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# Unsuspected Diabetic Ketoacidosis Complicating Acute Pancreatitis due to Hypertriglyceridemia

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**Abstract** Hypertriglyceridemia (HTG) is the third most common cause of acute pancreatitis, behind alcoholism and gallstones. We present a case of a 23 year old male with a history of hypertriglyceridemia presenting to our emergency department with complaints of nausea, vomitings and abdominal pain. Upon examination, he was toxic in appearance and tachycardic in the 130s bpm. Abdominal exam was remarkable for tenderness in the epigastric region with no peritoneal signs. Labs were remarkable for a leukocytosis of 20000/mm$^3$, random glucose of 419 mg/dL, Bicarbonate 8 mEq/L with AG of 18, lipase of 26000 IU/L, triglycerides of 3700 mg/dL. US abdomen showed no evidence of gall bladder stones. CT scan revealed moderate edema of pancreas and duodenum, with no stones, masses of CD dilatation. Patient was initially treated for acute pancreatitis with aggressive hydration and pain management. Over the hospital stay, patient continued to be tachycardic and became increasingly drowsy. Diabetic ketoacidosis (DKA), complicating pancreatitis, was entertained at this point and the patient was started on insulin infusion. A Quinton catheter was placed for therapeutic plasma exchange for severe hypertriglyceridemia. Patient was also started on heparin drip to assist with rapid clearance of triglycerides. Patient's condition significantly improved the next day with resolution of acidosis and amelioration of pain. This case illustrates the fact that DKA, Pancreatitis and HTG can occur as a clinical triad and it is important to diagnose an underlying DKA complicating HTG pancreatitis in patients with a rapidly deteriorating clinical course, profound metabolic acidosis and high glucose levels.

**Keywords** Triglycerides, Plasma exchange, Heparin, Abdominal pain

## 1. Case Report

A 23 year old gentleman with a past medical history of morbid obesity and HTG presented to our emergency department with complaints of nausea, vomiting and abdominal pain for a day. He described the pain to be sharp in the upper abdomen, radiating to the back. He reports drinking about 5 beers over the weekend. He had a similar episode of pain two weeks prior which resolved spontaneously. Upon exam, he was afebrile and tachycardic with heart rate in the mid-130s. Abdominal exam was remarkable for tenderness in the epigastric with no peritoneal signs. Labs were remarkable for a leukocytosis of 20000/mm$^3$; Na 130 mEq/L, random glucose of 419 mg/dL, bicarbonate 8 mEq/L with AG of 18, venous pH 7.3, venous lactate 2.0 mg/dL, lipase of 26000 IU/L, triglycerides of 3700 mg/dL. Urinalysis was positive for glycosuria, moderate proteinuria and ketonuria. US abdomen showed no evidence of gall bladder stones. CT scan revealed moderate edema of pancreas and duodenum, with no stones, masses or common bile duct dilatation. Patient was initially treated for acute pancreatitis with aggressive hydration and pain management. The ketonuria was attributed to starvation ketoacidosis. Throughout visit, patient continued to be tachycardic and became drowsy. Pt was transferred to the intensive care unit. Arterial blood gas revealed pH 7.25, $paCO_2$ 20 mmHg, $paO_2$ 80mmHg with lactate 1.8 mg/dL. Random glucose was 455 mg/dL. DKA was entertained and patient started on a continuous insulin infusion. A Quinton catheter was placed for therapeutic plasma exchange for severe HTG. Patient was also started on heparin drip to assist with rapid clearance of triglycerides. Patient's condition significantly improved the next day with a resolution of acidosis and amelioration of pain. Resolution of abdominal pain with correction of acidosis and lack of peritoneal signs on exam pointed towards DKA pseudoperitonitis. Therapeutic plasma exchange sessions were discontinued after TG dropped to the desired goal of <300 mg/dl. Complications included a thrombus of the Quinton catheter and left upper extremity for which he was started on rivaroxaban. Patient was transitioned from insulin infusion to a basal/bolus regimen and discharged on Insulin, Gemfibrozil, Omega-3 fatty acids and rivaroxaban.

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## 2. Discussion

Hypertriglyceridemia is the third most common cause of acute pancreatitis, behind alcoholism and gallstones, constituting about 5% of cases [1]. Diabetic ketoacidosis, HTG and pancreatitis occurring together as a clinical triad is infrequent and few case reports have been reported thus far. HTG induced pancreatitis along with DKA may be the initial presentation of unsuspected Diabetes or can complicate the natural history of uncontrolled diabetes [2, 3]. A prospective study involving 100 consecutive DKA patients, reported association with acute pancreatitis in about 10-15% of the patients [4].

DKA, Hypertriglyceridemia and acute pancreatitis is an interesting and rarely reported triad in medical literature [4, 5]. Insulin deficiency in DKA leads to accelerated lipolysis and inhibition of lipoprotein lipase in the peripheral tissues leading to elevated triglycerides precipitating pancreatitis [1, 4]. HTG pancreatitis usually have an underlying abnormality in lipid metabolism (as in our patient), compounded by a secondary complicating factor such as diabetes or alcoholism, raising the TG levels to levels sufficient to precipitate pancreatitis [6]. HTG pancreatitis is believed to be caused by acinar injury from the toxicity of unbound FFAs released from the action of pancreatic lipase activity on TGs [7].

DKA is characterized by high blood glucose levels, and ketoacidosis resulting in ketonemia and ketonuria. DKA and alcoholic ketoacidosis can present with profound metabolic acidosis, but starvation ketosis rarely presents with bicarbonate levels lower than 18 mEq/L, an important biochemical clue to the etiology of the ketoacidosis [8]. DKA complicating pancreatitis can clinically mask underlying complicating pancreatitis, present with higher lipase, amylase and TG levels and pursue a more severe clinical course with overestimating Ranson's prognostic scores compared to acute pancreatitis in isolation [4, 9]. DKA independently raises lipase apart from amylase making them less specific biochemical markers for pancreatitis [8]. Hypertriglyceridemia is a noted biochemical epiphenomenon associated with pancreatitis in up to 50% of the cases [10, 11] and it is important to differentiate this mild HTG from the marked HTG required to precipitate pancreatitis. Studies have shown TG levels to be greater than 1500 mg/dl to precipitate an episode of pancreatitis [12]. Lipoproteins are traditionally classified into five patterns based on electrophoretic motility patterns with Type IV being the most common lipoprotein abnormality in patients with DKA patients who present with HTG pancreatitis [11].

Hypertriglyceridemia pancreatitis should be treated promptly insulin infusion and IV fluids [11, 13, 14]. Insulin stimulates lipoprotein lipase levels on the endothelium leading to rapid clearance of triglycerides in the blood. Therapy with hypocaloric IV fluids causes further reduction of TG levels by decreasing the VLDL secretion from the liver [15]. Heparin infusion drip may also be associated with drop in TG levels by stimulating lipoprotein lipase into the circulation resulting in rapid clearance of TGs in the blood and has been used along with insulin to treat HTG [14]. However, theoretical concerns of release of FFAs from the action of LPL on TGs into the blood and reports of delayed HTG due to transient depletion of tissue LPL stores have raised scrutiny over the use of heparin in HTG pancreatitis [16]. Case reports and summaries support the use of lipid apheresis as a therapeutic option for the rapid clearance of TGs [17]. It is recommended to improve the TG levels to less than 500 mg/dl to avert further episodes of HTG pancreatitis [12].

This case illustrates the fact that DKA, Pancreatitis and HTG occur as a clinical triad and it is important to diagnose an underlying DKA complicating HTG pancreatitis in patients with a rapidly deteriorating clinical course, profound metabolic ketoacidosis and high glucose levels. Family and personal history of primary lipid disorder can very helpful in further determining the cause of pancreatitis and in the long term management of HTG to prevent further episodes of recurrent pancreatitis.

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