

## Case Report

# Postoperative Visual Loss in Orthopedic Spine Surgery in the Prone Position: A Case Report

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*Postoperative visual loss is rare but a devastating postoperative complication. It is a multifactorial etiology. The practice advisory for perioperative visual loss associated with spine surgery reported by the American Society of Anesthesiologists task force on perioperative visual loss was developed from several case reports and case series. We reported a new case of postoperative visual loss diagnosed as ischemic optic neuropathy after undergoing a spine surgery in prone position. This case should be added to the overall incidence of postoperative visual loss. The possible risk factors were categorized in order to identify the POVL-susceptible patients. The pathophysiology of ischemic optic neuropathy was briefly reviewed.*

**Keywords:** Postoperative visual loss (POVL), Spine surgery, Prone position

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### Case Report

#### **Patient characteristic**

A 64-year-old male with spinal canal stenosis at L2-L5 level, underwent a decompressive laminectomy, posterior instrumentation, and fusion of L2-L5. During the previous year before the surgery, he suffered from low back pain and sciatica but had no symptoms of motor weakness. He had systemic hypertension, which was well controlled with manidipine hydrochloride and atenolol. He also had diabetes mellitus and dyslipidemia, controlled with metformin and atorvastatin. He had no history of stroke, transient ischemic attack (TIA), amaurosis fugax, and any visual problems prior to the surgery. A preoperative physical examination included his weight and height, which were 82.5 kg and 172 cm respectively (BMI = 27.9 kg/m<sup>2</sup>). The vital signs were recorded as follows: blood pressure (BP) = 130/73 mmHg, resting heart rate (HR) was around 70 beats per minute, and respiratory rate (RR) = 16 breaths per minute. The concerns for airway assessment were that the patient had relative short neck and limited neck movement. Preoperative laboratory investigations included hemoglobin 14.2 gm/dL, fasting blood sugar 104 mg/dL, HbA1C 6.1, and serum creatinine

1.0 mg/dL. Electrocardiogram and chest radiography were within normal limits.

After standard monitoring and preoxygenation was adequately performed, thiopental 350 mg, atracurium 30 mg, and fentanyl 50 mcg were administered intravenously in order to induce anesthesia and facilitate tracheal intubation. Then the patient was turned into a prone position over the Wilson frame with the patient's head turned to the right side and supported by a silicone head ring. Although the patient's head was not repositioned during the operation, direct pressure on both eyes was frequently checked. General anesthesia was maintained with sevoflurane 1 to 1.5% in nitrous oxide and oxygen mixture with fraction of inspired oxygen (FiO<sub>2</sub>) = 0.33. The systolic blood pressure (SBP) was maintained around 85 to 100 mmHg throughout the procedure. The patient received 2,750 ml of intravenous fluid during the operation. Total surgical time was four hours 35 minutes and total blood loss was about 350 ml. Blood sugar was monitored every two hours with no hypo- or hyperglycemic episode during the intraoperative period.

At the end of the operation, the patient fully awakened from general anesthesia without any complications except there was swelling of left eyelid and chemosis of the left eye. In addition, he could not open his left eye. On postoperative day 1, the patient complained that he was unable to see with his left eye with variation from a reduction of visual acuity to no perception of light. The complete eye examination

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revealed that the left pupil was 5 mm in diameter reacting to light with a positive relative afferent pupillary defect (RAPD). Intraocular pressure (IOP), measured by tonometer, of right and left eye was 18 and 80 mmHg, respectively. An MRI of the brain and orbits was performed as an emergency procedure to evaluate the optic nerve, as there was a suspicion of ischemic optic neuropathy (ION). The study revealed slightly increased T2-signal intensity of the retinal and orbital segment of the left optic nerve with high signal intensity on diffusion-weight imaging (DWI) and decreased apparent diffusion, indicating ischemic optic neuropathy. Extra-ocular muscles and periorbital soft tissue swelling was also observed from the examination.

### Discussion

Postoperative visual loss (POVL) indicates a loss of sight during the administration of general anesthesia. The visual loss can be unilateral or bilateral and severity ranges from counting fingers vision to no light perception (NLP). It is a rare but devastating perioperative complication. The reported incidence varies depending on the method of study, the duration of study, types of surgical procedure, and specific ophthalmic lesion (i.e., anterior ischemic optic neuropathy, posterior ischemic optic neuropathy, cortical blindness, or central retinal artery occlusion). The previous studies reported an incidence range from 0.0008% to 0.2%<sup>(1-3)</sup>.

There are no identifiable preoperative patient characteristics that can predict the development of POVL. Routine preoperative ophthalmic and neurovascular ophthalmic evaluations are unnecessary. Based on preceding case reports and advisory practice, the risk factors of POVL can be categorized into patient, surgical, and anesthetic factors.

Patient risk factors: include male gender, preoperative anemia, vascular disease (i.e., hypertension, peripheral vascular disease, coronary artery disease, diabetes), use of tobacco, and obesity<sup>(4-6)</sup>. Some ocular conditions, such as angle-closure glaucoma, marked optic disc edema due to any cause, status post-cataract extraction, and location of the watershed zone of the PCA in relation to the optic disc are considered as ocular risk factors for POVL<sup>(7)</sup>. Different age groups tend to be susceptible to distinct causes of POVL. The primary cause of POVL in children below 18 years is cortical blindness, whereas ischemic optic neuropathy (ION) is a major cause of POVL in patients over 50 years<sup>(8)</sup>.

Surgical risk factors: involve types of surgical procedure (spinal surgery, cardiac surgery, head-neck surgery, hip and femur operations), lengthy procedures (exceed 6 hours), prone position, significant blood loss, a combination of lengthy procedure and significant blood loss, and Wilson frame use<sup>(4,8-11)</sup>. Prolonged steep Trendelenburg positioning, especially in robotic surgery and laparoscopic gynecologic surgery may be considered as one of the surgical risk factors. Ocular venous congestion and subsequently increased intraocular pressure are considered as mechanisms of injury<sup>(12)</sup>. However, the incidence of POVL related to this position has been rarely reported<sup>(13)</sup>.

Anesthetic risk factors: encompass an inadequate venous drainage-surgical position and limited intravascular fluid administration<sup>(6)</sup>. Furthermore, intravascular fluid overload, particularly excessive crystalloid administration, may impede the ocular venous outflow tract and lead to ocular compartment syndrome<sup>(5)</sup>. The high-risk of POVL occurrence should be fully disclosed to the patient before surgery<sup>(14)</sup>.

The surgeons, especially in spinal surgery, occasionally request intraoperative controlled hypotension or deliberate hypotension in order to provide a bloodless surgical field and reducing the possible requirement of blood transfusion<sup>(15)</sup>. Sustained controlled hypotension in high-risk patients may exaggerate the possibility of POVL. Thus, the decision of intraoperative controlling hypotension should be considered on a case-by-case basis. Correspondingly, blood pressure and intravascular volume status should be monitored continually in these patients.

One of the recommendations for high-risk patients is a staged procedure, especially in a procedure that might last longer than 6 hours. The aims are to reduce the operation duration together with the amount of blood loss. The head position should be adjusted to ensure neutral alignment and level with or slightly higher than heart level. Direct pressure on the eye globe and the abdomen must be avoided. The patients' vision should be evaluated as soon as the effects of the anesthetic are over. In suspected cases, correction of anemia and optimization of hemodynamic and oxygenation are imperative. Subsequently, ophthalmic specialist consultation should be sought to define the actual cause of visual loss<sup>(16)</sup>.

Even though perioperative anemia, intraoperative substantial blood loss, hypotension, and increased orbital venous pressure play a tremendous role in the pathogenesis of perioperative visual loss,

the causal relationship cannot be established. There are no lower limits of hemoglobin/hematocrit to indicate the need of transfusion necessary to preclude the POVL. Individual variation in ocular vessel autoregulation and anatomy of the orbital blood supply determine the susceptibility to POVL.

The pathogenesis of POVL is continually monitored during the surgical procedure. Not all the patients who have these risk factors develop POVL. Therefore, the initiation of treatment strategies such as hemodynamic correction, blood transfusion, corticosteroid administration, anti-platelet therapy or measures to lower the IOP (i.e., release CSF) are not proven to be effective in improving visual acuity<sup>(17-21)</sup>. Preventive approaches seem more reasonable to circumvent the occurrence of POVL.

For this patient, the risk factors for POVL were male gender, history of vascular disease (hypertension and diabetes mellitus), spine surgery in the prone position, use of the Wilson frame and relatively excessive fluid administration. His BMI was 27.9 kg/m<sup>2</sup>, which is considered overweight. The systolic blood pressure during the operation was maintained at  $\geq 20\%$  reduction from baseline. He was prone with his head turned to the right side. Therefore, the most probable cause was the obstruction of the venous return from the left orbit, which led to the increased IOP. Prolonged intraoperative hypotension and inappropriate positioning were considered as the major pathogenic causes of blindness of this patient.

### ***Ischemic optic neuropathy (ION)***

Ischemic optic neuropathy (ION) is defined as an acute ischemic injury to the optic nerve. ION is the most common factor reported as a cause of POVL (89.2%)<sup>(11,16,22)</sup>. It can be classified into two types: anterior and posterior lesion, consistent with the anatomically different vascular supply to the optic nerve. The posterior ciliary artery (PCA) solitarily supplies the anterior portion of the optic nerve. The arterial supply to the posterior part of the optic nerve comes from multiple sources excluding the PCA. The pathology of ION is confined only to the optic nerve. Clinical presentations of AION and PION are similar, including painless partial or total visual loss. The presence of optic disk edema, attenuation of vessels and flame-shaped hemorrhage on fundoscopic examination are only seen in AION but not PION<sup>(13,23)</sup>.

According to the American Society of Anesthesiologists (ASA) Postoperative Visual Loss Registry, 83 out of 93 cases of ION-caused postoperative

visual loss, 67.4%, were diagnosed with PION, 22.8% were diagnosed with AION and the remainders were reported as unspecified ION<sup>(5)</sup>.

The ischemic injuries may result from perioperative hemodynamic derangement, decreased oxygen delivery (i.e., anemia, hypotension), inadequate venous drainage, increased intraocular pressure (IOP) stemming from the prolonged head-down position, embolic occlusion or direct ocular compression<sup>(8,11)</sup>.

### **Conclusion**

One of the major limitations of studying the cause and pathophysiology of POVL is small sample number and the incomplete evidence of case reports. Several attempts have been made to advance POVL research particularly by the American Society of Anesthesiologists (ASA) Postoperative Visual Loss Registry, by setting up the ASA practice advisory for POVL, a population-based study and even a multicenter case-control study. Utilizing the detail from various proceeding reports may help the anesthesiologist to avoid this catastrophic complication. The present review has provided the concise recommendations as well as all possible causes of POVL.

### **What is already known on this topic?**

Postoperative visual loss (POVL) is a rare adverse event with a scarce case report. To draw a recommendation or a preventive guideline is moreover challenging. Several aspects must be concerned including patient factors, anesthetic factors, and surgical factors.

### **What this study adds?**

We reported a new case of POVL whom eventually confirmed by an ophthalmologist of the ischemic optic neuropathy. The case will be added to the overall incidence of POVL. We reviewed some previous reports and summarized all possible risk factors into the three categories (the patient factors, the anesthetic factors, and the surgical factors). The readers can use these categorized risk factors to identify their POVL-susceptible patients. More surveillance and preventive strategies should be applied to these high-risk patients.

### **What is/are the implication(s) for public health practice?**

The new case of POVL should be reported to local health department, such as the Royal College of Anesthesiologists of Thailand. Likewise, this

should be reported to the American Society of Anesthesiologists (ASA) Perioperative Visual Loss Registry. Regarding a sporadic occurrence, the case information is necessary for further practice advisory revision.

#### Potential conflicts of interest

None.

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## รายงานผู้ป่วยที่เกิดการตาบอดถาวรภายหลังการผ่าตัดกระดูกสันหลังในท่านอนคว่ำ

ปฐมพร ปิ่นอ่อน, เศรษฐพงศ์ บุญศรี

การตาบอดภายหลังการเข้ารับการผ่าตัดเป็นภาวะแทรกซ้อนที่พบได้น้อยแต่มีความรุนแรงมาก อีกทั้งยังส่งผลกระทบต่อคุณภาพชีวิตของผู้ป่วยอย่างมาก เกิดขึ้นจากหลาย ๆ ปัจจัยเสี่ยงร่วมกัน ปัจจุบันมีเพียงข้อแนะนำเพื่อป้องกันการเกิดการตาบอดภายหลังการผ่าตัดกระดูกสันหลังในท่านอนคว่ำจากสมาคมวิสัญญีแพทย์แห่งสหรัฐอเมริกา โดยอาศัยข้อมูลจากการรายงานผู้ป่วยหลาย ๆ ราย การศึกษาฉบับนี้เป็นการรายงานผู้ป่วยรายใหม่ที่เกิดการตาบอดอย่างถาวรภายหลังการผ่าตัดกระดูกสันหลังในท่านอนคว่ำ โดยจะเพิ่มเข้าไปในตัวเลขอุบัติการณ์ของการเกิดตาบอดภายหลังการผ่าตัด นอกจากนี้ผู้พิมพ์ยังได้รวบรวมปัจจัยเสี่ยงต่าง ๆ ของการทำให้เกิดตาบอดภายหลังการผ่าตัด โดยแยกเป็นหัวข้อย่อย ทั้งนี้เพื่อให้ง่ายต่อการระบุผู้ป่วยที่น่าจะมีความเสี่ยงสูงต่อการเกิดตาบอดภายหลังการผ่าตัด และได้รวบรวมพยาธิกำเนิดของภาวะเส้นประสาทตาขาดเลือดซึ่งเป็นสาเหตุของการเกิดตาบอดภายหลังการผ่าตัดของผู้ป่วยรายนี้

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