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Cortical Blindness Following Spinal Surgery: Very Rare Cause of Perioperative Vision Loss

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A 38-year-old man was operated with posterior spinal decompression and pedicle screw instrumentation for his L2 fracture with incomplete neurological deficit. In the recovery, he complained of blindness in both eyes after twelve hours. Computed tomographic scan and magnetic resonance angiography revealed bilateral occipital lobe infarcts. He remained permanently blind even after three years follow-up. Though rare, perioperative vision loss is a potential complication following spine surgery in prone position. We report a rare occurrence of cortical blindness following lumbar spine surgery.

Key Words: Blindness, Prone, Surgery, Spinal injuries, Postoperative vision loss

Introduction

Vision loss is a very rare but devastating complication of nonocular surgeries, and reported incidence is 0.003% to 0.0008% in the general surgical population [1]. The risk of perioperative vision loss (POVL) has been commonly noted after cardiac and spinal surgeries. The incidence as reported in literature is 8.64/10,000 for cardiac surgeries and 3.09/10,000 for spinal fusions [2].

Most cases of perioperative vision loss following spine surgery are mentioned as case reports in literature [3-8]. The specific pathogenesis of POVL remains elusive in most cases, with much controversy surrounding patient and surgical risk factors. Important causes of POVL include ischemic optic neuropathy (ION), retinal vascular occlusion (RVO) and cortical blindness. Among these major causes, cortical blindness is the rarest cause of POVL [7-9]. Myers et al. [7] reported only three cases of cortical ischemia leading to blindness while reviewing 37 patients of POVL after spine surgery. Because of the rarity of occurrence and as most of the data are retrospective, it is difficult to establish definite cause-effect relationship for the cortical blindness. In this article we report a case of POVL secondary to cortical ischaemia in a 38-year-old man following lumbar spinal fusion without any predisposing factors for vaso-occlusive disease.

Case Report

A 38-year-old manual labourer was brought to the emergency after a high energy motor vehicle accident. He was hemodynamically stable at the time of admission. On primary and secondary survey a lumbar spine fracture possibility was suspected. Radiographic evaluation confirmed our diagnosis and X-ray showed fracture of the second lumbar vertebra (Fig. 1A). Neurologically he had diminished power in both lower limbs (motor power of grade 3 or less around

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hip, knee and ankle joints). He had complete loss of bladder and bowel sensations, but no sensory impairment was elicited in the lower limbs.

His past medical history was not suggestive of any neurological problems, vision problems, diabetes, hypertension, coronary artery disease, deep vein thrombosis, peripheral vascular disease, collagen vascular disorder or previous chest or heart problems. He was a non smoker and a social drinker. His body mass index was 32.4.

Magnetic resonance imaging of the lumbar spine was performed to better delineate the severity of spinal cord injury and compression (Fig. 1B). He was operated after 72 hours of injury. Surgery was carried out with posterior decompression and fusion using pedicle screw instrumentation in the prone position under general anaesthesia (Fig. 2). Constant monitoring of the arterial blood pressure was performed during the surgery. Total duration of surgery was 105 minutes. Perioperative blood loss was 420 ml. Systolic blood pressure throughout the surgery was in the range of 90 to 110 mm Hg. The oxygen saturation as measured by pulse oxymeter was above 98% at all times. There was no other anaesthetic or surgical complication intraoperatively. In the recovery, patient complained of complete loss of vision after twelve hours. He was evaluated by an ophthalmologist, neurologist and a cardiologist. His ocular examination revealed complete bilateral loss of vision with preservation of papillary and corneal reflexes and normal ocular movements. Intraocular pressure and fundus examination were within normal limits. There was no new neurological deficit occurring after surgery and his cerebellar functions were intact. His postoperative haemoglobin was 10.6 g and he did not require any blood transfusion in the perioperative period.

The computed tomographic scan and magnetic resonance angiography of the brain revealed infarcts in bilateral occipital lobes (Fig. 3). Electrocardiography and echocardiography evaluation for underlying cardiac problem did not reveal any cardiac source of emboli. He was treated by the ophthalmologist, but no recovery in vision was observed even after three years follow-up.

Discussion

The article by Berg et al. [10] was surprising to the spine surgeons where they mentioned that the incidence of POVL following ocular surgeries is much lower than that seen in nonocular surgeries. Incidence estimates for POVL after nonocular surgery range from 0.013% for all surgeries up to 0.2% following spine surgery. Ischemic optic neuropathy is the most common cause of POVL accounting for more than 81%, followed by retinal artery thrombosis. Cortical blindness is the rarest cause of POVL with a handful of cases in the literature [11].

Work up of a patient with perioperative visual loss involves consideration of anatomy of the visual pathway. Anterior ischemic optic neuropathy and retinal vascular occlusion reveal remarkable changes on fundus examination, but no such changes are observed in posterior ischemic optic neuropathy (PION). The diagnosis in PION can be made

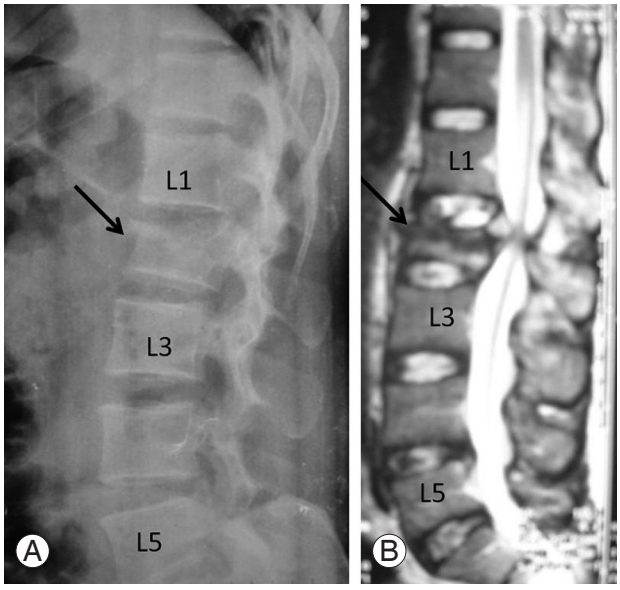

Fig. 1. Radiograph **(A)** and magnetic resonance imaging **(B)** of the lumbar spine showing fracture of the L2 vertebra (arrow).

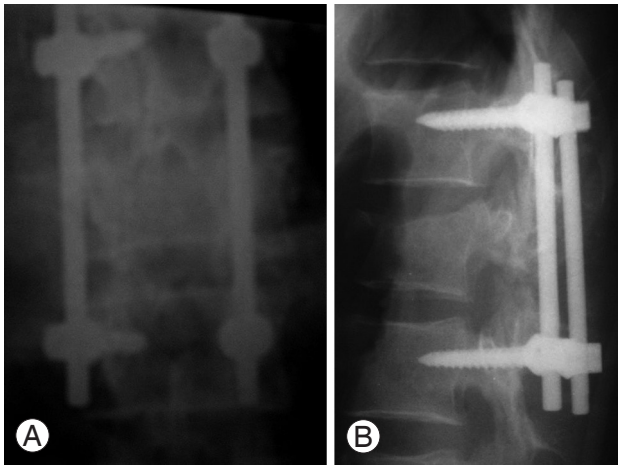

Fig. 2. Radiogarphs (antero-posterior and lateral views) after posterior spinal decompression and pedicle screw fixation for L2 vertebra fracture.

by contrast enhancement seen in the optic nerve on ocular magnetic resonance imaging. Cortical blindness is diagnosed by looking for the ischemic changes in the occipital lobe on intracranial imaging. In our case, the diagnosis was made by cortical occipital changes seen on magnetic resonance imaging with the absence of fundoscopic examination findings.

Despite numerous efforts and explanations, pathogenesis of POVL is still elusive. Various aetiologies' such as fall in systemic blood pressure, anaemia, direct ocular compression, hypercoagulable states, embolism, increased venous pressure, prone positioning during surgery and increased cerebrospinal fluid pressure have been elucidated but none has proved so far [12-15]. Two important factors in cortical blindness are generalised cerebral hypoperfusion and embolism. It has been suggested that more than one factor may be working in any patient making this a multifactorial event [7,10]. Pathogenesis in cardiac surgery is relatively easy to explain. Embolisation may take place due to cardiac and great vessel manipulation, atrial or ventricular fibrillation [7]. The source of emboli in spinal surgery is difficult to explain. It is also unclear why this phenomenon is commoner in surgeries carried out in the prone position. Direct pressure on eye, raised intraocular pressure or vascular congestion does not explain cortical infarcts, but may explain other causes of vision loss such as ION and RVO. Intraoperative hypotension, hypoxia, blood loss and anaemia are contributory factors, but are not found in this patient. Further, they should classically affect the watershed areas of blood supply in the brain which innervate the proximal muscles of upper and lower limb. Also, it has been shown that the use of deliberate hypotensive anaesthesia during spine surgery does not increase chances of POVL [7]. Huber and Grob [8] suggested that abnormal posture of the neck when the patient is being positioned prone for surgery could be a contributory factor for reducing perfusion in the vertebra-basilar area manifesting as stroke. This is purely hypothetical thought and cannot be definitively proved. It has been recommended to keep the neck at the level of heart or above in a neutral forward position at the time of surgery to avoid chances of hypoperfusion due to vertebra-basilar compression. The bilateral infarction as in the present case does not support the hypothesis. Review of POVL following general surgeries include many procedure carried out in the lower limb in prone position [7,16]. Irrespective of the types of surgeries, the prone position itself is a predisposing factor for POVL. Shen et al. [2] found some important finding on perioperative visual loss following spinal surgeries. Incidences were higher for age less than 18 or more than 65 years, male gender, anaemia, and posterior approach. Many cases occur in patients who have no identified preoperative risk factors, although hypertension, smoking, diabetes, and vascular disease appear to lead to increased risk. None of these risk factors was present in this patient. He had no cardio-pulmonary comorbidities and he had no fluctuation in hemodynamic status in the perioperative period as well. Considering these situations, other than the prone position we could not find any predisposing condition in our case.

Most of patients with cortical blindness have a partial vision loss. They often have other associated symptoms such

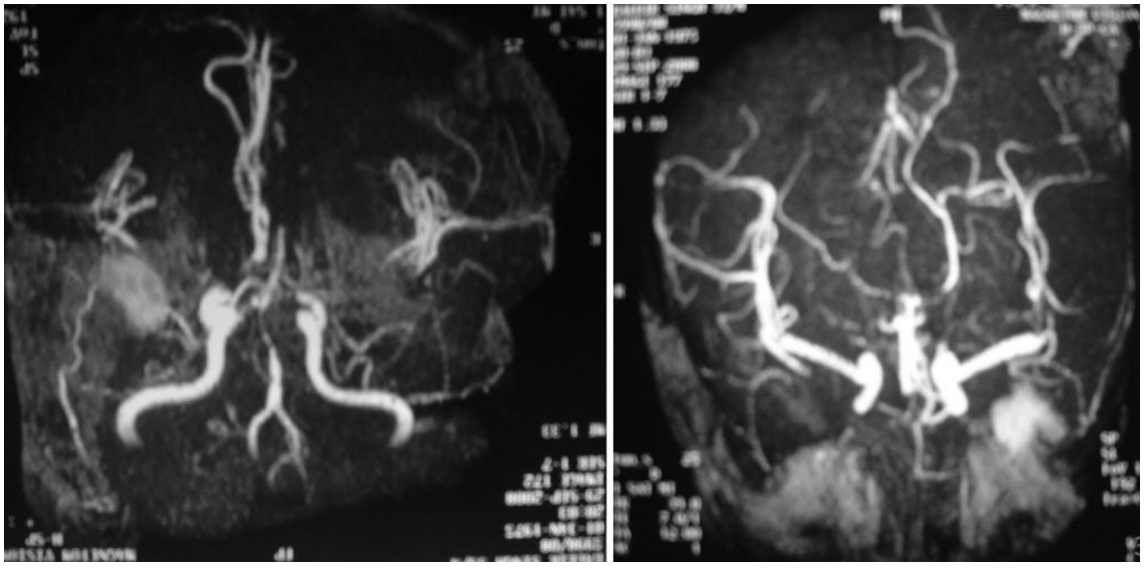

Fig. 3. Magnetic resonance angiography of brain reveals bilateral infarction of occipital lobe.

as cerebellar signs and other focal neurological deficits, depending upon the area of infarct. This patient had purely vision loss with no other deficit. There is no definite treatment for cortical blindness. No drugs including steroids have shown to reduce morbidity in these cases and most postoperative visual deficits do not show significant improvement with time. There is a hope of some improvement in initial months, but once the window time period has passed there is no hope of any further improvement.

This case report warns the spine surgeon about such fatal complication following spine surgery in prone position. Extreme cautions at every step should be taken to prevent the development of perioperative visual loss. Despite these precautions, some cases may still land up with visual loss and the patient should be explained about the grievous situation well before the surgical procedure.

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