Hypertriglyceridemic Induced Acute Pancreatitis

Elvira D. Kazemyan, MD; Bradley Loewer, MD
LSUHSC Family Medicine Residency, Lake Charles Memorial Hospital, Lake Charles, LA

Lake Charles Memorial Health System
LSUHSC Family Medicine Residency

LSU Health NEW ORLEANS

Introduction

Severe hypertriglyceridemia is the third most common cause for acute pancreatitis, and has been estimated to account for nearly 9 percent of patients with acute pancreatitis. Gallstones and alcohol abuse are the two most common causes of acute pancreatitis. And though, hypertriglyceridemia is an uncommon but a well-established etiology of acute pancreatitis, there is a reported incidence of 2-4 percent which can also lead to significant morbidity and mortality. The risk and severity of acute pancreatitis risks with increasing levels of serum triglycerides. There is an approximate 4 percent increase of incidental acute pancreatitis for every 100 mg/dL increase in triglyceride concentration.

Case presentation

19F w/ PMHx uncontrolled DM with hx DKA admission presents to ED with complaint of severe 101/10 stabbing and constant abdominal pain and nausea and vomiting x 2 days. Had four episodes of "yellow watery" vomit 1-day prior to admission and could not tolerate anything by mouth. Historically, patient has a history of noncompliance with insulin and will only take meds when she checks her sugars that often run in 200-300s. Previous admission was exacerbated by URI and reports symptoms of allergies (ie cough, sneezing, congestion) that spontaneously resolved approximately 2 weeks prior.

Imaging: CT abdomen/pelvis

1.) Prominence of the pancreatic tail region with regional moderate to severe adjacent strand change which is most suggestive of severe pancreatitis.
2.) Indistinct, prominent appearance of the bilateral kidneys raising concern for possible pyelonephritis.
3.) Moderate urinary bladder wall thickening suspicious for UTI and/or cystitis
4.) Central and L-paracentral posterior disc bulge at L5-S1; concern for cauda equina syndrome

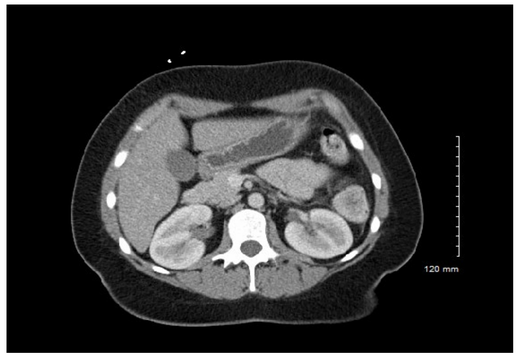

Figure 1. Axial field demonstrating acute findings of pancreas

Hospital Course

Our patient was immediately admitted to the ICU. Pulmonology and Gastroenterology were both consulted for possible need of MRCP. Insulin drip initiated and maintained. Aggressive fluid resuscitation with repletion of electrolyte was started and patient was to avoid oral intake at all costs. BMPs every 4 hours, 8-hour phosphorus levels, and every 12-hour triglyceride levels were closely monitored. Daily ABGs were done to ensure patient was not acidotic. The insulin drip was appropriately titrated based off blood glucose levels. Rosuvastatin was immediately started and when triglyceride levels were less than 100, gemfibrozil therapy initiated. Due to the need of the insulin drip and close monitoring, this patient remained in the ICU until triglyceride levels were less than 500. The ultimate goal in this case was to avoid necrotizing pancreatitis and multi organ failure. Our patient was in the ICU for a total of 12 days and 5 additional days were spent in a step-down unit until stable for discharge with severity and diabetes education appropriately relayed and understood by our patient and close in office follow up was scheduled.

Notable Laboratory Findings on Admission

Lab	Results
- Blood Glucose Level	282
- B-Hydroxybutyrate	87.1
- Lipase	1945
- Triglycerides	>4000
- Anion Gap	23
- ABG	PH: 7.04; PCO2: 12; PO2: 132

Table 2. Notable laboratory findings upon arrival to ED

Pathology

Triglycerides themselves are not toxic; instead, toxicity occurs as the are broken down to fatty acids by lipases. The severity depends on inflammatory responses along with lipotoxicity from triglyceride hydrolysis. Severe hypertriglyceridemia with lipase levels greater than 3 times the upper limit of normal can cause extremely high fatty acid levels which directly activated toll-like receptors 2&4 by free fatty acids. There are no clear biomarkers to determine lipotoxicity independent of acute pancreatitis with normal triglyceride levels.

Subtype	Frequency
Primary Etiologies	>90%
-Familial dyslipidemias	Chylomicron, VLDL, HTG
-Chylomicronemia	Younger age; Type III diabetes
-VLDL & HTG	Induced by alcohol, fatty meal, med
Secondary Etiologies	<10%
-Poorly controlled DMII	<1%
-Estrogen	<1%
-Tamoxifen	<1%
-Propofol	<1%
-Beta Blockers	<1%
-Pregnancy	<1%

Table 1. Common causes of Hypertriglyceride Induced Pancreatitis

Discussion

Overall, in Type II DM, insulin resistance leads to an over production and reduced catabolism of large triglyceride rich lipoproteins. Thus, insulin resistance leads to a decrease in lipoprotein lipase activity in both adipose tissue and muscle. This lack of insulin cascades into the triad of DKA, HTG, and acute pancreatitis. The increased production of very-low-density lipoprotein (VLDL) from the liver because of increased lipolysis, release and deliver of free fatty acids to the liver, coupled with the inhibition of lipoprotein lipase into the peripheral tissue. Lipoprotein apheresis, plasmapheresis, or plasma exchange have been used in patients with pancreatitis secondary to hypertriglyceridemia however, there have benefits in morbidity and mortality have not been shown, also the cost and potential adverse effects give additional reason to avoid it.

Conclusions

In our patient's case, the very rare hypertriglyceride induced acute pancreatitis was caused by her poorly controlled diabetes with a blood glucose greater than 500 and an A1c of 12.2. Not only was that a contributing factor, she was also on Seroquel which could have induced such situation long-term. As seen in this patient, pancreatitis occurred with severe acidosis and high anion gap metabolic acidosis. High triglycerides are a common cause, but unusual as fatty acid oxidation and ketone formation increases more than sole triglyceride production. This lack of insulin lead to lipolysis and fatty acid transport thus raising triglycerides. Our patient was extensively counseled on abstaining from fatty, greasy foods, alcohol, and very close monitoring of blood glucose levels and insulin administration. She currently is on an Omnipod pump with insulin administration with a near 50-pound weight loss. It was also important to note that since this was her second DKA admission with concomitant pancreatitis she is at ta great risk of organ failure if she were to remain poorly controlled or to suffer another episode of DKA.

Contact

Elvira Kazemyan, MD
Memorial/LSUHSC Family Medicine Residency
1525 Oak Park Blvd., Lake Charles, LA 70601
ekazemyan@lcmh.com
(337) 494-2023

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