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Case Report .

Symptomatic Thrombosis of Superior Mesenteric Vein Due to Diabetic Ketoacidosis

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ABSTRACT

A 24-year-old man, with history of type I diabetes mellitus, presented with a 3-day persistent abdominal pain despite control of his DKA contrary to his 1-year similar attacks. CT-scan with contrast revealed partial thrombosis of superior mesenteric vein (SMV). Following systemic anticoagulation, the abdominal pain disappeared. There was no evidence of hereditary thrombophilia or underlying autoimmune disease. Hence, patients with DKA and abdominal pains should be screened for SMV thrombosis.

Key words: CT Scan, Diabetes Mellitus, Diabetic Ketoacidosis, Venous Thrombosis.

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INTRODUCTION

Diabetic ketoacidosis (DKA) is an acute, major, life-threatening complication of type I diabetes mellitus (DM) due to insulin deficiency. The most common early symptoms of DKA are the insidious increase in polydipsia, polyuria and volume depletion. Gastrointestinal manifestations includes; nausea, vomiting and unexplained abdominal pain. The latter illusive manifestation remained mostly unexplained and rarely was attributed to a precipitating factor rather than metabolic acidosis.[1] We herein describe a successful outcome of a superior mesenteric vein thrombosis (SMVT) in a patient with recurrent DKA and abdominal pains in an attempt to improve the outcome of such diabetic crisis.

CASE PRESENTATION

A 24 year-old man seeked medical advice for management of recurrent severe abdominal pains for the past year which was associated with his admission with DKA. Previous attacks lasted only few hours but the present one persisted for 3 days. The pain was associated with vomiting, abdominal distension and constipation. It was also associated with fever. Vomiting did not relive the pains or antispasmodics. All his abdominal pains were associated with diabetic ketoacidosis and were relived after control of his hyperglycemic event. He admitted that he was not very compliant with his pre-meals novorapid insulin though was taking his 20 units of lantos every night. He also admitted that he was not compliant with his prescribed diabetic diet and used to eat sweats, fruits and juices. The patient denied shortness of breath, oedema, chest pain, skin rash and joint pains. Past history was significant for type I DM for the past 28 years and was treated with Lantos 24 units PM with novorapid 18+16+18 prior to respective meals. He did not have past history of significant medical illness, surgery, allergy or chronic intake of medications especially NSAIDs. On his initial physical examination, the patient was conscious, oriented X3 and was in severe distress of abdominal pain. Blood pressure was 120/80 mm Hg. Temp. was 39^{0}C. His body weight was 50 kg. He did not have lymphadenopathy, goiter, jugular venous distension or oedema. Systemic examination did not show abnormality. Laboratory investigations showed normal peripheral leucocytic and platelets counts. Hemoglobin was normal with normal MCV. Serum sugar was 17 mmol/L. Serum urea, creatinine, electrolytes and liver functions were normal except for HCO3 was 15 with blood pH at 7.2. Serum cholesterol and TSH were normal. Urine routine and microscopy was normal except for 4 (+) glycosuria and ketonuria. Stool testing for ova, parasites and occult blood was negative. Chest x-ray and ECG were normal. Abdominal and pelvic ultrasound was normal. Upper GI endoscopy showed only mild gastritis with negative Helicobacter infestation. His blood sugar was controlled with continuous insulin infusion. Moreover, he was hydrated and had received multivitamins that included vitamin B1. The initial deficiencies in magnesium, potassium and phosphorus were corrected. Since febrile, he was covered with broad-spectrum antibiotic (meropenum) till receiving the results of his blood cultures. The latter was available 2 days later and had shown a growth of pseudomonas aeuregenosa and streptococcus constelatus. He became afebrile 2 days after admission. Since both organisms were sensitive to the antibiotic; treatment was

continued for 10 days. CT scan, with contrast, of the abdomen was done in search for a septic focus. Interestingly, the latter showed partial SMVT (Fig 1). Subsequently, he was fully anticoagulated with heparin then warfarin. Thrombophila work up, drawn before anticoagulation, showed normal levels of protein S, protein C, antithrombin III, anticardiolipin antibodies, lupus anticoagulant and anti-factor V leyden. Moreover, serum complements (C3 & C4), IgA and protein electrophoresis were normal. ANA, anti-ds DNA, ANCA, RA, hepatitis B surface antigen and anti-HCV antibodies were negative. One day after heparin infusion, his abdominal pains disappeared. Repeat CT scan of the abdomen, 4 months later, showed resolution of the thrombosis (Fig 2). He was kept on warfarin for a total of 6 months. He was instructed to adhere to diet and tight insulin therapy.

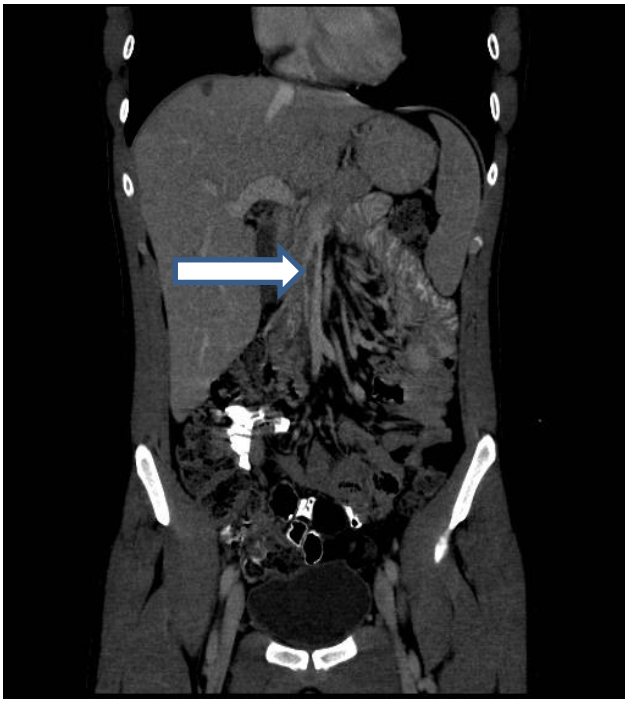

Fig 1: Coronal projection of a CT scan of the abdomen, with contrast, showing a filling defect in the superior mesetric vein indicating partial thrombosis of the vein.

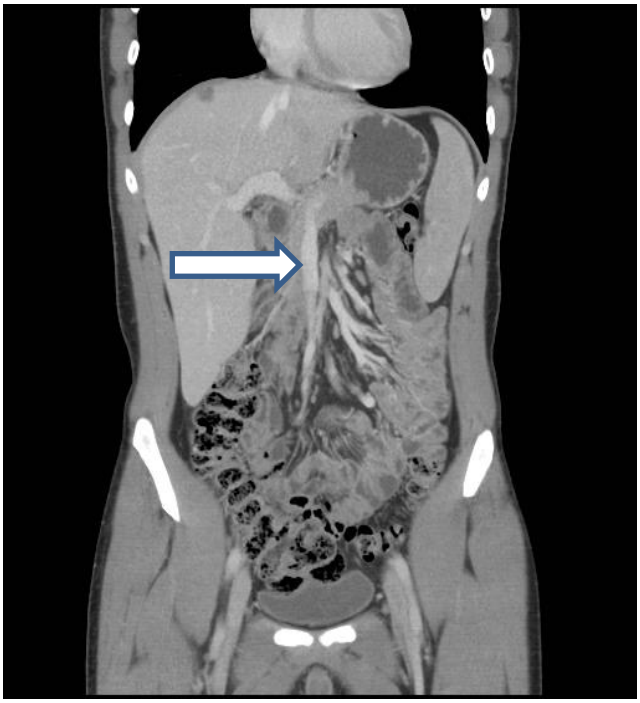

Fig 2: Coronal projection of a CT scan of the abdomen, with contrast, showing normal opacification of the superior mesenteric vein, 4-months after anticoagulation indicating resolution of SMVT.

DISCUSSION

Compared to arterial disease due to small bowel obstruction, acute SMVT is uncommon and accounts for 5-15% of all cases of acute mesenteric ischemia. The pathogenesis is due to venous back pressure leading to bowel wall thickening and hence inadequate tissue perfusion leading to intestinal ischemia, infarction and eventual necrosis. Often despite SMVT, small bowel necrosis does not occur, presumably due to persistent arterial supply and some venous drainage via collaterals. However, without anticoagulant therapy, the mortality approaches 30%, with a 25% recurrence rate.[2] Hence, prompt recognition is essential since early diagnosis and aggressive treatment may prevent bowel necrosis. Imaging is the only reliable way of making the diagnosis, especially as clinical presentation is vague. CT of the abdomen, with contrast, is the most accurate test available to us at present, with excellent sensitivity (up to 100%).[3] The majority of cases are considered secondary to an identifiable underlying condition such as hypercoagulable state, malignancy, pancreatitis, polycythemia, recent abdominal surgery and intra-abdominal sepses.[3] Our young patient with recurrent DKA had developed such phenomena without such underlying conditions. The only plausible explanation is that DKA is a prothrombotic state. Previous experimental and clinical studies as well as a post-mortem case report have indicated that type II DM is a risk factor for SMVT.[4-6] Moreover, adrenergia, dehydration and hemoconcentaration are common in DKA.[7] The localization of ischemia in the superior mesenteric vein is likely due to the preferential gut arterial hypoperfusion due to severe volume depletion.[8] Our case report represents the first, antemortem, report on progressive development of SMVT in a patient with type I DM with recurrent DKA and provides a possible explanation for the unexplained abdominal pains associated with it.

CONCLUSION

Our case report emphasizes the need for screening patients with DKA and abdominal pains with CT scan, with contrast, to assess for SMVT and its potential risk of gut necrosis.

REFERENCES

1. Umpierrez G, Freire AX. Abdominal pain in patients with hyperglycemic crises. J Crit Care 2002; 17: 63-67.
2. Gul, W., Markert, R.J., Abbass, K. Acute superior mesenteric vein thrombosis: A lethal disease. Practical Gastroenterol 2011; 35, 50-52.
3. Furukawa A, Kanasaki S, Kono N et-al. CT diagnosis of acute mesenteric ischemia from various causes. AJR 2009;192: 408-16.
4. Iba T, Aihara K, Yamada A, Nagayama M, Tabe Y, Ohsaka A. Rivaroxaban attenuates leukocyte adhesion in the microvasculature and thrombus formation in an experimental mouse model of type 2 diabetes mellitus. Thromb Res 2014; 133: 276-280.

5. Elkrief L, Corcos O, Bruno O, Larroque B, Rautou P-E, Zekrini K, Bretagnol F, Joly F, Francoz C, Bondjemah V, Cazals-Hatem D, Boudaoud L, De Raucourt E, Panis Y, Goria O, Hillaire S, Valla D, Plessier A. Type 2 diabetes mellitus as a risk factor for intestinal resection in patients with superior mesenteric vein thrombosis. Liver Int 2014; 34: 1314-1321.
6. Nicol KK, Davis GJ. An unusual complication of diabetes mellitus: The zebra that became a horse. Southern Med J 1997; 90: 83-85.
7. Yun AJ, Lee PY, Bazar KA. Can thromboembolism be the result, rather than the inciting cause, of acute vascular events such as stroke, pulmonary embolism, mesenteric ischemia, and venous thrombosis?: A maladaptation of the prehistoric trauma response. Med Hypotheses 2005; 64: 706-716.
8. Takala J. Splanchnic perfusion in shock. Intens Care Med 1994;20:403-404.

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