

Case Reports

Perioperative spontaneous bilateral suprachoroidal hemorrhage

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Summary

Suprachoroidal hemorrhage is a rare condition, occurring most commonly in the perioperative period, although cases of unilateral spontaneous hemorrhages have been reported. We report a case of bilateral spontaneous suprachoroidal hemorrhage and discuss the potential causative factors.

Introduction

In suprachoroidal hemorrhage, bleeding occurs in the potential space between the choroid and the sclera.¹ The condition, which usually occurs perioperatively, is rare and usually carries a poor prognosis, perhaps due to atypical presentations and/or a delayed diagnosis. Known risk factors include intraocular surgery, anticoagulant therapy, cardiovascular disease, and advanced age. Unilateral spontaneous suprachoroidal hemorrhage has been described previously. We report a case of bilateral, spontaneous suprachoroidal hemorrhage, causing devastating blindness. To our knowledge, this is the first such case reported.

Case Report

A 76-year-old non-Hispanic white man underwent uneventful, small-incision phacoemulsification cataract surgery to his left eye under topical anesthesia. He was already pseudophakic in his right eye and had a past history of retinoschisis and hypermetropia (preoperative refraction +4.50 D in the right eye and +4.25 D in the left). Axial lengths measured 22.27 mm and 22.23 mm. His relevant medical history included recurrent deep-vein thromboses, atrial fibrillation, chronic obstructive pulmonary disease, treated essential hypertension, and depression. Among his current medications were paroxetine, warfarin, aminophylline, mebeverine, amiloride, furosemide, a salmeterol inhaler, and a tiotropium bromide inhaler. His international normalized ratio (INR) on the day of surgery was 2.0.

On the first postoperative day, uncorrected visual acuity was 20/60 in the right eye and 20/120 (20/60 with pin-hole) in his left eye. His examination was essentially unremarkable apart from his intraocular pressure (IOP), which measured 22 mm Hg in the right eye and 27 mm Hg in the left eye. Twice daily oral acetazolamide 250 mg for 3 days was prescribed.

On day six, he represented with symptoms and signs of bilateral acute-angle closure and was found to have an IOP of 58 mm Hg in the right eye and 57 mm Hg in the left eye. His vision had dropped to light perception in the right eye and hand movements in the left eye. Anterior segment examination demonstrated flat chambers with closed angles visible on gonioscopy. Fundus examination was difficult due to the corneal edema and fixed pupils.

A diagnosis of choroidal effusion secondary to a systemic precipitant was considered, likely because of the acute, bilateral, angle-closure-type presentation in a pseudophakic patient. He was treated with intravenous acetazolamide and subsequently mannitol, with no response. His paroxetine was withheld since this was the only one of his medications known to possibly cause angle closure glaucoma.²

On further questioning, it was discovered that the patient was being treated by his primary care physician for a chest infection and had received four days of oral erythromycin. He had developed a productive cough over this

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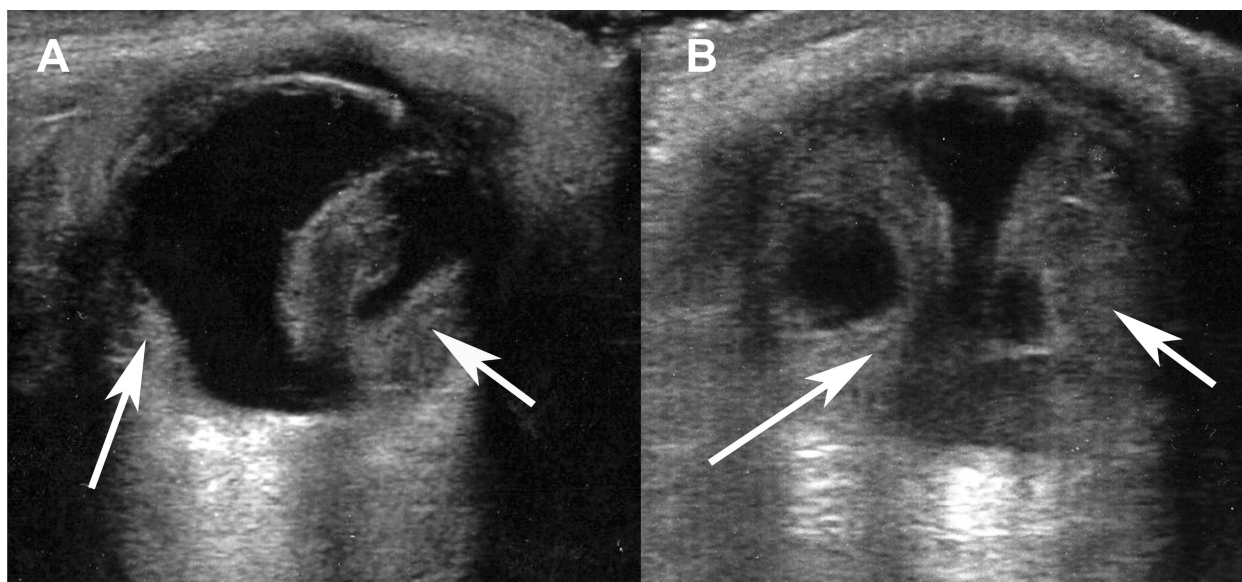


Figure 1. Transverse B-scan ultrasound of the right eye (A) and left (B) eye showing bilateral suprachoroidal hemorrhage (white arrows) with a serous component.

period but could not recollect a specific bout of coughing associated with the onset of his vision loss. His coagulation was rechecked and the INR was raised at 4.8. Warfarin was also withheld at this stage.

B-scan ultrasound was performed the following day (Figure 1). This revealed features consistent with bilateral suprachoroidal hemorrhage. Surgical intervention was not deemed possible due to his deteriorating respiratory function and his abnormally high INR (which peaked at 5.6). Management was therefore to treat his chest, normalize his INR, and continue topical therapy for raised IOP. He was admitted by the pulmonary physicians since he developed rigors and became septicemic.

His intraocular pressure returned to normal over the following week. Unfortunately, his vision deteriorated and remained at only light perception in both eyes. This was due to the persistently high initial IOP and the presence of extensive choroidal hemorrhages.

After 4 weeks he had recovered sufficiently for discharge. His INR had stabilized at 1.3 and warfarin was being reintroduced, due to the finding of anti-phospholipid antibodies. At this time, his vision remained perception of light only. This was due to break-through bleeding from the choroidal hemorrhages causing hyphema, in eyes that had also become hypotonus (IOP <4 mm Hg bilaterally).

Discussion

This case raises several issues, the first being diagnostic. Acute-angle closure in a pseudophakic patient is an atypical presentation, and when presenting bilaterally, pharmacological or systemic causes should be considered. The authors were therefore initially suspicious of a drug-related side-effect causing choroidal effusions as opposed to a direct postsurgical insult, especially since bilateral suprachoroidal hemorrhage has not been reported previously. Paroxetine is one of the many drugs implicated in bilateral, acute-angle closure, even presenting late, and was therefore withdrawn.² Further doses of acetazolamide were withheld because of the patient's general health but also because this drug has been implicated in bilateral, choroidal effusions following cataract surgery.³

Another, less likely, possibility was that the oral acetazolamide precipitated ocular hypotension, leading to choroidal effusions and subsequent bilateral suprachoroidal hemorrhage.

In our case, it is likely that the patient's lower respiratory tract infection and coughing (Valsalva), combined with his raised INR (both erythromycin and his intercurrent illness could have enhanced the effect of warfarin), were precipitating factors for the suprachoroidal hemorrhages.

Previously reported cases of unilateral spontaneous suprachoroidal hemorrhage have presented with either

shallow or open angles (combined with a raised or normal intraocular pressure).^{1,4} Ultrasound examination was ultimately crucial in confirming the diagnosis of bilateral suprachoroidal hemorrhage in this case⁵ since there was no view to the fundus due to the corneal edema in both eyes.

Suprachoroidal hemorrhage is a rare and dreaded intraoperative complication of cataract surgery. Changes in the choroidal vasculature associated with age are a widely acknowledged risk factor.⁶ Other risk factors include hypertension, atherosclerosis, glaucoma, aphakia, hypotony, sudden decrease in IOP, axial myopia, and inflammation.⁷ At least 11 of the 16 reported cases of spontaneous suprachoroidal hemorrhage have been associated with anticoagulant or thrombolytic therapy. Ocular hypotony and valsalva are important precipitating factors in suprachoroidal hemorrhage.

Literature Search

MEDLINE and the PubMed were searched in August 2010 (English-language results, no date restrictions) for the combinations of the following terms: *suprachoroi-*

dal, choroidal, hemorrhage, simultaneous, spontaneous, bilateral, unilateral.

References

1. Hammam T, Madhavan C. Spontaneous suprachoroidal haemorrhage following a valsalva manoeuvre. *Eye (Lond)* 2003;17:261-2.
2. Levy J, Tessler Z, Klemperer I, et al. Late bilateral acute angle-closure glaucoma after administration of paroxetine in a patient with plateau iris configuration. *Can J Ophthalmol* 2004;39:780-1.
3. Parthasarathi S, Myint K, Singh G, et al. Bilateral acetazolamide-induced choroidal effusion following cataract surgery. *Eye (Lond)* 2007;21:870-2.
4. Chandra A, Barsam A, Hugkulstone C. A spontaneous suprachoroidal haemorrhage: a Case Report. *Cases J* 2009;2:185.
5. Fukuchi T, Suda K, Matsuda H, et al. Secondary acute angle closure with spontaneous suprachoroidal haemorrhage suspected by ultrasound biomicroscopic examination. *Jpn J Ophthalmol* 2009;53:661-3.
6. Moshfeghi DM, Kim BY, Kaiser PK, Sears JE, Smith SD. Appositional suprachoroidal hemorrhage: a case-control study. *Am J Ophthalmol* 2004;138:959-63.
7. Chu TG, Green RL. Suprachoroidal hemorrhage. *Surv Ophthalmol* 1999;43:471-86.