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Case Report

A case report of severe hypertriglyceridemia induced acute pancreatitis managed with continuous insulin infusion

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ABSTRACT

31-year-old male, chronic alcoholic presented to the emergency room with epigastric pain, nausea and vomiting. On examination he was febrile, had abdominal distention with tenderness in the epigastric region. His lipase was elevated and computed tomography of abdomen showed evidence of acute pancreatitis. His past history was significant for chronic myeloid leukemia on imatinib and poorly controlled type 2 diabetes mellitus. Laboratory studies revealed elevated triglyceride levels (5254 mg/dl) and uncontrolled blood sugars (HbA1c-10.77%). Due to the severity of his pancreatitis presentation, he was admitted to the intensive care unit. He received aggressive intravenous fluid hydration and was started on continuous insulin infusion. He improved significantly with insulin therapy. His triglyceride levels decreased from 5254 mg/dl to 1891 mg/dl after 48 hours of initiating insulin therapy, by fifth day of admission triglycerides were below 500 mg/dl and was clinically better. He was discharged with a basal dose of insulin and fenofibrates. Intravenous insulin infusion is an effective, affordable, and accessible therapy for acute pancreatitis due to severe hypertriglyceridemia.

Keywords: Hypertriglyceridemia, Pancreatitis, Insulin infusion

INTRODUCTION

Hypertriglyceridemia (HTG) is a significant cause of acute pancreatitis. It occurs when high levels of triglycerides in the blood lead to inflammation of the pancreas.[1] Treating HTG-induced pancreatitis involves reducing serum triglyceride levels to less than 500 mg/dl promptly since the risk of pancreatitis significantly rises when levels exceed 1,000 mg/dl.[3-5] Factors such as poorly controlled diabetes, alcohol consumption, certain medications, and genetic factors can contribute to this condition as they affect lipid metabolism.[6] Effective treatment is crucial due to the high mortality rate associated with acute pancreatitis. Initial management typically involves intravenous hydration to maintain fluid balance, analgesics for pain relief, and antibiotics to prevent infection.[7,8] However there is currently no specific therapy tailored for HTG-induced pancreatitis, although insulin therapy is suggested as a viable option, when other interventions are not available or feasible.[9]

CASE REPORT

31-year-old male known case of chronic myeloid leukemia on imatinib presented with complaints of upper abdominal pain and multiple episodes of vomiting of one-day duration also associated with nausea. Pain was maximum in the epigastric region, continuous and radiating to the back. Pain aggravated on lying down in supine position and decreased in intensity on leaning forward. Vomiting was non bilious and non-projectile.

No history of fever, loose stools, hematemesis or melena. History of chronic alcoholism, last intake two days prior to presentation. No relevant surgical or family history.

Physical examination

On general examination, he was well built and nourished, conscious, oriented, no pallor, icterus, cyanosis, clubbing, lymphadenopathy, or edema; skin, hair, nails, eyes, thyroid were normal. Pulse: rate 98/min, regular, normal character, all peripheral pulsations present, no radio-femoral delay. Blood pressure: 120/70 mmHg, sitting, recorded in both upper arms. Temperature: 98.6°F. Respiratory rate: 28/min SpO_2: 98% (room air). Systemic examination abdomen was distended, soft with tenderness in epigastrium and right hypochondrium, rest of the systems within normal limits.

Investigations

Investigations done showed leukocytosis with raised inflammatory markers. Marked elevation of serum lipase (2024) and amylase (507), mild transaminitis and uncontrolled blood sugars (HbA1c-10.77). Ultrasonography (USG) abdomen was taken in view of the severity of the symptoms and showed features suggestive of acute pancreatitis. Contrast enhanced computed tomography (CECT) abdomen was taken to look for gall stones and was negative. Fasting lipid profile done showed severe hypertriglyceridemia (5254).

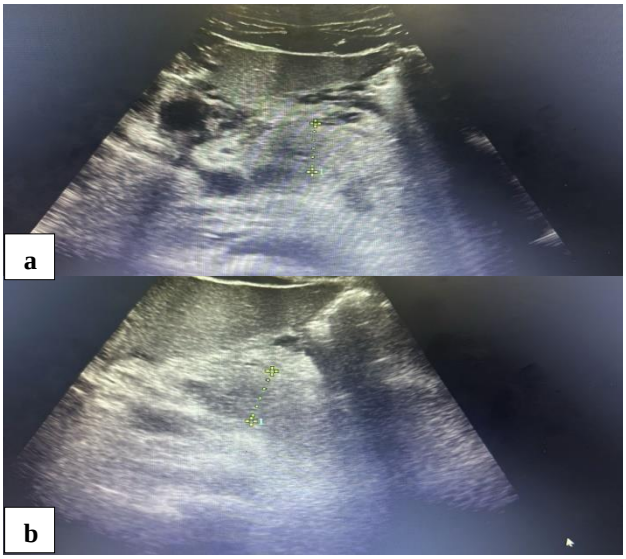

Figure 1: USG abdomen showing features suggestive of acute pancreatitis.

Diagnosis

A final diagnosis of severe hypertriglyceridemia induced acute pancreatitis was made. The diagnostic criteria for HTGP include the presence of at least two out of three following findings: acute-onset severe epigastric pain radiating to the back; serum lipase or amylase elevated three or more times the upper reference limit; and the three characteristic findings of acute pancreatitis on imaging investigations such as computed tomography, magnetic resonance imaging, or transabdominal ultrasonography. Another major clue for the diagnosis of HTGP includes biochemical evaluation remarkable for serum triglyceride levels more than 1000 mg/dl, in the absence of other potential causes of acute pancreatitis after detailed evaluation. The risk factors for hypertriglyceridemia are obesity, family history of hypertriglyceridemia, poorly-controlled diabetes mellitus, and alcoholism.

Treatment and follow-up

Currently, there are no clear therapeutic guidelines for HTGP. Insulin therapy has previously been used in these patients as a minimally invasive and economical strategy with promising outcomes. The mechanism by which insulin lowers the level of serum triglycerides is by triggering the enzymatic activity of lipoprotein lipase and inhibition of hormone-sensitive lipase. Lipoprotein lipase metabolizes chylomicrons and VLDLs into the free fatty acids and glycerol. Therefore, it ultimately decreases the serum triglyceride levels. Decreasing the activity of hormone-sensitive lipase causes decreased adipocyte-triglyceride breakdown, resulting in a decreased release of free fatty acids into the circulation, which controls the toxic effects on the pancreas, limiting its active inflammation. The initial symptomatic management comprised of bowel rest, intravenous fluids, and analgesics. It was given intravenously at a rate of 0.1-0.3 units/kg/hour. Serum triglyceride levels were monitored every 12 hours. With insulin therapy, it was pivotal to measure blood glucose levels and adjuvant 5% dextrose infusion was required when the blood glucose level fell below 200 mg/dl.

DISCUSSION

Acute pancreatitis can be triggered by various factors, with gall stones and alcohol abuse being the most common culprits. However, hypertriglyceridemia, although less frequent, is a recognized cause, occurring in approximately 2-4% of cases.[1] Triglycerides, essential components of fats, are either synthesized in the liver or obtained from the diet. In normal conditions, circulating triglycerides are metabolized into free fatty acids by an enzyme called lipoprotein lipase. Hypertriglyceridemia is diagnosed when fasting serum triglyceride levels exceed 150 mg/dl, and it becomes particularly concerning when levels reach or surpass 2000 mg/dl, significantly increasing the risk of acute pancreatitis to about 10-20%.[2-4] This elevation in triglyceride levels alters the composition of very low-density lipoprotein particles, making them larger and less efficiently processed by lipoprotein lipase.[5] Consequently, the breakdown of triglycerides during acute pancreatitis leads to the release of toxic free fatty acids, contributing to the damaging effects on the pancreas known as lipotoxicity.[6] Managing hypertriglyceridemia-induced pancreatitis poses challenges, with no universally agreed upon treatment protocol. Current options include supportive care such as hydration, pain management, and bowel rest, alongside interventions like continuous insulin infusion and, in

severe cases, plasmapheresis.[7] Plasmapheresis is a more aggressive approach aimed at removing triglycerides from circulation, but it is associated with significant complexities and costs, making it reserved for critical situations. Continuous insulin infusion (CII) emerges as a promising therapeutic option for hypertriglyceridemia-induced pancreatitis. Insulin stimulates the activity of lipoprotein lipase, facilitating the breakdown of triglycerides into free fatty acids and glycerol, thereby reducing serum triglyceride levels.[8,9] Additionally, insulin inhibits hormone-sensitive lipase, which decreases the breakdown of triglycerides stored in adipose tissue, further preventing the release of free fatty acids into circulation and limiting their toxic effects on the pancreas. Overall, continuous insulin infusion offers a safer, more economical, and minimally invasive alternative for managing hypertriglyceridemia-induced pancreatitis.[10]

CONCLUSION

Patients with HTGP require urgent management as the disease presentation is particularly severe and it may result in grave complications. The use of insulin therapy with close monitoring of blood glucose levels can be an appropriate therapeutic approach, especially in cases with no availability of apheresis. This paper not only highlights the utility of insulin therapy for HTGP but also sensitizes concerned physicians to evaluate this treatment approach in larger, multicenter studies. Long-term management using pharmacological and non-pharmacological therapies, directed at maintaining the serum triglycerides within normal limits, is required to prevent recurrent attacks of HTGP.

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