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AN EMERGENT MANAGEMENT OF ACUTE SUPERIOR MESENTERIC ARTERY EMBOLISM WITH ILEAL GANGRENE

Surgery

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ABSTRACT

Acute mesenteric ischemia (AMI) occuring due to sudden, partial or complete interruption of blood flow in main visceral arteries of the abdomen eventually resulting in intestinal ischemia and/or bowel gangrene is a surgical emergency. It represents 0.1% of hospital admissions and 2% of the revascularization operations for atheromatous lesions. 50% of AMI is caused by embolic phenomenon, 25% by thrombotic episode and rest 25% by both. The most common vessel involved in AMI is superior mesenteric artery. Acute mesenteric embolic ischemia (AMEI) arises typically from a cardiac emboli in patients with atrial fibrillation or following MI. Patients usually presents with central abdominal pain, out of proportion to the physical findings initially, later becoming diffuse associated with bloody diarrhoea during the episode. An early diagnosis, an aggressive resuscitation, intravascular or surgical restoration of blood flow and subsequent bowel resection based on bowel viability helps reduce morbidity and mortality.

KEYWORDS

Superior mesenteric artery, occlusion, early diagnosis, CECT abdomen.

INTRODUCTION

Acute mesenteric ischemia (AMI) is a surgical emergency, consisting of sudden, partial or complete interruption of blood flow in main visceral arteries of the abdomen eventually resulting in intestinal ischemia and bowel gangrene. It is a relatively uncommon entity representing 0.1% of hospital admissions [1, 2] and 2% of the revascularization operations for atheromatous lesions. 50% of AMI is caused by embolic phenomenon, 25% by thrombotic episode and rest 25% by both [3]. The most common vessel involved in AMI is superior mesenteric artery. Acute mesenteric embolic ischemia (AMEI) arises typically from a cardiac emboli in patients with atrial fibrillation or following MI [3]. Patients usually presents with central abdominal pain, out of proportion to the physical findings initially, later becoming diffuse associated with bloody diarrhoea during the episode [3]. Other conditions mimicking the severe abdominal pain of acute onset such as pancreatitis, cholecystitis, nephrolithiasis, intestinal obstruction and hollow viscus perforation should be ruled out. Despite advancement in the diagnostic and treatment modalities, AMI still continues to be a diagnostic challenge due to the non-specific nature of presentation resulting in delayed diagnosis and septic complications leading to very high morbidity and mortality. Treatment of AMI is focused on aggressive resuscitation, early diagnosis, intravascular or surgical restoration of blood flow and subsequent assessment of the bowel viability. Here, we report a rare case of AMEI admitted in our hospital with symptoms mimicking acute pancreatitis.

CASE STUDY

A 35 year gentleman, chronic smoker with ganja addiction and an alcoholic for 15 years, with no known co-morbidities presented to the casualty with sudden onset of crampy pain in the periumbilical region with multiple episodes of vomiting and melena for 2 days. His past history is suggestive of similar compliant 5 years ago which was relieved with conservative management. 1 year ago for similar complains, he underwent UGI endoscopy which revealed esophageal candidiasis with multiple D1 erosions. He was managed conservatively with medications and regular follow-up with the gastroenterologist till this acute presentation. On examination, pallor present, per abdomen examination revealed distended abdomen with ill defined tenderness all over the abdomen with no guarding or rigidity. Digital rectal examination revealed blood stained fingers. All blood investigations were within normal limits including a Hb 12.5 gm/dL, TLC 8.9 cumm, Platelet count 3.32 lakh/microL except CRP 426.54 mg/L, S. bilirubin 2.43 mg/dl, SGOT 116 U/L, SGPT 92 U/l ; serum amylase, lipase and serum electrolytes were within normal limits.

USG abdomen and pelvis showed GB sludge. UGI endoscopy revealed esophageal candidiasis and duodenal (1st part) erosions. CECT abdomen showed hepatomegaly with features suggestive of systemic infiltrative disease, near complete thrombosis of distal SMA with dilated small bowel loops (4.1cm) and reduced wall enhancement suspicious of ischemia and right renal infarct.

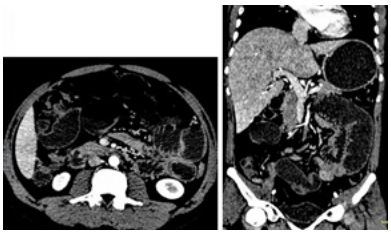

Figure 1: CECT abdomen and pelvis showing dilated small bowel loops and reduced wall enhancement suspicious of ischemia with near complete thrombosis of distal SMA.

An emergency exploratory laparotomy revealed patchy gangrene of ileum with bluish discoloration, absence of peristalsis and absence of mesenteric pulsation extending from 30 cm proximal to the ileocaecal junction till 130 cm cranially along the ileum.

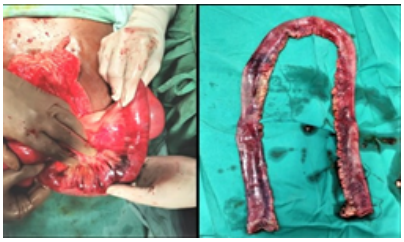

Figure 2: Intra-operative picture showing patchy gangrene of the distal ileum which was resected.

Infective tissue flakes were seen over gangrenous bowel loops without adhesions, rest of the bowel is normal with no intra abdominal collection. The nonviable ileal segment measuring ~130cm was resected followed by primary ileo-ileal end to end anastomosis and specimen was sent for HPE.

Post-operative period was uneventful. On 1st post-operative day, 2D echo as advised by cardiologist due to ECG changes (short PR Interval, poor T wave progression V3 and V4, ? suspect anterior myocardial infarction with T wave abnormality) revealed Large LV clot ~ 2.8cm X 0.7cm attached to LV apex following which patient was started on inj. Heparin 5000 IU I.V 6 hrly. He was started normal diet on POD-3, drain was removed on POD-4. A B/L lower limb arterial and venous doppler study revealed no abnormality.

HPE revealed non-specific gangrenous changes of ileum. Patient was discharged on POD-10. 2D ECHO done on subsequent follow up visit after 6 weeks revealed complete resolution of left ventricular clot and he was advised oral anti-coagulants (Tab. Apixaban 5mg twice daily, Tab. Clopidogrel 75 mg once daily) with follow-up after 3 months.

DISCUSSION

Mesenteric vascular occlusive diseases are relatively uncommon. The overall incidence is low at 0.09% to 0.2% [4] and found to be more common among females (3:1). Commonest cause is atherosclerotic vascular disease. Mesenteric ischemia occurs when 2 out of 3 visceral arteries are affected with severe stenosis or occlusion. It runs a high mortality upto 50-75%.

Types of mesenteric artery occlusive disease are:

1. Acute mesenteric ischemia (Embolic or thrombotic) [AEMI or ATMI]
2. Chronic mesenteric ischemia (CMI)
3. Non-occlusive mesenteric ischemia (NOMI).

Acute mesenteric ischemia can be caused by arterial emboli, arterial or venous thrombosis and non-occlusive obstruction. It characterized by reduced blood flow through mesenteric vessels which leads to bowel ischemia and eventually gangrene. Clinical presentation is non specific, often presenting as severe abdominal pain out of proportion to the physical findings, bloody diarrhoea due episodic pain, accompanied by nausea, vomiting, tachycardia and hypotension. Conventional plain X-ray abdomen have limited diagnostic value in evaluating AMI except showing signs of intestinal perforation. Biochemical and pathological parameters like elevated L-lactate, D-dimer, leukocytosis, elevated level of C-reactive protein, creatine kinase, hemoconcentration and a high anion gap reflects severity of the condition. A high serum amylase, aspartate aminotransferase and dehydrogenase can be found in some of the patients [5] and may act as predictive factors indicating possibility of the presence of ischemic or necrotic bowel. An emergency abdominal computed tomographic angiography (CTA) is the investigation of choice in all patients with suspicion for AMI [6]. Once AMI is diagnosed, immediate fluid resuscitation to enhance visceral perfusion, correction of electrolyte abnormalities, nasogastric decompression and institution of a broad spectrum antibiotics is to be followed routinely. Such patients should be anticoagulated with intravenous unfractionated heparin unless contraindicated. Emergency laparotomy for patients with overt peritonitis and endovascular revascularization procedures for cases with partial mesenteric arterial occlusion is life saving. Damage control surgery should be followed as an important adjunct in patients of compromised bowel viability with refractory sepsis, with reassessment by planned re-laparotomy and bowel resection when needed is an essential live saving part of AMI management. Mesenteric venous thrombosis can often be successfully managed by a continuous infusion of unfractionated heparin.

Most important factor in efficient management of such patients lies in the high level of suspicion and early diagnosis. Investigation of choice is Biplanar mesenteric arteriography which is even therapeutic. It shows occlusion or near occlusion of celiac artery and SMA at or near their origins from the abdominal aorta. Depending upon the etiology, the restoration of blood flow can be achieved by endovascular approach by catheter directed thrombolytic therapy or surgery that is, Transverse arteriotomy with extraction of embolus and restoration of blood flow and assessment of bowel viability.

In 1930, Cokkinis remarked, “Occlusion of the mesenteric vessels is apt to be regarded as one of those conditions of which the diagnosis is impossible, the prognosis is hopeless and the treatment is almost useless” [7].

Because diagnosis is vital to initiate treatment, new diagnostic strategies are required, that could intensify the suspicion of acute mesenteric ischemia (AMI) and help improve the selection of patients before proceeding to CT angiography or MRI and thus help timely management and enhance the outcome. Recent experimental studies are promising for alpha-glutathione S transferase (GST) and fatty acids bound to intestinal proteins (I-FABP) as early markers of intestinal ischemia with good prospects [8].

Non-occlusive mesenteric ischemia (NOMI) should be suspected in a critically ill patient with abdominal pain or distension, requiring vasopressor support and evidence of multi-organ dysfunction, who are especially older than 50 years with history of ischemic heart disease, congestive cardiac failure, renal and peripheral artery disease and patients following cardiac or vascular surgery. Treatments in such patients include correction of the underlying cause and prompt restoration of mesenteric perfusion. The infarcted bowel should be resected without delay.

CONCLUSION

AMI is an acute emergency surgical condition needing a high index of suspicion based on the patient's history of abrupt onset of abdominal pain, clinical and biochemical parameters like elevated l-lactate and D-dimer suggestive of acidosis and organ failure, an elevated alpha-glutathione S transferase (GST) and fatty acids bound to intestinal proteins (I-FABP) as early marker of bowel ischemia. This clinical scenario prompts to undertake CT angiography to establish the diagnosis. Management by rapid resuscitation with IV fluids and after a thorough assessment of the CTA, the patient should be subjected to emergency exploration to assess bowel viability, measures taken to re-establish vascular flow, and to resect non-viable bowel and appropriate corrective measures for bowel continuity. In selected cases, the employment of damage control surgery is essential and life saving. Planned re-assessment of the bowel with further resection or anastomosis and establishment of a stoma as needed is highly recommended. A team work involving acute care surgeons, vascular surgeons, radiologists and anesthetists is essential in optimizing the outcome in such patients.

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