

Idiopathic Acute Mesenteric Ischemia and Tuberculous Cervical Lymphadenitis in a 28-year-old Man

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Citation

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Abstract

We present a case of a 28-year-old man with tuberculous cervical lymphadenopathy who developed acute mesenteric ischemia with peritoneal signs in hospital. An emergency exploratory laparotomy with resection of 142cm of gangrenous small bowel and end-to-end anastomosis was performed. Subsequently, a thrombophilia screening was done which proved to be normal. By exclusion, the most likely cause of acute mesenteric ischemia in this patient was of unknown etiology which is indeed a rare condition. Presence of a dual pathology namely acute mesenteric ischemia with tuberculous cervical lymphadenopathy is equally rare. A high index of suspicion for diagnosis and prompt operative intervention when indicated improve prognosis in this otherwise grave condition.

CASE REPORT

A 28-year-old male presented with a 2-month history of bilateral neck swellings accompanied by a 3-day history of fever, diarrhea and vomiting. He was admitted initially for a lymph node biopsy in order to rule out lymphoma or tuberculosis.

In the hospital, he developed a severe diffuse abdominal pain of sudden onset. It was associated with distension and vomiting. The patient denied any past history of abdominal angina and had no history of palpitations, dyspnea, fever, melena or hematochezia. The patient was non-smoker, non-alcoholic and had no history of substance abuse.

On examination, the patient was toxic and in shock. General examination revealed multiple, non-tender, matted and firm lymph nodes in the neck. Abdominal examination showed features of frank peritonitis.

Radiological work-up included supine and erect plain X-rays, which revealed multiple central abdominal air-fluid levels (Fig 1).

Figure 1

Figure 1: X-ray with multiple air-fluid levels



Abdominal ultrasound showed mild splenomegaly and mild free pelvic fluid.

After initial resuscitation with intravenous fluids, an emergency exploratory laparotomy was performed under general anesthesia through a midline vertical abdominal incision. It revealed frank gangrene of the distal jejunum and proximal ileum with impending perforation (Fig 2) and also

mildly enlarged mesenteric lymph nodes.

Figure 2

Figure 2: Gangrenous bowel with impending perforation



The rest of the abdominal viscera and major abdominal vessels were grossly normal. There was no evidence of tuberculosis in the abdomen. Resection of the gangrenous bowel (distal jejunum and proximal ileum) with end-to-end anastomosis using 2/0 vicryl in a single layer, followed by approximation of the mesentery was done.

The patency of the intestinal lumen and integrity of the anastomosis were checked before abdominal closure in single layer. The resected segment measuring 142cm was sent for histopathological examination.

A right posterior cervical lymph node biopsy was also done in the same sitting. A cut section of the node revealed caseation. The histopathological examination of the intestine reported features compatible with gangrene of the small intestine due to ischemia. That of the lymph node biopsy was reported as features compatible with tuberculosis.

Antibiotic prophylaxis and thromboprophylaxis were given to the patient. The post-operative course was stormy with development of chest infection, paralytic ileus due to electrolyte imbalance, wound hemorrhage and infection.

Post-operative work-up to get to the cause of gangrene was done. A thrombophilia screen (Protein C, Protein S and anti-thrombin III done after stopping anti-coagulant therapy for 2 weeks prior to screen) was normal. Echocardiography and an abdominal Doppler ultrasound showed the heart chambers and valves to be normal and the caliber of the descending abdominal aorta and superior mesenteric artery also to be normal, thus ruling out embolism as cause of gangrene of

bowel.

The patient was discharged from the hospital on anti-tuberculous treatment with excellent response at seven months follow-up. The patient had no abdominal complaints as well.

DISCUSSION

The four major causes of acute mesenteric ischemia and their incidence are: acute mesenteric arterial embolus (AMAE – 50%), acute mesenteric arterial thrombosis (AMAT – 25%), mesenteric venous thrombosis (MVT – 10%) and non-occlusive mesenteric ischemia (NOMI – 20%).¹

AMAE is usually caused by an embolus of cardiac origin. AMAT frequently presents with a history of chronic mesenteric ischemia in the form of abdominal angina before the emergent event and usually has atherosclerotic disease at other sites. NOMI occurs more frequently in older patients who are already systemically ill, in the intensive care unit setting and on vasopressive drugs or digitalis. The symptoms in MVT may be present longer than in the typical case of acute mesenteric ischemia, sometimes exceeding 30 days.¹ Mesenteric ischemia in patients under the age of 40 years in the absence of cocaine use is rare and often causes a delay in diagnosis and appropriate treatment.²

According to the literature, a study of 4 cases of small intestinal ischemia secondary to superior mesenteric vein thrombosis showed three being due to hypercoagulable states (Protein-S deficiency, factor VII abnormalities) and one was idiopathic.³ It has been estimated that 20% of cases of MVT are idiopathic and 80% are secondary to some underlying condition like pancreatitis, trauma, haematological disease, inflammatory bowel disease, diverticular disease, hypercoagulability, cirrhosis, cancer or rheumatoid arthritis. There was no evidence of any of the above conditions in our patient.⁴

Differentiation of acute mesenteric ischemia on the basis of aetiology is of great importance because of variation in disease progression, response to treatment and outcome. Non-atherosclerotic and non-aneurysmal aorto-arterial thrombosis as a cause of acute abdomen or lower limb gangrene has been studied in autopsy cases and according to this study, the symptoms were attributable to hypercoagulable states and changes in the aortic wall. No aetiology was identifiable in 16.6% of cases and interestingly, there was associated tuberculosis in about 20%

of cases. Tuberculosis can rarely induce a hypercoagulable state by increased levels of type 1 plasminogen activator inhibitor and tissue factor.⁵

Based on the history, physical and operative findings and laboratory work-up, the most likely cause for acute mesenteric vascular insufficiency with bowel infarction and gangrene in our patient is idiopathic. The protocol for long term anticoagulation therapy is clear. Lifelong anticoagulation is recommended for patients with anti-thrombin III deficiency and factor V Leiden homozygous state. For patients with Protein C or Protein S deficiency or Factor V Leiden heterozygous state, there is low likelihood of recurrent thrombosis. These patients do not need life-long anticoagulation therapy.⁶

It needs to be re-emphasised that acute mesenteric ischemia needs a high index of suspicion for diagnosis, especially in a patient with no evidence of risk factors to develop this condition, as with longer delay in diagnosis, the prognosis becomes increasingly grave and morbidity and mortality proportionately increases. In the event of bowel infarction due to acute mesenteric ischemia of any cause, bowel resection is inevitable, but this has to be followed up with thorough biochemical and pathological investigations to determine or rule out the cause of the bowel infarction and also to decide on the necessity of long term anticoagulation therapy.

CONCLUSION

A relatively young patient presented to us with a relatively uncommon vascular emergency with grave prognosis,

namely bowel gangrene due to acute mesenteric ischemia of unknown aetiology associated with an unrelated co-existing pathology, namely tuberculous cervical lymphadenitis. The key to successful treatment of this patient included a high index of suspicion, prompt and aggressive surgical management and meticulous post-operative care.

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