Case Report

Unilateral central retinal artery occlusion in alcoholic cardiomyopathy

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ABSTRACT

Central retinal artery occlusion (CRAO) is an ocular emergency that may result in significant functional morbidity. Emboli of cardiac origin are an uncommon cause of retinal artery occlusion and a variety of cardiac disorders including dilated cardiomyopathies may be the source. We present a case of sudden unilateral vision loss in a 38-year-old man with alcoholic cardiomyopathy (ACM). Ocular examination revealed symptoms of a right CRAO. His visual outcome was poor despite emergency measures taken at presentation. This case should increase the awareness of CRAO as a possible embolic complication in patients with dilated cardiomyopathy.

Key words: *Alcoholic cardiomyopathy (ACM), dilated cardiomyopathy, retinal artery occlusion*

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Introduction

Central retinal artery occlusion (CRAO) is a rare disease with an incidence of 1:100,000.[1] It is an ocular emergency that results in significant functional morbidity with more than 75% of sufferers having a visual acuity (VA) of 20/400 or worse in the affected eye.[2,3] Occlusion can result from atherosclerosis-related thrombosis, embolic impaction, vasculitis, vasospasm, and systemic hypotension.[4] Emboli of cardiac origin are an uncommon cause of retinal artery occlusion and a variety of cardiac disorders including dilated cardiomyopathies may be the source.[5,6] Alcoholic cardiomyopathy (ACM) is a type of nonischemic dilated cardiomyopathy that develops following long-term heavy alcohol consumption.[7] Similar to other types of dilated cardiomyopathies (e.g., idiopathic, viral/immune), ACM is characterized by a dilated left ventricle, normal or reduced left ventricle wall thickness, and increased left ventricle mass.[7] Patients with dilated cardiomyopathy are at increased risk of thromboembolic events including symptomatic or silent peripheral arterial embolism, pulmonary embolism, and stroke.[8,9] There are, however, very few reports of retinal artery occlusion occurring in patients with cardiomyopathies,[6,10] and we did not come across any specific case of retinal artery occlusion occurring in a patient with ACM. Here, we report the case of a 38-year-old man with ACM who developed a right CRAO most probably arising from an embolus.

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Case Reports

The 38-year-old man presented to the eye clinic in June 2014 with a 6-hour history of sudden painless loss of vision in his right eye that was experienced on waking up in the morning. There was no history of photopsia, floaters, or prior transient loss of vision in the eye. He had no history of hypertension, diabetes, or sickle cell disease but had been on treatment for ACM for 2 years prior to presentation and was taking spironolactone, digoxin, lisinopril, and torsemide tablets. He was not on any anticoagulant. On examination, his right eye had a visual acuity (VA) of light perception with poor light projection and a relative afferent pupillary defect. Dilated funduscopy revealed a pink disc with a cup disc ratio of 0.4, with attenuated and segmented arterioles. The retina was pale and the macula was edematous with a cherry-red spot. No embolus was visualized. His left eye had a best corrected VA of 6/9. The anterior and posterior segments were essentially normal. The intraocular pressures were 10 mmHg and 9 mmHg in the right eye and the left eye, respectively. His pulse

rate was 96 beats/min, regular, good volume, while his blood pressure was 100/70 mmHg. A diagnosis of right CRAO was made. The affected eye was massaged, and he was placed on 250 mg of acetazolamide tablet every 8 h and 75 mg of clopidogrel tablet each day. He was then asked to see his cardiologist. There was, however, no immediate improvement in his vision with these measures as the VA remained the same after 24 h.

Blood investigations revealed that full blood count, erythrocyte sedimentation rate, fasting blood glucose, and lipid profile were all within normal limits. He had an electrocardiogram done that revealed biatrial enlargement with left ventricular hypertrophy, left axis deviation, and nonspecific conduction defect. Transthoracic echocardiography showed severely dilated cardiac chambers, left ventricular systolic dysfunction (ejection fraction 18%), globally hypokinetic left ventricle, severe tricuspid valve regurgitation, severe mitral valve regurgitation, and mild-to-moderate pulmonary valve regurgitation. The aortic valve had mild calcification with normal mobility. The other valves were normal morphologically. No pericardial effusion or intracardiac mass was seen.

One week later, vision in the right eye had improved to counting fingers at 2 m. As per the funduscopy report, the disc had become pale, the arterioles had recanalized, and there were few flame-shaped and dot retinal hemorrhages [Figure 1]. Fundus fluorescein angiography (FFA) and optical coherence tomography (OCT) could not be done as facilities for these were not available. He was last followed up 4 months after presentation with VA in the right eye, and his condition remaining unchanged. He had no evidence of retinal or iris neovascularization, and the macular edema had resolved. The intraocular pressure was normal.

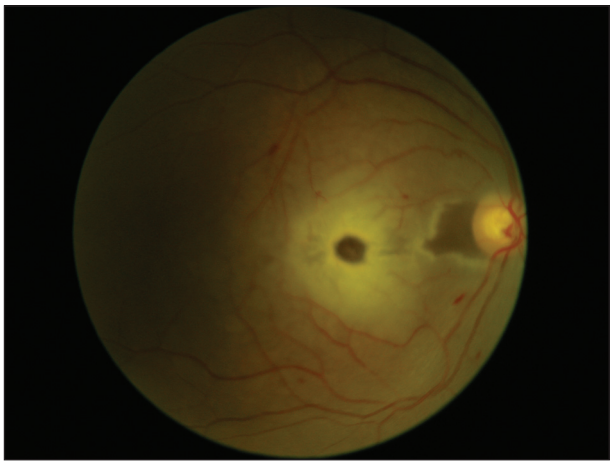

Figure 1: Right central retinal artery occlusion (CRAO) showing cherry-red spot, attenuated vessels, and few flame-shaped hemorrhages

Discussion

The occurrence of CRAO in this patient is highly suggestive of an embolic event even though an embolus was not visualized on retinal examination. Emboli are biomicroscopically detectable in only 20% of the cases with CRAO.[5] The calcifications on the aortic valve are the most likely source. Dilated cardiomyopathy patients present an important thromboembolic risk for reasons that include relative stasis of blood in dilated cardiac chambers, poor contractility and regional wall motion abnormalities, and concomitant atrial fibrillation.[8] Peripheral venous or right ventricular thrombus may lead to pulmonary emboli, whereas thromboemboli of left ventricular origin may lodge in any systemic artery, resulting in, for example, devastating cerebral, myocardial, and renal infarctions[11] as well as retinal infarction as in this case.

Acute CRAO presents with a sudden, painless loss of vision, unless a cilioretinal artery is present in which case central vision may be preserved.[1] Ocular findings are based upon funduscopy, FFA, and OCT. These findings vary based on time from event and type of CRAO. Early findings on funduscopy include retinal opacity in the posterior pole (58%), cherry-red spot (90%), cattle trucking (19%), retinal arterial attenuation (32%), optic disc edema (22%), and pallor (29%). All but optic disc edema was seen in our case. At the later stages, optic atrophy, retinal arterial attenuation (91%), cilioretinal collaterals (58%), and macular retinal pigment epithelial changes (11%) are seen on funduscopy.[12] Angiographic findings associated with CRAO include delayed filling of the affected vessels, reduced arterial caliber, and "cattle trucking" of the blood column in the branch arteries.[4] Fluorescein angiography is, however, not routinely indicated in the acute phase of arterial occlusive disease.[4] Findings from OCT may show increased inner retinal layer thickness in the acute stage due to retinal edema and optic nerve swelling.[13]

Presently, there is no guideline-endorsed evidence for the treatment of CRAO though a number of therapeutic interventions have been proposed.[1] The aim of the treatment is to increase the perfusion pressure of the retinal circulation, or to dislodge or lyse the obstructing thrombus/embolus.[4] The various treatment options

are as follows: Ocular massage; anterior chamber paracentesis; breathing a high oxygen (95%) and carbon dioxide (5%) mixture (carbogen); use of pharmacological agents (e.g., intravenous acetazolamide), hyperosmotic agents (e.g., mannitol, sublingual isosorbide dinitrate, etc.), and thrombolytics (e.g., tissue plasminogen activator).[1,5] For these treatments to be effective, however, they must be instituted within a few hours of occlusion of the retinal artery. Retinal ischemic tolerance time appears to be between 4 h and 6.5 h before any irreversible damage occurs.[14,15] Thus, any treatment instituted much later than 6 h from the time of loss of vision is not likely to restore the vision. Visual improvement was, therefore, unlikely in this case despite the measures taken.

The incidence of CRAO in this patient may have been prevented if he had been on an anticoagulant. There is however controversy about the necessity of routine anticoagulation in patients with dilated cardiomyopathy in sinus rhythm.[16,17] The only clear-cut indications for anticoagulation in most of the patients with dilated cardiomyopathy are atrial fibrillation, a previous thromboembolic event or a left ventricular thrombus.[8]

Visual prognosis for CRAO is generally poor due to retinal infarction.[5] Sixty one percent of the patients will achieve a VA of counting fingers or worse at the final visit, whereas only 16% will have a VA of 20/40 or better.[3] Affected eyes are also at risk of developing ocular neovascularization and neovascular glaucoma.[1,5] Rudkin *et al*.[18] showed that ocular neovascularization occurs at approximately 8 weeks, with a range of 2-16 weeks, so monitoring at 2-week intervals from CRAO occurrence up to 4 months post-CRAO would appear prudent.[15]

Conclusion

In conclusion, CRAO is a time-critical pathology, and the case reported here should heighten the awareness regarding the occurrence of this pathology as a possible embolic event in nonanticoagulated patients with dilated cardiomyopathy.

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