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Case Report

Ocular ischemic syndrome: A rare cause of postoperative visual loss following coronary artery bypass graft (CABG) surgery

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Introduction

Ocular ischemic syndrome is a chronic insufficiency of ocular blood supply due to carotid artery or ophthalmic artery occlusion. It is a consequential sequel of severe atherosclerosis,¹ hypertension, diabetes,² carotid artery dissection,³ or inflammatory conditions of carotid artery⁴ or emboli.⁵ The patient shows varied presentation as asymptomatic initially; gradually there is blurring, which may be transient or even may manifest as abrupt visual loss. The condition may present

with many posterior segment changes. In addition, if not detected early or followed periodically, it may present with anterior segment complications, such as inflammation, neovascularization, or secondary neovascular glaucoma (NVG). An early detection of this rare manifestation of chronic carotid insufficiency is very important, as the condition may herald life threatening thrombotic conditions of brain or myocardium.

Though NVG can be a cause of an abrupt as well as gradual visual loss in many systemic conditions, it has never found its place as a cause of visual disturbances and postop visual loss presenting immediate to postop or during periop period of nonophthalmic surgeries. The cause of perioperative visual loss (POVL) has suggested involvement of any structure of visual system, from cornea to occipital lobe, but among various vascular causes of POVL, ocular ischemic syndrome has never been implicated. The appearance or onset of the disease during postop or periop period in coronary arterial bypass graft (CABG) surgery has not yet been reported. The rarity of the case qualifies the need for reporting, which may emphasize multidisciplinary approach, follow-up, and intervention in such cases.

Case report

A 47-year-old female patient, a known case of diabetes mellitus, hypertension of 8 years duration, and coronary

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artery disease, underwent two sessions of CABG. She complained of onset of severe diminution of vision in right eye within 3 days of second CABG, which she ignored for almost 4 weeks. It was pain in the right eye and moderate to severe headache that compelled her to seek ophthalmic consultation. Examination of right eye revealed best-corrected visual acuity of perception of light with accurate projection of rays. Conjunctival and circumcorneal congestion was seen on presentation. Slit lamp biomicroscopy revealed grade III cells and flare, 360°, rubeosis iridis and ectropion uveae. Gonioscopy for the anterior chamber angles detected predominantly closed angles (Schaffer's grade 0) and in little less than two quadrants neovascularization of angles was seen. Crystalline lens was detected to have grade III (LOCS grade) cataract. Posterior segment and fundus were hazily seen due to corneal edema and intense anterior chamber reaction, which were further obscured due to cataract. Intraocular pressure (IOP) was 52 mmHg in right eye and 12 mmHg in left eye with Goldmann's applanation tonometer (GAT). She was treated with Injection 20%, Mannitol 250 ml IV stat over 20 min, tablet Dimox 500 mg four times per day, eye drop Timolol 0.5% (twice daily), Brimonidine 0.2% (three times per day), Dorzolamide 2% (three times per day), Predacetate 1% every two hours, and Homatropine 2% (three times per day). Patient when reviewed after 2 days was found to have no corneal edema, reduced AC reaction with IOP of 18 mmHg with GAT. Fundus evaluation revealed pale yellowish macula with cherry red spot (Fig. 1), attenuated arterioles, and dilated venules with few mid-peripheral intraretinal dot and blot hemorrhages. The fellow eye had no anterior segment or fundus abnormalities. A clinical and provisional diagnosis of post-CABG ocular ischemic syndrome was made based on the antecedent procedure, fundus picture, anterior segment neovascularization (NVI and NVA) complicated by neovascular glaucoma, and uveitis. The diagnosis was further supported by fluorescein angiography (FA), which detected prolonged arm-retina time, delayed retinal arteriolar filling time, and large scattered choroidal nonperfusion areas in right eye (Fig. 2). Poor arteriolar filling and narrowing and poor choroidal filling with no evidence of posterior segment neovascularization (Fig. 3) were observed. She underwent complete cardiovascular reevaluation and carotid Doppler imaging on both sides, which revealed no

Fig. 1 – Fundus picture right eye shows prominent cherry red spot.

Fig. 2 – Fluorescein angiography showing prolonged arm-retina time.

Fig. 3 – Fluorescein angiography showing poor arterial filling and poor choroidal perfusion.

abnormality. She was administered panretinal photocoagulation in right eye (Fig. 4) and was followed up for regression of anterior segment neovascularization and control of IOP secondary to NVG. She has shown regression of NVI and NVA within 3 weeks of follow-up and her IOP in right eye was controlled by three topical antiglaucoma drugs. Though on follow-up FA, she had improved arteriolar filling and choroidal perfusion (Fig. 5) but she had no recovery of vision in the affected eye.

Fig. 4 – Post panretinal photocoagulation.

Fig. 5 – Post panretinal photocoagulation fluorescein angiography showing improved arteriolar filling and choroidal perfusion.

Discussion

5% of the cases of severe carotid artery obstruction which has also been referred in literature as “venous stasis retinopathy” and later termed “ocular ischemic retinopathy (OIS)” which impediments blood supply to ipsilateral eye, has ocular involvement. Though the condition is restricted to higher age group from 5th to 8th decade, the mean age is 65 years⁶ and is twice more common in males. Ocular ischemic syndrome can occur even with 50% carotid artery stenosis.¹ Majority of cases of OIS show up to 90% carotid obstruction, which translates into 50% reduction of central retinal artery perfusion pressure.⁷ The stenosis may be located in common carotid, internal carotid, or may be in ophthalmic artery. This impairment and obstruction to the flow in the vessels is caused by atheroma in atherosclerosis,¹ dissecting aneurysm,³ inflammatory conditions like giant cell arteritis,⁴ Behcet's disease, or even trauma.

Postsurgical acute vision loss or POVL is an identified and known entity. Fortunately this severe and devastating complication has a very low incidence of about 0.0008% of all nonophthalmic surgeries. The cause though not clearly understood, it is encountered more commonly in cases after postspinal surgeries (0.11%) and cardiac surgeries (0.08%).⁸ Etiology and pathogenesis of this complication is still obscure

and not completely understood. Ischemic optic neuropathy, central retinal artery occlusion, and cortical blindness have been implicated and identified as possible causes in this condition. Anterior ischemic optic neuropathy (AION) has highest prevalence after cardiac surgery and other contributing risk factors are age, diabetes, long cardiopulmonary bypass time, and anaemia.⁹ POVL due to OIS and its complications have not yet been reported. Existence of this entity needs further investigations regarding etiology, prevention, and management on postoperative visual disturbances. In addition, it entails and demands a regular and prolonged follow-up during postoperative period following nonophthalmic surgeries.

Conflicts of interest

The authors have none to declare.

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