Argon Laser Trabeculoplasty for Intractable Glaucoma Following Intravitreal Triamcinolone

Based on experimental and clinical studies performed by Machemer and coworkers and other researchers, intravitreal injection of triamcinolone acetonide (IVTA) has been used as an alternative for the treatment of several diseases of the retina and uvea, such as chronic cystoid macular edema, age-related macular degeneration, and retinal vascular occlusion. However, it is an invasive procedure, with the risks of complications such as raised intraocular pressure (IOP), vitreous hemorrhage, and endophthalmitis. We describe the clinical course of 3 patients who developed secondary open-angle glaucoma following IVTA, uncontrollable by topical antiglaucomatous medication, treated with argon laser trabeculoplasty.

Report of Cases. Patient 1 was a 44-year-old man with subfoveal choroidal neovascularization in angioid streaks, despite 3 sessions of photodynamic therapy with verteporfin. Patient 2 and patient 3, respectively 65 and 67 years old, had diffuse diabetic macular edema. In an attempt to reduce subfoveal exudation and suppress subretinal angiogenesis (in patient 1), and to reduce diffuse diabetic macular edema (in patients 2 and 3), a transconjunctival intravitreal injection of 20 mg/0.1 mL of crystalline triamcinolone acetonide was administered to the affected eye. The procedure was carried out under topical anesthesia after paracentesis was performed to decrease the volume of the globe. All patients were phakic with normal baseline IOP and no evidence of glaucoma.

One month after the injection, IOP increased to 36 mm Hg in patient 1, 40 mm Hg in patient 2, and 37 mm Hg in patient 3, necessitating antiglaucomatous topical treatment with a combination of timolol maleate and dorzolamide hydrochloride ophthalmic solution twice daily. Two months after the injection, IOP was still high: 54 mm Hg in patient 1, 38 mm Hg in patient 2, and 32 mm Hg in patient 3. Latanoprost once daily (in the evening) and oral acetazolamide were added to the treatment regimen. During the next 2 months, maximally tolerable antiglaucomatous topical treatment failed to control the pressure. Five months after the injection, IOP ranged between 30 mm Hg and 40 mm Hg and the cup-disc ratio was increased in all 3 patients. Because IOP remained uncontrolled and triamcinolone crystals were still detectable in the vitreous cavity at the 6-o’clock position in the periphery of the fundus, an argon laser trabeculoplasty was performed over 360° in all 3 patients. One month after argon laser trabeculoplasty, the IOP was 20 mm Hg in patient 1, 15 mm Hg in patient 2, and 18 mm Hg in patient 3 while receiving a topical combination of timolol and dorzolamide ophthalmic solution, although triamcinolone crystals were still detectable in the vitreous cavity.

Comment. Secondary glaucoma is a risk with any form of corticosteroid therapy. Only 3 previous studies have reported secondary open-angle glaucoma uncontrollable by topical medication following IVTA, requiring trabeculectomy alone or removal of the depot corticosteroid by pars plana vitrectomy combined with trabeculectomy. To our knowledge, this is the first reported case of argon laser trabeculoplasty for intractable secondary glaucoma following IVTA. We decided to perform argon laser trabeculoplasty because of the intractable steroid-induced glaucoma, the still detectable presence of triamcinolone crystals in the vitreous cavity after 5 months, and pharmacokinetics of this drug after high-dose injections. The IOP-lowering effect caused by the natural absorption of the intravitreal triamcinolone could not be ruled out in our cases but seems unlikely, because triamcinolone crystals can be present in the vitreous and soluble triamcinolone can be detected in the aqueous humor for 9 months or longer after a single high-dose injection of IVTA, as previously described by Jonas et al. Additionally, 8 patients with secondary ocular hypertension (IOP range, 26-35 mm Hg; mean, 30 mm Hg) but without diagnosis of glaucoma at baseline, have been followed up since the high-dose IVTA for the eventual development of glaucomatous changes in the appearance of the optic nerve head. For 12 months after IVTA, triamcinolone crystals were still detectable in the vitreous cavity, the IOP was still elevated despite maximally tolerable antiglaucomatous topical treatment (range, 25-32 mm Hg; mean, 29 mm Hg), but the cup-disc ratio was apparently unaffected in all the patients. This small control group suggests that all patients with IOP elevation despite maximal medical therapy after high-dose IVTA need to be strictly observed; in some cases the drug effect seems to persist for more than 1 year. This long intraocular presence of triamcinolone may not only lead to secondary hypertension but also to steroid-induced secondary open-angle glaucoma necessitating more invasive laser or surgical procedures. The advantages of argon laser trabeculoplasty are numerous when considering the possible hazards of trabeculectomy, including anesthesia, hypotony, cataract, and endophthalmitis. It is also a time-efficient and cost-effective procedure when compared with filtering surgery. The clinical course of these patients suggests that argon laser trabeculoplasty may be considered as a primary option for secondary glaucoma following IVTA if medical therapy is unsuccessful. However, longer follow-up with a larger number of treated patients will be needed to assess whether argon laser trabeculoplasty permanently reduces the IOP, and if other medications could eventually be stopped as the steroid effect lessens.

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Lens capsular rupture has been reported to be traumatic or spontaneous. Traumatic capsular ruptures can occur from penetrating,1 surgical,2,3 or blunt trauma.4 Spontaneous capsular rupture has been described after cataract extraction in the fellow eye2 and associated with hypermature cataracts.5 To our knowledge, there have been no reports of spontaneous anterior capsular rupture causing lens-induced uveitis in the setting of anterior polar cataracts. We report the case of a 59-year-old woman with long-standing anterior polar cataract who developed spontaneous capsular rupture and lens-induced uveitis.

**Report of a Case.** A 59-year-old white woman complained of recurrent episodes of pain and photophobia in the left eye. The last episode was 1 year prior to initial examination, during which time she was diagnosed as having anterior uveitis and treated with topical steroids. Her ocular history was notable for congenital polar cataract diagnosed early in childhood and amblyopia in the affected left eye. She had no history of trauma or ocular surgery. Her medical history included controlled type 2 diabetes mellitus and hypertension but was otherwise unremarkable.

**Spontaneous Anterior Capsular Rupture Associated With Anterior Polar Cataract**

When seen at the University of Texas Health Science Center at San Antonio Cornea and Uveitis Service, her best-corrected visual acuity was 20/80 OS. Ophthalmic examination revealed a white mass protruding on the anterior lens capsule adjacent to the anterior polar cataract (Figures 1, 2, and 3). The remainder of the lens showed 1 to 2+ nuclear sclerosis. Flare and cells (2+) were present in the anterior chamber, but there was no fibrin or hypopyon. Guttata and pigment were present in the endothelium of both eyes. Uveitis evaluation was noncontributory.

Hourly 1% prednisolone acetate and once daily 1% atropine were prescribed. The anterior uveitis resolved in 26 days, and phacoemulsification without intraocular lens implantation was performed 5 weeks after initial examination. During surgery, the anterior capsule and anterior polar cataract were recovered. At the end of surgery, a sub-Tenon injection of 40 mg of triamcinolone acetonide was administered in the inferonasal quadrant.

The capsular specimen was fixed in 10% formaldehyde and stained with hematoxylin-eosin. The presence of a break in the anterior capsule, through which the cortical material was protruding, was confirmed histologically (Figure 4). Aside from a few isolated pigment-containing macrophages, no neutrophils, eosinophils, or other inflammatory cells were present at the site of the capsular break.

One month postoperatively, the eye continued to be quiet and topical steroids were tapered until administration was discontinued. Five months postoperatively, the patient obtained a visual acuity of 20/30 OS with a +8.50-diopter contact lens. At this time, both eyes were quiet during slitlamp examination.

**Comment.** Lens-induced uveitis has been recognized since 1919 by Straub7 and later by Verhoeff and Lemoine.8 The condition is traditionally explained by rupture of the lens capsule either by trauma or spontaneously; the latter is usually related to the presence of hyperma-

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