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Case Report

NEOVASCULAR GLAUCOMA SECONDARY TO CAROTID ARTERY ATHEROSCLEROSIS

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ABSTRACT

A 65 year old male patient presented with pain, redness and loss of vision in right eye. Slit lamp examination revealed mid dilated, fixed pupil with rubeosisiridis. Intraocular pressure was raised in right eye. Fluroscein examination showed dye leaking in right eye anterior chamber due to iris neovascularisation. Carotid Doppler and CT carotid angiography study showed right common carotid artery atherosclerotic plaque and reduced blood flow in right central retinal artery. Proper ophthalmological evaluation of patient having carotid artery disease is essential for prevention of intractable neovascular glaucoma and permanent blindness.

Keywords: Neovascular glaucoma, Carotid occlusive disease, chronic ocular ischemia

INTRODUCTION

Chronic ocular ischemia occurs due to carotid artery stenosis which can lead to neovascular glaucoma.[1]It is a potentially blinding condition. Neovascular glaucoma term is coined by Weiss et al in 1963. It was also known as haemolytic glaucoma and Rubeotic glaucoma, first described by Coats in 1906. It can be the result of carotid artery occlusion.[1] In one series, carotid occlusive disease was the fourth most common cause and accounted for 8 % of the cases of rubeosis iridis.[2] It eventually leads to "zipping up" of the angle resulting in the classical endpoint-neovascular glaucoma with high pressure, pain and corneal oedema.[2]

CASE REPORT

A 65 year old male presented with intermittent pain, loss of vision and redness since one year in the right eye. Patient was not a known case of hypertension or diabetes mellitus. There is no history suggestive of cerebrovascular accident in past.There was absence of light perception in right eye and visual acuity in left eye was 6/6. Intraocular pressure was 29 mm Hg in the right eye and 17mm of Hg in the left eye. Slit lamp biomicroscopic examination of the right eye revealed circumcorneal congestion of the conjunctiva with diffuse corneal edema. Anterior chamber depth was normal. Iris neovascularisation was noted along the pupillary margin with absence of hyphema (Fig.1).

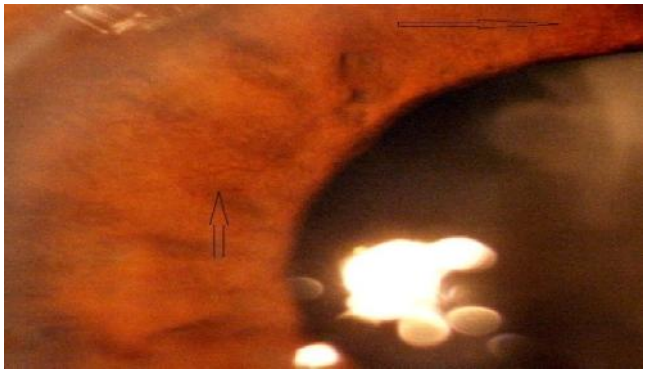

Fig1: Slit-lamp examination of Right eye showing Rubeosisiridis

Direct and consensual light reflexes were absent in the right eye. Ophthalmoloscopic examination revealed presence of pale optic disc. However rests of the details were not appreciated due to lenticular opacity. Anterior and posterior segment examination of the left eye was unremarkable. Blood pressure was 110/70 mm Hg in right arm supine position. Random blood sugar was 120 mg/dl and lipid profile was within normal limits except triglyceride level which was 154 mg/dl.

On Fundus Fluroscein angiography, leakage of fluroscein dye was noted in the anterior chamber of the right eye (Fig.2).

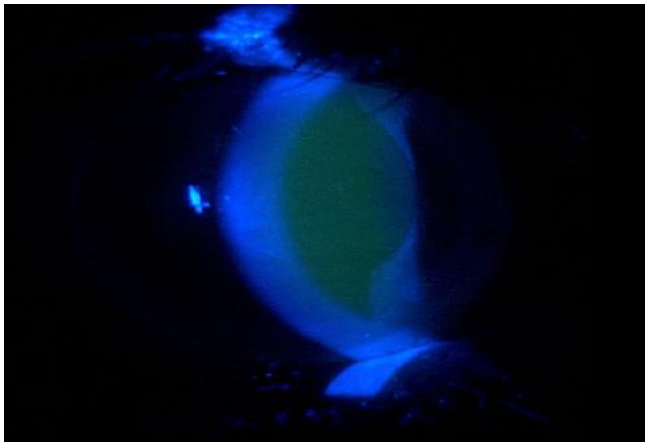

Fig 2: Leakage of flurosceinedye in aqueous on Cobalt blue filter.

Carotid Doppler study showed patterns suggestive of atherosclerotic changes in right and the left common carotid artery. Presence of an ulcerated, irregular plaque measuring 8.8×3.1 mm was noted at the bifurcation of the right common carotid artery which was confirmed on CT carotid angiography (Fig.3).

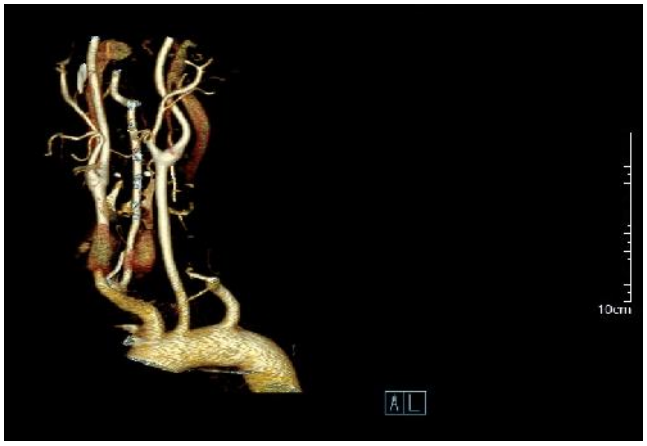

Fig 3: CT carotid angiography showing obstruction at the bifurcation of the right common carotid artery

Lumen of the right central retinal artery was 1.2sq.cm and that of left central retinal artery was 1.7 sq.cm The blood flow velocity in the right and left central retinal artery was 4.5 cm/sec and 8.8 cm/sec respectively.

DISCUSSION

In 1956, Wise proposed that production of diffusible angiogenic factors from ischemic retina was sufficient to induce neovascularisation of the iris, optic nerve and retina[3].With the advent of Fluroscein angiography it was demonstrated that the risk of neovascularisation was directly correlated to the extent and severity of the retinal ischemia[4].Retinal blood flow is autoregulated by balancing metabolic and myogenic factors. Numerous factors influence the vascular resistance. In ocular manifestations due to the carotid disease, most common cause is carotid artery atherosclerosis. Atherosclerosis of common or internal carotid artery usually affects the ipsilateral eye.

Presence of signs such as conjunctival congestion, presence of iris neovascularisation, fixed semi dilated pupil is suggestive of anterior segment ischemia. Fundus Fluroscein angiography of other eye was normal, but fluroscein dye is noted in the diseased eye which is due to the leaking neovascularisation of iris (Fig.2)

Fundus examination revealed pale optic disc but details could not be appreciated due to cataractous changes. Raised intraocular pressure in the affected eye is suggestive of development of neovascular glaucoma. One of the most striking features is the narrowed lumen and reduced blood flow in the right central retinal artery on carotid Doppler study. There is an absence of systemic diseases like Diabetes Mellitus, Hypertension etc which is an important finding because presence of these conditions have been found to be present in more than half of the patients of this condition.[5, 6] In 10-15 % of patients of ocular ischemic syndrome due to carotid insufficiency, there is history of an episode of transient visual loss.[6,7] But there is no such history of an episode of transient visual loss in this patient.

CONCLUSION

Chronic ocular ischemia resulting from the extracranial carotid artery occlusion leads to the neovascularisation in the eye and progressive loss of vision. Therefore, careful ophthalmological

evaluation of patient having carotid artery disease is essential for prevention of intractable neovascular glaucoma and permanent blindness.

Conflict of interest: Nil

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