Case Report

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Acute Mesenteric Ischemia: An Unusual Cause of Hematemesis in a 48 Year Old Man

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Abstract

Acute mesenteric ischemia is always an emergency and is associated with a high mortality rate. It predominantly affects elderly people with an underlying cardiovascular disorder. Although most of the cases present with sudden onset of abdominal pain, atypical symptoms including mental confusion, bacteremia, and gastrointestinal (GI) bleeding may be the foremost presentation.[1] GI bleeding may be the dominant symptom in rare cases and almost always presents as occult or overt lower GI hemorrhage.

We report the case of a 48-year-old man who presented to the Emergency Department (ED) of a university teaching hospital in northern India with the complaints of progressive abdominal pain and hematemesis.

Our reasons for highlighting this case are:

- The unusual presentation of an often clinically subtle disease made this case especially challenging to diagnose.
- The presence of confounding factors and "red herrings" caused further delay in the diagnosis of mesenteric ischemia.
- Mesenteric ischemia continues to remain a provisional diagnosis of low suspicion in a middle aged individual with abdominal pain and upper gastrointestinal hemorrhage presenting to the ED.

Keywords: Hematemesis; Gastrointestinal hemorrhage; Mesenteric ischemia.

INTRODUCTION

Acute mesenteric ischemia (AMI) may be defined as a sudden interruption of the blood supply to a segment of the small intestine, leading to ischemia, cellular damage, intestinal necrosis, and eventually patient death if untreated.[2] The incidence is low, estimated at 0.09 – 0.2% of all acute admissions to emergency departments. Therefore, although the entity is an uncommon cause of abdominal pain, diligence is always required because if untreated, mortality has consistently been reported in the range of 50%.[3]

AMI may be non-occlusive (NOMI) or occlusive, with the primary etiology further defined as mesenteric arterial embolism (50%), mesenteric arterial thrombosis (15-25%), or mesenteric venous thrombosis (5-15%).[4]

It is important to note that there are currently no level 1 evidence to guide the evaluation and treatment of suspected AMI, and the published literature contains primarily institutional reviews, case series and personal recommendations with no clearly defined treatment guidelines. Bearing this in mind, the emergency physician is left with only a high index of clinical suspicion to rule in the disease; the presence of multiple variegated factors pointing towards other causes make this entity even more difficult to suspect, evaluate and ultimately, treat successfully.

CASE PRESENTATION

A 48-year-old man presented to the Emergency Department (ED) of a university teaching hospital in Dehradun, India with a history of abdominal pain for 3 days which had increased in severity over the past 24 hours. This was associated with profound nausea, 3 episodes of "coffee-ground" vomiting, and progressive abdominal distension since a day. He did not have melaena, hematochezia, diarrhea or constipation, fever, or jaundice. The patient denied previous medical or surgical comorbidity, but mentioned a consistent intake of alcohol (CAGE 3) and a strong history of non-steroidal anti-inflammatory drug usage (diclofenac tablets) for frequent lower back and knee pains.

The initial assessment revealed a diaphoretic and restless patient with a patent airway and intact protective reflexes. Other than tachypnea, his breathing parameters were grossly normal. An examination of the circulatory system showed a pulse rate of 115 beats per minute (regular), capillary refill time of 4 seconds, and a blood pressure of 80/50 mmHg. He had a Glasgow Coma Scale of E4V5M6 (15). His random blood sugar, oxygen saturation on room air, and 12 Lead ECG were normal.

In the secondary survey, there was no pallor or icterus, and he was afebrile. Examination of the abdomen revealed tense tympanic distension with diffuse tenderness and guarding in all the quadrants. Rigidity was present and bowel sounds were absent. Hernial orifices and external genitalia were normal. Digital rectal examination revealed an empty rectum without the presence of blood. There were no physical signs of decompensated liver cell failure.

In the ED, a nasogastric aspiration revealed 150 ml of "coffee-ground" content. The patient was resuscitated successfully with large aliquots of saline through two peripheral lines, and subsequently administered empirical broad-spectrum antibiotics. He was also infused a proton pump inhibitor. Judging by the cascade of events and the patient's background, a provisional diagnosis of perforated peptic ulcer with peritonitis was made, and an emergent surgical consult was sought.

A blood gas analysis was done (pH 7.44, $PaCO_2$ 28 mmHg, PaO2 106 mmHg, bicarbonate 15 mEq/l, potassium 4.7 mmol/l, lactate 5.0 mmol/l). Plain radiographs of the chest and the abdomen were done, which did not show free air under the diaphragm or air fluid levels. The X-rays were also performed in lateral decubitus and propped up positions, but came out normal. A bedside ultrasound was negative for free intraperitoneal fluid but was hindered by significant bowel distension. A contrast enhanced computed tomography of the abdomen was suggested by the emergency physician, but this was deferred by the surgical team due to the patient's serum creatinine being 2.4 mg/dl. He was transferred to the Emergency High Dependency Unit (E-HDU) for continuum of medical care and observation, where he deteriorated over the next two hours. Laboratory findings showed white cell count 16,000/mm3, hemoglobin 11.1 g/dl, and platelet count 1,10,000/mm3. Liver enzymes and amylase were normal at admission.

A decision was taken to place him on invasive mechanical ventilation, insert central venous and arterial lines, and escalate therapy. Considering clinical findings consistent with peritonitis, acute kidney injury and septic shock, the patient was ultimately mobilized to the Operating Room (OR) for an emergency exploratory laparotomy by the attending general surgeon, four hours after admission to the E-HDU.

The intra-operative findings revealed gangrenous small bowel extending from the duodeno-jejunal (DJ) flexure to 30 cm proximal from the ileo-caecal (IC) junction. There was necrosis of the mesentery with approximately 400 ml of sero-sanguineous ascitic fluid. The rest of the bowel and solid organs were normal. A resection of the gangrenous small bowel was done (from jejunum to ileum, 30 cm proximal to IC junction) with closure of the remaining bowel stumps. There was no knowledge of the status of mesenteric circulation at surgery.

During the post-operative period in the ICU, the patient's general status got progressively worse. He died 2 days later.

DISCUSSION

The purpose of this case discussion is two fold:

- To emphasize the need of early diagnosis in acute mesenteric ischemia in the Emergency Department by establishing a high level of clinical suspicion.
- To correlate the available evidence to current practice with a sense of improving the way we evaluate and manage patients with acute mesenteric ischemia in the ED.

The clinical scenario of a patient complaining of excruciating abdominal pain with an unrevealing abdominal exam is classic for early AMI.[5] If the physical exam demonstrates signs of peritonitis, there is likely irreversible intestinal ischemia with bowel necrosis. In a study of AMI, 95% of patients presented with abdominal pain, 44% with nausea, 35% with vomiting, 35% with diarrhea, and 16% with blood per rectum.[6] Noticeably, none of these symptoms are specific. However, findings of subtle peritonitis, or patients in extremis with septic shock, almost always are predictive of intestinal infarction. Severe abdominal pain out of proportion to physical examination findings should be assumed to be AMI until disproved (Recommendation 1B) [7]

A radiograph is usually the initial test ordered in patients with acute abdominal pain but has a limited role in the diagnosis of mesenteric ischemia, especially in the early setting. Plain radiography only becomes positive when bowel infarction has developed and intestinal perforation manifests as free intraperitoneal air. Conventional plain X-ray films have limited diagnostic value in evaluating AMI, although signs of intestinal perforation may be seen (Recommendation 1B).[7]

Although laboratory results are not definitive, they may help to corroborate clinical suspicion. More than 90% of patients will have an abnormally elevated leucocyte count. The second most commonly encountered abnormal finding is metabolic acidosis with elevated lactate level.[8] It should be emphasized that the presence of lactic acidosis in combination of abdominal pain should lead to consideration for early CT angiography. Elevated serum lactate levels >2 mmol/l may assist in identifying the presence of ischemic or necrotic bowel (Recommendation 1B).[7]

Delay in diagnosis is the dominant factor that accounts for continued mortality rates as high as 30-70% despite vast clinical experience and recognition of this entity. Computed tomography angiography (CTA) should be performed as soon as possible for any patient with suspicion for AMI (Recommendation 1A).[7] CTA should be performed despite the presence of renal failure, as the consequences of delayed diagnosis, missed diagnosis, or mismanagement are far more detrimental to the kidneys and the patient than exposure to the contrast agent.

Fluid resuscitation with crystalloid and blood products is essential for the management of the patient with suspected AMI. To guide effective resuscitation, early hemodynamic monitoring should be implemented.[9] When the diagnosis of AMI is made, fluid resuscitation should commence immediately to enhance visceral perfusion. Electrolyte abnormalities should be corrected, and nasogastric decompression initiated (Recommendation 1B).[7] Vasopressors should be used with caution.

The high risk of infection among patients with AMI outweighs the risks of acquired antibiotic resistance; broad spectrum antibiotics should be administered immediately. Unless contraindicated, patients should be anticoagulated with intravenous unfractionated heparin (Recommendation 1B).[7]

There is overwhelming evidence in literature that peritonitis secondary to bowel necrosis mandates surgery without delay. Intestinal viability is the most important factor influencing outcome in patients with AMI. Prompt laparotomy should be done for patients with overt peritonitis (Recommendation 1A).[7]

CONCLUSION

We have reported a case of acute mesenteric ischemia that presented to us with abdominal pain, hematemesis, peritonitis and shock. We believe that the occurrence of an upper gastrointestinal hemorrhage was a relatively rare and unusual sign for underlying AMI. Despite the misleading symptomatology, a retrospective assessment of this case brought to light several shortcomings in the decision making processes that lead to a negative endpoint.

As emergency physicians, it is imperative that we familiarize ourselves with not only the atypical presentation of this entity, but also with the evidence that may be used to correctly guide our actions in evaluating and managing such patients. The successful treatment for atypical AMI, in our humble opinion, would strongly depend upon close cooperation between critical care specialists, acute care surgeons, radiologists, anesthetists, and vascular surgeons.

REFERENCES

1. Wadman M, Syk I, Elmstahl S. Unspecific clinical presentation of bowel ischemia in the very old. Digitalis treatment a reason for higher mortality? Aging Clin Exp Res 2004; 16:200-205.
2. Patel A, Kaleya RN, Sammartano RJ. Pathophysiology of mesenteric ischemia. Surg Clin North Am. 1992; 72:31-41.
3. Acosta S, Bjorck M. Acute thrombo-embolic occlusion of the superior mesenteric artery: a prospective study in a well-defined population. Eur J Vasc Endovasc Surg. 2003;26:179-83.
4. Clair DG, Beach JM. Mesenteric Ischemia. N Engl J Med. 2016; 374:959-68.
5. Carver TW, Vora RS, Taneja A. Mesenteric ischemia. Crit Care Clin. 2016;32:155-71.
6. Park WM et al. Contemporary management of acute mesenteric ischemia: Factors associated with survival. J Vasc Surg. 2002;35:445-52.
7. Miklosh Bala et al. Acute mesenteric ischemia: guidelines of the World Society of Emergency Surgery. World Journal of Emergency Surgery. 12: 38 (2017).
8. Kougias P et al. Determinants of mortality and treatment outcome following surgical interventions for acute mesenteric ischemia. J Vasc Surg. 2007;46:467-74.
9. Wyers MC. Acute mesenteric ischemia: diagnostic approach and surgical treatment. Semin Vasc Surg. 2010;23:9-20.