

Effects of Short-term Propofol Administration on Pancreatic Enzymes and Triglyceride Levels in Children

Gottschling S, Meyer S, Kreen T, Kleinschmidt S, Reinhard H, Graf N, Shamdeen GM. *Anaesthesia* 2005; 60: 660-3.

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A prospective study on the effects of short-term propofol administration on serum lipase, serum amylase, and triglyceride levels in pediatric patients undergoing magnetic resonance imaging (MRI) was conducted.

Forty children with cognitive and/or motor developmental delay, aged 4-178 months, undergoing MRI participated in the study. Inclusion criteria were ASA I or II children. Exclusion criteria were children aged 3 months or younger, hemodynamic instability, pre-existing hypotension, respiratory failure, seizures, or prior sensitivity to propofol. An initial venous sample was drawn prior to each propofol anesthetic, and a second venous sample was obtained four hours after the propofol infusion was discontinued. Blood samples were centrifuged at room temperature (18-23 deg Celsius), and measured within two hours after sampling. Serum amylase was measured by ethylidene liquid test, serum lipase by photometric enzyme colour test, and serum triglycerides by enzymatic colourimetric test.

Mean (SD; range) age was 67 (66; 4-178) months. Mean (SD; range) duration of anesthesia was 46 (29; 15-160) min, mean (SD; range) propofol loading dose was 2.2 (1.1; 1.5-4.5) mg/kg, mean (SD; range) continuous propofol infusion 6.9 (0.9; 5-8) mg/kg/h, mean (SD; range) total propofol dose 7.5 (1.7; 5-15.5) mg/kg. While no patients developed clinical signs of pancreatitis within 24 hours after stopping propofol infusions, mean (SD) serum lipase levels were elevated to 27.3 (13.1) IU/liter four hours after propofol versus 23.8 (7.7) IU/liter baseline ($P=0.035$); mean (SD) serum triglyceride levels were elevated to 141.9 (111.7) mg/deciliter four hours after propofol versus 106 (83.2) mg/deciliter baseline ($P=0.003$). It should be noted that these higher serum lipase and triglyceride values were still within normal laboratory limits. No significant difference was found between serum amylase baseline values and values at four hours after stopping propofol.

Comment: This is the first article in the published literature to report a possible association between propofol and acute pancreatitis in the pediatric population. Cases of presumed propofol-associated pancreatitis have been reported in adult patients—the postulated mechanism involves hydrolysis of triglycerides in the pancreas leading to toxic levels of high concentrations of unbound fatty acids, causing acinar and capillary injury, possibly via chylomicrons. The authors state that pancreatic cells in children might be more sensitive to propofol; propofol may have a direct damaging effect on acinar cells. The authors purport that pancreatitis as a possible complication of propofol administration should be considered in patients with abdominal pain even after uneventful short term propofol sedation, and that propofol be discouraged for patients with previously sensitized pancreas, history of pancreatitis, biliary tract disease or cystic fibrosis.

This study, while limited by its small sample size, comes at a point in time where many pediatric anesthesiologists are increasingly concerned over the safety of intraoperative propofol infusions for MRI. Propofol infusion syndrome (PIS) (defined as sudden or relatively sudden onset of marked bradycardia, resistant to treatment, with progression to asystole plus one of the following: lipemia, clinically enlarged liver secondary to fatty infiltration, severe metabolic acidosis with base deficit of >10 mmole/liter, or presence of muscle involvement with evidence of rhabdomyolysis or myoglobinuria) was originally described in pediatric intensive care unit (PICU) setting in patients undergoing prolonged (>24 -48 hours), high dose (>150 mcg/kg/min) long-term propofol sedation. To date, at least eighteen pediatric cases of propofol toxicity in intensive care settings have been reported in the literature providing evidence of an association between propofol and PIS. While propofol is widely believed benign for intraoperative use, so far only one published randomized study has validated the safety of propofol in a group of 36 ASA I children, aged 3-12 years, receiving varying rates of short term propofol infusions. In this study, no association with metabolic acidosis was found. Until further prospective studies examining the safety of intraoperative propofol infusions in pediatric patients are available, the clinician must be cautious and vigilant when administering such an anesthetic.