CASE REPORT

Hypertriglyceridemia-induced Acute Pancreatitis: A Case Report

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Abstract

Increased serum triglyceride level is the third most common cause of acute pancreatitis. Although a rare entity but results in severe pancreatitis with life-threatening organ dysfunction if untreated; therefore it requires a high level of clinical suspicion to be accurately diagnosed. We discuss the case of a 42-year-old man who initially presented in emergency department with suspected acute pancreatitis but who did not respond to the conservative management. On taking detailed history and clinical examination showed an evidence of hyperlipidemia. Subsequent investigations revealed acute pancreatitis secondary to hypertriglyceridemia. In this report, we tried to highlight few causes of hypertriglyceridemia, and the role of plasmapheresis and euglycemic insulin therapy in the management of hypertriglyceridemia-induced acute pancreatitis.

Keywords: Acute pancreatitis, Euglycemic insulin therapy, Hypertriglyceridemia, Plasmapheresis.

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Introduction

Fasting levels of serum triglyceride in excess of >150 mg/dL (1.7 mmol/L) is defined as hypertriglyceridemia (HTG). Based on the degree of elevation HTG is categorized as: mild, moderate, severe HTG, very severe HTG.[1]

Severity	*Triglyceride level*
Mild	150–199 mg/dL or 1.7–2.2 mmol/L
Moderate	200–999 mg/dL or 2.3–11.2 mmol/L
Severe	1000–1999 mg/dL or 11.3–22.5 mmol/L
Very severe	≥2000 mg/dL or >22.6 mmol/L

Very high risk for acute pancreatitis is considered when HTG levels are above 1000 mg/dL (11.3 mmol/L).[2] With such high triglyceride level of >1000 mg/dL (11.3 mmol/L), the risk of developing acute pancreatitis is approximately 5% whereas with triglyceride level >2000 mg/dL (22.6 mmol/L) the risk rises to 10–20%.[3] Patients with HTG-induced pancreatitis are more likely to have severe pancreatitis compared to those patients with other causes of pancreatitis. Elevated triglyceride levels are linearly associated with the severity of acute pancreatitis, higher the level of triglycerides more severe is the pancreatitis. Control of pancreatitis caused by TG involves immediate lowering of the lipid levels through plasmapheresis, heparin, and insulin infusions followed by oral fibrate therapy and long-term lifestyle modifications. We are going to report a case of severe hypertriglyceridemia-induced pancreatitis which responded to insulin and plasmapheresis with dramatic correction of the triglyceride levels.

Case Descriptions

- We report a case of 42-year-old man admitted to Medica Superspecialty Hospital, with complaints of sudden epigastric pain, radiating towards back, associated with nausea and vomiting (2–3 episodes/day) for 3 days. There was no association of pain with food intake. No history of fever, loose stools, jaundice, analgesic abuse, or no history of similar episodes in the past.
- He was known hypertensive on regular antihypertensive therapy and was also previously on statins in view of dyslipidemia since past 3 years. No history of any substance abuse.
- He was admitted at a local healthcare facility initially and was diagnosed as a case of acute pancreatitis based on USG whole abdomen and was treated with intra venous (iv) fluid, iv antibiotics, and other supportive drugs. There was no evidence of cholelithiasis in the USG whole abdomen. He was then transferred to Medica Superspecialty hospital to continue further treatment.
- On physical examination, he had normal vitals (heart rate—80/minute, blood pressure—127/78 mm Hg, temperature—98.7°F, respiratory rate—18/minute, SpO_2—96% in room air). He had epigastric tenderness, no distension but guarding present.
- Initial blood reports showed raised amylase and lipase of 195 and 981 U/L, respectively. Fasting lipid profile showed triglyceride—2560, LDL—269, VLDL—512, HDL—32, and total cholesterol—740. Patient was diagnosed as a case of hypertriglyceridemia-induced acute pancreatitis. He was initially managed with IV fluids at 20 mL/kg over 1 hour followed by maintenance fluid, analgesics, IV ulinastatin, and was kept nil per oral.

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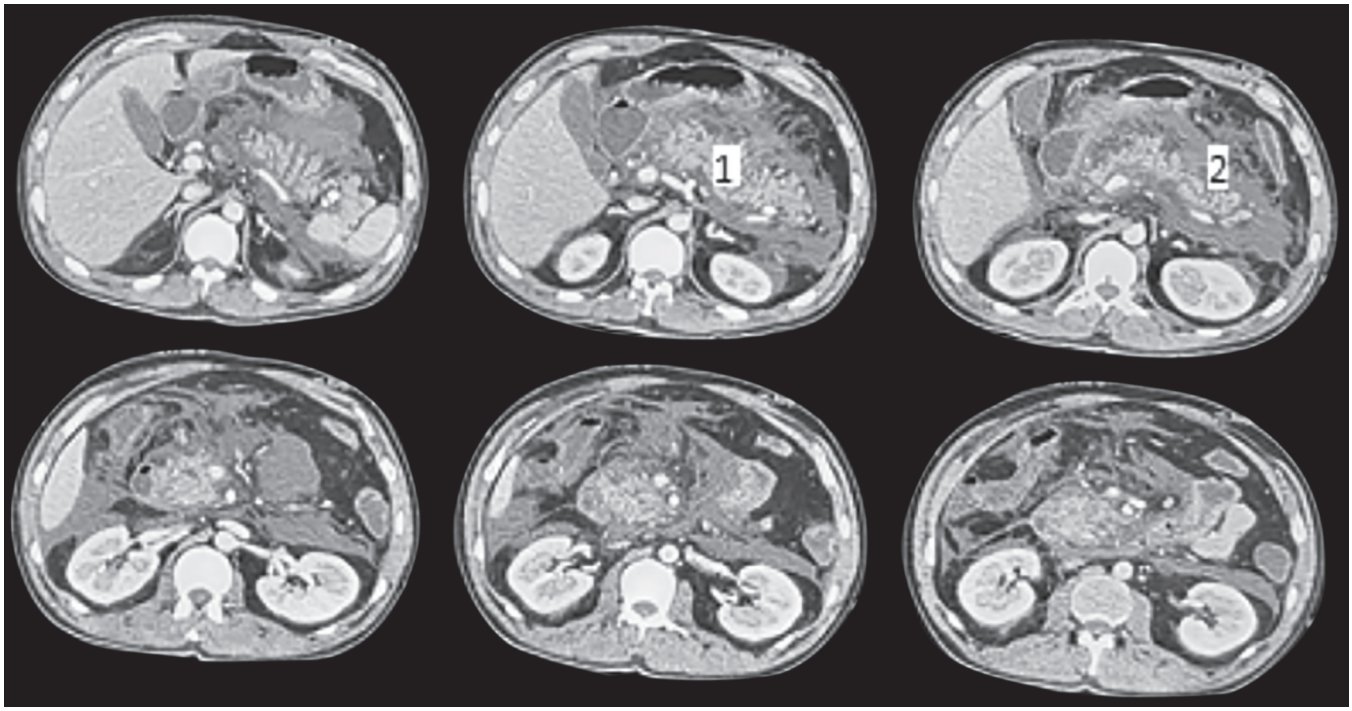


Fig. 1: CECT whole abdomen showing acute necrotizing pancreatitis. (1) Peripancreatic mesenteric fat necrosis; (2) Minimal ascites (modified CTSI—8)

- Euglycemic insulin therapy was started (at 0.25 units/kg/hour) with concurrent IV 25% dextrose infusion. CECT whole abdomen showed features of acute necrotizing pancreatitis with peripancreatic mesenteric fat necrosis and collection (modified CTSI—8) with minimal ascites (Fig. 1).
- Euglycemic insulin therapy was continued, and patient's triglyceride level now was 1575 mg/dL. He developed signs of increased inflammation as 104 beats/minute heart rate, respiratory rate >24 breaths/minute, and WBC count 15490 cells/µL. Plasmapheresis was initiated. Two sessions of plasmapheresis (2 L) were given on two consecutive days. Now, his triglyceride levels were lowered to 633 mg/dL and he was started on oral statin and fenofribrate, for long-term management of hypertriglyceridemia.

Discussion

This case demonstrates the successful treatment of a patient with acute severe pancreatitis due to the very high levels of triglyceride levels, being treated not only by conventional management for acute pancreatitis but also with euglycemic insulin therapy and plasmapheresis which is an unusual treatment in such ominous situation.

Apart from familial dyslipidemias (type I, IIB, IV, V), many secondary causes like obesity, metabolic syndrome, type 2 diabetes mellitus, alcohol consumption, pregnancy, etc., are the culprit for very high triglyceride levels leading to severe complications like acute pancreatitis.[4]

In hypertriglyceridemia-induced acute pancreatitis, with no signs of increasing inflammation or organ failure, intravenous insulin can be administered, to control hypertriglyceridemia. Insulin therapy is continued until triglyceride levels fall below 500 mg/dL. Intravenous (IV) infusion of regular insulin is given at a rate of 0.1–0.3 units/kg/hour while closely monitoring the blood glucose levels. Only side effect of this therapy is hypoglycemia which is prevented by concurrently infusing dextrose solution to maintain serum levels between 150 and 200 mg/dL. Insulin regimen has been reported to reduce triglyceride levels to less than 500 mg/dL (5.6 mmol/L) in 3.5–4 days.[5]

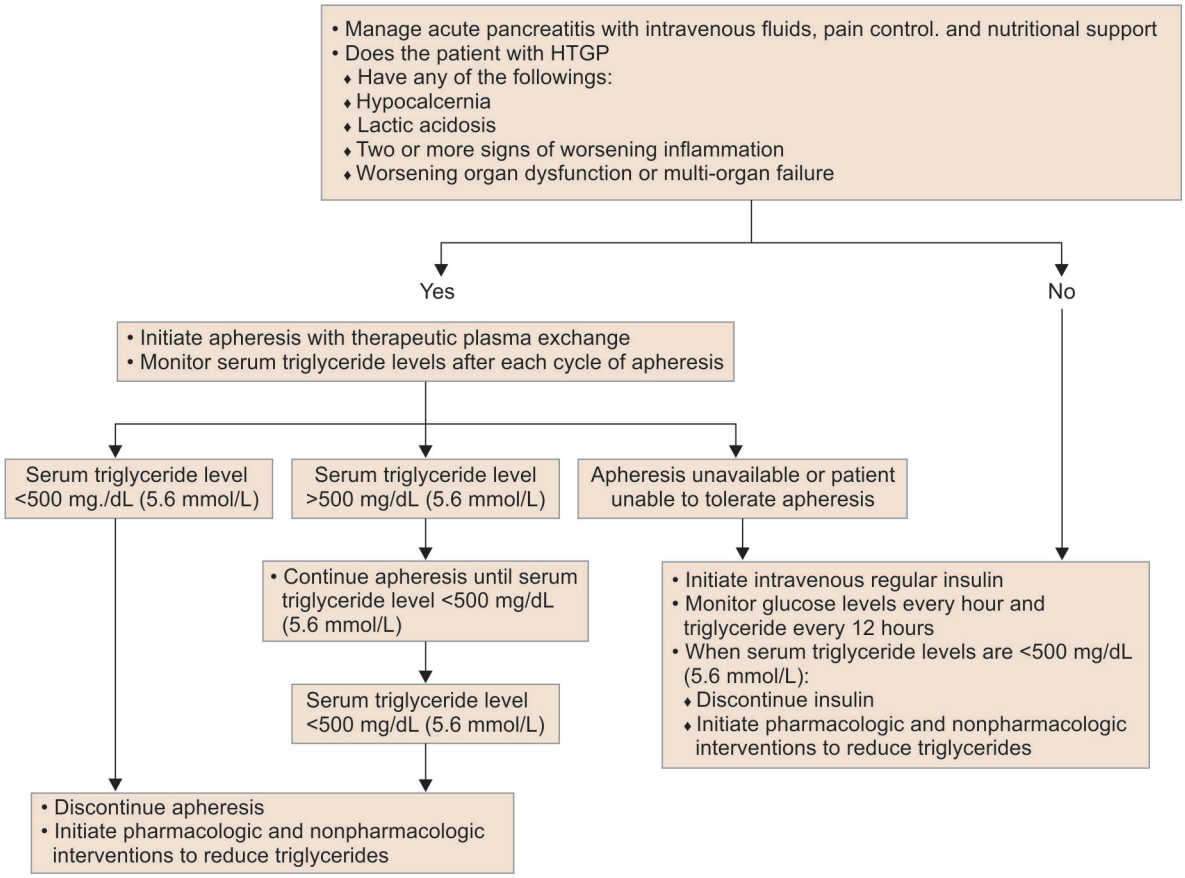
Therapeutic plasma exchange (TPE) is recommended in patients of hypertriglyceridemia-induced acute pancreatitis with very high serum triglyceride levels of >1000 mg/dL with serum lipase level >3 times the upper limit of normal. TPE is also recommended in patients with acute HTG induced pancreatitis with signs of hypocalcemia, lactic acidosis, or two or more signs of worsening inflammation (temp >38.5°C or <35.0°C; heart rate >90 beats/minute; respiratory rate >20 breaths/minute or $PaCO_2$ <32 mm Hg; WBC count >12000 <4000 or >10% immature band forms) or signs of worsening organ dysfunction or multiorgan failure as defined by Modified Marshall scoring system for organ dysfunction.[6,7]

Acute pancreatitis in patients with hypertriglyceridemia can be prevented by lowering the serum triglyceride level to 200 mg/dL (<2.2 mmol/L). Nonpharmacological interventions include weight loss in obese patients, aerobic exercises, avoid concentrated sugars, dietary fat restrictions. Pharmacologic therapy (fenofibrate, statins) and may be used as a long-term measure (Flowchart 1).[8]

Conclusion

In the case of severe pancreatitis not responding to treatment, a fasting lipid profile must be done. Lipid-lowering measures by insulin therapy and plasmapheresis coupled with long-term oral lipid lowering agents play a promising role. Chances of recurrence can be minimized by long-term follow-ups with blood lipid levels and keeping triglycerides below threshold by both pharmacological and nonpharmacological measures like diet and exercise.

Flowchart 1: Algorithm to treat hypertriglyceridemia-induced acute pancreatitis (Adapted from: https://painepodcast.com/2020/10/14/65-pancreatitis/)



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