Perioperative Bioccipital Watershed Strokes in Bilateral Fetal Posterior Cerebral Arteries During Spinal Surgery

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Key words
- Cerebral watershed infarction
- Circle of Willis
- Intraoperative hypotension
- Perioperative stroke
- Posterior cerebral artery
- Spinal surgery
- Vision loss

Abbreviations and Acronyms
BP: Blood pressure
CBF: Cerebral blood flow
ECG: Electrocardiogram
fPCA: Fetal posterior cerebral artery
ICA: Internal carotid artery
PCA: Posterior cerebral artery

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Citation: World Neurosurg. (2016) 85:367.e17-367.e21. http://dx.doi.org/10.1016/j.wneu.2015.09.098

Journal homepage: www.WORLDNEUROSURGERY.org

Available online: www.sciencedirect.com

BACKGROUND: **Vision loss due to cerebral infarction during spinal surgery is less described. Intraoperative hypotension would be a leading cause. Patients with variation of the circle of Willis could be more prone to present stroke in this context, but reports are lacking to sustain the theory. Bilateral occipital watershed ischemic strokes have never been described before. We report the case of a patient with a fetal origin of both posterior cerebral arteries (PCAs), presenting this particular anatomic stroke following lumbar laminectomy surgery for spinal stenosis during which intraoperative hypotension was observed. We discuss how this common anomaly associated with intraoperative hypotension could have promoted this serious complication.**

CASE DESCRIPTION: **A 55-year-old man woke up with cortical blindness after he had undergone lumbar surgery during which a marked decrease in blood pressure had occurred. Magnetic resonance imaging revealed bilateral symmetric infarctions of the occipital lobes in the distal territory of both PCAs and smaller anterior watershed ischemic strokes, suggesting a hemodynamic mechanism. Extended investigations, including conventional angiography, failed to find any cause of stroke but revealed bilateral fetal PCAs supplied by internal carotid arteries only. Two years later, the patient has not recovered and remains severely visually impaired.**

CONCLUSIONS: **The standing hypothesis would be posterior low-flow infarctions resulting from intraoperative hypotension on a variation of the circle of Willis more prone to decrease in cerebral blood flow. Moreover, this case supports the hypothesis of vascular insufficiency due to intraoperative hypotension as cause of stroke during spinal surgery.**

INTRODUCTION

Perioperative stroke is a rare complication of general surgery with an estimated incidence between 0.02% and 1%.[1-3] Intraoperative hypotension is considered to be at least a promoting factor, but mechanisms affecting cerebral circulation remain unclear.[4,5] More, it has been suggested that congenital variation of the circle of Willis could potentiate the risk of watershed strokes during intraoperative hypotension.[4,6] Patients undergoing spinal surgery are particularly exposed to intraoperative hypotension with a risk of vision loss due to ophthalmic and cortical infarctions. Watershed ischemic strokes in the posterior cerebral artery (PCA) territories have never been described before. It would support the hypothesis of cerebral hemodynamic insufficiency by decreased cerebral blood flow (CBF) as a mechanism of ischemia.[7] We report a case of a patient with bilateral fetal PCAs (fPCAs) who presented this particular anatomic infarction in the course of spinal surgery during which intraoperative hypotension was applied.

CASE

The patient gave written consent for the description of the case. The case was reported to the pharmacovigilance center.

A 55-year-old man, without any specific medical history or under any treatment (165 cm, 65 kg, body mass index of 23.9 kg/m^2), was admitted for scheduled lumbar laminectomy surgery for spinal stenosis. Blood pressure (BP) before surgery was 176/114 mm Hg. After premedication with 100 mg hydroxyzine orally, anesthesia was induced with inspired sevoflurane 5%, sufentanil 75µg, and propofol 120 mg. It was maintained with inspired sevoflurane 1% to 3% with an additional dose of 75 µg sufentanyl. After induction, the patient was placed in the genupectoral position for surgery. Electrocardiogram (ECG), blood oxygen saturation, capnia, and temperature were monitored throughout the anesthesia, as well as BP with a noninvasive cuff on the right arm. Systolic BP was between 61 and 145 mm Hg with the exception of a 12-minute period at 48 mm Hg. It was lowered to <80 mm Hg for 75 minutes to prevent bleeding. Diastolic BP averaged 53 mm Hg (Figure 1). Total anesthesia time was 95 minutes. No blood transfusion was required. Once ventilation

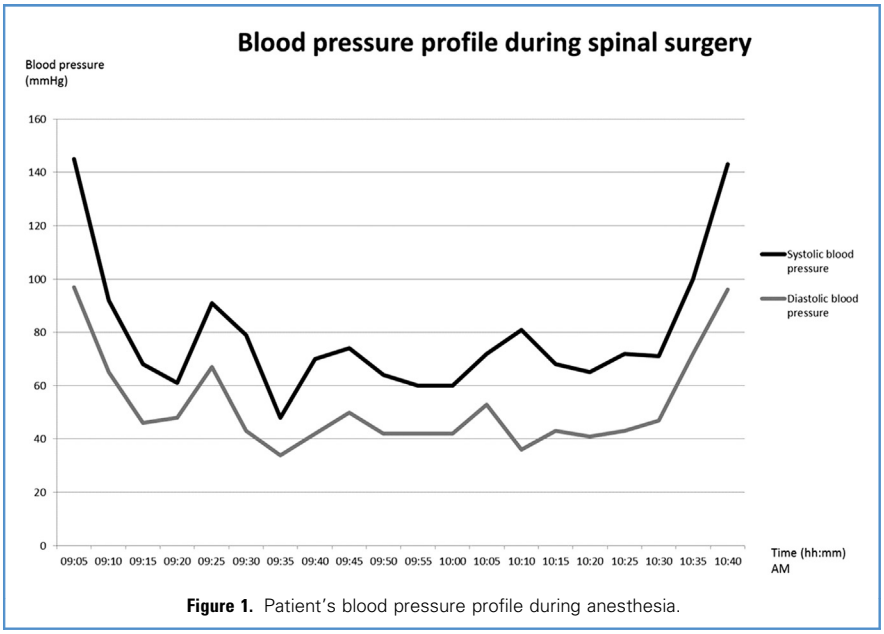

Figure 1. Patient's blood pressure profile during anesthesia.

resumed spontaneously, the patient's trachea was extubated and the patient was taken to the recovery area without any reported incident or cardiac arrhythmia. When he woke up, he complained of complete blindness. Ocular appearance was normal at funduscopic examination with complete pupillary responsiveness. No confusion or memory disturbance was noticed. Brain magnetic resonance imaging revealed extended symmetric areas of recent ischemia in both occipital lobes on diffusion-weighted sequences (Figure 2A) and a typical aspect of bilateral anterior watershed strokes at the junction of the anterior and middle cerebral arteries (Figure 2B). Cerebral infarctions were already visible as hyperintensities on fluid attenuated inversion recovery sequences. Time-of-flight magnetic resonance angiography did not show any vascular occlusion but revealed a bilateral fPCA with hypoplasia of proximal segments P1 (Figure 3A and B). Blood tests showed a hemoglobin level of 14 g/dL, platelet count of 200 G/L, C-reactive protein level of 9 mg/L, low-density lipoprotein cholesterol level of 3.16 mmol/L, and preprandial blood glucose level of 6 mmol/L. Coagulation tests were normal. ECG, 48-hour cardiac monitoring, transesophageal echocardiography, and examination of cervical and cerebral arteries by ultrasound were normal. BP monitoring in the stroke unit did not reveal arterial hypertension. Faced with this unusual aspect of ischemia in both PCA territories, a conventional angiography was performed. It showed normal arterial caliber but confirmed the variation of the circle of Willis with both PCAs originating from the carotids through posterior communicating arteries, hypoplastic P1 segment, and distal part of the basilar artery (Figure 3C and D). Unfortunately, the patient's clinical status did not significantly improve during hospitalization. The patient was discharged with paramedical assistance and a daily dose of aspirin. Severe visual impairment and left spatial neglect remain after 2 years.

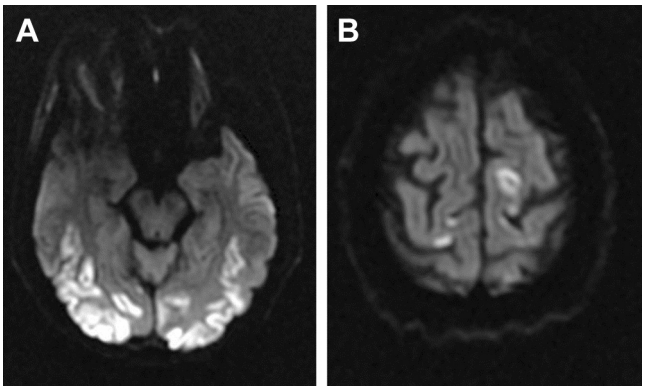

Figure 2. Axial diffusion weighted cerebral resonance magnetic imaging showing recent ischemia in both distal territories of cerebral posterior arteries (**A**) and recent bilateral anterior watershed ischemic strokes (**B**).

DISCUSSION

Vision loss is a complication encountered in 0.2% of spinal surgeries.[8,9] Most cases result from ischemia of the optic nerve or the retina promoted by anemia, intraoperative hypotension, and elevated intraocular pressure due to prone position during a long time.[10] Cortical blindness and cerebral infarction at large are less described.[11] Our case suggests intraoperative hypotension would be a leading cause of stroke in this context. Intraoperative hypotension is widely observed during major spinal surgery due to disruption of extensive vascular networks found in bones.[12] More, it can be applied by physicians to decrease blood loss and avoid transfusion.[13,14] The POISE trial showed that an episode of hypotension at any time during a patient's surgical procedure is related to a higher risk of perioperative ischemic stroke.[5] However, while vascular insufficiency resulting from decreased CBF would seem to be the most logical explanation, no solid evidence supports this theory. There is a discrepancy between the high frequency of intraoperative hypotension and the rare reports of stroke occurring during general surgery, particularly during the intraoperative phase, suggesting that another mechanism is involved. Cerebral