

Introduction

Acute pancreatitis (AP) presents with acute epigastric pain radiating to the back and elevated pancreatic enzymes. The most common causes are gallstones and alcohol. Hypertriglyceridemia-induced pancreatitis (HTGP), causes 14% of all AP cases, rising to 56% in pregnancy. We present a unique case of HTGP complicated by diabetic ketoacidosis (DKA) and pancreatic pseudocyst.

Case Presentation

A 27-year-old male with type 1 diabetes mellitus, hyperlipidemia, ADHD, and depression presented with sudden onset constant, sharp, diffuse abdominal pain with radiation to the back. He reported nausea, dry heaving, increased thirst and urinary frequency. Relevant family history includes familial hypertriglyceridemia. The patient was found to have DKA, lactic acidosis, leukocytosis, and hypocalcemia. Triglycerides (TG) and lipase/amylase were elevated. Computed tomography (CT) of the abdomen showed AP with fat stranding. He received fluids, an insulin drip, and was transferred to the intensive care unit. Patient met criteria for plasmapheresis and was transferred to a tertiary care center. On follow up, repeat CT three weeks post-presentation indicated a developing pseudocyst. CT four weeks later showed anterior pancreatic pseudocyst with a decrease of peripancreatic fluid collection and CT seven weeks later showed decreased peripancreatic and retroperitoneal fluid with 2 stents extending from stomach to collection.

Imaging

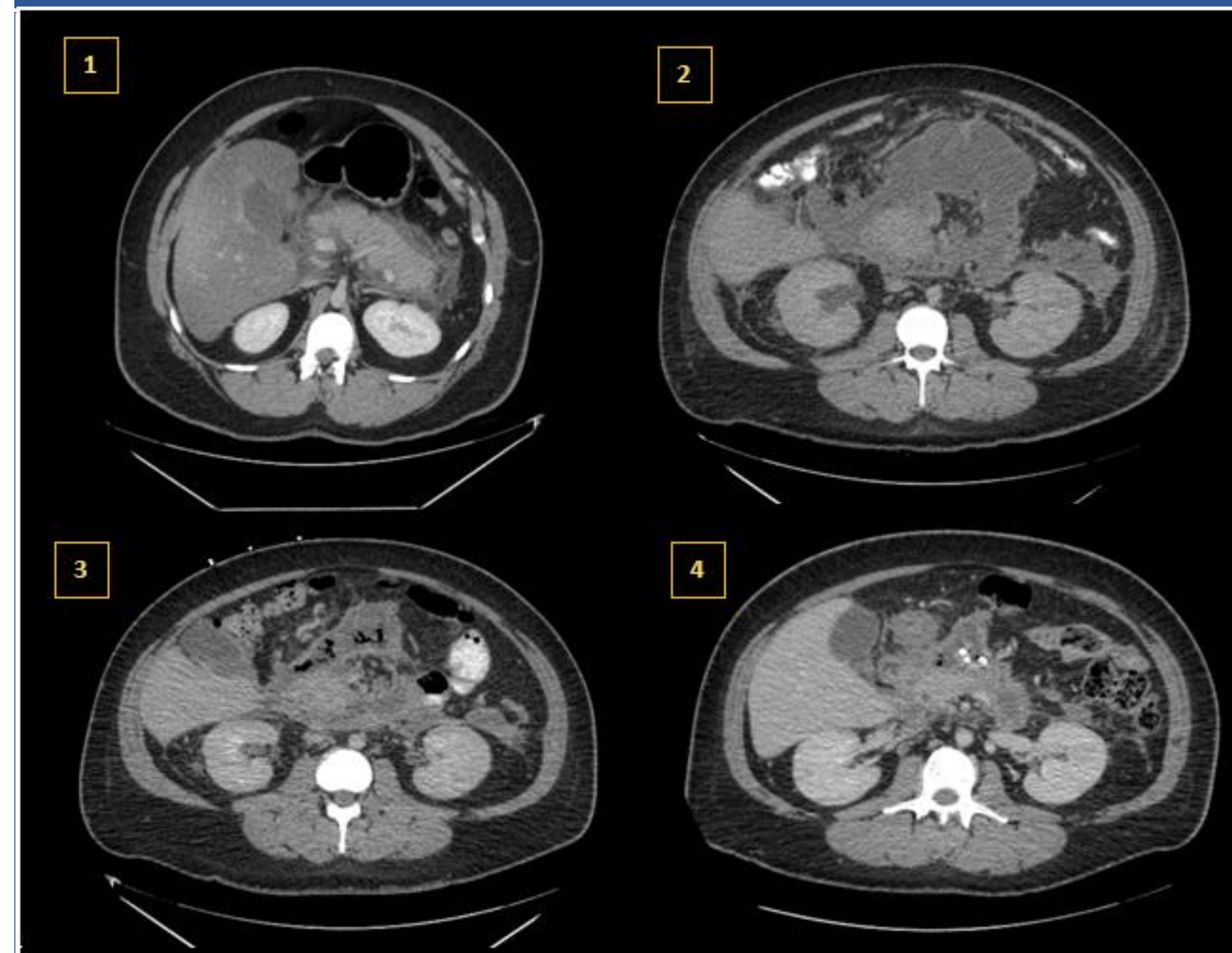


Figure 1: Progression of AP to development of anterior pancreatic pseudocyst and stent placement.

- 1:** AP with fat stranding.
- 2:** Developing anterior pancreatic pseudocyst.
- 3:** Ring-enhancing pancreatic pseudocyst
- 4:** Decreased peripancreatic and retroperitoneal fluid with 2 stents.

Discussion

TG > 1000 is required for the diagnosis of HTGP.

TG 1000-1999 carries a 5% risk of developing HTGP, increasing to 20% in TG >2000.

Patients are typically young, male, obese, and diabetic.

AP may precipitate or complicate DKA. DKA can mask AP, occurring in approximately 10-15% of cases. In 100 consecutive cases of DKA, 11 had a CT confirmed AP with most caused by hypertriglyceridemia and alcohol.

Hypocalcemia, lactic acidosis, or sepsis necessitate intensive therapy, including therapeutic plasma exchange (TPE), which replaces plasma with a colloid solution.

HTGP is managed with supportive care and insulin/TPE until TG <500. TG <200 prevents recurrence and is managed with pharmacotherapy and lifestyle modifications.

Conclusion

HTGP can cause AP and should be considered in the differential for acute abdominal pain, especially in younger males. It can be complicated by DKA and pancreatic pseudocyst, leading to a more severe presentation and necessitates the addition of TPE to insulin. Prompt recognition and management of HTGP is imperative in preventing complications and expediting recovery.

References

1. Forsmark, C. E., Bailly, J., AGA Institute Clinical Practice and Economics Committee, & AGA Institute Governing Board (2007). AGA Institute technical review on acute pancreatitis. *Gastroenterology*, 132(5), 2022–2044. <https://doi.org/10.1053/j.gastro.2007.03.065>
2. Yang, A. L., Vadavkar, S., Singh, G., & Omary, M. B. (2008). Epidemiology of alcohol-related liver and pancreatic disease in the United States. *Archives of internal medicine*, 168(6), 649–656. <https://doi.org/10.1001/archinte.168.6.649>
3. Fortson, M. R., Freedman, S. N., & Webster, P. D., 3rd (1995). Clinical assessment of hyperlipidemic pancreatitis. *The American journal of gastroenterology*, 90(12), 2134–2139. Scherer, J., Singh, V. P., Pitchumoni, C. S., & Yadav, D. (2014). Issues in hypertriglyceridemic pancreatitis: an update. *Journal of clinical gastroenterology*, 48(3), 195–203. <https://doi.org/10.1097/01.mcg.0000436438.60145.5a>

Contact

Garry Lachhar MD
Stony Brook Southampton Hospital
240 Meeting House Lane, Southampton, NY 11968
Garry.lachhar@stonybrookmedicine.edu
(631) 790-0482