



Posterior Reversible Encephalopathy Syndrome Causing Transient Postoperative Blindness Following Spine Surgery

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Postoperative vision loss (POVL) is devastating not only for the patient but also for the anesthesiologist. Posterior reversible encephalopathy syndrome (PRES) is an infrequent and treatable cause of POVL, which is reported predominantly in rapid hemodynamic perturbations, endothelial dysfunction, and massive volume resuscitation.^{1,2} To our knowledge, there are no reported cases of PRES following acute hypertension for a brief duration of 3 minutes and massive transfusion.

A 55-year-old female with a body mass index (BMI) of 21.82 kg/m², belonging to the American Society of Anesthesiologists (ASA) physical status class I, underwent posterior spine instrumentation (T8–L4) for correction of kyphoscoliosis. She was positioned prone for 9 hours under general anesthesia on a conventional orthopedic operating table, with soft chest and pelvic supports. Her head was positioned neutrally on a doughnut, and eye compression was circumvented by periodic confirmation of adequate periorbital space. Motor and somatosensory-evoked potential recorded no abnormal signals. Anesthesia was maintained with propofol infusion (75–100 mcg/kg/min), fentanyl (0.5–1 mcg/kg/h), and isoflurane (end-tidal < 0.5%) to allow optimal neurophysiological and entropy (target 40–60) monitoring. Normothermia was maintained. Ventilation was targeted to an end-tidal carbon-dioxide of 30 to 40 mm Hg and oxygen saturation > 94%. Intraoperatively, there was a drop in hematocrit to 25% from a baseline of 30%. Active resuscitation with crystalloids and blood products was initiated immediately. During resuscitation, there was a transient fall in arterial blood pressure (BP) to 70/40 mm Hg for 5 minutes. Noradrenaline infusion was initiated at 0.02 to 0.04 mcg/kg/min. Subsequently, there was an episode of acute rise of systolic BP to 190 mm Hg, presumably due to an inadvertent bolus delivery of noradrenaline

from the infusion pump. Noradrenaline was stopped immediately and propofol bolus was administered. BP normalized to 120/80 mm Hg in 3 minutes, and remained within normal range thereafter. Five packs of red blood cells (1250 mL), plasma (1000 mL), and 4000 mL of crystalloids were administered to cope with the total blood loss of 2000 mL and urine output of 2000 mL. The hematocrit was raised to 33% in the next hour. Due to prolonged prone positioning, postoperative mechanical ventilation was planned, anticipating airway edema. Two hours after shifting the patient to the postoperative unit, two episodes of generalized seizures occurred and were treated with phenytoin (1 g intravenous [IV] followed by 100 mg IV tid) and levetiracetam (500 mg IV bd). She had a persistent staring look. Blood sugar and electrolytes were normal. CT brain ruled out cerebral hemorrhage and edema, which were suspected due to the acute intraoperative hypertension and prolonged prone position.³ On the first postoperative day, there were no seizures, and extubation of trachea was done. Postextubation, she complained of painless vision loss bilaterally. Ophthalmic examination revealed bilateral loss of light perception and impaired optokinetic nystagmus, denoting a cortical pathology.⁴ Fundus and light reflex were normal, indicating an intact pathway till midbrain.⁵ Other causes of POVL like ischemic optic neuropathy (ION) and central retinal artery occlusion (CRAO) were ruled out by the absence of relative afferent pupillary defect and normal findings in fundus. The constellation of symptoms of seizures, altered sensorium, blindness and the association with acute intraoperative hypertension, and volume resuscitation directed toward the suspicion of PRES.^{1,2} MRI of the brain (►Fig. 1A,B) depicted typical bilateral symmetrical T2 hyperintensities in the watershed zones of

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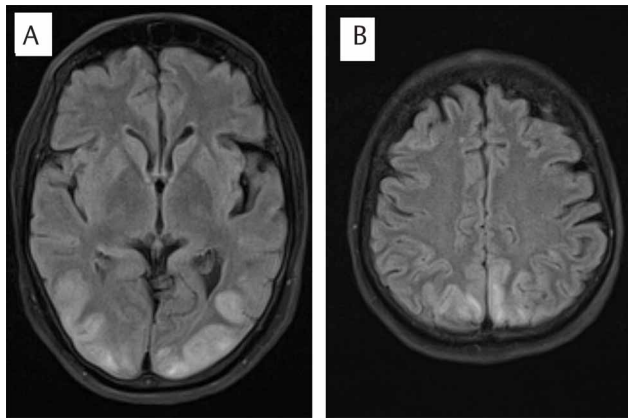


Fig. 1 (A, B) MRI brain–fluid attenuation inversion recovery (FLAIR) sequence in axial plane, showing hyperintensities in bilateral parietal and occipital lobes suggestive of posterior reversible encephalopathy syndrome (PRES).

occipital and parietal lobes, confirming PRES. Mannitol (20 g IV tid), head-end elevation, and antiepileptics were continued. On the third postoperative day, the vision recovered completely, and she was discharged a week later.

PRES is a vasogenic edema reported in cases of hypertensive emergencies, eclampsia, renal failure, cytotoxic chemotherapy, immunosuppressants, transplant recipients, and massive transfusion.^{1,2} Gopalakrishnan et al have reported PRES following sustained elevation in blood pressure.⁶ Singh et al reported PRES due to a sudden increase in blood viscosity following rapid blood transfusion in cases of uterine fibroid with menorrhagia.² The endotheliopathy precipitated by acute hemorrhagic shock could lead to a disruption in the blood-brain barrier (BBB). Although the exact pathophysiology remains unclear, disruption of the BBB and fluid extravasation due to acute hypertension exceeding the cerebral autoregulation threshold or endothelial dysfunction remains the most accepted mechanism.^{1,2,7} In our case, although the episode of hypertension was transient, the association with endotheliopathy from rapid volume resuscitation could have been the precipitating cause of PRES. The typical neuro-radiological findings in MRI with bilateral and symmetrical

parieto-occipital involvement were consistent with the diagnosis of PRES. POVL following spine surgery may result from CRAO, central retinal vein occlusion (CRVO), ION, cortical blindness, PRES, acute glaucoma, or corneal abrasion. Most of the causes except ION are potentially reversible if diagnosed and treated early. Having a low threshold of suspicion is cardinal for expeditious treatment of the reversible causes and prevention of permanent or secondary neurological injury.

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Conflict of Interest

None declared.

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