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Case Report

Secondary Hypertriglyceridemia Due to Diabetic Ketoacidosis Causing Acute Pancreatitis in A Pediatric Patient: A Case Report

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Abstract

Background: Diabetic ketoacidosis is a complication of diabetes mellitus. Diabetic ketoacidosis occurs mainly due to an insulin impaired state causing Hypertriglyceridemia. Hypertriglyceridemia is the third primary reason for acute pancreatitis and is challenging to diagnose in the presence of diabetic ketoacidosis due to the similarity of clinical signs and symptoms with acute pancreatitis.

Case presentation: A 13-year-old girl, known case of type 1 diabetes mellitus, presented to the emergency department with epigastric pain and repeated vomiting. Initial laboratory findings revealed elevated levels of glucose, amylase, lipase, cholesterol and triglycerides. Urine ketones were positive, and the patient exhibited metabolic acidosis blood gases. Based on the clinical picture, diagnosis was suggestive of diabetic ketoacidosis and secondary hypertriglyceridemia leading to acute pancreatitis. The patient was admitted to the intensive care unit and treated for diabetic ketoacidosis as well as hypertriglyceridemia. The patient improved on the seventh day and was discharged home.

Conclusion: The triad of hypertriglyceridemia, diabetic ketoacidosis and acute pancreatitis is very challenging to diagnose, as each disorder is causative to the other. Management should be focused on all three causes and utilize insulin, hydration and triglyceride lowering medication, and lifestyle modification.

Keywords: Hypertriglyceridemia, acute pancreatitis, pediatric, diabetic ketoacidosis.

Introduction

Type 1 Diabetes Mellitus (T1DM) is a common endocrine and metabolic disease that affects various groups in the pediatric age range (1). According to the latest data, there are approximately 35 thousand children and adolescents suffering from T1DM in Saudi Arabia (1). Meanwhile, there is an overall 3% annual increase ofT1DM, with a projected 79 thousand children estimated to develop it every year around the world (2). Saudi Arabia has one of the highest prevalence rates with an estimated annual increase of 17% (2). Diabetes can cause various metabolic, endocrine and systemic complications if not treated efficiently (3). Diabetic Ketoacidosis (DKA) is a severe metabolic complication that can develop among T1DM, occurring in up to 40% in newly diagnosed cases (4, 5). Cerebral edema is a lethal complication that can happen as a result of uncontrolled instances of DKA (6).

Non-lethal complications may also occur, including Hypertriglyceridemia (HTG) and Acute Pancreatitis (AP). However, these are not usually seen in the Pediatric Intensive Care Unit (PICU) (5). HTG can be attributed to genetic, dietary and other physiological factors (7). HTG may cause AP in approximately4% of patients with diabetes that have an increased level of serum triglyceride more than 1,000 mg/dL (8, 9). Therefore, understanding and reporting such incidents is crucial to providing more data for future research. Here, we report a case of hypertriglyceridemia with acute pancreatitis in a pediatric case of diabetic ketoacidosis.

Case presentation:

A 13-years-old female, who has been a known case ofT1DM since she was three years old, presented to the Emergency Department (ED) of Taif Children Hospital with a one-day history of repeated vomiting and epigastric pain. In terms of her family history, her father has hypertension. However, there is no family history of diabetes, hyperlipidemia or any other disease. Her regular medications include Lantus 20 units and Apidra 6 units per meal. She was 137 cm tall and weighed 35 kg with a Body Mass Index (BMI) of 18.4 Kg/m^2.She was lethargic and confused but conscious with a Glasgow Coma Scale (GCS) score of 12/15.Her blood pressure was 85\65 mmHg, heart rate130 beats/min, respiratory rate 36/min and temperature 36.7° C.

She had had multiple admissions due to DKA but with no previous incidences of pancreatitis. The physical examination revealed an altered mental status, accompanied by rapid deep breathing and signs of severe dehydration. Abdominal examination revealed epigastric tenderness and her bowel sound was positive.

Initial laboratory investigations revealed a glucose level of 698.4 mg/dl. Urine ketones were positive, and the patient exhibiteda clinical picture of metabolic acidosis. Her arterial blood gas picture was pH 6.8, PCO_2 25 mmHg, PO_2 34 mmHg, and HCO_3- 4.3 and SaO_2 98.0%. In addition, herHbA1c was 12.8 %, White Blood Cell counts (WBCs) $23.5x10^9$/L, hemoglobin 13.8 g/dL, blood urea nitrogen 4.4 mmol/L, creatinine 105 mmol/l, sodium 135 mmol/l, potassium 5.4 mmol/L and chloride 100 mmol/l. Meanwhile, her Liver Function Tests (LFTs), and coagulation profile was within normal limits. Serum levels of amylase and lipase were 359 U/L and 309 U/L, respectively. In terms ofher lipid profile, her total cholesterol was 221 mg/dL, triglyceride 321.20mg/dL, measured Low-Density Lipoprotein Cholesterol (LDL-C) 143 mg/dL and High-Density Lipoprotein Cholesterol (HDL-C) 51.6 mg/Dl. She received normal saline bolus of 20ml/kg and was treated with insulin infusion, nasogastric tube and morphine and was admitted to the PICU.

The patient's general condition, metabolic acidosis, gradually improved bar a persistent moderate-to-severe abdominal pain and her diabetic ketoacidos is resolved over the next 24 hours. The following day, the patient developed spikes of fever with no signs of jaundice or respiratory distress. However, her abdominal examination was remarkable for epigastric tenderness and guarding and normal bowel sounds. An ultrasound examination was undertaken and reported no features of acute pancreatitis gallstone or acute appendicitis. Based on her clinical picture, the patient was diagnosed with diabetic ketoacidosis, hyperglycemia, and acute pancreatitis. After the DKA resolution, the patient continued on intravenous fluid maintenance and insulin infusion for three days until her amylase and lipase levels subsided. She started on a low lipid and diabetic diet. Her Arterial Blood Gases (ABG) were checked every 12 hours while her lipase, amylase and lipid profiles were checked every 24 hours, as shown in **Table 1.**

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On the seventh day of hospitalization, the patient improved and demonstrated normal serum lipase and amylase levels. She was discharged without any complications. She was followed up in the endocrine clinic where her pancreatic enzyme levels were measured as normal and her lipid profile improved in the second week following a low-fat diet managed by a clinical dietician.

Table 1: Laboratory findings during hospitalization

Laboratory tests	Reference range	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Lipase (u/L)	13-60	—	309	214	72.5	62	50
Amylase (u/L)	28-100	—	359	206	258	99	105
Cholesterol (mg/dL)	0-200	—	221	200.5	194.9	180	180
Triglyceride (Mg/dL)	0-150	—	321.2	227.4	321	577	345
pH	7.35-7.45	6.9	7.36	—	7.34	7.37	7.39
PCO_2(mmhg)	35-45	10.8	25.4	—	23.6	30	34
HCO_3 (mmol/L)	22-26	5.1	15	—	14.8	17	19

Discussion:

DKA is a common complication of T1DM, and it is highly threatening with serious potential clinical complications (10). Patients tend to develop lethargy, acidosis and ketone production that may lead to a coma (11). It is distinguished by fundamental insulin inadequacy that creates hyperglycemia, hyperosmolar dehydration, and increasing serum ketones.

Hyperlipidemia and AP are amongst the unusual DKA complications that can be considered non-lethal (10). HTG is a multifactorial condition which can be due to either a primary or a secondary cause (7). Secondary causes are usually the result of metabolic or endocrine conditions such as DKA, Cushing's syndrome, hypothyroidism or kidney diseases (7). Furthermore, abdominal pain presented in association with DKA is usually caused by acute pancreatitis which can lead to vomiting, nausea and ileus. Hyponatremia is a common occurrence in cases of DKA.

As a result of the osmotic flux, hyperglycemia induces a water shift from the intracellular to the extracellular compartments, causing dilutional hyponatremia (12). As our patient was exhibiting abdominal pain, signs of dehydration, hyperglycemia, metabolic acidosis and mild hyponatremia with a sodium level of 135 mmol/L (Normal range: 136 -145 mmol/L), a diagnosis of DKA was established. Acute pancreatitis diagnosis in DKA is challenging (13). Nausea and vomiting are the most common symptoms, followed by abdominal pain that mimics acute pancreatitis pain, which is epigastric in origin (13). Moreover, around one-fourth of these cases have raised serum lipase and amylase levels without any clinical or radiological confirmation of AP (13). Our patient was admitted with a one-day history of repeated vomiting and epigastric pain and laboratory investigations reported a five-fold increase in lipase levels and a three-fold increase in amylase levels, with no signs of pancreatitis in the ultrasound scan.

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The triad of HTG, DKA and AP are very challenging and awareness of the root cause is difficult as amylase and lipase levels increase in both acute pancreatitis and DKA (14). Pathophysiologically, triglycerides are carried in circulation by specific lipoproteins that are released from the small intestine and hydrolyzed in the peripheral tissues (7). Afterwards, lipolysis occurs, causing the release of fatty acids. Consequently, the excess release of fatty acids causes hyperviscosity which can lead to acidosis and ischemia to the pancreatic capillary beds (15). In return, the ischemia and inflammation cause the pancreas to release high levels of amylase and lipase. A marked increase of triglyceride levels of more than 500 mg/dL increases the risk of developing pancreatitis, creating a vicious triad circle (15). Therefore, it is recommended that all three causes are addressed altogether via proper hydration, insulin and a lowering of triglyceride levels via lifestyle modification and medications including statins and fib rates (7, 15).

The treatment of HTG-induced acute pancreatitis is similar to any other cause of acute pancreatitis, and includes adequate hydration, pain management and decreased oral in take (16). There are no specific treatment guidelines for HTG, but insulin infusion is often useful to reduce triglyceride levels (16). Lipoprotein lipase activity is increased by insulin infusion, which degrades TG-rich chylomicrons and VLDL particles (16). Our case was primarily controlled via insulin infusion and intravenous fluid maintenance. The patient also had a low lipid and diabetic diet recommended. She had improved by the seventh day and was released without any complications. She was advised to follow up in our endocrine clinic for testing of her pancreatic enzyme and lipid levels, which had improved after two weeks of following up.

Conclusion:

The triad of hypertriglyceridemia, acute pancreatitis and diabetic ketoacidosis is very important and challenging to manage in pediatric patients. As elevated levels of serum lipase and amylase are not common in diabetic ketoacidosis, acute pancreatitis can be caused by severe hypertriglyceridemia. Insulin infusion is the leading choice for treatment, while intravenous fluid maintenance is also essential for completing recovery. A low lipid diabetic diet is highly recommended and necessary when controlling high levels of triglyceride, especially in cases of diabetic ketoacidosis.

Disclosure:

Statement:

The authors declare no conflict of interest.

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None.

Ethical consideration:

Written informed consent was obtained from the patient's guardians for publication purposes as long as personal information remains protected and anonymous. Ethical Approval was obtained from Children Hospital at Taif, Saudi Arabia to publish this case.

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