Central retinal artery occlusion following orbital tumor resection: Is rapid intervention effective?

Mohammad Taher Rajabi, Mohammad Naderan, Seyed Ziaeddin Tabatabaei Mohammadi, Mohammad Bagher Rajabi

A 52-year-old male patient presented at our hospital with unilateral proptosis and vision loss in his left eye. Imaging evaluations showed orbital tumor, so the patient underwent surgery. About an hour later after tumor removal, patient developed sudden vision loss and became no light perception. Fundus evaluation revealed central retinal artery occlusion (CRAO). The patient was treated immediately with ocular massage and anterior chamber paracentesis as well as systemic therapy with mannitol and intravenous administration of acetazolamide. After thirty minutes, he recovered perception to light and then hand motion and 2 h later, it was improved to 1 m counting finger. CRAO following orbital tumor has not been reported before. We recommend ocular examination in all patients that undergo orbital surgery immediately to 2–3 h after surgery.

Key words: Central retinal artery occlusion, orbital tumor, rapid intervention, tumor resection

Central retinal artery occlusion (CRAO) is a very rare condition that happens in 1/10,000 outpatient visit.[1,2] This event is sudden and devastating that leads to visual acuity (VA) of counting fingers or less,[2,3] thus it is one of the most important topics in ophthalmology needs to immediate intervention. For the first time, we report a patient with postoperation CRAO after orbital tumor resection.

Case Report

A 52-year-old male patient presented at our hospital with progressive unilateral proptosis since 2 years ago and vision loss in his left eye. His left eye VA was 2/10, and right eye was 10/10. Ocular examination revealed 2+ relative afferent pupillary defect in the left eye.

Computed tomography scan of the patient showed an intraconal mass sticking to superior ophthalmic fissure [Fig. 1]. He underwent tumor resection through inferior transconjunctival approach. The procedure was done without complication, and the early postoperative vision was fine but about an hour later, patient developed sudden vision loss and became no light perception (NLP). For ruling out postoperation hemorrhage, surgical site was evaluated but there was not any hemorrhage there. Fundus examination with indirect ophthalmoscopy revealed typical view of CRAO with the presence of cherry-red spot on the macula, white ground-glass appearance of the retina, and optic disc edema [Fig. 2]. For decreasing intraocular pressure and establishing retinal reperfusion, immediate ocular massage and anterior chamber paracentesis as well as systemic therapy with mannitol were done. After 30 min, the patient recovered perception to light and then hand motion. Two hours later, it was improved to 1 m counting finger, but he complained a large central scotoma. His VA improved to 20/200 the day after the surgery, and he complained of diplopia. Perimetry showed inferotemporal scotoma that involved central part. After 3 weeks, his VA improved to 4/10 and scotoma extension was decreased.
Figure 1: Orbital computed tomography-scan of the patient shows well-defined dumble shaped intraconal orbital mass that pushed the optic nerve superomedially and extending to the supra orbital fissure.

Medical workup did not show any evidence of collagen vascular or cardiovascular, hematologic, and neurologic disorders. Systemic investigations including fasting blood sugar, lipid profile, and homocysteine levels all were normal. Pathologic evaluation of the tumor was schwannoma with mucinous transformation.

Discussion

CRAO is usually caused by a thrombus or embolus that resulting to reduce blood perfusion of the retina. It clinically presents as sudden painless acute unilateral or bilateral vision loss in the range of counting fingers to NLP. The rate of spontaneously recanalization of the artery is about 15% with timely intervention, the prognosis is very poor as only 61% of patients can regain a final VA of 6/120 or less. One of the typical findings in CRAO is cherry-red spot that is found in about 90% of cases.

There is a golden time of 90–120 min after occlusion to perform interventions to improve vision, however there is no approved modality to be effective in the treatment of CRAO, but some methods such as immediate ocular massage and anterior chamber paracentesis, use of drugs such as intravenous acetazolamide and mannitol or inhalation of a mixture of 95% oxygen and 5% carbon dioxide (carbogen), all aimed to reduce intraocular pressure and improving blood flow to the eye. Without doing these modalities, <10% of patients can recover meaningful vision.

Multiple theories for CRAO during surgical procedures have been proposed; a prolonged hypotensive status and reduction in blood flow or also increasing intraocular pressure including ocular compression during certain ocular surgical procedures associated with ocular ischemia, causing ischemia of the retina and may lead to CRAO and vision loss after surgery. Although we did not face any significant drop in blood pressure (BP) during intervention but regarding differences in populations may be our patient was very sensitive to even small drop in BP that could lead to CRAO.

In another theory, Körner-Stiefbold explains that retrobulbar injections may compress central retinal artery and lead to arterial occlusion and decrease blood perfusion causing CRAO.

A more likely explanation in that ocular vascular damage may be happened by surgical maneuvers, and it will results in activation and aggregation of platelets and activated platelets release serotonin (5-hydroxytryptamine). Serotonin is a vasoconstrictor and inducing transient arterial spasm, causing transient or complete arterial occlusion leading to ischemia of retina and creating CRAO.

We recommend that in all patients with orbital tumors that undergo tumor removal, the ocular exam should be performed immediately, and every 1–3 h after the surgery to rule out CRAO, as delay in diagnosis and intervention could eventuate to severe vision loss.

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Conflicts of interest
There are no conflicts of interest.

References
1. Hayreh SS. Acute retinal arterial occlusive disorders. Prog Retin Eye Res 2011;30:359-94.
2. Brown GC, Magargal LE. Central retinal artery obstruction and visual acuity. Ophthalmology 1982;89:14-9.
3. Hayreh SS, Zimmerman MB. Central retinal artery occlusion: Visual outcome. Am J Ophthalmol 2005;140:376-91.
4. Chen CS, Lee AW. Management of acute central retinal artery occlusion. Nat Clin Pract Neurol 2008;4:376-83.
5. Hayreh SS, Podhajsky P. Ocular neovascularization with retinal vascular occlusion. II. Occurrence in central and branch retinal artery occlusion. Arch Ophthalmol 1982;100:1585-96.
6. Hayreh SS, Kolder HE, Weingeist TA. Central retinal artery occlusion and retinal tolerance time. Ophthalmology 1980;87:75-8.
7. Atebara NH, Brown GC, Cater J. Efficacy of anterior chamber paracentesis and Carbogen in treating acute nonarteritic central retinal artery occlusion. Ophthalmology 1995;102:2029-34.
8. Myers MA, Hamilton SR, Bogosian AJ, Smith CH, Wagner TA.
9. Körner-Stiefbold U. Central retinal artery occlusion – Etiology, clinical picture, therapeutic possibilities. Ther Umsch 2001;58:36-40.

10. Hayreh SS, Piegrs DJ, Heistad DD. Serotonin-induced constriction of ocular arteries in atherosclerotic monkeys. Implications for ischemic disorders of the retina and optic nerve head. Arch Ophthalmol 1997;115:220-8.