Plasmapheresis for recurrent acute pancreatitis from hypertriglyceridemia

Kathleen Kopecky, MD, Amber Moreland, MD, Christopher Hebert, MD, and Gates B. Colbert, MD

Acute pancreatitis is a known complication of severe hypertriglyceridemia. Therapeutic experience with plasmapheresis is less well reported but has been highly successful in life-threatening presentations. We describe a 38-year-old obese Hispanic woman with a previous history of acute pancreatitis from diabetic hypertriglyceridemia who presented to the emergency department with a 2-day history of worsening abdominal pain. Plasmapheresis was initiated with one calculated plasma volume exchange using 5% albumin replacement within 24 hours of admission. Following this treatment, the triglyceride level fell 74%. Another session was performed the following day. The final triglyceride level represented a 93% reduction. This case is novel in that the patient presented twice within the same year with hypertriglyceridemic pancreatitis and responded well to prompt plasmapheresis therapy.

Acute pancreatitis from hypertriglyceridemia is a rare but well-known condition. Elevated triglycerides result from a variety of compounding conditions that lead to direct damage of pancreatic endocrine and exocrine function and can cause severe ischemia to major organ systems. Treatment of hypertriglyceridemic pancreatitis (HTGP) with plasmapheresis is an extremely effective therapy, but is less well reported.

CASE PRESENTATION

A 38-year-old obese Hispanic woman with diabetes mellitus type 2, hyperlipidemia, and acute pancreatitis secondary to hypertriglyceridemia presented to the emergency department with a 2-day history of worsening abdominal pain. Ten months earlier, the patient was admitted for acute pancreatitis secondary to hypertriglyceridemia; laboratory results are shown in *Table 1.* The hospital course was complicated by altered mental status, hypoxemic respiratory failure, and respiratory mechanical support. She underwent two plasmapheresis treatments that quickly lowered her triglyceride level from >4000 mg/dL to normal range. Once stabilized, she was discharged and prescribed triglyceride- and lipid-lowering medications.

On the second admission, the patient recalled eating a meal the day of presentation and suddenly developing sharp epigastric abdominal pain with radiation to her back. Nausea followed with several episodes of nonbilious, nonbloody emesis. The patient had lapsed in taking her previously prescribed medications. She denied any fever, chills, chest pain, dyspnea, or diarrhea.

Table 1. Pertinent serum laboratory values in the patient described

Variables	10 months earlier	Current admission
Total cholesterol (mg/dL)	>1000	706
Triglycerides (mg/dL)	>4000	>4000
High-density lipoprotein (mg/dL)	35	40
Low-density lipoprotein (mg/dL)	492	93
Lipase (U/L)	19,536	8263
Glucose (mg/dL)	256	276
Blood urea nitrogen (mg/dL)	7	6
White blood cells (K/μL)	16.2	16.6
Oxygen (%)	83	54
Corrected calcium (for albumin, mg/dL)	5.9	8.2

In the emergency department, she appeared uncomfortable, with cardiac examination revealing tachycardia and tachypnea. Her abdomen was tender to palpation in the epigastric region with guarding, but there was no palpable hepatosplenomegaly. She had 2+ lower extremity edema but no rash. Admission vital signs showed a blood pressure of 104/72 mm Hg, heart rate of 104 beats per minute, temperature of 98.1°F, respiration of 18 breaths per minute, and body mass index of 39.8 kg/m^2. Laboratory tests are shown in *Table 1;* other pertinent results included serum sodium, 139 mEq/L; potassium, 4.3 mEq/L; bicarbonate, 12 mEq/L; glucose, 276 mg/dL; anion gap, 11; and creatinine, 0.8 mg/dL. Computed tomography of the abdomen and pelvis revealed acute pancreatitis with moderate inflammatory stranding predominantly surrounding the head and body of the pancreas, but without overt pancreatic necrosis. After

From the Department of Internal Medicine, Baylor University Medical Center, Dallas, Texas.

Corresponding author: Gates B. Colbert, MD, 3417 Gaston Avenue, Suite 875, Dallas, TX 75246 (e-mail: Gates.Colbert@BSWHealth.org; Twitter handle: @DoctorGates).

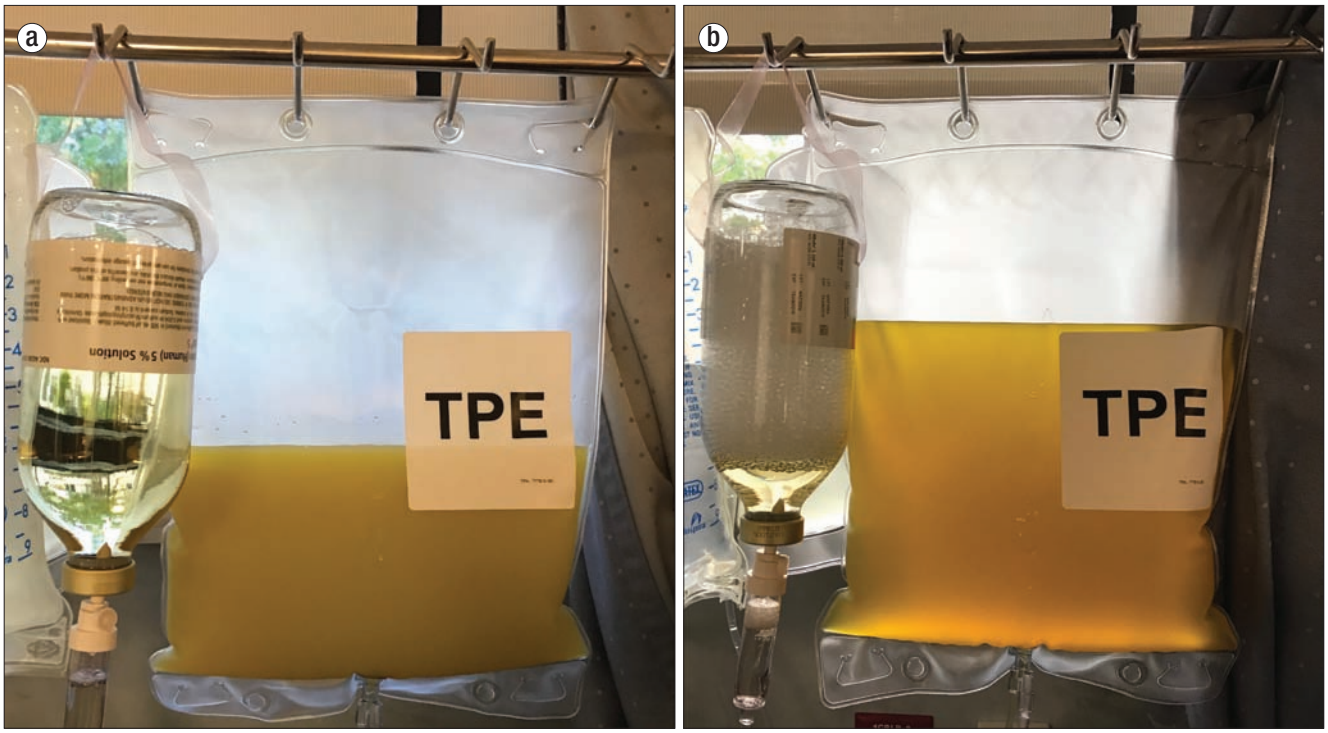

Figure 1. (a) Initial plasmapheresis session with lipemic, opaque effluent. **(b)** Translucent change of effluent after second plasmapheresis session with reduction in triglyceride levels.

examination and return of her studies, her cumulative Ranson score was 5 out of 11. With any Ranson score above 4, the risk of morbidity and mortality is high (1). The patient was aggressively rehydrated with Ringer's lactate solution and was started on an intravenous insulin infusion.

Plasmapheresis was performed with one calculated plasma volume exchange using 5% albumin replacement within 24 hours of admission. After the initial session, the patient's triglyceride level was reduced to 1026 mg/dL, a 74% reduction. The following day, a second plasmapheresis was performed with a reduction in triglyceride level to 360 mg/dL, an overall 90% reduction. The effluent of the initial exchange transitioned from lipemic and opaque to translucent after two sessions *(Figure 1).* Her final measured triglyceride level was 280 mg/dL, a 93% reduction, and her serum lipase was 399 U/L. By hospital day 5, she was tolerating a solid diet without abdominal pain and was transitioned to subcutaneous insulin.

DISCUSSION

HTGP is a well-established cause of acute pancreatitis, accounting for 1% to 4% of cases (2). HTGP is most common in patients with diabetes mellitus, diabetic ketoacidosis, chronic alcoholism, and women with increased estrogen (2). The risk of developing acute pancreatitis increases in proportion to the serum triglyceride level once a threshold of 1000 mg/dL is reached. However, the severity of acute pancreatitis does not correlate well with the serum triglyceride level (3). The exact mechanism by which elevated triglyceride levels trigger acute pancreatitis remains largely unknown (3). One theory behind HTGP is that pancreatic lipase hydrolyzes the excess triglycerides into free fatty acids, which subsequently produce injury to the pancreatic acinar cells. Ischemia is theorized to be from either hyperviscosity of excess chylomicrons or hyperviscosity of serum itself in the pancreatic capillaries (2).

Several modalities exist for treating HTGP. Maintenance of triglyceride levels below 500 mg/dL has been shown to greatly improve outcomes (4). In extreme levels of hypertriglyceridemia with life-threatening symptoms, plasmapheresis is recommended. This was first described as a treatment modality in 1978 by Betteridge (5). Plasmapheresis offers several advantages, including removing circulating triglycerides and chylomicrons, reducing blood viscosity, and lowering cytokines associated with pancreatitis (6).

Published experience with the use of plasmapheresis to treat HTGP remains scarce (7). This case is novel in that the patient presented twice within the same year with HTGP and responded well to prompt plasmapheresis therapy.

1. Ranson JH, Rifkind KM, Roses DF, Fink SD, Eng K, Spencer FC. Prognostic signs and the role of operative management in acute pancreatitis. *Surg Gynecol Obstet* 1974;139(1):69–81.
2. Tsuang W, Navaneethan U, Ruiz L, Palascak JB, Gelrud A. Hypertriglyceridemic pancreatitis: presentation and management. *Am J Gastroenterol* 2009;104(4):984–991.
3. Scherer J, Singh VP, Pitchumoni CS, Yadav D. Issues in hypertriglyceridemic pancreatitis: an update. *J Clin Gastroenterol* 2014;48(3):195–203.
4. Toskes PP. Hyperlipidemic pancreatitis. *Gastroenterol Clin North Am* 1990;19(4):783–791.
5. Betteridge DJ, Bakowski M, Taylor KG, Reckless JP, de Silva SR, Galton DJ. Treatment of severe diabetic hypertriglyceridaemia by plasma exchange. *Lancet* 1978;1(8078):1368.
6. Yeh JH, Chen JH, Chiu HC. Plasmapheresis for hyperlipidemic pancreatitis. *J Clin Apher* 2003;18(4):181–185.
7. Ramirez-Bueno A, Salazar-Ramirez C, Cota-Delgado F, de la Torre-Prados MV, Valdivielso P. Plasmapheresis as treatment for hyperlipidemic pancreatitis. *Eur J Intern Med* 2014;25(2):160–163.