

Intraoperative Management of Nonfunctioning Ahmed Glaucoma Valve Implant

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Abstract. A 62-year-old woman with neovascular glaucoma underwent filtering surgery with the Ahmed glaucoma valve (New World Medical, Inc., Rancho Cucamonga, CA). Intraoperative hypotony secondary to a defective valve was noted and managed by excising the valve mechanism. The implant was then treated as if it was non-valved by placement of an obturator suture. The postoperative course was unremarkable for a non-valved implant. This case demonstrates how intraoperative hypotony secondary to overfiltration of an Ahmed glaucoma valve implant can be successfully managed by converting to a non-valved implant. [Ophthalmic Surg Lasers Imaging 2006;37:62-64.]

INTRODUCTION

The Ahmed glaucoma valve (New World Medical, Inc., Rancho Cucamonga, CA) is an aqueous shunting device with a one-way valve designed to prevent postoperative hypotony. Since the mid-1990s, it has become a viable option in glaucoma surgery. The Ahmed glaucoma valve effectively lowers the intraocular pressure (IOP), increases the operative success rate, and preserves the visual function. Initial studies showed a

probability of success at 12 months similar to other drainage devices, whether valved or not.¹ Additionally, the Ahmed glaucoma valve may provide a predictable postoperative IOP.² We describe a case of a non-functioning valve resulting in excessive flow, forcing intraoperative modification of the Ahmed glaucoma valve into a non-valved device. A literature review using MEDLINE revealed no similar cases.

CASE REPORT

A 62-year-old woman with diabetes mellitus for 25 years complained of pain increasing for 1 month in the right eye. The best-corrected visual acuity was 20/100 in the right eye and 20/30 in the left eye. The IOP measured by Goldmann applanation tonometry was 52 mm Hg in the right eye and 10 mm Hg in the left eye. Examination of the right eye showed conjunctival injection, microcystic corneal edema, 3+ cells with flare in the anterior chamber, iris neovascularization, and a 2+ nuclear sclerotic cataract. Gonioscopy demonstrated peripheral anterior synechiae and neovascularization of the angle. Corneal edema obscured the fundus. The left eye was significant for neovascularization of the optic disc.

The IOP of the right eye was lowered to 40 mm Hg with topical and oral medications. Indirect panretinal photocoagulation was performed. Three days later, the visual acuity was 20/50 and the IOP was 21 mm Hg.

The patient underwent anterior chamber Ahmed glaucoma valve (Model S-2) surgery using a technique similar to that described by Coleman et al.,¹ including irrigation of the tube with balanced salt solution to open the valve. Before placing the scleral patch graft, the anterior chamber was uncharacteristically flat and re-formation was unsuccessful. The tube's limbal entrance wound and the paracentesis were watertight. The extraocular tubing had no perforations. The only locus of flow was through the valve. Retrograde placement of a 3-0 prolene obturator suture into the tube lumen required unroofing of the valve and a window cut in the elastomer membrane (Figs. 1 and 2), as re-

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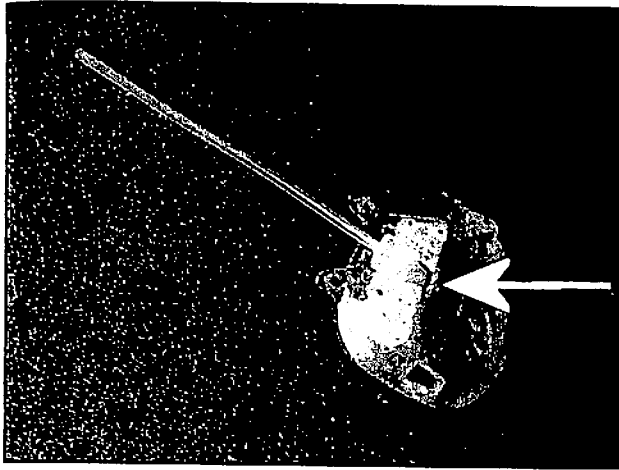


Figure 1. Ahmed shunt with cap removed and window cut in flow restrictor. Arrow points to removed window.

ported by Feldman et al. for non-opening valves.³ The tube was then ligated with 10-0 nylon suture to further obstruct flow. The anterior chamber retained its form when filled with balanced salt solution. The case was completed uneventfully in the standard fashion.

On the first postoperative day, the IOP was 10 mm Hg. Five weeks postoperatively, the IOP was 32 mm Hg. The 3-0 prolene obturator was removed, and the IOP dropped to 18 mm Hg. One month later, the pressure was 20 mm Hg without medications.

DISCUSSION

The Ahmed glaucoma valve implant was designed to minimize postoperative hypotony by adding a valve mechanism to an aqueous shunting device to maintain a more predictable IOP. Moreover, the tube does not require occlusion with a ligature or obturator, thus simplifying the initial surgery and eliminating the need for an additional procedure to remove the obturator.

In vitro, Eisenberg et al. calculated a mean opening pressure of 13.65 mm Hg, a facility flow of 1.2 $\mu\text{L}/\text{min}/\text{mm Hg}$, and a closing pressure of 6.1 mm Hg.² The IOP should therefore be maintained between the opening and closing pressures, without any incidence of overfiltration. However, Prata et al. suggested that the Ahmed Glaucoma Valve elastomer membrane is a flow regulator rather than a true valve because it does not fully close once perfused.⁴

Although a review of MEDLINE revealed no reports identical to this case, a few instances of postoperative hypotony with the Ahmed glaucoma valve have been reported. Of the 60 eyes studied in Coleman et

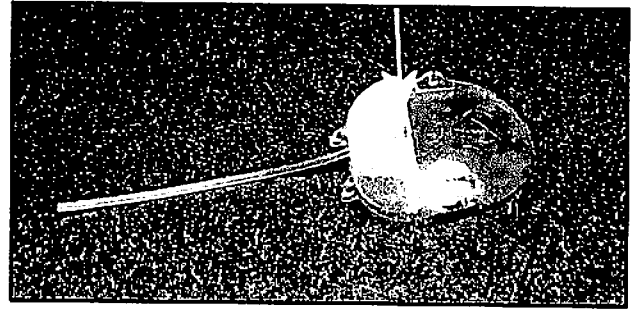


Figure 2. Ahmed shunt after removing window and replacing cap. Arrow points to removed window.

al.'s¹ initial study, 2 required re-formation of a shallow anterior chamber on the first postoperative day, ascribed to leakage around the tube. Similarly, Garcia-Feijoo et al.⁵ reported a case of peritubular filtration causing postoperative hypotony. Kee⁶ reported that partial ligation of the tube when implanting an Ahmed glaucoma valve lowered the rate of postoperative hypotony and surgical complications. However, this did not affect the final success rate.

Theoretically, overfiltration should not occur with the Ahmed glaucoma valve. However, if it occurs, as in this case, the Ahmed glaucoma valve behaves like a non-valved shunting device. Before implanting the Ahmed glaucoma valve, the surgeon must "prime the valve" by injecting balanced salt solution into the silicone tube. This will free adhesions that may be present between the two flaps of the elastomer membrane. On the other hand, if one flap of the membrane is adherent to the roof or floor of the valve, the flow may proceed unrestricted. By making intraoperative modifications, the defective Ahmed glaucoma valve can be successfully converted to a more predictable non-valved implant. The elastomer membrane can be accessed by carefully removing the roof of the valve. A small window can be excised in the portion of the membrane that overlies the posterior end of the tube (Fig. 1). The roof should then be replaced to inhibit fibroblast proliferation into the valve and tube (Fig. 2). Normal methods of occlusion for non-valved implants are then possible and will prevent postoperative hypotony.

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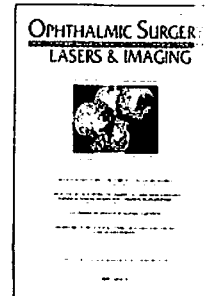
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