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Severe Acute Pancreatitis Triggered by Hypertriglyceridemia: Case Report

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Introduction

After gallstones and alcohol abuse, severe hypertriglyceridemia (HTG), defined as serum triglyceride levels exceeding 1,000 mg/dL (10 g/L), ranks as the third most common etiology of acute pancreatitis (AP). However, it remains a rare cause, accounting for only 1–2% of cases [1,3].

Recognizing hypertriglyceridemia as an underlying cause is crucial, as it represents a medical emergency with potentially severe outcomes if not promptly managed.

We report here the case of a patient admitted to our institution with severe acute pancreatitis secondary to hypertriglyceridemia.

Case Presentation

We report the case of Ms. M.E., a 46-year-old woman with a medical history of obesity and hypertension, treated with calcium channel blockers for the past three years. She presented to the emergency department with acute epigastric pain radiating to the back. The pain was initially postprandial but became constant, and was associated with nausea and vomiting. She was afebrile and in preserved general condition.

On admission, the patient was alert and oriented, afebrile, with a capillary blood glucose level of 1.9 g/L and a pain score (VAS) of 5. Physical examination revealed a distended abdomen with marked tenderness in the epigastric region. Respiratory rate was 24 breaths per minute without signs of respiratory distress. Oxygen saturation was 93% on room air and 99% with 3 L/min of supplemental oxygen. The patient was tachycardic with a heart rate of 110 bpm, normal blood pressure, and preserved urine output.

Initial laboratory workup showed elevated serum lipase at 338 IU/L, leukocytosis (WBC 11,100/µL), hemoglobin of 11.9 g/dL, and markedly elevated triglycerides at 10.8 g/L. HDL cholesterol was 0.45 g/L, and total cholesterol was 5.42 g/L. Liver and renal function tests, as well as serum electrolytes, were within normal limits. Abdominal ultrasound showed no evidence of biliary lithiasis or other potential causes of acute pancreatitis.

An abdominal CT scan performed 48 hours after symptom onset revealed severe acute pancreatitis, classified as grade E according to the Balthazar score, with a CTSI (Computed Tomography Severity Index) score of 4. No obstructive lesion was identified.

The patient was admitted to the intensive care unit for further management. The BISAP (Bedside Index for Severity in Acute Pancreatitis) score was 2, based on the presence of systemic inflammatory response syndrome (SIRS) and a pleural effusion, while age, mental status, serum urea level, and hemodynamics were within normal ranges. Endoscopic ultrasound later ruled out biliary etiology definitively.

Initial management included stabilization with non-invasive monitoring, aggressive intravenous hydration with 0.9% saline at 10 mL/kg/h, and fasting with initiation of parenteral nutrition. The patient received multimodal analgesia, full-dose statin therapy for lipid lowering, and respiratory physiotherapy including the use of an incentive spirometer.

The clinical course was favorable, with resolution of pain, epigastric tenderness, ileus, and vomiting, allowing for the progressive reintroduction of enteral nutrition. Biologically, there was significant improvement: white blood cell count decreased to 6,800/µL, serum lipase dropped from 338 to 78 IU/L, and triglyceride levels normalized to 2.6 g/L.

The patient remained in the ICU for 10 days. Given her clinical and biological improvement, she was subsequently transferred to the gastroenterology department for further management, including dietary counseling and lipid-lowering therapy.

Discussion

Severe hypertriglyceridemia (HTG) is responsible for approximately 1–2% of all acute pancreatitis (AP) cases [1,3]. The causes of HTG can be broadly classified as either primary (genetic) or secondary (acquired). Primary causes include genetic disorders such as familial chylomicronemia syndrome and mixed hyperlipidemias (Frederickson types I, IV, and V), whereas secondary causes encompass obesity, diabetes mellitus, certain medications (e.g., estrogens), pregnancy, and excessive alcohol consumption [4–6]. It is important to note that primary causes often result in more severe hypertriglyceridemia, and the coexistence of primary and secondary factors may exacerbate disease severity [7].

The exact pathophysiological mechanism linking HTG to AP remains incompletely understood. Experimental models have proposed several non-mutually exclusive hypotheses. One suggests that pancreatic lipase hydrolyzes the excess circulating triglycerides, generating high concentrations of free fatty acids that exert a direct cytotoxic effect on pancreatic acinar cells. Another theory involves pancreatic ischemia induced by increased blood viscosity due to chylomicron accumulation in pancreatic capillaries. More recent genetic investigations have added complexity: a Chinese study identified mutations in the CFTR gene (Cystic Fibrosis Transmembrane Conductance Regulator) in 26.1% of patients with HTG-associated AP, compared to only 1.3% in those with HTG without AP [8]. Similarly, a Spanish study linked mutations in the ApoE gene with HTG-induced AP [9]. These findings support a multifactorial etiology with genetic and metabolic interplay influencing individual susceptibility.

The clinical presentation of HTG-induced AP is indistinguishable from AP due to biliary or alcoholic causes. Diagnosis typically requires two of the following three criteria: characteristic epigastric pain radiating to the back, serum lipase elevation exceeding three times the upper limit of normal, and radiologic evidence of pancreatic inflammation. Dermatological signs such as eruptive xanthomas or palmar xanthomas may be seen in cases of very high triglyceride levels. Early measurement of serum triglycerides is essential, as levels can rapidly decline once fasting is initiated [7]. A triglyceride concentration >1,000 mg/dL (10 g/L) is generally considered diagnostic for HTG as the primary etiology of AP [10].

In a study involving 256 patients with HTG-induced AP between 2016 and 2018, serum triglyceride levels on admission were found to correlate with disease severity [11–12]. A 2017 meta-analysis involving over 1,000 patients with HTG-induced AP and more than 5,000 with AP of other etiologies confirmed this association and linked elevated TG levels with poorer clinical outcomes [12].

A French study showed that HTG-induced AP tends to be more severe than AP caused by alcohol or biliary disease. In that cohort, 71.5% of patients presented with severe AP, defined by CRP >150 mg/L, a Balthazar score >C, and ICU admission [13]. Similarly, our patient had a severe AP with CRP >300 mg/L, a Balthazar grade E, and required intensive care for over seven days. The same meta-analysis also reported higher rates of acute kidney injury, respiratory failure, and systemic inflammatory response syndrome (SIRS) in patients with HTG-induced AP. In our case, the patient developed early respiratory compromise, which was successfully managed with oxygen therapy and respiratory physiotherapy.

From a therapeutic standpoint, the initial management of HTG-induced AP mirrors that of AP from other etiologies and includes three main strategies: aggressive intravenous hydration, adequate nutritional support, and multimodal pain control. In cases of major HTG (>10 g/L), stricter dietary lipid restriction and metabolic monitoring are warranted. Therapies aimed at enhancing lipoprotein lipase (LPL) activity, such as insulin and heparin, have been described [14].

Heparin transiently increases LPL activity by mobilizing it from endothelial surfaces. However, this effect is limited, particularly in patients with homozygous LPL mutations. Moreover, due to the risk of rebound hypertriglyceridemia and potential hemorrhagic complications in pancreatitis, heparin is generally discouraged [15]. Insulin, on the other hand, promotes LPL activity in muscle and adipose tissues and is considered more effective. Although there are no consensus guidelines, a 2018 study proposed an insulin infusion protocol at 0.1–0.3 IU/kg/h to lower TG levels, regardless of diabetic status, with hourly glucose monitoring. Insulin can reduce TG levels by 50–75% within 2 to 3 days [7,16].

In severe forms of AP (e.g., APACHE II >7, Balthazar score D–E, or organ dysfunction), more aggressive measures may be needed. Non-pharmacological interventions such as plasmapheresis, combined blood purification therapy (CBPT), and hemofiltration have been used to rapidly and effectively lower triglyceride and chylomicron levels, thereby mitigating the inflammatory cascade in the pancreas [17–18].

Conclusion

In conclusion, hypertriglyceridemia has emerged as an increasingly recognized and potentially life-threatening cause of acute pancreatitis, particularly in severe forms. Prompt identification and appropriate management are essential to improve patient outcomes.

A multidisciplinary approach is crucial, involving emergency physicians, gastroenterologists, radiologists, and anesthesiologist-intensivists. Long-term follow-up is also necessary to address metabolic control, lifestyle modification, and dietary management to prevent recurrence and associated complications.

Reference

1. M.R.Fortson,S.N.Freedman,andP.D.Webster,“3rd.Clinical assessment of hyperlipidemic pancreatitis,”The American Jour- nal of Gastroenterology, vol. 90, pp. 2134–2139, 1995

2. 2. W. Tsuang, U. Navaneethan, L. Ruiz, J. B. Palascak, and A. Gelrud, “Hypertriglyceridemic pancreatitis: presentation and management,” American Journal of Gastroenterology, vol. 104, no. 4, pp. 984–991,2009.

3. P. Valdivielso, A. Ramirez-Bueno, and N. Ewald, "Current knowledge of hypertriglyceridemic pancreatitis," European Journal of Internal Medicine, vol. 25, no. 8, pp. 689–694, 2014.
4. Yadav D, Pitchumoni CS. Issues in hyperlipidemic pancreatitis. J Clin Gastro- enterol 2003;36:54–62.
5. C. L. Linares, A. L. Pelletier, S. Czernichow et al., "Acute pancreatitis in a cohort of 129 patients referred for severe hypertriglyceridemia," Pancreas, vol. 37, no. 1, pp. 13–22, 2008.
6. S. Sandhu, A. Al-Sarraf, C. Taraboanta, J. Frohlich, and G. A. Francis, "Incidence of pancreatitis, secondary causes, and treatment of patients referred to a specialty lipid clinic with severe hypertriglyceridemia: a retrospective cohort study," Lipids in Health andDisease, vol. 10, article 157, 2011.
7. Garg, R., & Rustagi, T. (2018). Management of Hypertriglyceridemia Induced Acute Pancreatitis. BioMed Research International, 2018, 1–12.doi:10.1155/2018/4721357
8. Chang YT, Chang MC, Su TC, et al. Association of cystic fibrosis transmembrane conductance regulator (CFTR) mutation/variant/haplotype and tumor necrosis factor (TNF) promoter polymorphism in hyperlipidemic pancreatitis. Clin Chem 2008;54:131–8.
9. Ivanova R, Puerta S, Garrido A, et al. Triglyceride levels and apolipoprotein E polymorphism in patients with acute pancreatitis. Hepatobiliary Pancreat Dis Int 2012;11:96– 101.
10. Heaney AP, Sharer N, Rameh B, et al. Prevention of recurrent pancreatitis in familial lipoprotein lipase deficiency with high dose antioxidant therapy. J Clin Endocrinol Metab 1999;84:1203-5.
11. Lu Z, Zhang G, Guo F, et al. Elevated triglycerides on admission positively correlate with the severity of hypertriglyceridaemic pancreatitis. Int J Clin Pract 2020;74:e13458.
12. Wang Q, Wang G, Qiu Z, et al. Elevated serum triglycerides in the prognostic assessment of acute pancreatitis: a systematic review and meta-analysis of observational studies. J Clin Gastroenterol 2017;51:586–93.
13. Lloret Linares C, Pelletier AL, Czernichow S, et al. Acute pancreatitis in a cohort of 129 patients referred for severe hypertriglyceridemia. Pancreas 2008;37:13–22.
14. Dubois, C., & Vergès, B. (2020). Les hypertriglycéridémies à l'origine de pancréatite aiguë. Médecine Des Maladies Métaboliques. doi:10.1016/j.mmm.2020.06.001
15. P. Jain, R. R. Rai, H. Udawat, S. Nijhawan, and A. Mathur, "Insulin and heparin in treatment of hypertriglyceridemia- induced pancreatitis," World Journal of Gastroenterology, vol. 13, no. 18, pp. 2642-2643, 2007.
16. A. Coskun, N. Erkan, S. Yakan et al., "Treatment of hypertriglyceridemia-induced acute pancreatitis with insulin," Przegląd Gastroenterologiczny, vol. 10, no. 1, pp. 18–22, 2015.
17. C. Stefanutti, G. Labbadia, and C. Morozzi, "Severe Hypertri- glyceridemia-Related Acute Pancreatitis," Therapeutic Apheresis and Dialysis, vol. 17, no. 2, pp. 130–137, 2013.
18. H.-L.WangandK.-J.Yu,"Sequentialbloodpurificationtherapy for critical patients with hyperlipidemic severe acute pancreati- tis," World Journal of Gastroenterology, vol. 21, no. 20, pp. 6304– 6309, 2015.