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CASE REPORT

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Acute pancreatitis secondary to moderate hypertriglyceridemia: A case report

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Key Clinical Message

It is crucial to remain vigilant about acute pancreatitis, even in cases with moderately elevated triglycerides. Triglycerides as a cause of acute pancreatitis must be considered even in the absence of other risk factors.

Abstract

Hypertriglyceridemia is one of the most common causes of acute pancreatitis (AP), with triglyceride levels greater than 1000 mg/dL being an established risk factor for AP. Cases of acute pancreatitis due to triglyceride levels less than 1000 mg/dL have rarely been reported in the literature. We report a case of a 26-year-old para-2, living-2 (P2L2) female who presented with epigastric pain and fever, with a serum triglyceride level of 579 mg/dL. A diagnosis of acute pancreatitis was made based on the clinical features and radiological findings, despite no identifiable risk factors. Hypertriglyceridemia was managed with no complications of acute pancreatitis during treatment.

KEYWORDS

acute pancreatitis, case report, hypertriglyceridemia, triglyceride

1 | INTRODUCTION

Hypertriglyceridemia is a common metabolic disorder encountered in clinical practice, characterized by elevated serum triglyceride (TG) levels. The widely accepted normal range of fasting triglyceride is less than 150 mg/dL.[1] As per The Endocrine Society 2010, hypertriglyceridemia is classified into the following categories in fasting condition as mild: 150–200 mg/dL (1.7–2.3 mmol/L), moderate: 200–999 mg/dL (2.3–11.2 mmol/L), severe: 1000–1999 mg/dL (11.2–22.4 mmol/L), and very severe: ≥2000 mg/dL (≥22.4 mmol/L).[2] When TG levels rise beyond 1000 mg/dL, the risk of acute pancreatitis increases markedly, making hypertriglyceridemia the third most common cause of acute pancreatitis[1] The complete understanding of how increased triglyceride levels result in acute pancreatitis remains unclear.

It has been hypothesized that the elevated levels of triglyceride-rich lipoproteins interacting with pancreatic lipase in the pancreatic capillaries cause the breakdown of triglycerides into free fatty acids and phospholipids into lysophosphatidylcholine.[3] Additionally, the hydrolysis of triglycerides by lipase induces the generation of toxic levels of free fatty acids and glycerol, thereby damaging the acini and capillaries and precipitating edema and hemorrhage.[4] Acute pancreatitis is one of the leading causes of hospitalization among gastrointestinal diseases. Although its diagnosis is simple, the major challenge lies in predicting its progression and outcome.[5]

The clinical diagnosis relies on a combination of history-taking, physical examination, and laboratory investigation. Abdominal pain radiating to the back, as well as high serum and urine levels of amylase, point toward acute pancreatitis.[6] The annual incidence of acute pancreatitis varies from 15.9 to 36.4 per 100,000 individuals, with its burden on healthcare resource utilization expected to rise shortly. Despite advancements in healthcare access, imaging tools, and treatments, acute pancreatitis continues to have significant morbidity and mortality. The overall mortality rate varies from 5% to 17% in severe cases and is about 1.5% in mild cases.[7] Herein, we report a rare case of a 26-year-old para-2, living-2 (P2L2) female with acute pancreatitis caused by moderate triglyceridemia with a triglyceride level of 579 mg/dL.

2 | CASE REPORT

2.1 | Clinical History

A 26-year-old para-2 living-2 (P2L2) female presented to the emergency department of our center with complaints of acute epigastric pain on and off for 4 days, radiating toward the back and associated with abdominal fullness. She also complained of fever on and off for 2 days, with a maximum temperature documented at 101.2 degrees Fahrenheit. She had a history of cervical spine surgery with a plate in situ 10 years back. She also gave a history of lower segment cesarean section (LSCS) and the placement of an intrauterine copper T 2 years ago after the second cesarean section. She is a non-vegetarian, non-alcoholic, non-smoker, and denies any substance abuse. Her menstrual cycles were regular, each lasting 28–30 days without dysmenorrhea. She was housewife and engaged in household chores. She didn't engage in physical activity and follow any exercise plan. Her daily diet included tea, biscuits, rice, lentils, vegetables, and meat. There was no history of diabetes or prediabetes, viral infections, abdominal trauma, or intake of any offending medications (like steroids or estrogen). Anticonvulsant, sulfonamides, and protease inhibitors autoimmune diseases, or any other surgical procedures. Her family history was not significant for pancreatitis, dyslipidemia, cardiovascular events, diabetes, gallstones, or autoimmune diseases.

2.2 | Examination

On examination, she was tachycardic with a heart rate of 116 beats per minute and normal blood pressure. The rest of her vital signs were within normal limits. She weighed 79 kg, and her height was 157 cm, with a BMI of 32. Her abdominal examination revealed abdominal distention with epigastric tenderness and sluggish bowel sounds.

2.3 | Investigations

An ECG was performed, which was found to be normal and ruled out the diagnosis of a cardiovascular accident. To rule out autoimmune diseases, serum antinuclear antibody test (ANA) was done, which was negative. Ultrasonography revealed marked hepatomegaly with grade III fatty infiltration, significant free fluid in the peritoneal cavity, copper-T in situ, and an enlarged pancreas measuring 13 cm in length, with peripancreatic fluid accumulation. USG findings ruled out common bile duct cholelithiasis, acute peritonitis, appendicitis, and abdominal aortic aneurysm. Her serum calcium level was low (8.1 mg/dL), which ruled hypercalcemia as a cause of pancreatitis. The chest X-ray and urine analysis were unremarkable, while the urine pregnancy test was insignificant.

Her laboratory parameters are shown in Table 1.

A Contrast-Enhanced Computed Tomography (CECT) abdomen was planned; however, a non-contrast CT was done due to low urine output and raised creatinine levels. The non-contract CT scan revealed a diffuse bulky pancreas measuring up to 43 mm at the body, with marked stranding and free fluid accumulation in the peripancreatic region, suggesting acute pancreatitis (Figure 1) with bilateral pleural effusions (Figure 2).

2.4 | Diagnosis and management

Based on the clinical findings, such as abdominal pain radiating to the back, elevated triglyceride, serum amylase, and lipase levels, along with positive CT findings, a diagnosis of acute pancreatitis was made. However, challenges arose while looking for the cause of pancreatitis and its severity.

Although the high triglyceride levels were not significant enough to directly cause the symptoms of pancreatitis, considering the absence of various other potential causes like alcohol or drug use, infections, and trauma, we had to explore moderately elevated triglycerides as a possible reason behind the symptoms and went ahead with its targeted treatment. We excluded various potential reasons for the elevated triglyceride levels.

Beginning with secondary factors, she showed no signs of endocrine disorders (treated or untreated

TABLE 1 Serum biochemical parameters at admission and 48 h after admission.

Parameters	Results at admission	Results at 48 h
Serum amylase	451	176
Serum lipase	603	276
Complete blood count	Total count: 16400	Total count: 9500
	Differential count	Differential count
	Neutrophils: 79	Neutrophils: 62
	Lymphocytes: 18	Lymphocytes:34
	Monocytes: 3	Monocytes:4
	Eosinophils: 0	Eosinophils: 0
	Basophils: 0	Basophils: 0
Hematocrit (%)	47	35
Serum hemoglobin (gm/dl)	14.3	11.8
Serum sodium (mmol/L)	133	145
Serum potassium (mmol/L)	4.1	3.6
Serum Urea (mg/dl)	89	46
Serum creatinine (mg/dl)	1.67	0.7
C-reactive protein	52	–
Serum total bilirubin (mg/dl)	2.1	1.7
Serum albumin (mg/dl)	3.4	3.2
Serum calcium	8.1	8.5
Corrected calcium	7.36	9.14
Serum aspartate aminotransferase	20	14
Serum alanine aminotransferase	50	24
Serum alkaline phosphatase	122	94
Prothrombin time (seconds)	16	13
Serum total cholesterol	200	188
Serum triglyceride level	579	367
Serum Low-density lipoprotein	72	68
Serum high-density lipoprotein	45	43

thyroid disorder or diabetes), was not under any medication, abstained from alcohol, was not pregnant, and had an unremarkable family history. Additionally, we eliminated possibilities of renal disease, liver disease, and autoimmune disorders. Apart from a high BMI, there were no other identifiable factors contributing to the elevated triglyceride levels. The severity was 2 according to the Bedside Index of Severity in Acute Pancreatitis (BISAP) score and 6 according to the CT severity index (CTSI) score, indicating acute moderate pancreatitis. However, the Ranson score was 1 at the time of admission, indicating acute mild pancreatitis and creating a dilemma in grading the severity of the disease.

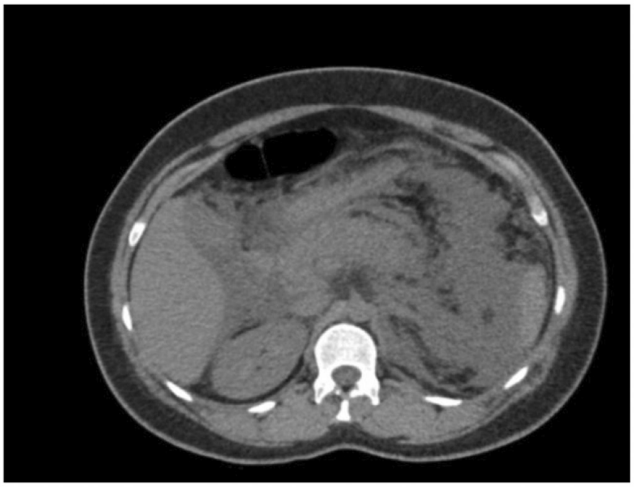

FIGURE 1 Non-contrast CT showing diffuse bulky pancreas and peripancreatic fluid with stranding.

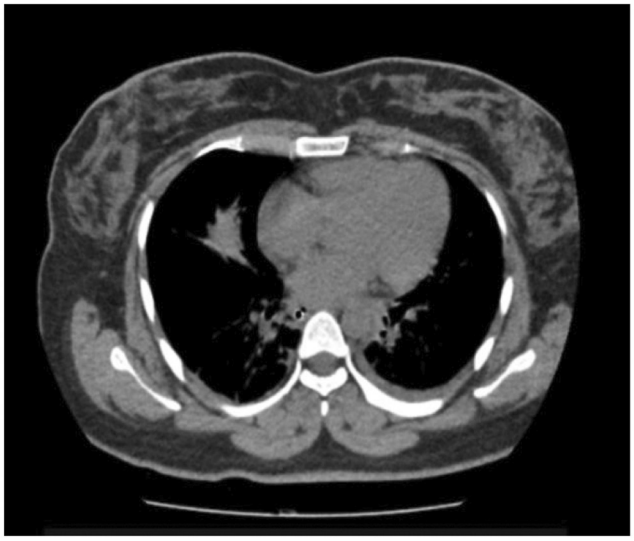

FIGURE 2 Non-contrast CT showing minimal pleural effusion.

The patient was transferred to an intensive care unit and initially managed with aggressive intravenous fluid resuscitation, fentanyl, and ondansetron. After collecting samples for blood cultures, empirical antibiotics such as piperacillin-tazobactam and metronidazole were started for high clinical suspicion of infection. Continuous insulin infusion decreased triglyceride levels to 367 mg/dL on the third day and 223 mg/dL on the seventh day. Fenofibrate, 160 mg once daily, was started to lower the triglyceride level further and prevent further episodes of pancreatitis. After 48 h, blood investigations were repeated, which revealed improvement. Empirical antibiotics were stopped as the blood culture reports were insignificant, and the patient was transferred from the ICU to the medical ward. She was discharged on the seventh day of admission, and her

vital signs were within normal limits at discharge. At the time of discharge, patient was counseled regarding aggressive lifestyle change, adherence to triglyceride-lowering medication, and with regular follow-up to prevent recurrent pancreatitis due to hypertriglyceridemia.

3 | DISCUSSION

High triglyceride levels can stem from primary factors in fewer than 5% of cases, often linked to genetic reasons. More commonly, however, hypertriglyceridemia is secondary to various factors such as diabetes, obesity, pregnancy, excessive carbohydrate intake, hypothyroidism, alcohol consumption, hepatitis, sepsis, renal failure, and specific medications, including estrogen, glucocorticoids, β blockers, bile acid binding resins, thiazides, tamoxifen, cyclosporine, protease inhibitors, and isotretinoin.[8] Hypertriglyceridemia is the third most common cause of acute pancreatitis, following alcohol and gallstones, and is classically considered a risk factor only when its levels are higher than 1000 mg/dL.[1,9] However, the case described above alerts us to the possibility of acute pancreatitis as a sequela of moderate hypertriglyceridemia.

Pancreatitis secondary to hypertriglyceridemia can sometimes be suspected during physical examination by detecting eruptive xanthomas or lipemia retinalis. Significant increases in triglyceride levels can lead to falsely low serum amylase and lipase, potentially necessitating reliance on pancreatic CT scans for diagnosis.[3]

Early management of acute pancreatitis and prevention of its complications are the mainstay of treatment. Initiation of conservative treatment, including aggressive intravenous hydration, initial bowel rest, and pain control, needs to be done soon after the diagnosis is suspected. In addition, several treatment modalities like insulin and heparin, plasmapheresis, combined blood purification therapy (CBPT), high-volume hemofiltration (HVHF), and hemoperfusion (HP) have been described for the targeted treatment of hypertriglyceridemic pancreatitis.[10]

Insulin infusion has been used to lower the triglyceride lever for more than a decade. Insulin lowers triglycerides levels by 50%–75% over 2–3 days.[10] However, frequent blood glucose levels should be checked to prevent hypoglycemia. Plasmapheresis rapidly removes triglycerides and chylomicron form the circulation removing the inciting factor and stoping further inflammation and damage to pancreas. It is reported that plasmapheresis lower triglycerides levels by 50%–80% in each session.[10]

Patients with severe hypertriglyceridemia-induced pancreatitis (APACHE II score ≥8, Balthazar grade D/E, multiple organ dysfunction, Marshalls criteria >2) should be treated more aggressively with plasmapheresis with albumin as a replacement fluid. Plasmapheresis should be stated within 36 h. Potential complications of plasmapheresis are infections and allergic reaction to donor plasma.[10]

If plasmapheresis is not availabe or the patient cannot tolerate plasmapheresis, alternative non-pharmacological therapy like high-volume hemofiltration and hemoperfusion can be considered. Nonpharmacological triglyceride-lowering treatment should be continued until triglyceride levels <500 mg/dL. If the patient is able to tolerate oral intake oral lipid-lowering therapy should be started.[10]

An observational study published by Nawaz et al. prospectively enrolled acute pancreatitis patients and categorized them into mild, moderate, and severe based on serum triglyceride levels. The study concluded that elevated serum triglycerides are independently associated with the development of complications like persistent organ failure, regardless of the underlying etiology of acute pancreatitis. Thus, targeting triglyceride-induced lipotoxicity could present an appealing approach for creating new interventions to treat acute pancreatitis.[11]

Zhang et al.[12] retrospective analysis showed that TG levels ≥500 mg/dL should raise a high degree of suspicion, particularly in cases where there is no other obvious cause of acute pancreatitis. There seems to be a direct correlation between the severity of hypertriglyceridemia, pancreatitis, and triglyceride levels. In prospective cohort research, Pedersen et al.[13] found that mild-to-moderate nonfasting hypertriglyceridemia is associated with a high risk of acute pancreatitis, starting at 177 mg/dL (2 mmol/L) with hazard risk estimates greater than myocardial infarction.

A similar case report with acute pancreatitis due to moderate hypertriglyceridemia was published by Atlani et al.[14] in 2023. In contrast to our case report, moderate hypertriglyceridemia is associated with systemic lupus erythematosus, and the disease progresses to severe hypertriglyceridemia during the course of illness.

4 | CONCLUSION

This case highlights the significance of considering moderately elevated triglycerides as a potential cause of acute pancreatitis, emphasizing the necessity for vigilance in diagnosis, especially in the absence of other risk factors. It prompts a re-evaluation of the thresholds for triglyceride levels that might precipitate pancreatitis and emphasizes the need for vigilance, even in moderately elevated triglycerides. More case reports and observational studies are warranted to understand the elevations in triglyceride levels as a causative factor for acute pancreatitis.

AUTHOR CONTRIBUTIONS

Anil Nepali: Conceptualization; data curation; supervision; validation; writing – original draft; writing – review and editing. **Satyam Kharga:** Conceptualization; data curation; supervision; validation; writing – original draft; writing – review and editing. **Malavika Jayan:** Conceptualization; supervision; validation; writing – original draft; writing – review and editing. **Prakriti Adhikari:** Conceptualization; data curation; supervision; writing – original draft; writing – review and editing. **Amit Shah:** Conceptualization; data curation; supervision; validation; writing – original draft; writing – review and editing. **Vivek Sanker:** Conceptualization; supervision; writing – original draft; writing – review and editing.

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None.

CONFLICT OF INTEREST STATEMENT

None declared.

DATA AVAILABILITY STATEMENT

The data supporting this article's findings are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

Ethical approval was not required for the case report per the country's guidelines.

CONSENT

Written informed consent was obtained from the patient to publish this report.

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