Glaucoma and Penetrating Keratoplasty

BY BRYCE FORD, MD, AND GEORGE SHAFRANOV, MD

CASE PRESENTATION

In 1999, a 79-year-old white female presented to the Yale Eye Center in New Haven, Connecticut. She had previously undergone 360° argon laser trabeculoplasty in both eyes. Upon presentation, she was not taking any topical medications. Her visual acuity measured 20/100 OD and 20/30 OS, and her right eye exhibited more corneal edema and guttatae than her left. She had moderate bilateral cataracts with pseudoexfoliation and early bilateral changes in the retinal pigment epithelium. Her IOP measured 29 mm Hg OD and 24 mm Hg OS. Her cup-to-disc ratio was 0.6 OU with bilaterally thin but intact rims (Figure 1).

Subsequently, the patient was followed by the cornea and glaucoma specialists at Yale for Fuchs’ corneal endothelial dystrophy, pseudoexfoliative glaucoma, and cataracts. A stepwise addition of topical medications controlled her IOP in the high teens to low 20s.

In December 2000, the patient underwent a combined penetrating keratoplasty (PKP) and cataract extraction in her right eye. Because her IOP was more difficult to control postoperatively, she underwent trabeculectomy with mitomycin C in her right eye in September 2001 and in her left eye in December 2001.

The patient’s right corneal graft failed in late 2001 and required a repeat PKP in December 2001. Unfortunately, the second graft also failed and vascularized within 1 year, at which point she lost functional vision in her right eye. Her left eye had a visual acuity of 20/200 secondary to corneal edema and cataract.

The patient had an episode of blebitis in April 2002 in her left eye that resolved with topical fortified tobramycin and amikacin. She maintained an IOP in the single digits in her left eye throughout 2002 without using medications.

In June 2003, the patient underwent combined cataract extraction and PKP in her left eye (Figure 1). Afterward, managing the patient’s IOP became difficult, and the pressure rose to the mid-30s on maximal tolerated medical therapy, including 250 mg of oral Diamox (Wyeth Pharmaceuticals, Philadelphia, PA) q.i.d.

In February 2004, the patient’s IOP increased to between 46 and 56 mm Hg in her left eye, while still on maximal tolerated medical therapy, and increased graft edema ensued. We noted glaucomatous progression in her left optic nerve. Her visual acuity was count fingers in her right eye and 20/400 in her left. We performed diode laser transscleral cyclophotocoagulation on her left eye (2,500 mJ, 2,000 ms,
24 applications, 360°). Although the procedure controlled the pressure initially, her IOP rose again into the 30s, necessitating repeat cyclophotocoagulation on that eye in June 2004 (2,500 mJ, 2,000 ms, 26 applications, 360°).

By October of that year, the patient’s visual acuity was hand motions in her right eye and 20/200 in her left. Her IOP measured 32 mm Hg OS on maximal tolerated medical therapy after a failed trabeculectomy and two cyclophotocoagulation procedures, but the corneal graft remained clear. The patient continued to live at home independently and was reluctant to undergo another cyclophotocoagulation, because her vision had decreased with each previous treatment.

HOW WOULD YOU PROCEED?
1. Would you follow the patient on maximal tolerated medical therapy?
2. Would you recommend repeating the cyclophotocoagulation for a third time? Repeating trabeculectomy with mitomycin C? Implanting a glaucoma drainage device?
3. Would you consider placing a glaucoma drainage device initially instead of performing cyclophotocoagulation after trabeculectomy failed?
4. Would endocyclophotocoagulation be an option during an initial combined PKP and cataract extraction?

SURGICAL COURSE
After an extensive discussion with the patient and her family, we implanted without complication a polypropylene, 184-mm² Ahmed Glaucoma Valve (model S-2; New World Medical, Inc., Rancho Cucamonga, CA) between the iris and the PCIOL of her left eye on November 11, 2004. We noted extensive conjunctival scarring from the previous trabeculectomy and cyclophotocoagulations. On the day after surgery, her IOP was 15 mm Hg, but it began to decrease over the following days.

On December 4, 2004, the patient presented with a complaint of significant vision loss. Her IOP measured 4 mm Hg, and her vision was hand motions secondary to serous choroidal detachments obscuring the macula.

By December 12, 2004, “kissing choroidals” were present. A retinal specialist drained the choroidal effusions, and we ligated the tube of the Ahmed Glaucoma Valve using 7–0 Vicryl sutures (Ethicon, Inc., Somerville, NJ) to prevent further postoperative hypotony. Afterward, the patient’s IOP was 15 mm Hg. Her “kissing choroidals” resolved, and her vision improved to 20/400. On January 20, 2005, however, the Vicryl sutures dissolved, and the tube opened, which dropped her IOP back to 4 mm Hg. At that time, the examination also revealed a large conjunctival defect and the exposed plate of the Ahmed Glaucoma Valve. On January 25, 2005, the patient returned to the OR, where we placed a Tutoplast graft (Tutogen Medical, Inc., West Patterson, NJ) over the exposed plate and closed the conjunctiva over the graft. The conjunctiva was fragile and difficult to suture.

On January 31, 2005, the patient’s IOP measured 4 mm Hg, and, again, she had developed a large conjunctival dehiscence, exposing the Tutoplast graft and the Ahmed Glaucoma Valve. The following day, she returned to the OR, and we removed the valve and placed a Tutoplast graft over the sclerostomy opening into the anterior chamber.

OUTCOME
On February 7, 2005, the patient’s visual acuity was 20/400 OS, and her IOP in that eye was 10 mm Hg. Again, the conjunctival flap overlying the Tutoplast graft had dehisced. The conjunctival defect has since epithelialized, and her IOP has remained in the mid-teens without medications. We expect, however, that her IOP will rise again in the near future, at which point we will confront the issue of controlling her IOP after a failed trabeculectomy, two cyclophotocoagulations, and the failed implantation of a glaucoma drainage device.

DISCUSSION
Irvine and Kaufman1 first reported the high incidence of elevated IOP following corneal transplantation in 1969. Since then, glaucoma has been recognized as a leading cause of graft failure and blindness after PKP.2,3 Glaucoma can be a preexisting condition, as in the case presented herein, or secondary to the PKP through various mechanisms such as the graft’s distortion of the trabecular meshwork, inflammatory glaucoma with synechial angle closure, steroid response, fibrous/epithelial ingrowth, and aqueous misdirection.4 The incidence of post-PKP glaucoma in the literature ranges from 9% to 31% in the early postoperative period and 18% to 35% in the late postoperative period. Risk factors for post-PKP glaucoma include preexisting glaucoma, aphakia/pseudophakia, and a history of prior surgeries.4

The treatment of post-PKP glaucoma tends to be challenging, because the IOP is often difficult to measure accurately due to highly irregular corneal astigmatism and corneal edema. Corneal opacification makes tracking glaucomatous damage more difficult, because visual field analyses are less reliable, and the visibility of the optic nerve may decrease.

Managing post-PKP glaucoma is challenging, because both elevated IOP and its treatment can have deleterious effects on the graft.5,6 Management usually follows the same algorithm as does primary open-angle glaucoma, including topical medications, laser trabeculoplasty, and surgery. The last treatment option entails a trabeculectomy with an antimitabolite, the implantation of a glaucoma drainage device, a cyclodestructive procedure, or a combination of these.
CHALLENGING CASES

Ayyala et al identified in their study the importance of intraocular pressure in patients with ocular hypertension or open-angle glaucoma.

**STANDARD**

**INDICATIONS AND USAGE**

STANDARD 0.05
days indicated in the treatment of elevated intraocular pressure in patients with ocular hypertension or open-angle glaucoma.

**CONTRAINDICATIONS**

STANDARD 0.05 contraindicated in patients with (1) bacterial conjunctivitis, (2) a history of bacterial conjunctivitis, (3) a history of chronic obstructive pulmonary disease (see WARNING), (4) sinus bradycardia, (5) severe retinal disease, (6) known retinal disease, (7) corneal disease (see WARNING), (8) cardiorespiratory disease, or (9) hypersensitivity to any component of the product.

**WARNINGS**

As with any topical surgical drug, this drug is absorbed systemically. The same adverse reactions found with systemic administration of beta-blocker drugs may occur with topical administration. Therefore, adverse reactions and contraindications are more common in patients with adverse reactions and rare death in association with cardiac failure, have been reported following systemic or ophthalmic administration of timolol maleate (see CONTRAINDICATIONS). Carbohydrate failure (see WARNING) (7) cardiorespiratory disease, or (9) hypersensitivity to any component of the product.

As seen in this patient, however, previous surgeries can decrease the efficacy and/or increase the complication rate of subsequent surgeries. Implanting a glaucoma drainage device after a failed trabeculectomy and cyclophotocoagulation has not been reported in the literature. In this case, it had less-than-ideal results in the setting of decreased aqueous production and a scarred conjunctiva.

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