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Review Article

Massive Intestinal Gangrene from Acute Mesenteric Ischemia: A Case Report and Literature Review

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Abstract:

Acute mesenteric ischemia(AMI) is a sudden interruption of the blood supply to a segment of the bowel resulting in reversible changes to bowel function and eventual gangrene. It may be occlusive or non-occlusive with acute mesenteric arterial embolism(AMAE) as the commonest. Clinical diagnosis is a challenge with the early clinical picture usually that of a discrepancy between severe abdominal pain and paucity of clinical signs on abdominal examination. Presented is a 72 year old man with sudden onset of severe abdominal pain. He had laparotomy that revealed massive intestinal gangrene with the distribution in keeping with superior mesenteric arterial embolism. He was offered resection and jejuno-colic anastomosis with an eventual mortality. Other findings were extrinsic gastric gastrointestinal stromal tumour(GIST), chronic cholecystitis and large bowel vascular ectasia. This is a surgical emergency that requires a high index of suspicion based on the history of sudden onset of abdominal pain in the setting of paucity of commensurate abdominal sign on clinical examination. Rapid resuscitation and emergency exploration to assess intestinal viability and institution of appropriate therapy to establish intestinal perfusion is recommended as resection of nonviable intestine may require extensive bowel resection with high morbidity and mortality.

Keywords: acute mesenteric ischemia; superior mesenteric embolism; massive intestinal gangrene

Introduction:

Acute mesenteric ischemia(AMI) is a term that describes a spectrum of bowel injury arising from a sudden interruption of the blood supply to a segment of the intestine resulting in reversible changes in bowel function to gangrene[1,2]. Antonio Beniviene was the first to describe mesenteric ischemia in the 15th century[2]. It may be non-occlusive or occlusive, the later, arterial or venous depending on the etiology. Based on the primary etiology, it is further described as: mesenteric arterial embolism(50%), mesenteric arterial thrombosis(15-25%) and mesenteric venous thrombosis(5-15%)[2,3]. AMI accounts for 0.1% of hospital admissions[4,5]. Clinical diagnosis is difficult, as the symptoms of AMI are non-specific with the early clinical picture usually that of a discrepancy between severe abdominal pain and paucity of clinical signs on abdominal examination[2,4,6]. The imaging of choice is selective CT angiography[4]. The first successful surgical therapy for AMI performed by Elliot in 1895 was a resection of gangrenous portion of the bowel followed by primary anastomosis of the viable parts2. Treatment options include a spectrum of percutaneous

angioplasty and stenting or thrombolytic therapy and eventually surgical exploration[4]. When there is bowel infarction, mortality is high and may be up to 90%[5]. Presented is a 72 year old man with sudden onset of severe abdominal pain. He had laparotomy that revealed massive intestinal gangrene and was offered resection and jejuno-colic anastomosis with an eventual mortality. This is to highlight the diagnostic and therapeutic challenges and call for a high index of suspicion and prompt therapy in patients with sudden onset of severe abdominal pain at variance with the clinical findings on abdominal examination.

Case report:

Here is a 72-year-old man with sudden onset of severe abdominal pain of 5 days duration who presented to the emergency department as a referral from a peripheral secondary health care facility after 3 days of the onset of symptoms. He was seen initially by the physicians in our facility who further referred the patient to surgery after 24 hours. Pain was generalized but worse at the right flank. It was aggravated with meals. There was no prior history of postprandial pain nor food fear. There was associated history of one episode of vomiting that was non-projectile with vomitus of small volume and consisting of recently ingested food. The vomitus was non-bloody and non-bilious. There was a history of anorexia but no history of weight loss, abdominal distension, jaundice, fever, nor diarrhoea. Patient opened bowel to stool 3 days prior to presentation to dark coloured stool. He was not a known peptic ulcer disease patient. Patient was a nonsmoker and did not admit to any history of cardiac disease. Drug history was unremarkable. There was a history of frequency and dysuria.

On examination, an elderly man, not pale, anicteric, acyanosed, dehydrated, nil pedal oedema. He had normal vital signs at presentation. The abdomen was asymmetrically distended with epigastric fullness. There was generalized tenderness with guarding marked over the epigastrium. There was positive renal punch test, no ascites and bowel sounds were reduced. Rectal examination showed reduced sphincteric tone and rectum containing scanty amount of dark-coloured faeces. An assessment of acute abdomen due to a perforated viscus was made.

He was resuscitated, investigated and had exploratory laparotomy the next day after presentation.

FBC: PCV 45%, WBC 6.5 X 10^5/L (Neutrophil 73%, Lymphocytes 27%), Platelet count was normal. E/U/Cr was unremarkable and Retroviral serology(RVS) non-reactive. Urinalysis showed bilirubinuria, proteinuria and haematuria. Abdominal USS reported 1). Ascending UTI, right pyelonephritis with hydronephrosis, 2) Simple left renal cortical cyst, 3) Biliary sludge. Plain Abdominal Xray showed no evidence of perforation but suggested ileus.

He had a laparotomy with the following intra operative findings: Gangrenous bowel extending from the jejunum (20cm from the ligament of Treitz) to the distal part of the ascending colon, inflamed gallbladder, Figure 2a, gastric mass on the lesser curvature of the stomach, Figure 3a and right renal cyst. He had the following procedures: Excision of gastric mass, Figure 3b, resection of gangrenous segment of bowel and jejuno-colic anastomosis and cholecystectomy, Figure 2b.

Abdomen was closed over a tube drain. About 12 hours after surgery, patient's G.C.S started deteriorating, could not maintain oxygen saturation and was commenced on oxygen and had oliguria. Attempts at resuscitation proved abortive and patient passed on about 18 hours after surgery.

Histology reported on macroscopy: Right hemicolectomy specimen that measured 206cm with haemorrhagic surface. Microscopy: Gastric mass- gastrointestinal stromal tumour, Figure 4ab, chronic cholecystitis, Figure 5a,5b, gangrenous bowel, Figure 6 and the large intestine- ascending colon- vascular ectasia, Figure 7. Based on the distribution of the gangrenous bowel sparing the proximal jejunum and extending to the ascending colon, a definitive diagnosis acute mesenteric arterial embolism(AMAE) was made with incidental findings of gastrointestinal stromal tumor, chronic cholecystitis and colonic vascular ectasia.

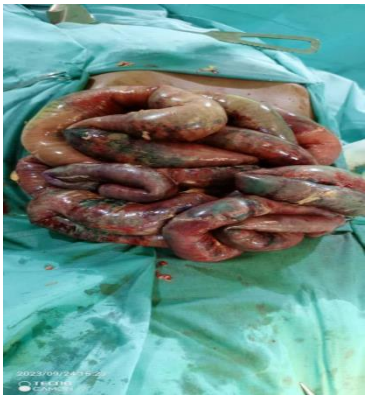


Figure 1: Gangrenous loops of bowel in situ.

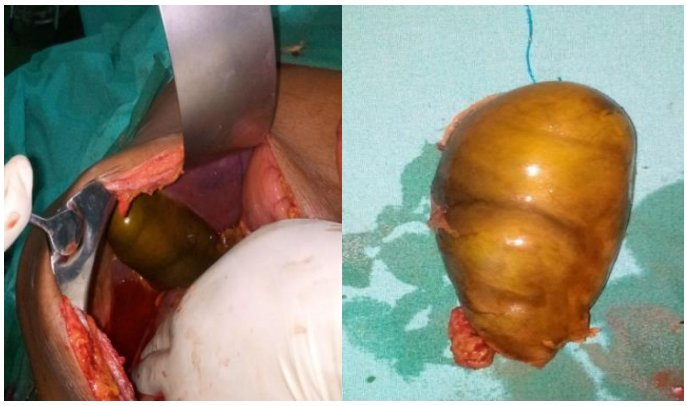


Figure 2a & 2b: Fig 2a- Inflamed gallbladder in situ. Fig 2b- Cholecystectomy specimen.

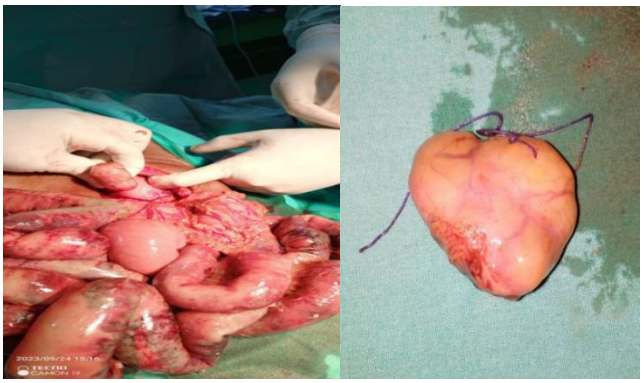
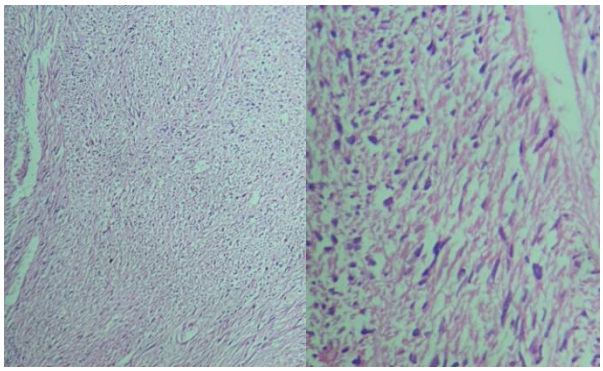
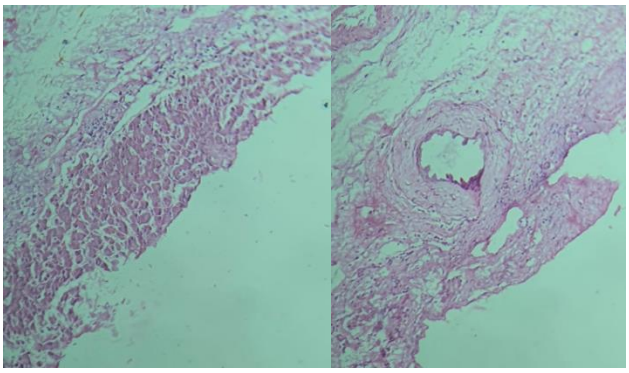


Figure 3a & 3b: Fig 3a- Gastric tumour attached to the lesser curvature of the Stomach. Fig 3b- Excision biopsy specimen.



Figures 4a & 4b: 4a- H & E x 40. 4b- H & E x 100- Gastric mass that showed tumour composed of bland spindle cell with faintly eosinophilic cytoplasm in a syncytial pattern with elongated nuclei and inconspicuous nucleoli. Elsewhere was seen round cells with clear eosinophilic cytoplasm in sheets. - Gastrointestinal stromal tumour.



Figures5a & 5b. 5a: 5b- H &E x40. Section of the gallbladder that showed variable amounts of predominantly lymphocytic inflammatory cell infiltrates in the lamina propria. There is thick and fibrotic wall- Chronic cholecystitis.

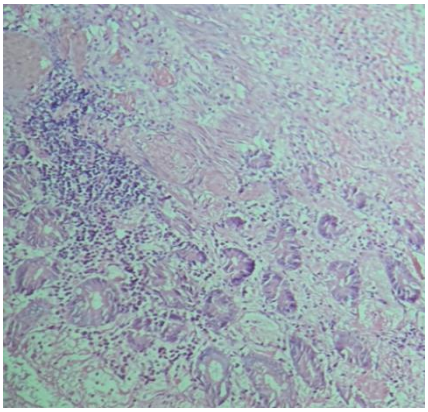


Figure 6: H & E X 40. Section of the intestine- jejunum that showed ghost-like appearance of gland and stromal elements. There was aggregate of mixed inflammatory infiltrates mainly mononuclear cells- gangrenous bowel.

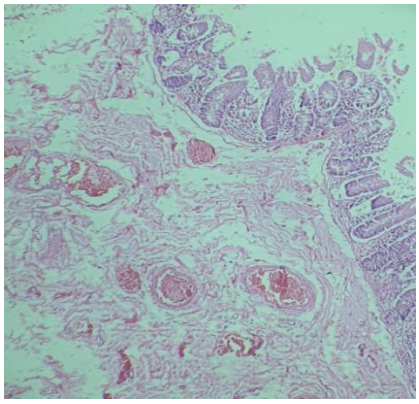


Figure 7: H & E x 100. Section of the large intestine with dilated and thin walled blood vessels in the mucosa and submucosa. The overlying mucosa shows partial erosion and the thinned out segments displays loss of mucosa with moderate inflammatory infiltrates- Vascular ectasia.

Discussion:

Acute mesenteric ischemia is a life-threatening pathology due to inadequate or complete interruption of blood supply through the superior or inferior mesenteric artery(SMA or IMA) resulting in hypoperfusion, ischemia and eventual gangrene of the bowel[4,5]. It is an uncommon condition with diagnostic and therapeutic problems; hence the prevalence is difficult to establish. The overall incidence is low and accounted for 0.09-0.2% of all acute admissions to the emergency departments as an uncommon cause of abdominal pain[6]. AMI is regarded as a disease of the elderly, typically in those older than 60 years[4,5]. It is rare below 45 years, most of the deaths occur after the 7th decade and it affects women more than males with a F:M ratio of 2:1[2]. Others have reported no overall sex preference including racial predilection[5].

There are 5 types of AMI based on the anatomy of the vessels and etiologies: Non occlusive mesenteric ischemia(NOMI)- 20-30%, acute mesenteric arterial embolism(AMAE)- 40-50%, acute mesenteric arterial thrombosis(AMAT)- 15-25% and mesenteric vascular thrombosis(MVT)- 5%. The 5th- 3% from mechanical scenarios like strangulation, intussusception, volvulus, tumour and trauma[4]. Majority of AMI cases are caused by emboli(40-50%) in keeping with our finding based on the distribution of the gangrene in the region supplied by the SMA. Mesenteric emboli can originate from the heart from atrial fibrillation and cardiac valves due to endocarditis. Sometimes, the emboli is generated from atherosclerotic aorta[1]. However, we did not have pointers to any of these in our patient from clinical evaluation. Majority of the emboli lodge 3-10cm distal to the origin of the SMA, thus classically sparing the proximal jejunum[1]. This is because the origin of the inferior pancreatico-duodenal branch is spared thereby maintaining perfusion of the proximal jejunum while the rest of the small intestine ischemic or gangrenous[2]. Diagnosis of SMA embolism can be made during surgery based on the distribution of the gangrene in the bowel[2]. This was in keeping with the finding of gangrenous bowel extending from the jejunum (20cm from the ligament of Treitz) to the distal part of the ascending colon during surgery. The second cause of AMI, AMAT is found in 15-25% of all AMI events. The thrombosis occurs commonly near the origin of the SMA usually in the setting of severe atherosclerosis. The

extent of intestinal involvement is greater than that due to embolism, extending from the duodenum to the transverse colon. MVT(5%) is the least common cause of mesenteric ischemia usually secondary to clotting disorders[2].

The clinical presentation may be based on the classic triad of AMI with the first as the most important: 1. Pain out of proportion with clinical examination as noted in our patient. 2. Bowel emptying from reflex spasm resulting in vomiting, diarrhoea. 3. Potential embolic source, however, this was not elicited in our patient. Severe pain in the middle and upper part of the abdomen out of proportion to the physical findings as was shown in our patient should be assumed to be AMI until disproven. This is the key to early diagnosis and should arouse a high level of clinical suspicion[7]. When physical examination demonstrates signs of peritonitis which may be subtle, then there is irreversible intestinal ischemia depicting gangrene[7]. In some studies of clinical presentation in AMI, 95% of patients presented with abdominal pain, 44% with nausea, 35% with vomiting, 35% with diarrhoea and 16% with blood per rectum(dark stool found in our patient) and constipation in 7%[1,2,4]. The signs of AMI overlap with acute pancreatitis, acute cholecystitis, small bowel obstruction and acute diverticulitis. It is remarkable to highlight the diagnostic challenge that resulted in the patient being offered an exploration after five days of onset of symptoms. This highlights the need for a high index of suspicion and prompt intervention.

Laboratory studies are generally nonspecific. Most common abnormalities are hemoconcentration as was noted in our patient, leukocytosis and metabolic acidosis[4]. There are no laboratory studies that are sufficiently accurate to identify the presence or absence of ischemic or necrotic bowel, although elevated I-lactate and D-dimer may be useful. Plain X-ray have limited diagnostic value in evaluating AMI as normal finding may be seen in 25% of cases. However, signs of intestinal perforation may be seen, other characteristic features are thumbprinting or thickening of bowel loops in <40% of cases. USS is a first line investigation for acute abdominal conditions. It is of little value in AMI even when combined with doppler. It is technically limited by the presence of air-filled distended bowel loops[2]. When there is clinical suspicion, MDCT(multi detector CT) scan angiography is the 1st line diagnostic imaging with a sensitivity of 83% and specificity of 93%. It is considered gold standard in the diagnosis of AMI and should be performed as soon as possible for any patient with suspicion of AMI[1,4,5]. Findings in AMI are thickened edematous bowel walls, hematoma, dilated loops of bowel, engorged mesenteric veins, pneumatosis, gas in the mesentery or portal veins, gangrene and frank arterial or venous thrombosis. Early angiography is a major factor for the decline in mortality in patients with AMI[2].

Following the diagnosis of AMI, Fluid resuscitation should be commenced to enhance visceral perfusion, correct electrolyte abnormalities and institute nasogastric decompression. Broad spectrum antibiotics should be commenced and unless contraindicated patient should be anticoagulated with IV heparin. Surgical revascularization remains the treatment of choice through a longitudinal arteriotomy embolectomy using a Fogarty catheter or distal bypass graft anastomosis[4]. Prompt laparotomy should be done and the finding of massive gut necrosis as was our experience required careful assessment of the patients underlying comorbidities. Our patient had an extrinsic GIST and chronic cholecystitis adjudged as incidental findings for which excision biopsy of the gastric mass and cholecystectomy done(Figures 4 & 5). Also noted was vascular ectasis of the large bowel(Figure 7). He was offered resection and jejuno-colic anastomosis. The length of the bowel resected was 206cm long. The patient would have had challenges associated with short bowel syndrome with its associated consequences. Patients lifestyle for survivors may be hindered by a lifetime of Total parenteral nutrition and in the alternative, small bowel transplantation may be required[2,5]. Prompt diagnosis and intervention are essential for the reduction of the high mortality (50-80%). Currently there are no level 1 evidence to guide the evaluation and treatment of suspected AMI[1].

The outcome in our patient was poor, patient died about 18 hours post-surgery from shock that resulted in multiple organ failure. The main reason for this poor outcome is the persistent difficulty in recognizing this condition prior to the onset of gangrene[2]. Even with good treatment as many as 50-80% of patients die[5]. AMI is a life threatening condition; prognosis is poor and accounts for a mortality from 50-90% in acute mesenteric arterial embolism and acute mesenteric thrombosis[4]. The most important prognostic factor is the early diagnosis[2]. Collaboration between the surgeon, radiologist and the anesthesiologist is recommended.

Conclusion:

AMI is a surgical emergency that requires a high index of suspicion based on the history of sudden onset of abdominal pain in the setting of paucity of commensurate abdominal sign on clinical examination. This clinical scenario in a strong indication for CT angiography which could be diagnostic and therapeutic. Rapid resuscitation and emergency exploration to assess intestinal viability and institute appropriate therapy to establish intestinal perfusion is recommended as resection of nonviable intestine may require extensive bowel resection with high morbidity and mortality.

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