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Diabetic Ketoacidosis and the Domino Effect

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABEF Bilal H. Shaikh
DEF Muneebah Sohaib
EF Raeda Alshantti
DE Francisco Barerra
E Farah S. Faridi
EF Natia Murvelashvili

Department of Internal Medicine, Presence Saint Francis Hospital, Evanston, IL, U.S.A.

Corresponding Author: Bilal H. Shaikh, e-mail: b.hussain1989@gmail.com
Conflict of interest: None declared

Patient: Male, 27
Final Diagnosis: Hypertriglyceridemia associated acute pancreatitis secondary to diabetic ketoacidosis
Symptoms: Abdominal pain
Medication: —
Clinical Procedure: —
Specialty: Endocrinology and Metabolism

Objective: Rare disease

Background: Severe hypertriglyceridemia is a well-known cause of acute pancreatitis. Mild elevations of triglyceride levels are common in patients presenting with diabetic ketoacidosis (DKA). Rarely, DKA can be accompanied by an elevation of serum triglyceride level severe enough to lead to AP.

Case Report: We report one such case of a young diabetic male who presented with DKA that was complicated by hypertriglyceridemia-induced acute pancreatitis (HTGAP). We were able to treat the condition with a slightly prolonged infusion of intravenous (IV) regular insulin in an efficient and cost-effective manner with a good outcome.

Conclusions: From our experience, DKA-associated HTGAP can be rapidly, efficiently, and cost-effectively treated with IV regular insulin and close biochemical monitoring. A high index of suspicion for acute pancreatitis is necessary in patients with DKA, especially with co-existing hypertriglyceridemia; and all efforts should be made to diagnose it in a timely manner to prevent subsequent complications.

MeSH Keywords: Diabetic Ketoacidosis • Hypertriglyceridemia • Pancreatitis

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/911747

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Background

Hypertriglyceridemia is one of the 3 most common causes of acute pancreatitis (AP) [1,2]. Serum triglyceride levels above 1000 mg/dL are most frequently implicated [3]. It has been proposed that high concentrations of toxic free fatty acids generated from the breakdown of triglycerides by pancreatic lipase contribute to pancreatic cell injury [4]. Acute elevations of circulating triglycerides may be observed in diabetic ketoacidosis (DKA). This is attributed to increased lipolysis, and decreased activity of the lipoprotein lipase enzyme in the capillary endothelial cells of adipose tissue, as a result of insulin deficiency. Occasionally, DKA may present with severe hypertriglyceridemia that leads to AP. A 4% incidence of hypertriglyceridemia-induced acute pancreatitis (HTGAP) was noted in one study that prospectively evaluated 100 DKA patients. The same study reported an 11% overall incidence of AP among these patients [5].

Case Report

We present the case of a 27-year old, obese, diabetic male who presented to the emergency department (ED) with mid-abdominal pain, nausea, and emesis. He reported no previous history of cholelithiasis, dyslipidemia, or recent alcohol use. He admitted to non-compliance with his anti-diabetic medications. On examination, his abdomen was diffusely tender. Laboratory testing revealed blood glucose of 383 mg/dL, anion gap 23, serum bicarbonate 14 mmol/L, urine ketones 80 mg/dL and a pH of 7.23, consistent with DKA. Serum lipase was elevated to 2595 IU/L. Serum samples were lactescent in appearance. Initial serum triglyceride level was reported as >3000 mg/dL. An abdominal computed tomography (CT) scan showed marked induration surrounding the head of pancreas consistent with pancreatitis (Figure 1). Treatment for DKA with intravenous (IV) fluids, IV regular insulin infusion, and potassium supplementation was begun per standard protocol. Oral feeding was held. The anion-gap normalized 36 hours later. Serum triglyceride levels were followed every 12 hours, and a decreasing trend became apparent after 24 hours of continuous insulin infusion. We continued the IV insulin beyond resolution of DKA to treat the acute severe hypertriglyceridemia; 5% dextrose-0.45% saline was infused simultaneously to prevent hypoglycemia. Our goal of serum triglycerides lower than 500 mg/dl was reached after 80 hours of continuous IV insulin, following which a transition to subcutaneous long-acting insulin was made. Serum lipase had trended down to 200 IU/L by this time. As the patient's symptoms resolved, oral feeding was resumed.

Discussion

Patients with AP secondary to hypertriglyceridemia tend to have more severe symptoms compared to AP due to other causes. This was concluded in a recently published systematic review of 38 studies on hyperlipidemic pancreatitis [6]. One study also reported observations of more severe episodes of HTGAP with co-existing DKA [7]. Nielson et al. estimated a mortality rate of 80% in patients with AP and co-existing DKA [8]. Currently, there is a lack of published mortality statistics in patients with HTGAP and co-existing DKA.

In our case, medication non-compliance led to poor glycemic control and DKA, which in turn led to severe hypertriglyceridemia causing AP. This has rarely been reported in available literature.

In the management of HTGAP, lowering the serum triglyceride level is a priority. Therapeutic plasma exchange (TPE) is often the preferred option when available [9]. However, in our case, triglyceride levels dramatically declined only with IV insulin, so TPE was deferred. A few other authors have reported similar successes in treating the DKA-HTG-AP triad only with IV insulin infusion [10,11]. In one randomized controlled trial involving 66 HTGAP patients comparing 2 triglyceride lowering therapies, no improvement in clinical outcomes was found with the use of early high-volume hemofiltration (HVHF) over the use of a low molecular weight heparin and insulin combination. However, hospital costs were 2-fold higher for patients receiving HVHF [12].

Additionally, our patient's abdominal pain prompted a CT scan in the ED, which revealed findings of AP early and unexpectedly. Signs and symptoms of DKA might mask those of co-existing AP [5]. Since hypertriglyceridemia, non-specific elevations in serum amylase, and lipase are known to be common in DKA, a diagnosis of AP might easily be overlooked [5,13].

Conclusions

From our experience, DKA-associated HTGAP can be rapidly, efficiently, and cost-effectively treated with just IV regular insulin and close biochemical monitoring, rather than having to utilize either TPE or HVHF.

A high index of suspicion for AP is necessary in patients with DKA, especially with co-existing hypertriglyceridemia >1000 mg/dL, serum lipase >3 times normal, prior history of AP or severe signs and symptoms. A timely CT scan of the abdomen may help in diagnosing AP early and prevent subsequent complications.

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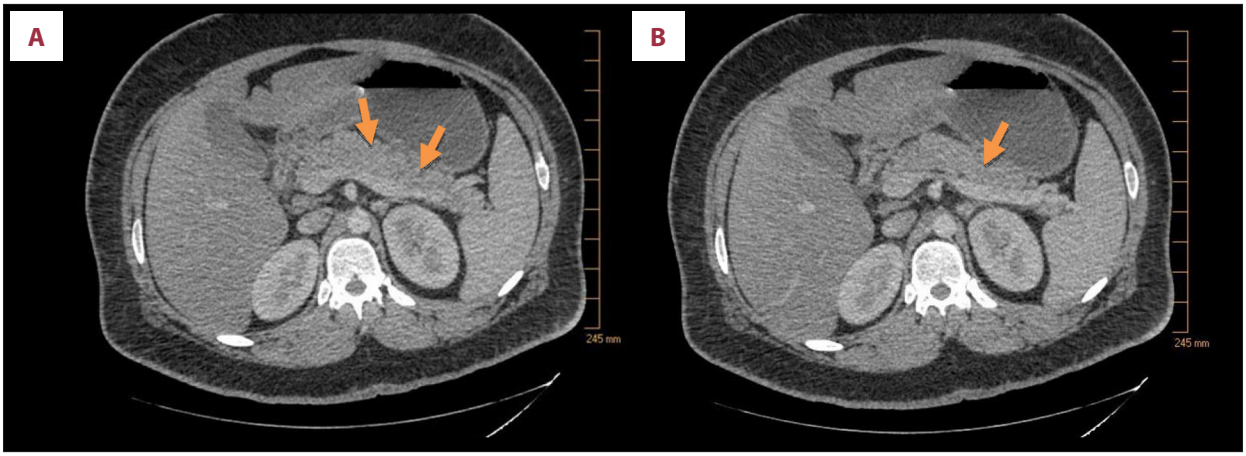

Figure 1. (**A, B**) Computed tomography images showing pancreatic inflammation (arrows) and induration surrounding the head of pancreas.

Conflict of interests

None.

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