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Hypertriglyceridemia induced severe acute pancreatitis (A rare presentation): A series of 3 cases

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Abstract
Hypertriglyceridemia is an unknown but well-established cause of acute pancreatitis following gall stones and alcohol. In addition to the supportive management of acute pancreatitis, specific therapeutic interventions for the reduction in levels of triglycerides like insulin, heparin and plasmapheresis have been studied and used in the management of hypertriglyceridemia induced pancreatitis.
We report three cases of Hypertriglyceridemia induced severe acute pancreatitis with multi organ dysfunction who recovered with insulin and heparin therapy for lowering triglycerides and supportive management of the organ dysfunction associated with acute pancreatitis.

Keywords: Hypertriglyceridemia, acute pancreatitis, insulin, heparin infusion, plasmapheresis

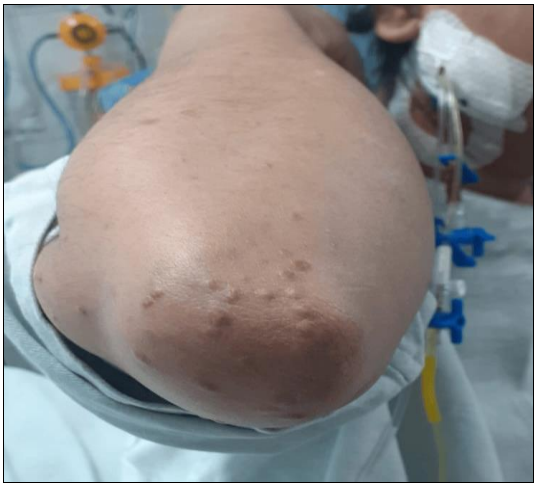
Introduction

Hypertriglyceridemia (HTG) is a rare but established etiology of acute pancreatitis, with a reported incidence of 6-7% [1]. Typically, TG levels >1000 mg/dL have been associated with Acute pancreatitis. The etiology of HTG can be broadly divided into 2 categories: primary and secondary. While primary one causes more severe HTG, more often it is the interplay of both primary and secondary factors that leads to severe HTG [2]. Secondary factors that are associated with HTG include obesity, alcohol abuse, uncontrolled diabetes mellitus, hypothyroidism, chronic renal failure, and drugs like estrogen, corticosteroids, and retinoids. Management of TG-induced pancreatitis involves lowering lipid levels through the use of plasmapheresis, heparin and insulin infusions and followed by fibrate therapy in the long term.

Case 1: A 54-year female, with a past medical history of diabetes on oral anti-hypoglycemics, presented with progressively worsening generalized abdominal pain associated with vomiting for four days along with decreased urine output for two days. She had no history of hematemesis or melaena. There was no history of alcohol or any substance abuse.
On physical examination the patient's pulse was 124 bpm, BP-106/64 mm of Mercury, Respiratory rate-32/minute, SO2- 88% on room air and right elbow had eruptive xanthomas (Picture 1). ECG was suggestive of sinus tachycardia, ABG - hypoxia with type I Respiratory failure with severe metabolic acidosis (Bicarbonate-6). RBS on admission was 460 mg %. Chest Xray- Bilateral heterogenous opacities suggestive of ARDS. Her hematological and biochemical investigations on admission were as follows.

Table 1: Hematological and Biochemical Investigations of Case 1

Hemoglobin	**13.5 g/dl**	**Serum Amylase**	**365 U/L**
Total Leucocyte count	22045 /ul	Serum Lipase	220 U/L
Platelets	288000/ul	Serum Cholesterol	558 mg %
Blood Urea Nitrogen	84 mg %	Serum Triglycerides	3200 mg %
Creatinine	6.5 mg %	Low Density Lipoprotein	150 mg %
HbA1C	10.2 %	Very Low density Lipoprotein	75 mg %
Procalcitonin	54.1		



Picture 1: Right elbow eruptive xanthomas

USG was suggestive of bulky pancreas with bilateral minimal perinephric fat stranding. A provisional diagnosis of Hypertriglyceridemia induced acute severe pancreatitis with acute respiratory distress syndrome with acute kidney injury was made. A Naso-jejunal tube was inserted and patient was kept nil by mouth (NBM) for the first 48 hours. Non-invasive mode of ventilation was started in view of the hypoxia. Intravenous continuous infusion of Heparin and Insulin was started to decrease the elevated triglyceride levels. Hourly glucose monitoring and coagulation profile monitoring every six hourly was done. In view of oliguria and severe metabolic acidosis patient was taken up for hemodialysis by Nephrology. The supportive management was continued over the next few days and gradual improvement in the patient's clinical and biochemical parameters was observed. The heparin and insulin drips were gradually stopped and patient was started on omega 3 fatty acids, a statin and fenofibrate. A CECT (Contrast Enhanced Computed Tomography) abdomen during the course of admission was suggestive of pancreatic pseudocyst formation for which the patient was advised conservative management and follow up under General Surgery.

Case 2: A 28-year male, chronic alcoholic since 7 years was admitted to the Medicine Intensive care Unit with history of altered sensorium with drowsiness since 2 days, acute onset generalized abdominal pain radiating to the back since 2 days and acute onset breathlessness since 1 day. On admission the clinical findings were Pulse- 130/min, BP- 100/60 mm Hg, Respiratory Rate -32/min, SO2- 92% on room air. ECG was suggestive of sinus tachycardia. ABG was suggestive of severe metabolic acidosis with compensatory respiratory alkalosis with mild hypoxia. On withdrawing blood for biochemical investigations, a highly lipidemic sample [Picture 2] was obtained repeatedly. After serial dilutions the following reports were available.

Table 2: Hematological and Biochemical Investigations of Case 2

Hemoglobin	**18.5 g/dl**	**Serum Amylase**	**582 U/L**
Total Leucocyte Count	12960/ul	Serum Lipase	156 U/L
Platelets	288000/ul	Serum Cholesterol	989 mg %
Blood Urea Nitrogen	36 mg %	Serum Triglycerides	5011mg %
Creatinine	0.5 mg %	Low density Lipoprotein	186 mg %
HbA1C	5.4 %	Very Low density Lipoprotein	110 mg %
Procalcitonin	22	Serum Sodium	113 meq/L
Uric acid	24.3 mg %	Serum Potassium	3.8 meq/L

Picture 2: Lipidemic blood sample of the patient

USG was suggestive of Bulky pancreatic tail with surrounding fat stranding suggestive of acute pancreatitis. A diagnosis of Hypertriglyceridemia induced pancreatitis with sepsis with metabolic encephalopathy was made based on the biochemical and radiological findings. Patient was kept Nil by Mouth (NBM). Naso-jejunal tube was inserted. In order to lower serum triglyceride levels patient was started on Intravenous infusions of Heparin and insulin along with Dextrose Normal saline with hourly blood glucose monitoring. Supportive treatment with Intravenous antibiotics and hydration according to Central Venous Pressure was continued. Serum sodium gradually improved to 133meq/L with improvement in patient's sensorium. Serial monitoring of Triglyceride levels was carried out. Patient gradually improved symptomatically with resolution of abdominal pain. Patient was shifted to oral statins and fenofibrate and was shifted out of the Medicine Intensive Care Unit after 8days of admission.

Case 3: A 66-year-old male patient without any history of comorbidities was admitted to the Medical Intensive Care Unit with history of generalized abdominal pain radiating to the back and persistent vomiting for 3 days. He denied any addiction. On examination his pulse was 130/min, BP- 100/70 mm of Mercury, Respiratory rate- 18/minute, SO2- 93% on room air. ECG was suggestive of sinus tachycardia, on ABG, pO2 was 68 mm Hg suggestive of Type I respiratory failure. Chest Xray showed bilateral costophrenic angle blunting. His hematological and biochemical investigations were as follows:

Table 3: Hematological and Biochemical Investigations of Case 3

Hemoglobin	**19.8 g/dl**	**Serum Amylase**	**635 U/L**
Total Leucocyte Count	22036/ul	Serum Lipase	142 U/L
Platelets	175340/ul	Serum Cholesterol	754 mg %
Blood Urea Nitrogen	50 mg %	Serum Triglycerides	4600 mg %
Creatinine	1.8 mg %	Low density Lipoprotein	170 mg %
HbA1C	5.5 %	Very Low density Lipoprotein	116 mg %
Procalcitonin	1.8	Serum Sodium	147 meq/L
Serum Uric acid	12 mg %	Serum Potassium	4.4 meq/L

USG Abdomen was suggestive of bulky pancreas with moderate ascites. CT Abdomen was suggestive edematous pancreas with peripheral fat stranding with moderate free fluid in the abdomen. On the basis of biochemical and radiological findings a diagnosis of Hypertriglyceridemia induced acute pancreatitis was made. A Ryle's tube was inserted and the patient was kept NBM. A central line was inserted and Intravenous fluid therapy was started with Central Venous Pressure monitoring. For the purpose of reducing levels of triglycerides patient was started on heparin and insulin infusions with close monitoring of coagulation profile and blood glucose levels. Supplemental oxygen therapy was provided in view of hypoxia. Patient started improving gradually symptomatically with decrease levels of lipids and increase in the oxygen saturation. Patient was eventually shifted out of intensive care after 5 days of treatment.

Discussion

Hypertriglyceridemia is the underlying cause of pancreatitis in 7% of the population, the third most common cause following gallstones and alcohol [1]. It is believed that a TG (Triglyceride) level of over 1000 mg/ dl (or 20 mmol/l) is needed to induce acute pancreatitis [3]. The exact mechanisms involved in hypertriglyceridemia-induced pancreatitis are not clear. Chylomicrons are thought to cause pancreatic inflammation when TG levels are elevated as they precipitate into the circulation [4]. It is believed that large lipoproteins could impair capillary bed circulation, causing oedema and ischemia to the pancreatic cells, disturbing the acinar structure and exposing them to lipases. The degradation of these particles into free fatty acids may produce a proinflammatory response, further damaging the acinar structure of the pancreatic cells [5]. Initial treatment is similar to that of acute pancreatitis due to other causes, which involves pancreatic rest by decreased oral intake, intravenous hydration, and pain management. Specific management for reducing the triglyceride levels include insulin infusion [6]. The mechanism of action behind this form of treatment suggests that insulin increases lipoprotein lipase (LPL) activity which can degrade chylomicrons and thus reduce serum triglycerides [7]. Another form of medical management which remains controversial is the use of heparin which can stimulate the release of endothelial lipoprotein lipase (LPL) into circulation; however, it may only result in transient rise in LPL followed by increased degradation of plasma stores causing LPL deficiency [8]. Another method of reducing triglyceride levels involves the use of plasmapheresis. This was first reported in 1978 by Betteridge *et al*. and can result in a rapid decrease in triglyceride levels over a short period of time compared to the other treatment options described [9]. However, in a resource limited setting insulin and heparin infusions are still considered cheaper and safer methods of emergency treatment. Fibrate therapy is used for long-term management as it has been proven to lower TGs, alongside a low-fat and high fiber diet. Omega-3-fatty acids have been shown to reduce TG levels when used in conjunction with other therapies [10].

Conclusion

Severe hypertriglyceridemia is a rare cause of acute pancreatitis especially in non-alcoholic, non-obese, non-diabetic patients. Management includes supportive treatment for the pancreatitis and specific therapies to reduce the high triglyceride levels. Although plasmapheresis is effective, it is found to be an expensive treatment option and is not available in all medical centers. Insulin and Heparin infusions still remain viable treatment options in a resource limited setting.

Conflict of Interest

The authors hereby declare that they have no conflicts of interest.

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