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Case report

Central retinal artery occlusion during vitrectomy: Immediate retinal revascularization following induction of posterior vitreous detachment

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ABSTRACT

Purpose: To describe a patient with acute central retinal artery occlusion (CRAO) during vitrectomy surgery and the possible role of vitrectomy in acute CRAO management.

Observations: An 84-year-old man presented with broad vitreomacular traction and epiretinal membrane in the right eye. Preoperative assessment clearly showed normal retinal vasculature. On starting vitrectomy, complete CRAO with marked segmentation of all retinal vessels was noted. Vitrectomy was performed in the usual manner and once the posterior hyaloid detached from the disc, immediate complete revascularization of the retinal vessels was noted. The patient had a complete visual recovery.

Conclusions and importance: Immediate vitrectomy with induction of posterior vitreous detachment may have a role in selected cases of acute CRAO, particularly if performed within a short window.

1. Introduction

Central retinal artery occlusion (CRAO) is a challenging ocular emergency that often leads to profound and irreversible visual loss. The main causes of CRAO are thrombosis due to atherosclerosis or emboli that mainly originate from the carotid arteries. Less commonly, CRAO may occur due to coagulopathies, collagen vascular diseases, and local ocular abnormalities. Several treatment modalities have been attempted, including medical therapies, laser embolysis/embolectomy and more invasive surgical maneuvers, with only limited success.[1,2]

Given the rarity of the disease and diverse clinical scenarios, the results of various treatment modalities in the literature are mainly based on single case reports or small case series.[2–7] In this report, we describe an unusual case of CRAO that was only noted at the beginning of vitrectomy. We speculate that immediate vitrectomy may have a role in selected cases of CRAO.

2. Case report

An 84-year-old male artist reported visual symptoms of distortion and aniseikonia. His optical coherence tomography (OCT) scans showed broad vitreomacular traction and an epiretinal membrane (Fig. 1a and b). He had cataract surgery five years previously. He was therefore listed for vitrectomy surgery under local anesthesia. He was known to be hypertensive and was on treatment with atenolol 50 mg once a day. On the day of the surgery, the preoperative assessment was performed in the usual manner. Blood pressure was 165/81 mmHg, pulse was 67 beat/min, and blood oxygen saturation (SpO2) was 99%. Subtenon's anesthesia was performed in the anesthetic room linked to the operating theatre using 6 ml of a mixture of lignocaine (2%) and bupivacaine (0.75%) warmed to body temperature in a warming cabinet, and injected infero-nasally. The globe was gently massaged following the injection and no compression device was used. The anesthetist did not use adrenaline or hyaluronidase in the anesthetic mixture.

Immediately at the commencement of vitrectomy, a complete CRAO was noted. This was associated with complete segmentation of all blood vessels (cattle-trucking), severe optic disc pallor and an area of pale retina inferior to the optic disc possibly demarcating the watershed zone (Fig. 1c). The eye pressure was checked and the vitrectomy machine sensor read 25 mmHg. Due to the inaccuracies that can often happen with the automated eye pressure sensors, the eye pressure was further checked digitally and felt to be within the normal range. There was no globe or eyelid tenderness. There was no proptosis or evidence of any retrobulbar hemorrhage. Retinal vessels were examined closely and no embolus was visible. The patient vital measures were rechecked and blood pressure was 168/84 mmHg, pulse was 64 beat/min and blood oxygen saturation (SpO2) was 99%. The patient was questioned and he did not report any change to his vision in the interval from the preoperative assessment to the time of the local anesthesia. He had no

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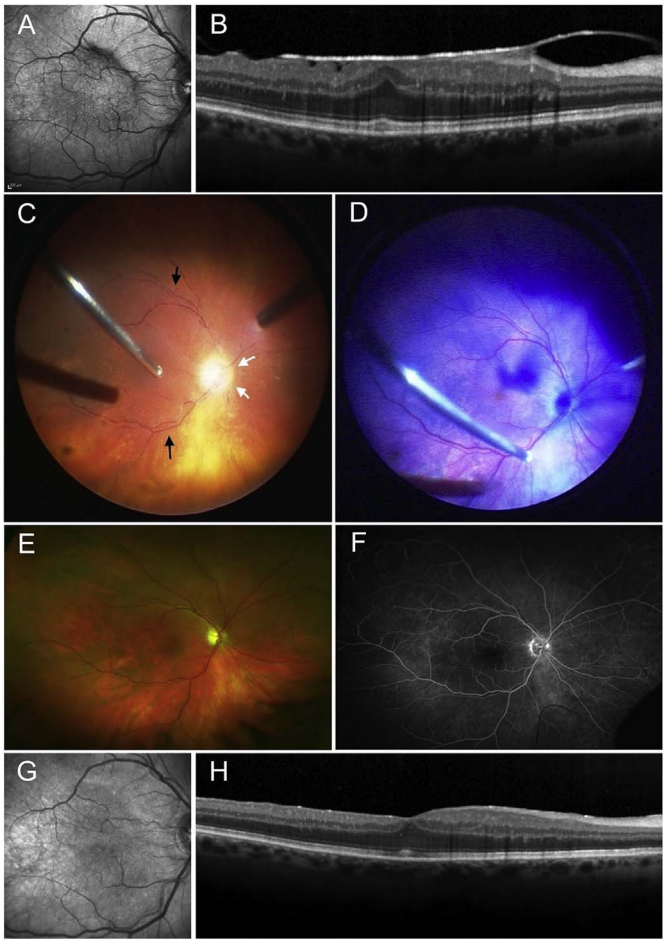

Fig. 1. Central retinal artery occlusion at the outset of vitrectomy. Preoperative (A) infrared reflectance and (B) foveal optical coherence tomography (OCT) scan of the right eye clearly depicting broad vitreomacular adhesion and epiretinal membrane. The retinal vessels appeared to be of normal caliber, with no obvious vascular changes at the arteriovenous crossing. Intraoperative video snapshot (C) at the beginning of the surgery demonstrating a complete central retinal artery occlusion. There is complete segmentation of the blood column in all major arcades (black arrows) and sever disc pallor (white arrows). (D) Video snapshot after PVD induction. The retinal vessels are completely reperfused. Postoperative (E) wide field color photography and (F) fluorescein angiography 10 days after the surgery depicting total restoration of the retinal circulation. At 10-days postoperatively, the (G) infrared reflectance and (H) foveal OCT scan show restoration of the retinal architecture following the peel of the epiretinal and internal limiting membranes. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

history of retinal vascular problems and no history of amaurosis fugax.

We proceeded with the vitrectomy. The core vitreous was removed in the usual manner. The posterior hyaloid was quite adherent and did not detach from the disc after several attempts. Membrane blue dual dye (DORC International, NL) was used to highlight the vitreous, and posterior vitreous detachment (PVD) was initiated by active suction at the disc. Surprisingly, once the hyaloid came off the disc, immediate complete revascularization of all retinal vessels occurred (Fig. 1d). The epiretinal and internal limiting membranes were peeled and the surgery continued uneventfully (supplemental video 1).

Supplementary video related to this article can be found at http://dx.doi.org/10.1016/j.ajoc.2018.01.008

Following surgery, the patient was further reviewed. His blood pressure was uncontrolled (175/85 mmHg) despite treatment. Blood work-up was performed to rule out arteritic causes. He was referred for vascular assessment and duplex ultrasound of the internal carotid arteries showed mild to moderate stenosis bilaterally. The patient was reviewed at 10 days following the surgery; his vision had improved to 6/12 and reported marked improved of the distortion and resolution of the aniseikonia. His OCT scans showed marked improved of the retinal architecture. Wide-field fluorescein angiography was performed. The retinal circulation was completely intact, with normal vessel caliber, no vascular changes at the arteriovenous crossings, and normal optic disc perfusion. There was no choroidal filling delay and no retinal emboli were evident on the angiogram (Fig. 1e and f). The patient was further reviewed at one and two months, reporting gradual improvement of vision and distortion and his vision improved to 6/9 at 2 months.

3. Discussion

In this case report, complete CRAO has occurred possibly during the interval between the administration of the subtenon's anesthesia and starting the surgery. Surprisingly, complete restoration of the circulation in all retinal vessels occurred immediately following the separation of the posterior hyaloid from the disc. The patient achieved complete visual recovery.

The exact mechanism of CRAO in this case is unknown, but the culprit is likely to be the local anesthesia. Previous reports show three possible mechanisms that can lead to CRAO during local ocular anesthesia.[8] These mechanisms include direct mechanical pressure on the central retinal artery by the anesthetic bolus, an elevation of intraocular pressure following the administration of periocular anesthesia, leading to occlusion of the central retinal artery (especially when a mechanical compression device is used), or a localized vasoconstrictive effect if adrenaline is used in the anesthetic mixture. These postulated mechanisms have been reported mostly in peribulbar or retrobulbar anaesthesia and rarely with subtenon's anesthesia.

These mechanisms do not fully explain the cause of CRAO in this report. In our patient, only subtenon's anesthesia, presumed to be safer, was given without adrenaline in the mixture. There was no evidence of compression on the central retinal artery and only a moderate anesthetic volume of 6 ml was used. The clinical picture of CRAO was complete, with segmentation of the blood column in all blood vessels, which often takes at least a few minutes to develop. It is notable that the patient had an area of severe retinal pallor inferior to the optic disc (Fig. 1c) that coincides with the watershed zone. This possibly implies underling vascular insufficiency at the level of the optic nerve head. Therefore, a mechanical effect of the volume of anesthetic on the central retinal artery is still a possible mechanism, particularly if the retinal circulation is compromised by underlying atherosclerosis and systemic hypertension. Therefore, it may be prudent to inject a smaller volume of local anesthetic for ophthalmic surgery, which can be further topped up when required during the procedure.

Several treatments have been tried for acute CRAO such as oral vasodilators, hyperbaric oxygen, and lowering the eye pressure to help CRA perfusion by intravenous acetazolamide, mannitol or paracentesis. However, the efficacy is very limited and sporadic. Translumenal Nd:YAG laser embolysis and/or embolectomy[6] has been attempted to photodisrupt the arterial emboli with promising visual outcomes in selected cases of branch or central retinal artery occlusion, but it can be complicated by significant vitreous hemorrhage, collateral tissue damage and propagation of the emboli to occlude other branches.

Vitrectomy has been attempted in few cases of central or branch retinal artery occlusion.[3,4,7,9] Almedia et al.[3] treated a patient with fovea threatening branch retinal artery occlusion by incision of the arterial sheath with a microvitreoretinal blade, and retrieval of the embolus by a microsurgical forceps. Tang et al.[7] treated one patient with surgical cannulation of the central retinal artery with a specially designed 50-gauge stylet, with an improvement in visual acuity from counting fingers to 20/25 at 4 months. Lu et al.[9] tried vitrectomy with central rental artery massage using a specially designed probe in patients with CRAO ranging between 1 and 6 days. The retina was reperfused, at least partially, particularly in patients with emboli at the level of CRA. However, these techniques mainly target displacement or

retrieval of an embolus at the level of the CRA. Retinal emboli are only visible in 20–40% of cases of CRA[10] while the remainder do not have visible emboli, as in our case.

In this case, CRAO was only noted at the start of the surgery and the options were to continue or terminate the surgery. Since there had been previous case reports on the possible role of vitrectomy in acute CRAO, and hypotony being one treatment option for CRAO, the surgeon decided to proceed. Interestingly, the retina was immediately re-vascularized following induction of PVD. The exact effect of detaching the posterior hyaloid on the CRA is unknown. We speculate that PVD induction can possibly lead to the release of abnormal adhesion at the level of the CRA. In this case, the hyaloid was quite adherent to the disc and only detached after several attempts. Alternatively, the active aspiration over the disc may have created a negative suction force that can open the collapsed central retinal artery lumen or dislodge a thrombus. Also, PVD induction may have dislodged a thrombus or an embolus. However, we didn't observe any retinal emboli after careful study of wide field angiography.

The retinal tolerance to ischemia is very short, and it has been demonstrated in experiential studies that irreversible damage occurs after 240 min or less.[5] Since the interval between CRAO and the surgery was relatively short in this case, the retinal reperfusion was complete and the patient had a complete visual recovery.

4. Conclusions

Surgical intervention for CRAO is still controversial with very limited evidence. In this case report, we describe an unusual occurrence of a CRAO noted at the outset of vitrectomy. We propose that prompt vitrectomy with PVD induction alone, if done within a short window of the occurrence of CRAO, can restore ocular circulation in selected cases. Detaching the hyaloid from the disc may have a role in reperfusion of the occluded CRA by releasing any abnormal adhesion around the artery or opening the collapsed lumen via the negative suction force created during active aspiration or dislodging a thrombus. The role of immediate vitrectomy for CRAO needs to be investigated on a larger scale.

Patient consent

The consent has been obtained from the patient in writing.

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Conflicts of interest

The authors have no financial disclosures.

Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.ajoc.2018.01.008.

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