A Life Threatening Cause of Abdominal Pain: Mesenteric Ischemia
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ABSTRACT

Introduction: Mesenteric ischemia can be divided into acute and chronic ischemia. These are two separate entities, each with their specific clinical presentation and diagnostic and therapeutic modalities. However, diagnosis may be difficult due to the vague symptomatology and subtle signs.

Case Presentation: We report the case of a 36-year-old male who presented with abdominal pain, and vomiting. A physical examination revealed mild abdominal tenderness. Computed tomography of his abdomen, angiography revealed occlusion of branches of the superior mesenteric artery. Local thrombolysis of the occluded branches was done. The patient got discharged in a stable condition. He however again presented with complaints of pain in abdomen and vomiting 15 days later. His CT angiography was one which was suggestive of chronic occlusion of branch of superior mesenteric artery with ischemic changes in the jejunal segment supplied by it. The ischemic segment was resected and an end-to-end anastomosis was performed.

Conclusion: Mesenteric ischemia is a pathology well-known by surgeons, gastroenterologists and radiologists. Acute and chronic mesenteric ischemia are two separate entities with their own specific clinical presentation, radiological signs and therapeutic modalities. Early diagnosis and thrombolysis helps in preserving the bowel.

Introduction:
Mesenteric ischemia can be divided into acute and chronic ischemia. These are two separate entities, each with their specific clinical presentation and diagnostic and therapeutic modalities. However, diagnosis may be difficult due to the vague symptomatology and subtle signs.

Case Presentation:
A 36-year-old male, ground staff of airlines was admitted to our Department of Interventional radiology for persistent abdominal discomfort, and vomiting for several months. He had no history of hypertension, Diabetes mellitus, no dislipidemia. He was a non smoker and non alcoholic.

On physical examination, abdomen was soft, non tender. Liver and spleen were not palpable. Bowel sounds were present.

His ECG and 2-D Echocardiography were within normal limits.

Laboratory investigations were within normal limits.

His mesenteric angiogram revealed acute thrombus in jejunal branch (Fig. 1) and ilio-colic branches (Fig. 2) of superior mesenteric artery. The iliocolic bronch was canulated with microcatheter and inj Urokinase 2 lac IU was given as bolus. Then jejunal branch was canulated with pulse spray catheter was placed and inj Urokinase 1 Lac IU was given as bolus. This was followed by infusion of Inj Urokinase @ 60,000 IU/hr. Check angiogram was done 24 hrs. later which revealed complete resolution of thrombus with good flow in the said branches. (Fig. 3 & 4)

The patient was discharged after 48 hrs. on antiplatelets, oral anticoagulants and kept on follow up. He presented 15 days later with complaints of pain in abdomen and vomiting. His CECT scan of abdomen revealed thickening in the jejunal loops resulting in stricture suggestive of likely ischemic changes. (Fig. 5 & 6)

Barium meal follow through was done which revealed stricture in jejunal segment. (Fig. 7 & 8)

He was referred to surgeon for exploratory laprotomy. The stenotic ischemic jejunal segment was resected and an end-to-end anastomosis was performed. Patient improved on treatment and got discharged in a stable condition.
developing AMI depend on the etiology: more often patients are older than 50 years and suffer from congestive heart failure, recent myocardial infarction, hypovolemia, hypotension or sepsis. Clinical presentation is nonspecific, often presenting as a sudden onset of severe abdominal pain, frequently out of proportion to what is found at physical examination. Accompanying symptoms and signs are nausea, vomiting & hypotension.

Discussion:
Acute mesenteric ischemia (AMI) accounts for 60% to 80% of all cases of mesenteric ischemia and has a mortality rate between 59% and 93%. AMI can be caused by arterial emboli, arterial or venous thrombosis and non-occlusive obstruction. In all causes, the sudden onset of intestinal hypoperfusion can lead to hypoxemia and intestinal hypoxia with irreversible bowel damage. Risk factors for
The absence of specific signs upon physical examination can make the diagnosis of AMI very challenging and the clinical consequences of missed AMI can be catastrophic.

**Acute mesenteric arterial embolism:**
Roughly, 50% of all cases of AMI are due to acute mesenteric embolism\(^9,10\). Mesenteric emboli can originate from the left atrium, associated with cardiac dysrhythmias such as atrial fibrillation, left ventricle with global myocardial dysfunction associated with poor ejection fraction, or cardiac valves due to endocarditis. Occasionally emboli generated from an atherosclerotic aorta. Emboli typically lodge at points of normal anatomic narrowing, and the SMA is particularly vulnerable because of its relatively large diameter and low takeoff angle from the aorta. The majority of emboli lodge 3 to 10 cm distal to the origin of the SMA, thus classically sparing the proximal jejunum and colon.

*Fig. 5 & 6: CECT abdomen showing thickening in Jejunal loops*

*Fig. 7 & 8: Stricture seen in jejuna segment*
Contrast enhanced multidetector CT angiography enables fast and detailed evaluation of the mesenteric circulation and abdominal viscera which provides accurate and rapid AMI diagnosis. Occlusive emboli in the SMA can be readily detected within the lumen and are generally localized beyond the middle colic artery origin, leading to abrupt cessation of contrast material flow within the vessel lumen. Few or no collaterals are seen secondary to the acute nature of the pathology. Because of its high sensitivity (90-100%) and specificity (about 100%), as well as therapeutic potential (possibility of intravascular administration of vasodilators and thrombolytics) selective catheter angiography is accepted as the gold standard for diagnosis of AMI. On angiography, emboli to the SMA are typically manifested as sharp, rounded filling defects in the contrast column with high-grade or subtotal occlusion of distal flow. Vasospasm, distal to the site of embolic occlusion, is frequently present. It is important to assess the development of collateral vessels from the coeliac axis or the inferior mesenteric artery connecting with distal branches when total occlusion of the SMA is seen. Concordantly with CT angiography findings, enhanced collaterals detected with catheter angiography may indicate chronic occlusion of the SMA. However, in some cases of AMI rapid establishment of a collateral vessel prevents development of bowel necrosis.

Treatment is dependent upon the type of AMI, but in most situations any patient with peritoneal signs should be operated upon without delay. Standard surgical therapy for AMI involves resection of irreparably damaged bowel and reestablishment of mesenteric blood flow through embolectomy. Patients with minor emboli, defined as emboli limited to SMA branches or to the SMA distal to the ileocolic artery, may be managed non-operatively with volume resuscitation, broad-spectrum antibiotics, vasodilators, and anticoagulants.

Conclusion:
Mesenteric ischemia is a pathology well-known by surgeons, gastroenterologists and radiologists. Acute and chronic mesenteric ischemia are two
separate entities with their own specific clinical presentation, radiological signs and therapeutic modalities. The natural history of AMI, in absence of treatment is nearly always fatal. Ischemia leads to infarction, perforation, and death in vast majority of patients. The focus of treatment should be to provide an aggressive and rapid diagnosis to minimize the amount of ischemic bowel that will progress to infarction, while rapidly instituting endovascular therapy.

In conclusion, AMI is a relatively uncommon cause of abdominal pain, but one with a high mortality rate. Its prompt recognition and aggressive treatment can prevent bowel infarction and improve outcomes. As in our case, early thrombolysis prevented ischemia to the distal ileal loops. In the proper clinical setting, it is crucial to maintain a high index of clinical suspicion so that a correct diagnosis can be made and treatment initiated expeditiously.

References: