East African Medical Journal Vol. 96 No. 7 July 2019
A UNIQUE CASE OF ACUTE MULTIPLE ARTERIAL THROMBOSIS COMPLICATING DIABETIC KETOACIDOSIS
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A UNIQUE CASE OF ACUTE MULTIPLE ARTERIAL THROMBOSIS COMPLICATING DIABETIC KETOACIDOSIS

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ABSTRACT

Acute aortic occlusion is a very rare but devastating occurrence in diabetic ketoacidosis (DKA). We report a unique presentation of DKA with aortic thrombi and distal arterial emboli. A 53-year-old woman, type 2 diabetes presented due to 7 days of abdominal complaint and DKA. She developed gangrene of her right leg. Contrast-enhanced CT revealed extensive thrombus formation in the aorta, and filling defects in other major arteries. The arterial occlusions most likely resulted from a hypercoagulable state associated with her DKA. Clinicians should maintain a high index of suspicion for arterial thrombosis in patients presenting with DKA.

INTRODUCTION

Vascular thrombosis is a little-known complication of diabetic ketoacidosis (DKA). However, it is important to recognize as it may accounts for as many as one-third of all DKA deaths (1,2). In those with arterial thrombosis and DKA, almost all muscular arteries may be involved, including carotid, coronary, mesenteric, iliac, renal, splenic, and pancreaticoduodenal arteries (3). But acute aortic occlusion is a very rare and yet devastating occurrence in DKA, associated with increased morbidity and mortality despite surgical intervention. We report a unique presentation of DKA with both thoracic and abdominal aortic thrombi and distal arterial emboli.

CASE REPORT

A 53-year-old woman with a known 12-year history of type 2 diabetes (off oral hypoglycemic agents for the past 12 month) was transferred to Tikur Anbessa Specialized Hospital (TASH) for further evaluation of 7 days of crampy abdominal pain, vomiting, and watery non-bloody diarrhea 2-3x/day. At an outside hospital, she had been managed for 4 days with isotonic fluids, regular insulin, and antibiotics for presumed DKA and acute gastroenteritis.

On admission, her physical examination was remarkable for: PR 125-145, RR 30-34, and abdominal distension. Her initial laboratory examination showed a blood sugar of 421 mg/dL (normal value 72-99 mg/dL); her urine was positive for ketone bodies. Additional labs included: white blood count 26,000/mm^3 with 90.7% neutrophils, serum creatinine 1 mg/dl, sodium 147.8mEq/L, potassium 3.3 mEq/L, and normal lipase and cardiolipin and beta 2 glycoprotein 1 antibodies. Initial non-contrast chest and abdominal computed tomography (CT) (figure 1) showed distal bowel obstruction, bilateral pleural effusion, bilateral lower lung atelectasis and consolidation, and minimal ascites. Electrocardiogram (ECG) and echocardiography were normal.

We initially considered DKA complicated by gastrointestinal induced sepsis, and she was admitted to the medical intensive care unit for the management of these conditions. She received ceftriaxone and metronidazole in addition to standard management of DKA with continuous low-dose regular insulin and isotonic fluids. The diagnosis of vascular thrombosis was considered on the 2nd day of admission to TASH (9th day of illness) due to persistent abdominal pain, abdominal distension, non-bloody diarrhea, and new onset of cyanosis of her lower extremities. Chest and abdominal contrast-enhanced CT (figure 2) revealed extensive thrombus formation in the descending thoracic and proximal abdominal aorta, non-obstructive filling defects in multiple mesenteric arteries, and right renal cortical necrosis. Anticoagulation with unfractionated heparin was initiated but thrombectomy was differed due to the poor clinical condition of the patient.

Her right leg rapidly developed gangrene requiring subsequent below the knee amputation. Eighteen days post amputation, her 26th day of admission, the patient had a sudden cardiac arrest; she was successfully revived with cardiopulmonary resuscitation (CPR). She was subsequently intubated and put on ventilator machine. Her level of consciousness dropped. Her blood pressure remained low, 70/40 mmHg, despite receiving 2 liters of normal saline and a noradrenaline continuous infusion. Her post-intubation chest x ray showed a large right pleural effusion; repeat thoracentesis revealed an empyema, for which chest tube drainage was performed. The antibiotics were switched to Meropenium and Vancomycin. She died on the 28th hospital day from uncontrolled septic shock despite aggressive management.

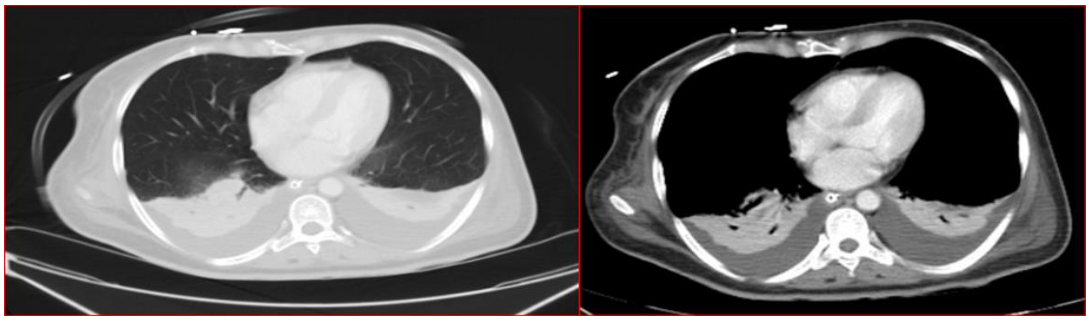

Figure1. Non-contrast CT: Bilateral pleural effusion, bilateral lower lung atelectasis and consolidation

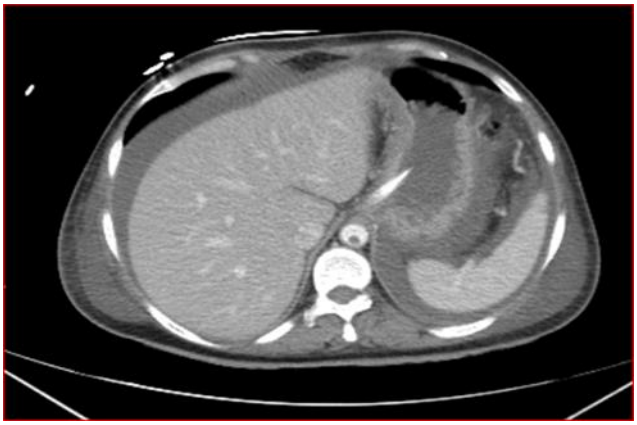

Figure 2. Extensive thrombus formation in the descending thoracic and proximal abdominal aorta, non-obstructive filling defects in multiple mesenteric arteries, and right renal cortical necrosis.

DISCUSSION

We report a unique case of a 53-year-old woman who presented with DKA, complicated by thoracic and abdominal aortic thrombi, and distal emboli involving mesenteric, right renal, and right femoral arteries. The arterial occlusions most likely resulted from a hypercoagulable state and activation of the vascular endothelium associated with her DKA. The metabolic insult of DKA may initiate or perturb the steady state of vascular endothelial cells, changing the hemostatic profile and resulting in a prothrombotic state (4).The prothrombotic state in DKA could be explained by increased platelet aggregation, coagulation activation, and diminution of the anticoagulation system (4-6).

Coagulation abnormalities that increase the risk of vascular thrombosis in DKA include decreased free protein S and protein C activity levels and increased von Willebrand factor (4).

In the literature, several clinical occurrences of hypercoagulability and the development of vascular thrombi in the setting of DKA have been described. Arterial thrombosis accounted for 33% of the deaths among the 9% mortality rate due to DKA in a study of 1,769 episodes of DKA (3). In another study of 528 patients with DKA and 82 patients with hyperosmolar coma, 6 of 38 deaths were due to mesenteric and iliac thrombosis, and 8 were due to myocardial infarction (7).

Arterial rather than venous thrombosis is more strongly associated with DKA (8), and may include many muscular arteries such as the carotid, coronary, mesenteric, iliac, renal, splenic, and pancreaticoduodenal arteries. However, abdominal aortic thrombosis, as in our case, is very rare. To the best of our knowledge, only two other similar cases have been reported (9, 10).

Establishing DKA as the etiology of the thrombosis involves excluding other causes of thrombosis such as a cardiac source and other prothrombotic states, including anti-phospholipid syndrome; both conditions were excluded in this case by demonstration of normal ECG and echocardiography and a normal antibody test.

In addition, abdominal pain is a common presenting symptom in DKA patients, occurring in as many as 45% of patients; however, this pain is expected to improve with the correction of acidosis, unlike the persistent pain of vascular thrombosis, as demonstrated in our patient.

The issue of prophylactic anticoagulation in DKA remains controversial. The mortality rate of arterial thrombosis in DKA is very high. However, accurate diagnosis of thrombosis is difficult. When thrombosis is diagnosed, the necessary rescue treatment

consists of invasive methods such as surgical thrombectomy and anticoagulation in addition to correcting the DKA.

CONCLUSION

We believe that DKA in our patient caused a prothrombotic, hypercoagulable state that led to an acute arterial thrombotic event and a devastating outcome. Thus, clinicians should maintain a high index of suspicion for arterial thrombosis in patients presenting with DKA.

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