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Case Report

An interesting case of acute pancreatits

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ABSTRACT

Hypertriglyceridemia (HTG) is the third most common cause of acute pancreatitis but it is relatively rare and requires a high level of clinic suspicion. We report a 30 years old male with no co-morbidities who is a non-alcoholic, non-smoker presented with epigastric pain associated with vomiting for two days. His vitals were stable and systemic examination was unremarkable. Serum lipase levels were elevated [7365 U/l (<40 U/l)]. Computed Tomography of the abdomen suggested acute pancreatitis with extensive peri-pancreatic inflammatory changes with no evidence of pancreatic necrosis. Serum triglycerides were found to be elevated (3450 mg/dl). A diagnosis of HTG induced acute pancreatitis was made. During evaluation he was found to be diabetic. He was treated with intravenous fluids, IV insulin infusion, and other supportive measures. He was started on statins, fenofibrate and omega 3 fatty acids. His condition improved and triglyceride levels after two days were in a declining trend and he was discharged on the same. He was currently under follow up and his serum amylase, serum lipase, serum triglycerides were normal. This case report provides insight into rare cause of acute pancreatitis-HTG which if diagnosed at the earliest can be treated promptly.

Keywords: HTG, Acute pancreatitis, IV Insulin

INTRODUCTION

Acute pancreatitis is an inflammatory condition of the pancreas that is painful and at times lethal. Despite the great advances in critical care medicine over the past 20 years, the mortality rate of acute pancreatitis has remained at about 20%. Acute pancreatitis secondary to HTG is seen in patients with disorders of lipid metabolism and is highly related to the subsequent development of cardiovascular comorbidities.[1,2] Alcohol consumption and gallstones represent the most common etiologies while pancreas divisum, annular pancreas, pancreatic trauma, hypercalcemia, hyperlipidemia, autoimmune, and medications are less common causes of acute pancreatitis.[3]

HTG is an uncommon but well-established cause of acute pancreatitis. Studies on patients with familial HTG and their long-term follow-up have shown that in the absence of other causes for pancreatitis, extreme elevations of triglycerides occur during episodes of acute pancreatitis and the so called "hyperlipidemic abdominal crisis." It is generally believed that a triglyceride level of more than 1,000 mg/dl is needed to precipitate an episode of acute pancreatitis.

A secondary factor such as alcoholism, obesity, uncontrolled diabetes mellitus, or a number of medications prone to increase lipid levels is present in the large majority of patients and should always be identified.[4] The etiology should be suspected in obese, diabetic or hyperlipidemic patients. Although the clinical course of HTG induced pancreatitis (HTGP) is often similar to other forms of acute pancreatitis with the only distinguishing clinical presentation observed initially being HTG, the complication rate observed is significantly higher in patients with HTG-induced pancreatitis than in patients

with other causes of acute pancreatitis.[5] We report a case of acute pancreatitis due to HTG which was effectively treated with intravenous fluids and insulin therapy.

CASE REPORT

A 30-year-old male with no co-morbidities who is a non-smoker and non-alcoholic presented with epigastric pain radiating to back associated with vomiting for 2 days. His BMI was 26 kg/m^2. Family history was unremarkable. His vitals were stable and systemic examination was unremarkable. His initial laboratory investigations showed hemoglobin-14.2 g/dl, total leucocyte count of 12,400 cells/cumm (N-72%, L-20%, M-6%, E-2%, B: 0%), Platelets-286,000 cells/cumm. Renal function tests, serum electrolytes, liver function tests, coagulation profile were normal. Cardiac evauation was normal. His serum lipase was increased several folds with value of 7365 U/l (<40 U/l). His CT abdomen findings were suggestive of acute pancreatitis and there were extensive peri-pancreatic inflammatory changes with no evidence of pancreatic necrosis. His fasting blood glucose was 300 mg/dl, post-prandial blood glucose was 350 mg/dl and HbA1c was 8.9%. He was diagnosed to have diabetes mellitus at time of evaluation. Lipid profile was total cholesterol-1450 mg/dl (160-200 mg/dl), triglycerides- 3450 mg/dl (50-150 mg/dl), HDL-35 mg/dl (30-60 mg/dl), LDL-725 mg/dl (80-175 mg/dl), VLDL-690 mg/dl (2-30 mg/dl). He was diagnosed to have HTG induced acute pancreatitis. In acute phase, he was treated with IV fluids, I.V insulin for hypertriglyceridemia and hyperglycemia and analgesics for symptomatic relief. He was started on statins, fenofibrate and omega 3 fatty acids. His condition was improved and trigyceride levels after two days were 680 mg/dl. He was discharged with statins, fenofibretes, omega 3 fatty acids and insulin. He was currently under follow up and his serum lipase, serum amylase, serum triglycerides were normal.

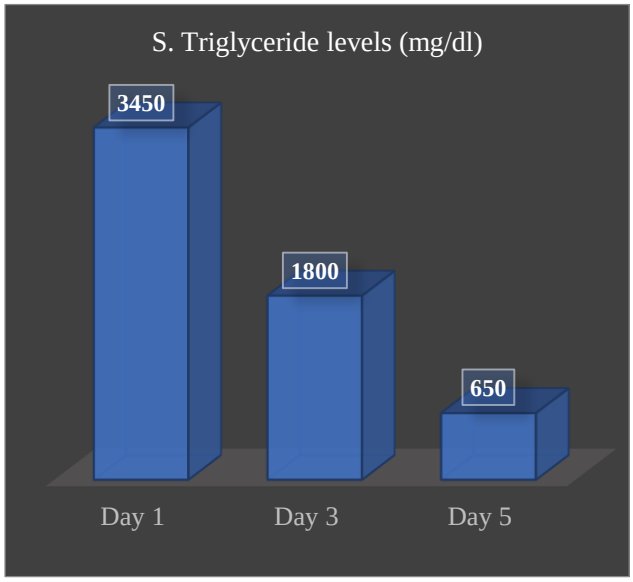

Figure 1: Serum triglyceride levels.

DISCUSSION

Patients who present with HTG induced pancreatitis usually have a preexisting abnormality in lipoprotein metabolism. The common scenarios in which a clinician would encounter a patient with HTG pancreatitis include a poorly controlled diabetic with or without a history of HTG, an alcoholic patient admitted with acute pancreatitis who is found to have a lactescent serum, a nondiabetic, nonalcoholic, non-obese patient who has HTG secondary to diet or drugs, and a patient with one of the familial hyperlipidemias presenting with acute pancreatitis in absence of a secondary factor.[4]

The mechanism by which HTG leads to pancreatitis is not clear. A well-accepted mechanism proposed in a study done by by Havel et al is hydrolysis of triglycerides in and around the pancreas by pancreatic lipase seeping out of the acinar cell leads to accumulation of free fatty acids in high concentrations.[6] Unbound free fatty acids are toxic and could produce acinar cell or capillary injury. Increased concentration of chylomicrons in the pancreatic capillaries causes capillary plugging and leads to ischemia and acidosis, and in the acidotic environment, free fatty acids cause activation of trypsinogen and initiate acute pancreatitis. Targeting acute phase of acute pancreatitis is necessary for successful management of HTG-induced pancreatitis. Initial management of acute pancreatitis includes bowel rest with no oral intake, aggressive intravenous hydration, symptomatic care including pain management. Acute phase treatment of HTG-induced pancreatitis includes IV insulin, IV Heparin and apheresis/Plasmapheresis for refractory cases.[5]

A recent prospective RCT by He et al on 66 hyper-triglyceridaemia-induced acute pancreatitis patients received either early high-volume haemofiltration (HVHF) or low molecular weight heparin (LMWH) plus insulin as an emergent triglyceride-lowering therapy. As per the results, HVHF lowered triglyceride levels more quickly than LMWH plus insulin therapy, but no difference was noted in terms of clinical outcomes, local pancreatic complications, need of surgery and cost.[7] Following the initial management, appropriate measures are necessary to decrease serum triglyceride levels to decrease the risk of relapse.

Sahu et al also reported a similar case of HTG-induced pancreatitis in which alcohol was a predisposing factor. Identification of secondary factor precipitating acute pancreatitis is most important for prompt management and for preventing further relapses.[7]

CONCLUSION

HTG induced pancreatitis is a rare entity, but well-established cause of acute pancreatitis. With new advances in therapeutic options identification of etiology in acute pancreatitis and timely intervention can reduce morbidity and mortality.

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