Vitreous Occlusion of Ahmed Glaucoma Tube in a Patient with Aphakic Glaucoma - A Case Report

Hina Qazi, Haroon Tayyab and Pir Salim Mahar

Department of Ophthalmology and Visual Sciences, The Aga Khan University Hospital, Karachi, Pakistan

ABSTRACT

This report presents an infrequent but well-recognised case of delayed Ahmed glaucoma valve tube obstruction caused by a vitreous plug in the anterior chamber of a 51-year Pakistani male with aphakic secondary open-angle glaucoma. The obstruction was successfully managed through an anterior vitrectomy and manual removal of the incarcerated vitreous plug *via* a pars-plana approach. This case highlights the importance of early recognition and appropriate intervention for vitreous-related Ahmed Glaucoma valve obstructions to ensure optimal intraocular pressure control in patients with aphakic glaucoma. This case contributes to the understanding of the complexities of managing Ahmed Glaucoma valve complications in aphakic patients, emphasising the need for tailored surgical interventions to prevent recurrent obstruction.

Key Words: *Ahmed glaucoma tube, Aphakic glaucoma, Glaucoma, Vitreous occlusion.*

How to cite this article: Qazi H, Tayyab H, Mahar PS. Vitreous Occlusion of Ahmed Glaucoma Tube in a Patient with Aphakic Glaucoma - A Case Report. *JCPSP Case Rep* 2025; **3**:299-302.

INTRODUCTION

The implantation of aqueous drainage devices for managing uncontrolled intraocular pressures (IOPs) in glaucomatous eyes has been increasingly utilised, especially for refractory cases.[1] A known complication following the implantation of a glaucoma drainage device is tube occlusion, which can result in decreased drainage and persistently elevated IOP postoperatively. Various substances, such as iris tissue, vitreous, blood, fibrin, and lens capsules, have been reported to cause tube obstruction.[2] In this report, the authors present a case of an Ahmed glaucoma valve (AGV) occlusion by incarcerated vitreous, which was successfully managed by pars-plana vitrectomy (PPV).

CASE REPORT

A 51-year male patient from Pakistan with a history of bilateral aphakia and a recent diagnosis of glaucoma presented for a second opinion due to progressive vision loss over the past two months, primarily affecting his left eye (OS). He was previously on dorzolamide 2% and timolol 0.5% twice daily, as well as travoprost 0.004% at night. Written informed consent was obtained from the patient, and the study received exemption from the Ethical Review Committee of the institute.

Correspondence to: Dr. Pir Salim Mahar, Department of Ophthalmology and Visual Sciences, The Aga Khan University Hospital, Karachi, Pakistan
E-mail: salim.mahar@aku.edu

Received: February 03, 2025; Revised: April 18, 2025; Accepted: May 02, 2025
DOI: https://doi.org/10.29271/jcpspcr.2025.299

Upon examination, his IOP was elevated to 50 mmHg bilaterally. His visual acuity (VA) was measured at 20/30 in the right eye (OD) and counting fingers at two feet OS. Ocular examination revealed a conjunctival injection, reactive but mid-dilated pupils, and aphakia in both eyes, with a history of lens extraction in early childhood. The anterior chamber (AC) was clear without any visible vitreous. Gonioscopy showed open angles (grade 2 on Schaffer's scale), while central corneal thickness measured 596 µm OD and 590 µm OS. A dilated fundus examination revealed a cup-to-disc ratio of 0.8 OD and 0.9 OS, with marked retinal nerve fibre layer (RNFL) loss as evident on optical coherence tomography (OCT) imaging.

He was confirmed with the diagnosis of bilateral aphakic glaucoma (secondary open-angle glaucoma) and was prescribed latanoprost 0.005%, brimonidine 0.2%, and dorzolamide 2% with timolol 0.5%. A week later, his IOP was 28 mmHg in OD and 18 mmHg in OS. Given the persistent IOP above the normal range, AGV implantation was recommended for the OD.

The patient received AGV implantation in the OD under general anaesthesia. A superotemporal conjunctival peritomy was performed, and the Tenon's capsule was dissected. Mitomycin-C 0.02% (0.1 ml) was injected into the subtenon space, and the scleral bed was irrigated with balanced salt solution. The AGV plate (Model FP7) was secured to the sclera with 8-0 Vicryl, 8 mm posterior to the limbus in the superotemporal quadrant. A partial-thickness scleral flap was created 3 mm behind the limbus, following the eye's curvature. Using a 23-gauge needle, a tunnel was formed from the scleral flap into the AC, which was maintained with 1% sodium hyaluronate. The AGV tube was passed into the AC, with its tip bevelled up. The conjunctiva and Tenon's capsules were repositioned and sutured over the device. A pars-plana or anterior vitrectomy

was not performed because no vitreous strands were visible in the AC, and the posterior capsule remnants laid behind the pupillary plane. Postoperatively, the patient was prescribed topical antibiotics and corticosteroids according to the hospital protocol.

On the first postoperative day, the AGV tube was well-positioned superotemporally in the AC, without hyphema, wound leak, or positive Seidel test. His IOP measured 20 mmHg and remained under 10 mmHg at subsequent follow-ups. A month later, a vitreous strand was observed touching the tube's internal ostium, though the IOP remained stable at 13 mmHg. Ten days thereafter, their IOP OD increased to 32 mmHg, with vitreous obstructing the tube (Figure 1). Nd laser vitreolysis (4-5 shots at 3.5 mJ) was attempted to disrupt the obstruction but failed to lower IOP. The patient was continued on dorzolamide 2% and timolol 0.5% and was referred for surgical management.

The patient underwent PPV using a 25-gauge system. The visualisation of the AGV tube confirmed vitreous in the lumen, which was confirmed with scleral depression and triamcinolone acetonide to identify residual vitreous. In the first instance, vitrectomy was carried out to remove excess vitreous and to provide access to the AGV tube and the vitreous plug. End-grasping forceps were employed to extract the dense vitreous plug located near the proximal tip of the tube, thereby minimising the traction applied. Additional debulking and delicate trimming of the vitreous base were conducted to ensure tube patency. A 29-gauge cannula flushed trypan blue dye into the tube lumen through a vitrectomy port to confirm clearance and tube patency.

On the first day after vitrectomy, the patient's IOP decreased to 10 mmHg, with a formed AC and no residual vitreous (Figure 2). One week later, the IOP was stable at 12 mmHg, and the AGV tube remained clear. The patient continued dorzolamide 2% and timolol 0.5% twice daily in OD, maintaining an IOP of 10 mmHg at the last follow-up.

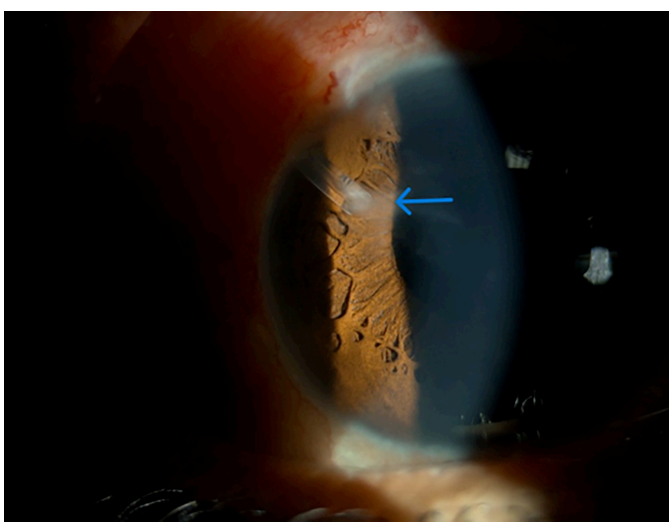

Figure 1: Vitreous strand in the AC being sucked in the AGV tube (arrow).

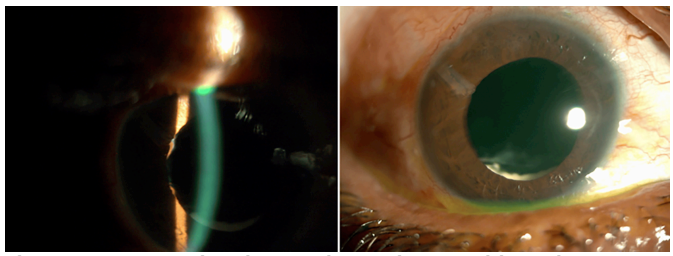

Figure 2: Postoperative pictures show a clear AC with no vitreous post vitrectomy.

DISCUSSION

Aphakic glaucoma secondary to congenital cataract surgery presents a significant clinical challenge, often requiring surgical intervention in approximately 50% of cases. The exact mechanism underlying glaucoma in aphakic eyes remains unclear; however, it is thought to involve intricate mechanical and biochemical alterations affecting both the vitreous and the structures of the anterior segment.[3] These cases frequently require multiple surgeries, with glaucoma onset reported anywhere from 0.3 to 94 months after initial cataract surgery.[3] Moreover, the likelihood of developing aphakic glaucoma increases with longer follow-up periods. Studies have shown that in patients followed for 5 years post-surgery, glaucoma incidence can reach up to 12 to 17%.[4]

Managing aphakic glaucoma remains challenging, with various treatment modalities employed over time. Medical therapies, including prostaglandin analogues, beta-blockers, carbonic anhydrase inhibitors, and miotics, are commonly used but may not always provide adequate IOP control.[3] Trabeculectomy with mitomycin-C, although more physiological, shows poor long-term success in aphakic eyes (≈25 % at 8.6 years);[5] therefore, most surgeons now favour an Ahmed or similar drainage device as the initial procedure when maximal medical therapy fails. In this case, the present patient was on maximum medical therapy, yet his IOP remained elevated. Previous studies suggest suboptimal outcomes for trabeculectomy combined with Mitomycin C in aphakic glaucoma.[6] In one large cohort, the success rate was only 25% over an average follow-up of 8.6 years.[7] Given these findings, glaucoma drainage device (GDD) implantation with Mitomycin C appears to be a more effective option, leading to the choice of the AGV implant in this case. The AGV functions on Bernoulli's principle, where a pressure differential between the AC and the plate facilitates aqueous flow through the tube.[8]

In aphakic eyes, reduced IOP can result in decompression, creating a pressure differential across the AC and vitreous cavity. This pressure differential can drive vitreous forward into the AC, potentially obstructing the GDD tube and raising IOP. This initiates a cycle where elevated IOP leads to further vitreous incarceration, dehydration, and condensation within the tube, creating a vitreous plug that penetrates deep into the lumen of the tube. In such cases, as reported by Vinod *et al*., end-grasping forceps are effective for extracting the vitreous plug.[9]

Vitreous obstruction of the AGV tube is typically observed within days postoperatively; however, in the present case, the obstruction was delayed, which is less commonly reported. Although factors such as eye rubbing can contribute to delayed obstruction, the patient denied any such activities, leaving the cause of the delayed obstruction uncertain. Cases of AC-placed AGV tube occlusion by vitreous are reported. Published series report an incidence of 4.7–10% depending on tube position, underscoring that vitreous blockage is a recognised, though infrequent complication.[10,11] Vitreous can migrate into the AC in aphakic or pseudophakic patients with posterior capsular defects or in a phakic patients with zonular dehiscence. When there are significant capsular defects not shielded by the surrounding iris or intraocular lens (IOL), it is advisable to perform PPV during the placement of the AGV. In this case, since the vitreous was situated behind the pupillary plane, anterior vitrectomy did not initially appear necessary.

Several options have been explored to clear vitreous obstructions in AGV tubes. Gu *et al*. reported success in dislodging vitreous obstruction in AGV tubes using an Nd laser.[12] Given its non-invasive nature, we attempted Nd vitreolysis first, but it failed to remove the vitreous obstruction. Consequently, an anterior vitrectomy *via* pars-plana was performed, which successfully reduced IOP to 12 mmHg. It could be argued that this complication might have been prevented by performing a thorough anterior vitrectomy or PPV during the initial AGV implantation. Desatnik *et al*. noted cases of vitreous entrapment in GDD tubes despite prior anterior vitrectomy in six of eight eyes, concluding that anterior vitrectomy alone may not suffice to prevent vitreous obstruction if the implant is in the vitreous cavity or if vitreous is present in the AC at the time of implantation.[13] PPV combined with GDD might increase the risk of posterior segment complications, such as retinal detachment, epiretinal membrane formation, and cystoid macular oedema. In the present case, PPV was not initially performed because the patient had no vitreous in the AC and was only-eyed. We aimed to minimise the risk of retinal detachment.

Vitreous-induced obstruction of an Ahmed valve tube, while uncommon, is a recognised complication in aphakic glaucoma. This case is notable for the delayed onset of tube obstruction by incarcerated vitreous, which is an unusual presentation. Successful management was achieved through anterior vitrectomy and the manual removal of the intraluminal vitreous plug, effectively restoring the patient's IOP to 10 mmHg.

PATIENT'S CONSENT:

Informed consent was obtained from the patient for the publication of data concerning this case.

COMPETING INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

HQ: Assisted with data collection, manuscript writing, editing, and critical revision of the manuscript.

HT, PSM: Involved in case management, surgical procedures, and critical revision of the manuscript.

PSM: Contributed to the conceptualisation of the study, manuscript drafting, and critical revision of the manuscript.

All authors approved the final version of the manuscript to be published.

REFERENCES

1. Arora KS, Robin AL, Corcoran KJ, Corcoran SL, Ramulu PY. Use of various glaucoma surgeries and procedures in medicare beneficiaries from 1994 to 2012. *Ophthalmology* 2015; **122(8)**:1615-24. doi: 10.1016/j.ophtha.2015.04.015.
2. Gupta S, Jeria S. A review on glaucoma drainage devices and its complications. *Cureus* 2022; **14(9)**:e29072. doi: 10.7759/cureus.29072.
3. Baris M, Biler ED, Yilmaz SG, Ates H, Uretmen O, Kose S. Treatment results in aphakic patients with glaucoma following congenital cataract surgery. *Int Ophthalmol* 2019; **39(1)**:11-9. doi: 10.1007/s10792-017-0777-y.
4. Freedman SF, Lynn MJ, Beck AD, Bothun ED, Orge FH, Lambert SR, *et al*. Glaucoma-related adverse events in the first 5 years after unilateral cataract removal in the infant aphakia treatment study. *JAMA Ophthalmol* 2015; **133(8)**:907-14. doi: 10.1001/jamaophthalmol.2015.1329.
5. Chen TC, Walton DS, Bhatia LS. Aphakic glaucoma after congenital cataract surgery. *Arch Ophthalmol* 2004; **122(12)**:1819-25. doi: 10.1001/archopht.122.12.1819.
6. Simons AS, Casteels I, Grigg J, Stalmans I, Vandewalle E, Lemmens S. Management of childhood glaucoma following cataract surgery. *J Clin Med* 2022; **11(4)**:1041. doi: 10.3390/jcm11041041.
7. Chen TC, Walton DS, Bhatia LS. Aphakic glaucoma after congenital cataract surgery. *Arch Ophthalmol* 2004; **122(12)**:1819-25. doi: 10.1001/archopht.122.12.1819.
8. Arikan G, Gunenc U. Ahmed glaucoma valve implantation to reduce intraocular pressure: Updated perspectives. *Clin Ophthalmol* 2023; **17**:1833-45. doi: 10.2147/opth.S342721.
9. Vinod K, Panarelli JF, Gentile RC, Sidoti PA. Vitreous occlusion of a glaucoma drainage implant-surgical management. *J Glaucoma* 2017; **26(7)**:669-72. doi: 10.1097/ijg.0000000000000694.
10. Ayyala RS, Zurakowski D, Smith JA, Monshizadeh R, Netland PA, Richards DW, *et al*. A clinical study of the Ahmed glaucoma valve implant in advanced glaucoma. *Ophthalmol* 1998; **105(10)**:1968-76. doi: 10.1016/S0161-6420(98)91049-1.
11. Gu J, Zhang Y, Zhai J, Ou Z, Chen J. Nd:YAG laser for Ahmed tube shunt blockage in patients implanted with boston type I keratoprosthesis. *Ophthalmol Ther* 2019; **8(2)**:333-9. doi: 10.1007/s40123-019-0186-6
12. Gu J, Zhang Y, Zhai J, Ou Z, Chen J. Nd:YAG laser for Ahmed tube shunt blockage in patients implanted with boston type I keratoprosthesis. *Ophthalmol Ther* 2019; **8(2)**:333-9. doi: 10.1007/s40123-019-0186-6.

13. Desatnik HR, Foster RE, Rockwood EJ, Baerveldt G, Meyers SM, Lewis H. Management of glaucoma implants occluded by vitreous incarceration. *J Glaucoma* 2000; **9(4)**:311-6. doi: 10.1097/00061198-200008000-00005.