

CLINICAL VIGNETTE

Metabolic Acidosis Associated with Propofol Use

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Propofol has been increasingly used as a sedative/anesthetic agent in endoscopic procedures. Most physicians prefer to use it in patients difficult to sedate or in prolonged therapeutic procedures. Patients often request it for its deep sedation. Most of the side effects of propofol are well-known, such as burning pain at the injection site, hypotension, hypertension, apnea, and hyperlipidemia.¹

The following case report represents an unusual side effect of propofol.

Case Report

A 62-year-old female presented to the emergency room with weakness and dizziness. She had not been eating well for several days. She had a history of hypertension and hypothyroidism. She consumed alcohol on a daily basis but had never been diagnosed with alcoholic liver disease. On presentation, she was found to be profoundly hypotensive with a blood pressure of 69/45, HR 61, T 97.4°F, and WT 52 kg. Physical exam was remarkable for a thin female with a protuberant abdomen without any abdominal tenderness. Her rectal exam revealed hemoccult negative stool.

She was admitted to the intensive care unit for sepsis syndrome. She was started on norepinephrine drip and on IV antibiotics. Paracentesis was negative for spontaneous bacteria peritonitis (SBP), and she was treated for a urinary tract infection. During the third hospitalization day, she passed tarry stool and her hemoglobin dropped from 9.4 gm/dl to 6.8 gm/dl. She received a blood transfusion, and her hemoglobin stabilized with negative subsequent stool occult blood.

Once stabilized, she was evaluated for her episode of gastrointestinal bleed. She was sedated with propofol and underwent diagnostic endoscopy and colonoscopy. She received 250 mg of propofol for a total procedure time of 27 minutes. She was found to have a clean base gastric ulcer, no varices, and a 3 mm colonic adenomatous polyp.

At the end of the procedure, the patient developed tachypnea with labored respirations. Arterial blood gas (ABG) was pH 7.14, pCO₂ 45, and pO₂ 145. Her previous day ABG was pH 7.44, pCO₂ 26, and pO₂ 76. She was given sodium bicarbonate and transferred back to the intensive care unit. Chest X-ray showed elevated diaphragms but no evidence of aspiration pneumonia or free air. She was subsequently intubated as she was fatigued from her respiratory distress.

Repeat paracentesis showed no evidence of SBP, and CT of abdomen revealed no evidence of perforation.

She was extubated 2 days later but again passed melena and dropped her hemoglobin requiring blood transfusion. A repeat endoscopy was performed to evaluate for the source of bleeding. Because of her previous respiratory distress from the procedure, the same anesthesiologist gave her a smaller amount of propofol. She was found to have an adherent clot on her gastric ulcer that was easily flushed away. There was no visible vessel under the clot. Her total sedation time was 10 minutes.

Post-procedure, she again developed tachypnea. Her ABG was pH 7.27, pCO₂ 37, and pO₂ 95. Her previous ABG was pH 7.44, pCO₂ 26, and pO₂ 127. She was given sodium bicarbonate and was observed in the intensive care unit without the need for intubation. She recovered uneventfully and was discharged without further episode of bleeding or respiratory distress.

Discussion

The patient developed metabolic acidosis following propofol administration. Her procedure took 27 minutes. Initial thoughts of her respiratory distress were iatrogenic perforation of her colon, aspiration of gastric content, or respiratory distress from an insufflated colon with her underlying ascites. Her ABG revealed metabolic acidosis. Once intubated and stabilized, paracentesis and CT scan of the abdomen revealed no evidence of SBP or perforation.

On her second endoscopy, the procedure took only 10 minutes, and again, she developed metabolic acidosis. The only commonality between the two episodes was the use of propofol, and because the second procedure took 10 minutes compared to 27 minutes the first time, her metabolic acidosis was not as severe.

Rare occurrence of severe and fatal complication associated with propofol use has been reported. Propofol infusion syndrome (PRIS) was described by Bray in 1998 when he reported 13 deaths in children associated with propofol use.² The syndrome was defined as sudden bradycardia, lipemic plasma, enlarged liver, metabolic acidosis, and rhabdomyolysis. This condition was associated with propofol infusion of 4 mg/kg/hr over 48 hour duration. Cardiovascular collapse is the common final pathway of PRIS. Profound metabolic acidosis, rhabdomyolysis, and respiratory and renal

dysfunction often complicate the clinical course leading to death.

PRIS was later described in adults. In 2009, Roberts et al³ evaluated critically ill patients receiving propofol infusions longer than 24 hours and reported the incidence of PRIS to be 1.1%. Mechanisms leading to PRIS are unknown. It is speculated that propofol inhibits intracellular energy production of mitochondria by inhibiting transportation of long chain fatty acids into the cell and/or having inhibitory effects on the intracellular mitochondrial respiratory chain.⁴ Inhibition of the mitochondrial respiratory chain will lead to impaired ATP production. Muscle necrosis may occur when metabolic demands exceed ATP production

There is no specific therapy for PRIS; therefore, prevention is the key. If possible, avoid using propofol for sedation longer than 3 days and at a maximum dosage of 4mg/kg/hr.⁵ During propofol infusion, one should monitor ABG, electrolytes, lactate, creatinine kinase, creatinine, and triglycerides.⁶

Metabolic acidosis associated with short-term propofol use has also been reported in ablation therapy for atrial fibrillation as a sedative agent for intubated patient and in neurosurgical procedures.⁷ However, the reported duration of propofol infusions varied from 2 to 7 hours. Cravens et al⁸ reported metabolic acidosis may occur in as high as 24% of the patients undergoing radiofrequency ablation, using propofol as a sedative agent. This was a retrospective chart review of patients undergoing radiofrequency ablation of arrhythmic foci. Arterial blood gases were obtained at the anesthesiologist's discretion. Of the 55 ABG obtained, 13 cases of metabolic acidosis were deemed to be due to propofol use. But these were often prolonged procedures, and ABGs were not performed on all the patients receiving propofol. So the true incidence is not likely to be this high. All recovered without sequelae.

Conclusion

Our patient developed documented metabolic acidosis after receiving propofol for as short as 10 minutes. The severity of metabolic acidosis in our patient appeared to be dependent on the duration of propofol use. The true incidence of metabolic acidosis associated with propofol use is not known, and most patients seem to recover with no long-term deleterious effects. With widespread use of propofol as a sedating agent, it may not be impractical to monitor ABG in all of our patients receiving propofol. However, in the patient who develops respiratory distress, one should obtain ABG to evaluate for possible metabolic acidosis from propofol use.

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