



A Stitch in Time Saved Nine: Early Recognition of Propofol Infusion Syndrome under Anesthesia

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Abstract

Keywords

- propofol infusion syndrome
- hyperkalemia
- acidosis

Though propofol infusion syndrome results from a larger dose of infusion over a longer period of time, we observed its development even when it was infused for a shorter period of time. In our patient, it was heralded by progressive acidosis and hyperkalemia. Discontinuation of propofol and simultaneous treatment of hyperkalemia averted any harm to the patient.

Introduction

Propofol is widely used as an anesthetic and sedative agent because of its favorable pharmacodynamics. Though a safe agent, deaths were reported in children who received infusion of propofol at a dose greater than 4 mg/kg/h for more than 48 hours.¹ Subsequently, similar incidents following propofol infusion were reported in adults too.² In 1998, Bray coined the term “propofol infusion syndrome” (PRIS) with various clinical features of progressive acidosis, rhabdomyolysis, hyperkalemia, cardiac asystole and cardiac failure, and death associated with propofol infusion.³ It was presumed to result from infusion of a large dose of propofol for a long duration. However, recently many cases of PRIS with favorable outcome have been reported following average to lower-dose infusion for shorter duration of time.⁴ In many instances, instead of complete picture of syndrome, only a subset of the classic syndrome features, chiefly hyperkalemia and lactic acidosis, were observed. Even a single induction dose of propofol culminating in hyperkalemia and cardiac arrest has been reported.⁵ We present a case report of an otherwise healthy adult who developed progressive hyperkalemia and acidosis from propofol infusion of 5 to 6 mg/kg/h, over a period of 10 hours only. However, timely recognition and intervention averted catastrophe.

Case Report

A 55-year-old, 85-kg, healthy man, underwent craniotomy for decompression of petroclival meningioma. Anesthesia was

induced with propofol and fentanyl, and tracheal intubation was facilitated with rocuronium. Continuous arterial pressure, in addition to routine monitoring, was instituted. Anesthesia was maintained with 50% oxygen in air, sevoflurane, infusions of propofol (5–6 mg/kg/h), rocuronium, and fentanyl. His baseline arterial blood gas (ABG) picture showed potassium 3.7 mEq/L, base excess 2.1, and lactate 0.8 mEq/L. Subsequent ABG analyses over the next 8 hours revealed gradual increases in potassium to 4.9 mEq/L, base excess to –8.1 and lactate to 2.8 mEq/L. Two hours after the last ABG, peaked T waves (without any other electrocardiographic [ECG] changes) were noticed on the monitor. ABG analysis at this stage (i.e., 10 hours after the baseline ABG) showed potassium of 6.2 mEq/L, with base excess of –8.8 and lactate of 4.3 mEq/L. Propofol infusion was discontinued, and hyperkalemia was managed with calcium gluconate, sodium bicarbonate, and glucose insulin infusion. Subsequent anesthesia was maintained with sevoflurane. Over the next 10 minutes, T wave reverted to normal shape and potassium decreased to 4.9 mEq/L with base excess of –11.3 and lactate of 4.3 mEq/L. The entire procedure lasted for nearly 13 hours. Estimated blood loss was 1200 mL, and the patient received 600 mL of blood, 1 L colloid, and 5.3 L of crystalloids. In view of prolonged surgical procedure, the patient was ventilated overnight under fentanyl and midazolam infusion. Next morning with his normal ABG picture, he was disconnected from ventilator and trachea was extubated. Further course in the hospital was uneventful.

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Discussion

Currently, PRIS is assumed to be related to disruption of fatty acid oxidation caused by impaired entry of long-chain acylcarnitine ester into the mitochondria and failure of the mitochondrial respiratory chain.⁶ Isolated hyperkalemia and acidosis, components of PRIS have been reported from even smaller or average doses of propofol infused over a shorter period of time.⁷ Exact mechanism of isolated hyperkalemia from propofol administration is unclear. In the body, the main store of potassium are the muscles and liver. PRIS usually leads to rhabdomyolysis, with secondary hyperkalemia. However, hyperkalemia has been reported without rhabdomyolysis.⁸ It is hypothesized that mitochondrial fatty acid oxidation disorder could disturb potassium homeostasis because of its relationship with mitochondrial metabolism.⁹ Meanwhile propofol could produce lactic acidosis through the impairment of free fatty acid utilization and mitochondrial activity in the muscles and heart.¹⁰ The patient was hemodynamically stable without evidence of hypovolemia or any pre-existing infection, thereby ruling out other possible causes of lactic acidosis. Hyperkalemia and acidosis during propofol infusion are harbingers of impending catastrophe, and failure to recognize these early warning signs and discontinuing its infusion would lead to florid PRIS with dangerous consequences.

In the light of the clinical circumstances, blood chemistry and recovery of deranged parameters following discontinuation of propofol infusion, it can be assumed that the patient may have developed PRIS. In this context, the role of serial ABGs and careful ECG monitoring cannot be overemphasized.

Conflict of Interest

None declared.

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Case Report

An Infant with Apert Syndrome and Tetralogy of Fallot for Craniosynostosis Correction: Anesthetic Challenges

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