CASE BASED LEARNING POINTS DOI: https://doi.org/10.18502/fem.v7i1.11700

Central retinal artery occlusion presenting with headache and sudden painless blurring of vision

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1. Case presentation

The patient was a 61-year-old smoker male, who presented to emergency department (ED) with complaints of sudden onset of headache followed by painless blurring of vision of the right eye that was started 10 hours prior to the admission. Due to blood pressure of 190/104 mmHg at home, the patient had taken amlodipine 10mg orally. The patient reported some episodes of transient ischemic attacks in his past medical history, for which he did not take any advice from physicians. The patient was also found to be hypertensive with deranged cholesterol. On examination in ED, the patient was afebrile, and had pulse rate= 88/min, blood pressure (BP)= 130/90 mmHg, respiratory rate=22/min, and O2 Saturation=99% in room air. There was not any positive finding in systemic examination.

Patient was admitted for further evaluation and management. Paraclinical lab tests were all reported in normal range. Echocardiography revealed left ventricular ejection fraction (LVEF) of 60%, with no regional wall motion abnormality (RWMA), mild concentric left ventricular hypertrophy (LVH) and normal cardiac chambers. In view of Headache, brain computed tomography (CT) scan was performed, in which, there was prominence of sulci, basal cistern, sylvian fissure and ventricular system suggestive of age-related diffuse cerebral atrophy. Ill-defined hypodensities were seen in bilateral periventricular white matter, suggestive of chronic ischemic changes.

Later, brain magnetic resonance imaging (MRI) was also performed, which revealed multiple discrete and confluent areas of hyperintensity scattered in subcortical deep and periventricular white matter of both cerebral hemispheres, suggestive of nonspecific small vessel ischemic changes, likely a combination of ischemic demyelination chronic lacunar infarcts and prominent perivascular space. The ventricular system and subarachnoid space were prominent, suggestive of age-related cerebral atrophy (Figure 1).

In the next step, cervical and brain MRI angiography was performed, which revealed 100% occlusion of right internal carotid artery at its origin, with no distal reformation of the artery in the neck and intracranial part (Figure 2). The right middle and anterior cerebral artery were filling via circle of Willis and were severely diffusely narrowed in calibre. There were mild atheromatous changes in the left common carotid artery and carotid bulb causing mild narrowing. Bilateral vertebral arteries were normal. There was evidence of diffuse severe narrowing and poor visualization of entire left anterior cerebral artery.

Ophthalmology reference was taken and fundus examination was done. On examination, the patient was found to have finger counting close to face with no improvement with glasses. In the right eye, anterior segment examination showed relative afferent pupillary defect (RAPD), while fundus examination revealed retinal background pale white with cherry red spot in macula and absent venous pulsation in the right eye, suggestive of Central Artery Retinal Obstruction (CRAO), and thread like blood vessels and Grade II Hypertensive retinopathy (Figure 3).

After starting the low molecular weight heparin, antiplatelet and steroid, vision improved from finger counting close to face to finger counting at 3 feet distance. Patient was later discharged under follow-up for further recovery.

2. Learning points

Central retinal artery occlusion (CRAO) is an ocular emergency (eye stroke) and is comparable to cerebral stroke, associated with monocular vision loss (1). CRAO occurs when the site of blockage is at or just proximal to lamina cribrosa of the optic nerve, narrowest part of the central retinal artery, where it pierces the dural sheath of the optic nerve (2). CRAO was first described by von Graefe in 1859 as an embolic occlusion of the central retinal artery in a patient with endocarditis. Its major cause is atherosclerosis in about 80% of cases, the retinal artery occlusion is either thrombotic or embolic in nature. Retinal emboli are usually of three types: calcific, cholesterol, and platelet fibrin. Inflammation, trauma, and arterial spasm are the other causes (3). Risk factors for CRAO are similar to stroke and heart disease, e. g. factors that promote atherosclerosis. Thus, patients who present with CRAO may be at risk of ischemic cerebral stroke; therefore, physicians' role is to not only manage CRAO and restore vision, but also evaluate the patient for risk factors that may lead to atherosclerosis and other vascular conditions like cerebral stroke (4). Patients with CRAO characteristically present with an acute, painless loss of vision, and 80% of affected patients

have a final visual acuity of only counting fingers close to the eye or worse along with resultant infarction of the retina due to loss of blood supply to the inner retinal layers (5). Once the central retinal artery is occluded, the recovery of retina depends on dislodgement of embolus or thrombus and also on the retinal tolerance time (6).

In the past, numerous therapies have been used in the treatment of CRAO but none of them have been shown to alter the natural course of disease. Therapies used include acetazolamide infusion, carbogen inhalation, paracentesis, and ocular massage, as well as various vasodilators such as glyceryl trinitrate. Thrombolytic therapy, either intravenously or intra-arterially, has also has been used in this regard. But one thing is common in various studies, which is in order for the treatment to be effective in CRAO, it must be deployed within a short time window, probably within 6 hours of symptom onset (7). Thus, CRAO is a disease that does not have definitive treatment, but needs to follow the same principles of treatment like other vascular end organ ischaemic diseases. That is, to provide thrombolysis therapy to re-perfuse ischemic tissue as soon as possible and to institute early secondary prevention modalities.

3. Declarations

3.1. Acknowledgment

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3.2. Authors' contribution

The authors meet the four criteria for authorship based on the recommendations of the International Committee of Medical Journal Editors (ICMJE).

3.3. Conflict of Interest

None.

3.4. Funding

None.

3.5. Consent for publication

The patient's written consent was obtained for the publication of this article.

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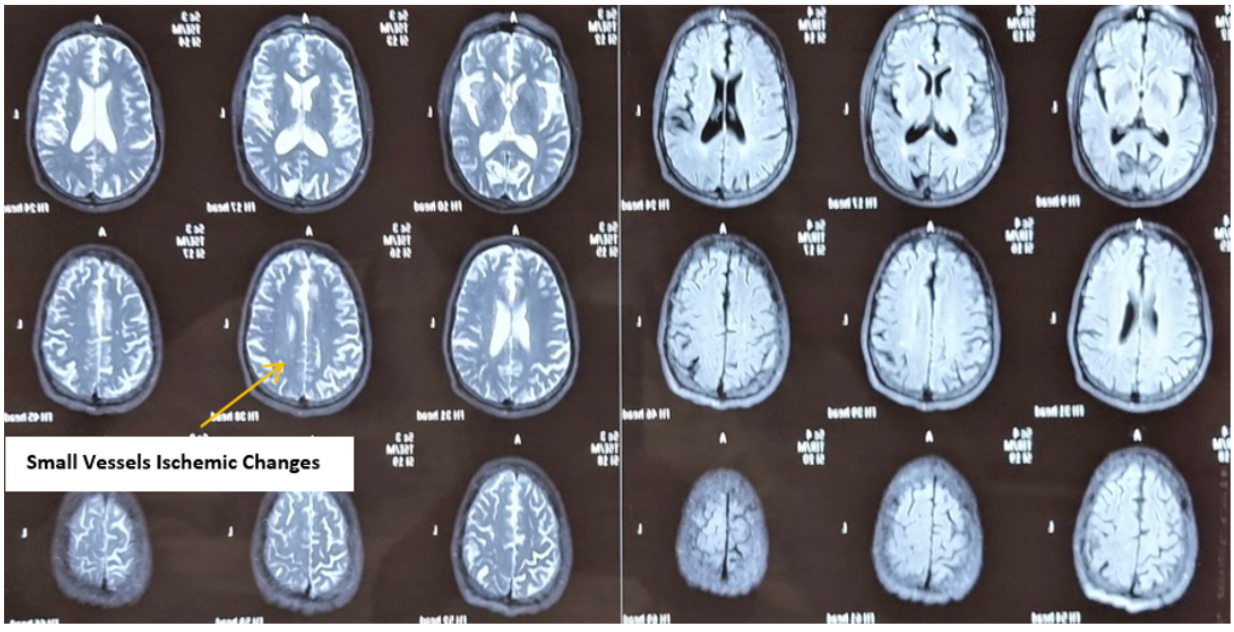

Figure 1 T1 and T2 axial views of the patient's brain magnetic resonance imaging (MRI) revealed multiple discrete and confluent areas of hyperintensity scattered in subcortical deep and periventricular white matter of both cerebral hemispheres, suggestive of nonspecific small vessel ischemic changes.

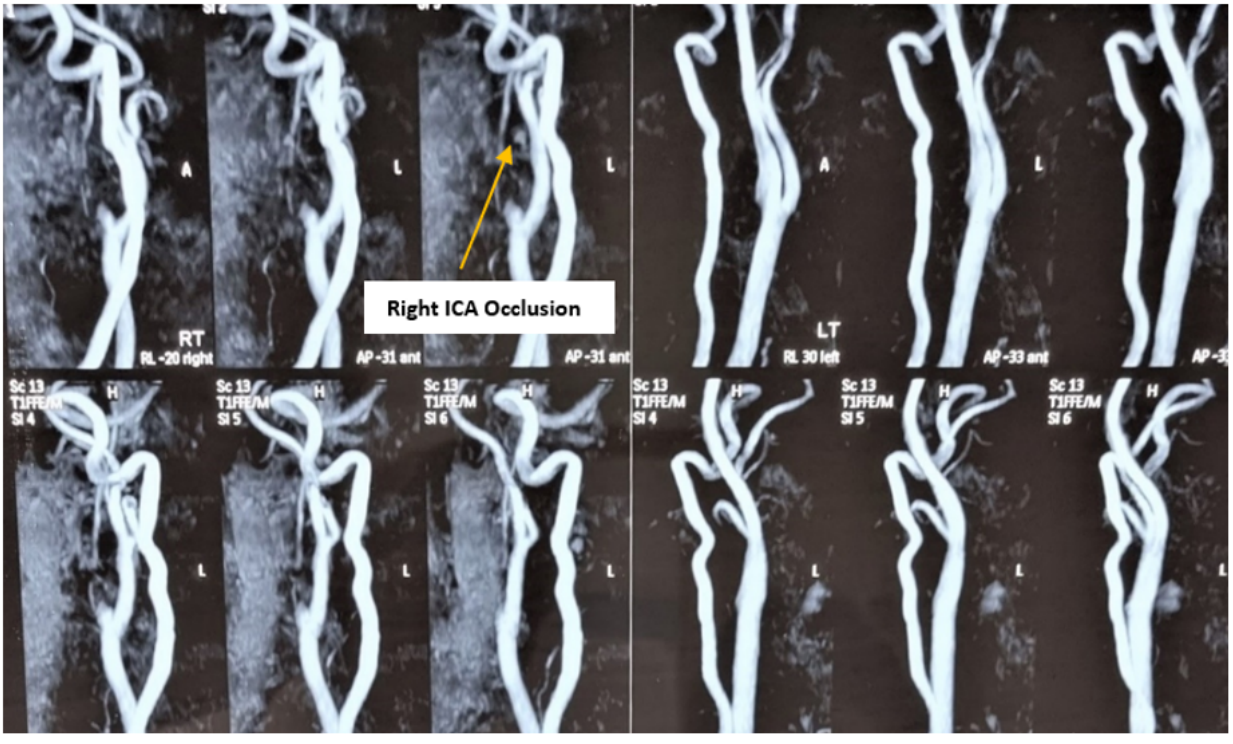

Figure 2 Cervical and brain magnetic resonance imaging (MRI) angiography showed 100% occlusion of right internal carotid artery at its origin, with no distal reformation of the artery in the neck and intracranial part.

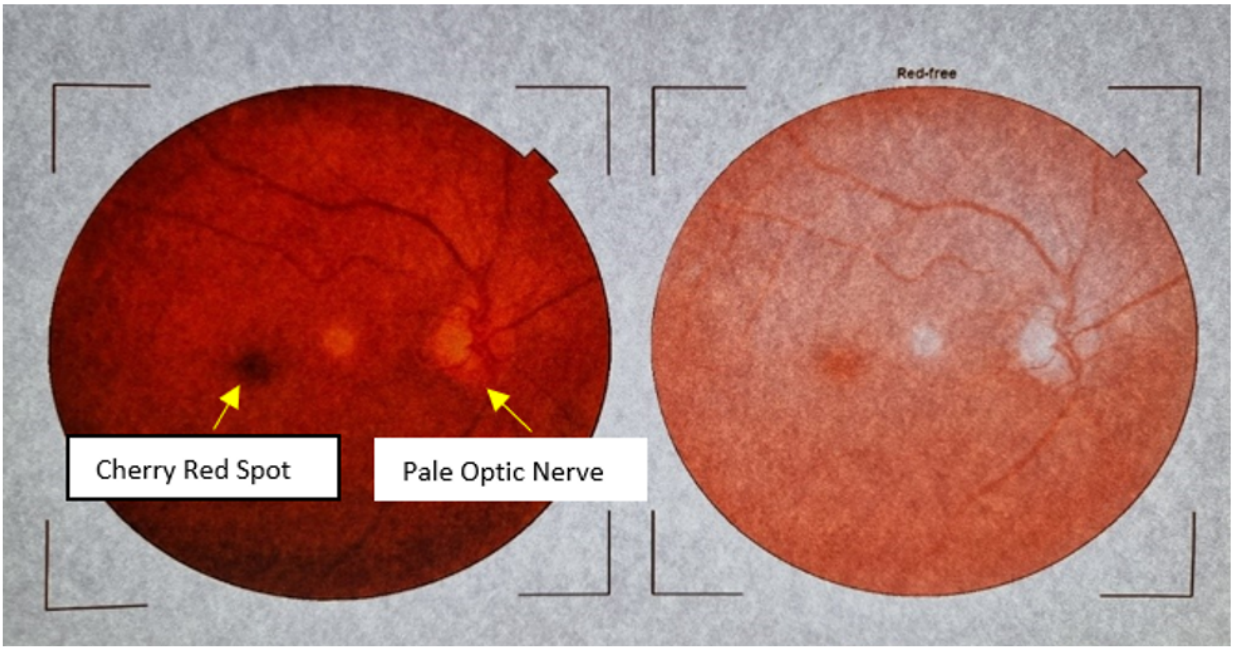

Figure 3 Fundus examination revealed retinal background pale white with cherry red spot in macula.