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Sudden painless monocular visual loss

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A 58-year-old African-American female presented to the ophthalmology clinic from the local emergency department with a history of sudden visual loss in her right eye.

The patient originally presented to the emergency department with the complaint of sudden painless monocular visual loss in her right eye. The ophthalmology clinic was notified of the patient's complaint and she was sent to the clinic for evaluation. Upon arrival the patient was subsequently diagnosed with a branch retinal artery occlusion. The patient was admitted to the hospital for a systemic work up by her primary care physician.

History

A 58-year-old African-American female presented to the ophthalmology clinic from the local emergency department with a history of right eye (OD) sudden visual loss since 9:00 am that morning, when she woke up. The patient described her vision as "complete blackness." The first episode lasted 10 minutes followed by an additional three episodes within the first hour of awakening. The patient denied any other associated symptoms—pain, photophobia, flashes of light, floaters, headache, jaw claudication, weight loss, scalp tenderness, or trauma.

Previous history

The patient's past medical history included uncontrolled hypertension. She was taking lisinopril. She had an allergy to penicillin and tetracycline. Her past surgical history included a total abdominal hysterectomy with bilateral oopherectomy. Her family history included diabetes mellitus and myocardial infarction in both of her parents. She was a nonsmoker, denied alcohol or illicit drug use and was retired.

Visual examination

Visual acuity in the OD was 20/30 and 20/30 –2 in her left eye (OS), with no improvement with pinhole. She was able to identify 11/11 Ishihara plates. The confrontational field in the OD revealed a superior nasal deficit and was full in the OS. Pupils were normal with no afferent papillary defect. Slit-lamp examination revealed anterior marginal blepharitis, normal conjunctiva, clear corneas, and mild cortical cataracts in both eyes (OU). Applanation tonometry at 3:30 pm revealed an intraocular pressure of 16/17. The vitreous was clear (OU) and retinal examination revealed normal-appearing optic nerves measuring .3/.3 OU with normal color and contour and no evidence of ischemia. The artery-to-vein ratio was 1:2 and veins were normal; examination of the OD inferior temporal arcade revealed emboli with decreased flow antegrade to the emboli. No edema was evident in the surrounding area.

The emergency department staff ordered a computed tomography scan of the head at 12:06 pm, which revealed

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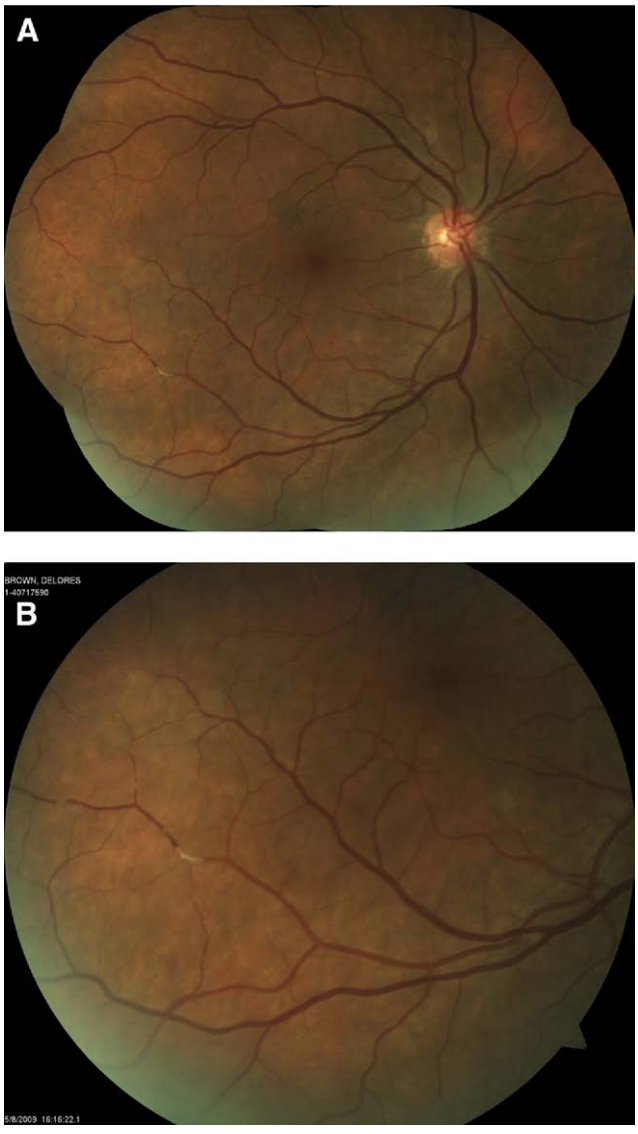

Figure 1 (a) BRAO with decreased flow below the obstruction. (b) Close-up view of the BRAO and boxcarring of the artery.

no acute processes. The ophthalmology clinic was notified at 2:55 pm of the patients' complaint and was sent over to the clinic with the instruction to perform digital massage. Upon arrival, the patient was subsequently diagnosed with a branch retinal artery occlusion (BRAO) (Figure 1a). She was admitted to the hospital for a vascular work up, magentic resonance imaging/angiography, and an echocardiogram.

Discussion

The purpose of this case report is to review an ophthalmologic emergency that may present to your primary care office or emergency department including the natural history, typical presentation and management of a BRAO and also central retinal artery occlusion (CRAO).

Blood supply to the retina originates from the ophthalmic artery. The ophthalmic artery is the first intracranial branch off of the internal carotid artery. The ophthalmic artery supplies the eye via two branches: the central retinal artery and ciliary arteries. The central artery supplies the retina via the superior and inferior intraretinal branches. A notable variant is the presence of a cilioretinal branch from the short posterior ciliary artery that gives collateral flow to the foveal area. This variant is present in approximately 14% of the United States population. An embolism occurs when an object migrates from one blood vessel and causes an occlusion of another vessel. This is in contrast to a thrombus, or clot, which forms at the blockage point within a blood vessel and is not carried from somewhere else. Several types of embolism exist—thromboembolus, an embolism of thrombus or blood clot, an embolism from a cholesterol plaque often resulting from atherosclerosis, and others such as fat, air, and septic emboli.

CRAO is a somewhat rare event, with an incidence of approximately 1 to 10 in 100,000.[1] Symptomatic BRAO is even less common. Demographic characteristics of patients with CRAO and BRAO are consistent with those seen for other vascular disorders.

CRAO usually presents with sudden, complete, painless visual loss in one eye. The retina becomes opaque and edematous and the intact choroidal vasculature beneath the foveola stands out in contrast to the surrounding opaque neural retina, thus producing a cherry red spot (Figure 2). In time, the central artery recanalizes and the retinal edema resolves. Visual outcome is dependent on presence of a cilioretinal artery that spares the central macula and time to treatment.[2]

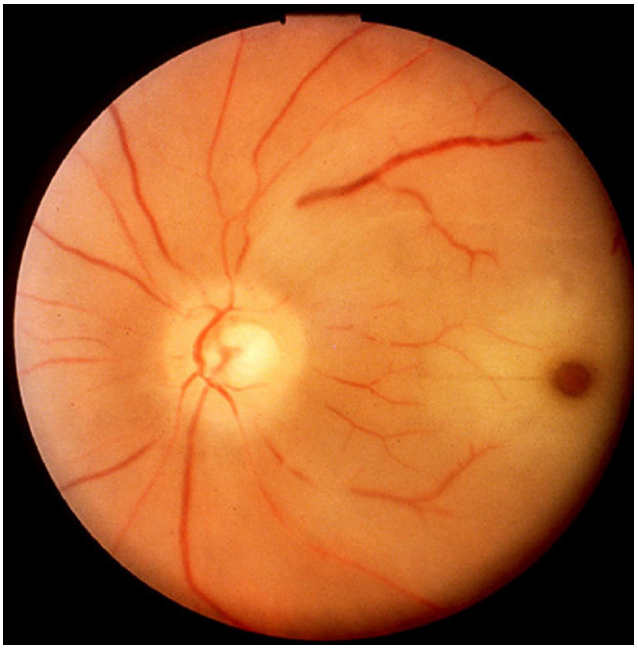

Figure 2 Cherry red spot. (Courtesy Thomas R. Hedges, III, MD).

BRAO is also a fairly rare ocular disorder. It can be grouped in several ways including permanent or transient, via location, and by the produced visual field defect. A BRAO occurs when an embolus flows from the central retinal artery to a more distal branch. Of recurring occlusions, BRAO is less common (38%) than CRAO (57%).

The patient presentation can vary; however, the most common presenting complaint is acute painless visual loss, which is only in a section of the peripheral vision depending on the location of the occlusion. The visual symptoms may originate with central visual loss and change depending on the final location of the emboli. The defect may be an altitudinal defect affecting the upper or lower hemifield but never respecting the vertical axis.

Fundus examination reveals an edematous opacification and whitening of the retina along the distribution of the occluded vessel within hours to days. Cotton-wool spots may be present and are limited to the area of the retina supplied by the occluded vessel. Emboli can be seen in approximately 20% to 40% of patients. Boxcarring is a sign of severe occlusion and slowing circulation. In the acute phase, the blood column in the artery becomes segmented with separation of serum from Rouleau stacking of the red corpuscles, leading to a boxcarring appearance of the blood column (Figure 1b). As with CRAO, the affected vessel recanalizes over time and perfusion returns, and the edema resolves with the visual field defect remaining.

Once a CRAO/BRAO is diagnosed, a work-up should be done to determine the etiology of the cause, although the yield is relatively low. A complete blood count, erythrocyte sedimentation rate, C-reactive protein (giant cell arteritis accounts for 1–2%

of CRAO cases), prothrombin time, partial thromboplastin time, fasting lipid panel and, if indicated, blood cultures should be ordered.[3] Imaging studies are also helpful in determining the etiology (e.g., echocardiogram to look for vegetations, valvular disease, and thrombi). Carotid Dopplers or magnetic resonance imaging should be ordered to evaluate the carotid circulation for atherosclerosis. An electrocardiogram should be done, or a 24-hour Holter monitor worn, to rule out atrial fibrillation.[4]

Treatment

Prognosis is related to the timeliness of treatment and the presenting visual acuity.[5,6] Depending on the location of the emboli, certain intervention may be beneficial, but efficacy is questionable.[7] Most patients (80%) with BRAO recover normal vision, whereas spontaneous clinical improvement from CRAO is rare.[8]

Hayreh studies concluded that in young healthy rhesus monkeys, CRAO retinal damage was reversible if the duration lasted for 97 to 98 minutes, but the retina suffers irreversible damage after 105 minutes.[9]

The retina of old, atherosclerotic, and hypertensive rhesus monkeys suffers no detectable damage with CRAO of 97 minutes duration, but suffers progressively more irreversible damage with increased duration. These findings are counterintuitive and may relate to the type of anesthesia used or hypoxic preconditioning.

The study suggested that a CRAO lasting 240 minutes results in massive, irreversible retinal damage.[10]

The general consensus from an evidence-based and medical legal aspect is to intervene by 90 minutes.[2] Ocular massage can be performed by applying direct pressure for 5 to 15 seconds, releasing, and repeating several times. Increased intraocular pressure causes a reflexive dilation of retinal arterioles by 16%. A sudden drop in intraocular pressure with release increases the volume of flow by 86%. Ocular massage may dislodge the embolus to a point farther down the arterial circulation and improve retinal perfusion, but the efficacy in improving visual outcome is unknown.

Anterior chamber paracentesis is advocated when visual loss has been present for less than 24 hours. Early paracentesis is thought to increase visual recovery by decreasing the intraocular pressure and allowing greater perfusion and pushing emboli farther down the vessel. This can be performed with a 27- to 30-gauge needle, with removal of 0.1 to 0.4 mL of aqueous humor, without decreasing the pressure lower than 4. There may be a marginal visual benefit associated with local intra-arterial fibrinolysis compared with conventional management of CRAO,[11-13] and hyperbaric oxygen may be of benefit. The data suggest that anterior chamber carbogen therapy offer little benefit for treating acute nonarteritic CRAO.[14]

Presence of a cilioretinal artery with foveolar sparing increases improvement in end outcome visual acuity. Ischemic retinal damage also may arise from oxidative damage and membrane damage once retinal tissue is reperfused.[2]

Long-term management focuses on preventing recurrent vascular events. Our patient's work-up revealed uncontrolled hypertension, dyslipidema, and diabetes. Her electrocardiogram, magnetic resonance imaging/angiogram were normal. Although no definitive etiology was evident we counseled the patient to control her hypertension and dyslipidemia, with the hope of decreasing the likelihood of future occurrences.

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