Contents lists available at ScienceDirect

American Journal of Ophthalmology Case Reports

Prompt vitrectomy for management of endophthalmitis in setting of unexposed glaucoma drainage implant

Tyler L. Junttila[a], Gareth MC. Lema[b], Sandra Fernando Sieminski[a,*]

[a] Ross Eye Institute, University at Buffalo, 1176 Main Street, Buffalo, NY, USA
[b] New York Eye and Ear Infirmary of Mount Sinai, 310 E. 14th Street, New York, NY, USA

ARTICLE INFO

ABSTRACT

Purpose: To highlight a rare case of fulminant endophthalmitis in the late post-operative stage after glaucoma drainage device implantation without evidence of device exposure, and to share the unique management that resulted in successful restoration of vision and intraocular pressure control.

Observations: Endophthalmitis after glaucoma drainage implantation (GDI) is a rare complication most often associated with exposure of the device. Management options are limited, but removal of GDI is a common approach in the setting of an exposed implant. Visual acuity outcomes are often significantly reduced despite adequate treatment. There is little in the existing literature about management of late-onset endophthalmitis in the setting of a GDI without implant exposure. Here we present such a case that was successfully managed by prompt pars plana vitrectomy and removal of tube from the anterior chamber with subsequent re-insertion and patch graft. Our case results in a restoration of baseline visual acuity and IOP control at 7 months follow up.

Conclusions and importance: Endophthalmitis occurring after GDI implantation is a challenging complication to manage. Many physicians resort to removal of device for treatment, and a majority would treat initially with intravitreal antibiotic injection of antibiotics rather than prompt pars plana vitrectomy. This article provides a different approach that avoids removal of the device.

1. Introduction

Endophthalmitis after glaucoma drainage implantation (GDI) is a rare complication most often associated with exposure of the device. While there are no definitive guidelines, many prefer to remove the GDI as a part of the treatment of endophthalmitis in the setting of an exposed implant. However, there is little in the existing literature about management of late-onset endophthalmitis in the setting of a GDI without implant exposure. Here we present such a case that was successfully managed by prompt pars plana vitrectomy and removal of tube from the anterior chamber with subsequent re-insertion and patch graft.

2. Case report

A 70-year-old female with uncontrolled primary open-angle glaucoma (POAG) with a history of cataract extraction with intraocular lens and Baerveldt glaucoma implant (BGI) (Abbott Laboratories, Inc. Abbott Park, IL, USA) placement in the left eye. Her pre-operative visual acuity was 20/20 and the IOP was 34 mmHg in the left eye on

brimonidine 0.2% BID and travaprost 0.004%. Her past medical history was significant for type 2 diabetes mellitus, obesity, hypertension, hyperlipidemia, asthma, and sleep apnea. The BGI was placed in the superotemporal quadrant using a fornix-based conjunctival flap and the tube was covered anteriorly with a corneal patch graft. A sub-conjunctival injection of mitomycin C (0.2 cc of 0.2 mg/mL strength) was placed over the plate to prevent the early scarring and conjunctival injection that occurred in the fellow eye after Baerveldt placement months earlier. The surgery was without complication. At the two-month post-operative visit her best-corrected visual acuity (BCVA) was 20/25 in the left eye with and IOP of 15 mmHg on no drops. She did, however, continue to take 250 mg daily of acetazolamide to control IOP in the fellow eye, as she exhibited prominent conjunctival injection with all topical glaucoma medications.

Seven months after this procedure she presented with a sudden, atraumatic onset of left eye pain, redness, and significant vision loss of 12 hours' duration. She reported having a preceding upper respiratory infection. On examination, her visual acuity in the left eye was hand motion at 1 foot and IOP was 35 mmHg. Her left eye showed significant conjunctival injection with chemosis and no clinical sign of tube

* Corresponding author. Ross Eye Institute, University at Buffalo, 1176 Main Street, Buffalo, NY, 14209, USA.
E-mail addresses: sandrafe@buffalo.edu, smfernando17@gmail.com (S.F. Sieminski).
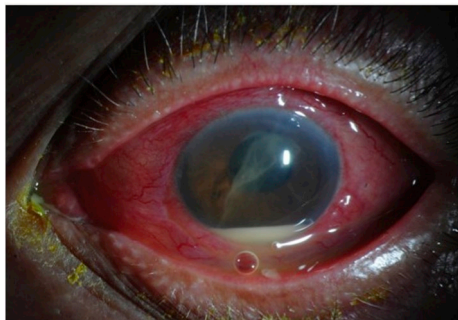
Fig. 1. Slit lamp photo of hypopyon and fibrin in anterior chamber at presentation.

exposure. There were 4 + white cells in the anterior chamber with a 1.5 mm inferior hypopyon and a fibrous pupillary membrane [Fig. 1]. B-scan ultrasonography showed cells in the anterior vitreous [Fig. 2].

Eight hours after presentation, the patient was evaluated by a retina specialist. Although vision was still hand motion at 1 foot, given the rapid presentation and severity of the AC reaction, the decision was made to perform emergent pars plana vitrectomy (PPV) with pre-operative IV moxifloxacin. In the operating room, the eyelids were noted to be more edematous, suggesting an orbital process. The cornea and conjunctiva were thoroughly inspected and noted to be without erosion. An anterior chamber washout with pupillary membrane excision was performed to improve the view to the posterior pole. A vitreous biopsy was taken and followed by full vitrectomy. The tube contained thick opaque fluid; whether this originated from the globe or the orbit could not be determined. The tube was removed from the anterior chamber and repositioned by coiling the open end posteriorly suturing it to the sclera to hold it in position. It was then covered by conjunctiva. Intravitreal injections of 1mg/0.1mL of vancomycin and 2.25mg/0.1mL of ceftazidime were given at the end of the case as well as an intra-operative dose of intravenous moxifloxacin. Given the worsening periorbital edema from presentation to the operating room hours later, a CT scan of the orbits was ordered post-operatively which did not show orbital cellulitis.

Immediately post-operatively the patient was given 400 mg PO moxifloxacin to use daily along with the following topical medication regimen in the left eye: prednisolone acetate 1% QID, polymyxin B sulfate-trimethoprim QID, dorzolamide 2%-timolol 0.5% BID, brimonidine 0.2% TID, and atropine 1% BID. Three days after the procedure, the microbiology results yielded pan-sensitive beta-lactamase negative *Haemophilus influenza*. Given the sensitivity results of the bacteria, moxifloxacin was discontinued and the patient was started on 200 mg cefpodoxime BID for 10 days. At the 1-week post-operative visit the best corrected visual acuity (BCVA) in the left eye was found to be 20/70

with an IOP of 21 mmHg via applanation.

At the two-month post-operative visit, the BCVA in the left eye was found to be 20/30 with an IOP of 33 via applanation. On examination, the tube had loosened from fixation posteriorly to the sclera, eroded through the conjunctiva, and was now laying uncoiled on top of the cornea. Her anterior chamber and vitreous was otherwise quiet. At the request of the patient, and against medical advice, surgery to revise the tube was delayed by two weeks. She was placed on polytrim QID, dorzolamide 2%-timolol 0.5% TID, brimonidine 0.2% TID, and prednisolone acetate 1% QID OS in the interim. During this tube revision, the tube was replaced into the anterior chamber under a both partial thickness scleral flap and overlying new corneal patch graft. There was some concern about epithelial downgrowth into the tube lumen, as well as potential inoculation of the tube with bacteria due to the two week exposure. To mitigate these factors, the tube was flushed vigorously with balanced salt solution on a cannula prior to reinsertion, and intracameral vancomycin and ceftazidime were administered at the end of the case. The 1-day post-operative visit showed a BCVA of 20/80 with IOP of 10 via applanation in the left eye. The tube was sufficiently covered and only trace anterior chamber cells were present. At the most recent visit 7 months status-post PPV and 5 months status-post tube revision, the uncorrected visual acuity was 20/20 and IOP of 13 by applanation in the absence of topical IOP-lowering medications.

3. Discussion

Endophthalmitis is an uncommon complication of GDI surgery with a reported incidence of 0%–2.2%.[2,3,7,8,13] The five year incidence of endophthalmitis in two major studies pertaining to GDI implantation, the Ahmed Baerveldt Comparison (ABC) Study and the Tube versus Trabeculectomy (TVT) study was relatively low: 0% in the Ahmed group and 2.2% in the Baerveldt group in the ABC study, and 1% in the TVT study.[7,8] Zheng et al. recently published a large retrospective study of 1,891eyes that undergone GDI from 2007 to 2014 and found 14 cases of endophthalmitis.[2] Similarly, a 9-year retrospective study found that 9 of 542 eyes had developed endophthalmitis after Ahmed glaucoma implantation.[3]

Endophthalmitis after GDI implantation is commonly associated with conjunctival erosion over the implant, likely owing to a decompensated barrier to pathogens. The incidence of erosion over GDI has been reported to occur at a rate of 0.09 ± 0.14% per month.[4] The ABC study reported a 5 year incidence of erosion of 1% and 3% in the Ahmed and Baerveldt groups, respectively, and the TVT reported a 5 year incidence of 5%.[7,8] However, other studies have reported a higher incidence of conjunctival erosion over GDI including Zhou et al. and Trubnik et al. at 7.52% and 8.3% respectively.[9,12] Erosion over the GDI associated with endopthalmitis was found to be present in the majority of cases by Zhang et al. and Al-Torbak et al.[2,3] Since conjunctival
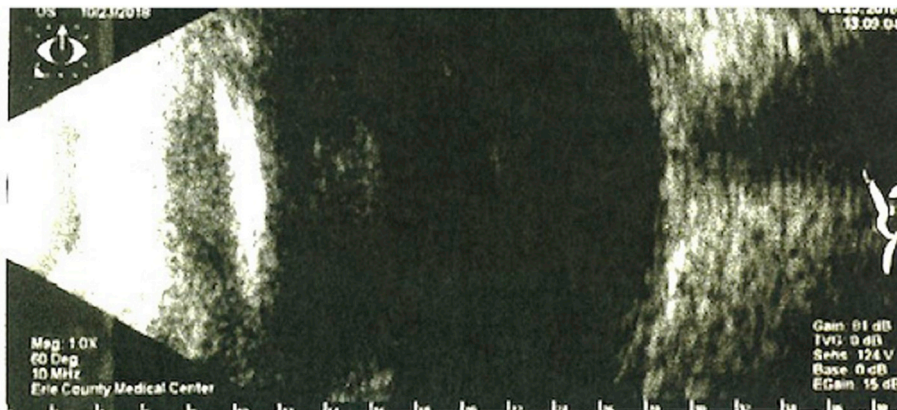

Fig. 2. B scan ultrasonography demonstrating cells in anterior vitreous at presentation.

erosion long been recognized as risk factor for endophthalmitis, patch graft is recommended to not only prevent tube exposure on initial implantation, but to prevent re-exposure upon repair.[2,3] While patch graft placement may help to prevent or delay erosion, it does not eliminate the risk. Zhang et al. noted that all 9 cases of endophthalmitis associated with erosion had prophylactic patch grafting at the time of GDI surgery.[2] While multiple studies have shown that the rate of conjunctival erosion is similar among different patch graft material, Zhou et al. identified aphakia, uveitic glaucoma, and longer post-operative topical steroid use as risk factors for erosions over Ahmed valve implants.[9,10,11]

Endophthalmitis after GDI placement occurring in the absence of conjunctival erosion is a rarer phenomenon. Zheng et al. noted that 5 of the 14 cases and Al-Torbak et al. noted 3 of 9 cases of endophthalmitis that did not involve conjunctival erosion.[2,3] Overall there is a paucity of information regarding endopthalmitis in setting of GDI without erosion. One case report documented such a situation, however it was confounded by transscleral cyclophotocoagulation which may have induced a microabrasion offering an alternative route of entry for atypical pathogens.[14] Sterile endophthalmitis has also been reported in the setting of a GDI possibly as a response to the composition of the GDI material.[15] However, in our case we document the absence of erosion in the setting of a positive intravitreal culture.

It has been shown that *Streptococcus* species are the most common offending microbe in cases of endophthalmitis after GDI surgery, however *Haemophilus influenzae* and *Staphylococcus* species have also been noted to occur but with less frequency.[2,3,5] Regardless of the approach to treatment, intravitreal antibiotics aimed at covering these microbes is recommended. While the Endophthalmitis Vitrectomy Study (EVS) group has guided management of endophthalmitis after cataract surgery, these results may not be directly applicable to other forms of endophthalmitis, namely as coagulase negative *Staphylococcus* was the most common offending organism after cataract surgery compared to more virulent organisms such as *Streptococcus* and *Haemophilus influenzae* which occur more frequently in the setting of GDI.[2,3,5,6]

Treatment of endophthalmitis associated with GDI implantation is quite varied in the literature. Several different approaches to treatment have been documented all of which include intravitreal antibiotics with or without vitrectomy and GDI removal.[1,2,3] Some have recommended removal of GDI in all cases over concerns that it may serve as a nidus of infection whereas others have reported successful outcomes while leaving the GDI in place.[1,16] Al-Torbak et al. noted no correlation between final visual acuity and whether or not the GDI was removed during treatment, although the sample size was small.[3] Some studies have noted better final BCVA in patients who have had a shorter duration of symptoms prior to presentation.[2,3] However, Francis et al. reported a poor outcome in a patient with a BGI and endophthalmitis despite aggressive treatment with vitrectomy, intravitreal antibiotics and removal of GDI within 8 hours of symptom onset.[17] At present there is a lack of sufficient data to conclude the best approach to treatment of endophthalmitis in the setting of GDI. Visual acuity outcomes are often significantly reduced despite adequate treatment. Of the 14 cases of endophthalmitis reported by Zheng et al., the BCVA had decreased (Snellen lines > 2) in 9 of 14 eyes compared to baseline with an average BCVA of 20/800.[2] Similarly, Al-Torbak et al. reported that none of the 9 eyes that had developed endophthalmitis after Ahmed valve implantation resulted in a BCVA better than 20/200 after treatment.[3]

In our patient, who presented with endophthalmitis in the setting of an unexposed GDI, and hand motion vision within 24 hours of symptom onset, a prompt PPV was chosen over tap and injection with a successful outcome. Subsequent PPV after tap and inject is sometimes necessary when clinical deterioration persists, which was the case in 3 of the 14 patients as documented by Zhang et al.[2] This was taken into consideration for our patient given the acuity and severity of symptoms in the setting of our patient's immunocompromised status from diabetes.

To our knowledge, this is the first report of a return to baseline visual acuity in the setting of endophthalmitis without erosion or GDI involvement after treatment with primary PPV. The mechanism of endophthalmitis in our patient is presumably via bacterial inoculation through imperceptible breaks in conjunctiva or perhaps passage across intact or thinned conjunctival tissue.

This case report also highlights the management of GDI in the setting of endophthalmitis, suggesting that device explantation may not be requisite, but tube removal may be beneficial. This is evidenced by the fact that our patient has been able to maintain target IOP goal independent of medications for up to 7 months follow up. Given the inevitable occurrence of conjunctival inflammation and scarring in a case of endophthalmitis, the fact that the GDI is maximally functional may be attributed to removing the tube from the anterior chamber.

While it would be ideal to have a randomized controlled trial to compare outcomes of tap and inject to PPV for endophthalmitis without tube exposure, it is unlikely that a sample size would ever be large enough to be statistically significant. As a result, we rely on case reports and clinical experience to guide decision making in such complicated cases.

4. Conclusion

Endophthalmitis with an unexposed tube is an uncommon complication after GDI surgery. Tube exposure is not pre-requisite to development of endophthalmitis, and in such cases bacteria such as *Streptococcus, Haemophilus influenzae,* and *Staphylococcus* species should be considered as likely pathogens. As was demonstrated here, endophthalmitis in the presence of GDI does not necessitate explantation of the device in all cases, but prompt vitrectomy should be considered, given the acuteness of symptom development.

Patient consent

The authors obtained the patient's full written consent to document the case and include clinical images.

References

1. Gedde SJ, Scott IU, Tabandeh H, et al. Late endophthalmitis associated with glaucoma drainage implants. *Ophthalmology*. 2001;108(7):1323–1327.
2. Zheng CX, Moster MR, Khan MA, et al. Infectious endophthalmitis after glaucoma drainage implant surgery: clinical features, microbial spectrum, and outcomes. *Retina*. 2017;37(6):1160–1167.
3. Al-Torbak AA, Al-Shahwan S, Al-Jadaan I, et al. Endophthalmitis associated with the Ahmed glaucoma valve implant. *Br J Ophthalmol*. 2005;89(4):454–458.
4. Stewart WC, Kristoffersen CJ, Demos CM, et al. Incidence of conjunctival exposure following drainage device implantation in patients with glaucoma. *Eur J Ophthalmol*.

2010;20(1):124–130.

5. Al Rashaed S, Arevalo F, Al Sulaiman S, et al. Endophthalmitis trends and outcomes following glaucoma surgery at a tertiary care hospital in Saudi Arabia. *J Glaucoma.* 2016;25(2):e70–e75.

6. Endophthalmitis Vitrectomy Study Group. Results of the Endophthalmitis Vitrectomy Study: a randomized trial of immediate vitrectomy and of intravenous antibiotics for the treatment of postoperative bacterial endophthalmitis. *Arch Ophthalmol.* 1995;113(12):1479–1496.

7. Budenz DL, Feuer WJ, Barton K, et al. Postoperative complications in the Ahmed Baerveldt comparison study during five years of follow-up. *Am J Ophthalmol.* 2016;163:75–82 e3.

8. Gedde SJ, Herndon LW, Brandt JD, et al. Postoperative complications in the tube versus trabeculectomy (TVT) study during five years of follow-up. *Am J Ophthalmol.* 2012;153(5):804–814 e1.

9. Zhou D, Zhou XY, Mas-Ramirez AM, et al. Factors associated with conjunctival erosions after Ahmed glaucoma valve implantation. *J Ophthalmic Vis Res.* 2018 Oct-Dec;13(4):411–418.

10. Levinson JD, Giangiacomo AL, Beck AD, et al. Glaucoma drainage devices: risk of exposure and infection. *Am J Ophthalmol.* 2015;160(3):516–521 e2.

11. Smith MF, Doyle JW, Ticrney Jr JW. A comparison of glaucoma drainage implant tube coverage. *J Glaucoma.* 2002;11(2):143–147.

12. Trubnik V, Zangalli C, Moster MR, et al. Evaluation of risk factors for glaucoma drainage device-related erosions: a retrospective case-control study. *J Glaucoma.* 2015;24(7):498–502.

13. Stein JD, Ruiz Jr D, Belsky D, Lee PP, Sloan FA. Longitudinal rates of postoperative adverse outcomes after glaucoma surgery among medicare beneficiaries 1994 to 2005. *Ophthalmology.* 2008;115(7):1109–1116.

14. AlHadlaq A, AlMalki S, AlShahwan S. Late onset endophthalmitis associated with unexposed glaucoma valved drainage device. *Saudi J Ophthalmol.* 2015;30(2):125–127. https://doi.org/10.1016/j.sjopt.2015.12.005.

15. Kim JH. Delayed sterile endophthalmitis after glaucoma drainage implantation. *Semin Ophthalmol.* 2011;26(4-5):290–294.

16. Ellis BD, Varley GA, Kalenak JW, et al. Bacterial endophthalmitis following cataract surgery in an eye with a preexisting Molteno implant. *Ophthalmic Surg.* 1993;24(2):117–118.

17. Francis BA, Diloreto DA, Chong LP, et al. Late onset bacterial endophthalmitis following glaucoma drainage implantation. *Ophthalmol Surg Lasers Imaging.* 2003;24(2):128–130.