Acute Mesenteric Venous Thrombosis with a History of Recurrent DVT in Young Age: a Case Report

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Introduction

Acute mesenteric ischemia (AMI) may be defined as a sudden interruption of the blood supply to a segment of the small intestine, leading to ischemia, cellular damage, intestinal necrosis, and eventually patient death if left untreated.¹ Mesenteric venous thrombosis (MVT) is an emergency condition that is rarely found as a cause of AMI compared to arterial thrombosis, and accounts for only 16% of all mesenteric ischemia cases.² The overall incidence is low (0.09-0.2% of all acute admissions to emergency department), prompt diagnostic and intervention are essential in order to reduce the high mortality rate (50-80%).¹,10

Case Presentation

A 26 year-old man presented to the ER with severe abdominal pain with a visual analogue scale (VAS) of 8-9, which started 4 days as an on-and-off localized abdominal discomfort and got worsened in the past 12 hours prior to hospital admission. He had a recurrent history of deep vein thrombosis (DVT) in the past one year. His vital signs were unremarkable, a tender sensation by palpation at the left lumbar region, distended abdomen and guarding to palpation were absent. Abdominal plain x-ray, electrocardiogram, echocardiogram and laboratory tests results were within normal range except for D-dimer that was 13.298 µg/ml. Ultrasonography showed ascites and loop bowel dilatation. Abdominal computed tomography (CT) angiography revealed a free fluid in all region of the abdomen with edematous thickening; also a partially dilated small bowel wall of the jejunal segment without contrast filling was seen in the superior mesenteric veins, splenic veins, and portal veins with lumen dilatation.

The patient started to receive subcutaneous enoxaparin twice daily, 1,470 mg Disolf (lumbrokinase DLBS1033) three times daily, morphine 1 mg/hour, 1 g cefoperazone/subbactam twice daily, and bowel rest. The patient then developed peritonitis at the 4th day of admission and had undergone an urgent laparotomy, during which a 30 cm segment of ischemic small bowel was resected. The patient showed a good recovery in ICU.

Discussion

Like all thrombotic events, the formation of MVT is a result of Virchow’s Triad (endothelial injury, stasis of flow and hypercoagulability).³ Although we did not found any history that supported endothelial injury (e.g. recent intraabdominal surgery, colitis ulcerative, or trauma)
and stasis of flow (e.g. cirrhosis, congestive heart failure/CHF) in this case, hypercoagulability state, however, was the most likely cause of MVT in this patient considering his history of recurrent DVT.4

Acute MVT presents almost universally with abdominal pain, often out of proportion with physical exam in earlier phase.5 Our finding that laboratory test results were within normal value except D-dimer suggests that plasma D-dimer detection might be a useful means of identifying patients with acute intestinal ischemia, as also indicated in a former metaanalysis.6

Abdominal computed tomography angiography (CTA) revealed a bowel ischemia and suggested MVT. Sensitivity and specificity of CT angiography are 93% and 100%, respectively, with positive and negative predictive values between 94% and 100%.7 Acute intestinal ischemia due to MVT was diagnosed and management options were explored. Although the CT images were suggestive of intestinal ischemia, the patient had no signs of peritonitis at early time of admission. Thus, conservative management was chosen to manage this patient.8,9 Low molecular weight heparin (enoxaparin) was commenced on the third day of admission, followed by an oral thrombolytic, Disolf (lumbrokinase DLBS1033) and the patient was placed on bowel rest. Studies have shown that lumbrokinase inhibits platelet activation and aggregation and blocks the intrinsic coagulation pathway and might be useful as an adjunctive maintenance therapy that appears very promising.10 However, the patient developed fever and peritoneal signs emerged in the fourth day of admission, which was suggestive for peritonitis and the patient was proceeded to an urgent laparotomy. After surgery, enoxaparin was continued for 10 days, while high dose Disolf were prescribed for three months then continued for life.

Conclusion

A high suspicion has to be made when finding abrupt onset of abdominal pain without proportion to physical exam plus a history of recurrent DVT or other hypercoagulability state. A prompt diagnosis by CT angiography has to be done and initial management such as fluid resuscitation, initiation of anticoagulant, adjunct oral thrombolytic, bowel rest, antibiotic if needed. Continuous monitoring of a peritonitis sign become the mainstay management of acute mesenteric ischemia.

DAFTAR PUSTAKA