Update on the Management of Central Retinal Artery Occlusion

Michael Dattilo, MD, PhD[a,b], Valérie Biousse, MD[a,b,c,*], Nancy J. Newman, MD[a,b,c,d]

KEYWORDS

• Central retinal artery occlusion • Branch retinal artery occlusion • Stroke • Ischemia • Management • Treatment • Thrombolysis

KEY POINTS

- Acute central retinal artery occlusion (CRAO) and branch retinal artery occlusion (BRAO) are the ocular equivalent of a cerebral infarction in the anterior circulation.
- The risk factors for a CRAO or a BRAO and acute cerebral ischemia are very similar.
- Patients with acute CRAO and BRAO need to be evaluated emergently in a stroke center similar to patients with cerebral ischemia.
- Up to 24% of patients with acute retinal ischemia have concomitant cerebral infarctions on brain diffusion-weighted MRI.
- Because no current therapeutic intervention has been shown to improve visual outcome compared with the natural history of CRAO, management of CRAO should be focused on secondary prevention of vascular events, such as cerebral ischemia, myocardial infarction, and cardiovascular death.

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[a] Department of Ophthalmology, Emory University School of Medicine, 1365-B Clifton Road, Northeast, Atlanta, GA 30322, USA; [b] Neuro-Ophthalmology, Emory Eye Center, 1365-B Clifton Road, Northeast, Atlanta, GA 30322, USA; [c] Department of Neurology, Emory University School of Medicine, 12 Executive Park Drive, Northeast, Atlanta, GA 30329, USA; [d] Department of Neurological Surgery, Emory University School of Medicine, Atlanta, GA 30322, USA
* Corresponding author. Neuro-Ophthalmology, Emory Eye Center, 1365-B Clifton Road, Northeast, Atlanta, GA 30322.
E-mail address: vbiouss@emory.edu

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BACKGROUND

Central retinal artery occlusion (CRAO) was first described in 1859 in a patient with endocarditis[1] and is caused by partial or complete obstruction of the central retinal artery (CRA) leading to retinal ischemia (**Figs. 1** and **2**). The CRA originates from the ophthalmic artery, which is the first branch of the internal carotid artery. The CRA and its branches supply blood to the inner retina, including the macula and fovea. Occlusion of a branch of the CRA causes a branch retinal artery occlusion (BRAO) (**Fig. 3**). In approximately 15% to 30% of the population, a cilioretinal artery is present, originating from the posterior ciliary circulation and not the CRA, often supplying part of the macula and fovea (**Fig. 4**).[2–4] Because the cilioretinal artery does not originate from the CRA, it is spared in a CRAO. If the cilioretinal artery supplies the fovea, central visual acuity may be near normal (20/50 or better) following a CRAO, whereas peripheral vision in the effected eye will be severely impaired (see **Fig. 4**).[3,5]

CRAO and BRAO are most often embolic, and the causes and risk factors are similar to those of patients with cerebrovascular ischemic events; these are classically referred

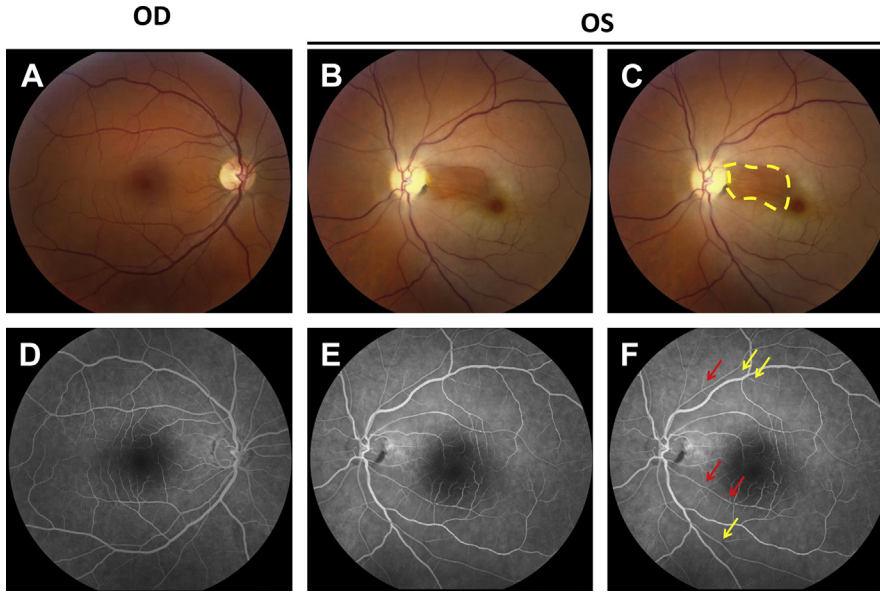

Fig. 1. Left CRAO with cilioretinal artery sparing. Color fundus photographs (*A–C*) and retinal fluorescein angiography (*D–F*) in acute CRAO. (*A*) Color fundus photograph of the right eye (OD) showing a normal appearing fundus. (*B*) Color fundus photographs of the left eye (OS) showing a CRAO with cilioretinal artery sparing. Compared with the right eye, the fundus of the left eye has a white hue, indicating inner retinal edema. The papillomacular bundle is perfused by patent cilioretinal arteries. (*C*) Same photograph as in (*B*) outlining the area perfused by the cilioretinal arteries (*area within the yellow lines*). Retinal edema is seen outside of the area perfused by the cilioretinal arteries. (*D*) Fluorescein angiography of the normal right eye taken 59 seconds from the injection of fluorescein dye into the patient's arm. The retinal arteries are of normal caliber and the retina is well perfused. (*E*) Fluorescein angiogram of the left eye taken 46 seconds after injection of fluorescein dye. The retinal arteries in the left eye are attenuated as compared with the retinal arteries in the right eye (*red arrows in* [*F*]), and areas of discontinuity in the retinal arteries of the left eye are also noted (*yellow arrows in* [*F*]).