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Anesthesiology

Propofol Infusion Syndrome in A Child Undergoing Thoracoabdominal Scoliosis Surgery: A Case Report

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Abstract Case Report

Propofol-Related Infusion Syndrome (PRIS) is a rare but potentially fatal complication, particularly in children and adolescents exposed to continuous propofol infusion. It typically manifests as cardiac failure, metabolic acidosis, or rhabdomyolysis. We report the case of a 13-year-old girl undergoing thoracolumbar scoliosis surgery who developed an early presentation suggestive of PRIS immediately after prolonged propofol anesthesia. Early discontinuation of the drug and multidisciplinary management led to a rapid improvement. This case highlights the need for close postoperative monitoring, consistent with observations previously reported in the literature [1, 4].

Keywords: Propofol infusion syndrome; pediatric anesthesia.

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Introduction

Propofol is a commonly used intravenous anesthetic agent valued for its rapid onset and favorable recovery profile. However, prolonged infusion may lead to Propofol-Related Infusion Syndrome (PRIS), a rare but potentially fatal condition characterized by cardiac instability, metabolic disturbances, and rhabdomyolysis. Children and adolescents appear particularly vulnerable due to their increased metabolic demands and susceptibility to mitochondrial dysfunction.

We report the case of a 13-year-old girl undergoing thoracoabdominal scoliosis surgery who developed early signs suggestive of PRIS in the immediate postoperative period, highlighting the importance of early recognition and prompt management in pediatric anesthesia.

CASE REPORT

We describe the case of a 13-year-old girl with Turner syndrome admitted for surgical correction of thoracolumbar scoliosis. The procedure was performed in the dorsal decubitus position. Anesthetic induction included 100 mg of propofol, 150 micrograms of fentanyl, and 20 mg of neuromuscular blocker, allowing

easy intubation. Maintenance anesthesia relied on total intravenous anesthesia with propofol throughout the procedure, combined with a lidocaine infusion. The sixhour surgery was marked by an estimated blood loss of 1500 mL, infusion of 2 liters of crystalloids, and a transfusion protocol including five units of packed red blood cells, two units of fresh frozen plasma, two units of platelets, and two grams of tranexamic acid. At the end of the procedure, the patient exhibited hemodynamic instability requiring the initiation of norepinephrine.

Upon admission to the ICU, the patient presented with persistent circulatory instability and green-colored urine, observed within the first hours and consistent with massive elimination of propofol, as previously described. Postoperative laboratory findings showed a lactate level below 2 mmol/L, platelets at 149,000/mm³, prothrombin time of 46%, INR of 1.76, CPK of 994 U/L, LDH of 271 U/L, normal transaminases, creatinine of 3 mg/L, and urea of 0.17 g/L. Immediate discontinuation of propofol, correction of hypovolemia, and multimodal analgesia resulted in clinical improvement progressive normalization of metabolic parameters. The overall presentation strongly suggested an early form of propofol infusion syndrome.



Figure 1: Green discoloration of urine in the collection bag

DISCUSSION

Propofol infusion syndrome was first described in children by Bray in 1998 [1] and remains a rare but particularly feared complication in anesthesia and critical care. Its pathophysiology is dominated by mitochondrial dysfunction, characterized by inhibition of the respiratory chain and impaired fatty acid oxidation, as reported by Kam and Cardone in 2007 [4]. This leads to insufficient energy production, intracellular lactate accumulation, and multiorgan cellular failure, especially affecting cardiac and skeletal muscle. The heart, highly dependent on fatty acids during stress, becomes unable to meet its energy demand, explaining the occurrence of cardiovascular collapse and refractory bradycardia in severe cases.

The pediatric population is especially vulnerable. Wolf et al. demonstrated in 2001 that children exhibit increased sensitivity to metabolic inhibitors due to higher energy requirements, lower lipid reserves, and limited metabolic adaptability [2]. Several risk factors have been identified, including prolonged or high-dose propofol infusion, major surgery, hypovolemia, malnutrition, severe infection, hypothermia, and particularly the concomitant use of catecholamines or corticosteroids [3, 4]. The patient in our report presented multiple risk factors: prolonged spinal surgery, significant blood loss, continuous exposure to propofol, major metabolic stress, and postoperative norepinephrine use.

Typical clinical manifestations include metabolic lactic acidosis, rhabdomyolysis, acute cardiac failure, and occasionally acute renal failure. In our case, although lactate levels remained below 2 mmol/L, the elevated CPK, green urine discoloration, hemodynamic instability, and rapid improvement after discontinuation of propofol were all consistent with an early-stage PRIS. Observations by Fudickar in 2010 indicated that rapid normalization following propofol withdrawal is a key diagnostic clue in early forms [5]. Furthermore, Hemphill in 2019 emphasized that PRIS can be suspected even in the absence of severe acidosis when clinical context and risk factors are strongly suggestive [6].

Optimal management relies on immediate discontinuation of propofol, substitution with an alternative hypnotic agent, correction of metabolic disturbances, optimization of intravascular volume, and circulatory support. Monitoring should include blood gas analysis, lactate levels, CPK, liver and kidney function tests, and cardiac evaluation. The favorable outcome observed in this case is consistent with published early forms of PRIS that were identified and treated promptly, underscoring the need for increased vigilance in the postoperative period, especially in pediatric scoliosis surgery.

CONCLUSION

Propofol infusion syndrome should be considered in any child or adolescent receiving continuous propofol perfusion who develops metabolic or hemodynamic abnormalities. Early recognition and immediate discontinuation of propofol are crucial for improving prognosis. This case illustrates that even early forms of PRIS can be reversible when promptly identified.

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