Suspected Propofol-Related Infusion Syndrome After Lumbar Spinal Fusion With Total Intravenous Anesthesia: A Case Report

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Funding The authors received no financial support for the research, authorship, and publication of this article.

Conflict of Interest The authors report no conflicts of interest.

Informed Consent The patient was informed that the data concerning his case would be submitted for publication, and he provided verbal consent.

ABSTRACT

Propofol-related infusion syndrome (PRIS) is a well-documented yet rare complication of prolonged infusions of propofol. It is characterized by a myriad of metabolic abnormalities, including cardiac arrhythmias, rhabdomyolysis, acute kidney injury, metabolic acidosis, and other disturbances. First described in children receiving extended propofol infusions to maintain sedation while in the intensive care unit, PRIS has now been described in every age group. It typically results in death. Management of this potentially devastating complication involves supportive treatment of the metabolic problems encountered and discontinuing the use of propofol. We describe a patient with suspected PRIS who underwent a two-stage lumbar spine procedure with total intravenous anesthesia, using propofol as the anesthetic. At 6-weeks postoperatively, he could walk without assistive devices and did not require pain medication. Findings of the current case may help inform healthcare providers of the possibility of PRIS after spinal fusion, allowing for a potentially life-saving diagnosis.

Keywords: Propofol-Related Infusion Syndrome, Lumbar Vertebrae, Spinal Fusion, Intravenous Anesthesia

INTRODUCTION

Propofol is a common medication used for its sedative properties in the intensive care unit and as an anesthetic agent in the operating room. It is typically well tolerated by patients and considered relatively safe. Propofol-related infusion syndrome (PRIS) is a well-documented complication of the use of propofol. The condition was initially described in children but has been observed in all age groups.[1,2] Symptoms typically include cardiac arrhythmias, metabolic acidosis, acute kidney injury, rhabdomyolysis, and metabolic disturbances.[3]

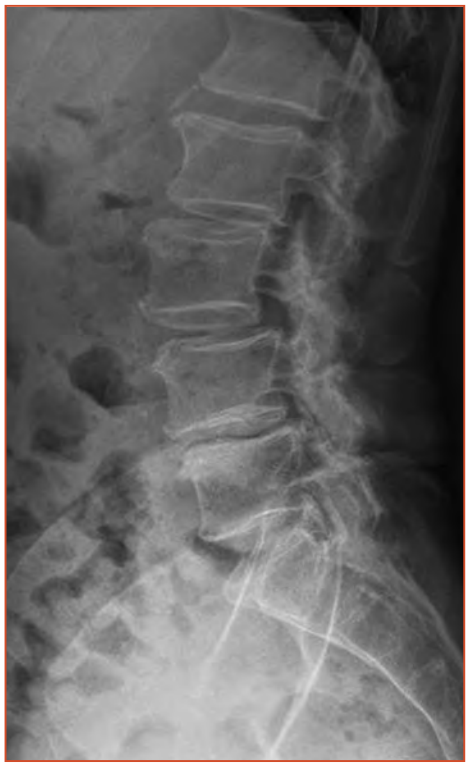
Patients with this life-threatening condition are typically critically ill in intensive care units and receive prolonged infusions for more than 48 hours for sedation.[4] Most patients described in studies died as a result. PRIS has rarely been noted in patients with short-term infusions as is typical with most surgical procedures.

To our knowledge, PRIS has not been described in patients who underwent spinal fusion using total intravenous anesthesia. Surgical treatment of spinal deformity in adults is complex owing to combined anterior and posterior approaches for circumferential correction and fusion. It is associated with considerable complications and can be performed in a single day or staged fashion. Operating times range from 6 to 12 or more hours, with a common estimated blood loss of more than 2 L.[5] Propofol is often used as the anesthetic because of it allows intraoperative neurophysiological monitoring. We present a patient who developed PRIS after lumbar spine decompression and fusion for treating lumbar degenerative scoliosis.

CASE REPORT

A 60-year-old man presented with severe degenerative scoliosis of the lumbar spine with stenosis, neurogenic claudication, and radiculopathy (Figures 1 and 2). Nonoperative treatment was unsuccessful. Operative correction such as lateral interbody fusion with posterior spinal fusion was discussed with the patient. His medical history included alcohol abuse, tobacco abuse, hyperlipidemia, and chronic pain. He had no previous complications with surgical procedures but did report “difficulty waking up” after a remote procedure several years before, of which he said he did not remember the details. There was no family history of difficulties with anesthesia.

The patient was taken to the operating room and underwent anterior decompression and interbody fusion



Fgure 1. Preoperative lateral view of the lumbar spine, showing severe multilevel facet arthropathy and degenerative disc disease with anterolisthesis of levels L3-L4 and L4- L5.

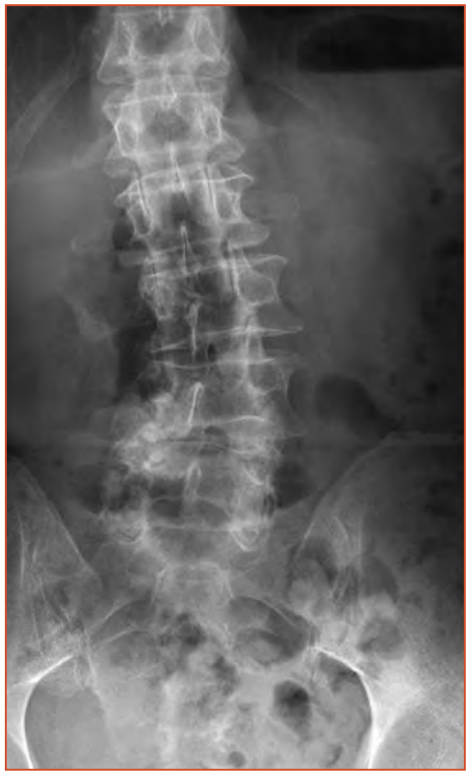


Figure 2. Preoperative anteroposterior view of the lumbar spine, showing severe multilevel facet arthropathy, degenerative disc disease, and degenerative levoconvex scoliosis.

using an extreme lateral technique at the levels L2-L3, L3-L4, and L4-L5.[6] While under the same anesthetic, he was repositioned and underwent posterior decompression and instrumented fusion of level L2-L5. No intraoperative complications were noted, with successful correction of the complex degenerative scoliosis (Figures 3 and 4).

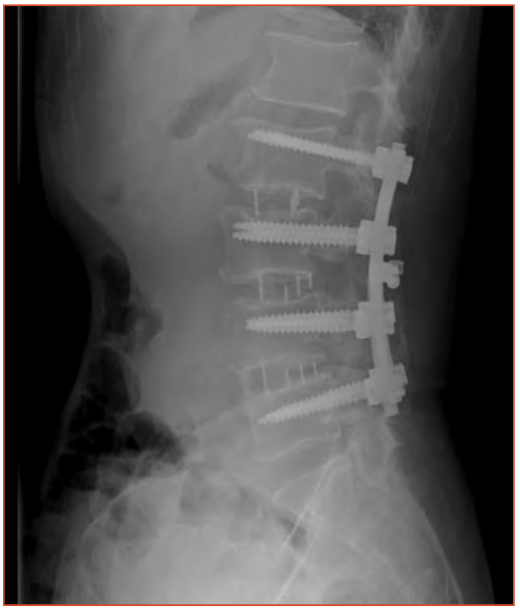


Figure 3. Lateral view of the lumbar spine, showing interbody fusions at levels L2-L3, L3-L4, and L4-L5 with posterior instrumentation at level L2-L5.

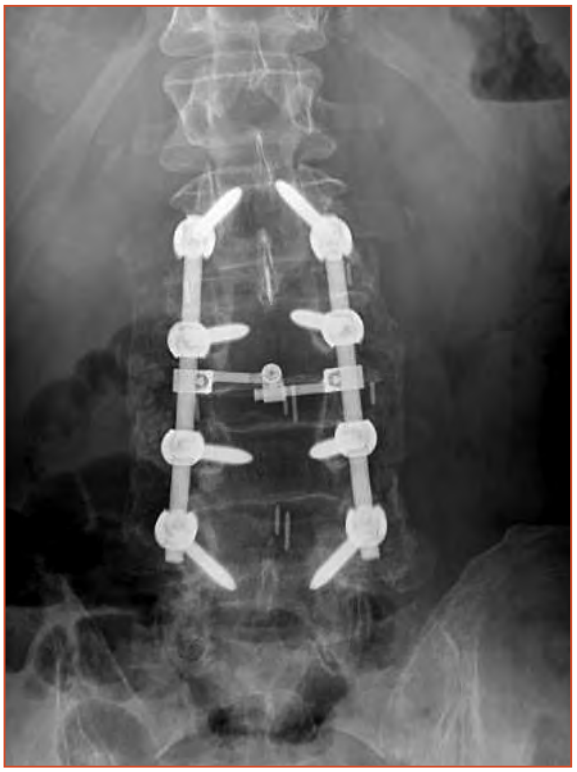


Figure 4. Anteroposterior view of the lumbar spine, showing interbody fusions at levels L2-L3, L3-L4, and L4-L5, with posterior instrumentation at level L2-L5.

Before incision, tranexamic acid was administered to minimize blood loss.[7] The patient was in the operating room for about 10 hours and 48 minutes, with a total anesthesia time of 9 hours and 30 minutes and a mean propofol infusion rate of 6.8 mg/kg per hour. During the second stage of the procedure, an arterial blood gas was drawn that showed a mild metabolic acidosis to a pH of 7.31. At the completion of the procedure, the patient was awoken from anesthesia-induced sleep and extubated without observed complication. Estimated blood loss throughout the surgical procedure was 850 mL, with urine output measured at 550 mL. A total of 5000 mL of intravenous crystalloid was administered, and no blood products were given. Postoperatively, hematocrit volume was 0.3 (30%), down from a preoperative measurement of 0.4 (40%).

In the immediate postoperative period, the patient could move extremities on command but had decreased mental activity and was severely agitated, requiring restraints for safety. A cardiac arrhythmia comprising tachycardia with premature atrial complexes and ST segment changes were detected on continuous cardiac monitoring, and he was subsequently transferred to the intensive care unit. Results of initial workup revealed an acute kidney injury with a 0.36 increase in creatine levels, severe rhabdomyolysis, metabolic acidosis, and elevated troponins. No obvious cause of the sudden symptoms was identified. He was treated with close monitoring and aggressive resuscitation. The patient's creatine kinase level peaked at 312.3 µkat/L (18,693 U/L), and the troponin I level elevated to 0.938 µg/L. During the next 5 days, the patient's metabolic abnormalities resolved, his kidney function returned to baseline levels, and his arrhythmia resolved without intervention. His mental activity improved considerably, and he was discharged to an inpatient rehabilitation facility.

At his 6-week postoperative visit, the patient walked without assistive devices and did not require any pain medications. The previous symptoms caused by neurogenic claudication and radiculopathy were resolved. His mental activity was at its baseline level, with no reported residual effects of his prolonged hospital stay. He did report "very little recollection" of the several days after the operation.

DISCUSSION

PRIS is a rare but potentially life-threatening condition associated with the use of a common and relatively safe medication. It has been described mostly in patients undergoing prolonged sedation for treating critical illness such as respiratory failure or traumatic brain injuries.[2] Bray[8] described diagnostic criteria to include sudden bradycardia progressing to asystole in addition to one of either metabolic acidosis, derangement of liver function, lipemic plasma, or rhabdomyolysis. The definition now includes hypotension, acute kidney injury, hyperkalemia, and hypoxia.[4] PRIS may be dose dependent, with patients most at risk who receive dosages higher than 4 mg/kg per hour for a prolonged duration.[9] Treatment typically involves cessation of propofol and then treatment of the sequelae.[9] PRIS often results in patient death. Many authors have tried to identify risk factors or develop screening tests to prevent PRIS.[10,11]

To our knowledge, PRIS has not been documented to occur after spinal fusion. PRIS is typically a diagnosis of exclusion because many of the signs and symptoms often result from much more common pathological features. In our patient, there was concern he was under-resuscitated or suffering from medication withdrawal. Under-resuscitation was unlikely, however, because of standard levels of blood loss, adequate urine output, and only minor abnormalities found on intraoperative blood gas. Medication withdrawal was unlikely owing to near immediate onset of symptoms postoperatively and the failure to identify any offending agents initially during review of medical records, interviews with the patient's friends and family, and discussion with the patient when the symptoms resolved.

Our patient exhibited typical findings of PRIS. At our institution, degenerative scoliosis procedures with multiple stages are typically performed using one anesthetic agent. Our experience has been similar to that of others, in which this single prolonged procedure is safe. That is, major complications are rare, with similar outcomes to staged procedures performed during several days. These procedures are also usually performed with total intravenous anesthetic. A review of our patient's medical history did not indicate the risk of developing PRIS.

Although PRIS is rare, surgeons performing prolonged procedures with the use of continuous infusion of propofol should be aware of the condition. It can be difficult to diagnose, and delay in doing so may result in death of patients.

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