

INTERMITTENTLY BLURRY VISION AND IOP SPIKES

Two years after complicated cataract surgery, a patient needed help.

BY JOANN GACONI, MD; THOMAS PATRIANAKOS, MD; ANGELA TURALBA, MD; AND DOUGLAS J. RHEE, MD

CASE PRESENTATION

An 80-year-old man of European ancestry presented with a complaint that the vision in his left eye had been blurry for the past few weeks. The patient was referred for the management of uncontrolled IOP.

He had a history of bilateral cataract extraction, and the surgery on his right eye was unremarkable. Surgery on his left eye was significant for a posterior capsular tear, anterior vitrectomy, and the

placement of an anterior chamber IOL. The following day, the patient underwent pars plana vitrectomy and lensectomy with an IOL exchange. The subsequent surgeon sutured a posterior chamber IOL to the sclera. For the ensuing 2 years, the patient continued to experience IOP spikes and inflammation, and he later developed iris neovascularization in his left eye.

His current medications include brimonidine, a fixed combination of dorzolamide and timolol, travoprost, difluprednate, and intermittent bevacizumab. Upon examination, his visual acuity was 20/20 OD and 20/200 OS. His IOP measured 13 mm Hg OD and 28 mm Hg OS. Central corneal thickness measured 550 μ m OD and 555 μ m OS (Figures 1–4).

—Case prepared by Douglas J. Rhee, MD

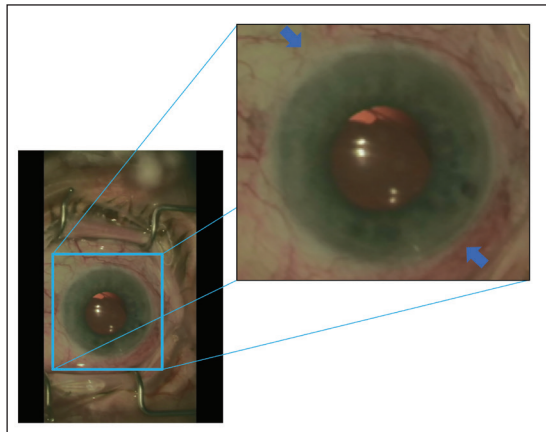


Figure 1. The view through the OR microscope shows corneal edema and a partially subluxated scleral-sutured IOL. The blue arrows indicate the location of the two polypropylene stabilization sutures.

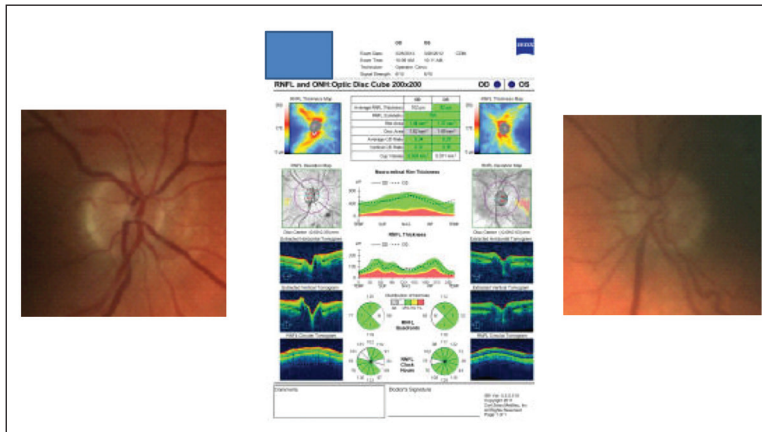


Figure 2. Optic nerve photographs and an OCT scan of the corresponding retinal nerve fiber layer.

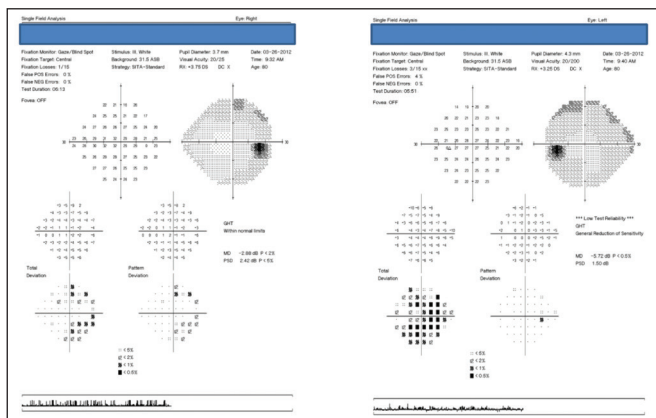


Figure 3. Corresponding automated achromatic visual fields show damage.

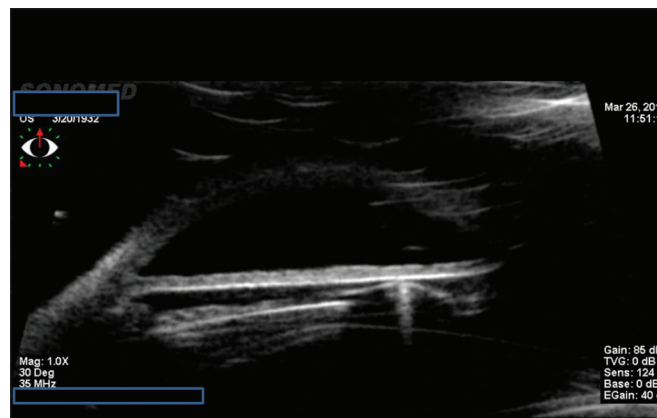


Figure 4. Imaging with ultrasound biomicroscopy shows a tilted IOL that is in close proximity to the iris.

**JOANN GIACONI, MD**

The retinal nerve fiber layer of the left eye is in the green zone but thinner than that of the right eye, and the former is presumably thinner than its own baseline measurement 2 to 3 years ago. The patient has therefore experienced nerve damage from the past 2 years of IOP spikes, and the IOP is uncontrolled despite maximal topical medical therapy. The visual field depression with a mean deviation of -5.72 dB in the left eye is most likely due to corneal edema rather than glaucoma.

The patient's history of complicated cataract surgery, IOP spikes, inflammation, a decentered scleral-sutured IOL (Figure 1), and lens tilt with iris contact (Figure 4) all point to uveitis-glaucoma-hyphe-ma (UGH) syndrome. Given the iris neovascularization and bevacizumab injections, my first step would be to carefully examine the retina for other causes of neovascularization because iris neovascularization is more common with retinal ischemic conditions. Assuming other causes of neovascularization are ruled out, the offending IOL will have to be repositioned or replaced to avoid further chafing of the iris, the IOP must be controlled, and the corneal edema must be addressed to rehabilitate the eye.

Rather than resuture the posterior chamber IOL or externalize its haptics, both of which could lead to renewed iris chafing, my preference would be to replace the posterior chamber IOL with an anterior chamber IOL because there appears to be sufficient iris support. I would combine this procedure with glaucoma surgery, either ab interno trabeculectomy or ab interno placement of a subconjunctival shunt, because the IOP is likely to remain high postoperatively and the glaucoma is

still at an early stage. Descemet stripping endothelial keratoplasty can be performed later to address the corneal edema once the inflammation and IOP are controlled to increase likelihood of the procedure's success if corneal edema persists.

**THOMAS PATRIANAKOS, MD**

The cause of elevated IOP appears to be mechanically induced inflammation from a displaced sutured IOL, as seen clinically and on ultrasound biomicroscopy (UBM). This diagnosis can be confirmed on examination by looking for IOL instability during saccade of the patient's left eye. This scenario is becoming more common as the 10-0 polypropylene sutures often used to fixate IOLs break, and it helps to explain a shift in surgeon preference to PTFE (Gore-Tex) sutures for IOL fixation.

After undergoing many procedures on his left eye, this patient may not wish to proceed with additional surgery. Fortunately, the optic nerve is still preglaucomatous, according to OCT imaging and visual field testing. It would therefore be acceptable to observe him or to attempt to optimize topical medical therapy before recommending further surgical intervention.

After a discussion of potential side effects, an oral carbonic anhydrase inhibitor could be started in the short term to lower IOP, as long as there are no contraindications. The steroids could also be tapered to determine if the IOP spike is due to a steroid response, although this possibility is highly unlikely.

If none of the aforementioned options works and the patient is willing, surgical intervention should be attempted to relieve the mechanical

apposition of the IOL against the posterior portion of the iris. Either the IOL can be removed (leaving the patient aphakic), or it can be refixated with a PTFE suture. An attempt can be made to rethread the eyelet, or the entire haptic can be lassoed and sutured to the sclera.

**ANGELA TURALBA, MD**

A unifying diagnosis that explains the findings in this case is UGH syndrome. Although uveitis, glaucoma, and hyphe-ma are the hallmarks of this condition, corneal decompensation and iris neovascularization have also been reported. UBM was diagnostic and confirmed chafing of the lens against the iris.

The poor visual acuity in the patient's left eye may be due to refractive error, corneal decompensation, optic neuropathy, or cystoid macular edema. No significant glaucomatous defects are evident on OCT or visual field testing. The latter showed a generalized reduction in sensitivity, which could be due to corneal decompensation, but central corneal thickness was symmetric between the eyes and in the average range.

Assuming there is no macular edema, it is possible that the patient's decline in vision is due to refractive error from the tilted or decentered IOL. If so, the eye has good visual potential, and I would attempt visual rehabilitation by resuturing the superior haptic that seems to be chafing the iris. Alternatively, the IOL could be replaced with a sutureless scleral-fixated three-piece IOL, which might be less prone to dislocation or tilt. A third option would be to explant the IOL and leave the patient aphakic if his visual potential is poor or he is a contact lens candidate.

If the IOP did not improve after the iris chafing was relieved and all steroid drops had been discontinued, I would implant a tube shunt. I would opt for a staged approach in this case because the patient has early glaucoma.



WHAT I DID:
DOUGLAS J. RHEE, MD

I felt that the chronic inflammation and IOP spikes were consistent with UGH/iris chafing syndrome. In my experience, an IOL exchange alone is insufficient when the inflammation has been present for more than 6 months. Chronic inflammation had caused the iris neovascularization.

During surgery, I used iris hooks to improve visualization, and I placed a Sheets glide behind the IOL. I cut the polypropylene sutures and removed the IOL. I then performed scleral fixation of a replacement IOL with polypropylene sutures 2.5 mm posterior to the limbus. With three interrupted 10-0 nylon sutures, I closed the 6.5-mm near-clear beveled sclerocorneal incision via a temporal approach. I placed an Ahmed Glaucoma Valve (model FP-7, New World Medical) superotemporally and covered the tube with donor pericardium prior to conjunctival closure (Figure 5).

The patient's visual acuity improved from counting fingers on postoperative day 1 to 20/60 1 week after surgery and 20/30 2 months after surgery. IOP decreased to 14 mm Hg on postoperative day 1, a level that was maintained at 1 week. He began using timolol approximately 1 month after surgery, and IOP measured 13 mm Hg at 2 months. Eight months after surgery, the

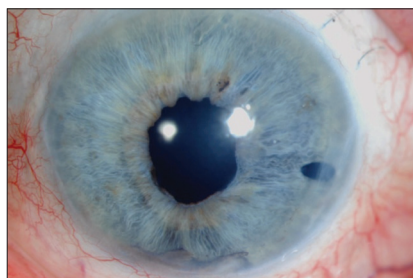


Figure 5. A slit-lamp examination of the patient's left eye shows slight corneal edema, a round pupil, and a well-positioned posterior chamber IOL. At approximately 12:15 o'clock, the silicone tube of an Ahmed Glaucoma Valve can be seen entering the anterior chamber.

patient reported that his left eye was comfortable. His visual acuity was 20/25, and IOP was 13 mm Hg on once-daily timolol. ■

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