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Case Reports

Medical Education Research Forum 2020

5-2020

A Case of Triglyceride-Induced Pancreatitis

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Introduction

The incidence of acute pancreatitis has increased over the years, with gallstones and alcohol abuse being the leading causes of pancreatitis⁵. Many people develop mild pancreatitis, but 20% develop severe or necrotizing pancreatitis⁷. Seven-percent of the cases of pancreatitis are attributed to (HTG) hypertriglyceridemia². The diagnosis is often missed secondary to coexisting factors such as medications and alcohol abuse². Treatment options include bowel rest, intravenous fluids, insulin drip, and plasmapheresis³.

Case Presentation

CC: “Abdominal pain, N/V/D”

HPI: A 55-year-old female presented to the ED with epigastric abdominal pain radiating to her back, nausea, vomiting and diarrhea x one day. Patient woke up in the morning with these symptoms. Denies sick contacts. Patient denied any melena, hematochezia, or hematemesis. Patient denied alcohol abuse, no history of alcohol abuse is noted in the EMR. Denied recent travel or recent antibiotic use. Patient did not take prescription medications. Of note, patient had a negative colonoscopy one year ago.

ROS: Positive for abdominal pain, nausea, vomiting, diarrhea

Vitals: BP 96/56 mmHg | Pulse 138 | 36.2°C | RR 20 | SpO2 97%

Physical Examination:

Constitutional: Well-developed. Alert. **Diaphoretic**

Eyes: Pupils are equal, round, and reactive to light. EOM are normal.

Neck: No neck rigidity.

Cardiovascular: **Tachycardia**, no murmurs

Pulmonary/Chest: Effort normal and breath sounds normal. No respiratory distress.

Abdominal: Soft. **Generalized tenderness. More prominent epigastric region. +Guarding**

Musculoskeletal: Normal range of motion.

Neurological: AAOX3. 5/5 strength B/L UE and LE. No cranial nerve deficit. Negative finger to nose test

Skin: No rash



Lipemic blood³

Hospital Course

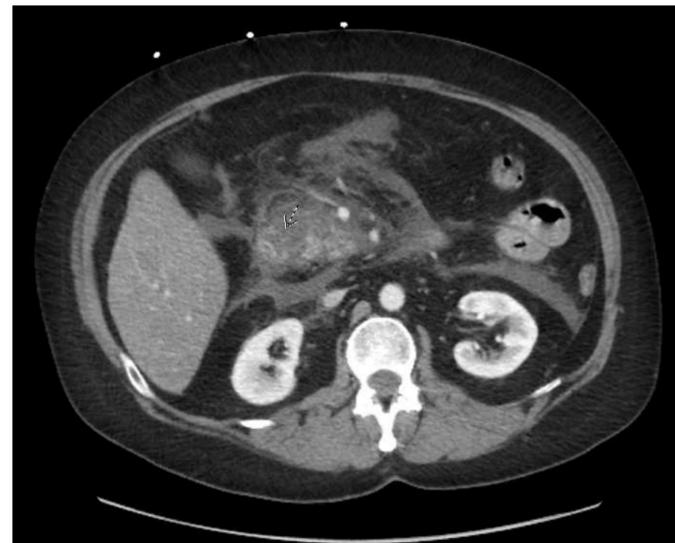
Henry Ford Macomb ED and ICU course 5/17/2019:

Given initial vital signs - code sepsis was called – patient was immediately given 30 cc/kg IVF bolus – Labs orderd - CBC, BMP, Mag, Troponin, lactic acid, blood cultures, liver enzymes, lipase, coags, ethanol level, UA, blood cultures -EKG, acute abdominal series, and CT abdomen/pelvis was ordered

Labs were initially lipemic - therefore redone and labs were significant for Creatinine 1.55 Ca 6.9 ALT 99 AST 246 Lactic Acid 3.9 Triglycerides (TG)1582 Lipase 2365 ETOH =0.07

CT abdomen/pelvis results:

1. Pancreatic necrosis with severe surrounding fluid and inflammation. No gas or discrete fluid collections identified.
2. Submucosal edema of the second portion of the duodenum with possible breakdown of the outer serosal walls along the pancreatic head.



Patient was started on IV regular insulin 0.1 units/kg/hour with D5W+0.9NS and Meropenem for suspected triglyceride-induced necrotizing pancreatitis. With these findings patient was admitted to the ICU and general surgery was consulted. US abdomen was ordered and showed a 12 mm common bile duct with possible choledocholithiasis. No evidence of cholelithiasis. Patient’s electrolytes were being replaced and glucose, urine output, and respiratory status were monitored.

General surgery recommended transfer to higher level of care given multiple pathologies (triglycerides, choledocholithiasis, ETOH) causing pancreatitis and patient was transferred to Henry Ford Main from Henry Ford Macomb ICU.

Henry Ford Main Hospital Course:

Patient was admitted to the SICU on 5/17/2019. In the SICU, patient reported nausea and abdominal pain on admission. Vitals were stable. Patient was treated conservatively with pain management and serial abdominal exams. Once patient’s pain improved - diet was advanced. Repeat triglycerides (TG) 5/20/19 were 210. Patient progressed well and was discharged on 5/25/19 with follow up with PCP.

Discussion

As demonstrated in this case – there are various pathologies that can cause pancreatitis. This case is challenging as the patient did have other factors involved – such as possible choledocholithiasis vs ETOH abuse. Patient did not have a GI consult or ERCP as it was deemed that patient’s necrotizing pancreatitis was from her TG levels. Additionally, there is no record of ETOH abuse in the patient’s medical records though she was positive for ETOH the day she first presented.

When reviewing literature - the key treatment for any type of pancreatitis is early resuscitation, nutrition and pain control⁷. This patient had TG induced necrotizing pancreatitis without infection, which improved with conservative treatment. Additionally, current guidelines have not yet defined first-line (TG)-lowering therapies – with insulin and and plasmapheresis as being options³. Insulin decreases TG by promoting synthesis and activity of lipoprotein lipase (LPL), which hydrolyzes TG into fatty acids and glycerol and facilitates storage of the fatty acids in adipocytes⁶. Plasmapheresis quickly reduces triglycerides making it an effective treatment, however it is expensive and studies have not shown it to be superior to insulin therapy¹. Overall, aggressive resuscitative measures and conservative treatment with insulin and intravenous fluids is preferred over plasmapheresis as mortality benefit has not been proven with its use¹.

Resources

1. Berberich, A et al. “Conservative management in hypertriglyceridemia associated pancreatitis”. *Journal of Internal Medicine*. 286 (2019):644–650.
2. Gan, S-Ian et al. “Hypertriglyceridemia-induced pancreatitis: A case-based review.” *World journal of gastroenterology* vol. 12,44 (2006): 7197-202.
3. Khan, Rafay et al. “Hypertriglyceridemia-Induced Pancreatitis: Choice of Treatment.” *Gastroenterology research* vol. 8,3-4 (2015): 234-236.
4. Ramesh, B N R et al. “Severe Hypertriglyceridemia-induced Acute Pancreatitis: Successful Management by Plasmapheresis.” *Indian journal of nephrology* vol. 29,3 (2019): 217-219.
5. Shah, Adarsh P et al. “Acute pancreatitis: current perspectives on diagnosis and management.” *Journal of inflammation research* vol. 11(2018): 77-85.
6. Song, X., et al. ”Intensive insulin therapy versus plasmapheresis in the management of hypertriglyceridemia-induced acute pancreatitis (Bi-TPAI trial): study protocol for a randomized controlled trial”. *Trials*. vol 20, 365 (2019).
7. Trikudanathan, G et al. “Current Concepts in Severe Acute and Necrotizing Pancreatitis: An Evidence-Based Approach”. *Gastroenterology*. Vo. 156(7) (2019):1994-2007.

