

Case report

Unilateral visual loss due to central retinal artery occlusion with total ophthalmoplegia following cervical spine surgery in prone position

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Abstract

Background: Blindness after spinal surgery is a rare complication, but it is serious, irreversible and incurable. Central retinal artery occlusion (CRAO) is rare after spinal surgery and ophthalmoplegia is even rarer. **Case:** A 52-year-old male patient complained of loss of vision in right eye immediately after cervical spine surgery. On examination, the patient's visual acuity in right eye was absent perception of light. Right eye pupil was dilated and relative afferent pupillary defect (RAPD) was present. Extraocular movements were absent in all gazes in right eye. Intra-ocular pressure (IOP) was 26 mmHg in right eye and 16 mmHg in the left. Posterior segment examination revealed blurred disc margin with ischemic whitening of retina, thin and attenuated retinal arterioles and a central cherry red spot in right eye. Left eye was essentially normal. **Observations:** The causal factors of blindness in the patient were likely ischemia of the retina after venous congestion or temporary arterial occlusion resulting from changes in pressure to the tissues of the orbit. Factors including prolonged prone positioning with head end dependent position and possibility of orbital compression by the headrest could have contributed to impaired venous drainage, increase in IOP and reduction in perfusion pressure. **Conclusion:** Loss of vision post spinal surgery is a rarest of complication yet grave and irreversible. Because the problem involves mainly prone positioning of the patient, an appropriate position should be found so that facial and ocular compression can be avoided.

Keywords: Visual loss, Central retinal artery occlusion, Ophthalmoplegia, spine surgery, Prone position

Introduction

Postoperative visual loss is a rare but devastating complication, with estimates for spinal and cardiac surgery as high as 0.2% and 4.5% (Stevens et al, 1997). Ophthalmoplegia

following surgery is even rarer (West et al, 1990). Multiple preoperative risk factors hypertension, diabetes mellitus, polycythemia, smoking, renal failure, narrow angle glaucoma and collagen vascular disease and intra-operative risk factors such as prone positioning and raised intraocular pressure have been implicated as a cause of vision loss (Myers et al 1997; Warner 2006; Hunt et al 2004; Kamming

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& Clarke 2005). We report a case of central retinal artery occlusion and ophthalmoplegia following spinal surgery in prone position.

Case report

Ophthalmologist's opinion was sought for 52-year-old male patient who complained of loss of vision in right eye immediately after the surgery. Patient was operated upon in prone position in which C1 lateral support and C2 pedicle plate screw fixation from posterior approach was done. Perioperative parameters during surgery were systolic blood pressure between 100 to 140 mm Hg, heart rate between 70 to 90 per minute, blood loss was 600-700 mL and duration of surgery was 210 minutes.

On examination the patient's visual acuity was absent perception of light in right eye (OD), and 20/20 in left eye (OS). Right eye pupil was dilated and relative afferent pupillary defect (RAPD) was present. Left eye pupil was normally reacting. Ecchymosis was present in right sided periorbital region. Extraocular movements were absent in all gazes in right eye and left eye was found to be normal. Anterior segment examination was grossly normal in both eyes. IOP was 26mm Hg in OD and 16 mmHg in OS. Posterior segment examination revealed blurred disc margin with ischemic whitening of retina, thin and attenuated retinal arterioles and a central cherry red spot in right eye (Fig 1a). Left eye posterior segment examination was essentially normal (Fig 1b). Provisional diagnosis of Right sided central retinal artery occlusion (CRAO) with total ophthalmoplegia was made. As Immediate measure ocular massage and anterior chamber paracentesis was done. Intravenous 20% mannitol was started according to his body weight. After emergency management there was no improvement in visual acuity of right eye. Then, he was started on intravenous methyl prednisolone at 1g/day for three days along with antiglaucoma medication. MRI Scan showed edema in right medial rectus and inferior

oblique muscles (Fig 2). It showed no obvious signal abnormalities in the right optic nerve in its intraorbital, orbital apex or in suprasellar region. Visual evoked potential (VEP) was done which showed reduced amplitude and delayed latency in right eye (Fig 3). Intravenous methyl prednisolone was stopped and patient was shifted to oral steroid at 1.5mg/kg/day once daily and tapered in one month. After three month follow up, there was no improvement in vision but extraocular movement was improved in all gazes.

Discussion

The three recognized causes of post operative visual loss are ischemic optic neuropathy, CRAO and cortical blindness, the most common being ischemic optic neuropathy (Myers et al 1997; Kamming & Clarke 2005). The mean age of patients at presentation was 46.5 years, average operative time was 410 minutes while average blood loss was 3500 mL (Myers et al 1997). Though in our case, age at presentation was 52 years but operating time was 210 minutes and blood loss was 600-700 mL. A cherry red spot at the macula with a white ground glass appearance of the retina and attenuated arterioles with preserved choriocapillaris and an afferent pupillary defect or reduced pupillary light reflex are diagnostic of CRAO.

In most cases of postoperative visual loss, the etiology is not clear and a number of potential risk factors have been implicated. Preoperative risk factors include hypertension, diabetes mellitus, polycythemia, smoking, renal failure, narrow angle glaucoma and collagen vascular disease (Myers et al 1997; Warner 2006).

Increased intraocular pressure (IOP) has been recognized as another important risk factor for post operative visual loss. It has been demonstrated that IOP increases significantly in anesthetized prone patients (Hunt et al 2004). Significant increase in IOP has also been demonstrated in healthy subjects when

subjected to prone position (Lam & Douthwaite 1997). A 10-degree reverse Trendelenburg position has been found to normalize IOP in the prone position (Ozcan et al 2004). Prolonged prone position with dependent position of the head might be a significant contributing factor in increasing IOP due to increased central venous pressure. Factors causing raised central venous pressure are reduced venous return and inadvertent pressure on abdomen in prone position (Kamming & Clarke 2005).

The causal factors of blindness in our patient were likely to be ischemia of the retina after venous congestion or temporary arterial occlusion resulting from changes in pressure to the tissues of the orbit. Factors including prolonged prone positioning with head end dependent position and possibility of orbital compression by the headrest could have contributed to impaired venous drainage, increase in IOP and reduction in perfusion pressure. Inadvertent shift of head and subsequent entrapment of right eye in headrest could have led to undue pressure on eye and adnexae. Stagnation of blood flow could have led to thrombus formation in the orbital circulation.

Conclusion

Loss of vision post spinal surgery is a rarest of complication yet grave and irreversible. Because the problem involves mainly prone positioning of the patient, a right position should be found so that facial and ocular compression can be avoided. Any change in the patient's position must be taken care of so as to prevent aforementioned sequelae. Particular attention must be given to bradyarrhythmias as they signal vagal stimulation caused by increased intraorbital pressure.

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