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Case Report

Unilateral Vision Loss after Prolonged Prone Position in Spinal Surgery

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ABSTRACT

A 68 year old male developed unilateral vision loss after prolonged spinal surgery under general anesthesia in prone position. Left eye examination revealed hand movements close to face, proptosis, afferent papillary defect and complete ophthalmoplegia. Despite prompt medical treatment vision had deteriorated but there was an improvement in eye movements and fundus showed a pale optic disc at 4 weeks follow-up. Postoperative visual loss (POVL) is a rare but serious complication with an estimated incidence of 0.01–1% after non-ocular surgery. The known causes for POVL are ischaemic optic neuropathy, central retinal artery occlusion and cortical blindness. We suspect the potential aetiological factor for POVL in our case is ischemic optic neuropathy due to prolonged compression of orbital contents.

Keywords: postoperative visual loss (POVL), prone position, ischemic optic neuropathy, spinal surgery

INTRODUCTION

It is estimated that 1 case per 100 spine surgeons annually will have a significant vision complication after surgery. [1] Studies have shown that spinal surgery is the leading cause of postoperative vision loss, replacing cardiac surgery.[2] The three recognized causes of postoperative visual loss are ischaemic optic neuropathy, central retinal artery thrombosis and cortical blindness.[3] Sudden unilateral or bilateral visual loss occurring after general anesthesia has been reported and attributed to various causes including hemorrhagic shock, blood dyscrasia, hypotension, hypothermia, coagulopathy disorders, direct trauma, embolism and prolonged compression of the eyes.[4] However, visual loss with total ophthalmoplegia as a surgical complication has rarely been described as a consequence of prolonged compression of the eye. [5,6]

CASE REPORT

A 68 year old male presented with pain in upper and lower back since past 1 year, radiating to both lower limbs and associated with tingling sensation. Since past 5 months patient was unable to walk without support. A magnetic resonance imaging (MRI) scan revealed cervical

myelopathy from C7-T1 with lumbar canal stenosis at L3-L4 and L4-L5 vertebrae. The patient had a history of hypertension but had never received medical treatment. Blood pressure recorded on admission was 160/100 mmHg and the patient was started on Tablet Amlodipine (5 mg) once a day. His blood pressure preoperatively was 140/90 mmHg. The preoperative blood investigations were normal.

Laminectomy and fixation at C7-T1 and laminectomy at L3-L4 and L4-L5 was performed. Under general anesthesia, the patient was put on the operating table in the prone position with his head resting over a horseshoe shaped head support with patching over both the eyes. The total time for the surgery was six hours and an additional half hour was required for induction of anesthesia. Intraoperative blood pressure was maintained on an average 90/60 mm of Hg. Total fluid loss was 1000 milliliters and two units of packed RBCs were transfused. The surgery was uneventful.

After recovering from general anesthesia the patient complained of pain and swelling over the left eye. On examination left eye was found to have visual acuity of hand movements with a positive light perception and accurate projection of rays, periorbital swelling, 4 mm of axial proptosis, conjunctival chemosis, corneal abrasion with absent corneal sensation, afferent pupillary defect and complete ophthalmoplegia. Digitally intraocular pressure was on the higher side. The fundus was not visualized due to the severe chemosis, corneal haze and cataract. Exposure keratitis with positive fluorescein stain developed after 48 hours. Right eye anterior segment was normal and posterior segment showed grade II hypertensive retinopathy. MRI brain with orbit revealed proptosis enlarged extra ocular muscles with inflamed retro orbital fat on the left side and cerebro-cortical atrophy with periventricular ischemic changes. Magnetic resonance angiography (MRA) and venography (MRV) were normal. Cavernous sinus thrombosis was suspected, but not confirmed by magnetic resonance imaging and venography.

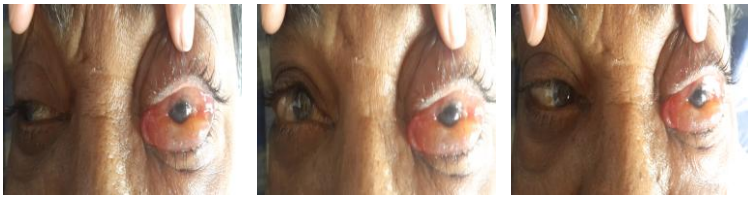
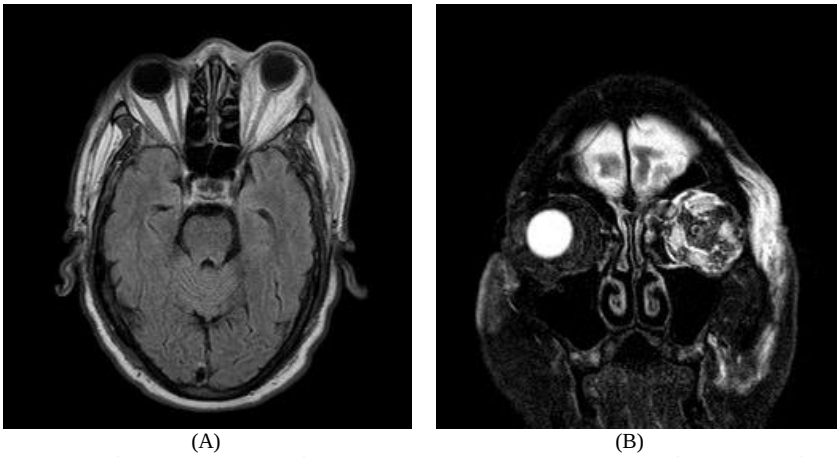


Figure 1: Left eye showing periorbital swelling, severe chemosis, corneal abrasion and complete ophthalmoplegia.

Patient was started on IV Ceftazidime (2 gm) 8 hourly, IV Vancomycin (1 gm) 12 hourly, IV Metronidazole (400 mg) 8 hourly, topical lubricating and antibiotic eye drops at frequent instillations and cycloplegic eye drops. Low molecular weight heparin (Enoxaparin sodium) 0.6 mg subcutaneously was started on second postoperative day. Tablet Prednisolone (60 mg) once a day was started on fifth postoperative day and continued up to 15 days in tapering doses. After 1 week conjunctival chemosis had reduced but there was no improvement in visual acuity, pupil reaction and extraocular movements. Left eye patching was advised on discharge. On 2 weeks follow up there was no improvement in visual acuity and ocular movements. At 4 weeks follow up visual acuity in left eye was light perception with mild lid edema, no chemosis, exposure keratitis, afferent pupillary defect and dense

cataract. There was an improvement in eye movements with mild restriction on lateral side. Fundus was not clearly visualized however disc showed changes suggestive of optic atrophy.



(A) (B)

Figure 2: (A) Axial scan: Orbital fat and overlying soft tissue show hypointensity on T1W. Inflammation of extraocular muscles with involvement of overlying soft tissue and proptosis of left eyeball (B) Coronal STIR: Orbital fat and overlying soft tissue show hyperintensity.

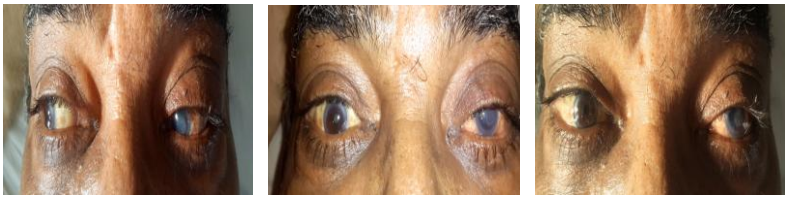


Figure 3: 4 weeks follow up- left eye showing mild lid edema, exposure keratitis and mild restriction of ocular movements on lateral side.

DISCUSSION

It is estimated that the incidence of visual disturbances and blindness after anesthesia for the major surgical procedures varies between 0.05% and 1%. [7] The American Society of Anesthesiologists (ASA) database reports that 81% of postoperative visual loss is diagnosed as ischaemic optic neuropathy (ION) and the remainder as retinal artery thrombosis. 67 % of all the reported cases of postoperative visual loss occurred after spinal surgery in the prone position. [8]

Various mechanisms have been postulated for postoperative vision loss. The most obvious is the effect of direct external pressure by a headrest or other support on the orbital contents causing an increase in intraocular pressure, which may lead to retinal ischaemia and visual loss. This has been named 'Hollenhorst syndrome' and is usually linked with examination findings consistent with central retinal artery occlusion. [9]

Prone positioning tends to increase venous pressure and peak inspiratory pressure, which in turn increase intraocular pressure. Raised IOP superimposed with intraoperative anemia and hypotension can lower the perfusion pressure to the optic nerve head and contribute to ischemic optic neuropathy. [9,10] The above changes along with kinetics of movement of intravascular fluid into extravascular tissues and spaces in the orbit manifests postoperatively as profound lid and orbital edema. [2] Patients with pre- operative risk factors (glaucoma, hypertension, diabetes, smoking and atherosclerosis) having prone spinal surgery at risk of severe blood loss are now known

to be at significantly increased risk of postoperative visual loss secondary to ischaemic optic neuropathy. [8]

Mukherjee *et al* reported a similar case where the patient developed CRAO with complete ophthalmoplegia after an uneventful spinal surgery. The suggested mechanism was, collapsed arterial and venous channels of the orbit dilated on subsequent release of the external pressure which led to the transudation of fluid through the permeable walls into the tissue spaces. This accumulation of fluid resulted in orbital edema, proptosis, paresis of ocular movement, and retinal edema. [10]

Ho *et al* reviewed published case reports on ION after spine surgery with the patient in the prone position. Most of the patients were diagnosed with posterior ION (17/22) and only a minority (5/22) had anterior ION. They found that visual loss was frequently bilateral (40% of anterior ION and 47% of posterior ION cases). They suggested that long surgery in prone position, blood loss, hypotension, hemodilution, and administration of large amounts of intravenous fluids are potential risk factors for occurrence of postoperative ION. [11]

Roth *et al* surveyed 60,965 patients who underwent anesthesia for non-ocular surgery, of which ocular findings were found in 34 patients (0.056%). Corneal abrasion was the most common injury (21 patients) followed by conjunctivitis or red eye (7 patients), blurred vision persisting longer than 1 day (3 patients), chemical injury, permanent vision loss, and eyelid hematoma (1 patient each). Mechanisms postulated were compression, hypotension, hypoxia, and anemia. [12]

Ophthalmoplegia after spinal surgery is also an exceptional complication. West *et al* reported a case that developed unilateral total external ophthalmoplegia and unilateral visual loss following scoliosis surgery. The ophthalmoplegia was initially thought to be due to cavernous sinus thrombosis but MRI scan showed no evidence of thrombosis. The authors came to the conclusion that prolonged compression of the orbital contents during anesthesia led to blindness and ophthalmoplegia. [5] Halfon *et al* suggest that reversibility of ophthalmoplegia probably depends on the degree of ischaemia suffered by the extraocular muscles and the III, IV, and VI cranial nerves. [6]

Prolonged compression of orbital contents during surgery in prone position can give rise to ischaemia of optic nerve and other orbital contents. This is believed to be possible mechanism for visual loss and complete ophthalmoplegia in our patient. Corneal abrasion was thought to be due to improper patching of eyes intraoperatively. Increased orbital venous pressure and extravasation of intracellular fluid into orbital space might be responsible for periorbital oedema and chemosis.

Thus, sight threatening ophthalmic complications is well recognized in patients who undergo prolonged spinal surgery under general anaesthesia. Some are preventable by taking appropriate measures but others are not, this may be due to poorly understood mechanism. The importance of head positioning by keeping the head above the heart to avoid venous pooling in the orbit, avoidance of excessive use of replacement fluids and preoperative high-risk recognition like glaucoma, hypertension can help in minimizing any impairment of ocular perfusion.

CONCLUSION

Postoperative unilateral visual loss in non-ocular surgery is a very devastating complication. The anesthetist and surgeon should be well aware about the head position of patient to avoid unnecessary ocular compression. Patients with high

preoperative risk factors should be counseled prior to surgery. Appropriate measures should be taken intra operatively as well as in early postoperative period to prevent this visual complication.

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