

Case Report

Open Access, Volume 5

Role of low-dose insulin infusion in hypertriglyceridemia-induced acute pancreatitis in a patient with type 2 diabetes mellitus: A case report

Haromi Licdan^{1*}; Rene Xavier Manalo^{1,2}

¹Department of Internal Medicine, Saint Louis University – Sacred Heart Medical Center, Baguio City, Philippines.

²Department of Medicine, Notre Dame de Chartres Hospital, Baguio City, Philippines.

***Corresponding Author: Haromi Licdan**

Department of Internal Medicine, Saint Louis University
– Sacred Heart Medical Center, Baguio City, Philippines.
Email: haromi.licdan@yahoo.com.ph

Received: Jun 10, 2025

Accepted: Jul 11, 2025

Published: Jul 18, 2025

Archived: www.jjgastro.com

Copyright: © Licdan H (2025).

Keywords: Hypertriglyceridemia; Acute pancreatitis; Low-dose insulin therapy; Lipid-lowering treatment; Diabetes mellitus.

Abstract

Background: Hypertriglyceridemia is a recognized but uncommon cause of acute pancreatitis, accounting for up to 10% of cases. Management focuses on supportive care and rapid reduction of triglyceride levels. While insulin infusion is one treatment modality, the use of low-dose insulin protocols remains underreported. **Case presentation:** We report the case of a 37-year-old Filipino female with a history of type 2 diabetes mellitus and hypertriglyceridemia who presented with acute epigastric pain following alcohol intake. Laboratory tests revealed elevated lipase and triglyceride levels (>3,000 mg/dL). She was diagnosed with acute pancreatitis of mixed etiology (alcohol and hypertriglyceridemia). Management included bowel rest, fluid resuscitation, analgesia, and a low-dose insulin infusion at 0.05 units/kg/hour. Serum triglyceride levels decreased to 95 mg/dL within 24 hours, and the patient was discharged with lipid-lowering therapy and lifestyle counseling. **Conclusion:** This case demonstrates that low-dose insulin infusion is a safe and effective option for rapidly lowering triglyceride levels in HTG-AP, particularly in resource-limited settings where plasmapheresis may not be available.

Background

Hypertriglyceridemia-Induced Acute Pancreatitis (HTG-AP) is a well-recognized but less common cause of acute pancreatitis, accounting for approximately 4%-10% of cases, with a rising incidence in parallel with global increases in obesity and metabolic syndrome [1]. HTG-AP typically occurs when serum triglyceride levels exceed 1,000 mg/dL, with significantly increased risk as levels surpass 2,000 mg/dL [2]. The proposed pathophysiology involves the hydrolysis of triglycerides by pancreatic lipase, leading to the release of excessive free fatty acids, which are directly toxic to pancreatic acinar cells and vascular endothelium, triggering inflammation and tissue injury [3].

Management of HTG-AP includes standard supportive measures—such as fluid resuscitation, pain control, and nutritional support—as well as targeted reduction of triglyceride levels. Among triglyceride-lowering therapies, insulin infusion has emerged as an accessible and effective approach. Insulin enhances Lipoprotein Lipase (LPL) activity, accelerating the metabolism of chylomicrons and Very-Low-Density Lipoproteins (VLDL), thereby reducing circulating triglyceride levels [4]. While plasmapheresis can rapidly lower triglycerides, it is costly and not universally available [5]. Although high-dose insulin regimens are commonly used, recent studies suggest that low-dose insulin infusions may offer comparable efficacy with a lower risk of hypoglycemia and other metabolic complications [6].

Citation: Licdan H, Manalo RX. Role of low-dose insulin infusion in hypertriglyceridemia-induced acute pancreatitis in a patient with type 2 diabetes mellitus: A case report. *J Gastroenterol Res Pract.* 2025; 5(3): 1233.

This case report highlights the successful management of HTG-AP using a low-dose insulin infusion protocol, underscoring its potential as a safe, effective, and resource-conscious alternative.

Case presentation

We present a 37-year-old Filipino female housewife with a two-day history of crampy epigastric pain that began following the intake of approximately one bottle of gin during a drinking session. The pain evolved into a severe, boring epigastric discomfort radiating to the back. Two hours prior to hospital consult, the pain was accompanied by nausea and multiple episodes of non-bloody, non-bilious vomiting.

The patient had a prior diagnosis of type 2 diabetes mellitus and hypertriglyceridemia since five years prior to admission for which she was prescribed metformin and fenofibrate. She reported adherence to both medications until she self-discontinued her medications one year prior to admission. Her family history was notable for hypertension, dyslipidemia, and hypertriglyceridemia on the maternal side, and a strong history of type 2 diabetes on the paternal side. She denied smoking and reported occasional alcohol consumption, limited to 1-2 glasses of gin per session on an average of 1-2 occasions per month.

On initial examination, the patient was alert, oriented, and ambulatory with assistance, though in mild cardiopulmonary distress. Her vital signs revealed a heart rate of 102 beats per minute, with all other parameters within normal limits. Her Body Mass Index (BMI) was 27.3 kg/m² (weight: 61 kg, height: 149 cm). Abdominal examination revealed direct and rebound tenderness in the epigastric region, more pronounced on the left side. Examination of other systems was unremarkable.

Initial laboratory evaluation showed leukocytosis with a white blood cell count of 12,000/ μ L (neutrophils 72%, lymphocytes 21%, monocytes 1%), normal hemoglobin (13.9 g/dL), hematocrit (37%), and platelet count (346,000/ μ L). Capillary blood glucose was 137 mg/dL. Serum lipase was significantly elevated at 371.26 U/L (approximately 6.2 times the upper limit of normal), while alanine aminotransferase (ALT) was mildly increased at 35.33 U/L. Renal function was within normal limits (BUN 8.12 mg/dL, creatinine 0.36 mg/dL, eGFR 134 mL/min/1.73 m²). Electrolytes, including sodium (136 mEq/L), potassium (3.9 mEq/L), and ionized calcium (1.21 mmol/L), were normal.

Lipid profile revealed markedly elevated triglyceride levels (3,254.15 mg/dL), total cholesterol (432.61 mg/dL), and Low-Density Lipoprotein Cholesterol (LDL-C: 231.19 mg/dL). Chest radiograph was unremarkable. Hepatobiliary ultrasound showed mild diffuse fatty infiltration of the liver but no abnormalities in the pancreas or gallbladder.

A diagnosis of acute pancreatitis of mixed etiology (alcohol and hypertriglyceridemia-induced) was made. The patient's BISAP score was 0. She was managed with bowel rest (NPO) and fluid resuscitation using lactated Ringer's solution (initial bolus at 20 mL/kg), alongside analgesics for pain control. Hypertriglyceridemia was addressed based on the 2012 Endocrine Society Clinical Practice Guidelines with a continuous low-dose insulin infusion at 0.05 units/kg/hour. Blood glucose

was monitored hourly, while serum triglycerides, potassium, hematocrit, and BUN were monitored every 12 hours.

After 24 hours of insulin infusion, serum triglyceride levels normalized to 95 mg/dL, allowing discontinuation of the insulin drip. The patient's symptoms improved, and she was subsequently discharged with appropriate lipid-lowering medications and dietary and lifestyle counseling.

Discussion

Hypertriglyceridemia-Induced Acute Pancreatitis (HTG-AP) is most commonly seen in patients with poorly controlled diabetes, metabolic syndrome, or genetic lipid disorders. Triglyceride levels above 1,000 mg/dL significantly increase the risk of pancreatitis, particularly when levels exceed 2,000 mg/dL [2]. In our case, the patient presented with severe hypertriglyceridemia (>3,000 mg/dL), likely exacerbated by alcohol intake and discontinuation of lipid-lowering therapy.

The pathogenesis of HTG-AP is thought to involve excessive hydrolysis of triglycerides by pancreatic lipase, releasing free fatty acids that damage pancreatic acinar cells and the microvascular endothelium [3]. Management strategies aim not only to support pancreatic recovery but also to reduce circulating triglycerides quickly to prevent further pancreatic injury.

While plasmapheresis has demonstrated efficacy in rapidly lowering triglyceride levels, it is often inaccessible due to its cost and limited availability [5]. Insulin therapy, on the other hand, offers a more widely available alternative by stimulating Lipoprotein Lipase (LPL), thereby enhancing chylomicron clearance [4]. Most reported cases use high-dose insulin, but this approach carries risks such as hypoglycemia and hypokalemia.

In our patient, low-dose insulin at 0.05 units/kg/hour successfully reduced serum triglycerides from >3,000 mg/dL to <100 mg/dL within 24 hours without significant adverse effects. This approach aligns with the Endocrine Society's 2012 Clinical Practice Guidelines, which recommend insulin as a first-line therapy for HTG-AP in diabetic patients. It offers a safer and potentially more practical method, particularly in low-resource settings or in patients at higher risk for insulin-related complications.

This case reinforces the effectiveness of low-dose insulin infusion in treating HTG-AP and suggests that it can be a viable alternative to plasmapheresis or higher-dose insulin protocols.

Conclusion

This case highlights the successful use of low-dose insulin infusion in the management of hypertriglyceridemia-induced acute pancreatitis in a diabetic patient. With triglyceride levels exceeding 3,000 mg/dL, timely and effective reduction of serum triglycerides was achieved within 24 hours using an insulin infusion at 0.05 units/kg/hour, without the need for plasmapheresis. This approach underscores the therapeutic value of low-dose insulin as a safe, accessible, and cost-effective option for managing HTG-AP, especially in resource-limited settings or in patients at increased risk of insulin-related complications. Clinicians should consider this strategy in similar cases to improve outcomes while minimizing treatment-related risks.

Author declarations

Statement of ethics: Ethical approval is not required for this study in accordance with local or national guidelines.

Informed consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in- Chief of this journal.

Conflict of interest statement: The authors have no conflicts of interest to declare.

Funding sources: This study was not supported by any sponsor or funder.

Author contributions: H.L. was primarily involved in the data collection, literature review, patient care, and drafting the initial manuscript. R.X. was responsible for patient care while providing clinical expertise and supervised data analysis, and significantly revised the manuscript. Both authors reviewed and approved the final manuscript.

Data availability statement: The data supporting the findings of this case report are available upon reasonable request from the corresponding author due to patient privacy concerns.

Acknowledgement: I extend my heartfelt gratitude to my institution, Saint Louis University Sacred Heart Medical Center, our training corps and consultants, and my co-residents from the Department of Internal Medicine for their invaluable guidance and support throughout this case report.

References

1. Li X, Ke L, Dong W, Liu X, Li W, Yang Y. Trends in hypertriglyceridemia-induced acute pancreatitis: A 10-year retrospective study in China. *World J Gastroenterol.* 2024; 30: 3996–4007.
2. Scherer J, Singh VP, Pitchumoni CS, Yadav D. Issues in hypertriglyceridemic pancreatitis: An update. *J Clin Gastroenterol.* 2021; 55: 488–496.
3. Carr RA, Rejowski BJ, Cote GA, Pitt HA. Systematic review of hypertriglyceridemia-induced pancreatitis: A more virulent etiology? *Pancreatology.* 2016; 16: 469–476.
4. Kota SK, Jammula S, Kota SK, Meher LK, Modi KD. Hypertriglyceridemia-induced recurrent acute pancreatitis: A case-based review. *Indian J Endocrinol Metab.* 2012; 16: 141–143.
5. Chen JH, Yeh JH, Lai HW, Liao CS. Therapeutic plasma exchange in patients with hyperlipidemic pancreatitis. *World J Gastroenterol.* 2020; 26: 3801–3810.
6. Rawla P, Bandaru SS, Vellipuram AR. Hypertriglyceridemia-induced pancreatitis: Updated review of current treatment and preventive strategies. *Clin J Gastroenterol.* 2017; 10: 537–548.