

Central retinal artery occlusion after uneventful glaucoma valve implantation surgery with retrobulbar anesthesia: a case report

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Dear Editor,

I am Dr. Fang-Yu Lin from the Department of Ophthalmology, Shanghai General Hospital, Shanghai, China. I am writing to present to you a case of central retinal artery occlusion after routine glaucoma valve implantation surgery with retrobulbar anesthesia. Retrobulbar anesthesia has been extensively applied in intraocular surgery for many years. It is generally considered a low risk procedure. However, there is a possibility of retinal vessel occlusion, such as central retinal artery occlusion (CRAO), a rare yet devastating complication causing a sudden and permanent loss of vision. This refers to an acute ocular vascular occlusive disorder with dramatic onset of painless vision loss. We report a case of CRAO after uncomplicated glaucoma valve implantation surgery with retrobulbar anesthesia.

A 17-year-old boy with a history of congenital cataract, was found to have an intraocular pressure (IOP) in the right eye of 25 mm Hg, and he was diagnosed as juvenile glaucoma. After undergoing bilateral phacoemulsification cataract extraction and intraocular lens (IOL) implantation surgery, the patient's IOP returned to below 20 mm Hg, with postoperative best corrected visual acuity (BCVA) of 20/20 in both eyes. During outpatient follow-up two years after, his right-side IOP again increased to 28 mm Hg, but visual field acuity test showed no abnormalities, therefore he had not been prescribed eye drops at follow-up. Three years after, the IOP in the right eye increased to 45 mm Hg with normal anterior chamber

angles and could not be controlled with topical levobunolol, brinzolamide along with travoprost. He had no history of vascular disease nor was he taking any oral medication.

The decision to go forward with the surgery was made. Eye examination did not show any vascular abnormalities prior to surgery, BCVA was 20/20 and IOP was 44.7 mm Hg. A glaucoma valve implantation surgery was performed in the right eye and a retrobulbar injection of 2.5 mL of lidocaine was used for anesthesia. A compression was applied for around 15s. Surgery was uneventful, and the eye was padded with ofloxacin ointment at the end of the procedure. Epinephrine was not used in any fluids before and during surgery.

The next day, his right visual acuity was hand motion, not improving with pinhole. IOP in the right eye was 17 mm Hg. Ocular examination of the right eye revealed no corneal edema, no cells in the anterior chamber, well-positioned valve and IOL, and there was a right relative afferent pupil defect. Vitreous was clear, and a cherry red spot could be observed at the macular central fovea with surrounding pale swelling of the fundus posterior retina (Figures 1A, 2A). The retinal vein was tortuous and dilative, while the retinal artery was thinner than normal. No obvious emboli were visible in the retinal circulation.

The patient was urgently given a retrobulbar injection of 0.5 mg of atropine, oxygen inhalation therapy, and 70 mg prednisone orally per day. As the patient noticed only minimal improvement with the treatment, he was further administered immediate 200 mg salvianolate with 250 mL normal saline and 5 µg alprostadil injection with 20 mL normal saline treatment per day, and also 500 µg methycobal with 20 mL normal saline twice a day. Carotid artery ultrasound and color doppler echocardiography were performed and showed no abnormalities. The patient was given extracorporeal counterpulsation treatment with a pressure of 0.02 MPa per day.

Fundus photography and the optical coherence tomography (OCT) examination showed most of the retinal edema subsided but the optic disk became pale at one week (Figures 1B, 2B). After two weeks, fundus fluorescein angiography (FFA) showed normal arm-retinal circulation time (A-RCT) and retinal vein filling time (RVFT) with no visible emboli in the retinal circulation (Figure 3). Only extracorporeal counterpulsation treatment was continued for another month.

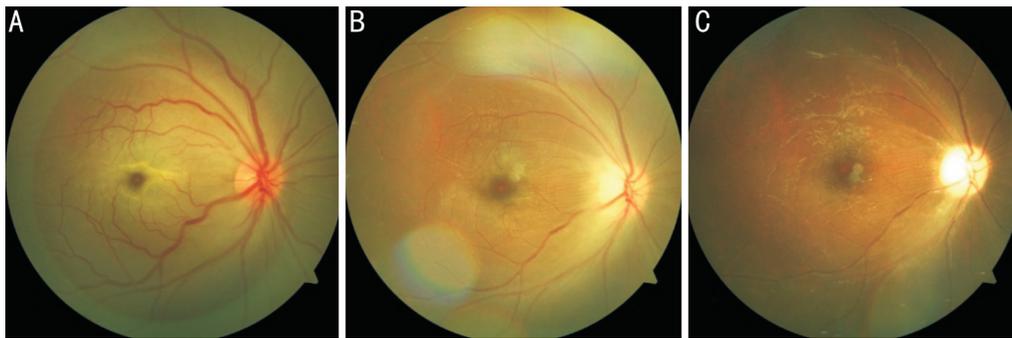


Figure 1 Fundus photography showed cherry red spot with diffuse retinal edema on the first postoperative day (A); Retinal edema subsided and optic disk was slightly pale a week later (B); A pale optic disk with attenuated retinal veins and arteries one month later (C).

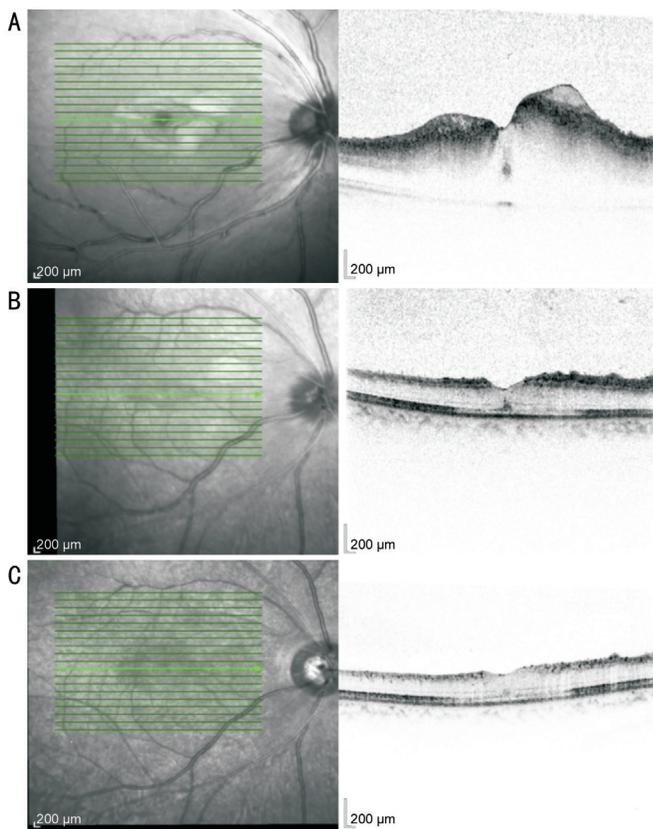


Figure 2 Macular region OCT scans showed retinal edema on the first postoperative day (A) and edema gradually faded away one week (B) and one month later (C).

One month later, there was no improvement in vision acuity, which remaining hand motion. Ocular funduscopy examination showed a pale optic disk with attenuated retinal vessels (Figure 1C).

Carotid artery disease is the most common etiological factor of retinal arterial occlusion (RAO), resulting in RAO by embolism, which can hemodynamically induce retinal ischemia. On the other hand, serotonin (5-hydroxytryptamine) secreted by rough vascular endothelium can induce arterial spasm^[1]. The patient in this case was a 17-years-old young boy with no pre-existing general illness except congenital cataract and juvenile glaucoma that was diagnosed during outpatient follow-up. Our literature review did not show any association

between congenital cataract and definite vascular abnormalities or increased risk of thrombosis. On the other hand, the glaucoma valve implantation surgery itself was uneventful. Although there is a very small probability that the valve may cause vascular compression, this impact would be negligible. Therefore we consider, in this case, that this complication is possibly attributable to retrobulbar anesthesia rather than surgical procedures or potential general artery disease.

There are many case reports describing CRAO after several types of ocular surgeries, such as cataract surgery^[2-3], anti-glaucoma surgery^[4], vitrectomy^[5] or even pterygium excision^[6] which were performed under sub-Tenon's, peribulbar or retrobulbar anesthesia. To our knowledge, this is the first report of CRAO after uneventful glaucoma valve implantation surgery.

Retrobulbar anesthesia has been widely used in ocular surgeries, and complications of this procedure (e.g. retrobulbar hemorrhage, injection of lidocaine and air into the optic nerve sheath, trauma to and partial injection of lidocaine in the central retinal artery) have been described by Morgan *et al*^[7]. These complications could result in emboli in both choroidal and retinal circulations, occlusion of the central retinal artery and vein, or outer retinal ischemic infarction^[7-8]. This implies that when anesthesia is administered, there are ocular blood flow changes^[8], which have been shown by color Doppler imaging (CDI)^[9]. We suggest that pulsatile ocular blood flow (POBF) falls during anesthesia may be the possible mechanism for CRAO in our case. A reduced ocular blood flow may be hazardous to patients regardless of whether IOP is raised or not^[10-11]. This effect recovers slowly and is still present after surgery^[8,12].

Several hypotheses have been proposed herein to explain the possible mechanism of a dramatic reduction of the blood flow velocity after retrobulbar anesthesia. First, there may be a central retinal artery vasospasm in response to the injection^[13] or the anesthetic agent itself could possibly diffuse into the artery^[2]. This disturbs the autoregulation of the retinal circulation. Meyer *et al*^[14] found that porcine

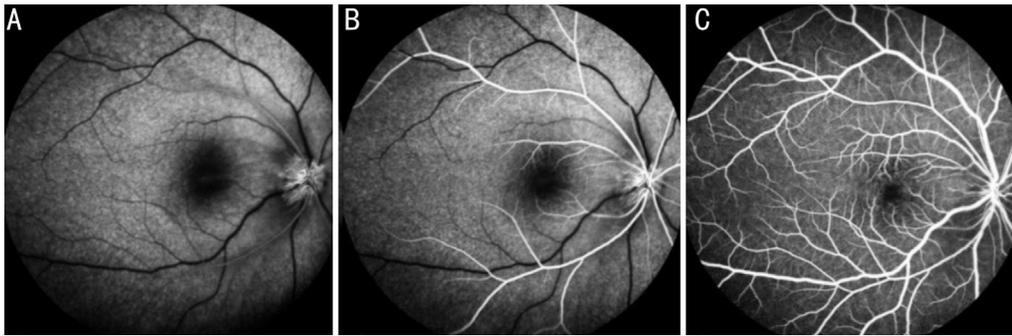


Figure 3 FFA showed normal A-RCT and RVFT at 15s (A), 17s (B) and 41s (C) two weeks after surgery.

ciliary arteries were prevented from relaxation by agents like lidocaine. Riva *et al*^[15] showed that local anesthetic agents (lignocaine, bupivacaine, and ropivacaine) could cause relative vasoconstriction at lower concentrations and vasodilation at higher concentrations. Findl *et al*^[16] also demonstrated in human that choroidal and central retinal artery blood flow decreased 15% one minute after peribulbar anesthesia, which is supposed to be safer than retrobulbar anesthesia. This effect persisted at five to ten minutes^[10,16]. This may be an explanation for the decrease of POBF but it is difficult to determine its clinical relevance as it is impossible to measure concentration of local anesthetic agent around the artery.

Secondly, the mechanical compression of the retrobulbar tissue space by local anesthetic agent (2.5 mL in our case) may cause a volume effect to the globe. The central retinal artery is most likely to be compressed within the orbit, especially since the anesthetic agent forms a “trapped bolus” before gradually diffusing throughout the peribulbar space^[2,8]. This volume effect may initiate a sudden blockage of the central retinal artery. Huber and Remky^[9] reported that a reduction could be detected in retrobulbar velocity after a 2 mL injection, while systolic retinal and ciliary perfusion pressure were reduced after an injection of 5 mL rather than 2 mL.

Thirdly, a rise in IOP secondary to globe compression might also cause CRAO. It is well known that an extreme and prolonged increase in IOP is required to produce CRAO^[13]. However, several studies showing similar results indicated that there were not a statistically significant change in IOP following retrobulbar anesthesia^[10-11,17], and others^[8,18] demonstrated a rise in IOP by 3-4 mm Hg which only lasted for a short time. Moreover Gillart *et al*^[19] and Watkins *et al*^[10] described that this transient change in IOP was unlikely to affect retinal perfusion. And Findl *et al*^[16] also reported no correlation between high IOP and decrease in retinal blood flow following anesthesia agent injection. Therefore it is likely that the effect of automatic adjustment in patients with compromised vasculature is limited^[2] which causes them to be extremely sensitive to even a tiny change in IOP. In our case, on the other hand, the patient’s preoperative IOP was already

high, so a little increase in IOP might have a great influence on POBF, potentially leading to occlusion of the retinal artery.

In conclusion, CRAO is a rare complication after retrobulbar anesthesia in several kinds of ocular surgeries, and it is always associated with poor visual outcome. The mechanism of this unusual complication is still uncertain, but alternative and possibly safer methods such as topical anesthesia may be preferred, especially in patients who may have preexisting vascular compromise or high preoperative IOP level.

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Conflicts of Interest: Lin FY, None; Fu MS, None.

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