Hemorrhagic Pancreatitis Following Short- term Propofol Infusion in a Fifteen Year Old with Acute Fulminant Hepatic Failure (FHF)

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Introduction: Although extremely rare, there have been an increasing number of reports of acute and sometimes severe pancreatitis following short-term and even single dose propofol infusion in adults. In this case report we would like to present the case of a fifteen year old girl with acute FHF who developed severe hemorrhagic pancreatitis after a 48 hour infusion.

Case Description: A fifteen year old, previously healthy, 80 kg female with a history of acute fulminant hepatic failure (FHF), presumably autoimmune in nature presented to our institution for evaluation and possible orthotopic liver transplant (OLT) after her PCP noticed jaundice and abnormal liver enzymes. Upon admission she was started on Solumedrol (for presumed fulminant autoimmune hepatitis), Ppcecid, Vitamin K, and Lactulose (for developing encephalopathy). Her amylase and lipase on hospital day #2 were 86 U/L (0-137) and 17 U/L (normal 12-70) respectively. Pt. also had an abdominal CT and U/S on day #2 which showed a normal pancreas and no ascites. On day 3 she was intubated for stage 3 encephalopathy and decreased oxygenation. Pt. was intubated with Etomidate 0.3mg/kg, Rocuronium 1mg/kg, and Fentanyl 2mcg/kg. After intubation she was started on propofol infusion of 35 mcg/ kg/ min for sedation. Over the next 48 hours the patient’s condition worsened and she became increasingly acidic (lactic acid prior to propofol infusion was 2.8 mm/L and increased to 20 mm/L). She developed abdominal distention and required volume and pressors for hemodynamic support. Presuming her hepatic failure was the cause for the deterioration the patient was emergently taken to the OR for hepatectomy pre-OLT as a bridge to transplant. Upon exploration of the abdomen, the surgeon noted severe hemorrhagic pancreatitis with saponification and pancreatic ascites. The plan for OLT was abandoned due to the patient's medical condition. After return to the floor the propofol infusion was not restarted. Her lipase and amylase at that time were 1305 U/L and 5502 U/L, respectively. She continued to deteriorate and on hospital day #9 the family decided to withdraw care and extubate. Patient expired soon afterwards.

Discussion: Drug induced pancreatitis is an extremely rare disorder, accounting for just 1.4% of all cases. Although also rare, there are increasing numbers of case reports linking short term propofol infusions to pancreatitis. This is the first reported incident of a pediatric patient to develop hemorrhagic pancreatitis in this manner. Clinical and laboratory evidence of pancreatitis is routinely screened for in pediatric patients admitted to our institution for FHF. While a possible association of corticosteroids and pancreatitis has been previously established, our patient had normal pancreatic laboratory and imaging studies while on steroids. The temporal relationship between the initiation of the propofol drip in our patient with a deteriorating medical condition including extremely high lactic acid levels and hemodynamic instability and the subsequent development of hemorrhagic pancreatitis suggests a possible link between these two events. The possibility that the pancreatitis could have occurred secondary to hypertriglyceridemia induced by the propofol infusion must be addressed. Boyle reported that serum triglycerides were increased in 2/22 patients on a prolonged propofol infusion exceeding 100 mcg/ kg /min and Gottschling reported 4/44 patients that had elevated triglyceride levels after a propofol infusion for less than 160 minutes. Further studies need to be done to establish a relationship in these situations since propofol is generally the drug of choice used to sedate encephalopathic patients. Monitoring during the care of patients with fulminant hepatic failure might therefore include daily amylase, lipase, triglyceride and lactic acid levels especially if they are on propofol. Other risk factors need to be elucidated.
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