

General

Perioperative Visual Loss After Spine Surgery: A Case-Based Review of Posterior Ischemic Optic Neuropathy

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Perioperative visual loss (POVL) is an uncommon but life-altering complication that has been reported most often after prolonged spine procedures performed in the prone position. Among the recognized causes, posterior ischemic optic neuropathy (PION) accounts for the majority of cases in this setting and is believed to result from impaired perfusion of the retrobulbar optic nerve. Clinical and registry data consistently associate PION with intraoperative anemia, sustained or relative hypotension, substantial blood loss, large-volume fluid administration with hemodilution, and preexisting vascular disease that limits autoregulatory reserve. Advanced age and systemic atherosclerotic burden further increase susceptibility by reducing the optic nerve's tolerance to ischemic stress.

This case-based educational review describes an elderly patient with significant systemic diseases, including vasculopathy, who developed acute, painless visual loss after lumbar spinal surgery. Using this clinical scenario, we examine the interplay between patient-specific risk factors and intraoperative physiology, review the underlying vascular anatomy and pathophysiology of PION, and outline an approach to early recognition and differential diagnosis of postoperative visual deficits. Management principles are discussed with an emphasis on hemodynamic optimization, prompt specialist consultation, and realistic counseling regarding prognosis. The goal of this structured discussion is to provide anesthesiology trainees and perioperative clinicians with a practical framework to recognize high-risk situations, implement preventive strategies, and respond appropriately should this rare but catastrophic complication occur.

TEACHING CASE

An 80-year-old man with a history of hypertension, peripheral arterial disease, coronary artery disease with prior stenting, chronic kidney disease stage III, and hyperlipidemia presented for elective posterior lumbar decompression and fusion at L2-L5 for severe spinal stenosis and neurogenic claudication.

Preoperative hemoglobin was 12.1 g/dL. Blood pressure was well controlled on beta-blockers and ACE inhibitors. He denied any baseline neurological or visual disturbances.

General anesthesia was induced uneventfully. The patient was positioned prone on a Jackson table with the head in a foam headrest and eyes checked to be free of external pressure. Tranexamic acid 10 mg/kg intravenous bolus administered prior to incision, followed by 1 mg/kg/hr infusion throughout the procedure. The procedure lasted 9.5 hours. Estimated blood loss was 1.8 liters. He received 3 liters of crystalloid, 1 liter of 5% albumin, and 2 units of packed red blood cells. Permissive hypotension was utilized

with mean arterial pressure (MAP) ranging between 55-65 mmHg for prolonged periods to reduce surgical bleeding.

He was extubated at the end of the case and transferred to PACU hemodynamically stable.

On postoperative day 1, the patient reported profound painless vision loss in both eyes, describing only light perception. Pupils were sluggishly reactive, and there was no ocular pain. Fundoscopic examination showed normal optic discs without edema. The anesthesiologist was called bedside and an urgent ophthalmology consultation was obtained.

TEACHING QUESTIONS

QUESTION 1

Which diagnosis is most consistent with painless bilateral visual loss and a normal early fundoscopic exam after prolonged spine surgery?

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- A. Central retinal artery occlusion
- B. Cortical blindness
- C. Posterior ischemic optic neuropathy
- D. Acute angle-closure glaucoma

QUESTION 2

Which intraoperative factor is most strongly associated with posterior ischemic optic neuropathy?

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- A. Short procedure duration
- B. Hypertension throughout surgery
- C. Prolonged anemia and hypotension
- D. Use of volatile anesthetics

QUESTION 3

Why is the optic disc often normal in the early stages of posterior ischemic optic neuropathy?

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- A. The retina is unaffected
- B. The injury occurs posterior to the optic nerve head
- C. The condition is functional rather than ischemic
- D. Fundoscopy is not sensitive

QUESTION 4

Which patients are at greatest baseline risk for perioperative ischemic optic neuropathy?

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- A. Young patients without comorbidities
- B. Patients with glaucoma only
- C. Elderly patients with vascular disease and anemia
- D. Patients undergoing short supine procedures

QUESTION 5

What is the most important immediate management step once perioperative visual loss is suspected?

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- A. Start high-dose steroids
- B. Emergent return to the operating room
- C. Urgent ophthalmology consultation and hemodynamic optimization
- D. Ocular massage

ANSWERS AND DISCUSSION

QUESTION 1

Correct Answer: C - Posterior ischemic optic neuropathy

Posterior ischemic optic neuropathy (PION) is the most common cause of perioperative visual loss after prolonged spine surgery in the prone position. It classically presents as acute, painless visual loss, often bilateral, noted upon emergence from anesthesia (typically occurs within 24-48 hours after surgery). A defining feature of PION is a normal early fundoscopic examination, because the ischemic insult

occurs in the retrobulbar portion of the optic nerve, posterior to the lamina cribrosa.^{1,2} This absence of early optic disc edema can be misleading and may falsely reassure clinicians, delaying recognition of the injury; optic atrophy often does not become apparent until weeks after the initial insult.^{2,3}

Similar to PION, anterior ischemic optic neuropathy (AION) presents with sudden painless vision loss upon emergence from anesthesia. AION is more often associated with cardiac surgery, whereas PION is commonly found following spinal surgery.⁴ The defining difference between PION and AION is that AION is characterized by acute optic disc edema and paleness on fundoscopic examination due to acute involvement of the optic nerve head. PION will eventually develop optic disc pallor by 4-6 weeks after onset.⁴

Central retinal artery occlusion typically occurs in spine surgery in the setting of external compression of the eye, leading to elevated intraocular pressure.³ It is characteristically defined by retinal whitening and a classic cherry-red spot on fundoscopic examination.³

Cortical blindness arises from occipital lobe injury and presents with a normal fundoscopic exam and preserved pupillary light reflexes despite loss of vision.

Acute angle closure glaucoma (AACG) presents as sudden onset severe ocular pain, often associated with headache, blurred vision, nausea and vomiting.³ On exam, AACG is associated with a mid-dilated pupil that is sluggishly reactive and elevated intraocular pressure up to 60 mmHg.³

QUESTION 2

Correct Answer: C - Prolonged anemia and hypotension

The leading mechanism of PION is impaired optic nerve perfusion resulting in ischemia. The posterior optic nerve is supplied by small pial vessels with limited autoregulatory reserve, making it susceptible to reductions in oxygen delivery.^{2,5}

Several intraoperative factors are thought to contribute to the development of posterior ischemic optic neuropathy, including sustained hypotension, significant blood loss, anemia with hemodilution, prolonged surgical duration, large-volume crystalloid administration, and prone positioning that increases venous pressure to the head and lead to impaired optic nerve perfusion. Case-control analyses have demonstrated strong associations between greater estimated blood loss, longer operative times, lower postoperative hemoglobin levels, and the occurrence of PION following spine surgery.^{6,7} Findings from the American Society of Anesthesiologists postoperative visual loss registry further support hypotension and anemia as major potentially modifiable contributors to this complication.⁶

QUESTION 3

Correct Answer: B - The injury occurs posterior to the optic nerve head

Unlike AION, PION affects the retrobulbar optic nerve, which is not visible behind the optic disc. Because the optic disc and retinal circulation are initially spared, fundoscopy appears normal in the early postoperative period.^{1,2} Over time, typically after 4 – 6 weeks, optic atrophy and pallor will become evident.⁴ Vascular studies have demonstrated that posterior optic nerve perfusion depends on a fragile microvascular network vulnerable to systemic hypoperfusion.⁸ MRI may show diffusion restriction of the optic nerve in some cases, but imaging is not always diagnostic early.⁹

QUESTION 4

Correct Answer: C - Elderly patients with vascular disease and anemia

Advanced age and systemic vasculopathy impair vascular autoregulation and reduce the optic nerve's tolerance to ischemia. Conditions associated with increased risk include hypertension, diabetes mellitus, hypercholesterolemia, peripheral arterial disease, coronary artery disease, chronic kidney disease, and anemia. Large retrospective studies of spine surgery patients have shown that older individuals with vascular comorbidities are disproportionately represented among cases of posterior ischemic optic neuropathy.^{7,10} Registry analyses have also identified obesity and male sex as additional associated risk factors.^{6,10} In this patient, extensive underlying vasculopathy likely reduced baseline optic nerve perfusion reserve, increasing susceptibility to the combined effects of intraoperative hypotension and anemia.

PION is associated with prolonged operative time and prone positioning. PION is most commonly associated with advanced age, not young age. A nationwide study that looked at perioperative visual loss in spine fusion surgery

from 1998 to 2012 in the United States found that the incidence risk ratio for ischemic optic neuropathy increased by 24% per decade of life.¹¹ Glaucoma is not a risk factor for PION.

QUESTION 5

Correct Answer: C - Urgent ophthalmology consultation and hemodynamic optimization

There is no proven therapy to reverse established posterior ischemic optic neuropathy, and high-dose corticosteroids have not demonstrated consistent benefit in ischemic optic neuropathy.^{8,12} Accordingly, immediate management priorities focus on urgent ophthalmologic evaluation, optimization of mean arterial pressure, correction of anemia to improve oxygen delivery, avoidance of further hypotension, and assessment for compressive etiologies such as external ocular pressure or orbital compartment syndrome. The American Society of Anesthesiologists 2012 and updated 2019 Practice Advisory emphasizes that prevention, rather than rescue therapy, remains the most effective strategy in managing this complication.^{6,13} Recommended preventive approaches include avoiding prolonged deliberate hypotension in high-risk patients, maintaining adequate hemoglobin concentration and systemic perfusion, minimizing excessive crystalloid administration that may contribute to tissue edema, and counseling high-risk patients preoperatively about the rare but serious risk of perioperative visual loss. Despite these measures, visual recovery is frequently incomplete, and many patients experience permanent visual deficits.^{6,10}

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