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Case Report

CRAO: Early Indicator of Stroke

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Abstract

Central retinal artery occlusion (CRAO) is an ophthalmic emergency and warrants a detailed vascular evaluation besides appropriate ocular management. Further, these patients are at a high risk of secondary ischaemic events. The incidence rate of ischemic stroke is about 20 times higher within a month and 70 times higher within one week after CRAO occurrence. Hence, the optimal management of CRAO needs to address restoration of perfusion to CRA (if possible), prevent neovascular ocular complications and investigate systemic atherosclerotic risk factors to reduce ischaemic events to end organs.

We report a case of a young male with no systemic co-morbidity who presented with acute CRAO and subsequently suffered cerebral stroke two days later.

Keywords: *Central Retinal Artery Occlusion (CRAO); Ischemic Stroke; Vaso-occlusion*

Introduction

Central retinal artery occlusion (CRAO) is an ophthalmic emergency presenting with sudden, severe, painless monocular visual loss and can be an early harbinger of impending occlusive systemic sequelae like ischemic heart disease or cerebral stroke [1]. In addition to the similarities in pathogenesis, CRAO and stroke share risk factors and also show similar incidence patterns with respect to age (peaks at age 80 - 84 years) and gender (predominance in men) [2-3].

Two recent, retrospective, hospital-based, cross-sectional studies have documented a 25% incidence of concurrent stroke with retinal ischemia, corroborated by diffusion-weighted MRI [4,5].

Occurrence of CRAO in young age is relatively rare and often associated with medical co-morbidity like hyperhomocysteinemia, temporal arteritis, collagen diseases and coagulation disorders like sickle cell disease and migraine [6].

Case Report

A 35-year-old male presented with sudden onset loss of vision in right eye of 12 hours duration, noted during driving at night. The patient was a commercial auto driver by occupation. Vision in right eye (OD) was light perception with projection of rays (PR) accurate only in temporal quadrant. Examination revealed right relative afferent pupillary defect, quiet anterior segment with intraocular pressure of 16mm Hg (Goldmann applanation tonometry). Retinal examination documented optic disc edema, retinal edema at posterior pole, arteriolar attenuation with "*cattle trucking*" of vessels and "*cherry red spot*" at macula suggestive of CRAO. No intra-arterial emboli could be visualized (Figure 1a).

Left eye (OS) examination was normal, with BCVA of 20/20 (Log MAR 0.00), no evidence of hypertension or vascular occlusive crisis on fundoscopy (Figure 1b). A clinical diagnosis of right eye CRAO was made and anterior chamber paracentesis was done in emergency setting under aseptic conditions followed by carbogen inhalation for two hours. Patient was admitted for monitoring and intravenous hyperosmotic therapy was instituted to reduce vitreous pressure in an endeavour to re-establish retinal perfusion. No visual gain was evident over a 48 hour hospital stay.

During second day of hospital stay, patient complained of weakness in his left upper limb. A neuro consult and neuroimaging (NCCT head) documented ill-defined white matter edema in right parietal and occipital regions (Figure 2). A diagnosis of cerebral stroke with right ACA/ MCA watershed infarcts with left upper limb monoplegia was made.

A thorough work-up to assess the risk factor(s) of stroke was done; no history of tobacco consumption, diabetes, hypertension, dyslipidemia or pre-existing vaso-occlusive disorders could be elicited. Blood investigations viz. CBC, ESR, CRP, LFT, KFT, serum lipid profile, PT-INR were within normal limits. ECG, 2D Echo, carotid doppler and CT angiography also revealed no abnormalities. Serum homocysteine levels were normal. Coagulation profile (factor V Leiden, protein C and S) was non-contributory. Patient was put on anticoagulation therapy- oral aspirin 75mg OD and clopidogrel 75mg OD. Fundus fluorescein angiography at two weeks post occlusion revealed retinal reperfusion OD and normal perfusion OS (Figure 1c, d). One month post ischemia, retinal and disc edema OD had subsided and BCVA improved marginally to hand movement close to face (HMCF) with PR accurate only in temporal quadrant.

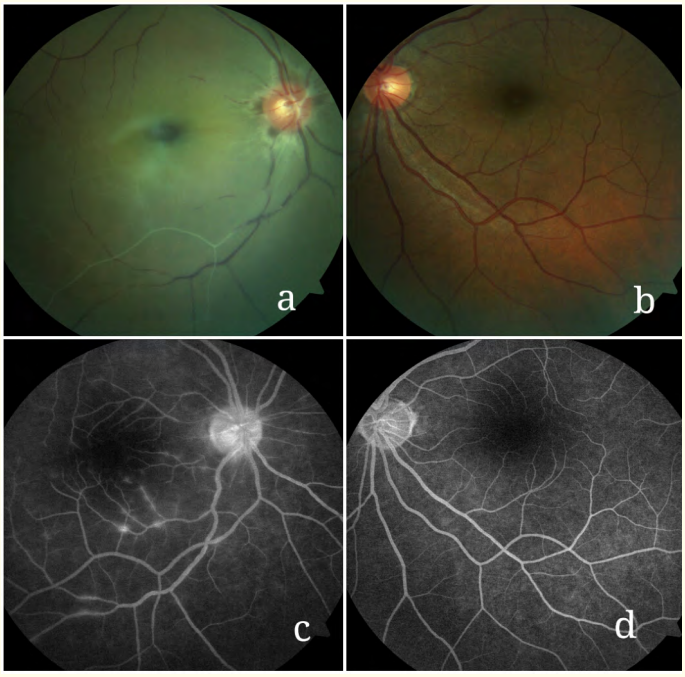

Figure 1: *Retinal examination suggestive of CRAO right eye (a), and normal fundus left eye (b); Fundus fluorescein angiography at two weeks post occlusion revealing retinal reperfusion OD (c), and normal retinal perfusion OS (d).*

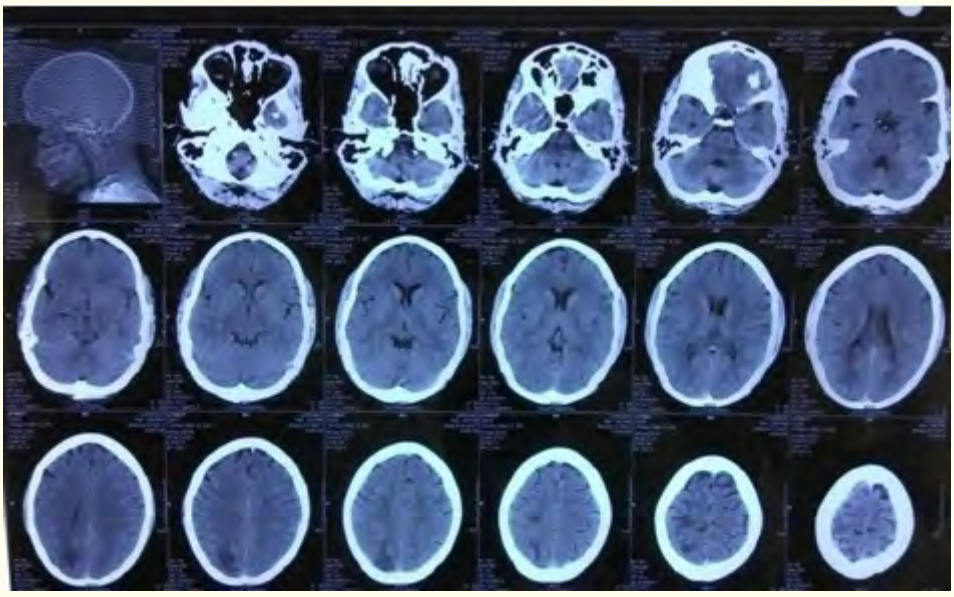

Figure 2: *NCCT head showing ill-defined white matter edema in right parietal and occipital regions.*

Discussion

Central retinal artery occlusion (CRAO) is a visually disabling ophthalmic emergency causing profound monocular visual loss, it results in 80% of patients having a visual acuity of 20/400 or worse [1].

Risk factors include diabetes mellitus, arterial hypertension, transient ischaemic attacks (TIAs) or cerebral vascular accidents, carotid artery disease, coronary artery disease, tobacco consumption and giant cell arteritis. In patients younger than 50 years; pro-atherogenic states (hyperhomocystenemia, factor V Leiden, protein C/S, anti-thrombin deficiencies), anti-phospholipid antibodies or prothrombin gene mutations, sickle cell disease, migraine (vasospasm) and paraneoplastic syndromes all contribute to non-arteritic CRAO [6].

CRAO and other types of sudden monocular vision loss such as branch retinal arterial occlusion or retinal transient ischemic attack are known to increase the risk of silent brain infarcts. The incidence of ischemic stroke has been found to be almost 20 times higher within one month and phenomenally about 70 times higher within a week after CRAO occurrence [7].

Hence, the optimal management of CRAO not just needs to address restoration of ocular perfusion to the CRA (if possible), but also prevent secondary neovascular complications to the eye and investigate systemic atherosclerotic risk factors to reduce secondary ischemic events to other end organs. Patients presenting with CRAO often have a previously undiagnosed vascular risk factor that may be amenable to medical or surgical treatment and hence help prevent a secondary ischemic event. However, no preventable or treatable risk factor was found in our patient.

The importance of a systemic workup, specifically the urgency of a stroke evaluation, should not be undermined as the highest window of stroke risk is within the first week.

Conclusion

Central retinal artery occlusion warrants a detailed vascular evaluation besides appropriate ocular management. Further, these patients are at a high risk of secondary ischaemic events, so risk factor modification is prudent.

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Conflict of Interest

None.

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