# Formation of a Pupillary Membrane After Ocular Trauma

BY SANDRA M. JOHNSON, MD, AND JOSH NUNN

## **CASE PRESENTATION**

A 68-year-old black man with a history of primary open-angle glaucoma was referred for ongoing care to the University of Virginia Glaucoma Service in June 2011 by an outside ophthalmologist. The patient had had a very shallow anterior chamber and hypotony in his right eye, since the ripcord suture had been removed from his Baerveldt glaucoma implant (Abbott Medical Optics Inc.) within 1 week of the referral. The patient's right eye had undergone phacotrabeculectomy with laser suture lysis (LSL) and adjunctive 5-fluorouracil in 2010, followed by the placement of a Baerveldt implant in 2011.

Upon examination, the patient's BCVA was 6/200 OD and 20/20 OS. His ophthalmic medical regimen included prednisolone 1% q.i.d. in his right eye and brimonidine 0.2%-timolol 0.5% b.i.d. and bimatoprost 0.01% q.h.s. in his left eye. Applanation IOP measured 2 mm Hg OD and 12 mm Hg OS. Prior Humphrey visual field testing (Carl Zeiss Meditec, Inc.) of the patient's right eye showed dense superior loss. The average central corneal thickness by ultrasound pachymetry was 596 mm OD and 597 mm OS. A gonioscopic examination revealed an open angle to a ciliary body band in both eyes. The patient's past ocular history was otherwise negative.

We deepened the anterior chamber of the patient's right eye with Viscoat (Alcon Laboratories, Inc.), a procedure repeated 4 days later, with recurrence of hypotony and a shallow anterior chamber each time. The patient was brought to the OR 6 days later to tie off his tube with multiple nylon sutures. After the revision, the IOP in his right eye initially returned to a baseline value of 31 mm Hg, and his BCVA improved to 20/50. Therapy with brimonidine 0.2%-timolol 0.5% was started for his right eye. Initially, the IOP dropped down into the normal range. On postoperative day 40, however, the IOP in his right eye measured 38 mm Hg,

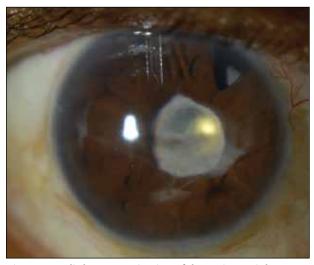


Figure 1. A slit-lamp examination of the patient's right eye revealed a dense pupillary membrane across the pupil and organized fibrin strands from the iris to the cornea.

and we performed LSL. Two hours postoperatively, the IOP in his right eye decreased to 4 mm Hg, and the brimonidine 0.2%-timolol 0.5% was stopped.

Three days after LSL, the patient rubbed his right eye while sleeping and dislodged his eye shield. The next day, the IOP in his right eye was 3 mm Hg. A slit-lamp examination revealed that the trauma had resulted in iridocorneal touch, as there were fibrin strands in the anterior chamber from the iris to the cornea in several places. We instructed the patient to instill prednisolone 1% six times per day. Over the next 2 weeks, the IOP in his right eye stabilized at between 10 and 12 mm Hg, and a significant pupillary membrane (PM) formed. After 1 month, the IOP in his right eye was 23 mm Hg, and the prednisolone, which was dosed six times a day, was tapered to q.i.d and then replaced with loteprednol 0.5% q.i.d. in the event that a steroid response was

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developing. Brimonidine 0.2%-timolol 0.5% b.i.d was also prescribed, and the IOP returned to a range of 10 to 12 mm Hg. Three months after the trauma, however, an organized fibrin membrane remained intact across the pupil, and the patient's BCVA was 20/80 (Figure 1).

### **HOW WOULD YOU PROCEED?**

- Would you continue medical management?
  If so, would you implement any changes?
- Would you perform membrane lysis with an Nd:YAG laser?
- · Would you surgically excise the membrane?

## **SURGICAL COURSE**

After 3 months of medical treatment with prednisolone and loteprednol, the patient underwent three sessions of Nd:YAG laser treatment at 0.7 to 2.1 mJ with more than 100 applications per session spaced over 8 weeks for the removal of the PM (Figure 2). He was given a 1-week course of difluprednate 0.05% q.i.d. after each session and was switched from loteprednol 0.5% to prednisolone 1% q.i.d. after the second session. The Nd:YAG treatments successfully disrupted the strands between the cornea, iris, and membrane, but the PM re-formed after each session. One month after the third Nd:YAG session, the PM persisted and remained visually significant, leaving the patient with a visual acuity of 20/400. We decided to return to the OR for surgical excision of the PM.

We made a paracentesis at 6 o'clock and injected lidocaine hydrochloride solution (Xylocaine MPF 1%; AstraZeneca LP) followed by Healon (Abbott Medical Optics Inc.). We used a cystotome to puncture the white membrane across the pupil and a Utrata forceps to peel it. We also used intraocular disposable Greishaber scissors (Alcon Laboratories, Inc.) to snip the superior membrane in several locations and then peeled it. We made a second paracentesis at 10 o'clock and then bimanually stretched the pupil. With the scissors, we snipped an adhesion between the pupil and IOL at the 7-o'clock position. A 10–0 nylon suture was placed across both paracenteses, and the knots were

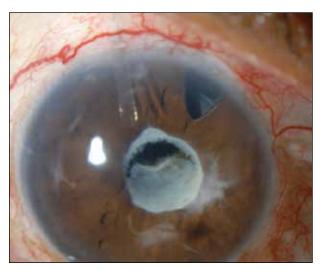


Figure 2. Slit-lamp view of the patient's right eye after one Nd:YAG laser treatment.

buried. We performed a sub-Tenon injection of triamcinolone 40 mg and a subconjunctival injection of an antibiotic and dexamethasone.

## **OUTCOME**

On the first postoperative day, no fibrin could be seen on slit-lamp examination, and the BCVA of the patient's right eye improved from hand motions preoperatively to 20/100. The IOP in his right eye remained stable at 10 to 12 mm Hg. We prescribed ciprofloxacin 0.3% q.i.d., prednisolone 1% every 2 hours while awake, and cyclopentolate 1% q.h.s. for his right eye. On postoperative day 20, the eye remained clear of fibrin, and his BCVA improved to 20/40. The ciprofloxacin was stopped, and the prednisolone 1% was decreased to q.i.d. and gradually tapered to b.i.d. (Figure 2).

## **DISCUSSION**

PMs may form as an early postoperative complication of cataract extraction and combined phacoemulsification and trabeculectomy. 1-3 It is believed that the mechanism for the postoperative formation of a PM is inflammation after intraocular surgery, which disrupts the blood-aqueous barrier, allowing plasma proteins, including fibrin, to enter the anterior chamber. 4 Our patient developed a PM after rubbing an eye with a shallow anterior chamber. To our knowledge, this is the first reported case of a PM's forming as a result of ocular trauma associated with a flattening of the anterior chamber.

We believe the trauma to the eye combined with the previous shallow anterior chamber caused intraocular inflammation similar to that after cataract

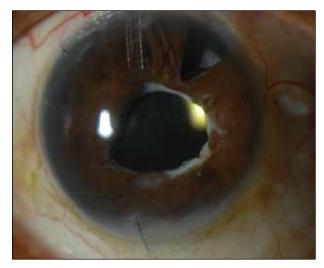


Figure 3. Slit-lamp view of the patient's right eye after the PM was excised with Grieshaber scissors.

surgery. We immediately increased the frequency of the patient's prednisolone 1% eye drops in an effort to decrease inflammation and stabilize the bloodaqueous barrier.5 Increased IOP is a well-known side effect of prednisolone. The patient was switched to loteprednol, which is still effective at decreasing intraocular inflammation but induces less IOP elevation than prednisolone, 6,7 and brimonidine 0.2%-timolol 0.5% was started. This new regimen treated the ocular hypertension but, as expected, failed to have any effect on the fibrin membrane.

Nd:YAG laser treatment has been shown to be effective in resolving secondary PMs in several case series as well as in a randomized controlled trial conducted by Angra et al.<sup>8-10</sup> Angra et al were able to achieve a 3- to 4-mm opening in the PMs of all of their Nd:YAG patients using 16 to 36 laser spots at 5 mJ per burst. Three membranes with thicknesses of 1.20 to 1.26 mm required a second Nd:YAG session to create an adequate opening. For our patient, laser treatment was initially successful in disrupting the membrane, but the PM re-formed after each session (Figure 3).

Others have described removing PMs with a vitrophage through a pars plana or pars plicata approach. 11 We instead used the previously described approach. The surgery successfully removed the PM, and the patient's IOP has remained in the normal range without medical therapy since the surgery.

Another possible treatment option would have been an anterior chamber injection of recombinant tissue plasminogen activator (tPA) when the fibrin was initially present. In a randomized, prospective, multicenter study, Heiligenhaus et al showed that a single 10-µg

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injection of tPA accelerated the lysis of postoperative fibrin in the anterior chamber. 12 We did not choose this approach because the fibrin membrane became too organized for tPA to be effective.

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