Revision of a Valved Glaucoma Device

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CASE PRESENTATION

A 78-year-old Hispanic woman with longstanding open-angle glaucoma presented to the Faculty Practice Glaucoma Service at Mount Sinai Medical Center in New York in April 2010. She had been treated at another facility since the mid-1990s. Her ocular surgical history was significant for cataract extraction and multiple glaucoma surgical procedures in both eyes. In her right eye, she had undergone combined phacoemulsification with the implantation of a posterior chamber IOL and trabeculectomy with mitomycin C (MMC) in 1999, a trabeculectomy with MMC in 2004, implantation of an Ahmed Glaucoma Valve (New World Medical, Inc.) in 2007, and a revision of the device with irrigation of the tube in May 2009 (1 year prior to the April 2010 examination). In her left eye, the patient had undergone cataract extraction with implantation of a posterior chamber IOL in 1992. Subsequently, she developed uncontrolled glaucoma. A trabeculectomy with MMC in 1999 failed, and a bullous keratopathy required penetrating keratoplasty in 2010, which was complicated by a bacterial corneal ulcer. She subsequently developed absolute glaucoma in her left eye that required diode transscleral cyclodestruction in 2004.

Since April 2008, the patient had been on topical ocular hypotensive therapy in both eyes with brimonidine 0.15% t.i.d., dorzolamide 2% t.i.d, travoprost q.h.s., and methazolamide 50 mg by mouth b.i.d. These medications were not changed following the tube irrigation in May 2009. She was diagnosed with cystoid macular edema in her right eye in January 2010 and subsequently treated with nepafenac b.i.d and prednisolone acetate 1% t.i.d. Her past medical history was significant for essential hypertension, diabetes mellitus type 2, asthma, and a cerebrovascular accident in 1985. In January 2010, her systemic medications, which have remained unchanged for many years, consisted of aspirin, atenolol, diltiazem, lisinopril, insulin, metformin, albuterol, singulair, gabapentin, lovaza (omega-3), simvastatin, folic acid, calcium, and vitamin C.

On June 17, 2010, the patient’s BCVA was 20/30 OD and no light perception OS. She had mild bilateral ptosis. The pupil in her right eye was round and reactive, whereas that of her left eye was fixed and nonreactive and measured 6 mm. A slit-lamp examination of her right eye revealed mild conjunctival injection and no bleb over the superotemporal plate, a clear cornea, and a deep and quiet anterior chamber with an Ahmed Glaucoma Valve implant between the iris and the cornea at the 12-o’clock position. A slit-lamp examination of her left eye was notable for an opaque cornea. Posterior chamber IOLs were present in both eyes. The patient’s IOP by Goldmann applanation tonometry was 36 mm Hg OD but decreased to 20 mm Hg after massage. A fundoscopy examination of her right eye revealed mild conjunctival injection and no bleb over the superotemporal plate, a clear cornea, and a deep and quiet anterior chamber with an Ahmed Glaucoma Valve implant between the iris and the cornea at the 12-o’clock position. A slit-lamp examination of her left eye was notable for an opaque cornea. Posterior chamber IOLs were present in both eyes. The patient’s IOP by Goldmann applanation tonometry was 36 mm Hg OD but decreased to 20 mm Hg after massage. A fundoscopy examination of her right eye revealed a cup-to-disc ratio of 0.9. A Heidelberg Retina Tomograph (Heidelberg Engineering GmbH) scan assessed the right eye as outside normal limits in all sectors.

A Goldmann visual field examination of the right eye showed an enlargement of the superior arcuate defect compared to the field performed 7 months previously (Figure 1).
HOW WOULD YOU PROCEED?

• Would you treat the patient medically or surgically?
• Do any aspects of the patient’s past medical history contraindicate specific medical or surgical treatments?
• If you choose surgery, which procedure would you perform?

SURGICAL COURSE

We elected to perform a second revision of the Ahmed Glaucoma Valve implant. After administering topical anesthesia, we placed a 7–0 Vicryl corneal traction suture (Ethicon, Inc.) in the superotemporal quadrant and reflected the globe inferonasally to expose the superotemporal quadrant. We created a paracentesis at the 5:30-o’clock position and injected viscoelastic into the anterior chamber. A 25-gauge spinal needle stylette was passed through the tube via the paracentesis site, and resistance was noted at the junction between the tube and the plate. A 3–0 Prolene suture (Ethicon, Inc.) was passed through the tube and met resistance at the same junction. A second paracentesis site was made at the 3-o’clock position, and irrigation through the tube was unsuccessful. Next, we created a peritomy over the plate and opened the capsule overlying the plate to expose the Ahmed Glaucoma Valve implant.

During dissection, we observed that fibrovascular tissue had infiltrated the valve chamber (Figure 2A) and removed this growth. We incised the silicone casing on the posterior portion of the valve chamber with a Wescott scissors (Figure 2B). Next, we disengaged the valve mechanism from the body of the main plate and cut the valve from the tube (Figure 2C). The tube remained attached to the connecting piece, which was retained and sewn back onto the body of the main plate using two 10–0 nylon sutures (Figure 2D). The tube was easily reinserted into the anterior chamber through the tissue track from which it had been removed (Figure 2E), and it was secured with two interrupted 10–0 nylon sutures without dissection of the track. We closed the capsule surrounding the plate using interrupted and running 8–0 Vicryl sutures. The conjunctival peritomy was then closed using interrupted and running 8–0 Vicryl sutures. The wound was tested with fluorescein and found to be Seidel negative. Dexamethasone and moxifloxacin q.i.d. were dripped on the ocular surface, and a shield was placed over the eye.

OUTCOME

On the first postoperative day, the patient’s visual acuity in the right eye was count fingers. Goldmann applanation tonometry was 5 mm Hg. No choroidals were noted. We instructed the patient to discontinue methazolamide and all of her glaucoma drops and to start difluprednate q.i.d., moxifloxacin q.i.d., and nepafenac b.i.d. in that eye. On postoperative day 3, her visual acuity had improved to 20/40, and the IOP was 7 mm Hg. A large bleb had formed over the plate. On the most recent postoperative visit, 10 months after surgery, her visual acuity and Goldmann visual field were stable. The IOP measured 11 mm Hg on dorzolamide b.i.d. (Figure 3).

DISCUSSION

The Ahmed Glaucoma Valve implant is often implanted in patients whose glaucoma is inadequately...
controlled by medical therapy or for whom filtration surgery has been unsuccessful, is contraindicated, or is unlikely to succeed. Multiple studies have shown this device to be safe and effective for the lowering of IOP.1-9

Most recently, Budenz et al concluded that, although the average IOP after 1 year was slightly higher in patients who received an Ahmed Glaucoma Valve, there were fewer early and serious postoperative complications associated with use of the Ahmed Glaucoma Valve implant versus the Baerveldt glaucoma implant (Abbott Medical Optics Inc.).10 A cumulative probability of success of 80% and 49% for the Ahmed Glaucoma Valve implant at 1 and 5 years, respectively, was reported by Souza et al.11

In cases where the Ahmed Glaucoma Valve has failed, revision is an option, because this procedure can be performed without the dissection of another quadrant—particularly helpful in eyes with limited conjunctival tissue. However, studies comparing the efficacy of procedures after seton failure have not been published. Because this patient’s prior revision had been effective for 1 year, we decided to proceed with a second revision.

During the procedure, we noticed that the valve mechanism had been infiltrated by fibrovascular tissue, a reported cause of the Ahmed Glaucoma Valve’s failure in both adults and children.12,13 Hill et al studied six eyes that underwent revision of the Ahmed Glaucoma Valve due to distal occlusion secondary to fibrovascular ingrowth within the valve mechanism.13 They proposed that distal occlusion is caused by fibrovascular ingrowth between the plate of the Ahmed Glaucoma Valve and the silicone leaflets, causing failure of the device. They found that the ingrowth of tissue occurs through a gap produced by the surgeon’s grasping the device along its centerline, which indents the valve cover into the chamber containing the silicone leaflets. As a result, the valve cover lifts up at the sides, leaving a space for fibrovascular tissue to grow. The researchers found that handling the device laterally or posteriorly to the rivets did not damage the rivets or produce a gap between the valve cover body junctions. They proposed a no-touch zone on the Ahmed Glaucoma Valve as a way to prevent damage to the valve mechanism and fibrovascular ingrowth.13

In contrast, Thieme et al discussed encapsulation of the Ahmed Glaucoma Valve as an early complication in young patients that leads to inhibition of fluid exchange and failure of the procedure. The investigators found that the IOP could be controlled through removal of only the encapsulated blebs in all four of their cases.14 Thieme et al proposed that the valve mechanism was blocked by contracted scar tissue but that the device itself was not affected by the encapsulation. Their conclusion was based on the fact that surgical excision of the capsule immediately led to aqueous flow and a drop in IOP.14 Thieme et al investigated the capsule that forms around the plate macroscopically, microscopically, and ultrastructurally in four patients between 2 and 17 years of age. The investigators found that the smooth inner surface (facing the base plate of the implant) consisted of compressed collagen fibers with elastoid degeneration and formation of a pseudo-endothelium toward the base plate with a pronounced transformation of fibroblasts into myofibroblasts. The outer area was highly vascularized. In those vessels, electron microscopy revealed thrombosis. Inflammatory responses were nearly absent in all areas of the excised material. Trigler et al similarly noted minimal inflammatory response in the fibrous tissue.15

Despite our implantation of the Ahmed Glaucoma Valve using the no-touch-zone technique, we found a significant amount of fibrovascular ingrowth in this case. We decided to remove the fibrovascular growth inside

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Figure 3. Postoperative day 7 (A). Postoperative day 45 (B).
the valve chamber and the valve mechanism, while sparing the already formed capsule and plate. Because the capsule was present, we elected not to tie off the tube. We found no descriptions of this type of revision during our literature review. This procedure carries a minimal risk of additional complications, and it is a reasonable alternative to consider in patients with a failed Ahmed Glaucoma Valve.

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