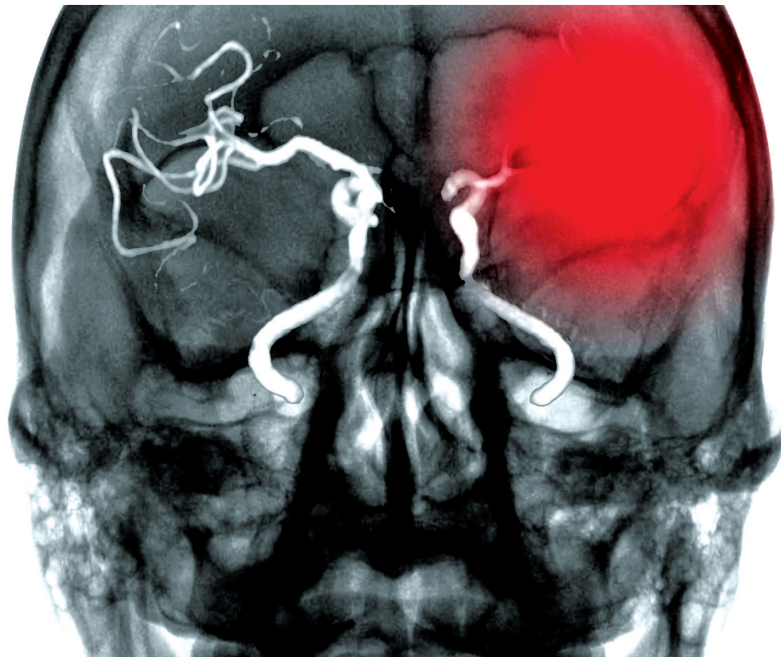




RETINAL ARTERY OCCLUSION: A SIGN OF STROKE



Know what to look for in this potentially life-threatening presentation.

BY JACQUELINE THEIS, OD, FAAO

It's a busy Friday afternoon in your office, and you're interrupted by a knock on your door. "Ms. Smith is on the phone. She says she has lost vision in her left eye. Your schedule is full for the day. Should she be scheduled for tomorrow?"

What immediately runs through your mind when you imagine this scenario? For me, the first thing I want to know is whether this is *blurry* vision or *vision loss*? Other questions follow: Was it sudden? Was it transient? Full vision loss or partial? How long has it been going on? Is there any associated pain? These are the questions I *need* to know in order to answer the receptionist's question.

In general, vision loss of sudden onset is an ominous complaint. If the patient calls your office immediately, you may not be able to establish over the phone whether the vision loss is transient and possibly due to a neurologic condition such as papilledema, vertebrobasilar artery insufficiency, or migraine; or if it is more permanent vision loss and due to a retinal vascular occlusion, ischemic optic neuropathy, ocular ischemic syndrome, giant cell arteritis, vitreous hemorrhage, retinal detachment, or optic neuritis. None of these differentials are benign, and some of them, such as retinal artery occlusion and arteritic ischemic optic neuropathy, are both sight-threatening and life-threatening.

So the short answer in this scenario is, "No, this patient needs to be seen right now."

ACUTE CLINICAL FINDINGS

If the aforementioned patient presents with acute retinal arterial ischemia from loss of vascular perfusion due to a branch retinal artery occlusion (BRAO), central retinal artery occlusion (CRAO), or ophthalmic artery occlusion (OAO), the nonperfused retina will appear acutely swollen and opacified, giving the fundus a white appearance. Due to the presence of a thin retinal nerve fiber layer within the fovea, the macula may appear pink or red despite nonperfusion, a finding commonly



Figure. Fundus photo of an eye with nonarteritic CRAO. The arteries are attenuated with surrounding retinal whitening, and the optic nerve is hyperemic and nonedematous. Note the cherry red spot appearance of the macula, commonly found in CRAO.

known as a *cherry red spot* (Figure).

Acute retinal arterial ischemia causes sudden, painless, monocular central and peripheral vision loss, which some patients may characterize as having a purplish hue.¹ The amount of central vision loss depends on the perfusion of the macula, and visual acuity in CRAO averages between 20/200 and counting fingers. Patients with a BRAO or CRAO who

have a cilioretinal artery allowing macular perfusion may present with better and even normal central visual acuity.² The prognosis for final visual outcome depends upon the duration of occlusion; if the occlusion lasts 4 hours or longer, the retinal damage is likely irreversible.^{3,4} In addition to retinal whitening, the retinal arteries may appear attenuated due to lack of blood flow and may have segmented

blood flow, referred to as *boxcarring*. The patient will have a relative afferent pupillary defect and reduced color vision in the affected eye.

Acutely, the optic nerve may appear normal, pale, or hyperemic in nonarteritic CRAO. However, in arteritic, or giant cell arteritis–related CRAO or OAO, the optic nerve may be edematous due to simultaneous disruption of the posterior ciliary artery blood flow.^{1,2}

PATHOPHYSIOLOGY

A CRAO, BRAO, or OAO is an ophthalmologic and life-threatening emergency that is pathophysiologically analogous to an ischemic stroke. CRAO is relatively uncommon, occurring in two per 100,000 individuals.⁵ The most common cause of nonarteritic CRAO is an embolic occlusion of the central retinal artery at its narrowest part, either where it enters the optic nerve sheath or where it enters the globe as it crosses the lamina cribrosa.⁶ Most often the embolus originates from an atherosclerotic plaque in the ipsilateral carotid artery or from the aortic arch of the heart. Less commonly, the CRAO can be due to a vasculitis such as giant cell arteritis, chronic systemic autoimmune disorders, or hematologic disorders.^{7,8}

STROKE RISK

Both the American Heart Association and American Stroke Association consider retinal arterial ischemia to be the equivalent of acute cerebral ischemia.⁹

A 2018 study by Lavin et al evaluated the diagnostic yield of a standardized stroke evaluation in patients with acute CRAO. The standard stroke workup included lab testing of low-density lipoprotein levels, hemoglobin A1c, erythrocyte sedimentation rate, C-reactive protein level, platelet count, and troponin level, along with imaging studies including echocardiography, cardiac telemetry, computed tomography angiography (CTA) or magnetic resonance angiography (MRA) of the head and neck, and MRI of the

AT A GLANCE

- Vision loss of sudden onset is an ominous complaint; patients should be seen right away and referred immediately when appropriate.
- Patients with acute central retinal artery occlusion (CRAO) are at heightened risk of having a subsequent stroke or heart attack and need to be sent to an ER immediately.
- Optometrists may be less likely to see an acute presentation than to see a patient with chronic findings that are suggestive of a previous CRAO.

“[T]HE BEST MANAGEMENT FOR PATIENTS WITH ACUTE RETINAL ISCHEMIA IS TO CHECK THE BLOOD PRESSURE OF THE PATIENT AND TO REFER IMMEDIATELY TO THE NEAREST CERTIFIED STROKE CENTER FOR EVALUATION.”

brain. The study found that 26.7% of patients had critical carotid disease (atherosclerosis or dissection); 37.3% had coincident acute stroke, usually on the ipsilateral side of the CRAO; 33% had a hypertensive emergency (blood pressure >180/100); and 20% had a myocardial infarction or critical structural cardiac disease. Results of the cardiovascular-cerebrovascular evaluation led to a change in medication for 93% of patients and urgent surgical intervention in 25% of patients.¹⁰

Not only do patients with acute CRAO have a higher incidence of a recent prior ischemic event such as stroke or heart attack, but they also are at higher risk of having a subsequent stroke or heart attack. This risk is highest within the first 1 to 2 weeks after acute retinal ischemia.^{11,12}

MANAGEMENT

At this time, there is no proven effective treatment to improve visual outcome after BRAO or CRAO. However, given the aforementioned statistics regarding stroke risk, the best management for patients with acute retinal ischemia is to check their blood pressure and to refer them *immediately* to the nearest certified stroke center for evaluation. A comprehensive evaluation usually includes MRA or CTA of the head and neck, diffusion-weighted imaging or MRI of the brain, cardiac evaluation including blood pressure,

electrocardiogram, echocardiogram, and cardiac monitoring, and further evaluation if an embolic origin cannot be ascertained. Treatment for these patients is focused on secondary prevention measures to decrease subsequent ischemic cardiovascular and cerebrovascular events.²

CHRONIC CLINICAL FINDINGS

The retinal opacification will usually start to resolve within a month of onset, by which time it may appear normal on clinical fundus examination. Approximately 3 months after the onset, the optic disc may appear atrophic with or without cilioretinal collateral vessels. Collateral vessels must be differentiated from neovascularization, as there are instances of anterior and posterior segment neovascularization and neovascular glaucoma developing after retinal arterial occlusions.²

In the aforementioned study by Lavin et al, subsequent follow-up of patients at 1 year after their CRAO found that 25.3% of patients had had a stroke or myocardial infarction or had died.¹⁰ This percentage is higher than the 6.2% rate found in patients 1 year after transient ischemic attack or minor stroke,¹³ and it remains higher than that found in the general population for up to 10 years after a CRAO.²

Optometrists may be less likely to see an acute presentation than to see a patient with chronic findings that

are suggestive of a previous CRAO, such as collateral vessels on the disc. When we see such chronic findings, knowing the increased combined risk of stroke, myocardial infarction, and death in this population, it is important to ask the patient if he or she has had a systemic ischemic (cerebrovascular-cardiovascular) evaluation. If not, check the patient's blood pressure and refer this patient to his or her primary care physician.

RECOGNIZE AN EMERGENCY

Acute retinal arterial ischemia (ie, transient monocular vision loss, CRAO, BRAO, and OAO) is considered equivalent to acute cerebral ischemia (ie, transient ischemic attack and stroke). On presentation, these patients must be immediately evaluated and sent to the nearest certified stroke center to prevent subsequent stroke, heart attack, or death. ■

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JACQUELINE THEIS, OD, FAAO

- Optometrist, Virginia Neuro-Optometry, Richmond, Virginia
- Affiliate Assistant Clinical Professor, UC Berkeley School of Optometry
- drtheisod@virginianeurooptometry.com
- Financial disclosure: None