



Vitreotomy With Intrasurgical Control of Ocular Hypotony as a Treatment for Central Retina Artery Occlusion

Central retinal artery occlusion (CRAO) is an ocular emergency usually caused by blockage of a vessel by a thrombus or embolus. The central retinal artery supplies the inner two thirds of the retina; the occlusion of this vessel causes ischemia and subsequent necrosis.¹ Experimental studies have shown that irreversible retina damage can occur 240 minutes after CRAO.² Clinically, the patient experiences a sudden painless and unilateral loss of vision. The natural course of CRAO is devastating: 92% of patients have a visual acuity of counting fingers or worse. However, up to 8% of patients have improved visual acuity because of spontaneous remission of the occlusion.³

There are numerous treatments for CRAO, but none have proved to be successful. Conventional conservative options include carbogen inhalation, acetazolamide infusion, ocular massage and paracentesis, and intravenous glyceryl trinitrate. However, none have been able to alter the natural history of the disease.^{4,5} There has been recent interest in other techniques: the use of tissue plasminogen activator, intraarterial thrombolysis,⁶ surgical embolus removal,⁷ Nd:YAG laser embolysis,⁸ systemic prostaglandin E1,¹ and vitreous surgery with direct central retinal artery massage⁹ or intraarterial verapamil and alteplase infusion.¹⁰ However, these treatments have shown only limited efficacy in improving vision.

As CRAO is the ocular equivalent of a cerebral stroke, the risk factors for this occurrence are the same as for stroke or heart disease. It is important to manage

these risk factors as they may lead to other vascular conditions.⁴

Surgical Technique

This surgical technique is indicated in central artery occlusion with an evolution of less than 24 hours. Standard 3-port pars plana 23-gauge vitrectomy (Platform Constellation; Alcon, Ft Worth, TX) is undertaken ensuring the removal of the posterior hyaloid, especially in the papillary area. Then, intraocular pressure is lowered to 17 mmHg (minimum autocompensated intraocular pressure), and using a magnifying lens and aspirating with a silicone-tipped cannula on the papilla, the intraocular pressure is brought to 0 mmHg. Thus, vascular resistance is reduced generating a positive pressure difference in the central retinal artery and increasing retinal blood flow to the eye. If this is achieved, when aspiration is active, the central artery is filled and consequently the central vein dilates, indicating an active flow when the intraocular pressure is low; when aspiration is stopped, the central retinal vein contracts. When blood flows through the area where the emboli or thrombus is located, the velocity of flow increases because at this point the lumen is narrower. As a result, turbulences are generated, which can cause the thrombus to move or fragment (see **Video, Supplemental Digital Content 1**, <http://links.lww.com/IAE/A353>, which shows this surgical technique). In most cases of CRAO, the thrombus is not visible. Nevertheless, to perform this technique, it is not essential to have visualization of the thrombus because even if it is located behind the lamina cribosa, blood flow can also be regulated with this technique. Using cycles of aspiration–nonaspiration, the thrombus is moved toward more distal vessels causing a re-permeabilization of the central retinal artery. During surgery, restoration of retinal circulation can be seen, but a postoperative angiography must also be performed for confirmation. After surgery, the trocars are removed ensuring there are no fluid leaks and the eye should be left slightly hypotonic.

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Case Study

A 65-year-old male patient presented sudden loss of vision in his left eye that had started 1 hour previously. His medical history included cardiac arrhythmia treated with propafone hydrochloride 600 mg daily. The visual acuity was counting fingers at 1 m. Fundoscopy revealed retina edema, and a CRAO was diagnosed.

A vitrectomy with intrasurgical control of ocular hypotony as described above was performed five and half hours after the onset of symptoms. Complete revascularization was achieved, and visual acuity tested 5 days after the surgery was 20/20.

Discussion

Pars plana vitrectomy is not in itself an alternative treatment but allows for the alteration of eye pressure resulting from continuous changes in aspiration. The resistance to blood flow in front of the thrombus is lowered, creating a flow around and behind the thrombus. Thus, a discontinuous flow acting in a pulsatile way can displace the thrombus leading to a complete resolution of the occlusion.

Final visual recovery depends on the time of evolution and the nature of the thrombus. According to experimental models of CRAO, the time limit to cause irreversible damage to the retina is 240 minutes.² However, unlike animal models, humans rarely have a complete occlusion of the artery, and there is always a remaining flow, which increases retinal viability. As a result, treatment for CRAO has been recommended within 24 hours of symptom onset.

There are mainly three types of thrombus: 1) cholesterol emboli, which originate from the carotid atheromatous plaques in the ipsilateral carotid artery and also from the aorta or heart valves; 2) fibrin platelet emboli, which are associated with carotid or cardiac thrombosis; and 3) calcific emboli, which are related to calcified heart valves or the aorta.

This technique has been shown to be effective with fibrin platelet emboli, which can be deformed, and probably with cholesterol emboli. However, it seems

less able to mobilize calcific emboli, which are often impacted in the lumen of the artery. Although other treatments for CRAO have not proven to be effective, this technique opens up the possibility of permanently resolving occlusive artery disease.

Key words: central retinal artery occlusion, treatment, cholesterol emboli, platelet emboli, nonperfusion, perfusion, blood flow, pars plana vitrectomy, ocular hypotony, fluorescein angiography.

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References

1. Menzel-Severing J, Siekmann U, Weinberger A, et al. Early hyperbaric oxygen treatment for nonarteritic central retinal artery obstruction. *Am J Ophthalmol* 2012;153:454–459.
2. Hayreh SS, Zimmerman MB, Kimura A, Sanon A. Central retinal artery occlusion. Retinal survival time. *Exp Eye Res* 2004;78:723–736.
3. Takai Y, Tanito M, Matsuoka Y, et al. Systemic prostaglandin E1 to treat acute central retinal artery occlusion. *Invest Ophthalmol Vis Sci* 2013;54:3065–3071.
4. Cugati S, Varma DD, Chen CS, Lee AW. Treatment options for central retinal artery occlusion. *Curr Treat Options Neurol* 2013;15:63–77.
5. Varma DD, Cugati S, Lee AW, Chen CS. A review of central retinal artery occlusion: clinical presentation and management. *Eye (Lond)* 2013;27:688–697.
6. Noble J, Weizblit N, Baerlocher MO, Eng KT. Intra-arterial thrombolysis for central retinal artery occlusion: a systematic review. *Br J Ophthalmol* 2008;92:588–593.
7. García-Arumí J, Martínez-Castillo V, Boixadera A, et al. Surgical embolus removal in retinal artery occlusion. *Br J Ophthalmol* 2006;90:1252–1255.
8. Lim JY, Lee JY, Chung HW, et al. Treatment of branch retinal artery occlusion with transluminal Nd:YAG laser embolysis. *Korean J Ophthalmol* 2009;23:315–317.
9. Lu N, Wang NL, Wang GL, et al. Vitreous surgery with direct central retinal artery massage for central retinal artery occlusion. *Eye (Lond)* 2009;23:867–872.
10. Kovach JL, Mason B. Visual recovery and OCT evolution following central retinal artery occlusion treated with intra-arterial verapamil and alteplase infusion. *Ophthalmic Surg Lasers Imaging Retina* 2015;46:77–79.