Propofol Infusion Syndrome: A Life Threatening Condition Blunted by Continuous Renal Replacement Therapy

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Introduction
Propofol is a common intensive care unit anesthetic which was approved by FDA in 1989. It possesses sedative, anxiolytic, and anticonvulsant properties. Case reports of Propofol related infusion syndrome (PRIS) started in early 1990s. Reported signs and symptoms included acute refractory bradycardia, severe metabolic acidosis, cardiovascular collapse, lactic acidosis, rhabdomyolysis, hyperlipidemia, renal failure, and hepatomegaly. It is associated with both high dose cumulative and short-term infusions. This case report highlights the development of PRIS in a patient admitted to the ICU, symptoms of which were assumed to be blunted because of continuous renal replacement therapy support.

Case Report
A 35-year-old man with bipolar disorder was brought to the emergency room for altered mental status, agitation was heavily sedated and intubated for airway protection. Propofol was started after intubation. Initial bloodwork showed leukocytosis, acute kidney injury (AKI) and lactic acidosis. He was started on empirical antibiotics. Urine toxicology screen was positive for cocaine and benzodiazepines. He subsequently developed septic shock refractory to IV fluids and was started on norepinephrine infusion. CRRT was initiated for oliguric AKI due to rhabdomyolysis and ischemic acute tubular necrosis. His condition subsequently improved and he was weaned from pressor support and CRRT on day 9. Creatine kinase (CK) levels had improved from 30000 U/l on admission to 7035 U/l. Propofol infusion was maintained at 80mcg/kg/min. On day 10, he developed unexplained anion gap metabolic acidosis and hyperkalemia. Triglyceride levels were 1864 mg/dl (baseline 159 mg/dl). He was also noted to have rising transaminases. Given these manifestations, PRIS was suspected. Propofol infusion was stopped. Over the next few days, he was noted to have improvement in acidosis, liver enzymes and triglyceride levels [1-3].

Discussion
PRIS is a rare but potentially fatal complication seen in critically ill patients. Management of overt PRIS includes immediate discontinuation of propofol infusion and problem-driven management, including hemodialysis, hemodynamic support, and extracorporeal membrane oxygenation in refractory cases. Given the high mortality of PRIS, the best management is prevention. Although bradycardia, lactic acidosis and cardiovascular collapse are part of the classic presentation of PRIS, our patient lacked these features possibly because he was undergoing CRRT which can be a treatment for PRIS. Only when the CRRT was interrupted on high doses of propofol did our patient manifest potential early manifestations of PRIS. Clinicians should consider alternative sedation agents in patients who are receiving prolonged or high dose propofol infusions.

References

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