Gluacoma drainage device complicated by fungal Paraconiothyrium endophthalmitis

Abstract

Ten years after Baerveldt™ implantation, a woman underwent immunosuppressive therapy for necrotizing scleritis. She subsequently developed symptoms suggestive of endophthalmitis in the setting of tube exposure. Cultures isolated fungal Paraconiothyrium. She underwent two vitrectomies with anterior chamber washouts, Baerveldt™ device removal, and intraocular and systemic antifungal therapy. The endophthalmitis has since resolved.

Keywords: Endophthalmitis; Eye Infections, Fungal; Mycoses; Glaucoma Drainage Implants; Antifungal Agents.

Introduction

Glaucoma drainage devices (GDD) are becoming increasingly popular in the surgical management of elevated intraocular pressure. GDD-associated endophthalmitis is relatively uncommon, with cumulative incidence rates ranging from 0.4% at one year to 6.4% at six years follow-up. These infections are almost entirely bacterial and are often associated with tube exposure.

We present a case of fungal endophthalmitis developing 10 years after GDD implantation.

Case Report

A 74-year-old woman with a history of primary open angle glaucoma had undergone Baerveldt™ glaucoma implantation (BGI) in 2005, penetrating keratoplasty (PKP), and posterior chamber intraocular lens implantation (PCIOL) in the right eye. A 350-mm2 BGI had been placed superotemporally and covered with a corneal patch graft.

Ten years later, she presented to an outside facility with pain and injection OD. She was diagnosed with necrotizing scleritis and started on topical and high dose oral prednisone. Rheumatologic history and a new rheumatologic work-up were negative. She was started on oral methotrexate and subsequently developed corneal infiltrates, subacute vision loss, and worsening pain. Topical moxifloxacin, voriconazole, and difluprednate were prescribed for presumed fungal keratitis.

Her infiltrates persisted 2.5 weeks later, and she was referred to our institute. At presentation, visual acuity (VA) was count fingers, and IOP was 8 mmHg. The BGI tube had eroded through a melted corneal patch graft and was exposed at the limbus. The PKP was edematous, and superior scleromalacia was seen without active inflammation. There were dense white infiltrates in the anterior chamber (AC), vitreous opacities, and white plaques on the PCIOL surface (Figure 1). B-scan ultrasound revealed dense vitreous infiltrates suggestive of fungal endophthalmitis.

The following day, she underwent pars plana vitrectomy (PPV), AC washout, and removal of the BGI and PCIOL. Intraoperatively, extensive infiltrates were visualized in the AC and posterior chamber that covered the underside of the iris and ciliary body. Intravitreal amphotericin B was injected on postoperative days 1 and 3. Ten injections of voriconazole were administered into the AC (unicameral eye). Surgical cultures grew the fungus Paraconiothyrium, which was sensitive to voriconazole, amphotericin, and fluconazole. Oral fluconazole and oral prednisone taper were started postoperatively, and her methotrexate was discontinued. One month later, she underwent AC washout, PPV, and silicone oil injection for persisting AC infiltrates.

At her most recent visit, two months after initial presentation, VA was hand motion, and IOP was soft, with a formed AC in a silicone oil filled eye. Exam showed resolution of AC fungal infiltrates, attached retina, but edematous corneal graft (Figure 2).

Discussion

Endophthalmitis is an uncommon complication of GDD implantation, with incidence rates for BGI ranging from 0.9% to 3%. Development of endophthalmitis may occur many years post-implantation. The risk of infection is significantly increased with GDD exposure, especially over inferiorly-placed implants.
Risk of erosion is increased without covering the tube with a patch graft, lack of coverage of tube shunt with eyelid, eye rubbing, and when extensive conjunctival scarring is present. During primary implantation, using a scleral patch graft and placing the tube superiorly may help to reduce the risk of erosion. Additionally, any sources of mechanical irritation should be managed, e.g., surface lubrication or tube repositioning. Any evidence of ocular infection, conjunctival erosion, implant extrusion, or tube exposure should prompt quick management.

Several microorganisms have been isolated in GDD-associated endophthalmitis, including Streptococcus, atypical Mycobacteria, Nocardia, and Bacillus, among others. Although exogenous fungal endophthalmitis is a known complication of intraocular surgery, our case of GDD-associated fungal endophthalmitis is highly unusual. A literature review revealed only one case of fungal endophthalmitis in a GDD eye. Furthermore, Paraconiothyrium spp. is an endophytic fungus, which has only been implicated in cutaneous lesions in immunocompromised patients. As this fungus is largely found in soil and vegetation, it is unclear how or where our patient’s infection originated, as she had no history of overt trauma. However, it is possible that some vegetative debris came into contact with her eye and was exacerbated by the combination of immunosuppressive therapy in the setting of an exposed seton.

Fungal endophthalmitis must be treated aggressively. In addition to topical, intraocular, and systemic antifungals, vitrectomy or PKP may be warranted. Given the scarcity of fungal cases, it is unclear whether treatment should include GDD removal. Unfortunately, even with aggressive treatment, the visual prognosis for exogenous fungal endophthalmitis is tenuous. As such, removal of the implant may be preferred.

References