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Case Report

Recurrent bowel necrosis from non-occlusive mesenteric ischemia induced by diabetes ketoacidosis in an adult patient causing mortality: a rare cause of mortality associated with poorly controlled diabetes in adults

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ABSTRACT

Diabetes ketoacidosis (DKA) is associated with severe dehydration resulting in non-obstructive mesenteric ischemia (NOMI), which can contribute to severe patient complications. Recurrent episodes of NOMI with DKA in adults can be associated with mortality rarely. We presented a case of an adult patient with poorly controlled insulin-dependent type 2 diabetes mellitus due to poor socioeconomic background who presented to the emergency department with multiple episodes of bowel necrosis causing mortality.

Keywords: Diabetes ketoacidosis, Non-occlusive mesenteric ischemia, Type 2 diabetes mellitus, Bowl necrosis, Mortality

INTRODUCTION

Diabetes ketoacidosis (DKA) is one of the commonest and most severe acute complications associated with T1DM in childhood.[1] DKA and its complications can rarely develop in adults with poorly controlled insulin-dependent T2DM associated with lower socioeconomic status.[2] Patients commonly present to the emergency department with nausea, vomiting, polyuria, polydipsia, weakness, and sudden onset of abdominal pain. Signs of dehydration, hypotension, tachycardia and tachypnoea are the typical findings of physical examination.[3]

Abdominal pain can present in 40-75% of DKA cases due to various reasons, and the prevalence of abdominal pain rises with acidosis in the blood.[3] Non-occlusive mesenteric ischemia (NOMI) is a known rare complication of DKA that can present with severe abdominal pain, which can cause necrosis of the small bowel and large bowel due to sudden volume depletion, causing severe morbidity and mortality.[3,4] Literature suggests DKA with NOMI is common in children with T1DM, and they have a good prognosis after the treatment.[4-6] We present a case of recurrent bowel necrosis with NOMI from DKA in an adult with poorly controlled insulin-dependent T2DM causing mortality.

CASE REPORT

A 58-year-old male presented to the emergency department feeling unwell for two days and with severe bilateral flank pains. He was febrile at home with some nausea without vomiting. He denied urinary symptoms. He is known to have insulin-dependent (IDDM) type 2 diabetes mellitus (T2DM) with poor blood sugar control. He has had multiple previous episodes of diabetes ketoacidosis due to poor compliance with medications. He underwent right hemicolectomy with a transverse colostomy for acute mesenteric ischemia from a DKA episode one year ago. He lives independently by himself

with a poor socioeconomic background and is a heavy smoker with more than 50 pack years.

He was febrile and tachycardic on examination. He was drowsy with Glasgow coma scale (GCS) 14. His blood pressure was at a lower normal level. Abdominal examination was unremarkable except for bilateral flank tenderness. The colostomy bag was functioning, and the bowel sound was typical. His blood investigations showed glucose 60 mmol/l (3.0-7.8 mmol/l) and ketones 5.7 mmol/l (<1 mmol/l), pH 6.89, lactate 5.8 mmol/l (0.5-2.2 mmol/l), Potassium 5.6 mmol/l (3.5-5.2 mmol/l), Na 116 mmol/l (135-145 mmol/l), GFR 36 ml/min, white blood cells 14.8×109/l. Chest X-ray shows no consolidation.

The initial diagnosis was DKA triggered by possible pyelonephritis, as the patient had significant flank pain. Computed tomography of kidney, ureter and bladder (CT KUB) without contrast was organised as the patient had an acute kidney injury (AKI) from severe dehydration. CT KUB shows no signs of pyelonephritis or urinary tract obstruction. It shows thickening of the small bowel with gas within the bowel wall (pneumatosis intestinalis), portal venous system and liver consistent with ischemic bowel.

The patient was resuscitated with fluid management and insulin infusion. Intravenous antibiotics were started. He underwent an emergency midline laparotomy due to haemodynamic instability. Small bowel walked through and was found to have 55 cm of jejunum was ischemic. The rest of the small bowel was healthy and had normal peristalsis. A damage control procedure was performed where the ischemic small bowel segment was resected, and proximal and distal ends were brought as a stoma without anastomosing as the patient was on inotropes during the operation.

He underwent a relook laparotomy the following day. The remaining small and large bowel parts were healthy. Small bowel limbs were anastomosed side-to-side with a GIA stapler, and the staple line was oversewn with 3-0 polydioxanone (PDS). ICU care was given during the postoperative period and transferred to the ward on the eighth day of the postoperative stay.

He was investigated for coagulopathy and infective endocarditis to identify the causes of recurrent mesenteric ischemia. The thrombophilia screening was normal, and the echocardiogram showed good ejection fraction without vegetations or regional wall motion abnormalities. His inflammatory and tumour markers were normal.

He recovered uneventfully and was discharged home on the 19th day after the initial surgery. He presented to the ED a month after the previous discharge with a one-day history of severe right lower abdominal pain with vomiting. He was haemodynamically stable, and abdominal examination revealed signs of small bowel obstruction. His blood investigations were unremarkable. CT abdomen showed a closed loop bowel obstruction with two transient points in the right lower quadrant with evidence of bowel ischemia. He underwent emergency laparotomy and was found to have a near-frozen abdomen due to dense adhesions. Extensive adhesiolysis was performed to release the bowel obstruction. He recovered well and was discharged home on the seventh postoperative day.

After three months, he was brought again to the emergency department by ambulance with low GCS and hypotension. He was haemodynamically unstable with hypotension and tachycardia. The temperature was high. The abdomen was soft on examination, but the bowel sounds were sluggish. His blood glucose level was 66.6 mmol/l, and the ketones were 5.4 mmol/l. The pH was 6.78 and lactate was 6.4 mmol/l. CT abdomen showed extensive pneumatosis in the small and large bowel with portovenous gas in the liver. The surgical team and the ICU team with the family decided on palliation for him, given the futility of the surgical treatment due to extensive bowel ischemia and frozen abdomen.

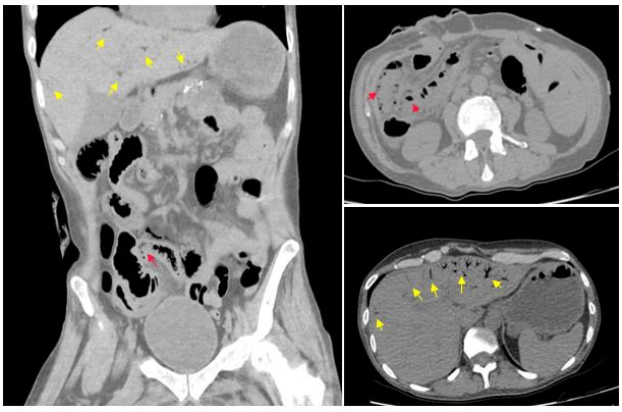

Figure 1: CT KUB shows pneumatosis intestinalis (red arrowheads) and gas within the portal system (yellow arrowheads) during the second admission with NOMI.

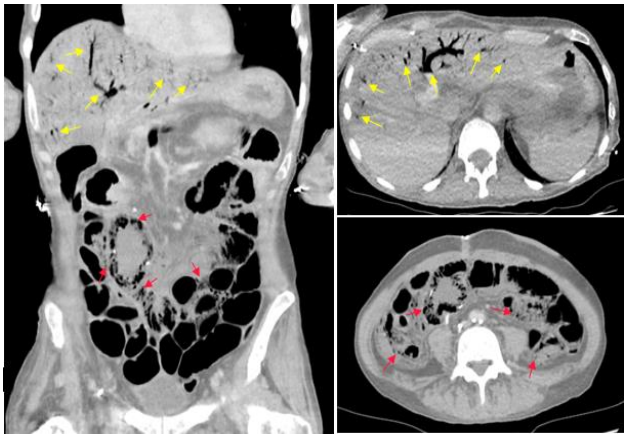

Figure 2: CT abdomen showing pneumatosis intestinalis (red arrowheads) and gas within the portal system (yellow arrowheads) during the third admission with NOMI.

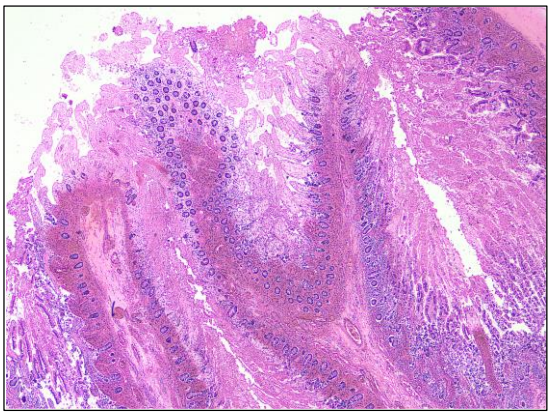

Figure 3: Ischaemic small intestinal mucosa with diffuse superficial mucosal necrosis, patchy preservation of crypts and associated submucosal vascular congestion (H and E at 20X).

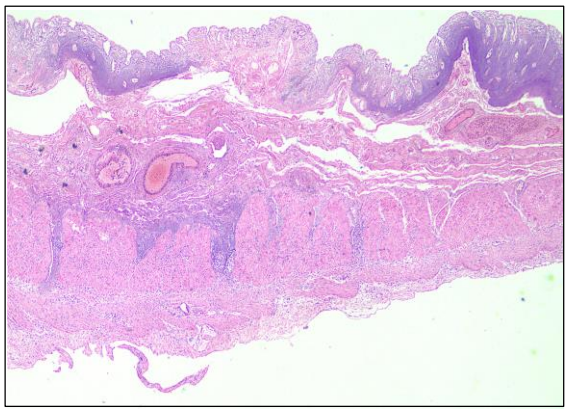

Figure 4: Ischaemic large intestinal mucosa with diffuse mucosal necrosis, patchy mucosal bacterial overgrowth and areas of mural ischaemia with attenuation of the bowel wall (H and E at 20X).

DISCUSSION

DKA occurs due to a relative or absolute fall in insulin levels in the blood, which is associated with a rise in counterregulatory hormones such as adrenaline, glucagon and cortisol.[7] Normal physiological mechanisms are deranged due to this hormonal imbalance causing hyperglycaemia, hyperosmolality and ketoacidosis.[6]

The diagnosis of DKA is made when the biochemical criteria of hyperglycemia (glucose level more than 11.1 mmol/l, acidosis pH less than 7.3 and ketonemia in the blood.[8,9] Polyuria, polydipsia, polyphagia, and loss of weight are the classical signs of DKA. Patients might present to the emergency department with rapidly progressing symptoms of abdominal pain, vomiting, dehydration, and lethargy. The reasons for the abdominal pain are acidosis, potassium depletion and inadequate perfusion of splanchnic vessels. Initial metabolic stabilisation usually improves clinical symptoms.[10] Initial management of DKA includes immediate fluid resuscitation to improve the intravascular volume, rectify the electrolyte derangement and start insulin treatment to prevent further lipolysis and ketogenesis, which contributes to stabilising the acid-base imbalance.[4,11] It is crucial to consider acute mesenteric ischemia (AMI) if the severe acidosis persists after the initial treatment, as AMI is associated with high morbidity and mortality.[4,6]

AMI occurs due to an embolic event (40-50%) or thrombosis of a previously stenosed vessel by atherosclerosis (20-35%) or dissection of a mesenteric vessel (5%) or NOMI which is responsible for 5-15% of the cases.[12]

In our case, the patient had bowel necrosis in the context of DKA. Superior mesenteric artery occlusion or NOMI should be the possible reason for small and large bowel necrosis. Intraoperative findings and histology indicated that NOMI was the cause of mesenteric ischemia. The intestines showed patchy and segmental necrosis; mesenteric arteries were patent without having evidence of occlusion. Histology confirmed ischemic enteritis of the intestines.

NOMI can occur in DKA due to various reasons. Severe dehydration from hyperglycaemia causes collapse of intravascular volume, decreased cardiac output and rise of blood viscosity, causing hypoperfusion of splanchnic vessels.[3] DKA can stimulate vasopressin excretion and activation of the renin-angiotensin system, which triggers the mesenteric artery spasms contributing to hypoperfusion of the intestines.[13,14]

CT abdomen angiogram is the first line of imaging to diagnose AMI as it is quick, non-invasive, and accurate.[15] CT imaging features of AMI are filling defects in mesenteric vessels and thickening or hypoattenuation of the bowel wall, bowel distension, mesenteric fat stranding, bowel wall oedema and pneumatosis with portal venous gas.[16,17]

In our case, on the second presentation, the patent underwent CT KUB to rule out pyelonephritis as the clinic features favoured urinary tract infection causing DKA. CT KUB did not show features of urinary tract infection; instead, it showed gas in the mesenteric veins with branching gas throughout the periphery of the liver and multiple thickened small bowel loops adjacent to the mesenteric gas.

On the third presentation, the CT abdomen showed extensive pneumatosis of the small and large bowel and a large volume of gas throughout the portal vein and the liver. Treatment of AMI includes revascularisation of the ischemic bowel, assessing the viability of the segments and resecting the necrotic bowel segments.[12] It has been reported that the mortality rate with delayed interventions can be up to 90%.[18,19] In our case, the patient had multiple episodes of DKA due to poor compliance with diabetes treatment due to poor socioeconomic background. He

survived following two bowel resection surgeries, but the treatment was futile on the last presentation due to extensive bowel necrosis and frozen abdomen due to dense adhesions from the previous surgeries in the abdomen.

CONCLUSION

Complications of diabetes keto acidosis (DKA) are rare in adults with type 2 diabetes mellitus (T2DM) compared to children with type 1 diabetes. In this case report, we present a rare case of mortality associated with DKA of an adult patient with poorly controlled T2DM following recurrent non-obstructive mesenteric ischemia (NOMI) due to severe dehydration. Multiple bowel resections following recurrent episodes of severe DKA may lead to poor outcomes due to limited reserves.

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