Monocular blindness: a complication of intraoperative positioning in posterior cervical spine surgery

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ABSTRACT

Postoperative visual loss after spine surgery is a rare but devastating complication. Although uncommon, reports of postoperative blindness are documented in spine and cardiopulmonary bypass surgeries with an incidence varying between 0.05 percent and one percent. A 62-year-old man presenting with cervical myelopathy underwent C3-C6 posterior laminectomy, decompression and lateral mass plating. Unfortunately, he developed unilateral blindness in his right eye. The potential aetiological factors and ways to prevent this disastrous complication are discussed.

Keywords: blindness, cervical spine surgery, monocular blindness, postoperative blindness, spinal surgery

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INTRODUCTION

Postoperative visual loss after spine surgery is an uncommon but severe complication[1-5]. The reports of postoperative blindness are documented in spine and cardiopulmonary bypass surgeries, with an incidence varying between 0.05% to 1%[6]. The three recognised causes for blindness are ischaemic optic neuropathy, central retinal artery thrombosis and cortical blindness. We present a 62-year-old man who underwent C3-C6 posterior laminectomy and decompression for cervical myelopathy, and who unfortunately developed unilateral blindness in his right eye.

CASE REPORT

A 62-year-old man who had carcinoma of the mandible was scheduled for radical excision of his left mandible tumour. Incidentally, he presented with neck pain radiating to the right upper limb, and progressive weakness in the right upper and lower limb for several weeks. On examination, he was found to have hyperreflexia and hypertonia in his lower limbs, with grade IV power in the right upper and lower limbs. His radiographs and magnetic resonance (MR) imaging were suggestive of cervical spondylotic myelopathy, with severe central canal and bilateral foraminal narrowing at C4/5 and C5/6 levels. Milder degrees of spinal and foraminal stenosis were noted at C3/4 and C6/7 levels.

The patient underwent surgery consisting of C3-C6 posterior laminectomy and decompression with lateral mass plating. The patient was placed in a procubitus (prone) position on a horseshoe headrest intraoperatively, with reverse Trendelenburg position. The duration of surgery was 120 minutes. The haemodynamic status of the patient was within normal range intraoperatively and postoperatively. Postoperatively, the patient developed painless unilateral loss of vision in right eye. There was no swelling of the eyeball or any corneal haziness. Based on ophthalmoscopical findings of a cherry red spot over the retina, a diagnosis of a central retinal artery occlusion was made.

Computed tomography (CT) of the brain was normal, with no suggestion of any cortical damage. The haematological results were suggestive of thrombocytosis, with a raised erythrocyte sedimentation rate (ESR) preoperatively and postoperatively. The patient was treated with anti-inflammatory drugs and steroids but no improvement was observed.

DISCUSSION

Ischaemic optic neuropathy following spinal surgery in the prone position has been reported previously, although the incidence and risk factors are not fully appreciated by many anaesthetists and surgeons. The plausible reasons for blindness were evaluated and literature search was done. The reasons, which could be attributed to the ischaemic optic neuropathy, were: (a) *Preoperative causes* such as hypertension, diabetes mellitus, smoking, polycythemia, renal failure, narrow angle glaucoma, atherosclerosis, valvular diseases and collagen disorders[7].

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(b) *Intraoperative causes*, with recognised intraoperative factors for developing ischaemic optic neuropathy including hypotension and anaemia[8]. With an increase in intraocular pressure in the prone position, even modest hypotension can lead to critical ischaemia, and decreased perfusion pressure and blood supply to the retina, leading to blindness.

In this patient, the blood pressure was maintained throughout at an acceptable level and there was no sudden or wide fluctuation in the blood pressure. The preoperative haemoglobin level of this patient was 9.8 g/dL. The anaemia could have accentuated the effect of ischaemia of the optic nerve. Considering increased blood loss, sudden or excessive intraoperative blood loss can cause a decrease in the oxygen-carrying capacity of blood. Since oxygen is required for retinal activity to function normally, hypoxia may lead to irreversible damage to the retina.

(c) *Postoperative causes*. Raised intraocular pressure is the sole postoperative cause for ischaemic optic neuropathy. The various reasons for raised intraocular pressure are:

1. Due to increase pressure over the globe: Inappropriate pressure from padding while lying on the horseshoe headrest, leading to extrinsic pressure over the eyeball, causing raised intraocular pressure and ischaemic optic neuropathy.
2. Raised central venous pressure: Central venous pressure is raised due to decreased venous return in the low position of the head and obstruction to venous outflow on turning the head to one side. Central venous pressure may also be increased if there is pressure over the abdomen. Due to absence of venous valves, changes in central venous pressure translate into concomitant changes of ocular venous pressure and therefore can affect intraocular pressure.
3. Prone position: Recently, it has been demonstrated that intraocular pressure increases in the prone position[9]. A study in awake patients noted that there was a significant increase in intraocular pressure when positioned prone. It was also found that ten degrees of reverse Trendelenburg position normalised the pressure while prone[10].
4. Intraoperative change in the position of the patient. The surgeon and the anaesthetist confirmed the position of the head and eye preoperatively. Intraoperative change in this position during surgical procedure could have led to an extrinsic pressure over the eyeball. Also, an enlarged left mandibular tumour swelling may have forced the head to tilt, with resulting extrinsic pressure over the eyeball.

(d) *Miscellaneous causes* include a hypercoagulable state leading to sluggish blood flow and possible ischaemia of the optic nerve. This patient was a known case of carcinoma of the mandible with multiple metastases to the spine, femur, humerus and sternum. The raised ESR and thrombocytosis were secondary to the carcinoma leading to a hypercoagulable state. There is also a possibility of thrombus (metastatic cells) arising due to carcinoma of the mandible. Thrombi arising from the carotids or the heart leading to a sudden occlusion of the central retinal artery could have been another cause, but carotid Doppler ultrasonography and 2D echocardiography ruled out the probability of thrombi arising from carotid arteries or the heart.

The mechanism of blindness is retinal ischaemia secondary to raised intraocular pressure, either due to venous congestion or arterial occlusion. There is a decreased blood flow resulting from venous collapse when intraocular pressure exceeds venous pressure[11]. The retina has a dual arterial supply; one from the central retinal artery supplying the inner layer, and the outer layer of retina depends on diffusion from the choroidal plexus. Any obstruction to the central retina, either external or internal, may thus lead to blindness. A constant oxygen supply is essential for the maintenance of retinal electric activity. Arterial or venous occlusion modifies the haemodynamics in the retinal circuit and produces hypoxia in the affected area. In the case of arterial occlusion, severe hypoxia rapidly (in less than 45 minutes) produces irreversible cell damage in the inner layers of retina[12].

Blindness during spinal surgery is a rare complication but it is serious, irreversible and incurable. It is important for us to be aware and prevent its occurrence, since it is the only rational treatment for the unfortunate condition. On the basis of the available information, the authors recommend the following considerations for prevention of postoperative blindness in spinal surgery:

1. Mayfield headrest or Gardner Wells tongs may be useful to avoid direct pressure over the eyeballs. Also, some non-compressible goggles with soft edges should be used to avoid direct pressure over the eye in the prone position, if soft foam or a headrest is used instead of tongs.
2. A ten-degree reverse Trendelenburg position is recommended for all spine surgery done in the prone position to decrease intraocular pressure.
3. The surgeon should make sure that the eyes are visibly free from contact with the head holder, once the patient is prone. The anaesthetist must continuously check the eyes throughout the procedure, especially after application of traction

or after the movement of the patient during the procedure. The operating surgeon should inform the anaesthetist regarding a change in position, so that the eyes are kept free from external pressure.

4. The anaesthetist should look for sudden inadvertent intraoperative bradyarrhythmias. Increase in intraocular pressure may lead to secondary vagal stimulation resulting in arrhythmias. The arrhythmias should alert the anaesthetist about the possible external pressure over the globe.
5. Careful use of hypotensive anaesthesia. Although hypotensive anaesthesia is preferable in spinal surgery, it would be better to establish a baseline systolic and mean blood pressure for each patient, and not allow the pressure to drift below the predetermined value, thus balancing the risk-benefit ratio in the patient.
6. The surgeon should be vigilant about the intraoperative blood loss. If more blood loss is expected, use of cell savers and autologous blood transfusion is recommended.
7. To identify and optimise preoperative risk factors like hypertension, diabetes mellitus, hypercoagulable states, smoking, anaemia, increased blood loss and collagen disorders, which increases the risk of postoperative blindness. It would be preferable to explain the possibility of such a complication to high-risk patients.
8. There is one report in Japanese literature suggesting the use of Urokinase, PGE1 and hyperbaric oxygen therapy with stellate ganglion block as the treatment for central retinal artery obstruction[13], but there is no other supporting literature.

More research in these treatment modalities is needed to find a solution for this disastrous complication. The current case is unusual because there was no excessive intraoperative blood loss, hypotension or prolonged duration of surgery. But the patient did have a hypercoagulable state due to carcinoma of the mandible, which was a perioperative risk factor. The facial bone tumour could be an important factor, which can change the position of head and eye intraoperatively. This suggests that, despite the various reports of perioperative visual loss in the past, the aetiological factors still remain an enigma. The factors, which still need attention, are the variations in ocular vascular anatomy and haemodynamics.

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