Viscocanalostomy for Refractory Glaucoma Secondary to Intravitreal Triamcinolone Acetonide Injection

Intravitreal injection of triamcinolone acetonide (IVTA) has gained widespread use in the treatment of a variety of neovascular and inflammatory intraocular conditions, including proliferative diabetic retinopathy, chronic cystoid macular edema, and chronic uveitis. Intraocular pressure (IOP) greater than 21 mm Hg occurs in approximately 42% of eyes following IVTA. Most of these patients are successfully treated with medical therapy, while a small percentage (1%) require glaucoma surgery for pressure management.1 We report a series of 3 patients who developed refractory glaucoma secondary to IVTA. All 3 were successfully treated with viscocanalostomy.

Report of Cases. Patient 1 was a 54-year-old man with a previous central retinal vein occlusion. Patient 2 was a 51-year-old woman with a history of a superotemporal branch retinal vein occlusion. Both had undergone IVTA for treatment of macular edema associated with the vein occlusions. Patient 3 was a 77-year-old woman who had received IVTA for exudative age-related macular degeneration. All 3 patients developed raised IOPs following a single IVTA (20 mg/0.1 mL) that were not controlled despite maximal medical therapy. Preoperative IOPs were 38 mm Hg, 40 mm Hg, and 40 mm Hg and preoperative visual acuities were 20/200, 20/125, and 20/40 for patients 1, 2, and 3, respectively.

All patients underwent viscocanalostomy by P.K.W. after informed consent was obtained. Details of the procedure in all cases are as follows. A superficial scleral flap measuring 5 mm × 5 mm × 250 µm was created with the base at the limbus (Figure 1). The corner of the scleral flap was elevated and dissected forward with a crescent blade kept parallel to the scleral surface advancing 2 mm into the clear cornea (Figure 2).

Figure 1. Angle structures including Schlemm’s canal (arrowhead). The white dotted line shows the superficial scleral canal.

Figure 2. Superficial scleral flap (5 mm × 5 mm × 250 µm) extending 2 mm into the clear cornea (arrowhead).
The lateral edges of the deep flap were then extended 1 mm into the clear cornea. As the deep flap was dissected forward, the posterior then anterior trabecular meshwork were exposed before the flap was extended into the clear cornea, stripping off 1 to 2 mm of the Descemet membrane, creating a trabeculo-descemetic window. The endothelial lining of Schlemm’s canal and the underlying juxtacanicular tissue were removed with a cellulose sponge until aqueous was seen seeping from the anterior chamber. The deep flap was excised with the roof of Schlemm’s canal (Figure 3).

An ophthalmic viscoelastic device (Viscoat, Alcon Laboratories Inc, Fort Worth, Texas) was injected into the deep scleral lake and toward the cut ends of Schlemm’s canal. Four 10.0 Vicryl sutures (Ethicon Inc, Somerville, New Jersey) closed the superficial scleral flap; the conjunctiva was also closed with 10.0 Vicryl sutures (Figure 4). Prednisolone, 1%, drops were used 4 times a day for 4 weeks followed by ketorolac, 0.5%, drops 4 times a day for 4 weeks. No perioperative or postoperative complications occurred, except for a small perforation of the trabeculo-descemetic window in patient 2. No antimetabolites, suture lysis, or postoperative needling were necessary.

Patients 1 and 3 had 6 months of follow-up and patient 2 completed 1 year. At 1 month, IOPs were 14 mm Hg in patient 1, 26 mm Hg in patient 2, and 10 mm Hg in patient 3. At 6 months postoperatively, IOPs were 10 mm Hg in patient 1, 16 mm Hg in patient 2, and 10 mm Hg in patient 3. Patient 2 had an IOP of 14 mm Hg at 1 year of follow-up. No antiglaucoma medication was necessary at any time postoperatively. Visual acuities at 6 months were 20/125, counting fingers, and 20/40 in patients 1, 2, and 3, respectively.

Comment. A few options have been suggested for the successful management of persistently elevated IOP following IVTA. These include trabeculectomy,1 laser trabeculoplasty,2 and pars plana vitrectomy.1

Viscocanalostomy has been shown to be effective in treating open-angle glaucoma with a reduced risk of the potentially sight-threatening complications, such as hypotony and endophthalmitis, compared with trabeculectomy.4 We report a series of 3 patients who were treated successfully with viscocanalostomy for refractory glaucoma secondary to IVTA.

Steroids induce glaucoma by lowering outflow facility through an unknown mechanism. Theories include the deposition of extracellular matrix in the trabecular meshwork.5 The complete success of viscocanalostomy in lowering IOP to acceptable levels without using antiglaucoma medication suggests that stripping the juxtacanalicular tissue and the inner waterproof endothelial lining of Schlemm’s canal relieves the steroid-induced resistance to aqueous outflow. We feel this case series illustrates that the obstruction to aqueous outflow in IVTA-induced secondary glaucoma may lie predominantly in the juxtacanalicular tissue and the endothelium of Schlemm’s canal. These structures

Figure 3. Excised deep scleral flap, including the roof of Schlemm’s canal (arrowhead), leaving the trabeculo-descemetic window (asterisk).

Figure 4. An ophthalmic viscoelastic device is used to form the intrascleral lake (arrow). The superficial flap is replaced and sutured.
We present 3 cases of endogenous *S apiospermum* endophthalmitis and histopathologic findings.

**Report of Cases. Case 1.** A 59-year-old woman with pre-B-cell acute lymphocytic leukemia developed neutropenia (absolute neutrophil count, <100 cells/µL) and blurred vision in her left eye. She received intravenous piperacillin sodium–tazobactam sodium, amphotericin B, vancomycin hydrochloride, acyclovir sodium, and sulfamethoxazole–trimethoprim. Her vision deteriorated, and skin lesions appeared on the right arm and left hip. Results of peripheral blood and cutaneous and pulmonary biopsy cultures were negative.

Visual acuity was 20/20 OD and 20/40 OS, with central scotoma and pain in the left eye. There was vitritis with preretal exudation and surrounding areas of focal retinitis and retinal hemorrhages (Figure 1A). Vitreous aspiration and intravitreal administration of amphotericin B (5 µg/0.05 mL) were performed in conjunction with intravenous voriconazole treatment (6 mg/kg). Four days later, 20 colonies of *S apiospermum* were identified, sensitive only to voriconazole (minimum inhibitory concentration [MIC], 0.5 µg/mL) and resistant to amphotericin B (MIC, >16 µg/mL), flucytosine (MIC, >64 µg/mL), and itraconazole (MIC, 2 µg/mL). The patient received 2 subsequent intravitreal injections of voriconazole (100 µg/0.1 mL), a vitrectomy with intravitreal voriconazole (150 µg), and 3 weekly intravitreal injections of voriconazole (100 µg). Cutaneous blood and lung cultures yielded *S apiospermum*, and voriconazole monotherapy was continued (4 mg/kg intravenously twice daily in the hospital and then 200 mg orally twice daily at home). Initial and repeated vitreous voriconazole levels (Fungus Testing Laboratory, University of Texas, San Antonio) were evaluated by high-performance liquid chromatography and exceeded the MIC (Table). The patient’s visual acuity progressed to no light perception with eye pain, and she chose to undergo enucleation. Only the initial ocular culture had positive findings; results of all subsequent cultures were negative.

Limited exenteration was performed because of gross scleral extension. Histopathologic examination demonstrated a disorganized and necrotic retina with inflammatory infiltrate and scleral thickening (Figure 1B). Morphologically normal-appearing *S apiospermum* organisms were identified (Figure 1C and D). The patient died 6 months later of overwhelming sepsis.

**Case 2.** A 37-year-old woman with pre-B-cell acute lymphocytic leukemia developed pain and redness in her left eye. She had profound neutropenia and systemic sepsis with pulmonary nodules. Visual acuity was 20/20 OD and 20/25 OS with conjunctival injection, chemosis, and decreased abduction in the left eye; the results of the remainder of the anterior and posterior examination were normal in both eyes. Magnetic resonance imaging showed nonspecific temporal, orbital soft-tissue enhancement. Intravenous vancomycin, imipenem, piperacillin sodium–tazobactam sodium, and liposomal amphotericin B treatment resulted in resolution of her ocular symptoms. Results of repeated magnetic resonance imaging were normal.

Fever and sepsis persisted. Intravenous voriconazole (4 mg/kg twice daily) and azithromycin were added. Four days later the patient developed dull pain in the left eye with decreased vision (visual acuity, 20/100). Examination showed conjunctival injection, vitritis, a yellow-white pre-retinal mass over the temporal macula, and a flocculent whitish vitreous mass in the inferotemporal macula. The papillomacular bundle demonstrated similarly colored yellow-white infiltrates with surrounding intraretinal hemorrhages.

The patient underwent vitreous aspiration and injection with vancomycin (1 mg/0.1 mL), cefazidime sodium (2 mg/0.1 mL), and amphotericin B (5 µg/0.05 mL), followed by vitrectomy and injection of vancomycin (1 mg), cefazidime sodium (2 mg), and voriconazole (100 µg). Four days later, vitreous cultures identified 4 colonies of *S apiospermum* sensitive only to voriconazole (MIC, 0.25 µg/mL) and resistant to amphotericin B (MIC, >16 µg/mL) and caspofungin acetate.

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**Endogenous Scedosporium apiospermum Endophthalmitis**

*Scedosporium apiospermum* is the asexual form of *Pseudallescheria boydii*, a ubiquitous saprophytic filamentous fungus.1 Neutropenia predisposes patients to infection with this organism.2 Endogenous endophthalmitis from *S apiospermum* is rare but grave sequela.3