

Posterior Ischemic Optic Neuropathy After Extensive Spine Surgery: A Case Report and Review of the Literature

Ben Levinson, DNP, CRNA

Sundara Reddy, MBBS, FRCA

Postoperative vision loss (POVL) after spine surgery is a rare but devastating complication. Because of its rarity (incidence < 0.2%), POVL might not be considered for inclusion in an informed consent by surgeons and anesthesia providers. We present a case of POVL due to posterior ischemic optic neuropathy following prone spine surgery. Posterior ischemic optic neuropathy is characterized by acute painless vision loss that is progressive and irreversible. Our case is atypical because the patient experienced moderate improvement of

visual acuity. Increased awareness and understanding of risk factors associated with POVL is an important and timely patient safety topic. In this report we review different pathophysiologies and risk factors for POVL following spine surgery along with recommendations for informed consent and strategies to reduce the incidence of POVL.

Keywords: Blindness after spine surgery, posterior ischemic optic neuropathy, postoperative vision loss.

Postoperative vision loss (POVL) has been reported for years, and it is important for anesthesia providers to understand the pathophysiology and risk factors for POVL. Surgeries most frequently associated with POVL include cardiac bypass, spine surgery, and radical neck dissection, although a wide range of other surgical procedures have also been implicated, such as total joint replacement.¹ Frequently in the literature, POVL is mentioned without differentiation between the specific causes of visual loss, creating confusion about the current evidence, risk factors, and recommendations.

Several pathologies may contribute to POVL; however, the most commonly referenced forms of POVL in the literature are corneal abrasions, central retinal artery occlusion (CRAO), and ischemic optic neuropathy (ION). Both CRAO and ION have been reported in prone spine surgery. Central retinal artery occlusion is a retinal ischemia that develops from occlusive events of the central retinal artery and is exacerbated by external pressure on the globe.^{2,3} Maintaining the eyes free of pressure is essential in preventing CRAO, and ocular pressure must be assessed frequently in prone cases by the anesthesia provider.⁴ A diagnosis of CRAO is made by the presence of a cherry-red spot and retinal opacity in the posterior pole on fundoscopic examination.^{5,6} The reported incidence of CRAO in prone spine cases is 0.0089% and has not changed significantly over the last several decades.⁷ Ischemic optic neuropathy is an optic nerve or optic disc ischemia. In contrast to CRAO, there is a decreasing trend in the incidence of ION over the same time period. In prone spine fusion cases, the incidence of ION is

0.006%.⁷ It is unclear why the incidence of ION has been decreasing, but it has been suggested that this trend could be related to changes in patient positioning devices, decreased surgical times, and increased provider awareness.⁸

There are 2 types of ION: anterior (AION), which is due to ischemia of the optic disc, and posterior (PION), which is due to ischemia of the retrobulbar optic nerve (Figure 1).⁹ The distinction can be made by fundoscopic findings and the clinical picture. In AION the optic disc is acutely edematous, whereas in PION the optic disc is acutely normal but becomes atrophic weeks to months after the event.⁶

We present a case of PION with an atypical vision loss pattern in the setting of unexplained delayed emergence from anesthesia after complex spine surgery. Our case is atypical because the patient's vision improved over time.

Case Summary

A 70-year-old, 80-kg man with a history of lower back pain arrived at a large academic medical center's emergency department with severe uncontrollable back pain. His medical history included diabetes mellitus type 2, smoking, hypertension, hyperlipidemia, and gastroesophageal reflux disease. Magnetic resonance imaging (MRI) revealed a suspicious, lytic lesion in the body of L4, combined with a pathologic fracture (Figure 2). Results of biopsy of the lesion indicated metastatic clear cell renal carcinoma. The orthopedic surgeon planned to undertake L4 corpectomy and laminectomy, combined with fusion from L2 to the pelvis. The patient initially underwent partial tumor embolization by an interventional radiologist the day before spine surgery, to reduce

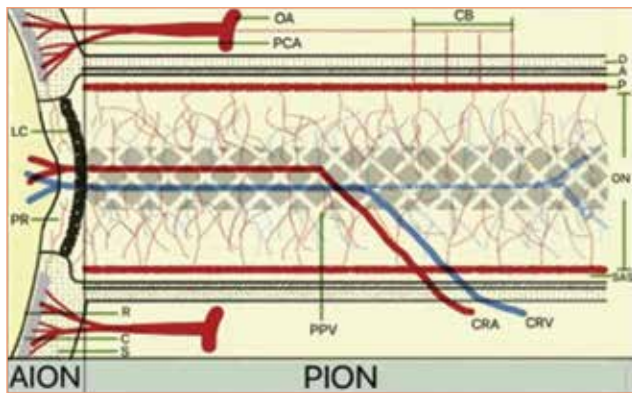


Figure 1. Diagram of Vascular Supply to Optic Nerve, With Anatomical Location of Ischemic Optic Neuropathy^a

Abbreviations: A, arachnoid; AION, anterior ischemic optic neuropathy; C, choroid; CB, collateral branches arising from ophthalmic artery (OA); CRA, central retinal artery with penetrating branches; CRV, central retinal vein; D, dura; LC, lamina cribrosa; ON, optic nerve; P, pia; PCA, posterior ciliary arteries; PION, posterior ischemic optic neuropathy; PPV, penetrating pial vessels arising from CB; PR, prelaminar region; R, retina; S, sclera; SAS, subarachnoid space.

^aDiamond-shaped area in center represents inner part of optic nerve, a watershed zone susceptible to ischemia.

(Reprinted with permission from Fandino, 2017.⁹ Original image from: Hayreh SS. Management of ischemic optic neuropathies. *Indian J Ophthalmol.* 2011;59(2):123-136.)

vascularity in an attempt to minimize surgical bleeding.

On the day of surgery, the patient was seen in the preoperative area, where risks and benefits of surgery were discussed, including the risk of POVL. Laboratory values were within normal limits other than hemoglobin (Hb) level of 11.8 g/dL, platelet count of $127 \times 10^9/L$, and glucose level of 210 mg/dL. Preoperative noninvasive blood pressure (NIBP) was 105/57 mm Hg, with an average reading of 135/63 mm Hg obtained from inpatient records. Preinduction vital signs showed a heart rate of 63/min, sinus rhythm, NIBP of 145/58 mm Hg (mean arterial pressure [MAP] of 83 mm Hg), and an oxygen saturation of 100%.

The patient was induced with propofol, 150 mg, fentanyl, 100 µg; lidocaine, 100 mg; and succinylcholine, 100 mg. A 7.5-mm cuffed endotracheal tube was inserted and placement confirmed. Postinduction vital signs showed a heart rate 64/min with normal sinus rhythm, NIBP of 85/51 mm Hg (MAP, 62 mm Hg), and oxygen saturation of 100%. The patient was placed on volume-controlled mechanical ventilation 8 breaths/minute, tidal volumes of 545 mL, and positive end-expiratory pressure of 3 cm H₂O. Anesthesia was maintained with sevoflurane at exhaled concentrations of 1.2% to 1.4%, which were briefly increased to 2.8% to facilitate head placement in Gardner-Wells tongs. A MAP of 50 mm Hg was recorded at this time and treated with ephedrine, 10 mg, followed by phenylephrine, 200 µg, to achieve normotension. A right-sided radial 20-gauge arterial line was



Figure 2. Sagittal Magnetic Resonance Image of Lumbar Spine Showing Tumor in Body of L4, With Arrow Highlighting Impingement of Cauda Equina

placed, as well as a 16-gauge peripheral intravenous line in the left arm. Additional medications given included dexamethasone (10 mg), tranexamic acid (bolus and infusion for a total dose of 1,520 mg), cefazolin (2,000 mg), and vancomycin (1,250-mg infusion).

The patient was repositioned with care to maintain neck neutrality and placed prone on a Jackson table (Orthopedic Systems Inc) with the head slightly elevated and suspended in 15 lb of traction. The arms were placed in the upright position, and all pressure points were padded appropriately. Somatosensory and motor evoked potential monitoring was conducted intraoperatively with sevoflurane maintenance of 0.6% to 0.7% exhaled concentrations, along with a propofol infusion titrated to 100 to 160 µg/kg/min and remifentanyl infusion titrated to 0.1 to 0.2 µg/kg/min. The patient also received midazolam, 5 mg, and ketamine, 100 mg, in incremental doses. Because of the patient's unusually strong respiratory drive, causing asynchronization with the ventilator on several occasions, end-tidal carbon dioxide values were maintained between 30 and 35 mm Hg for the duration of the case. A phenylephrine infusion was continued throughout the case with infusion rates of 0.2 to 0.7 µg/kg/min, which were increased up to 1.5 µg/kg/min during times of acute hemorrhage, to maintain the MAP within the surgeon's requested range of 65 to 75 mm Hg.

The surgery was complicated by episodic large blood loss during various stages of the corpectomy and tumor manipulation. At 3 points during the case, the patient became hypotensive, with a nadir MAP of 45 mm Hg during one of the events. In each event the MAP was



Figure 3. Intraoperative Record of Episodic Hemorrhages With Hypotensive Hemorrhage Events (arrows)^a

Abbreviations: ART BP, arterial blood pressure; CVP, central venous pressure; EKG, electrocardiogram; ETCO₂, end-tidal carbon dioxide; FIO₂, fraction of inspired oxygen; inject, injection; N, normal; NSR, normal sinus rhythm; Plasma-lyte A, multiple electrolytes injection (Plasma-Lyte A, Baxter International Inc); Ring..., Ringer's solution; SpO₂, oxygen saturation measured by pulse oximetry.

^aOn x-axis, each small box represents 5 minutes, and each large box represents 15 minutes. On y-axis, heart rate is in beats per minute. Noninvasive blood pressure (NIBP), mean arterial pressure (MAP), and invasive blood pressure are measured in millimeters of mercury. Central venous pressure (CVP) is measured in centimeters of water.

restored within 2 to 5 minutes (Figure 3) with a combination of volume replacement and bolus doses of vasoactive drugs (ephedrine and phenylephrine). There were no changes noted in somatosensory and motor evoked potential monitoring during the episodes of hypotension. Over a 9-hour procedure, the patient received a total of 5.3 L of crystalloid, 500 mL of 5% albumin, 4 units of packed red blood cells, and 200 mg of calcium chloride. Total estimated blood loss (EBL) for the case was 2.3 L. Several blood samples were drawn throughout the case, with the lowest recorded intraoperative Hb value being 10.3 g/dL. (The morning after surgery, Hb level was 9.9 g/dL.)

During closure, the propofol infusion was decreased and eventually discontinued, along with the sevoflurane, remifentanyl, and phenylephrine infusions. On return of spontaneous respirations, 0.5 mg of hydromorphone was administered. Care was taken to maintain neutrality of the neck and extremities while positioning the patient supine. Pressure support ventilation was discontinued, and the patient continued to breathe spontaneously, 350-mL to 500-mL tidal volumes, at a rate of 21 to 29/min.

Despite the strong respiratory effort, the patient was deeply obtunded and had no response to aggressive sternal rub. Blood glucose level was 302 mg/dL. A single dose of flumazenil (0.4 mg) was administered, with no improvement. Arterial blood gas analysis showed a metabolic

acidosis with partial respiratory compensation, pH 7.31, PCO₂ of 33 mm Hg, PO₂ of 394 mm Hg, base excess of -10 mEq/L, and bicarbonate (HCO₃) value of 17 mEq/L.

With the propofol infusion discontinued for more than 2 hours and the patient showing no signs of increased responsiveness, the protocol of acute cerebrovascular accident was initiated. The patient was seen by the neurology team and underwent computed tomography and MRI, with results showing no evidence of stroke. An electroencephalogram showed no abnormalities suggesting seizures. Over the course of the night in the intensive care unit, the patient became arousable and followed commands. He was extubated the morning after surgery.

Following extubation the patient was oriented and without cognitive impairments but had reduced strength and sensory deficits on the left side. At this time, the patient reported a painless dim blurry vision, as if "someone turned the lights off in the room." An ophthalmologist was consulted. Visual field testing showed loss of vision predominantly in the central zones bilaterally. Reported visual acuity was 20/160 in both eyes, and pupils were not reactive. Ocular pressures were in the normal range. Slit-lamp findings were unremarkable except for known laser scars from previous diabetic retinopathy treatment, and the optic discs appeared normal. Posterior ION was suspected given the clinical setting and largely unremarkable

results of the examination. According to ophthalmologic recommendations, vasopressor agents were avoided and volume resuscitation was solely used to manage hypotension. Over the next several days the patient reported some improvement in vision.

At a 7-week follow-up, the patient had visual acuities of 20/60 –2 in the right eye, and 20/70 –1 in the left eye. The ophthalmologist believed that some of the patient's vision loss in the left eye was likely secondary to previous panretinal photocoagulation surgery. Fundoscopic findings at 7 weeks' follow-up revealed new atrophy of the optic discs, confirming PION.

Discussion

Although a rare complication, PION is typically the ION associated with prone spine surgery.¹⁰⁻¹⁴ Posterior ION in the postoperative period presents as a sudden painless loss of visual acuity, and it may continue to degenerate but typically does not improve.² Treatments such as high-dose corticosteroids, antiplatelet agents, reduction of intraocular pressure (IOP), and reduction of cerebrospinal fluid pressures, have all proved ineffective.³ A case report *has* attributed partial and complete visual recovery secondary to optimization of hemodynamic parameters and anemia.¹⁵ However, due to the lack of adequate study size and randomized controlled trials, little is known about the prevention and management of perioperative PION, as well as other forms of POVL.

Unfortunately, numerous publications discussing ION do not distinguish between AION and PION. Since 2012, two large multicenter studies have emerged, revealing the following factors as significantly and independently associated with ION after spinal fusion surgery: obesity, male sex, Wilson frame use, longer anesthetic duration, greater EBL, aging, and decreased percent colloid to crystalloid administration (Table).^{7,16} In addition, many publications suggest other risk factors in the development of ION during spine surgery; however, they are largely based on opinion, case reports, and pathophysiologic assumptions. Two small observational studies frequently cited in the literature give weak support to other risk factors, which include preoperative anemia, hypertension, diabetes, peripheral vascular disease, coronary artery disease, and tobacco use.^{13,17} In addition, the use of high-dose α -adrenergic agonists, hypotension, and large volumes of crystalloid (which may or may not be equivalent to decreased percent colloid) have been implicated in the development of ION, but there is currently insufficient evidence to validate these claims.⁴ Nevertheless, because the optic nerve's blood flow is dependent on perfusion pressure, it makes physiologic sense to optimize many of the proposed risk factors that relate to organ perfusion, despite the lack of confirmatory evidence. Because adequate blood pressure is essential to organ perfusion, hypotension is likely deleterious,

Definitive risk factors	Associated risk factors
Obesity	Preoperative anemia
Male gender	Hypertension
Aging (24% increased incidence risk ratio per 10 y)	Diabetes
Wilson frame use	Tobacco use
Prolonged surgery (> 6.5 h)	Peripheral vascular disease
Large blood loss (> 40% EBV)	Coronary artery disease
Increased crystalloid proportion of total fluids administered	

Table. Risk Factors for Ischemic Optic Neuropathy During Prone Spine Surgery

Abbreviation: EBV, estimated blood volume.

although the ideal MAP will vary from person to person. Regarding the implication of large crystalloid volumes, a lower percentage of colloid administration is associated with ION during spine fusion, leading to recommendations for a balanced colloid to crystalloid fluid replacement during substantial blood loss.^{4,16}

It is important to understand the different perfusion effects on AION vs PION. Whereas AION involves circulation in the globe, PION involves circulation that is retrobulbar. To prevent both AION and PION, theoretically this would involve the variables increasing oxygen delivery, which include optimizing Hb levels, blood volume, oxygen saturation, MAP, and cardiac output. In addition, in the case of AION it is theoretically important to minimize IOP because perfusion pressure to the optic disc equals MAP minus IOP.^{1-3,18} Therefore, pressure on the globe could affect perfusion to the optic disc (AION) by increasing IOP but would not affect perfusion to the retrobulbar optic nerve (PION). Interestingly, although pressure on the globe can cause various eye injuries and POVL secondary to CRAO, there is currently *no* evidence that pressure on the globe causes either form of ION in spine surgery, despite the possible mechanism of increasing IOP.^{4,13,19-33} This lack of evidence may result from the fact that the ION associated with spine surgery is primarily PION, and because the development of PION is retrobulbar in nature, changes in IOP would not affect perfusion to the retrobulbar optic nerve.^{1,3,10,13}

In contrast to IOP, venous congestion likely does not play a role in the development of AION, as the venous outflow system does not significantly affect blood flow to the optic disc.¹⁸ Unlike AION, however, venous congestion leading to increased venous pressure may affect perfusion in PION, because of its anatomically different retrobulbar pathology.^{1,3,10,13} It has been theorized that the mechanism relating Wilson frame use to ION is due to increased abdominal compression, and a tendency for the patient's head to be in a more dependent position, causing increased venous congestion.^{8,16}

Conclusion

Improving the outcome for patients undergoing prone spine surgery has been limited with regard to ION. The low incidence and lack of scientific evidence has made recommendations difficult to make, with only 7 variables being identified by large studies as significantly and independently associated with ION after spinal fusion: obesity, male sex, Wilson frame use, longer anesthetic duration, greater EBL, aging, and decreased percent colloid administration.^{7,16} Because many of these variables cannot be controlled, anesthesia providers have an obligation to understand the current data regarding ION and to identify patients at increased risk of ION. An expert panel survey, composed of mainly anesthesia professionals, by the Anesthesia Patient Safety Foundation discovered that 86.6% of respondents believed that “most surgeons do not recognize the risk of ION in the susceptible patient population, whereas 52.2% felt the same was true for anesthesia professionals”.³⁴ The use of a spine team consisting of anesthesia providers who regularly do difficult prone spine cases may be useful to help organizations identify what defines a “complex” spine case and to create guidelines for providers. Institutional identification of a “complex” spine surgery would likely be determined by a higher risk of hemodynamic instability, EBL, or anesthetic duration. When possible, interventions such as vascular embolization, tranexamic acid, large-bore intravenous access, and staging procedures, may be useful to limit the elevated risks for complications in “complex” spine cases.^{35,36} Proper positioning to decrease venous congestion such as patient head elevation above the level of the heart, decreased abdominal compression, and avoidance of Wilson frames, may also limit the risks of developing ION.^{8,16} Theoretically, other interventions that decrease venous congestion may also be helpful.

Due to the devastating effects of POVl and the unfamiliarity of most patients with this complication, extensive discussion becomes mandatory when patients have multiple risk factors for ION. During the informed consent process, anesthesia professionals and surgeons should include discussion of the remote risk of visual impairment ranging from partial vision loss to complete blindness in both eyes for patients considered to be at risk of POVl from ION.

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AUTHORS

Ben Levinson, DNP, CRNA, is an anesthesia provider at Winneshiek Medical Center, Decorah, Iowa. Email: IowaLevinson@gmail.com.

Sundara Reddy, MBBS, FRCA, is in the Department of Anesthesiology at the University of Iowa Hospitals and Clinics, in Iowa City, Iowa.

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The authors have declared no financial relationships with any commercial entity related to the content of this article. The authors did not discuss off-label use within the article.

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