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Case report

Central retinal artery occlusion on postoperative day one after vitreoretinal surgery

Jonathan F. Russell[a], Nathan L. Scott[a], Luis J. Haddock[a], Alexander M. Eaton[b], Harry W. Flynn[a,*]

[a] Department of Ophthalmology, Bascom Palmer Eye Institute, University of Miami, Miller School of Medicine, Miami, FL, 33136, USA
[b] Retina Health Center, Fort Myers, FL, 33907, USA

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 peribulbar[1,4,9,12,17,21] and sub-Tenon's[5,6] 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.

The patient underwent surgery under monitored anesthesia care with a deep peribulbar block using a 25 gauge, 7/8 inch Atkinson needle containing 6mL of a 1:1 mixture of 4% lidocaine and 0.75% bupivicaine with hyaluronidase. Cryotherapy surrounding the retinal breaks was performed, and a scleral buckle (41 band with 70 sleeve) was placed. No subretinal fluid was drained. An anterior chamber

Table 1
Patient characteristics.

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-dorzo., brimon., hyper-vent.	20/15	LP	20/400
2	70, F	retrobulbar	HTN	Vitreous opacities	PPV	1	none	20/25	E 3′	20/40 (ecc)

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

* Corresponding author. 900 NW 17th St, Miami, FL, 33136, USA.
E-mail address: hflynn@med.miami.edu (H.W. Flynn).

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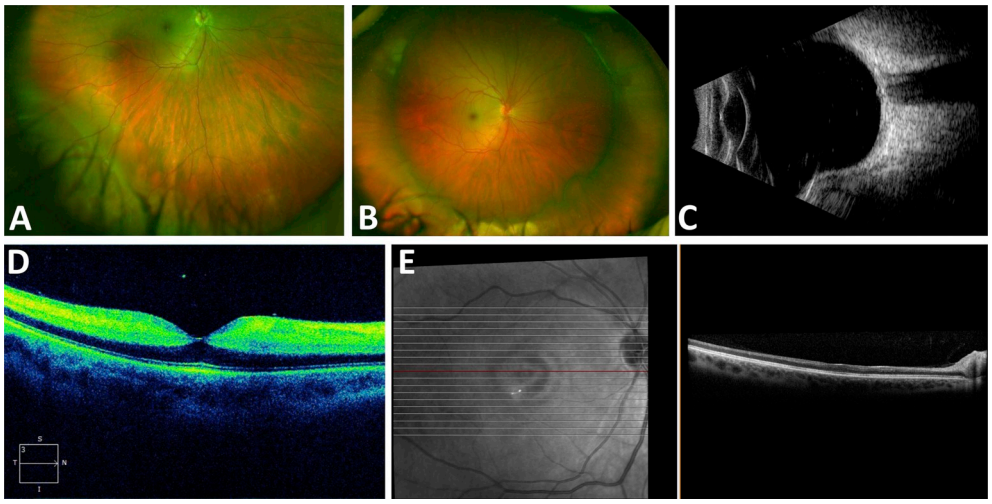

Fig. 1. CRAO one day after uncomplicated scleral buckling procedure. **A**. Preoperative wide-field fundus photograph of right eye showed macula-sparing inferotemporal rhegmatogenous retinal detachment. Preoperative VA was 20/15. **B**. One day postoperative wide-field fundus photograph showed macular whitening and cherry-red fovea. **C**. One-day postoperative B-scan ultrasound showed scleral buckle and optic nerve head drusen but normal retrobulbar optic nerve thickness. **D**. One-day postoperative OCT showed inner retinal hyperreflectivity. **E**. Ten month postoperative OCT showed severe retinal thinning and loss of foveal contour. Last VA was 20/400. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

paracentesis was performed and the intraocular pressure was confirmed to be at physiologic level by digital palpation. The optic nerve and retina were perfused. At the conclusion of the procedure, 500mg of intravenous acetazolamide was administered, and supplemental sub-Tenon's block was administered with a blunt cannula. No gas bubble was instilled, there were no episodes of hypotension during the surgery, and postoperatively the patient did not sleep in the prone position.

On postoperative day one, VA in the right eye was light perception. Intraocular pressure (IOP) was 14. There was a trace afferent pupillary defect by reverse in the right eye. The posterior segment examination showed retinal whitening in the macula and a cherry-red spot (Fig. 1B). 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 bupivicaine; there were no episodes of intraoperative hypotension, there was no gas bubble, and postoperatively the patient did not sleep in the prone position. Her preoperative 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 cilioretinal 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. 2E). 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.[1,3–6,8–12,14,17–21] Calenda et al.[4] reported CRAO after peribulbar anesthesia for PPV for RRD repair, and Jung et al.[10] reported two cases of CRAO after PPV using retrobulbar anesthesia. Ascaso[1] reported transient CRAO after peribulbar anesthesia for PPV. Ellabban et al.[5] noted CRAO upon initiation of PPV for vitreomacular traction in a patient who had received sub-Tenon's anesthesia; upon induction of posterior vitreous detachment the retina completely re-perfused.

CRAO after PPV with scleral buckling has been reported,[7] 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[7]; both occurred in patients with known vascular risk factors. Malinowski and Pesin[13] 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 mepivicaine injection have also

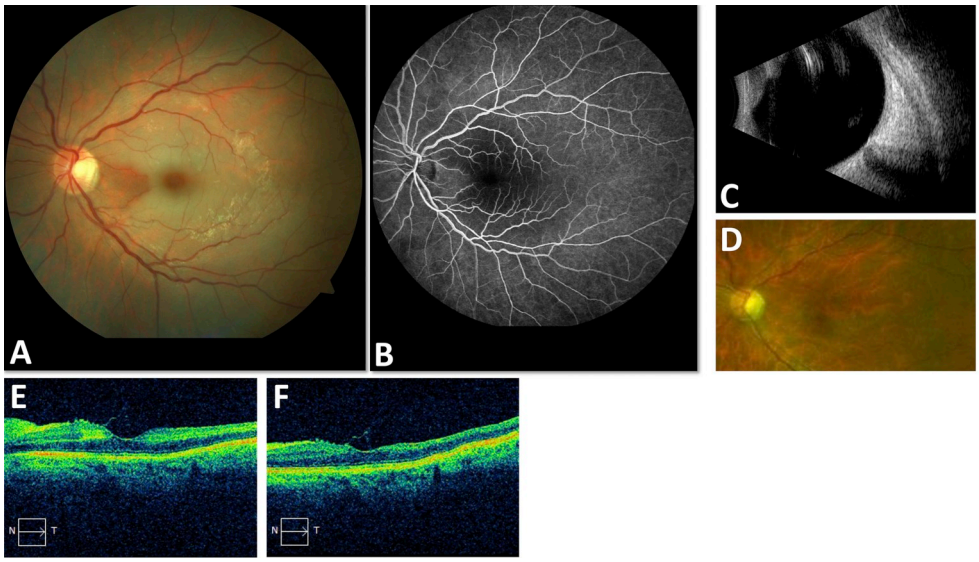

Fig. 2. CRAO one day after uncomplicated pars plana vitrectomy for symptomatic vitreous opacities. **A**. One-day postoperative fundus photograph of left eye showed macular whitening with cilioretinal sparing, and cherry-red fovea. **B**. One-day postoperative fluorescein angiography showed decreased foveal and parafoveal fluorescence, likely due to a combination of blockage of choroidal flush from macular edema and lack of retinal capillary perfusion. **C**. One-day postoperative B-scan ultrasound demonstrated normal optic nerve head and normal retrobulbar optic nerve thickness. **D**. Two-month postoperative wide-field fundus photograph showed vascular attenuation and macular pigmentary changes. **E**. One-day postoperative OCT showed inner retinal hyperreflectivity. **F**. Two-month postoperative OCT showed severe retinal thinning. VA was 20/40 eccentric, and remained 20/40 (eccentric) six months after surgery. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

been reported.[19] Interestingly, the CRAOs were not noted on postoperative day one, but occurred between 2 and 14 days postoperative. These three cases were part of a cluster that included three additional patients with BRAO over a three month period who had received retrobulbar mepivicaine containing methyl- and propyl parahydroxybenzoate preservatives, whereas no episodes of retinal arterial occlusion were noted in patients who had received retrobulbar injection with preservative-free mepivicaine, leading the authors to speculate that the preservatives may have caused a delayed vasoconstrictive effect leading to vascular occlusion.

To our knowledge, there have only been two documented cases of ocular arterial occlusion immediately after scleral buckling. In one case, the authors noted simultaneous occlusion of 3 cilioretinal arteries, whereas the central retinal artery was not occluded.[15] In the second case, general anesthesia was used without any supplemental retrobulbar/peribulbar/sub-Tenon's injections, in a patient that was later found to have underlying coagulopathy.[2] The authors noted delayed choroidal perfusion consistent with an ophthalmic artery occlusion.[2] Thus, the current study is the first report of CRAO in the immediate postoperative period after scleral buckling.

Proposed mechanisms for CRAO in cases where local anesthesia is used include direct transection of the central retinal artery by the needle, a local vasoconstrictive or vasospastic effect of the injection or the injected medications, increased orbital pressure leading to vascular compression or stasis, and iatrogenic retrobulbar hemorrhage. Scleral buckling is known to decrease retinal arterial flow,[16,22] but intra-operative examination of the retina both before and after application of the scleral buckle did not reveal a CRAO. Thus, whether the CRAO observed after scleral buckling was a consequence of anesthesia, 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.ajoc.2018.10.001.

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