Case Report

Estrogen-Induced Severe Hypertriglyceridemia Presenting as Recurrent Acute Pancreatitis Successfully Managed with Intravenous Insulin Infusion

Salma Salman[1], Sabiha Banu[2], Nazish Fatima[3], Araj Jamil[4]

ABSTRACT

Introduction: Hypertriglyceridemia is an underrecognized cause of acute pancreatitis, especially in reproductive-age females without obvious risk factors. Careful history taking is essential, particularly drug history. Estrogen use, often for menstrual irregularities, may be over looked but can significantly elevate triglyceride levels and precipitate pancreatitis, warranting targeted clinical inquiry.

Case Presentation: We report a 26-year-old female admitted to the Medical ICU with severe abdominal pain who was diagnosed with acute pancreatitis. Laboratory evaluation revealed markedly elevated triglyceride levels of 4694 mg/dL. She had a history of menstrual irregularities, hirsutism, and recent use of oral estradiol for menstrual period regulation, secondary to suspected underlying polyendocrine metabolic ovarian syndrome. The patient was managed in the intensive care unit with continuous intravenous insulin infusion, resulting in a rapid decline in triglyceride levels to 640 mg/dL within 24 hours, along with significant clinical improvement. She was simultaneously started on fibrates and high doses omega-3 fatty acids.

Conclusion: Estrogen triggered hypertriglyceridemia with metabolic predisposition can cause pancreatitis; intravenous insulin offers rapid, effective, and accessible treatment in resource-limited settings.

KEY WORDS: Hypertriglyceridemia, Acute pancreatitis; Insulin infusion; Estrogen therapy and Polycystic ovary syndrome.

1. Dr Salma Salman
Endocrine Fellow,
National Institute of Diabetes and Endocrinology (NIDE),
Dow University of Health Sciences (DUHS),
Karachi – Pakistan.
2. Dr Sabiha Banu
Assistant Professor,
National Institute of Diabetes and Endocrinology (NIDE),
Dow University of Health Sciences (DUHS),
Karachi – Pakistan.
3. Dr Nazish Fatima
Senior Registrar,
National Institute of Diabetes and Endocrinology (NIDE),
Dow University of Health Sciences (DUHS),
Karachi – Pakistan.
4. Dr Araj Jamil
Senior Registrar in Critical Care Medicine (CCM)/ Intensivist,
Dow University of Health Sciences (DUHS),
Karachi – Pakistan.

Address for Correspondence:

Salma Salman, FCPS (Med),
Fellow Endocrinology,
National Institute of Diabetes and Endocrinology (NIDE),
DIMC, DUHS, Dow University of Health Sciences,
Karachi – Pakistan.
E-mail: salma.salman@duhs.edu.pk

INTRODUCTION

Acute pancreatitis is a common clinical condition with gallstones and alcohol use accounting for most of the cases. Hypertriglyceridemia-induced acute

Submitted: January 02, 2026 **Revision Received:** May 20, 2026

Accepted for Publication: May 30, 2026

How to cite this: *Salman S, Banu S, Fatima N, Jamil A. Estrogen-Induced Severe Hypertriglyceridemia Presenting as Recurrent Acute Pancreatitis Successfully Managed with Intravenous Insulin Infusion. JPES. 2026;3(1):44-48.*

Access this Article Online

URL: https://jpes.org.pk/index.php/jpes/article/view/87

pancreatitis (HTG-AP) is a less frequent but important etiology, particularly in younger individuals and those without classical risk factors.[1] The risk of pancreatitis increases significantly when serum triglyceride levels exceed 1000 mg/dL and rises further at levels above 2000 mg/dL.[2] The underlying mechanism involves hydrolysis of triglycerides into free fatty acids, resulting in lipotoxicity, endothelial dysfunction, and pancreatic inflammation.[3]

Several secondary factors may contribute to severe hypertriglyceridemia, including uncontrolled diabetes, pregnancy, medications, and endocrine disorders. Estrogen therapy is known to increase hepatic verylow-density lipoprotein production and reduce lipoprotein lipase activity, thereby exacerbating triglyceride levels.[4] In individuals with underlying insulin resistance, such as those with polycystic ovary syndrome (PCOS), this effect may be amplified, predisposing them to severe metabolic complications.

Despite increasing global recognition of HTG-AP, there is limited data from Pakistan specifically addressing insulin infusion as a primary modality for rapid triglyceride reduction, particularly in patients with coexisting endocrine abnormalities.[5] We report a case of recurrent HTG-AP in a young woman likely precipitated by estrogen therapy in the setting of suspected PCOS, successfully managed with intravenous insulin infusion.

CASE PRESENTATION

A 26-year-oldfemale married for 2 years presented with a one-day history of severe, continuous abdominal pain localized to the epigastrium and right hypochondrium, radiating to the back and associated with nausea. There was no history of fever, alcohol use, or known gallstone disease. She reported irregular menstrual cycles for several years, along with hirsutism and delayed menstruation for the preceding two months. The patient had been prescribed Diane-35®(ethinylestradiol 0.035 mg/cyproterone acetate 2 mg) by the gynecology team for menstrual irregularity and had been taking it for approximately four months before presentation. She had difficulty conceiving because of irregular menstrual cycles secondary to polycystic ovarian syndrome. The patient had experienced a previous episode of acute pancreatitis approximately four months earlier, during which her serum triglyceride level was 450 mg/dL. She had already been receiving Diane-35 (ethinylestradiol 0.035 mg/cyproterone acetate 2 mg) at that time. The patient denied any family history of dyslipidemia, hypertriglyceridemia, or recurrent pancreatitis. Although a genetic predisposition was considered, no clinical or family history suggestive of an inherited lipid disorder was identified.

On presentation, she appeared anemic but was hemodynamically stable, with a blood pressure of

Table-IA: Work up done during admission.

Investigation	*Value*	*Reference Range*
HB	12.0 gm/dL	12.0-15.0
TLC	7.3 × 10^9/L	4.0-10.0
PLATELET	300 ×10^9/L	150-400
CREATININE	0.6 mg/dl	0.6-1.1
SERUM TSH	0.527 uIU/ml	Adults 21 - 54 years: 0.4 - 4.2
SERUM HCG	< 2.5	Normal Females: < 2 mIU/ml
AMYLASE	190 U/L	28-100
Lipase	162 U/L	< 38
HBA1C	5.41 %	Normal <5.7.
CALCIUM	8.40 mg/dl	8.6-10.2
Magnesium	1.72 mg/dl	1.6-2.6
ANA	Positive (Fine Speckled +1)	
L.D.H	245	125-220
HBsAg/Anti-HCV	Nonreactive	

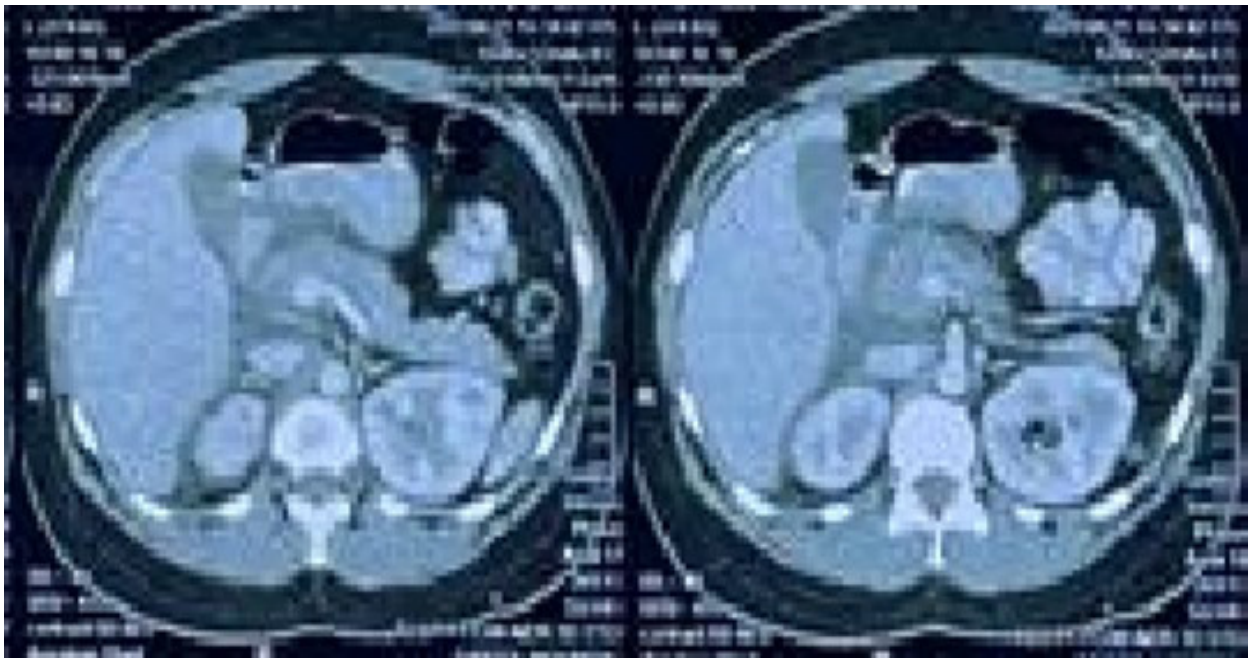

Fig.1:Contrast-enhanced CT scan demonstrating non-necrotizing acute interstitial pancreatitis with a CT severity index of 6.

140/75 mmHg. There was no jaundice or thyroid enlargement. Abdominal examination revealed grade 3 tenderness in the epigastrium and right hypochondrium. Clinical features were suggestive of hyperandrogenism (acne, hirsutism with a Ferriman-Gallwey score of 10). Labs shown in Table-I.

Laboratory evaluation revealed markedly elevated triglyceride levels of 4694 mg/dL, while random blood glucose ranged between 123 and 136 mg/dL. Tables-IA and IB show the laboratory investigations. A CT abdomen with a pancreatic protocol shows a swollen and edematous pancreas. Significant peripancreatic fat stranding is seen with fluid extending along the mesentery, into the abdomen and pelvis, within the right perinephric space, and tracking along the right paracolic gutter, with thickening of bilateral Gerota's and lateroconal fascia. No evidence of pancreatic necrosis or vascular compromise seen. as shown in Fig.1.

A diagnosis of recurrent acute pancreatitis secondary to severe hypertriglyceridemia, likely precipitated by estrogen therapy in a patient with suspected PCOS, was established. The patient's Ranson score was 1, which is associated with a low mortality risk (approximately 1%).

The patient was admitted to the intensive care unit and managed conservatively with bowel rest, intravenous fluids, and analgesia. A multidisciplinary team was involved in her management. The patient was started on a continuous intravenous insulin infusion at 0.1 U/kg/hour together with a 5% dextrose infusion. Capillary blood glucose was monitored every 2 hours, and glucose levels were maintained within the target range of 120–180 mg/dL throughout the 48-hour treatment period. Serum potassium monitoring was planned every 6 hours; however, due to local ICU resource constraints, potassium levels were assessed every 12 hours and supplemented as required when hypokalemia was detected. Serum triglyceride levels were measured at 12, 24, and 48 hours, demonstrating a progressive decline of greater than 50% from baseline. Gemfibrozil and omega-3 fatty acids were initiated concurrently and continued after discontinuation of insulin infusion. This is also accompanied by drastic clinical improvement in abdominal pain and vomiting. She continued oral lipid-lowering therapy with gemfibrozil 600 mg twice daily and omega-3 fatty acids (1 g three times daily). The patient remained clinically stable and was discharged with advice for endocrinology follow-up to evaluate underlying hormonal abnormalities, including suspected PCOS, and to address long-term metabolic and reproductive health. The consent of the patient, as well as her husband, was taken prior to the writing of this manuscript.

DISCUSSION

Hypertriglyceridemia (HTG) is among the underrated causes of acute pancreatitis. The risk of acute pancreatitis increases with the increasing level of TGs, especially when they reach the critical value of above 1000 mg/dl.[6] The higher level of triglyceride compromises the

Table-IB: Lipid profile pre and post insulin infusion along with reference range.

Test	*Pre-insulin infusion (2nd march 2026)*	*Post-insulin infusion after 24 hours (3rd march 2026)*	*Result after 48 hours: 4th/march (post insulin infusion)*	*Reference value*
Triglycerides	4694 mg/dl	2463 mg/dl	646 mg/dl	<150
Serum Cholesterol	607 mg/dl	457 mg/dl	286 mg/dl	<200 (Desirable) / 200–239 (Borderline) / >239 (High)
HDL cholesterol	16 mg/dl	20 mg/dl	33 mg/dl	<40 (Low) / >60 (High)
LDL cholesterol	71 mg/dl	58 mg/dl	72 mg/dl	<100 (Optimal) / 100–129 (Near Optimal) / 130–159 (Borderline High) / 160–189 (High) / >189 (Very High)
VLDL cholesterol	N/A	N/A	129.2 mg/dl	≤30
Non-HDL Cholesterol	N/A	N/A	253 mg/dl	Goal: <130 (high risk), <160 (moderate), <190 (low)

microcirculation of pancreas due to increase in viscosity. Concurrently, hydrolysis of triglycerides by pancreatic lipase releases free fatty acids (FFAs), which exert direct cytotoxic effects, promote endothelial dysfunction, and trigger an inflammatory cascade that culminates in pancreatic injury.[7]

Multiple contributing factors appear to have acted synergistically in our case. The use of exogenous estrogen is a recognized but frequently under-appreciated cause of secondary hypertriglyceridemia. Estrogen increases hepatic synthesis of very low-density lipoproteins (VLDL) while reducing lipoprotein lipase (LPL) activity, thereby impairing triglyceride clearance. Although this effect is typically mild in otherwise healthy individuals, it may become clinically significant in patients with underlying metabolic susceptibility.[8]

Polycystic ovary syndrome (PCOS) is commonly associated with insulin resistance, obesity, and dyslipidemia, all of which predispose patients to elevated triglyceride levels. The coexistence of PCOS-related metabolic dysfunction and exogenous estrogen therapy may have amplified the risk of severe hypertriglyceridemia in our patient. This highlights the importance of careful metabolic assessment before initiating estrogen-containing therapies in susceptible individuals.[8]

Intravenous insulin infusion has emerged as an effective and increasingly utilized therapeutic modality for rapid reduction of triglyceride levels in HTG-associated pancreatitis (HTG-AP), as is evident by multiple sources.[9] Insulin enhances LPL activity, facilitating the clearance of triglyceride-rich lipoproteins such as chylomicrons and VLDL. The recommended insulin dose typically ranges from 0.1 to 0.3 units/kg/hour. However, due to the risk, especially in non-diabetic patients, concurrent glucose infusion and close monitoring of blood glucose levels are essential.[10] In our patient we started infusion at 0.1 unit/kg/hour, which is similar to many of the case studies published in literature with good recovery.

In our patient, insulin monotherapy resulted in a marked reduction in triglyceride levels within 24 hours, accompanied by significant clinical improvement. This rapid response is consistent with findings from previous studies and case series demonstrating that insulin infusion can effectively lower triglyceride levels within 24–72 hours.[11] A review of outcomes in patients with HTG-AP has shown that the overall prognosis is favorable, with the majority of patients achieving complete recovery following intensive insulin therapy.[12] These findings reinforce the role of insulin not only as a metabolic regulator but also as a practical frontline intervention in acute settings.

In resource-limited environments such as Pakistan, insulin infusion represents a particularly valuable treatment option. Unlike plasmapheresis, which is costly, resource-intensive, and often unavailable, insulin is widely accessible and can be administered with relative ease in most clinical settings.[13] Plasma exchange (plasmapheresis) remains an alternative therapeutic modality, particularly in severe or refractory cases. While plasmapheresis can achieve rapid reduction of triglyceride levels, current evidence suggests that it may not significantly outperform insulin therapy in terms of speed of triglyceride reduction or overall mortality benefit.[13]

Early identification of underlying endocrine and metabolic disorders is critical to preventing recurrence. Long-term management should focus on

sustained lipid control through lifestyle modification, pharmacotherapy (e.g., fibrates or omega-3 fatty acids), and appropriate endocrine evaluation and follow-up.[14]

CONCLUSION

In conclusion, this case highlights the multifactorial nature of severe hypertriglyceridemia and underscores the pivotal role of insulin monotherapy as an effective, safe, and accessible treatment option. It also emphasizes the importance of recognizing reversible precipitating factors, particularly estrogen therapy, and the need for comprehensive metabolic assessment in young patients presenting with pancreatitis. Further prospective studies are warranted to establish standardized treatment protocols and to better define the role of insulin relative to other therapeutic modalities in diverse clinical settings.

Grant support: None.

Financial disclosure: None.

IRB approval: Not applicable.

Patient consent: Written informed consent taken from patient prior to publication.

REFERENCES

1. Gao L, Li W. Hypertriglyceridemia and acute pancreatitis: clinical and basic research—a narrative review. J Pancreatol. 2024;7(1):53-60. doi:10.1097/JP9.0000000000000153.
2. Rygiel K. Hypertriglyceridemia - Common Causes, Prevention and Treatment Strategies. Curr Cardiol Rev. 2018;14(1):67-76. doi: 10.2174/1573403X14666180123165542. PMID: 29366425; PMCID: PMC5872265
3. Stinkens R, Goossens GH, Jocken JW, Blaak EE. Targeting fatty acid metabolism to improve glucose metabolism. Obes Rev. 2015;16(9):715-57. doi: 10.1111/obr.12298. Epub 2015 Jul 16. PMID: 26179344.
4. Parhofer KG, Laufs U. The Diagnosis and Treatment of Hypertriglyceridemia. Dtsch Arztebl Int. 2019 Dec 6;116(49):825-832. doi: 10.3238/arztebl.2019.0825. PMID: 31888796; PMCID: PMC6962767.
5. Inayat F, Zafar F, Baig AS, Chaudhry NA, Aslam A, Khan ZH, Iqbal MJ. Hypertriglyceridemic Pancreatitis Treated with Insulin Therapy: A Comparative Review of 34 Cases. Cureus. 2018;10(10): e3501. doi: 10.7759/cureus. 3501. PMID: 30648042; PMCID: PMC6318129.
6. Qureshi TM, Khan A, Javaid H, Tabash A, Hussein MS, Othman MO. Secondary Causes of Hypertriglyceridemia are Prevalent Among Patients Presenting With Hypertriglyceridemia-Induced Acute Pancreatitis. Am J Med Sci. 2021;361(5):616-623. doi: 10.1016/j.amjms.2021.01.005. Epub 2021 Jan 11. PMID: 33618838.
7. De Pretis N, Amodio A, Frulloni L. Hypertriglyceridemic pancreatitis: Epidemiology, pathophysiology, and clinical management. United European Gastroenterol J. 2018;6(5):649-655. doi: 10.1177/2050640618755002. Epub 2018 Jan 22. PMID: 30083325; PMCID: PMC6068776.
8. Feingold KR. The Effect of Endocrine Disorders on Lipids and Lipoproteins. [Updated 2026 Mar 20]. In: Feingold KR, Adler RA, Ahmed SF, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK409608/.
9. Mateo RCI, Ganda OP. Severe hypertriglyceridemia in the hospitalized patient. In: Garg RK, Hennessey JV, Garber JR, editors. Handbook of Inpatient Endocrinology. Cham: Springer; 2025. doi:10.1007/978-3-031-86099-7_28.
10. White BN, Carter BL, Bradford JL. Analysis of Intravenous Insulin Dosing Requirements for Treatment of Severe Hypertriglyceridemia. Hosp Pharm. 2023;58(1):79-83. doi: 10.1177/00185787221126339. Epub 2022 Oct 12. PMID: 36644746; PMCID: PMC9837331.
11. Maric N, Mačkovic M, Bakula M, Mucic K, Udiljak N, Marusic M. Hypertriglyceridemia-induced pancreatitis treated with continuous insulin infusion-Case series. Clin Endocrinol (Oxf). 2022;96(2):139-143. doi: 10.1111/cen.14554. Epub 2021 Jul 14. PMID: 34263462
12. Giri S, Singh RK, Jena SK, Sahu SK, Sahu SK, Praharaj D, Mallick B, Nath P, Panigrahi SC, Meher D, Sahu MK, Anand AC. Incidence and Outcome of HTG-AP at a Tertiary Care Center in Eastern India: A Retrospective Analysis. Euroasian J Hepatogastroenterol. 2025;15(1):50-53. doi: 10.5005/jp-journals-10018-1471. Epub 2025 Jun 18. PMID: 40718609; PMCID: PMC12288593.
13. Gubensek J. The role of apheresis and insulin therapy in hypertriglyceridemic acute pancreatitis-a concise review. BMC Gastroenterol. 2023;23(1):341. doi: 10.1186/s12876-023-02957-3. PMID: 37789261; PMCID: PMC10546782.
14. Wierzbicki AS. Advances in the pharmacological management of hyperlipidemia through the use of combination therapies. Expert Opin Pharmacother. 2025;26(2):157-165. doi: 10.1080/14656566.2024.2444986. Epub 2024 Dec 23. PMID: 397096

Author's Contribution:

SS: Concept, literature review, lab interpretation, manuscript writing and final approval.
SB: Patient management, literature review, and critical revision of the manuscript.
NF: Provided expert guidance, and approved the final version for publication.
AJ: Patient management, Data collection, literature review, final approval.