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A Rare Case of Combined CRAO and Cilioretinal Vein Occlusion due to Carotid Artery Disease

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ABSTRACT

Central retinal artery occlusion (CRAO) is an ophthalmic emergency and the ocular analogue of cerebral stroke. Best evidence reflects that over three quarters of patients suffer profound acute visual loss with a visual acuity of 20/400 or worse. This results in a reduced functional capacity and quality of life. There is also an increased risk of subsequent cerebral stroke and ischaemic heart disease. Here we present a case of combined central retinal artery and cilioretinal vein block secondary to carotid plaque.

Keywords: Central retinal artery occlusion, carotid thrombus, cilioretinal vein occlusion, ischaemic ocular syndromes.

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INTRODUCTION

Carotid artery disease is known to cause a variety of ischemic ocular syndromes[1] secondary to embolism or perfusion failure[2]. Atherosclerosis-related thrombosis at the level of the lamina cribrosa is by far the most common underlying cause of central retinal artery occlusion (CRAO), accounting for about 80% of cases.

Visual loss from CRAO occurs from the loss of blood supply to the inner layer of the retina. The ophthalmic artery is the first branch of the internal carotid artery. The central retinal artery is the first intraorbital branch of the ophthalmic artery.

The incidence of atherosclerosis increases with age and is accelerated by hypertension, hyperlipidaemia, diabetes, oral contraceptives and hyperhomocysteinaemia. Other risk factors include obesity, tobacco smoking and a sedentary lifestyle. CRAO can be present with venous occlusion and time should not be wasted in finding the etiology of the disease process and hence saving the life of the patient.

CASE REPORT

A 40 year old male presented to out patient department of jawahar lal nehru medical college with chief complaints of sudden loss of vision in right eye 3 months back. According to patient, he was apparently well 3 months back when he noticed loss of vision in his right eye. Loss of vision was sudden in onset, painless and non progressive. There was also one episode of transient loss of vision for 2 minutes in right eye on the day before.

The patient is a diagnosed case of hypertension and is on medication. There is no history of diabetes mellitus, coagulopathies, cardiac disease, and any chronic illness. There is no history of any chronic drug intake.

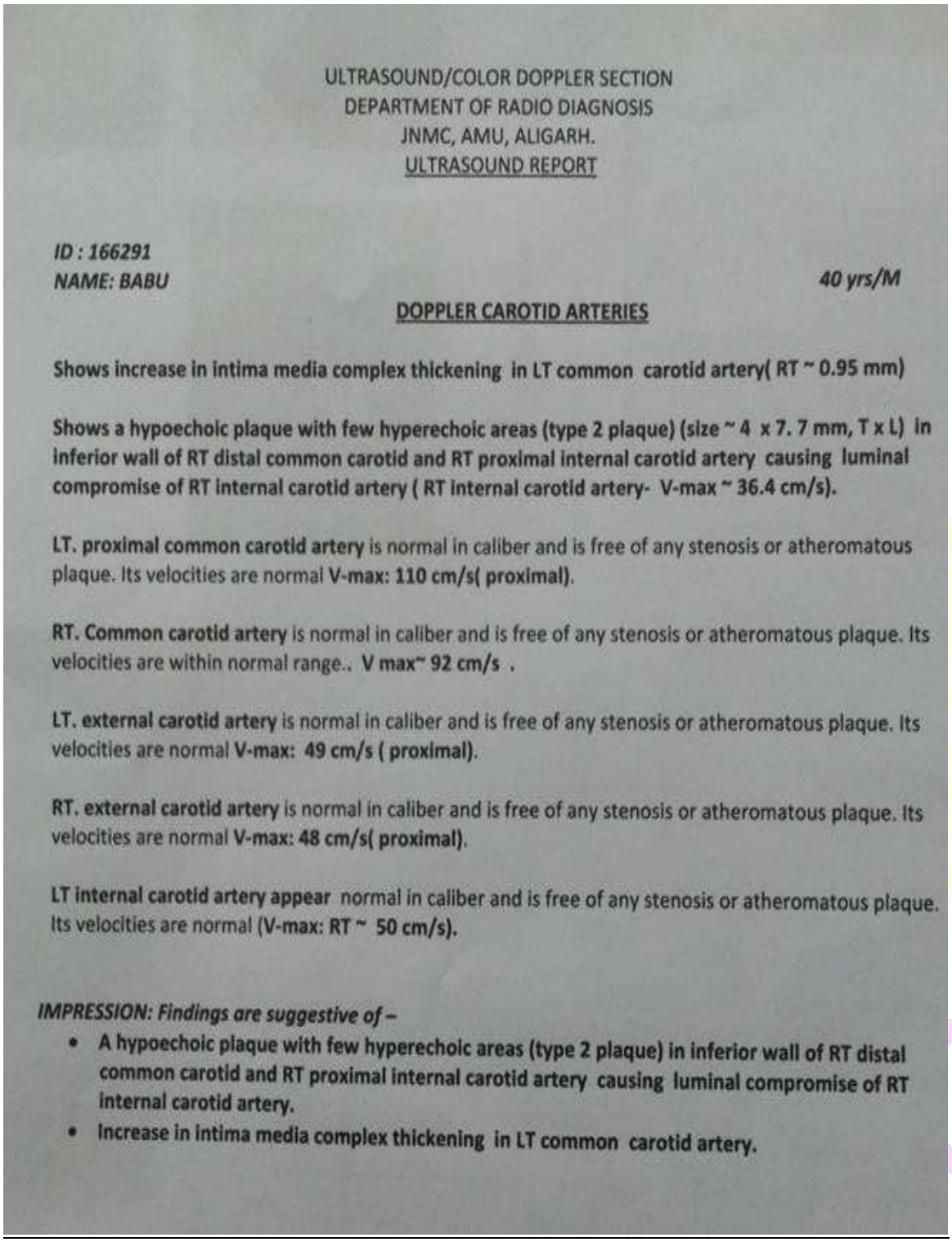
On ocular examination, the best corrected visual acuity in right eye of the patient was Hand Movement positive with projection of rays inaccurate in all the four quadrants. Relative afferent pupillary defect grade 3 was present. Rest of the examination was normal with no neovascularization at the angle or iris. The visual acuity in left eye was 6/6 and rest of the ocular examination in left eye was within normal limits.

Upon examination of the cardiovascular system, a bruit was heard at the bifurcation of carotid artery. Rest of the cardiovascular and examination of other organ systems was normal.

Patient was advised fundus fluorescein angiography after fundus examination and carotid Doppler study after a cardiology reference.

Carotid Doppler study showed a hypoechoic plaque (size- 4/7 mm) in inferior wall of right distal common carotid and right proximal internal carotid artery (figure 1).

Fundus photographs (figure 2) along with fundus fluorescein angiograms (figure 3) are given below.



ULTRASOUND/COLOR DOPPLER SECTION
DEPARTMENT OF RADIO DIAGNOSIS
JNMC, AMU, ALIGARH.
ULTRASOUND REPORT

ID : 166291
NAME: BABU *40 yrs/M*

DOPPLER CAROTID ARTERIES

Shows increase in intima media complex thickening in LT common carotid artery(RT ~ 0.95 mm)

Shows a hypoechoic plaque with few hyperechoic areas (type 2 plaque) (size ~ 4 x 7. 7 mm, T x L) in inferior wall of RT distal common carotid and RT proximal internal carotid artery causing luminal compromise of RT internal carotid artery (RT internal carotid artery- V-max ~ 36.4 cm/s).

LT. proximal common carotid artery is normal in caliber and is free of any stenosis or atheromatous plaque. Its velocities are normal **V-max: 110 cm/s(proximal).**

RT. Common carotid artery is normal in caliber and is free of any stenosis or atheromatous plaque. Its velocities are within normal range.. **V max~ 92 cm/s .**

LT. external carotid artery is normal in caliber and is free of any stenosis or atheromatous plaque. Its velocities are normal **V-max: 49 cm/s (proximal).**

RT. external carotid artery is normal in caliber and is free of any stenosis or atheromatous plaque. Its velocities are normal **V-max: 48 cm/s(proximal).**

LT internal carotid artery appear normal in caliber and is free of any stenosis or atheromatous plaque. Its velocities are normal **(V-max: RT ~ 50 cm/s).**

IMPRESSION: Findings are suggestive of –

- **A hypoechoic plaque with few hyperechoic areas (type 2 plaque) in inferior wall of RT distal common carotid and RT proximal internal carotid artery causing luminal compromise of RT internal carotid artery.**
- **Increase in intima media complex thickening in LT common carotid artery.**

Figure 1: Carotid Doppler report

Figure 2: Fundus photograph-L

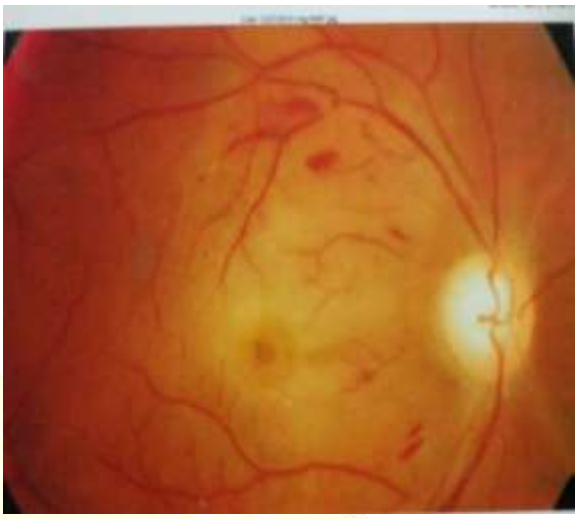


Figure 3: Fundus photograph- RE

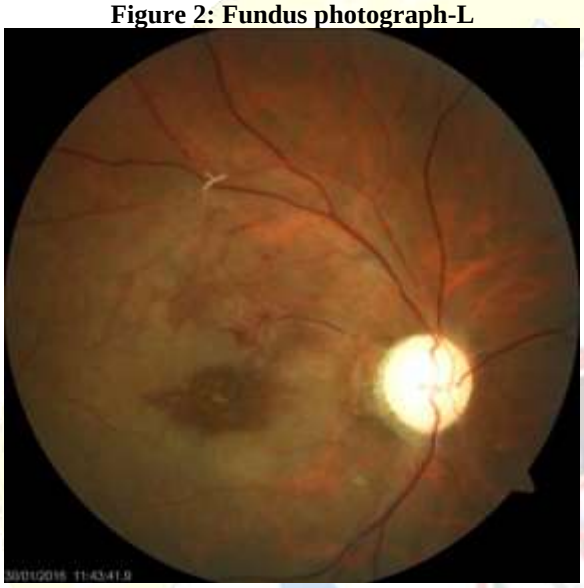


Figure 4: Fundus photograph- RE (after 15 days)

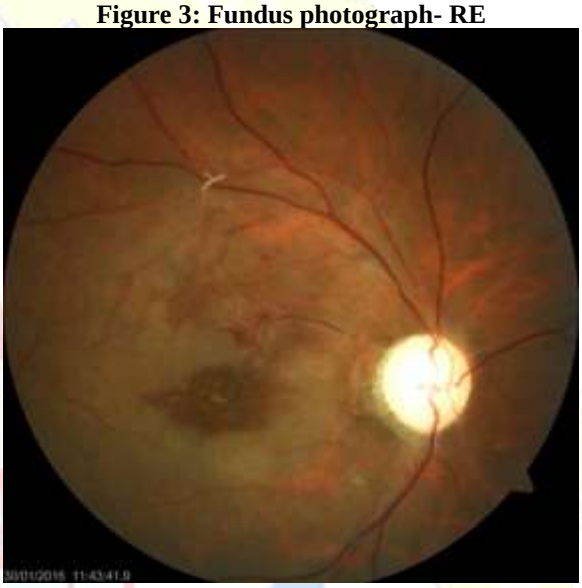


Figure 5: Fundus photograph- RE (after 45 days)

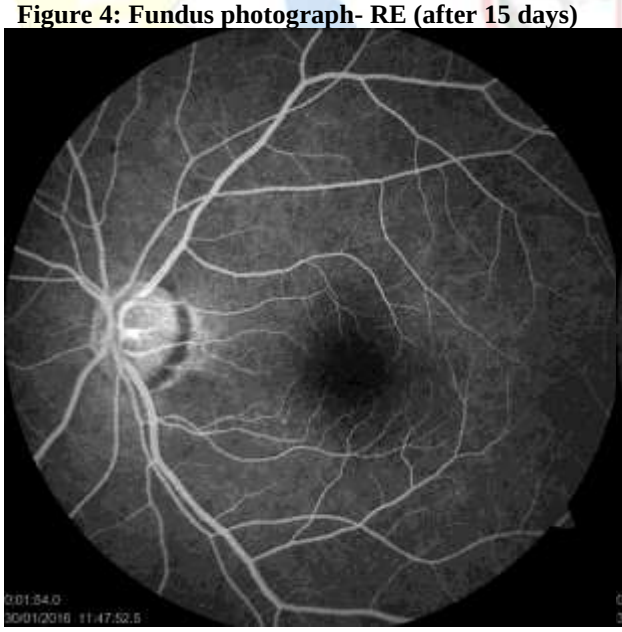


Figure 6: Fundus Fluorescein Angiogram- LE

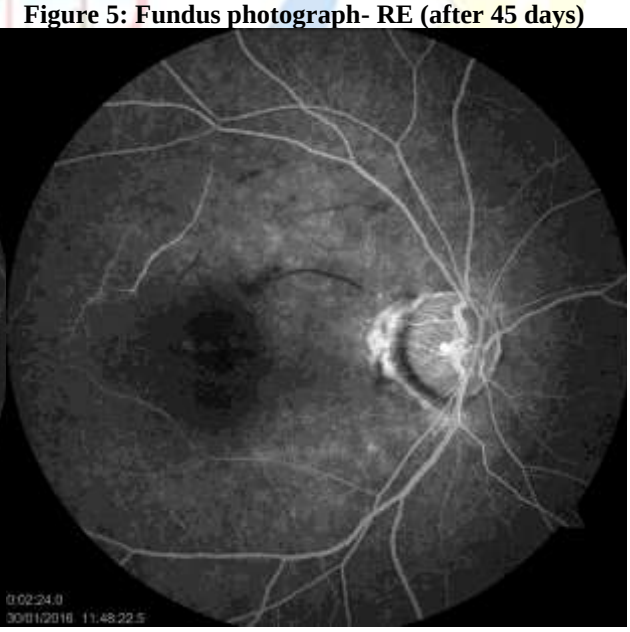


Figure 7: Fundus Fluorescein Angiogram-RE

DISCUSSION

In 1859, Van Graefe first described central retinal artery occlusion (CRAO) as an embolic event to the central retinal artery in a patient with endocarditis. In 1868, Mauthner suggested

that spasmodic contractions could lead to retinal artery occlusion. There is a multitude of causes of CRAO, but patients typically present with sudden, severe, and painless loss of vision.

Epidemiology

The incidence is estimated to be 1 in 100,000 people and accounts for 1 in 10,000 ophthalmological outpatient visits[3]. The mean age of presentation is in the early 60s, although a few cases have been reported in patients younger than 30 years. The etiology of occlusion changes depending on the age of presentation. Men are affected slightly more frequently than women.

Pathophysiology

Acute obstruction of the central retinal artery results in inner layer edema and pyknosis of the ganglion cell nuclei and ultimately ischemic necrosis results. Furthermore, the foveola assumes a cherry-red spot because of a combination of 2 factors: (1) the intact retinal pigment epithelium and choroid underlying the fovea, and (2) the foveolar retina is nourished by the choriocapillaris. The late stage shows a homogenous scar replacing the inner layer of the retina. Hayreh has shown that irreversible cell injury occurs after 90-100 minutes of total CRAO in the primate model[4].

Clinical Features

CRAO usually presents with sudden, painless monocular vision loss. Snellen VA of counting fingers or worse is found in 74% of patients with a visual field defect[5]. If a cilioretinal artery is present, the central vision may be preserved. Some patients may reveal a history of amaurosis fugax involving transient loss of vision lasting seconds to minutes but which may last up to 2 hours.

On ocular examination, the fundoscopic findings in CRAO vary based on time from event and by type of CRAO[6]. Early findings performed within 7 days of CRAO showed the following results: retinal opacity in the posterior pole (58%), cherry red spot (90%), cattle trucking (19%), retinal arterial attenuation (32%), and optic disk oedema (22%) and pallor (39%).

At later stages based on survivorship curves, fundoscopic findings showed optic atrophy (91%), retinal arterial attenuation (58%), cilioretinal collaterals (18%), and macular retinal pigment epithelial changes (11%)[5,7,8].

CRAO can also be divided into stages based on the severity of VA loss and fundoscopy. This is useful in predicting prognosis.

Investigations

Arteritic CRAO and subsequent visual loss results in patients with temporal arteritis[9]. As a result, all patients over 50 years of age with CRAO and a waxy pale optic disc should be investigated for temporal arteritis. FFA and inflammatory markers, such as erythrocyte sedimentation rate (ESR), Creactive protein (CRP), and platelet count, should be performed[9]. These cases should be treated promptly with steroid to prevent contralateral eye vision loss. A recent single centre retrospective audit demonstrated that 64% of patients suffering a CRAO had at least one new undiagnosed vascular risk factor, the most common being hyperlipidaemia (36%), followed by hypertension (27%) and diabetes (12%)[10].

Management

The management of CRAO should be divided into:

Acute:

Attempt to restore ocular perfusion to the CRA. This includes use of sublingual isosorbide dinitrate or systemic pentoxifylline or inhalation of a carbogen, hyperbaric oxygen, to increase blood oxygen content and dilate retinal arteries[11-13].Ocular massage to attempt to dislodge emboli[14].

Subacute:

Preventing secondary neovascular complications to the eye. Another complication of CRAO is the risk of neovascularization and subsequent glaucoma. Neovascularization after CRAO tend to occur around 8 weeks (range 2–16 weeks)[15]. Therefore, prudent clinical practice would be to review all patients with acute CRAO at regular intervals as early as 2 weeks, and then monthly up to 4 months after CRAO.

Long term:

Preventing other vascular ischaemic events to the eye or other end organ. The optimal management of CRAO needs to address systemic atherosclerotic risk factors to reduce secondary ischaemic events. Rudkin *et al* noted that 64% of CRAO patients had at least one new vascular risk factor found after the retinal occlusive event, with hyperlipidaemia being the most common undiagnosed vascular risk factor at the time of the sentinel CRAO event (36%)[10]

CONCLUSION

CRAO should be considered as an ocular emergency and is the ocular analogue of cerebral stroke. The same atherosclerotic risk factors that predispose to cardio, peripheral, and cerebrovascular disease are present in CRAO, and these must be actively evaluated to prevent further medical comorbidities.

Current management in acute CRAO has limited efficacy to improve vision and studies suggest that for treatment to be effective in CRAO, it must be deployed within a short time

window, of about 6h of symptom onset within the retinal tolerance time for any therapy to be effective. The management follows the same principles of treatment as any other vascular end organ ischaemic disease, and includes vascular review to prevent further end organ ischemia and ocular complications.

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