The Surgical Management of Uveitic Glaucoma

Advice on diagnosis and treatment.

BY ANDREW G. YOUNG, MD; JONATHAN G. CROWSTON, MD, PhD; AND ROBERT N. WEINREB, MD, WITH KEITH BARTON, MD, FRCP, FRCS, FRCOphth

CASE PRESENTATION

A 48-year-old black male was referred with a 1-month history of cloudy vision, photophobia, and periorbital pain in his right eye. An outside ophthalmologist had made the diagnosis of ocular hypertension and uveitis in the patient’s right eye and initiated treatment with a timolol-dorzolamide combination b.i.d., brimonidine-P 0.15% t.i.d., bimatoprost q.h.s., acetazolamide 500 mg p.o. b.i.d., and prednisolone acetate 1% q2h.

The patient’s IOP on presentation was 40 mm Hg OD. There was low-grade nongranulomatous uveitis, mild corneal edema, and a circumciliary flush but no keratic precipitates or iris nodules (Figure 1). The angles were open (grade 4). There was no posterior uveitis or retinitis, and the optic nerve had mild thinning of the inferotemporal rim (Figure 2). The patient’s left eye appeared to be within normal limits (Figure 1).

Comments on the Differential Diagnosis

KB: The differential diagnoses include inflammatory glaucoma and steroid-induced glaucoma. The degree of angle pigmentation can give some indication as to the cause of the elevated IOP. Occasionally, a patient is referred to me with pigmentary glaucoma that has been misdiagnosed as uveitic glaucoma. Eyes with uveitis can certainly have increased pigmentation of the angle, probably caused by a loss of pigment from the iris. The pigmentation of the angle in uveitic eyes is usually not as heavy as it is in pigment dispersion syndrome, and increased pigment may only be present in the inferior angle.
The topical prednisolone acetate was tapered, and the IOP decreased to 11 mm Hg. One week after the medication was stopped, however, the anterior chamber inflammation recurred, and the IOP rose to 39 mm Hg. Prednisolone acetate was therefore restarted, and topical flurbiprofen was added to the drug regimen.

**Comments on the Benefit of Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) in Treating Inflammatory Glaucoma**

**KB:** NSAIDs can assist in the control of anterior uveitis, but they are less effective than steroids. Care must also be taken, as there is some evidence that NSAIDs may reduce alpha agonist- and prostaglandin-mediated IOP lowering.1–3 For short-term treatments, I tend to use systemic NSAIDs more than topical formulations. For longer-term treatments, I prescribe topical formulations to reduce the gastrointestinal side effects. An alternative approach would be to use 1% rimexolone (Vexol; Alcon Laboratories, Inc., Fort Worth, TX), but this agent may only be appropriate for treating low-grade inflammation. Unfortunately, one cannot pharmacologically separate the anti-inflammatory effect of corticosteroids from their IOP-elevating effect.

Diffuse peripheral anterior synechiae developed over the next few months, despite intensive steroid treatment. The IOP in the patient’s right eye remained elevated at around 30 mm Hg. Repeat optic nerve examination revealed progressive thinning of the neuroretinal rim inferotemporally compared with the initial presentation. The surgical lowering of IOP was recommended.

**Comments on Trabeculectomy Versus Glaucoma Drainage Devices in Patients With Uveitis**

**KB:** I try to control the inflammation when possible prior to surgery. In patients with significant inflammation, I use systemic steroids.

**JGC:** In whom do you use systemic steroids, and what doses do you prescribe?

**KB:** I prescribe these drugs for patients with panuveitis or in cases in which there have been other forms of posterior uveitis or cystoid macular edema. In addition, I consider systemic steroids for patients with aggressive or recurrent anterior uveitis such as that associated with HLA-B27. Patients will typically take 40 mg of prednisone for 3 to 14 days before surgery and taper by 5 mg per day per week postoperatively.

**RNW:** What are your views regarding a glaucoma drainage device versus trabeculectomy?

**KB:** Glaucoma drainage devices probably work better in uveitic glaucoma than in almost any other type of secondary glaucoma. In my experience, however, a trabeculectomy with mitomycin C (MMC) achieves pressure control (less than 21 mm Hg off medications) in approximately 70% of eyes with uveitic glaucoma. In this series,4 my coworkers and I used a relatively low concentration of MMC (0.2 mg/mL for 3 minutes) and active postoperative management, including the removal of releasable sutures, bleb massage and needling, or 5-fluorouracil (5-FU) injections when necessary.

My long-term aim is to create a risk profile of uveitic glaucoma to identify in advance those patients who will require the implantation of an aqueous shunt as the primary surgical treatment and those in whom trabeculectomy will be sufficient. In patients with juvenile rheumatoid arthritis and in those who are aphakic, I usually perform primary aqueous-shunt implantation, because the success rate of trabeculectomy is so poor.

**Comments on the Use of an Antimetabolite**

**KB:** My coworkers and I completed a small study comparing 5-FU and MMC intraoperatively in uveitic glaucoma.5 After 3 years of follow-up, the success rate was sig-
nificantly higher in the MMC group. When we followed these patients for a longer period, up to 7 years, however, there was no significant difference in bleb survival. In the longer term, there is probably not much difference in IOP control. The eyes receiving 5-FU failed earlier, however, than those with MMC.

**CONCLUSION**

Managing uveitic glaucoma can be challenging. Intraocular inflammation and steroids can both lead to elevated IOP, and systemic and topical NSAIDs may have limited benefit as adjunctive therapy for uveitis. When surgical intervention is indicated, trabeculectomy with MMC can provide effective long-term IOP control. Lower concentrations of the agent may help reduce the incidence of postoperative hypotony in these patients. Primary glaucoma drainage devices may be preferable in cases of juvenile rheumatoid arthritis.

**Section editors Jonathan G. Crowston, MD, PhD, and Robert N. Weinreb, MD, are glaucoma specialists at the Hamilton Glaucoma Center, University of California, San Diego. Dr. Crowston is Assistant Professor of Ophthalmology. Dr. Weinreb is Distinguished Professor of Ophthalmology and Director. They acknowledged no financial interest in the products or companies mentioned herein.**

**Drs. Crowston and Weinreb may be reached at (858) 534-6290; jcrowston@ucsd.edu.**

**Keith Barton, MD, FRCPh, FRCS, FRCOphth, is Consultant Ophthalmologist and Glaucoma Service Director for Moorfields Eye Hospital in London. He acknowledged no financial interest in the products or companies mentioned herein.**

**Dr. Barton may be reached at +44 207 566 2256 or +44 207 566 2154; keith1barton@aol.com.**

**Andrew G. Young, MD, is a Senior Clinical Fellow at the Hamilton Glaucoma Center, University of California, San Diego. He acknowledged no financial interest in the products or companies mentioned herein. Dr. Young may be reached at (858) 534-8824.**