# Postoperative vision loss following non ocular surgery- A case report

**A. Sivaraja Gowthaman[1,*], Hannah Ranjee Prasanth[2], Elfride Sanjana[3]**

[1]Junior Resident, [2]Associate Professor, [3]Professor, Dept. of Ophthalmology, Pondicherry Institute of Medical Sciences Kalapet, Puducherry, India

***Corresponding Author:**
Email: sivaraj0270@gmail.com

## Abstract
We describe a rare and classical presentation of anterior ischemic optic neuropathy following aortic valve replacement surgery. This case is being presented for its rarity and its morbidity which is bilateral irreversible loss of vision.

**Keywords:** Anterior Ischemic Optic Neuropathy, Aortic valve replacement surgery, Disc edema, Steroids, Sudden vision loss.

## Introduction
Post operative sudden visual loss is a serious devastating complication that follows uneventful spinal or cardiac surgery leading to irreversible loss of vision. The most common causes are anterior ischemic optic neuropathy (AION), posterior ischemic optic neuropathy (PION), central retinal artery occlusion (CRAO), cortical blindness.[1] The percentage of postoperative optic neuropathy ranges from 0.002% to 0.1% following all surgical procedures and after coronary artery bypass graft (CABG) ranges from 0.06% to 0.113%.[2]

## Case Report
A 47 year old male presented with sudden diminution of vision in both eyes, right eye more than left eye, for 5 days following aortic valve replacement surgery done 7 days back. He had one episode of syncope 15 days back and history of dyspnoea on exertion for the past 2 years. He was diagnosed to have severe calcified aortic stenosis, other arteritic systemic diseases such as Takayasu arteritis were ruled out by cardiologist and patient underwent aortic valve replacement surgery. On ocular examination best corrected visual acuity OD was ½ /60 NIP, NIG and OS 6/12 NIP, NIG. Rest of anterior segment examination was unremarkable. Stereoscopic fundus examination of right eye revealed a clear media, average sized optic disc with crowded cup, pallid disc oedema, engorged and tortuous veins, peripapillary nerve fiber layer edema and optic disc showed optociliary collaterals in superonasal quadrant, AV ratio was 1:3. There were no disc haemorrhages. Macula and peripheral retina were normal. No cotton wool spots or exudates were present. There were no visible emboli along retinal vessels (Fig. 1). Disc edema was more in right eye when compared to left eye. Left eye fundus examination revealed a clear media, average sized optic disc with crowded cup, pallid disc oedema, engorged and tortuous veins, peripapillary nerve fiber layer edema, AV ratio was 1:3.There were no disc haemorrhages. Macula and peripheral retina were normal. No cotton wool spots or exudates were present. There were no visible emboli along retinal vessels. (Fig. 2). Colour vision could not be assessed in right eye as vision was ½ /60. Left eye colour vision testing showed defect for red and green colour plates. Visual field examination could not be done as patients vision in right eye was ½ /60 and general condition was poor. Common visual field defect seen in ischemic optic neuropathy is altitudinal field defect. On General examination pulse rate was 82 beats per minute, blood pressure of 100/60mm of Hg, mean arterial pressure was 65 mm of Hg., respiratory rate was 26 per minute.

On cardiovascular systemic examination, patient had palpable thrill at apex, ejection systolic murmur heard in apex (aortic region). Other systemic examination were unremarkable.

Patient was started on intravenous methyl prednisolone 1 g / day and patient was reviewed after 3 days and followed by oral prednisolone 60 mg per day with slow taper. Best corrected visual acuity in right eye improved to 2/60 while left eye remained 6/12 NIP, NIG. Disc edema reduced in both eyes. (Fig. 3 and 4)

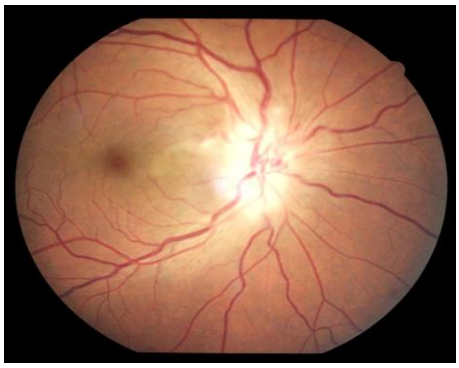

**Fig. 1: Right eye showing pallid disc edema, peripapillary nerve fibre layer edema**

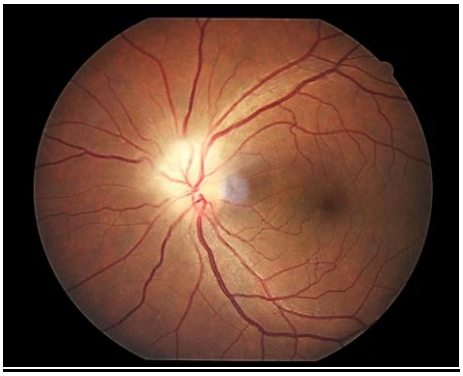

**Fig. 2: Left eye showing pallid disc edema and peripapillary nerve fibre optociliary collaterals on the disclayer edema**

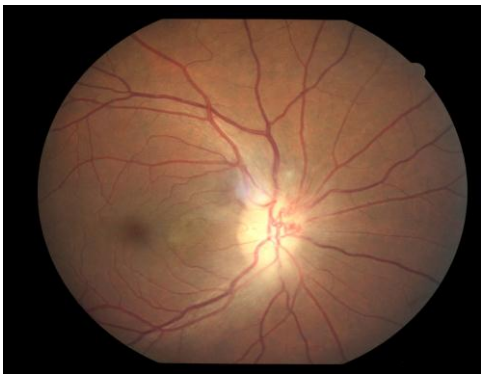

**Fig. 3: Right eye showing resolving disc edema after 3 days of treatment**

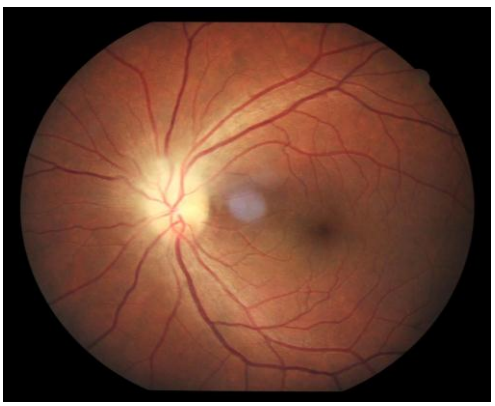

**Fig. 4: Left eye showing resolving disc edema after 3 days of treatment**

## Discussion

The incidence of postoperative vision loss varies from 0.06-4.5% in cardio pulmonary bypass surgery with AION being the most common etiology.[2] Most commonly seen in the age group of 50 years.[3] The incidence varies in different studies. In a Mayo clinic study of cardiac patients, among 27,915 patients who had undergone cardiac bypass surgery 17 (0.06%) were identified with perioperative vision loss.[3,4] The common causes of sudden loss of vision in Mayo clinic study includes anterior ischemic optic neuropathy, posterior ischemic optic neuropathy, central retinal artery occlusion and occipital infarctions[3,4] The incidence varies from 0.0008% -0.002% in non ocular non cardiac surgery such as spinal surgery with posterior ischemic optic neuropathy being the most common cause.[5]

Ischemic optic neuropathy is usually unilateral. Bilateral involvement is more common among arteritic form than in non arteritic form. According to American society of Anaesthesiologists postoperative visual loss registry bilateral ischemic optic neuropathy was seen in 55 (66%) patients out of 83 patients with ischemic optic neuropathy.[6] In another study by KT Berg et al, out of 75 patients, 43 (57%) cases had bilateral involvement of AION.[5]

AION occurs at the optic nerve head where the optic nerve and retinal vessels enters the globe. Posterior ischemic optic neuropathy occurs from optic nerve head to optic chiasma. There is presence of disc edema in case of AION whereas in posterior ischemic optic neuropathy fundus appears normal in early days. Both can lead to optic nerve pallor in few days to weeks.[1]

AION is due to decreased blood flow in short posterior ciliary artery which reduces oxygen delivery to optic nerve head. There are many factors for decreased blood flow in posterior ciliary artery which includes prolonged surgical procedures, intrinsic small vessel disease such as diabetes mellitus, hypertension, prolonged hypotension, prolonged vasospasm following inotrope infusion after surgery.[1,2] PION is due to infarction of the intraorbital optic nerve supplied by pial plexus, but it is less common than AION.[5]

According to Mayo clinic study the risk factors for perioperative vision loss following cardiac procedures include advanced age, post-operative haemoglobin concentrations, longer pump times, history of severe vascular disease, blood transfusions and other blood component transfusions.[4]

AION may occur due to anaemia and intraoperative hypotension. Blood loss leads to release of endogenous vasoconstrictors due to sympathetic nervous system which can cause choroidal and optic nerve ischemia[5].Patients after extracorporeal surgical procedures have lower concentrations of haemoglobin because of hemodilution due to pump and greater blood loss. This further leads to reduced oxygen supply to retina and then ischemia.[2]

Prolonged prone position of patients during cardiac and spinal surgery is an important risk factor. Prolonged prone position leads to increased intraocular pressure.[7] IOP is doubled in prone position according to Cheng et al.[8] Increase in central venous pressure leads to increased episcleral pressure and intraocular pressure. Increased IOP reduces perfusion pressure. Perfusion pressure in anterior optic nerve is the difference between mean arterial pressure and

intraocular pressure.[1] Head up position and use of loose endotracheal tubes can help in reducing intraocular pressure.

The other cause for AION is embolic phenomenon which occurs due to aortic cross clamping and may lead to presence of embolus in retinal vessels.[9] Use of intra operative epi aortic ultrasound can reduce risk of emboli in aortic cross clamping.

Increased use of inotropes following surgery also leads to AION. Adrenaline causes vasospasm of the ocular vessels.[10]

There may be mild to moderate hypothermia during cardiac bypass surgery which may cause increased blood viscosity and lead to watershed infarction of optic nerve.[11] It is also shown that cerebral blood flow decreases upto 6-7% for every centigrade decrease in body temperature.

There is no proven treatment for postoperative ischemic optic neuropathy. There are certain proposed treatments which includes correction of hemodynamic derangements, systemic corticosteroids, anti platelet therapy and lowering of intraocular pressure.[3] Prevention of ischemic optic neuropathy should be the ultimate goal. Careful documentation in the chart is essential and statements regarding causation should be made only when the evidence is clear.

## Conclusion

Postoperative ischemic optic neuropathy is a rare, devastating event, which may be difficult to detect immediately after surgery due to anaesthesia following cardiac surgery. The signs are subtle and are often present late. There is no effective treatment as of now, so the condition may lead to permanent vision loss. Because of rising number of cardiac surgical procedures, cardiac surgeons and ophthalmologists should consider the possibilities of this serious postoperative event.

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