Elevated IOP After the Implantation of a Valved Drainage Device

BY MARK WERNER, MD

CASE PRESENTATION

A 93-year-old male with end-stage glaucoma was referred to my private glaucoma consultation service when the vision in his left eye deteriorated from 20/200 (measured several months previously) to hand motions only. His right eye had been diagnosed with no light perception quite some time earlier.

Over the past year, the IOP in the patient’s left eye had measured in the high-to-midteens with occasional fluctuations into the 20s despite a medical regimen of Cosopt (dorzolamide hydrochloride-timolol maleate ophthalmic solution; Merck & Co., Whitehouse Station, NJ) and Lumigan (bimatoprost ophthalmic solution 0.03%; Allergan, Inc., Irvine, CA). No visual fields were available, and I could not obtain an accurate surgical history. The neighbor who brought the patient to his appointments also shopped and cooked for him. She was very concerned about him and took notes during each visit. Although the patient was confined to a wheelchair, he was fairly lucid.

On examination, the patient’s IOP measured 22 mm Hg OD and 32 mm Hg OS with Goldmann applanation tonometry. A slit-lamp examination of his right eye showed a vascularized cornea and a blood clot that completely filled the anterior chamber, making it impossible for me to perform gonioscopy or a fundus examination. The patient’s left eye had a clear cornea, a deep and quiet anterior chamber, a surgical iridectomy, and a posterior chamber IOL inside a clear capsule. Gonioscopy showed trabecular meshwork posteriorly for 360º. Fundoscopy revealed a cup-to-disc ratio of 0.99 OS and a normal retina.

I decided to operate in an effort to preserve the patient’s remaining vision. On February 26, 2009, I implanted an Ahmed Glaucoma Valve (New World Medical, Inc., Rancho Cucamonga, CA) and covered the tube anteriorly with a pericardial patch graft in the superotemporal quadrant of his left eye.

After an uncomplicated procedure, I did not note any shallowing of the anterior chamber, and I estimated that the patient’s IOP was 10 mm Hg by palpation. The valve was primed with balanced salt solution prior to its implantation.

During his first postoperative visit, the patient’s IOP measured 10 mm Hg OS, but his visual acuity remained hand motions only. The patient had postoperative ptosis, mild edema of the eyelid, and marginal reflex distances of approximately +1 mm OD and 0 mm OS. The tube was well positioned deep in the anterior chamber and did not touch any intraocular structures. The anterior chamber was deep with a moderate cellular reaction and no frank hyphema. Fundoscopy showed a flat retina.

I prescribed a routine regimen of prednisolone acetate 1% q2h while awake, atropine 1% b.i.d., and Vigamox t.i.d. (moxifloxacin 0.5%; Alcon Laboratories, Inc., Fort Worth, TX). On the fourth postoperative day, the patient’s IOP measured 6 mm Hg OS, and the ocular examination was essentially unchanged, except for a minimal choroidal effusion in the temporal area of the retina.

On the eleventh postoperative day, applanation tonometry by the ophthalmic technician showed that the patient’s IOP had increased to 33 mm Hg OS. A second measurement showed the IOP was slightly lower at 28 mm Hg. Except for a resolution of the choroidal effusion, the remainder of the patient’s examination was unchanged, and the vision in his left eye remained hand motions only. The eye’s anatomy and the patient’s lack of cooperation prevented me from visualizing the plate of the Ahmed valve.

I restarted the patient on Cosopt and instructed him to stop using the atropine and Vigamox. When he returned 2 weeks later, his vision remained hand motions only, and his IOP (as measured by a technician) was 30 mm Hg OS.
**HOW WOULD YOU PROCEED?**

1. What is the likely cause of the patient’s elevated IOP?
2. Would you add new agents to his medical regimen? If so, which one would you choose next?
3. Would you consider another operation on the patient’s left eye? If so, what procedure would you perform (bleb needling, laser trabeculoplasty, or a cyclodestructive procedure)?

**CLINICAL COURSE**

While examining the patient to determine the next step in his treatment, I asked him to look inferiorly so I could examine the bleb. When the patient insisted he was complying with my request, I noticed that his left eye was not properly adducted. When I turned on the lights, sat back, and directed the patient to look to the right, I noticed that his right eye was fully abducted but that his left eye was exotropic and did not cross the midline (Figure 1).

Additional tonometric measurements taken at the slit lamp showed an IOP of 25 mm Hg when the patient turned his head slightly to the right and 17 mm Hg when he turned his head farther to the right (approximately 30° relative to the slit lamp). I decided that the apparent increase in IOP in the left eye was due to restriction of ocular motility in the direction of greatest resistance, rather than a true increase in IOP. The patient’s IOP was within the normal range when his eyes were in a passive position. Because the patient had lost the ability to fixate and was unlikely to perform this maneuver in the course of his daily activities, I felt that observation was indicated. I instructed the patient to continue his current medical regimen and to return for follow-up in 1 week.

**OUTCOME**

Several subjective examinations over the next month showed that the exotropia remained but had improved slightly. The patient experienced significant improvement in his ptosis, but the vision in his left eye remained hand motions only.

During the next follow-up visit, the patient’s IOP measured 31 mm Hg OS when he looked straight ahead and 22 mm Hg OS when he turned his head approximately 25° to 30° to the right.

I instructed the patient to taper his use of steroids, to continue using Cosopt in his left eye b.i.d, and to begin using Lumigan in both eyes at bedtime. Approximately 3 months after the implantation of the drainage device, IOP measurements obtained by a technician and the physician with the patient’s left eye in the resting position (somewhat abducted) were 19 and 18 mm Hg, respectively. I determined that these IOPs were acceptable for the patient. Furthermore, because his poor vision prevented him from fixating and he was of advanced age, I felt that additional surgical intervention for the exotropia was not indicated.

**DISCUSSION**

I implanted a valved tube in this case due to evidence of a prior trabeculectomy in the patient’s left eye. I believed that, in my hands, the patient had a better chance of success and a lower risk of hypotony with an Ahmed implant than with another trabeculectomy. I prefer to use a 350-mm² Baerveldt implant (Abbott Medical Optics Inc., Santa Ana, CA), in younger patients, because in my and other clinicians’ experience, it controls IOP better and causes fewer hypertensive phases postoperatively than the Ahmed valve. The literature appears to support the use of Baerveldt implants in young patients, but the benefits have not been confirmed by randomized controlled trials. Anecdotally, I have observed a high incidence of hypotony and related complications in patients of advanced age who received nonvalved implants with greater surface area. I also chose an Ahmed valve for the elderly patient described in this case, because I felt it would control his IOP during the early postoperative period more reliably than the Baerveldt implant.

When the patient’s IOP rose precipitously in the weeks after the tube’s implantation, I initially thought that he was experiencing a hypertensive phase secondary to the device’s encapsulation. I was concerned about this development, because postoperative hypertension is predictive of worse outcomes with glaucoma drainage devices. The patient was already effectively blind in his right eye and had experienced a rapid loss of vision in his left eye preoperatively.

The patient was typically seated in the examination chair in the technician’s room during his evaluations,
because it was difficult to move him. The room lights were kept low for his comfort. My ability to detect the patient's exotropia was initially complicated by these conditions and the presence of ptosis and a swollen eyelid immediately postoperatively.

Because the patient's IOP remained high despite his use of two aqueous suppressants, I was more alert to confounding factors than I would have been otherwise. I did not assess his motility initially, however, because I usually perform this examination only on patients who complain of diplopia, and my patient was monocular with poor vision in his seeing eye. I began to suspect that the drainage device was affecting the patient's ocular motility when I had difficulty examining the plate of the implant at the slit lamp.

“I would consider the restriction of ocular motility to be in the differential diagnosis for an apparent increase in IOP after the implantation of a glaucoma drainage device.”

Investigators have observed restricted ocular motility with the Ahmed valve as well as other glaucoma drainage devices. Motility is usually impeded by the device’s encapsulation in muscle, contact between the muscle and the implant, or the physical mass of the implant or bleb. Although diplopia usually resolves over time or can be addressed with prism glasses, patients occasionally need surgical revision to restore full ocular movement. Surgical options that could allow patients to achieve proper fixation include revision of scar tissue around the implant, with or without concurrent surgery on the extraocular muscles. Additional surgery to restore the patient's ocular motility was contraindicated by his advanced age, poor vision, and the potential for serious complications (eg, a complete loss of vision in his left eye, corneal decompensation, exposure of the drainage tube, or a suprachoroidal hemorrhage).

Although a gaze-evoked elevation in pressure is a well-known phenomenon in patients whose ocular motility is restricted by thyroid ophthalmopathy, I believe that the current case is the first to describe this phenomenon after the implantation of a glaucoma valve. As glaucoma specialists, we need to ask which IOP—the one obtained while the eye is in a resting versus the forced primary (straight ahead) position—is clinically relevant for glaucomatous progression. This patient could achieve a straight-ahead gaze only with forcible adduction of his left eye. If the patient frequently and actively adds his eye, this maneuver may raise his IOP and damage the optic nerve.

Surgery on the extraocular muscles reportedly reduced gaze-related rises in IOP in patients with thyroid-related ophthalmopathy. Because my patient could not fixate, however, I did not believe he was vulnerable to this kind of damage and therefore decided not to pursue additional surgery.

My patient was one of an increasing number of individuals over the age of 90 who have uncontrolled or rapidly progressing glaucoma. In this case, the patient’s age and overall condition made it difficult to follow his end-stage glaucoma adequately and may have contributed to his severe loss of vision. He may not have been able to perform visual field testing reliably, and it is impossible to evaluate progression of very advanced glaucoma with structural testing. The patient's IOPs did not provide reliable information about the stage of his disease, because the measurements obtained in the office only occasionally fluctuated above the normal range.

I estimate that a patient who is older than 90 years with severe, uncontrolled glaucoma presents to me at least every 2 months. When I treat these patients, I carefully consider their life expectancy and their socioeconomic status before deciding on treatment. Fortunately, my patient had a kind and conscientious neighbor who ensured he could keep up with his medications and medical appointments. Losing light perception from untreated glaucoma would have adversely affected his quality of life.

Based on my patient's clinical course, I would consider the restriction of ocular motility to be in the differential diagnosis for an apparent increase in IOP after the implantation of a glaucoma drainage device. □

Mark Werner, MD, is a voluntary assistant professor of ophthalmology with a specialization in pediatric glaucoma at the Bascom Palmer Eye Institute at the University of Miami/Miller School of Medicine. He acknowledged no financial interest in the products or companies mentioned herein. Dr. Werner may be reached at wernerma75@yahoo.com.