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CLINICAL INFORMATION

Post operative visual loss after cervical laminectomy in prone position



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PALAVRAS-CHAVE

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Abstract Postoperative visual loss is a rare and devastating complication. The estimated incidence is 0.01–1% after non ocular surgery. It has been reported after spine, cardiac and head and neck surgeries. We report a patient who was operated for cervical laminectomy in prone position and complained of loss of vision in one eye postoperatively. He was thoroughly investigated after visual loss. The case was diagnosed as central retinal artery occlusion (CRAO) of the left eye. Here we consider the potential etiological factors causing this unilateral loss of vision and try to suggest strategies to reduce the incidence of the complication in spinal surgery.

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Perda visual no pós-operatório de laminectomia cervical em pronação

Resumo A perda visual pós-operatória é uma complicação rara e devastadora. A incidência estimada é de 0,01–1% após cirurgia não oftalmológica. Há relatos de sua ocorrência após cirurgias da coluna, cardíaca e de cabeça e pescoço. Relatamos o caso de um paciente submetido à laminectomia cervical em pronação que se queixou de perda de visão em um dos olhos no pós-operatório. O paciente foi profundamente investigado após a perda visual. O caso foi diagnosticado como oclusão da artéria central da retina (CRAO) do olho esquerdo. Aqui consideramos os potenciais fatores etiológicos que causam essa perda unilateral da visão e tentamos sugerir estratégias para reduzir a incidência dessa complicação em cirurgia de coluna vertebral.

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Introduction

Postoperative visual loss is an unexpected and rare complication and estimated incidence is 0.01–1% after non ocular surgery.^{1–4} Review of literature reveals such cases as isolated case reports. Patil et al.⁵ found an overall rate of 0.094% in spine surgery discharges in US Nationwide Inpatient Samples (NIS). Most common causes of postoperative vision loss are ischaemic optic neuropathy, central retinal artery thrombosis and cortical blindness. Among these ischaemic optic neuropathy is frequently observed cause of postoperative visual loss following general anesthesia.¹ The second most prevalent cause of postoperative visual loss in patients operated in the prone position for spinal surgery is central retinal artery occlusion (CRAO). There are some recognized preoperative risk factors which include diabetes mellitus, hypertension, smoking, renal failure, narrow angle glaucoma, polycythemia, atherosclerotic vascular disease and collagen vascular disorders.⁶

Case report

A 56 year old man (170 cm, 88 kg, BMI 30.45 kg.m⁻²) came to our hospital in the Neurosurgery department with the presenting complain of pain in left shoulder radiating to left arm for 2 years. He had tingling sensation in left arm and pain in the right arm for last 2 years. Patient had history of diabetes mellitus since 4 years. His blood sugar was deranged because he had not taken oral hypoglycemic agent for last fifteen days. He was put on a diabetic diet and antidiabetic drug was started. There was history of smoking since ten years. The patient had no other significant disease.

On examination there was mild upper limb weakness more on left than right. Sensory component was intact. There was no restriction of movement in upper limb. A Magnetic Resonance Imaging (MRI) scan of cervical spine was performed which revealed advanced cervical spondylosis with disco-degenerative nerve root compression at C3–4 to C6–7 level with secondary central canal stenosis. His all preoperative routine investigations were within normal limit except blood sugar which was 216 mg.dL⁻¹. His blood pressure was 126/78 mm Hg and his heart rate was 82 beats/min.

Considering the neurological involvement the surgeon decided to perform Endoscopic laminectomy at C5–6 and C6–7 level. General anesthesia was planned for the patient. Two 16 gauge peripheral intravenous cannulae was put in both the hands. Anesthesia was induced with midazolam 1 mg, propofol 150mg and a bolus of fentanyl 150 mcg. Tracheal intubation with a size of 8.5 mm reinforced oral endotracheal tube was facilitated with vecuronium 6 mg. Urinary catheter was inserted and patient was positioned prone. The eyes were protected with chloramphenicol ointment before being taped and padded. Patient was subsequently put on the operating table in the prone position. His neck was maintained in the midline position using a horse-shoe head holder with slight flexion, so that his back would remain in neutral position with the head remaining slightly dependent. Anesthesia was maintained with isoflurane 0.8–1.4%. Total duration of surgery was around 170 min and additional 70 min were required for the induction and cessation of anesthesia. Systolic blood pressure



Figure 1 Fundus picture of left eye showing pallor. Retinal pallor with attenuated arteries.

of the patient was maintained between 90 and 120 mm Hg throughout the procedure. There was 400 mL of blood loss during the surgery which was replaced with 1500 of normal saline and maintain the blood pressure on acceptable level.

At the end of operation, the patient was turned into supine position and transported to the critical care unit with endotracheal tube in place for elective ventilation. There was mild facial swelling and bilateral conjunctival congestion. During overnight elective ventilation patient maintained his vital parameters. After reevaluation he was extubated in the morning. On the first postoperative day following extubation, he complained of reduced vision in the left eye. Immediate ophthalmological examination was conducted by an ophthalmologist.

On examination visual acuity was found to be reduced to perception of light in the left eye while in the right eye it was normal (6/6). Left eye had puffiness of lids, mild proptosis and conjunctival congestion. Extraocular movement showed restricted adduction and also in elevation and depression in left lateral gaze. Intraocular pressure of both eyes were normal. Left pupil was moderately dilated with Relative Afferent Pupillary Defect (RAPD). Fundus examination revealed pallor with oedema of central retina and a dull foveal reflex (Fig. 1). Retinal arteries were thin with altered AV ratio. Fundus examination of right eye was normal (Fig. 2). Patient was given a course of methylprednisolone IV 1 g daily for 3 days. Despite this, the vision deteriorated to no perception of light. On fifth day MRI of right eye was normal but left orbit showed diffuse swelling of belly of medial rectus muscle. Case was diagnosed a central retinal artery occlusion (CRAO) of left eye.

Discussion

Postoperative loss of vision after spine surgery is a rare but disastrous complication. Most probable intraoperative causes of visual loss include patient positioning, blood loss, intraoperative hypotension, long duration of surgery and excessive hydration or combination of these factors. The



Figure 2 Normal fundus picture of right eye.

commonly reported causes of postoperative visual loss are central retinal artery occlusion (CRAO) or vein occlusion, ischaemic optic neuropathy and cerebral ischaemia.^{7,8} Diabetes mellitus, smoking, chronic hypertension, vascular disease and coagulopathy are the potential risk factors for this complication.⁶ The role of these factors in the pathogenesis of visual loss in the setting of spinal surgery still remains unknown. Our patient was a known smoker and was also suffering from diabetes mellitus.

External compression over the orbit, retro bulbar haemorrhage, or too small orbital space can also adversely affect retinal circulation leading to CRAO and retinal ischaemia. Leibovitch et al.⁹ in their case report have mentioned about ischaemic orbital compartment syndrome in prone position. It was considered that in prone patient a 10° reverse Trendelenberg position normalized the intraocular pressure.¹⁰ Our patient was heavy built and obese (BMI 30.45 kg.m⁻²), so orbital space could already have been compromised due to abundant orbital fat, and the slightest insult from prolonged prone position may have precipitated retinal ischaemia due to central retinal artery occlusion. Moreover diabetes is known to cause vascular changes which would have precipitated the event in our case. Jampol and colleagues¹¹ has also reported ischaemia of the ciliary arterial circulation from ocular compression. Retinal embolus is another potential cause for artery occlusion but this generally occurs after a cardiac surgery.

In review of published cases of CRAO after spine surgery, Kumar and colleagues¹² described signs and symptoms that included unilateral vision loss, no light perception, afferent pupillary defect, periorbital, eyelid oedema or both, chemosis, proptosis, ptosis, paraesthesia of the supra-orbital region, hazy or cloudy cornea, loss of eye movements, ecchymosis or other trauma near the eye. Proptosis and extraocular muscle swelling has been seen in some cases in early orbital Computed Tomography (CT) or Magnetic Resonance Imaging (MRI). Macular or retinal oedema, cherry red spot or attenuated retinal vessels were typical. Our patient's clinical presentation resembled the case reported by Kumar et al.

Ischaemic optic neuropathy (ION) has been reported in wide variety of surgical procedures like cardiothoracic

surgery,¹³ instrumented spinal fusion operations,¹⁴ head and neck surgery,¹⁵ nasal and sinus surgery.¹⁶ Anterior ischaemic optic neuropathy typically appears as optic disc oedema and peripapillary haemorrhage and occurs due to abnormal optic nerve blood flow from hypotension, anaemia or an emboli. Prolonged prone position also increases the intraocular pressure causing acute angle closure glaucoma. It is found that raised IOP significantly decreases retinal and choroidal blood flow and damages the retinal ganglion cells. But in this condition the eyes become painful and red along with corneal oedema. The condition is bilateral and accompanied by headache, nausea and vomiting. Vision can be partially salvaged in both these conditions with the use of high dose steroids and with immediate use of IOP lowering drugs. In our patient, blood pressure was maintained throughout the operation and blood loss was also not significantly high. Visual loss was painless and unilateral. Unfortunately our patient had no features of either ION or glaucoma but showed a clear picture of a CRAO with no recovery of vision.

Some treatment strategies can be attempted in cases of CRAO, like ocular massage to lower intraocular pressure and improve blood flow in arteries, or to dislodge any embolus. 5% CO₂ in oxygen can be inhaled to enhance dilation of arteries and improve O₂ delivery. Intravenous acetazolamide may be given to increase retinal blood flow. All this attempt may only be beneficial if given within 60–100 min of occurrence of CRAO, otherwise permanent damage ensues. Hayreh and Weingast found that irreversible retinal damage followed 100 min of ischaemia where their studies surgically occluded the central retinal artery.

Conclusion

Reduction in ocular blood flow is possible by numerous factors, which in presence of risk factor can cumulatively lead to postoperative visual loss. Patients with preoperative risk factors (hypertension, diabetes, smoking, atherosclerosis, coagulopathy) should undergo preoperative ophthalmologic examination. Patient and relatives should also be prewarned about the unpredictable but rare risk of perioperative visual loss. High risk patients should be positioned with the head level little higher than the heart when possible. In addition the head should be maintained in a neutral forward position i.e. 10° reverse trendelenberg position in the mid-line, when possible. Perioperative hypotension, anaemia and excessive hydration should be avoided. Immediate ophthalmologic examination should be done for this unusual complication.

Conflicts of interest

The authors declare no conflicts of interest.

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