

Case Report

A Case of Acute-on-Chronic Mesenteric Vascular Occlusion.

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Chettinad Health City Medical Journal 2017; 6(3): 145 - 147

Abstract

Mesenteric ischemia is an acute abdominal vascular emergency that has various causes, which carries a high mortality rate ranging from 60% - 80%, and in which prognosis depends on the pathologic findings.

Prompt diagnosis of this cause of acute abdomen requires a high index of suspicion. Rapid restoration of blood flow is the goal in these patients.

We hereby report a 61-year hypertensive, dyslipidemic female on treatment also having coronary artery disease and past history of rheumatic mitral stenosis having undergone mitral valvotomy twice. Once as closed mitral valvotomy and second as balloon mitral valvotomy in 2015. She was admitted for dyspeptic symptoms and fever. She developed acute abdominal pain while on evaluation for pain. On evaluation, patient was found to have complete occlusion of the superior mesenteric artery and was diagnosed as a case of acute-on- chronic mesenteric vascular occlusion due to cardiac emboli.

Key Words: Mesenteric Ischemia, Superior Mesenteric Artery Occlusion, Rheumatic Mitral Stenosis.

Introduction

Among the less common causes of abdominal pain is mesenteric ischemia, which accounts for less than 1 of every 1000 hospital admissions¹. A high degree of suspicion is required for the clinician to diagnose a case of intestinal ischemia. The difficulty in diagnosing the condition early usually leads to delay in treatment which results in extensive intestinal infarction².

We hereby report a case of a 61-year female who initially presented with non-specific symptoms of fever with chills and rigors.

Case report

A 61-year female presented with chief complaints of fever with chills and rigors and low back ache of 3 days duration. She also gave a history of nausea, bloating sensation, loss of appetite and inability to pass stools for 2 days prior admission. On second day of hospitalization she developed acute onset abdominal pain in right and left upper quadrants which she described characteristically as band like and it was episodic. The pertinent facts in medical history were that she had Rheumatic heart disease and had undergone closed mitral valvotomy at 28 years of age; later coronary angiogram revealed single vessel disease in September 2015 and she underwent a balloon mitral valvotomy in October 2015. She was a hypertensive for the past 5 years and had dyslipidemia, on regular treatment with penicillin G, Aspirin, rosuvastatin and amlodipine.

On clinical examination patient was febrile (99.5 F), pulse rate was 94/min regular, BP was 130/90 mmHg and hydration was poor. There was no pallor, icterus, cyanosis, clubbing, lymphadenopathy or pedal edema. On examination of the cardiovascular system, a loud pulmonary component of second heart sound was heard. Abdominal examination revealed tenderness in right hypochondrium. There was no guarding or rigidity, and no hepatosplenomegaly. Bowel sounds were normal and rectal examination did not show any abnormality.

Patient was evaluated for fever. Initial laboratory investigations revealed leukocytosis (WBC- 19800/cumm) and normal ESR. Platelet count was 2 lacs/cumm. Renal function, liver function, serum amylase and lipase, and electrolytes were within normal limits. Dengue serology was negative. Blood and urine culture showed no growth. Her chest roentgenogram revealed cardiomegaly and bilateral increased bronchovascular markings and presence of incomplete right bundle branch block (RBBB) on ECG. To rule out infective endocarditis patient underwent ECHO, and it demonstrated severe tricuspid regurgitation with severe PAP (EPAP- 78), mild residual mitral stenosis, dilated LA, RA, and normal LV systolic function with no regional wall motion abnormalities.

Abdominal ultrasound was performed for persistent abdominal pain which showed suspicious right sided ureteric calculus. Since X-ray KUB failed to visualize

any ureteric calculus, a plain CT KUB was done which had features suggestive of a superior mesenteric artery occlusion. CT angiogram was done to confirm the superior mesenteric artery (SMA) occlusion that showed complete occlusion in SMA distal to the origin of middle colic artery (Fig 1 and Fig 2) with reconstitution of flow, in distal SMA, through collateral vessels (Fig 3). The presence of collaterals was suggestive of chronic obstruction in SMA.

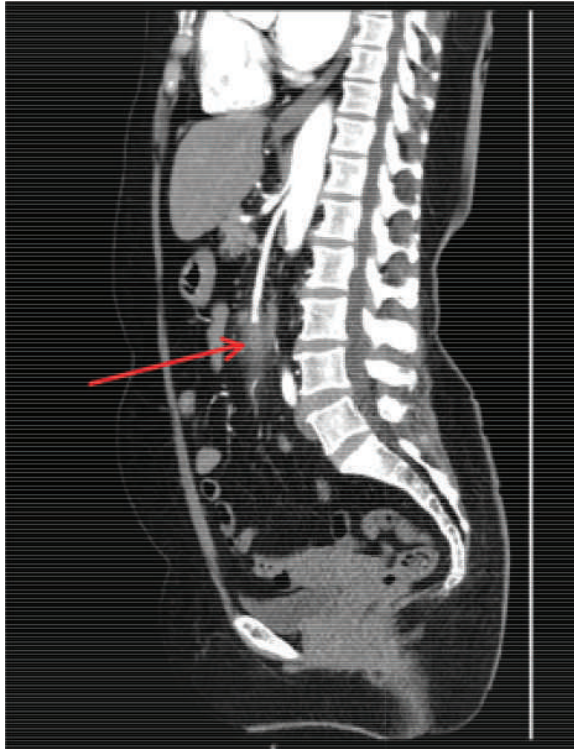


Fig 1 : Arrow pointing toward the thrombus in the SMA resulting in non-visualization of contrast media.



Fig 2 : 3-Dimensional reconstructed image revealing absence of flow in SMA beyond the arrow

5% of cardiac emboli can lodge into mesenteric artery and most often they lodge distally³, as in our case. In view of residual mitral stenosis, we concluded the possibility of a transient atrial arrhythmia that could have produced an embolus lodging in superior mesenteric artery. There was apparently no evidence of atherosclerotic plaques.

Patient was started on Inj. heparin 5000 IU/ fourth hourly. The patient improved clinically and her abdominal pain subsided and she was referred to a vascular surgeon for further management.



Fig 3 : Red arrow shows patent SMA with contrast filling. Blue arrows show the portion of SMA completely occluded by thrombus, with resultant total absence of contrast filling. Green arrow shows filling of the SMA distal to the obstruction. Yellow arrows point towards the collaterals from SMA, proximal to obstruction, which result in filling of contrast in SMA distal to obstruction - as shown by green arrow.

Discussion

There are 4 causes of acute mesenteric ischemia: arterial embolism (50%), arterial thrombosis (15–25%), non-occlusive mesenteric ischemia (20–30%), and mesenteric venous thrombosis (5%)⁴.

Nearly 5% of peripheral emboli which is usually from the heart, are discovered as mesenteric arterial embolism⁵.

Almost 15% of emboli get lodged near origin of the SMA, whereas most often the emboli lodge distally in the tapered segment of the SMA⁶.

Mesenteric ischemia ultimately leads to gangrene of the bowel. The onset of peritoneal signs indicates bowel infarction, as do signs of sepsis and hemodynamic instability. There is only 50% survival if the diagnosis is made in first 24 hours, and survival decreases to 30% or less if diagnosis is delayed^{7,8}.

Laboratory investigations are nonspecific. Normal laboratory values do not rule out the diagnosis of mesenteric vascular occlusion. It is advisable not to delay angiography when there is strong clinical suspicion. Distended loops of bowel and dynamic ileus are some of the subtle signs of acute mesenteric ischemia on plain radiographs⁹. The gold standard for the diagnosis of acute mesenteric ischemia is selective mesenteric angiography with a sensitivity of about 90% and specificity nearly 100%. Nevertheless the pillars of modern diagnostic technique in mesenteric ischemia are computed tomography angiography and magnetic resonance angiography¹⁰.

Restoration of blood flow to the ischemic intestine, resection of nonviable bowel, and supportive care are among the early interventions in mesenteric ischemia. Papaverine, a phosphodiesterase inhibitor, is the most common agent used as intraarterial vasodilator therapy in practice today. It is given directly into the SMA at 30 to 60 mg/h. Operative embolectomy has been the traditional procedure in the acute thromboembolic event with reasonable short and long-term outcomes. Endoluminal therapy has emerged as a main therapy recently for intestinal ischemia. Many centers use intracatheter fibrinolysis as the first treatment for SMA thromboembolism¹¹.

Conclusion

Acute abdominal pain without much abdominal signs with forceful evacuation of gut, along with vomiting in a patient should greatly increase the suspicion for acute mesenteric ischemia. As acute mesenteric ischemia may lead to intestinal infarction within no time, prompt diagnosis and treatment are very essential. In the setting of a suspicious history and physical examination, the physician must keep a high index of suspicion for early diagnosis of mesenteric ischemia.

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