CASE REPORT

DOI: 10.4274/csmedj.galenos.2026.2026-2-1

Cam and Sakura Med J

CSMJ

# Chylomicronemia-Induced Necrotizing Pancreatitis Treated with Therapeutic Plasma Exchange: A Case Report

Melike Delipoyraz, Kamuran Uluç, Gülçin Hilal Alay

University of Health Sciences Türkiye, Çam and Sakura City Hospital, Clinic of Intensive Care Unit, İstanbul, Türkiye

**What is known on this subject?**

Severe hypertriglyceridemia (HTG) is a well-established but relatively uncommon cause of acute pancreatitis, particularly in patients with underlying familial hyperlipidemia or chylomicronemia. HTG-induced pancreatitis may follow a severe course, with an increased risk of necrotizing disease, organ failure, and intensive care unit admission. Rapid reduction of triglyceride (TG) levels is considered a key component of management in severe and complicated cases.

**What this study adds?**

This case demonstrates that early therapeutic lipoprotein apheresis can provide rapid biochemical improvement and clinical stabilization in severe necrotizing pancreatitis secondary to chylomicronemia. It highlights the potential role of lipoprotein apheresis as an effective adjunct to standard medical therapy in selected high-risk patients with familial hyperlipidemia. The report emphasizes the importance of early recognition and aggressive TG-lowering strategies in the intensive care management of HTG-induced pancreatitis.

## ABSTRACT

Acute pancreatitis associated with severe hypertriglyceridemia, especially when it develops in the context of familial hyperlipidemia and chylomicronemia syndrome, is characterized by rapid clinical deterioration, a necrotizing course, and multiple organ dysfunction. Therapeutic plasma exchange (TPE) may be considered a treatment option in severe cases due to its rapid triglyceride (TG)-lowering effect. In our patient, TG levels above 2000 mg/dL, together with necrotizing pancreatitis, metabolic acidosis, and acute kidney injury, indicated rapid clinical deterioration. The observed reduction in TG levels and the clinical improvement in this patient suggest that TPE could be considered in selected severe cases. Early use of TPE in intensive care settings may be an option in selected cases; however, further studies are required.

**Keywords:** Critical care medicine, therapeutic plasma exchange, acute necrotizing pancreatitis, hypertriglyceridemia, chylomicronemia

## Introduction

Acute pancreatitis (AP) associated with severe hypertriglyceridemia (HTG) can progress rapidly with multiple organ dysfunction, particularly when it develops in the context of familial hyperlipidemia and chylomicronemia syndrome (1). HTG-AP develops from lipotoxicity resulting from disruption of the pancreatic microcirculation by triglyceride (TG)-rich lipoproteins, particularly chylomicrons, which circulate in the bloodstream and are hydrolyzed by pancreatic lipase into FFAs. These mechanisms lead to pancreatic ischemia, inflammation, and necrosis, resulting in a severe course of the disease (2). Recurrent pancreatitis attacks and the need for intensive care unit (ICU)

**Corresponding Author:** Melike Delipoyraz, MD, University of Health Sciences Türkiye, Çam and Sakura City Hospital, Clinic of Intensive Care Unit, İstanbul, Türkiye

**E-mail:** melikedelipoyraz@gmail.com **ORCID ID:** orcid.org/0000-0001-7347-1629

**Received:** 12.02.2026 **Accepted:** 06.04.2026 **Epub:** 15.04.2026

**Cite this article as:** Delipoyraz M, Uluç K, Alay GH. Chylomicronemia-induced necrotizing pancreatitis treated with therapeutic plasma exchange: a case report. Cam and Sakura Med J. [Epub Ahead of Print]

follow-up are more frequently reported in patients with hereditary lipoprotein metabolism disorders (3,4).

The treatment of hypertriglyceridemic pancreatitis essentially involves the standard approach to AP; however, rapidly lowering TG levels is critically important in the course of the disease. In addition to medical treatment, TPE has been proposed as a potential treatment option for the rapid control of TG levels in these patients (2).

In this report, we present the management and clinical course of severe hypertriglyceridemic necrotizing pancreatitis developing on the basis of chylomicronemia associated with familial hyperlipidemia in patients treated with TPE in the ICU.

### Case Report

A 25-year-old male patient with known type 1 diabetes mellitus and familial hyperlipidemia presented to the emergency department with complaints of abdominal pain, nausea, and vomiting. Blood samples drawn in the emergency department yielded lipemic serum. The patient's tests revealed an amylase level of 1741 U/L and a lipase level of 1424 U/L; contrast-enhanced computed tomography showed acute necrotic pancreatitis, and the patient was admitted to the ICU. The patient's history was notable for recurrent episodes of pancreatitis and one prior episode of TPE. Tests performed in the ICU revealed metabolic acidosis (pH 7.273, bicarbonate 14.8), a TG level of 2786 mg/dL, a urea level of 61.4 mg/dL, a creatinine level of 2.75 mg/dL, and a GFR of 31. The patient was diagnosed with acute necrotizing pancreatitis due to chylomicronemia. In the ICU, in addition to intravenous hydration, fibrate therapy for HTG, omega-3 supplementation, and insulin infusion therapy, TPE was performed. Therapeutic plasma exchange was initiated 24 hours after ICU admission and on day 3. The procedure was performed using a centrifugal technique. Each session lasted approximately 1.5 hours. Two sessions were conducted. No procedure-related complications were observed. TG levels were monitored. During follow-up, the patient's TG level decreased to 482 mg/dL and did not increase thereafter. The percentage reduction and temporal changes in TG levels are summarized in Figure 1. After a 9-day stay in the ICU, the patient was transferred to the internal medicine ward upon achieving clinical stabilization and improvement.

Written informed consent was obtained from the patient for publication of clinical data and accompanying images.

## Discussion

AP associated with HTG is a well-defined clinical entity that is often subject to diagnostic delay in clinical practice and can be severe, particularly when it occurs in the context of a genetic predisposition. According to the Endocrine Society guidelines, TG levels above 1000 mg/dL significantly increase the risk of AP, and this risk becomes even more pronounced at levels above 2000 mg/dL (1). A TG level of 2786 mg/dL was the biochemical basis for the development of necrotizing pancreatitis.

The pathophysiology of HTG-AP is related to lipotoxicity resulting from disruption of the pancreatic microcirculation by chylomicron-rich lipoproteins and from hydrolysis of TG into free fatty acids (FFAs) via pancreatic lipase. Unbound FFAs trigger acinar cell necrosis by causing local acidosis, endothelial dysfunction, and mitochondrial damage (2). These mechanisms biologically explain why HTG-AP is associated with a more frequent necrotizing course, organ failure, and ICU requirements (3). The presence of metabolic acidosis and acute kidney injury in the this case can be considered a clinical manifestation of these systemic effects.

An important clinical pitfall in the diagnosis of HTG-AP is that TG levels can decrease rapidly in the acute phase, and the etiology may be overlooked in later measurements (5). Findings reported in the literature suggest that measuring serum TG levels early may be useful in clinical practice for patients evaluated for idiopathic pancreatitis.

The primary approach for the treatment of HTG-AP involves rapid reduction of TG levels, as reducing the TG load during the acute phase, in conjunction with standard

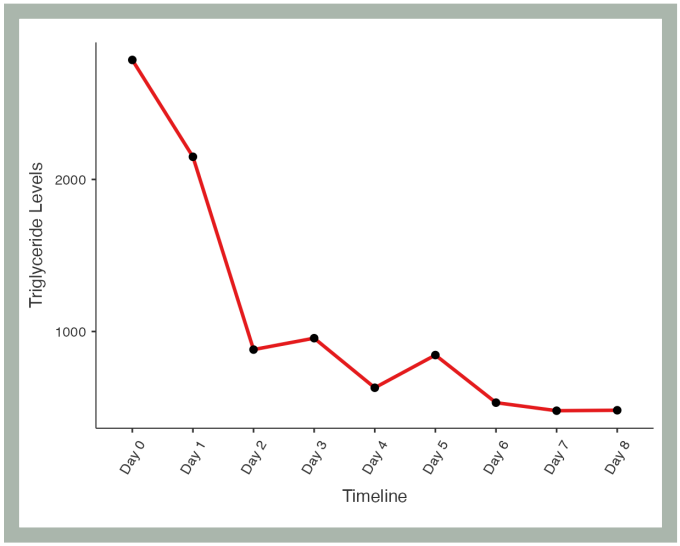


**Figure 1.** Trend of serum triglyceride levels during intensive care follow-up and after therapeutic plasma exchange

AP management, may limit pancreatitis and the systemic inflammatory response associated with lipotoxicity (6). For this purpose, intravenous insulin infusion is commonly used and may facilitate TG reduction (7). Additionally, fibrate therapy, which suppresses hepatic TG production, and omega-3 fatty acids, which reduce TG synthesis, contribute to long-term TG control (1). To our patient, intravenous insulin infusion, fibrate therapy, and omega-3 fatty acid therapy were administered in addition to standard intensive care.

TPE has been reported to produce rapid biochemical improvement in patients with HTG-AP by removing circulating chylomicrons and TGs. However, its superiority in terms of mortality and long-term clinical outcomes is unclear (8). According to the American Society for Apheresis guidelines, TPE may be considered in selected severe cases (9). Because of the clinical characteristics of our patient, TPE was performed, and clinical stabilization was achieved, with TG levels falling below 500 mg/dL within a short period.

## Conclusion

AP associated with HTG can be severe, particularly when it occurs in the setting of chylomicronemia. Our patient had a history of recurrent pancreatitis characterized by high TG levels, and TPE was followed by a rapid reduction in TG levels and clinical improvement. This case suggests that TPE may be considered an adjunctive option for selected patients with severe HTG-AP. Further studies are required to clarify its clinical impact.

### Ethics

**Informed Consent:** Written informed consent was obtained from the patient for publication of clinical data and accompanying images.

### Footnotes

### Authorship Contributions

Concept: M.D., G.H.A., Design: M.D., K.U., Data Collection: M.D., Literature Search: M.D., K.U., Writing: M.D.

**Conflict of Interest:** No conflict of interest was declared by the authors.

**Financial Disclosure:** The authors declared that this study received no financial support.

## REFERENCES

1. Berglund L, Brunzell JD, Goldberg AC, et al. Evaluation and treatment of hypertriglyceridemia: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2012;97:2969-2989. Erratum in: J Clin Endocrinol Metab. 2015;100:4685. Erratum in: J Clin Endocrinol Metab. 2026;111:e647.
2. Valdivielso P, Ramírez-Bueno A, Ewald N. Current knowledge of hypertriglyceridemic pancreatitis. Eur J Intern Med. 2014;25:689-694.
3. Saddatgah S, Larouche M, Naderian M, et al. Recognition and management of persistent chylomicronemia: a joint expert clinical consensus by the National Lipid Association and the American Society for Preventive Cardiology. Am J Prev Cardiol. 2025;22:100978.
4. Scherer J, Singh VP, Pitchumoni CS, Yadav D. Issues in hypertriglyceridemic pancreatitis: an update. J Clin Gastroenterol. 2014;48:195-203.
5. Yadav D, Pitchumoni CS. Issues in hyperlipidemic pancreatitis. J Clin Gastroenterol. 2003;36:54-62.
6. Feingold KR. Pancreatitis secondary to hypertriglyceridemia. 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/NBK279082/
7. Garg R, Rustagi T. Management of hypertriglyceridemia induced acute pancreatitis. Biomed Res Int. 2018;2018:4721357.
8. Chen JH, Yeh JH, Lai HW, Liao CS. Therapeutic plasma exchange in patients with hyperlipidemic pancreatitis. World J Gastroenterol. 2004;10:2272-2274.
9. Padmanabhan A, Connelly-Smith L, Aqui N, et al. Guidelines on the Use of Therapeutic Apheresis in Clinical Practice - Evidence-Based Approach from the Writing Committee of the American Society for Apheresis: The Eighth Special Issue. J Clin Apher. 2019;34:171-354.