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Delayed Suprachoroidal Hemorrhage during Anterior Chamber Decompression One Day following Tube Shunt Implantation

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Introduction

Suprachoroidal hemorrhage is a rare but potentially devastating condition that most often occurs during but can occur after intraocular surgery. Glaucoma surgery carries the highest rate of suprachoroidal hemorrhage, particularly delayed suprachoroidal hemorrhage.[1] Delayed suprachoroidal hemorrhage can occur hours, days, or even weeks after surgery and is generally characterized by the sudden onset of severe ocular pain, vision loss, and high intraocular pressure (IOP). Despite its potentially devastating visual consequences, delayed suprachoroidal hemorrhage remains a poorly understood condition, and its management remains challenging.

We present a case that had a good visual outcome following the development of a non-appositional delayed suprachoroidal hemorrhage that occurred while decompressing the anterior chamber on postoperative day one following glaucoma tube shunt implantation.

Case Report

An 86-year-old myopic female with advanced primary open angle glaucoma (POAG) underwent uncomplicated Baerveldt-350 tube shunt implantation with scleral patch graft placement in the left eye. A peribulbar block was administered along with monitored anesthesia care. Preoperative intraocular pressures were 15 mmHg in the right eye and 18 mmHg in the left eye. Preoperative intravenous mannitol was not administered. Intraoperatively, the tube was positioned in the supertemporal sulcus, and 1–2 venting slits were created using a super sharp blade to facilitate aqueous outflow. The surgery was uncomplicated but was notable for bleeding from the very friable conjunctiva during closure.

Past ocular history was notable for advanced uncontrolled POAG that was greater in the left eye, IOP of 21 mmHg despite maximally tolerated IOP lowering eye drop therapy (MTIT), and a central corneal thickness of 498 µm. Visual field constriction had progressed so that only central islands remained in both eyes. Very advanced optic nerve cupping was present in both eyes despite MTIT which included brimonidine-timolol twice daily, netarsudil 0.02% once daily, brinzolamide 1% every 8 hours, and latanoprostene bunod 0.024% nightly in both eyes. She had a history of cataract surgery with intraocular lens implantation in both eyes. She was otherwise systemically healthy, except for a tendency for skin hematomas without use of antithrombotics.

On postoperative day 1, uncorrected visual acuity was 20/100 in the left eye (20/20 preoperatively), with IOP of 26 mmHg, moderate depth anterior chamber, and a sulcus tube in good position. Due to the elevated IOP, the anterior chamber was decompressed at the slit lamp by applying direct pressure next to the previous paracentesis site using a cotton-tipped applicator, resulting in IOP reduction to 5 mmHg. The patient felt immediate severe pain during this procedure, and her IOP 15 minutes later spiked to 36 mmHg with visual acuity of hand motions, diffuse corneal edema, a well-formed anterior chamber. A temporal suprachoroidal hemorrhage involving the macula was noted.

Two rounds of dorzolamide hydrochloride 2% and brimonidine-timolol 0.2%-0.5% eye drops were placed in the left eye, and 500 mg acetaminophen given orally. Atropine 1% eye drops twice a day and acetazolamide 500 mg capsule every 12 hours were initiated, to resume her preoperative IOP-lowering eye drops as noted above, and continue her peri-operative eye drops of moxifloxacin 0.5%, prednisolone acetate 1%, and ketorolac 0.5% four times per day.

On postoperative day 3, the patient reported symptomatic improvement in pain. Visual acuity improved to 20/60, and IOP was 14 mmHg with only mild corneal haze and a deep anterior chamber. The tube was well-covered tube and no leaks were identified. Fundus examination revealed evidence of limited, non-appositional suprachoroidal hemorrhage involving the fovea, confirmed by B-scan ultrasonography (Figures 1, 2). Observation with continued medical management was suggested.

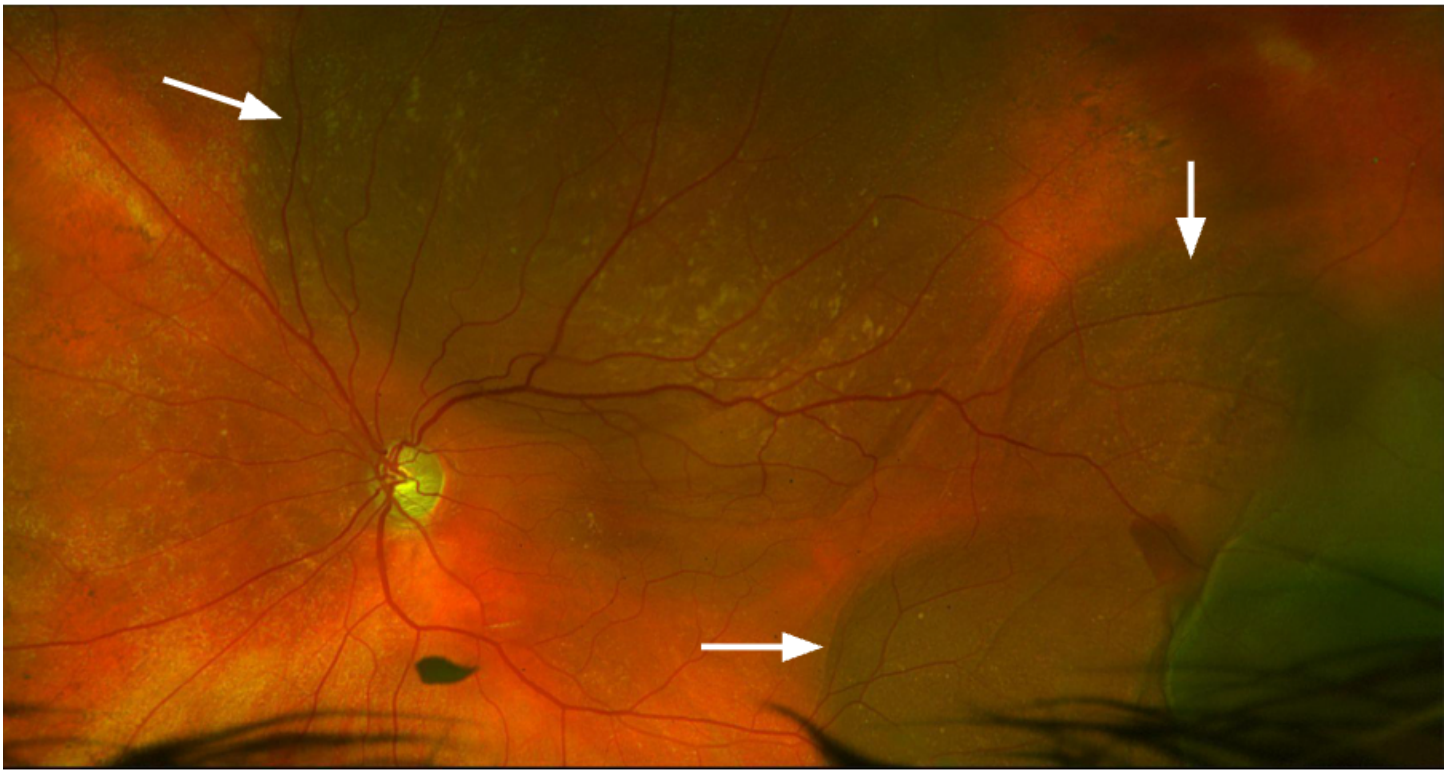

Figure 1. Ultra-widefield color fundus photograph (California, Optos) image of the left eye on postoperative day 3 with localized suprachoroidal hemorrhage involving the macula (white arrows) along with a small retinal

hemorrhage. The optic disc demonstrated cupping. Note the presence of a posterior vitreous detachment inferiorly.

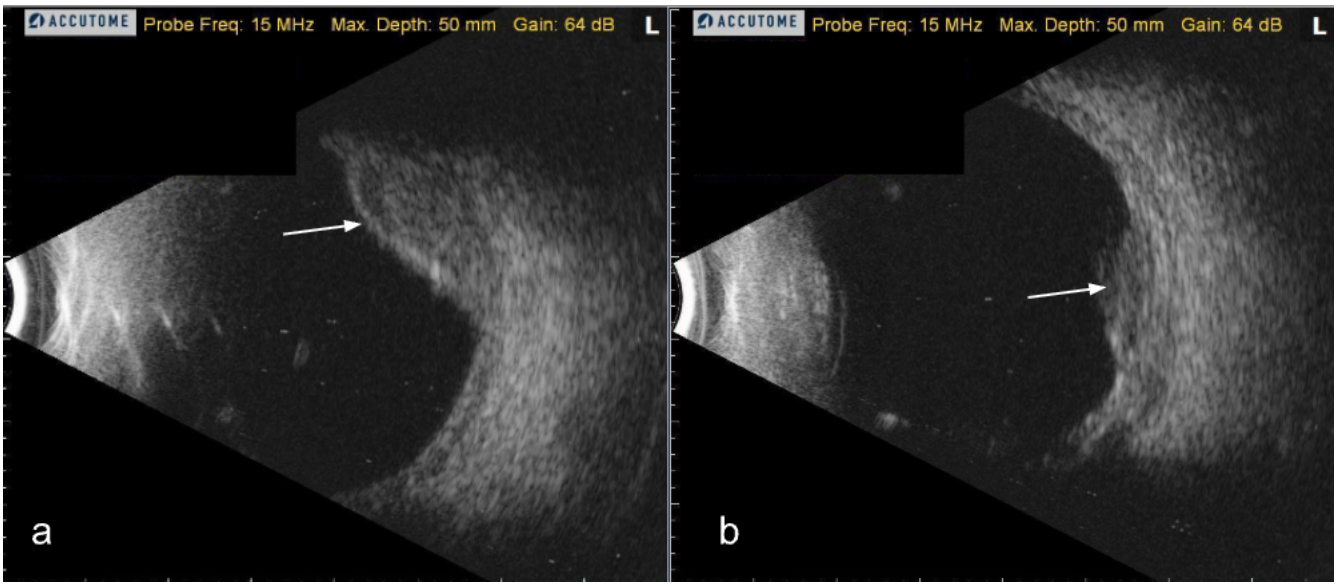

Figure 2 a-b. Ultrasound B-scan of the left eye on postoperative day 3 showed the limited, non-appositional suprachoroidal hemorrhage as dense with hyper-echogenicity (white arrows).

Over the next 4 weeks, there was continued improvement in vision to 20/30, and IOP ranged from 11-18 mmHg. Fundus exam showed improved suprachoroidal hemorrhage (Figure 3).

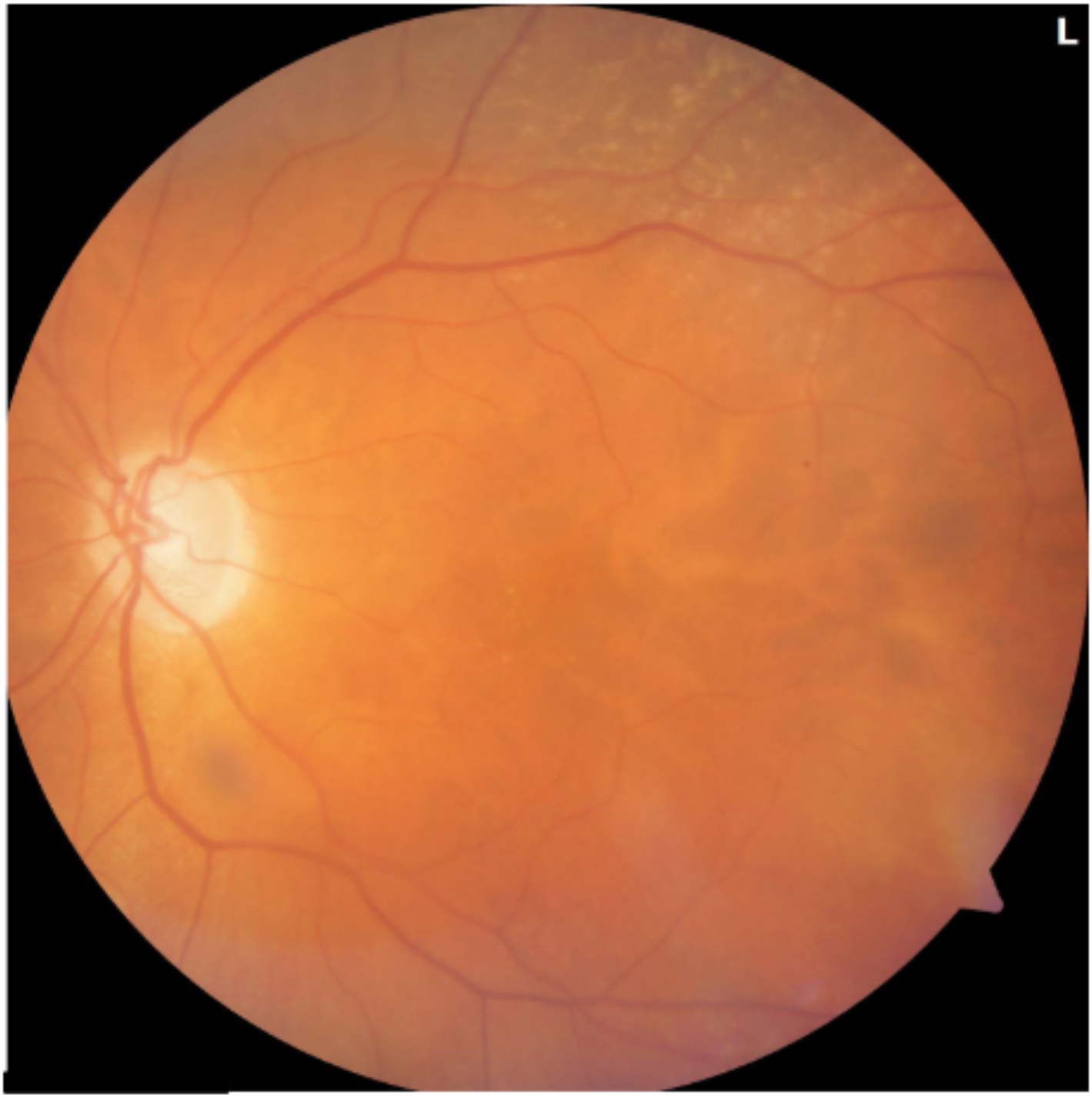

Figure 3. Fundus image of left eye on postoperative day 50 with significant improvement in suprachoroidal hemorrhage.

Discussion

Delayed suprachoroidal hemorrhage has been reported after various intraocular surgeries, with the highest incidence following glaucoma filtering procedures (0.6%–7%).[1,2] Notably, tube shunt implantation carries a higher risk (1.2%–2.7%) than trabeculectomy (0.6%–1.4%), possibly due to more marked IOP reduction in eyes

with elevated preoperative pressures.[1] Among tube types, non-valved implants manifest greater risk (7%) compared to valved implants (2.8%), with postoperative hypotony being a primary risk factor.[2]

This patient had several established risk factors: advanced age, higher preoperative IOP, myopia, and an abrupt IOP drop following globe decompression. Prior studies have similarly highlighted hypotony (IOP ≤3 mmHg), prior intraocular surgery, myopia, aphakia, and sudden IOP drops within 24 hours postoperatively as key risk factors.[2,3] Risk profiles vary by procedure type—hypotony predominates following tube implantation, while aphakia and anticoagulation are more strongly associated with delayed suprachoroidal hemorrhage after trabeculectomy. [2] Known systemic risk factors include advanced age, hypertension, anticoagulation, and cardiopulmonary disease—all of which may compromise choroidal vascular integrity.[2,4–6] Visual outcomes are known to depend on suprachoroidal hemorrhage severity, with poor prognostic indicators including prolonged appositional suprachoroidal hemorrhage, retinal or vitreous incarceration, or afferent pupillary defect at presentation.[7,8] Even so, only about 34% of cases achieve a final visual acuity of 20/200 or better.[9]

Management remains controversial. While some advocate for early drainage, especially in eyes with appositional suprachoroidal hemorrhage, others report no significant difference between surgical and conservative approaches—particularly in smaller, non-appositional cases.[4,10,11] In our case, the suprachoroidal hemorrhage was identified immediately, allowing prompt decision-making. Given the limited, non-appositional nature of the hemorrhage, observation was elected with care to avoid episodes of hypotony. This patient had complete resolution of suprachoroidal hemorrhage along with visual improvement.

Conclusion

Though rare, delayed suprachoroidal hemorrhage can cause permanent vision loss. Identifying systemic and ocular risk factors is critical to minimize risk. This case highlights the potential for suprachoroidal hemorrhage to develop following decompression of the anterior chamber soon after tube shunt surgery. Since the hemorrhagic choroidal detachment was non-appositional, a conservative approach with cycloplegics, IOP-lowering therapy, and avoidance of hypotony may lead to favorable outcomes even in high-risk patients.

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Statement of Ethics

This case series adheres to patient confidentiality and ethical principles in accordance with the guidelines of the Declaration of Helsinki and relevant local regulations.

Conflict of Interest Statement

The authors declare no conflicts of interest related to this topic.

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