J Clin Case Rep 2017, 7:9 (Suppl)
DOI: 10.4172/2165-7920-C1-015

Severe hypertriglyceridemia-induced pancreatitis managed with plasmapharesis. A case report

Sheref Abdelgawad Elseidy, Ahmed Abd Allah Khalifa and **Ahmed Mahmoud Khalifa**
Ain Shams University Hospital, Egypt

Hypertriglyceridemia (HTG) is reported to cause 1-4% of acute pancreatitis (AP) episodes. Disorders of lipoprotein metabolism are conventionally divided into primary (genetic) and secondary causes, including: diabetes, hypothyroidism, and obesity. Serum triglyceride (TG) levels above 1,000 mg/dl are usually considered necessary to ascribe causation for AP. The mechanism for hypertriglyceridemia pancreatitis (HTGP) is postulated to involve hydrolysis of TG by pancreatic lipase and release of free fatty acids that induce free radical damage. Interestingly, serum pancreatic enzyme levels may be normal or only minimally elevated in such cases. The reduction of triglyceride level to below 1000 mg/dL effectively prevents further episodes of pancreatitis. The mainstay of treatment for the hypertriglyceridemia associated with pancreatitis includes dietary restriction of fat and administration of lipid-lowering agents. It is thought that within 24 to 48 hours of the onset of pancreatitis, in the majority of patients, triglyceride levels fall rapidly as a result of fasting status, as the absorption of chylomicrons to the blood is cut off. Experiences with plasmapheresis are limited. This study reported a young female, 42 years old patient presented to our Emergency Room (ER) presented by epigastric pain radiating to the back, vomiting and fever. On examination (O/E) she was fully conscious, feverish, with epigastric tenderness. While withdrawing her blood for laboratory tests, her blood looked milky white. Laboratory results showed: elevated kidney functions (creatinine 3.4 mg/dL) and serum amylase 209 IU. Pelvi-abdominal ultrasonography (PAUS) showed bulky heterogeneous pancreas and was diagnosed with pancreatitis. She had hyperlipidemia. Her TGs level was (1335mg/dL) and serum cholesterol was (750mg/dL). She did not improve on full dose of HypolipidemicsShe developed acute kidney injury and her serum blood urea nitrogen (BUN) was 72 mg/dL and serum creatinine was 6.3 mg/dL and received one session of hemodialysis then plasmapharesis was initiated and she received 8 sessions with full dose of hypolipidemics. She responded only to plasmapharesis. Her TGs and cholesterols started to decrease and reached 545mg/dL and 194 mg/dL respectively and she clinically started to improve and her serum amylase was 11 IU. Plasmapharesis was stopped. Unfortunately she was deteriorated again with more severe symptoms, re-elevation of TGS and cholesterol again. CT abdomen revealed bulky heterogeneous pancreas suggestive of necrotizing pancreatitis with moderate ascites and bilateral pleural effusion. And then she had had 2 attacks of generalized tonic clonic fits followed by deterioration of conscious level and the condition was deteriorated more and she developed disseminated intravascular coagulopathy (DIC) and acute respiratory distress syndrome (ARDS) and unfortunately she passed away.

drsherif.elseidy@gmail.com