Acute Pancreatitis in a Four Year Old Following a Sixty Minute Propofol Infusion

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**Introduction:** Hypertriglyceridemia has been well-established as a cause of acute pancreatitis reported to account for 1.3% to 3.8% of all cases. Propofol, a lipid-based anesthetic agent, has been associated with hypertriglyceridemia after prolonged infusion. However, there have been no case reports of propofol causing acute pancreatitis in a patient younger than twelve years of age. We describe the case of a four year old girl who developed acute pancreatitis following a brief propofol infusion.

**Case Description:** A four year old, 18.8 kg girl with a history of glycogen storage disease Type IA presented for tonsillectomy and adenoidectomy with pressure equalizing tube insertion. The indication for surgery was obstructive sleep apnea and chronic serous otitis media. She was born via SVD and was diagnosed with glycogen storage disease at 2 months of age when her enzyme deficiency was confirmed with liver biopsy and peripheral blood studies. Due to hypoglycemia with prolonged fasting, she required continuous tube feeding overnight. Her prior anesthetic for G-tube insertion was uneventful. A triglyceride level six months prior to the indexed surgery was 792mg/dL (normal 25-120mg/dL). Induction of general anesthesia was accomplished with fentanyl 35mcg, propofol 45mg, and rocuronium 12mg. She was maintained with a propofol/remifentanil infusion and nitrous oxide 60% in oxygen. The surgery, which lasted about sixty minutes, proceeded uneventfully and the patient was extubated and transferred to the PACU in stable condition. Ten hours postoperatively, she developed nausea and vomiting refractory to anti-emetics. Over the next 24 hours, the patient developed abdominal pain, confusion, and lactic acidosis. Blood work obtained 36 hours postoperatively revealed amylase of 393U/L (normal 0-137), lipase of 1782U/L (normal 12-70) and triglycerides of 400mg/dL (normal 25-120 mg/dL). The diagnosis of acute pancreatitis was made. Ultrasound of the abdomen revealed a diffusely echogenic liver (secondary to glycogen and fat deposition), a distended gallbladder with no stones, a normal biliary system, and a well visualized pancreas with no pseudocysts or abnormal fluid collections. The patient was treated supportively with total parenteral nutrition. She did well and her abdominal pain improved on day three. Her labs also improved to amylase-174U/L, lipase-577U/L, triglycerides-334mg/dL. The patient was restarted on enteral nutrition on day six and was discharged home on day seven. Upon discharge, her amylase was 81U/L and lipase was 134U/L. She did not have any further negative sequelae and her abdominal pain had completely resolved at the time of her one month follow-up. Her serum amylase and lipase at follow-up were both 54U/L.

**Discussion:** There are 2 case reports of pancreatitis (one acute and one chronic) associated with glycogen storage disease type I. Both of these patients had a history of hyperlipidemia unresponsive to dietary therapy. Acute pancreatitis secondary to hypertriglyceridemia is well established. The proposed mechanism is that hydrolysis of triglycerides in the pancreas leads to high concentrations of free fatty acids (FFA) which are toxic and can produce capillary and cellular injury. In addition the increase in pancreatic lipase in the capillaries results in lipolysis, ischemia, capillary damage, and microthrombi. The resulting combination of an acidic environment and FFA cause activation of trypsinogen which initiates acute pancreatitis. Pancreatitis typically does not occur unless the serum triglyceride level reaches 1000 to 2000 mg/dL. Propofol is 2,6-diisopropylphenol available in the US as a 1% solution in an aqueous solution of 10% soybean oil, 2.25% glycerol, and 1.2% purified egg phosphatide (lecithin). Because it is a lipid emulsion, it has been shown in some studies to raise the triglyceride level, though usually only after prolonged infusion and usually not to clinically significant levels. It is this
A case of a twelve year-old girl who developed acute pancreatitis after propofol sedation for MRI was recently published. Upon retrospective chart analysis that patient was noted to have elevated triglyceride levels (400-800mg/dl) for 2.5 years prior to the event. In our case, our patient had mild hypertriglyceridemia likely secondary to her glycogen storage disease type I. There is a case in the literature of propofol causing pancreatitis in a patient whose triglyceride level, while elevated at the time of the event, was lower than at that patient’s baseline (as in our case). We feel that it is most likely that propofol was the triggering agent which caused the acute pancreatitis in a patient who was predisposed due to her preexisting hypertriglyceridemia. We did not find any case reports in the literature of pancreatitis associated with the other drugs used in our case: fentanyl, remifentanil, or rocuronium. On the basis of these cases, we would suggest that special consideration should be given to avoid the use of propofol in children with preexisting hypertriglyceridemia.

Refs:
1. Gottschling, S. et al., Pediatric Anesthesia, 2005
2. Kumar, A. et al., Chest, 1999