CASE REPORT

A Rare Case of Diabetic Ketoacidosis with Type-1 Diabetes Mellitus with Bowel Obstruction

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ABSTRACT
Diabetic ketoacidosis (DKA) is a common consequence of electrolyte and dehydration abnormalities that affect individuals with type-1 diabetes more commonly. However, mesenteric bands are extremely rare and usually observed in childhood. We here present a case of a male patient aged 19 years who represented with fever, abdominal pain, nausea, and vomiting(non-projectile). The laboratory revealed metabolic acidosis, high blood glucose, urine glucose and strongly positive high circulating blood ketones. Based on the laboratory findings patient has confirmed DKA. Despite correction of acidosis, the abdominal pain occurred for which an ultrasonography of the abdomen was performed which suggested features of bowel obstruction and exploratory laparotomy was performed which revealed multiple mesenteric bands present at various levels of small bowel with largest band present at the terminal part of ileum obstructing the small bowel. Mesenteric bands should thus be suspected in patients with diabetic ketoacidosis if the pain is persistent despite correction of acidosis.

Keywords: Diabetes; Hypokalaemia; Insulin; Ketoacidosis; Ketone bodies

INTRODUCTION

Diabetic ketoacidosis (DKA) is a biochemical triad of hyperglycaemia, ketoacidosis and ketonaemia and one of the potentially life-threatening acute metabolic complications of diabetes mellitus.[1]Abdominal pain, sometimes mimicking an acute abdomen, is a frequent manifestation in patients with DKA.[2] DKA has a case fatality rate of 1 to 5 percent. Although the highest rate of mortality is in older adults and persons with comorbid conditions, DKA is the leading cause of death in persons younger than 24 years with diabetes, most often because of cerebral edema.[3]

Abdominal pain is a frequent DKA manifestation (present in 40-75% of the cases) and its prevalence increases as arterial pH and serum bicarbonate levels decline. Furthermore, abdominal rebound tenderness, suggesting the presence of an acute abdomen, affects 12% of DKA patients with abdominal pain. Many mechanisms have been suggested to underlie the abdominal symptoms in DKA, namely acute hyperglycemia mediated impaired gastrointestinal motility (esophageal, gastric and gallbladder), rapid expansion of the hepatic capsule, and mesenteric ischemia precipitated by volume depletion.[4]

Laboratory evaluation of the patient with ketoacidosis and abdominal pain shouldinclude an electrocardiogram, flat and upright abdominal films, complete bloodcount, and measurement of serum enzymes (amylase, lipase, alkaline phosphatase, SGOT, SGPT, CPK isoenzymes), in addition to measurements of glucose, pH, electrolytes, and urea nitrogen. This evaluation will help in identifying diabetic patients with abdominal pain secondary to atypical presentation of myocardial infarction, intestinal perforation, gastric atony, generalized ileus, or obstruction.[5]Furthermore, there is a need to improve patient education and attendance at clinics and to raise awareness of diabetic ketoacidosis and its management amongst healthcare professionals. The mortality rate remains high in developing countries and among non-hospitalized patients.[6]

We here present a case of a male patient aged 19 years in which clinical symptoms and laboratory investigations revealed a case of diabetic ketoacidosis and further despite correction of acidosis, the abdominal pain was reported for which an ultrasonography of the abdomen was performed which suggested features of bowel obstruction and

further, laparotomy confirmed a sub acute intestinal obstruction (SAIO) which is a rare complication of DKA.

CASE REPORT

A 19 years old male patient with a history of type 1 diabetes mellitus presented with fever, abdominal pain, nausea, and vomiting (non-projectile) for 8 days. The patient's vital signs on presentation were a temperature of 37.2^0C, heart rate of 102 beats/min, respiratory rate of 28 breaths/min, blood pressure of 132/80 mmHg, and oxygen saturation of 97 % on room air.

The laboratory results reported that patient had severe metabolic acidosis, high blood glucose, urinary glucose, and strongly positive high circulating blood ketones (table 1).

Table 1: Laboratory parameters at the time of admission and at the time of discharge

Laboratory parameters		At the time of admission	At 18th day
CBC	Hb	12.4 g/dl	10.4g/dl
	WBC-	$11.97x10^3$	$9.94x10^3$
	RBC	4.68	3.9
	Platelet	1.73 lakh/mm^3	3.9 lakh/mm^3
Serum blood sugar	**Serum blood sugar**	656mg/dl	84 (Fasting)
	HbA1c	>14	
Renal function	Urea	35.1 mg/dl	42 mg/dl
	Creatinine	0.6 mg/dl	0.6 mg/dl
Arterial blood Gas (ABG)	pH	7.00	7.46
	PCO_2	15 mmHg	37mmHg
	PO_2	130 mmHg	83 mmHg
	BE	26.2 mmol/L	83 mmHg
	HCO_3	3.9 mmol/L	25 mmol/L
Urine analysis	Pus cells	1-2	1-2
	Urine sugar	2+	1+
	Urine ketone	3+	negative
Liver function test	Total protein	6.2 g/dl	5.8g/dl
	Albumin	3.5 g/dl	Albumin 2.9 g/dl
	Globulin	2.7 g/dl	Globulin 2.9 g/dl
	SGOT	79 U/L	79 U/L
	SGPT	165 U/L	165 U/L
	ALP	304 U/L	108 U/L
Serum electrolytes	Sodium	137 mmol/L	140 mmol/L
	Potassium	3.5 mmol/L	4.5 mmol/L
	Chloride	92 mmol/L	98 mmol/L

Based on the laboratory findings patient confirmed with DKA. The patient was aggressively resuscitated with isotonic crystalloid solution and started insulin infusion. After the initiation of insulin treatment, potassium levels were shifted intracellularly and serum levels declined and patient has developed with hypokalaemia which was corrected along with glucose levels. He was treated empirically for sepsis with broad spectrumantibiotics.

On5thday, patient started complaining of severe pain in abdomen which was associated with constipation, X-Ray Abdomen and USG abdomen was suggested of features of bowel obstruction for which he was started on conservative management but patient did not improve, after surgery opinion patient was taken for exploratory laparotomy which revealed multiple mesenteric bands located at various levels of small bowel, with largest band present at terminal part of ileum obstructing the small bowel. The band was resected; the postoperative course was uneventful. The patient reported no previous history of abdominal surgery or trauma.

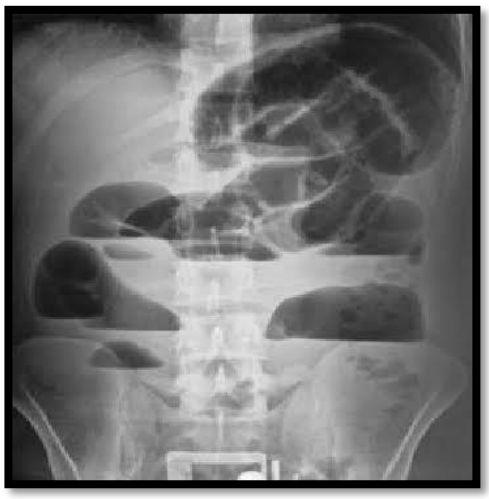

Figure1: X-ray abdomen shows multiple fluid level

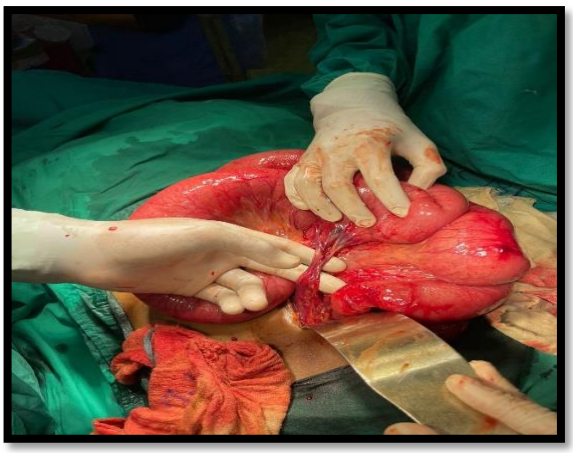

Figure 2:Shows the mesenteric bands

On 18th day, patient clinically and vitally stable. Laboratory parameters (table 1) were normal, patient was shifted to ward for observation and was later discharged.

DISCUSSION

The diagnostic criteria for diabetic ketoacidosis are: (i) ketonaemia 3 mmol ⁄ l and over or significant ketonuria (more than 2 + on standard urine sticks); (ii) blood glucose over 11 mmol ⁄ l or known diabetes mellitus; (iii) venous bicarbonate (HCO_3) below 15 mmol ⁄ l and ⁄ or venous pH less than 7.3. Savage MW In the present case, urine sugar was 2+ and ketonuria was 3+, serum blood sugar was 656mg/dl and HbA1c was >14, venous bicarbonate (HCO_3) was 3.9 mmol/L and pH was 7.00 which confirmed the diagnostic criteria for diabetic ketoacidosis.

DKA results from insulin deficiency from new-onset diabetes, insulin noncompliance, prescription or illicit drug use, and increased insulin need because of infection.[3]In the present case, insulin demand was increased due to sepsis which was further treated empirically with broad spectrum antibiotics.

This insulin deficiency stimulates the elevation of the counterregulatory hormones (glucagon, catecholamines, cortisol, and growth hormone)[3] and peripheral insulin resistance lead to hyperglycemia, dehydration, ketosis, and electrolyte imbalance which underlie the pathophysiology of DKA.[7]Without the ability to use glucose, the body needs alternative energy sources. Lipase activity increases, causing a breakdown of adipose tissue that yields free fatty acids. These components are converted to acetyl coenzyme A, some of which enter the Krebs cycle for energy production; the remainder are broken down into ketones (acetone, acetoacetate, and β-hydroxybutyrate). Ketones can be used for energy, but accumulate rapidly. Glycogen and proteins are catabolized to form glucose. Together, these factors promote hyperglycemia, which leads to an osmotic diuresis resulting in dehydration, metabolic acidosis, and a hyperosmolar state.[3] Hence, hyperglycemia-induced osmotic diuresis, if not accompanied by sufficient oral fluid intake, leads to dehydration, hyperosmolarity, electrolyte loss, and subsequent decrease in glomerular filtration. With decline in renal function, glycosuria diminishes and hyperglycemia/hyperosmolality worsens. With impaired insulin action and hyperosmolality, utilization of potassium by skeletal muscle is markedly diminished leading to intracellular potassium depletion. Also, potassium is lost via osmotic diuresis causing profound total body potassium deficiency. Therefore, DKA patients can present with broad range of serum potassium concentrations. Nevertheless, a “normal” plasma potassium concentration may indicate that potassium stores in the body are severely diminished and the institution of insulin therapy and correction of hyperglycemia will lead to future hypokalemia.[7]

The patients with severe DKA had more electrolyte abnormalities (hypokalemia, hypomagnesemia, hypophosphatemia) compared with the mild and moderate forms of the disease.[8]In the present case,after the initiation of insulin treatment, potassium levels were shifted intracellularly and serum levels declined and patient has developed with hypokalaemia which was corrected along with glucose levels. He was treated empirically for sepsis with broad spectrum antibiotics.

The abdominal pain is significantly associated with metabolic acidosis, rather than hyperglycemia or dehydration. The cause of abdominal pain in most patients may not be determined by clinical and imaging studies, but the pain can be relieved by itself after the elimination of ketoacidosis. In the case of acidosis correction and normal blood glucose, the patient still has severe abdominal pain and nausea and vomiting. However, once upon rehydration, the ketone body in the urine disappears, the patient's abdominal pain symptoms can be improved immediately.[9]But in the present case abdominal pain did not subside, patient complained of severe pain in abdomen which was associated with constipation, X-Ray Abdomen and USG abdomen was suggested of features of bowel obstruction for which exploratory laparotomy was performed which revealed multiple mesenteric bands located at various levels of small bowel, with largest band present at terminal part of ileum obstructing the small bowel. Sub acute intestinal obstruction (SAIO) implies incomplete obstruction. It is one of the important causes of morbidity and mortality in the surgical practice. The latter is true in patients presenting as sub-acute intestinal obstruction (SAIO) with atypical features that cause delay in diagnosis. Diagnosis is difficult, and in about 50% of cases it is made intra-operatively. Delayed diagnosis, co-morbidities and advanced age are the causes of the high related mortality rate (7.5-15%).[10]

An intestinal obstruction is caused by one of three mechanisms: compression of the bowel, partial volvulus, or entrapment of an intestinal loop between the band and mesentery.[11] In the present case,

intestinal obstruction was due to mesenteric bands as the exploratory laparotomy revealed multiple mesenteric bands located at various levels of small bowel, with largest band present at terminal part of ileum obstructing the small bowel.

Intestinal bands caused by inflammation and surgery in pediatric patients are common and can lead to intestinal obstruction. However, anomalous congenital bands that are not related to abdominal conditions such as laparotomy, trauma, or peritonitis are extremely rare causes of intestinal obstruction in children. Their exact incidence is unknown. The etiology of anomalous congenital bands is still unknown, but they are not secondary to known embryologic remnants such as omphalomesenteric duct or vitelline vessel remnants.[11]In the present case, sub acute intestinal obstruction (SAIO) is a rare complication of DKA.

Literature evaluation reports another case with of DKA with acute colonic pseudobstruction. Afocused evaluation for potential aetiologies of diabetic ketoacidosis is warranted in all patients in order to not miss rare causes.[12]

CONCLUSION

This case draws clinician attentionon how important the investigation of a clinical precipitant is a fundamental component in the management of diabetic ketoacidosis. SAIO (Sub acute intestinal obstruction) is a rare complication of DKA, early initiation of management of diabetic ketoacidosis and surgical intervention in SAIO has a better outcome and prognosis for the patient. Hence, sub acute intestinal obstruction due to mesentric bands should thus be suspected in patients with diabetic ketoacidosis if the pain is persistent despite correction of acidosis.

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