrhage, including a retinal tear. Because a routine vitrectomy was performed, however, I assume that the patient has asymmetric diabetic retinopathy. Preoperatively, optical biometers may struggle to penetrate the lens opacity, so immersion ultrasound biometry may be required.

Therapy with topical antihypertensive drops should be continued, but any prostaglandin analogue should be discontinued because this drug class can exacerbate inflammation. If not already started, therapy with a topical steroid and NSAID would be initiated immediately and the agents dosed every 2 hours and four times daily, respectively. Oral acetazolamide (Diamox, Wyeth Pharmaceuticals) 250 mg three times daily would also be started; the goal is to reduce the IOP to below 30 mm Hg before surgery to decrease the risk of vascular occlusion.

Surgery should be performed within the next 2 days if possible. Peribulbar anesthesia would be optimal for two reasons: (1) the duration of surgery may be prolonged, and (2) the eye is inflamed and has a recent history of multiple surgeries. The instillation of trypan blue dye would help identify the location and extent of the capsular breach. An OVD with a high molecular weight such as Healon GV (Johnson & Johnson Vision) would be injected to repel flocculent material and enhance control of the capsule. If the capsular breach is isolated, then it would be incorporated into the capsulorhexis. If the extent of the capsular breach is unclear, then a can opener capsulotomy with multiple relieving incisions would be performed.

Soft, swollen lens material would be removed with I/A suction. The phaco machine settings would be lowered to conservative values and the bottle height lowered as well. Slow, controlled phacoemulsification is the goal. It is important to avoid grabbing capsular remnants and free edges. Additionally, hydrodissection would be avoided to minimize expansion volume within the capsule and reduce the risk of further tears. Minimal hydrodelineation would be performed to define the nucleus. If the capsulotomy is incomplete, the nucleus would be bowled out to avoid rotation and unnecessary instrumentation.

The intracapsular placement of a one-piece IOL is the primary goal, but sulcus options should be available. I prefer to use a monofocal toric IOL and a distance target in patients with poorly controlled diabetes. I would have a low threshold for implanting a MIGS device because of the high risk of bleb failure. At the conclusion of surgery, a needle would be used to viscodissect the bleb in hopes of restoring function.

The eye is at high risk of postoperative macular edema. The postoperative treatment regimen would include a topical steroid and a topical NSAID administered every 2 hours and twice daily, respectively. IOP would be assessed 1 day after surgery.

In the short term, the difficulty level of the case depends on the extent of damage to the capsule identified at the start of surgery. The long-term challenge is managing the patient's ocular hypertension. Both points must be considered during surgical planning.

ROBERT J. NOECKER, MD, MBA

The patient appears to have lens-particle glaucoma (LPG), presumably caused by rupture of the lens capsule during trabeculectomy. She received medical therapy and close observation. When a large amount of lens material is present in the anterior chamber, surgical removal of the material and probably the lens is indicated.

Therapy with hypotensive medications and atropine would be continued before surgery. Intensive steroid treatment would be initiated to quiet the eye. Suture lysis, if possible, would be performed immediately to enhance aqueous outflow and reduce the IOP. A-scan biometry would be performed to calculate the IOL power.

The procedure would be scheduled as complex cataract surgery combined with bleb revision. After the eye is entered, irrigation and aspiration would be performed to evacuate lens material from the anterior chamber. The lens capsule would be stained with trypan blue dye to improve visualization, and the capsulorhexis would be created. Hydrodelineation would be performed, but hydrodissection would be avoided to prevent a posterior extension of the tear. The lens would be debulked centrally, and cortex would be removed with irrigation and aspiration. Placement of the IOL would be guided by the status of the capsule.

Next, a cyclodialysis spatula would be inserted through the flap and into the bleb to ensure patency. I would consider performing a subconjunctival injection of mitomycin C and dexamethasone depending on how inflamed the conjunctiva is. The postoperative drug regimen would consist of topical steroids and antibiotics. Glaucoma medications would be discontinued.

MONISHA MANDALAYWALA VORA, MD

The patient is likely experiencing LPG caused by unintended surgical trauma. LPG results when the lens capsule is disrupted by trauma, laser posterior capsulotomy, or intraocular surgery. Glaucoma typically develops a few weeks after the precipitating event but can occur within a few days of the inciting trauma or surgery.[1] Key to the diagnosis are a history of surgery or trauma and a compromised anterior capsule; the latter feature differentiates LPG from other lens-induced glaucomas such as phacolytic glaucoma. Slit-lamp findings for eyes with neovascular glaucoma and uveitic glaucoma may be similar, but these entities can be ruled out with a thorough history and examination.[2]

The patient's elevated IOP and flat bleb are secondary to blockage of the trabecular meshwork and trabeculectomy ostium by flocculant cortical material in the anterior chamber. In situations like this, the extent of IOP elevation typically corresponds to the amount of lens material present in the anterior chamber. Prompt surgical intervention to evacuate the cortical material is crucial to minimize the risk of irreversible damage to the optic nerve.

Before surgery, therapy with acetazolamide would be initiated. An injection of mannitol would be performed to soften the eye and reduce the risk of iris prolapse when the initial incision is made. A paracentesis incision would be made, followed by an anteriorly positioned, longer wound to prevent iris prolapse. A subincisional iris hook would be placed directly underneath the main wound to protect the iris during phacoemulsification. Next, cortical material would be aspirated to provide a clearer view of the anterior lens capsule, which would be painted with trypan blue dye to facilitate inspection. If the rupture is small, the area would be incorporated into the capsulorhexis. For a larger tear, the use of a Kuglen hook with or without iris hooks can facilitate an evaluation of the capsule's edges.

Given the patient's age, bimanual irrigation and aspiration of the cortical and nuclear material should be sufficient to remove the entire lens. If no further compromise of the capsule is detected, a one-piece acrylic IOL would be placed in the bag. The edges of the capsule should just overlap the edge of the optic. A cystotome and microforceps may be used to aid in preventing anterior capsular phimosis.

Before removing the OVD from the eye, the iris and ostium of the trabeculectomy would be gently swept in an ab interno fashion to ensure that no cortical material remains to block the drainage pathway to the outside of the eye. The bleb would be needled in an ab externo fashion as necessary with a 27-gauge needle to optimize postoperative flow. If needed, IOP-lowering medication would be prescribed, and bleb-salvaging

procedures such as laser suture lysis would be performed.

WHAT I DID: LISA BROTHERS ARBISSER, MD

Because of the antigenic nature of the dispersed cortical material in this rapidly developing hypermature cataract and the uncontrolled IOP, therapy with a topical steroid, NSAIDs, and an oral acetazolamide sequel was initiated immediately. The patient was taken to the OR the next day. A 3-mL peribulbar block was administered inferiorly with manual massage. Mannitol 0.25 g/kg was delivered as an intravenous bolus. A paracentesis and a small clear corneal incision were created in a normotensive eye. (Scan the QR code to watch the procedure.)

An Osher cannula was used to paint the capsule sparingly with trypan blue dye under a cohesive OVD. Dye was reinstilled as necessary throughout surgery. A 23-gauge cannula on a 3-mL syringe was used to aspirate the nucleus and flocculant material. A 26-gauge cannula was used for cortical removal. Additional OVD was injected as needed to maintain the chamber. Automated irrigation was avoided to ameliorate the risk of deepening or shallowing of the anterior chamber, which could have caused extension of the capsular tear.

Once the lens material had been removed, the inferior edge of the anterior capsular tear was easily identified with staining. The tear extended from equator to equator without involving the posterior capsule. No lens material was visible in the posterior segment.

WATCH IT NOW

Despite repeated instillation of dye and retraction of the superior iris, I could not locate the superior edge of the anterior capsular tear. As I strategized my next move, I noticed intense staining of the superior pupillary margin. This was unexpected because there was no basement membrane to stain at the iris edge. I suddenly realized that the superior capsular edge must have fused with the pupillary margin for nearly 180º.

Using blunt and limited sharp dissection combined with viscodissection, the synechial adhesion of the anterior capsule to the iris was pried apart in the proper plane, and the position of the superior hemicapsule was restored.

A tangential cut was made with Vannas scissors in the inferior edge of the capsular leaflet to create a continuous hemicircular capsulorhexis. The cut increased exposure of the visual axis because the spontaneous split of the capsule by intumescence was not central. A continuous hemicircle was torn in a slightly superior position, and the visual axis was exposed. A one-piece acrylic IOL was inserted into the bag, and the haptics were sequestered in the fornix. The anterior capsular leaflets prevented the haptics from entering the sulcus, thereby eliminating the risk of uveitis-glaucoma-hyphema syndrome.

Acetylcholine (Miochol-E, Bausch + Lomb) was instilled, and the OVD was removed manually. Constriction of the pupil was surprisingly symmetric. The chamber was irrigated with triamcinolone acetonide (Triesence, Alcon) diluted 1:10 with balanced salt solution to confirm the absence of residual vitreous and to act as an antiinflammatory. An intracameral injection of moxifloxacin (off-label use) was performed. The incisions were confirmed to be watertight, and the IOP was found to be within a normal range with a Barraquer tonometer.

The IOP remained in a normal range after surgery as the hypotensive medications were tapered, but the bleb was still flat. The patient's UCVA was 20/30. After a 1-month taper of the steroids and a 3-month taper of the NSAIDs, the eye was quiet, and no macular edema was observed. Gonioscopy showed no peripheral anterior synechiae or obvious ostomy for the bleb.

The cause of the patient's uncontrolled IOP after vitrectomy is mysterious. I suspect that the trabeculectomy blade breached the anterior capsule as it entered the anterior chamber of the soft, vitrectomized eye. The subsequent formation of a hypermature cataract might have caused the capsule to split from equator to equator. ■

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