



CRAO WITH CAD AND CVA-RARE PRESENTATION

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ABSTRACT Central retinal artery occlusion presents with acute painless loss of monocular vision. It is considered a form of stroke with same clinical approach and management. Incidence of 1 to 10 in 100,000. 59 years old man k/c/o type 2 Diabetes Mellitus and systemic hypertension, poor compliance with medication, developed chest pain and breathlessness treated conservatively for NSTEMI with acute pulmonary edema. The next day he had Severe headache in the left frontal region with sudden onset of painless loss of vision in left eye. Left eye no perception of light. Relative afferent pupillary defect present. FUNDUS showed bilateral NPDR with Cherry red spot in left eye. Perimetry 120-2 showed severe constriction and absent visual fields in the left eye. MRI BRAIN- Acute infarct in left mamillary body, left half of optic chiasma and canalicular segment of the left optic nerve, thrombotic occlusion of the intracranial segment of the left ICA. CAROTID DOPPLER- Eccentric soft plaque in the right common carotid artery, left ICA causing moderate luminal narrowing, patchy flow at the origin of the left ICA with absent flow in the remaining visualized ICA. Treated with Dual antiplatelets and strict glyceemic control.

KEYWORDS : CRAO, Cherry red spot, carotid doppler

INTRODUCTION

Central retinal artery occlusion presents with acute painless loss of monocular vision. It is considered a form of stroke with same clinical approach and management. Incidence of 1 to 10 in 100,000. Here we report a case of CRAO with concurrent ipsilateral left Internal carotid artery critical narrowing.

CASE REPORT

59 years old man with Type 2 Diabetes Mellitus and systemic hypertension, poor compliance with medications, developed chest pain and breathlessness treated conservatively with anticoagulants, antiplatelet agents for NSTEMI with acute pulmonary edema. The next day he had Severe headache in the left frontal region with sudden onset of painless loss of vision in left eye. On examination, vitals were stable. Left eye no perception of light. Relative afferent pupillary defect present. However, there was no evident motor weakness in all 4 limbs. FUNDUS showed bilateral NPDR with Cherry red spot in left eye.

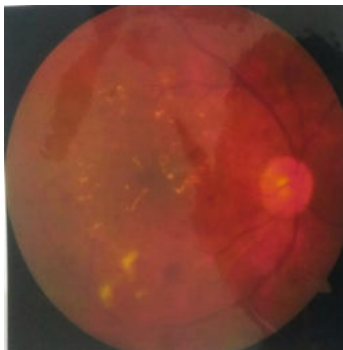


Figure1. Fundus-cherry red spot

Perimetry 120-2 showed severe constriction and absent visual fields in the left eye. CBC, RFT, Electrolytes were within normal limits. HbA1c -11.0. Trop T-250. 2D Echo shows Anteroseptal, Apical and inferior wall Hypokinesia with Moderate LV dysfunction (EF-40%). MRI BRAIN- Acute infarct in left mamillary body, left half of optic chiasma and canalicular segment of the left optic nerve, thrombotic occlusion of the intracranial segment of the left ICA.

CAROTID DOPPLER- Eccentric soft plaque in the right common carotid artery, left ICA causing moderate luminal narrowing, patchy flow at the origin of the left ICA with absent flow in the remaining visualized ICA. Treated with Dual antiplatelets, Antihypertensives and strict glyceemic control. The poor prognosis for visual recovery in the left eye was explained to the patient. The patient was followed up at regular interval as the chance of developing CRAO in right eye is high.

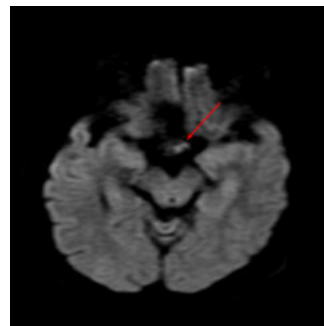


Figure2, Acute infarct in optic chiasma

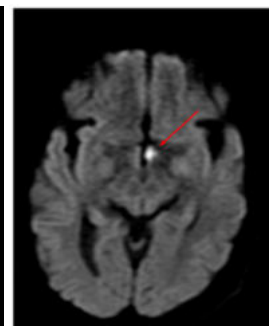


Figure3, Acute infarct in Mamillary body

DISCUSSION

In the setting of sudden, painless vision loss, the diagnosis of CRAO must be at the top of the differential, as it portends the most vision and life threatening underlying diagnosis and with associated high mortality. Typical patients have severe monocular vision loss with 80% of patients having a visual acuity of 20/400 or worse as a result of loss blood supply to the inner retinal layers. Analogous to ischemic cerebral stroke, the pathology is thromboembolic and majority are due to carotid artery disease, primarily due to atherosclerotic plaques. As demonstrated in this case, one cannot neglect carotid stenosis and the heart as other potential sources of emboli. Risk factors mimic those of cerebral stroke and include hypertension, diabetes mellitus, carotid artery disease, coronary artery disease, history of transient ischemic attacks or cerebral vascular accidents. There have also been several case series of CRAO during and post angiography and stenting. Given the emergent nature of condition, swift imaging and treatment is critical. It is suspected that irreversible retinal damage occurs without recovery of vision within six and half hours.

CONCLUSION

Patients with CRAO are at risk of cardiovascular as well cerebrovascular events and reduced life expectancy. Long term management to prevent vascular events is cause specific. Unless the etiology is known at presentation, carotid artery imaging study is recommended. CRAO has poor prognosis for spontaneous recovery of vision.

REFERENCES

1. Leavitt JA, Larson TA, Hodge DO, et al. The incidence of central retinal artery occlusion in Olmsted County, Minnesota. *Am J Ophthalmol.* 2011;152(5):820-3.e2.
2. Merchut MP, Gupta SR, Naheedy MH. The relation of retinal artery occlusion and carotid artery stenosis. *Stroke.* 1988;19(10):1239-42.
3. Zhang LY, Zhang J, Kim RK, et al. Risk of acute ischemic stroke in patients with monocular vision loss of vascular etiology. *J Neuroophthalmol.* 2018;38(3):328-33.
4. Hayreh SS, Zimmerman MB. Central retinal artery occlusion: visual outcome. *Am J Ophthalmol.* 2005;140(3):376-91.
5. Hayreh SS, Podhajsky PA, Zimmerman MB. Retinal artery occlusion: associated

- systemic and ophthalmic abnormalities. *Ophthalmology*. 2009;116(10):1928-36.
6. Georgalas I, Pagoulatos D, Koutsandrea C, et al. Sudden unilateral painless loss of vision. *BMJ*. 2014;349:g4117.
 7. Sun R, Peng XY, You QS, et al. Branch retinal artery occlusion following carotid stenting: a case report. *Exp Ther Med*. 2016;12(4):2183-6.
 8. Tomsak RL, Hanson M, Gutman FA. Carotid artery disease and central retinal artery occlusion. *Cleve Clin Q*. 1979;46(1):7-11.
 9. Chen CS, Lee AW, Campbell B, et al. Efficacy of intravenous tissue-type plasminogen activator in central retinal artery occlusion: report from a randomized, controlled trial. *Stroke*. 2011;42(8):2229-34.
 10. Hattenbach LO, Kuhl-Hattenbach C, Scharrer I, et al. Intravenous thrombolysis with low-dose recombinant tissue plasminogen activator in central retinal artery occlusion. *Am J Ophthalmol*. 2008;146(5):700-6.
 11. Olsen TW, Pulido JS, Folk JC, et al. Retinal and ophthalmic artery occlusions preferred practice pattern. *Ophthalmology*. 2017;124(2):P120-P143.