Case report

Central retinal artery occlusion on postoperative day one after vitreoretinal surgery

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A B S T R A C T

Purpose: To report two cases of central retinal artery occlusion (CRAO) associated with vitreoretinal surgery.

Observations: Two patients underwent vitreoretinal surgery and were diagnosed with CRAO on postoperative day one. Both had received retrobulbar anesthetic blocks, followed by pars plana vitrectomy in one patient and scleral buckling in the other patient. Best-corrected visual acuity at last follow-up was 20/40 and 20/400.

Conclusions/Importance: CRAO is a rare but serious adverse event after vitreoretinal surgery. The causative mechanism is not known in these patients.

1. Introduction

Central retinal artery occlusion (CRAO) is known to be a rare complication associated with ocular surgery.11 Retrobulbar anesthetic injections are a known association,3,8,10,14,18–20 though there are also reports of CRAO occurring after peribulbar1,4,9,12,17,21 and sub-Tenon’s5,6 injections. Visual recovery is generally poor in these patients.

CRAO after vitreoretinal surgery has been reported,1,5,10,19 but to our knowledge, there have only been two documented cases of ocular arterial occlusion identified on postoperative day one after scleral buckling alone, neither of which were CRAOs.2,15 One was simultaneous occlusion of 3 cilioretinal arteries,15 and the other was an ophthalmic artery occlusion.2 Here we report two patients, one who had undergone scleral buckling, and one who had undergone pars plana vitrectomy, who were noted to have CRAO one day after vitreoretinal surgery (Table 1).

2. Findings

2.1. Case 1

A 28 year old man with a history of high myopia had repair of a macula-sparing rhegmatogenous retinal detachment in the left eye with scleral buckle. Seven months later, he presented with a macula-sparing rhegmatogenous retinal detachment in the right eye (Fig. 1A). Preoperative VA was 20/15 in the right eye and 20/50 in the left. In the right eye, there were optic nerve head drusen, and multiple retinal breaks adjacent to lattice retinal degeneration.

Here we report two patients, one who had undergone scleral buckling, and one who had undergone pars plana vitrectomy, who were noted to have CRAO one day after vitreoretinal surgery (Table 1).

Table 1

| Case | Age, Sex | Anesthesia | Vascular risk factors | Preop Dx | Operation type | CRAO Dx (days postop) | CRAO Tx | Preop VA | VA at CRAO Dx | Last VA |
|------|----------|------------|----------------------|----------|----------------|----------------------|---------|----------|---------------|---------|
| 1    | 28, M    | retrobulbar| Brother died of MI   | RRD      | Scleral buckle | 1                    | AC tap, timolol-dorz., brimon., hyper-vent., none | 20/15   | LP       | 20/400        |         |
| 2    | 70, F    | retrobulbar| HTN                  | Vitreous opacities | PPV | 1                    |         | 20/25   | E 3′         | 20/400  |         |

Abbreviations: M, male; F, female; MI, myocardial infarction; HTN, hypertension; RRD, rhegmatogenous retinal detachment; PPV, pars plana vitrectomy; AC tap, AC paracentesis; dorzo., dorzolamide; brimon., brimonidine; LP, light perception; ecc, eccentric fixation.

Abbreviations: CRAO, Central retinal artery occlusion; PPV, pars plana vitrectomy; BRAO, branch retinal artery occlusion; OCT, optical coherence tomography

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Anesthesia included IV sedation, monitored anesthesia care, and a plana vitrectomy for symptomatic vitreous floaters in the left eye. Pertension, and thyroidectomy presented one day after undergoing pars plana vitrectomy for symptomatic vitreous floaters in the left eye. The retinal vasculature appeared normal without attenuation, emboli, or box-carring. The retina was attached. There was no proptosis or orbital fullness. Optic nerve head drusen were again noted on B-scan in the right eye, but there was no optic nerve sheath hematoma (Fig. 1C). OCT demonstrated inner retinal thickening and hyperreflectivity (Fig. 1D). Fluorescein angiography demonstrated normal arterial and venous perfusion (not shown).

The patient was treated with anterior chamber paracentesis (subsequent IOP was 5), timolol-dorzolamide and brimonidine drops, and 500mg oral acetazolamide. Induced hyperventilation into a paper bag was also performed. On the second postoperative day the VA was 1/200. IOP was normal, and the exam was unchanged. During the review of systems, the patient stated that his brother had died of myocardial infarction at age 23 from early-onset advanced atherosclerotic disease. The patient himself had previously undergone echocardiography, chest CT and MRI which had been normal. He had never had a hematologic workup. The patient was started on prednisone 60mg PO daily for two weeks, which was then tapered. Hematology consultation with a complete hypercoagulable workup revealed elevated factor VIII which was felt to likely be related to a healing response rather than underlying coagulopathy. A follow up test 6 months after surgery showed a normal factor VIII level. A carotid ultrasound was normal.

At one month after surgery, the patient felt his central scotoma had improved, and the VA had improved to 20/400. At ten months after surgery, the patient reported a stable central scotoma, and VA remained 20/400. OCT showed severe retinal thinning with loss of foveal contour (Fig. 1E).

2.2. Case 2

A 70 year old woman with a history of psoriatic arthritis, hypertension, and thyroidectomy presented one day after undergoing pars plana vitrectomy for symptomatic vitreous floaters in the left eye. Anesthesia included IV sedation, monitored anesthesia care, and a retrobulbar block using lidocaine and bupivacaine; there were no episodes of intraoperative hypotension, there was no gas bubble, and postoperatively the patient did not sleep in the prone position. On postoperative day one the VA was 20/25. On postoperative day one the VA was 3/200. IOP was 11. Exam showed macular whitening with a cherry red spot (Fig. 2A), but the remainder of her retinal exam was normal. A fluorescein angiogram demonstrated decreased foveal and parafoveal fluorescence with chorioretinal artery sparing (Fig. 2B). B-scan ultrasonography demonstrated a normal optic nerve head and normal retrobulbar optic nerve thickness (Fig. 2C). OCT showed inner retinal thickening and hyperreflectivity (Fig. 2D). The patient underwent carotid ultrasound, echocardiogram, and electrocardiogram which were reported to be normal. Lab tests including thyroid function studies, ESR and CRP were normal. She was managed with observation. Six months later, her VA was 20/40 with searching. The posterior segment examination showed vascular attenuation, macular atrophy, and pigmentary changes (Fig. 2D). OCT showed inner retinal atrophy and thinning (Fig. 2F).

3. Discussion

We report two patients with CRAO that occurred in the immediate postoperative period after vitreoretinal surgery. One patient was diagnosed one day after scleral buckling, and the other patient was diagnosed one day after PPV. CRAO is a known but very rare complication of ocular surgery that can occur after retrobulbar, peribulbar, or sub-Tenon’s anesthesia. One case of CRAO after PPV with scleral buckling has been reported, but the first case occurred 19 days after surgery in a patient with known bilateral severe carotid artery stenosis, and the second case occurred 21 days after surgery and was a combined CRAO-central retinal vein occlusion. In addition, one branch retinal artery occlusion (BRAO) occurred 23 days after PPV with scleral buckling, and another case of BRAO 14 days after PPV; both occurred in patients with known vascular risk factors. Malinowski and Pesin have also reported BRAO 12 days after PPV. In both studies, the occurrence outside of the immediate postoperative period calls into question whether the retinal arterial occlusions were causally related to the surgical procedures.

CRAOs after PPV with retrobulbar mepivacaine injection have also
buckling is known to decrease retinal arterial flow, but intra-operative compression or stasis, and iatrogenic retrobulbar hemorrhage. Scleral buckling, or both, is not clear. Underlying vascular risk factors and/or coagulopathy may precipitate CRAO, but as evidenced in our report are not always present.

A variety of treatments, from lowering intraocular pressure to cannulating the ophthalmic artery and directly applying recombinant tissue plasminogen activator, have been attempted for CRAO. However, none have demonstrated clinical benefit. Visual recovery from CRAO is generally poor, though can be somewhat variable as it was in our study.

In summary, CRAO is a rare but serious complication associated with vitreoretinal surgery. We report two patients with CRAO in the immediate postoperative period after vitreoretinal surgery, one after scleral buckling and one after pars plana vitrectomy. Vitreoretinal surgeons should be aware of this potential, albeit rare complication when considering surgical intervention.

**Patient consent**

No consent was obtained, as there is no identifiable information reported here.

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**Conflicts of interest**

All authors have no financial disclosures.

**Authorship**

All authors attest that they meet the ICMJE criteria for authorship.

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**Appendix A. Supplementary data**

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ajo.2018.10.001.

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