

Hypertriglyceridemia-induced acute pancreatitis: Yet to be explored

Abstract

Hypertriglyceridemia is an important yet often missed cause of acute pancreatitis. The diagnostic dilemma and no specific guidelines for management of this condition further add to difficulty in treating a patient with hypertriglyceridemia induced acute pancreatitis. We present a thirty five year old, diabetic male patient with hypertriglyceridemia induced pancreatitis, enumerating the difficulties in diagnosis and treatment of our patient. A brief review of literature summarizes the various modalities of treatment proposed.

Key words: Acute pancreatitis, hypertriglyceridemia, plasmapheresis

INTRODUCTION

Acute pancreatitis is a condition with high morbidity and mortality and hypertriglyceridemia as the cause is often underestimated. Moreover, typical findings like raised lipase and amylase levels are not seen which further increases the diagnostic dilemma.^[1] We report a case of 35-year-old male patient with hypertriglyceridemia-induced acute pancreatitis who had atypical findings, making the management challenging.

CASE REPORT

A 35-year-old diabetic, nonalcoholic, male patient presented with complaints of nausea, vomiting, and severe abdominal pain for last 3 days. He had tachycardia, tachypnea but was hemodynamically stable. Investigations revealed hematocrit 44, white blood cell count 9800, and thrombocytopenia (platelet count: 27,000). Serum lipase level was 975 U, and serum amylase level was 112 U. Chest X-ray suggested mild bilateral pleural effusion and ultrasonography of the abdomen revealed edematous pancreatitis with minimal ascites. Arterial blood gas analysis showed no hypoxemia, but there was uncompensated metabolic acidosis with high anion gap. Renal function tests were deranged, blood sugars were high and urine tested positive for ketones. His serum calcium levels were too low (1.8 mg%), and serum phosphorus was only 0.2 mg%. The blood sample was repeated, and lipid profile was also done as per laboratory feedback of sample being lipemic in appearance. Serum triglyceride levels were very high (2572), very low-density lipoprotein (VLDL) and serum cholesterol were also raised. Bedside index of severity in acute pancreatitis score was 3, and APACHE 2 was calculated 14. Fluid resuscitation was initiated, and hematocrit and blood urea nitrogen were done sixth hourly. Insulin infusion was started to control blood glucose levels. No antibiotic was initially given as per our unit protocol, and fentanyl infusion was started at 50 mcg/h. Patient was administered fenofibrate 160 mg at night; dyselectrolytemia correction and fluid resuscitation were continued. Nasojejunal tube was placed endoscopically on day 2 of the presentation, and low volume feeding was initiated. We could not use heparin infusion in view of severe thrombocytopenia. Bilateral lower limb compression device was applied for deep vein thrombosis prophylaxis.

After 48 h, serum triglyceride levels decreased to 1804 and thrombocytopenia started to improve. Blood glucose levels were controlled, and serum calcium was also 6.9 mg%. On day 3, patient showed gross improvement and serum triglyceride levels decreased to 604. Contrast-enhanced computed tomography (CT) of the abdomen revealed acute edematous pancreatitis with CT severity index of 2. Patient passed flatus and bowel sounds could be appreciated. He was given clear liquids orally which he tolerated well. Platelet count increased to 1.3 lacs, his urine output was adequate, and he was shifted to the room in stable condition.

**Neeru Sahni,
Amit Kumar Mandal¹,
Namrata Maheshwari²**

Department of Anaesthesia and Intensive Care, PGIMER, Chandigarh, Departments of ¹Pulmonary Medicine and Critical Care and ²Critical Care, Fortis Hospital, Mohali, Punjab, India

Address for the Correspondence:

Dr. Neeru Sahni,
Department of Anaesthesia
and Intensive Care,
PGIMER, Chandigarh, India.
E-mail: neerunalin@yahoo.com

Access this article online

Website: www.oghr.org

DOI: 10.4103/2348-3113.152326

Quick response code:



DISCUSSION

In hypertriglyceridemia, triglyceride-rich chylomicrons block the circulatory flow in the pancreas which leads to swelling and hemorrhages in pancreas. The triglycerides hydrolyze to form free fatty acid molecules which then activate trypsinogen leading to acute pancreatitis.^[1] Diagnostic dilemma begins with not so markedly raised serum lipase and amylase levels, as also in our patient.^[2] Triglyceride >500 mg% also shows normal level of amylase due to *in vitro* interaction with a high level of lipid particles.^[2] The abnormally low levels of serum calcium and phosphorus were due to a highly lipemic sample in our patient. Various laboratory abnormalities expected in a lipemic sample are hemoglobin, blood sugar, serum phosphorus, calcium, total protein, and serum creatinine.^[3] Hence, one should be very cautious in the interpretation of these values when a patient is found to have hypertriglyceridemia.

Treatment of hypertriglyceridemia-induced pancreatitis is challenging due to lack of consensus or randomized controlled trials comparing efficacy of the suggested treatment modalities. The literature does not suggest any particular protocol to be followed for a patient with hypertriglyceridemia induced pancreatitis. Suggested treatment in acute setting is use of heparin, insulin infusion, plasmapheresis, and fibrates.

Heparin and insulin infusion enhance the activity of lipoprotein lipase and lower the level of circulating triglyceride. Intravenous insulin also decreases the production of both VLDL1 and VLDL2 from liver and intestine, respectively.^[4] However, there is no insulin infusion protocol suggested for lowering triglyceride levels. For heparin administration, the point of concern remains the lowering of activity of lipoprotein lipase after an initial peak. This happens due to degradation of this enzyme by liver.^[5] Hence, use of heparin infusion remains controversial in the treatment of hypertriglyceridemia-induced acute pancreatitis. In our patient, we used insulin infusion alone as heparin infusion was contraindicated due to persistent thrombocytopenia.

Plasmapheresis allows early clearance of triglyceride from patient's serum as well as supplements lipoprotein lipase and apolipoprotein from fresh frozen plasma of the donor plasma used during the procedure.^[6] Plasmapheresis also reduces proinflammatory cytokines decreasing the inflammation of pancreatic tissue.^[7]

A single plasmapheresis has been shown to remove about 66.3% of the triglyceride while a second exchange was shown to remove 83.3%

of the triglyceride.^[8] The number of sessions of plasmapheresis did not predict better outcome as also in another study by Syed *et al.*, about 89.3% reduction in triglyceride levels was observed with the first plasmapheresis itself.^[9] Bota reported good outcome when plasmapheresis and insulin infusion were combined together.^[10]

Despite rapid lowering of triglyceride level, the serious side effects of this procedure cannot be ignored. There is a high risk of anaphylactoid and/or transfusion-related infections.^[11] High cost, no randomized trials to prove its efficacy over other modalities and lack of easy availability restrict its use.

Hence, the diagnosis of this condition is associated with diagnostic difficulties due to absence of grossly elevated amylase and lipase levels, discrepancies in actual and measured laboratory parameters in view of highly lipemic sample along with lack of suspicion for this etiology. Despite diagnosis, there is no consensus on which treatment modality is best, and there are no guidelines till date advocating the use of plasmapheresis.

REFERENCES

1. Akay S, Ardic ME, Erkan N. Hypertriglyceridemia-induced acute pancreatitis following hyperlipidemic abdominal crisis. *Eurasian J Med* 2011;43:122-4.
2. Yadav D, Agarwal N, Pitchumoni CS. A critical evaluation of laboratory tests in acute pancreatitis. *Am J Gastroenterol* 2002;97:1309-18.
3. Brooks AM, Paisey RB, Waterson MJ, Smith JC. Diagnostic difficulties with a lipaemic blood sample. *BMJ* 2010;340:b5530.
4. Pavlic M, Xiao C, Szeto L, Patterson BW, Lewis GF. Insulin acutely inhibits intestinal lipoprotein secretion in humans in part by suppressing plasma free fatty acids. *Diabetes* 2010;59:580-7.
5. Neuger L, Vilaró S, Lopez-Iglesias C, Gupta J, Olivecrona T, Olivecrona G. Effects of heparin on the uptake of lipoprotein lipase in rat liver. *BMC Physiol* 2004;4:13.
6. Ewald N, Kloer HU. Severe hypertriglyceridemia: An indication for apheresis? *Atheroscler Suppl* 2009;10:49-52.
7. Stefanutti C, Labbadia G, Morozzi C. Severe hypertriglyceridemia-related acute pancreatitis: Myth or reality? *Ther Apher Dial* 2013;17:464-5.
8. Yeh JH, Chen JH, Chiu HC. Plasmapheresis for hyperlipidemic pancreatitis. *J Clin Apher* 2003;18:181-5.
9. Syed H, Bilusic M, Rhondla C, Tavarria A. Plasmapheresis in the treatment of hypertriglyceridemia-induced pancreatitis: A community hospital's experience. *J Clin Apher* 2010;25:229-34.
10. Bota VM. Urgent apheresis combined with insulin infusion in hypertriglyceridemia-induced acute pancreatitis. *Am J Emerg Med* 2013;31:452.e1-2.
11. Dodd RY. The risk of transfusion-transmitted infection. *N Engl J Med* 1992;327:419-21.

How to cite this article: Sahni N, Mandal AK, Maheshwari N. Hypertriglyceridemia-induced acute pancreatitis: Yet to be explored. *Onc Gas Hep Rep* 2015;4:91-2.
Source of Support: Nil, **Conflict of Interest:** None declared.