May 6th, 12:00 AM

Triad of Acute Pancreatitis Coexisting with Diabetic Ketoacidosis and Very Severe Hypertriglyceridemia Successfully Resolved with Insulin Therapy Alone

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Introduction

- Diabetic ketoacidosis (DKA) is known to cause mild elevations in triglyceride levels.
- Severe hypertriglyceridemia (HTG) is a well-known cause of acute pancreatitis however, in unique cases, DKA has been shown to cause severe HTG leading to the development of acute pancreatitis (AP).
- HTG causing AP only accounts for 1-4% of cases of AP
- From the few cases in literature that have reported this triad of DKA, HTG, and AP, even fewer have shown to be successfully treated with insulin therapy alone with the severe degree of HTG that we discuss.

Case Presentation

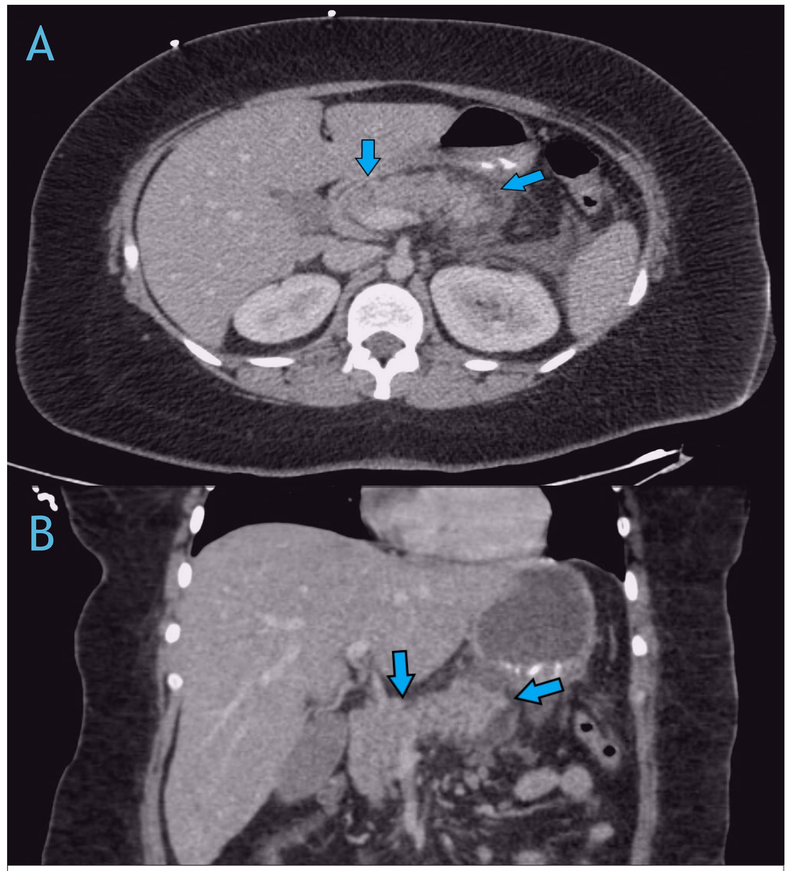
- A 20-year-old Caucasian female who presented for evaluation of 2-day history of worsening epigastric pain, associated with nausea and vomiting. She has a pmh significant for SLE and a family history significant for HLD. Social history was negative for alcohol or tobacco dependence. Patient had a RUQ ultrasound which showed AP and a confirmatory CT abdomen and pelvis which showed severe AP of the distal body and tail of the pancreas.
- Labs was significant for a blood glucose 370 mg/dL, high anion gap metabolic acidosis (anion gap 21), lipase of 1095 U/L, triglyceride level of 16076 mg/dL, cholesterol 1629 mg/dL and HbA1c of 11.2%.
- She was diagnosed with AP and admitted to the ICU for treatment with intravenous insulin infusion for the treatment of DKA and HTG with aggressive IV fluids and electrolyte replacement.
- Patient's DKA resolved and her triglyceride levels decreased from 16076 to 283 mg/dL over the next 5 days.

	Total Cholesterol	HDL	LDL	Triglycerides	VLDL
Day 1	1629	24	203	16076	-
Day 2	-	-	-	906	-
Day 2	-	-	-	759	-
Day 3	-	-	-	474	-
Day 4	377	51	170	356	71
Day 5	-	-	-	283	-

Pathophysiology

- DKA is known to cause mild elevations in triglyceride levels via insulin deficiency however severe HTG (> 2000 mg/dL) is rare.
- Insulin deficiency seen in DKA leads to the production of free fatty acids and amino acids from adipose and muscle tissue.
- As these free fatty acids (FFA) are circulating in the blood they get taken up by the liver and leads to increased production of very low density lipoproteins (VLDL) which causes HTG.
- Insulin deficiency also reduces the activity of lipoprotein lipase in peripheral tissues leading to decreased removal of VLDL from plasma which allows more time for liver uptake. (1)
- Even though the true mechanism is still not clear, It has been proposed that high concentrations of toxic free fatty acids generated from the breakdown of triglycerides by pancreatic lipase contributes to pancreatic cell injury (2)

Imaging and Labs



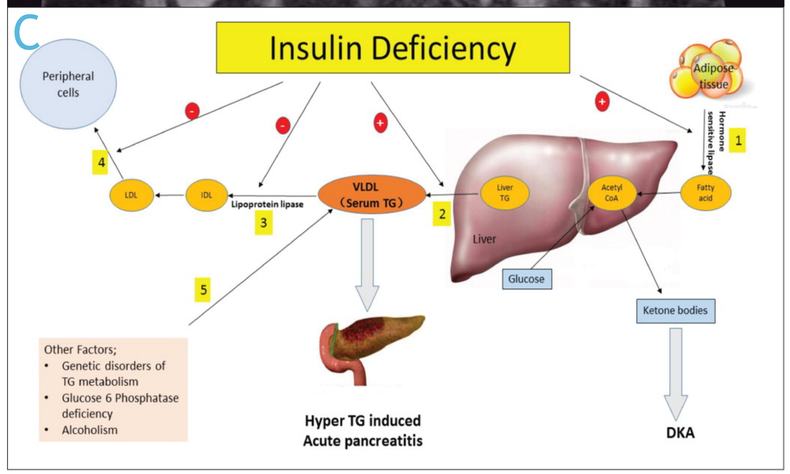


Figure 1. (A, B): Computed tomography images showing pancreatic inflammation and edema (arrows) surrounding the head and tail of the pancreas (C): The role of insulin deficiency in causing HTG leading to AP. Decreased inhibition hormone-sensitive lipase leads to increased FFA which will be converted to serum HTG causing AP. (3)

Treatment

- Management includes aggressive fluid administration, electrolyte repletion and intravenous insulin infusion. Studies show that other treatment options include low-molecular-weight heparin, therapeutic plasma exchange, and double-filtration. (3)
- Plasmapheresis is considered especially those with organ failure such as renal insufficiency, who failed with conservative treatment as mentioned above. (4)
- The use of lipid-lowering agents, such as fibrates, can be used in most conditions accompanied by HTG but not beneficial in patients with lipoprotein lipase deficiency (5)
- Omega oils are essential fatty acid naturally present in fish, algae, and other seafood; their use as a TG-lowering drug in the management of severe HTG is recommended by both the National Lipid Association and the American Heart Association (6)

Discussion

- Previous studies have shown that an estimated mortality rate of up to 80% can be seen in patients with AP and co-existing DKA (2).
- Therapeutic plasma exchange (TPE) or plasmapheresis is the modality of choice for treating patients with pancreatitis due to severe HTG as it involves the removal of plasma and substitutes it with a colloid solution such as donated plasma or albumin.
- TPE is associated with increased cost, availability issues, and its mortality benefits have not been completely established. In many cases, patients are transferred to another institution for management.
- IV insulin therapy is used until triglyceride levels are less than 500 mg/dL, however its use in severely elevated HTG has not been well studied in the literature. In our case, we were able to successfully and safely lower severely elevated triglyceride levels without the need for plasmapheresis.

Conclusion

In our case, we have treated a triad of DKA, AP and severe HTG with a value of 16076 mg/dL with insulin therapy alone which has not been documented in our literature search.

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