

# Postoperative visual loss following dorsal root entry zone rhizotomy: A dreaded complication after a benign procedure

## ABSTRACT

Postoperative visual loss (POVL) is a rare but grave postoperative complication. It has been mainly reported in patients undergoing cardiac and spinal surgeries. Dorsal root entry zone (DREZ) is pain relieving procedure performed in patients with refractory neuropathic pain with minimal complication rate. We present a case of unilateral POVL following DREZ rhizotomy in prone position in a patient having brachial plexus neuropathy. Exact etiology of vision loss was though not clear; hypotension, use of vasopressors and hemodilution may have led to vision loss in this patient. This case report highlights the associated risk factors for development of this hazardous complication.

**Key words:** Postoperative vision loss; prone; rhizotomy

## Introduction

Postoperative vision loss (POVL) is a dreaded complication reported after several surgical procedures but mostly after spinal, cardiac, and head-neck surgeries. The actual incidence may not be known definitively but is highest for cardiac and spinal surgeries. Estimated rate of POVL in the USA over 10 years period was 8.64/10,000 in cardiac operation and 3.09/10,000 patients in spinal fusion surgeries.<sup>[1]</sup>

Ischemic optic neuropathy (ION) and central retinal artery occlusion (CRAO) are the main ophthalmic lesions forming 89% and 11% of the cases of vision loss reported in spine surgery cases.<sup>[2]</sup> Blood loss amounting to 1 L or more or anesthetic duration more than 6 h was present in majority of cases of ION.<sup>[2]</sup>

Dorsal root entry zone (DREZ) rhizotomy is pain relieving procedure performed in patients having refractory neuropathic pain. It is a relatively benign procedure carried out in patient placed prone with head resting over horseshoe. Although all precautions for prevention of vision loss were undertaken, yet our patient developed unilateral vision loss in immediate postoperative period, the exact etiology of which could not be ascertained. Herein, we describe the presentation and associated risk factors for development of this complication.

## Case Report

A 52-year-old male presented to the hospital with history of pain and weakness in the right upper limb for last 15 years following trauma. The pain gradually increased over the years and for last 6 months, it was not being relieved even

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## MISHRA RK, MAHAJAN C<sup>1</sup>, BINDRA A, GOYAL K

Department of Neuroanaesthesiology and Critical Care, All India Institute of Medical Sciences, <sup>1</sup>Department of Neuroanaesthesiology and Critical Care, Neurosciences Center, All India Institute of Medical Sciences, New Delhi, India

**Address for correspondence:** Dr. Charu Mahajan, Department of Neuroanaesthesiology and Critical Care, Neurosciences Center, 7<sup>th</sup> Floor, Room No. 711, All India Institute of Medical Sciences, New Delhi - 110 029, India. E-mail: charushrikul@gmail.com

by medications. Patient had history of trauma right chest for which he underwent some chest surgery. On examination, the patient had Glasgow Coma scale of E4V5M6, average body build, bilateral pupils normal sized reacting to light, and normal cranial nerve function. He had weakness in the right upper limb with motor power 0/5 at shoulder, elbow, and wrist. Rest all limbs had normal power. Patient had no other comorbidity. After radiological investigations, diagnosis of brachial plexus neuropathy was made, and patient was scheduled for right C4-D1 DREZ rhizotomy for pain relief in prone position.

The routine preoperative investigations were normal with hemoglobin of 13.2 mg/dl. In the operating room, standard monitors such as electrocardiogram, pulse oximeter, and noninvasive blood pressure (BP) were connected. Anesthesia was induced with intravenous (IV) administration of fentanyl 100 µg and propofol 120 mg followed by rocuronium 50 mg to facilitate tracheal intubation. Isoflurane with 60% nitrous oxide in oxygen and intermittent boluses of fentanyl along with propofol infusion 50 mcg/kg/min were used for maintenance of anesthesia. In view of intraoperative use of nerve stimulator by neurosurgeon, muscle relaxant was not administered. Patient was mechanically ventilated to achieve end-tidal CO<sub>2</sub> of 35 ± 5 mm Hg. After proper padding of eyes and pressure points, patient was placed in prone position on a horseshoe ensuring that eyes are free of compression. Intraoperatively, patient had an episode of sudden blood loss of around 500 ml and lowest recorded BP was 87/54 mm Hg. Hypotension (systolic BP [SBP] <90 mm Hg) remained for almost 10 min. It was managed by administration of fluids and blood and intermittent bolus of IV mephentermine. Total intraoperative blood loss was about 2 L and two units of packed red blood cell were transfused to achieve an end hematocrit value of 28. Apart from this, patient received 5 L of IV fluid and a total of 600 ml of urine output was recorded. The surgery lasted for 6 h. At the end of surgery, neuromuscular blockade was reversed but patient did not become fully awake, so he was shifted to Intensive Care Unit (ICU) for further management. After an hour in the ICU, patient became fully conscious and trachea was extubated. In the immediate postoperative period, patient complained of decreased vision in the right eye. On examination, patient was able to only perceive light. Noncontrast computed tomography (NCCT) head was done which did not reveal any hemorrhage or visual cortex pathology. Neuro-ophthalmological consultation was obtained. Patient had positive relative afferent pupillary defect with mild proptosis and partial paresis of the 3<sup>rd</sup> and 6<sup>th</sup> nerve on the right side. On funduscopy, slight blurring of disc margin and hyperemia was observed but overall disc and macula were healthy. "Cherry red spot" pathognomic of CRAO

was not seen. Provisional diagnosis of orbital apex syndrome was made. Contrast enhanced CT scan of head and orbit was done but there were no signs of optic nerve compression or other pathology. Magnetic resonance imaging (MRI) brain and orbit were also unremarkable. Methyl prednisolone 1 mg/kg IV was prescribed for 3 days followed by oral prednisolone 1 mg/kg for 11 days. Visual evoked potential testing was performed but patient was unable to perform it and it was postponed until stitches are removed. Repeat CT done on the 3<sup>rd</sup> postoperative day was also normal. Patient was discharged on the 7<sup>th</sup> postoperative day with instruction for follow-up in ophthalmological retinal outpatient department for further evaluation and management. At the time of discharge, there was no improvement and patient was only able to perceive hand movements. After an interval of 14 days of treatment, on follow-up, the vision remained the same without any significant improvement.

## Discussion

Though rare, POVL is a devastating complication for patient and disturbing for the physicians involved. There are case reports of blindness after various surgeries but POVL following DREZ rhizotomy is being described for the first time.

Common causes of postoperative blindness are ION, CRAO, branch retinal artery occlusion (BRAO), cortical blindness (CB), and POVL of unknown origin. Among these, causes of unilateral vision loss can be ION, CRAO, BRAO, and acute angle glaucoma.

An ischemic insult to the optic nerve can result in ischemic optic neuropathy which may be anterior or posterior depending on the part of the nerve affected. Posterior ischemic optic neuropathy (PION) is most commonly seen after spine surgeries and anterior ischemic optic neuropathy (AION) frequently reported after cardiac operations.<sup>[3]</sup> Posterior part is most commonly affected due to its poor vascular supply. The posterior portion of the optic nerve has its main vascular supply from pial vessels derived from branches of the ophthalmic artery. These vessels are incapable of autoregulatory control which is why this part of the nerve is particularly vulnerable to a fall in perfusion pressure or anemia. ION occurs because of hypoperfusion or decreased oxygen delivery to the optic nerve. Potential etiologic factors causing postoperative ION are prolonged surgery in the prone position, decreased ocular perfusion pressure, blood loss and anemia/hemodilution, and infusion of large quantities of IV fluids.<sup>[4]</sup> Almost all risk factors were present in our case also. An interplay of several factors such

as blood loss, episode of hypotension, and long surgery could have led to vision loss in this patient. Although an SBP above 80 mm Hg cannot be labeled as very low, in prone position, venous congestion of head leading to raised intraocular pressure may occur. Hence, even SBP >80 mm Hg may not have been enough for adequate perfusion of optic nerve. Though there is no recommended transfusion threshold for elimination of POVL, regular hematocrit monitoring should be done intraoperatively in patients with substantial blood loss. Vasopressors are also implicated in development of POVL.<sup>[5,6]</sup> Vasoconstriction of the peripheral arterioles when BP is already on the lower side may lead to ischemia of the optic nerve. Large fluid resuscitation and resulting hemodilution can also be the pathogenic mechanism in development of PION. Abnormal autoregulation in an individual adds to the woes. Fluorescein fundus angiography is normal in PION but could not be performed in this patient because of logistic reasons.

CRAO is a common cause of unilateral blindness caused either by external compression of eye, decreased retinal blood flow, or impaired venous drainage from retina. Signs and symptoms of CRAO are unilateral vision loss, no light perception, afferent pupil defect, periorbital/eyelid edema or both, chemosis, proptosis, ptosis, paresthesias of the supraorbital region, hazy/cloudy cornea, and corneal abrasion.<sup>[7]</sup> Macular/retinal edema, cherry red spot, or attenuated retinal vessels are typical. External compression by horseshoe headrest may increase the risk of eye compression and CRAO, but there is no relation of type of headrest used and development of ION.<sup>[8]</sup> In American Society of Anesthesiologists POVL Registry, 19.2% of patients who developed ION had their head supported by Mayfield pins.<sup>[2]</sup> Several new devices such as eye protectors, pillows, and tables with mounted mirrors or video cameras for continuous monitoring of eyes are used intraoperatively.<sup>[9]</sup> There are no recommendations on type of headrest devices to be used, but regular intraoperative assessment of eyes and documentation is important. In our patient, no periorbital pressure signs or cherry red spot was visible on eye examination. However, absence of cherry red spot does not exclude CRAO. It is considered equivalent to cerebral stroke and window time for retinal ischemic time is up to 6 h.<sup>[10]</sup> Therapeutic options include nitrate, inhalation of hyperbaric oxygen, ocular massage, IV acetazolamide and mannitol, and paracentesis of anterior chamber and steroids without proven advantage. Vision loss is usually profound and permanent.

BRAO will lead to only field cuts and not complete blindness as in this case. Findings of acute angle closure glaucoma, such as painful, red eye were also not seen. Orbital apex syndrome is characterized by multiple cranial nerve deficits

associated with a mass lesion near the apex of the orbit. Raised intraorbital pressure (IOP) can compress arterial and venous circulations leading to CRAO and optic nerve injury. It was a possible differential diagnosis but on fundus examination, no features of raised IOP could be obtained. MRI orbit did not show any mass effects near the orbital apex.

The exact etiology of vision loss could not be determined but CRAO seemed to be one of the probable causes. In this case, NCCT and MRI head revealed no abnormal findings suggestive of CB or pituitary apoplexy. MRI orbit was also unremarkable. Patient was discharged with advice for further evaluation and management in Neuro-ophthalmology Department.

The exact etiology of POVL in this case could not be found out and we may encounter this dilemma more often than it is described in literature. The diagnosis of CRAO and PION can be the probable lesions responsible for vision loss in our patient. However, the vision outcome in both is poor and risk factors should be taken care of before taking up the patient for surgery.

Despite our best efforts for prevention of POVL, it may still occur in any case. This makes it important to obtain informed consent for POVL.<sup>[11]</sup> No doubt, it sounds alarming to the patients and may cause anxiety for which opinion regarding obtaining consent varies widely.

To conclude, vision loss is being described for the first time in patient undergoing surgery for brachial plexus neuropathy. Hypotension, use of vasopressors, hemodilution along with probably individual anatomic circulatory variation could have led to vision loss in this patient. A 10°-20° head up position of a patient placed prone, can prevent increase in intraocular pressure. Throughout the surgery, we should repetitively ensure that there is no extrinsic compression on the eyes. POVL is not totally preventable, so an informed consent if obtained preoperatively may help in medicolegal cases.

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#### Conflicts of interest

There are no conflicts of interest.

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