Case Report

Spontaneous glaucoma drainage device extrusion after early postoperative orbital cellulitis – Case report and literature review

Abdulrahman AlDarrab a,b,⇑, Abdullah AlBahlal 1, Mohammed Dibaji a, Abdulrahman AlZaid c, Ibrahim AlJadaana, Sahar Elkhamary e, Silvana Schellinid

Abstract

A 70-year-old glaucoma patient who underwent Ahmed Glaucoma Valve (AGV) implantation developed orbital cellulitis (OC) 5 days postoperatively. On presentation, the valve was not exposed and no intraocular involvement was noted. After successful treatment of OC with intravenous and oral antibiotics, the patient presented to the clinic with the AGV completely extruded from the eye. There are only 10 previous cases of OC reported in the literature following glaucoma drainage device (GDD) implantation and no previous reports of spontaneous AVG extrusion. Close observation and thorough evaluation of the tube and plate positions are warranted to achieve better outcomes.

Keywords: Orbital cellulitis, Glaucoma, Glaucoma drainage device

Introduction

Orbital cellulitis (OC) is a rare sight threatening complication of glaucoma drainage device (GDD) implantation. Currently there are only 10 cases of postoperative OC after GDD implantation in the literature (Table 1).1–8 Additionally, there is only one report of spontaneous GDD extrusion9 which was documented with an Ex-PRESS shunt. There are no reports of spontaneous extrusion with the Ahmed Glaucoma Valve (AGV).

To the best of our knowledge, we are presenting the first case of early OC after AGV implantation that evolved to spontaneous AGV extrusion.

Case report

A 70-year-old male with bilateral primary angle closure glaucoma (PACG) presented for a routine visit at the glaucoma clinic at King Khaled Eye Specialist Hospital (KKESH), Riyadh, Saudi Arabia. The patient had a previous long-term history of bilateral trabeculectomy and cataract extraction. Two years prior to presentation that patient had undergone uneventful Ahmed Glaucoma Valve (AGV) implantation in the right eye (OD) and was stable.

At presentation, the Snellen visual acuity (VA) was 20/300 OD and 20/40 OS, intraocular pressure (IOP) was 13 mmHg OD and 23 mmHg OS with Goldmann applanation tonometry...
(GAT). The patient was using topical brimonidine tartrate 0.15% twice daily, combined dorzolamide and timolol maleate 0.5% twice daily and Travoprost 0.004% once daily at bedtime in both eyes. On slit lamp examination, he had mild conjunctival hyperemia bilaterally with flat blebs in the superior temporal aspect of the eyes, clear corneas, deep and quiet anterior chambers with the tube in place in the right eye. The pupil was round with a relative afferent pupillary defect, and the intraocular lenses were in place. Fundus examination indicated advanced cupping (0.9) and flat atrophic retina OU. Patient was hypertensive and blood pressure was controlled with medications, otherwise, his systemic history was unremarkable.

Given that the OS was the eye with good vision, yet had high IOP under maximal topical medication, a ligated AGV (FP7 model, New World Medical Inc., Rancho Cucamonga, CA) was implanted in the superior temporal quadrant OS under local anesthesia. The AGV was tightly sutured to the sclera with two interrupted #9-0 Prolene sutures and the tube was covered with Tutoplast pericardial patch. Mitomycin C was not applied. The procedure was uneventful and the immediate postoperative recovery was normal and drops were used to control the IOP due to the tube ligation.

Five days postoperatively, the patient presented to the Emergency Room complaining of pain OS and reduced VA for one day duration. On examination OS, the VA was 20/200, IOP was 35 mmHg, and the patient had swollen, tender upper and lower eyelids, with marked proptosis with limited ocular motility (~2) in all directions of gaze, conjunctival chemosis but no erosion or leakage over the tube and plate, corneal edema with epithelial micro-cysts, deep anterior chamber with small blood clot, micro-hyphema and tube in place (Fig. 1). The left pupil had a relative afferent pupillary defect that had been previously documented. Fundus examination OS indicated a flat retina and clear vitreous with no signs of inflammation. He received two tablets of acetazolamide (250 mg) and the IOP dropped after two hours to 25 mmHg. B-scan ultrasonography OS was unremarkable, with no intraocular involvement.

A non-contrast computed tomographic scan (CT scan) of brain and orbit confirmed the diagnosis of OC without intraocular involvement, revealing left periorbital soft tissue thickening extending to the medial canthus, associated with mild scleral thickening of the OS globe, as well as faint stranding of the surrounding intraconal fat with no evidence of sinusitis. Left extraocular muscles with mildly swollen, mostly notably the lateral rectus (Fig. 2A&B). Magnetic resonance imaging (MRI) of brain and orbits with contrast was done and revealed evidence of significant degree of thickening related to the episcleral area with evidence of enhancement related to the episcleral space and Tenon with extension in the proximal aspect of the optic nerve sheath on the left side (Fig. 2C&D).

The patient was admitted for medical treatment using topical moxifloxacin 0.5% four times daily OS and intravenous cefazoline sodium 1000 mg three times daily associated to intravenous gentamicin 80 mg three times daily and oral acetazolamide 250 mg three times daily to control the IOP.

### Table 1. Previous cases of postoperative Orbital cellulitis (OC) after glaucoma drainage device (GDD) implantation.

| Author            | Age     | Sex | Glaucoma type          | Glaucoma device | Time after surgery | Erosion over the valve | Endophthalmitis | Tube remained | Outcome                     |
|-------------------|---------|-----|------------------------|-----------------|--------------------|------------------------|------------------|---------------|-----------------------------|
| AlDarrab et al.   | 70 years| Male| PACG                   | Ahmed           | 5 days             | No                     | No               | No            | Device extrusion            |
| Beck et al.       | 53 years| Male| POAG                   | Baerveldt       | 3 months           | No                     | No               | Yes           |                             |
| Esporcatte et al. | 18 months| Male| PCG                    | Ahmed           | 1 month            | No                     | Yes              | No            | Phthisis bulbi              |
| Farid et al.      | 11 months| Female| Congenital glaucoma   | Ahmed           | 4 months           | No                     | Yes              | No            |                             |
| Kassam et al.     | 3 years | Male| POAG                   | Baerveldt 350 mm² | 3 months           | No                     | Yes              | No            | Cataract and retinal detachment |
| Chaudry et al.    | 11 years| Female| Congenital glaucoma   | Ahmed           | 5 months           | No                     | Yes              | No            |                             |
| Marcet et al.     | 44 years| Male| Secondary to uveitis   | Ahmed           | 2 days             | No                     | No               | No            |                             |
| Lavina et al.     | 18 years| Male| Traumatic CACG        | Ahmed           | 4 days             | No                     | No               | Yes           |                             |
| Karr et al.       | 1 year  | Male| Childhood glaucoma    | Molteno         | 1 month            | Yes                    | No               | No            |                             |

PACG; Primary angle closure glaucoma, POAG; Primary open angle glaucoma, PCG; Primary congenital glaucoma, CACG; Chronic angle closure glaucoma.

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The patient was admitted for medical treatment using topical moxifloxacin 0.5% four times daily OS and intravenous cefazoline sodium 1000 mg three times daily associated to intravenous gentamicin 80 mg three times daily and oral acetazolamide 250 mg three times daily to control the IOP.
Day one after admission, the patient felt better with reduction of eyelid swelling and decreased conjunctival chemosis and corneal edema. Two days after admission, the ocular motility continued to improve as well as the ocular examination. Five days after admission, the patient continued to improve and was discharged on the same topical medication regimen plus oral amoxicillin 875 mg and clavulanic acid 125 mg twice daily for a week instead of intravenous antibiotics. Five days after the discharge, he presented to the clinic and the ocular signs and symptoms had improved significantly and the IOP was well controlled. The patient was advised to continue the medication regimen.

Three weeks after discharge, the patient presented to the clinic with a complete extrusion of the AGV completely OS. There was no history of trauma or eye rubbing in the intervening three weeks. On examination OS, VA was 20/100, with mild conjunctival hyperemia at plate site superotemporally with no leak, clear cornea, deep and quiet anterior chamber with flat retina and clear vitreous. His IOP at that time was 7 mmHg. The patient was advised to decrease his topical glaucoma medications and we elected observation with very regular follow up visits.

Discussion

Our search of the English peer reviewed literature indicates the current case is the 11th case of OC following GDD implantation and the first case of spontaneous extrusion of the AGV.

The patient was 70 years of age but OC following GDD implantation can occur in children also. Previous reports of patients with OC after GDD indicated they underwent surgery to treat congenital glaucoma, glaucoma secondary to uveitis, primary open angle glaucoma or primary angle closure glaucoma, similar to our patient. Previous reports of OC had been documented with the Baerveldt, Krupin-Denver and Molteno implants.

Five days after AGV implantation, our patient developed OC but this complication was reported as soon as 2 days or as late as 15 months after the procedure in the literature.

Imaging studies (CT scan and MRI) confirmed our clinical diagnosis of OC showing inflammation around the plate, reaching the orbital fat and excluding the possibility of OC secondary to sinusitis, which is the primary cause of OC. Most likely, intraoperative contamination of the orbital tissues explains the OC in the current case. Other causes of OC include trauma to the periorbital area, endogenous systemic infection or secondary to endophthalmitis. However, the clinical evaluation and imaging studies excluded endophthalmitis in the current case.

Differential diagnosis includes orbital inflammatory syndrome, scleritis or myositis.

Alkheraiji et al., reported a case of necrotizing scleritis following Baerveldt tube implantation in a patient who presented later with decrease in vision, severe anterior chamber reaction, hypopyon, hyphema and conjunctival melting in which the tube was removed and scleral biopsy revealed Mycobacterium abscessus.

Myositis has been reported as well with the Baerveldt tube shunt.

Our patient had mainly lateral rectus involvement likely due to the proximity between the GDD and this extraocular muscle. However successful treatment with systemic antibiotics, likely indicates a bacterial infection rather than inflammatory process as the favorable cause.

At presentation our patient had no conjunctival erosion. Previous reports have documented only 3 of the 10 cases of OC after GDD presented with conjunctival erosion. Erosion of the conjunctiva and exposure of the GDD has been described in the early or late postoperative period, predisposing to endophthalmitis. Our patient had no concomitant endophthalmitis and only 3 cases of OC after GDD presented with endophthalmitis, all without GDD exposure. The outcome of OC when endophthalmitis is present is generally poor even with intensive care.

Our case was notable because complete extrusion of the AGV occurred after successful medical therapy for OC. We believe that early inflammation around the plate and the thickening of soft tissue including the thickening of the extraocular muscles and sclera that all subsided over time, played a major role in tissue fragility and loosening of the plate sutures changing the plate position until complete extrusion occurred.

Alternate explanations for extrusion include trauma or improper plate fixation secondary to compromised scleral resistance, low scleral rigidity originating from high myopia or insufficient viscoelastic material utilized in the anterior chamber.

The outcome of OC following GDD depends on the infection control, intraocular involvement and the stage of glau-
coma. After intensive treatment to control the infection, the patient can develop phthisis bulbi, cataract with retinal detachment, uncontrolled IOP, requiring diode cyclophotocoagulation and topical therapy. Although the area around the plate might be scarred secondary to the inflammation, it can still be partially functioning and can help in lowering the IOP.

In conclusion, we observed a glaucomatous patient who underwent GDD implantation and then developed the rare complication of postoperative OC. During the acute phase, the GDD was not exposed and intraocular inflammation was not present and a good outcome was achieved with medical management. However, he presented later with spontaneous GDD extrusion.

To the best of our knowledge, this is the first case in the literature of spontaneous GDD extrusion secondary to OC following GDD implantation. Close observation is required even after resolution of the OC episode and meticulous evaluation of the tube and plate position is imperative.

Conflict of interest

The authors declared that there is no conflict of interest.

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