Propofol Infusion Syndrome Associated with Steroid and Catecholamine Administration in a Patient with Cervical Neck Trauma

Background
- Propofol infusion syndrome is a well documented entity that can involve acute refractory bradycardia, metabolic acidosis, rhabdomyolysis, lipemic plasma, or a clinically enlarged liver or fatty liver
- Cases have been described where short durations of propofol infusions at relatively high doses induced propofol infusion syndrome.
- Administration of exogenous steroids and/or catecholamines may increase the risk of developing propofol infusion syndrome.1

Propofol Dosing and Lactate Trend

Case Description
- 19 year-old patient (61 kg) status post cervical neck trauma from a motor vehicle accident (MVA).
- History significant for scleroderma-induced cardiomyopathy status post orthotopic heart transplant in 2008.
- Following the MVA, he was intubated in the emergency room at an outside hospital and started on a propofol infusion for sedation.
- Boluses totaling 9 grams of IV methylprednisolone were administered 3 hours after MVA and an infusion was started at 5.35 mg/kg/hr.
- The patient was transferred to our institution 13 hours later on propofol 100 mcg/kg/min and phenylephrine 8 mcg/min to maintain a MAP of 90 mmHg.
- He was promptly brought to the operating room for anterior cervical spinal decompression.
- In order to facilitate somatosensory evoked potential monitoring (SSEP), anesthesia was maintained with propofol at 150 mcg/kg/min and remifentanil in combination with sevoflurane.
- After initial assessment of SSEPs it was determined that he had no signals below the level of C5, and SSEP monitoring was not indicated. Sevoflurane was increased and propofol decreased to 50 mcg/kg/min. Phenylephrine was increased to 50 mcg/min to maintain a goal MAP of 90 mmHg.
- The case was complicated by a metabolic acidosis that increased from a baseline pH 7.44 and lactic acid 0.8 mmol/L to a peak of pH 7.31 and lactic acid 12.3 mmol/L. There was no evidence of trauma or crush injury, and electrolytes were normal.
- During this time, there was approximately 1.7 liters (L) of blood loss with concomitant administration of 3 L of LR and 2 units of packed red blood cells. After transfusion the hematocrit was 31% and urine output was 3.74 ml/kg/hr.
- Hemodynamic instability increased requiring epinephrine 0.05 mcg/kg/min.
- Propofol was discontinued at this point, but lactic acidosis continued to worsen. Pressor support escalated requiring norepinephrine and vasopressin. Propofol infusion was stopped at approximately 5 hours into the case due to suspicion of propofol infusion syndrome.
- Several hours after discontinuation of propofol, lactate and pressor requirements started to decrease.
- Lactate was back to baseline within 6 hours of stopping propofol infusion.

Discussion
- The rapid onset of lactic acidosis without any other clear etiology and its resolution with discontinuing propofol leads us to believe our patient developed propofol infusion syndrome.
- A previous study has suggested risk factors include propofol infusion duration greater than 48 hours and dose over 5mg/kg/hr. Our case had a total infusion time of 15.5 hours and a mean dose of 3.3 mg/kg/hr.
- Our case highlights the pitfalls of using propofol for ICU sedation and the possible increased risk of patients on high dose steroids and catecholamines in the pediatric population.

<table>
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<th>Number of hours</th>
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<th>mg/kg/hr</th>
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References