Acute Mesenteric Ischemia (AMI): A surgical perspective

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Abstract

Acute mesenteric ischemia (AMI) is a rare surgical emergency as it involves perfusion and need frequent surgical interventions. It is a life threatening emergency with a poor prognosis. It is classified according to the etiological basis thromboembolism, non-obstructive and venous origin. AMI has various risk factors ranging from cardiac arrhythmias to the intraabdominal hypertension. Apart from the signs and symptoms, the multi-detector computer tomography remains a diagnostic tool with accuracy. In the management of AMI patients’ initial resuscitation, hydration and analgesia play important role, however the operative /interventional management is either endovascular thrombectomy with or without stenting when there is no bowel involvement or peritonitis. The laparotomy with open vascular thrombectomy with vascular graft, it is indicated when bowel involvement and peritonitis patients. Often the whole bowel is gangrenous and no further treatment is needed because of certain mortality. The 2nd step is to know the extent of bowel involvement meticulously and resection of bowel with necrosis or gangrene with anastomosis, further relook laparotomies and resection of the bowel may be need. Initially abdomen will be closed with Bagota bag or VAC (Vacuum Assisted Closed) dressing and final staged abdominal closer, once pathology resolves completely. As far as prevention of all
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types of AMI is considered, to date there are no proven preventive measures are available.

Key words: Acute mesenteric ischemia, anti-coagulation, bowel ischemia, bowel resection, endovascular interventions, inferior mesenteric artery and vein open surgical thrombectomy, small and large bowel, superior mesenteric artery and vein, staged abdominal closer.

Acute Mesenteric Ischemia

The acute mesenteric ischemia (AMI) is a life threatening surgical clinical condition. As the ischemia may lead to gangrene and necrosis of the functional bowel, it is mandatory to do urgent surgical interventions. From the increased awareness among physicians, primarily in the fields of gastroenterology as well as abdominal and vascular surgery, a reduction of AMI-related mortality from 90 to 50% in last 3 decade.¹

Epidemiology

Acute mesenteric ischemia is a potentially life threatening surgical emergency. AMI is rare fortunately; it constitutes 0.1% of hospital admissions. Older literature describes the incidence of AMI as 8.8 cases per 100,000 patients.²

In Japan, the incidences are comparatively lower 1:10,000. Most common cause is (up to 50 %) embolism causing acute mesenteric ischemia (AMI), acute thrombus is culprit in 25% of patients, non-obstructive mesenteric ischemia occurs in 20% and in 10% of acute mesenteric ischemia is caused by mesenteric venous thrombosis. The incidence of AMI is increasing with aging population and increasing co-morbidities. Majority of these patients will need surgical interventions. Earlier surgery have better patient’s outcome.³

Classification

AMI is classified as follows:

1. Acute mesenteric arterial embolism, When the embolism is the etiology, it is also occlusive mesenteric ischemia.

2. Non occlusive acute mesenteric ischemia, when there is no complete obstruction and commonly atherosclerosis.

3. Acute mesenteric venous thrombosis, when there is venous obstruction and thrombosis.⁴

Applied Anatomy

Acute Mesenteric Ischemia (AMI) is caused by hindrance or blockage of SMA (Superior mesenteric artery), SMV (superior mesenteric vein) and Inferior mesenteric Arterial supply and drainage. The SMA arises from the abdominal aorta and supplies blood to the midgut from ileum to the transverse colon through various branches (Figure1). IMA is the smallest mesenteric artery
originating from the abdominal Aorta supplies branches to distal transverse colon to the rectum. The superior mesenteric vein is formed by joining various smaller veins draining blood from small intestine, caecum, ascending colon, transverse colon, stomach and pancreas. Inferior mesenteric vessels are rarely involved in blockage due to the presence of collaterals, when compared to the Superior Mesenteric vessels.

**Figure1**: Mesenteric circulation.

**Pathophysiology**
Superior Mesenteric Artery is commonly involved in embolism and thrombosis due to its small off angle and vessel gets narrowed near the origin of the middle colic artery. Pathophysiology of acute mesenteric ischemia changes could be due to thromboembolic phenomenon or non-occlusive processes could be due to thrombotic venous occlusion. The damage to the small bowel and colon is inversely proportional to the blood flow in the mesenteric system. Factors affecting the mesenteric flow are systemic mean arterial pressure, vessels involved, ischemia and patency of
collateral circulation. The affected small and large bowel may be in the state of reversible ischemia to transmural infarction and necrosis. Initial ischemia can cause bowel spasm, but if it persists the bowel mucosal barrier is damaged and luminal contents, bacteria, toxins and venous pro-inflammatory mediators are released into the circulation leading to sepsis and septic shock and multi organ failure. The bowel could be affected by embolism from the heart; SMA becomes narrowed after the first branch, and is at a higher risk of embolism in visceral arteries. The acute angle at origin creates greater flow turbulence and it is a common site of lodging of embolism.

Typically, acute mesenteric arterial thrombosis is usually a complication of atherosclerosis disease and usually occurs at the origin of the aorta. The lower cardiac output and lower flow in SMA stimulate the formation of thrombus resulting in complete cessation of bowel perfusion.

The non-occlusive mesenteric ischemia is commonly seen in patients with sepsis, hypovolemic shock or heart failure, also in critically ill patients on vasopressors. The mesenteric venous thrombosis commonly due to congenital or acquired prothrombotic conditions and it is usually a chronic entity developing thrombus with time.

**Etiology**

The AMI occurs due to venous etiologies as mentioned below:

a) The embolic phenomenon, the emboli from heart, aorta gets lodged in SMA causing acute bowel ischemia.

b) Arterial thrombosis leading to mesenteric ischemia severe, Atherosclerotic disease, severe dehydration, reduction in cardiac output, vasopressors and vascular dissection.

c) The non-occlusive mesenteric ischemia occurs due to hypotension of venous etiology, medication such as vasopressors, cocaine, digitalis and ergotamine. Case reports of intraabdominal hypertension and coeliac artery compression syndrome causing acute mesenteric Ischemia.

Majority of mesenteric venous thrombosis patients had predisposing factors. The most common is congenital or acquired prothrombotic conditions such as deficiency of natural anticoagulants, polycythemia, sickle cell disease, dysfibrinogenemia and medications such as contraceptives. Paraneoplastic syndrome, where tumor causes compression and hypercoagulability, Infectious appendicitis and pancreatitis, venous trauma, and intraabdominal hypertension.

**Risk factors**

There are various risk factors for embolic AMI such as the coronary artery disease, atherosclerosis, hyperlipidemia, smoking, hypertension and endocarditis. Atherosclerosis is described to be the risk factor for arterial superior mesenteric thrombosis; literature described diabetes mellitus the foremost in the causes of acute mesenteric ischemia in these patients. Common risk factor for mesenteric venous thrombosis described in the literature is prothrombotic condition, but Al-Shraim et al. described cirrhosis of liver as the commonest cause of mesenteric thrombosis in their group of patients.
Diagnosis
The abdominal pain out of the proportions is the diagnostic manifestation of mesenteric ischemia. It should always be suspected in patients with above-mentioned co-morbidities with sudden onset abdominal pain. Nausea/vomiting and bloody diarrhea also are manifestations of mucosal bleeding and bowel spasm in mesenteric ischemia patients. Abdominal distention and constipation is also present in most of these patients but diarrhea may also occur. The hemodynamics is dependent on stage of mesenteric ischemia and patients may present in septic shock with organ dysfunction. The laboratory indicators of the mesenteric bowel ischemia are increased D-Dimer and raised serum lactate levels. One has to be careful as both these parameters can be raised in different conditions. The common laboratory finding in AMI is, leukocytosis, metabolic acidosis. The Multi Detector Computed Tomography (MDCT-A) angiography is the imaging of choice to diagnose the mesenteric ischemia. In a recent study MDCT shown to have the specificity of 100 % and the sensitivity of up to 94% to diagnose the mesenteric ischemia.

Therapeutic approach and management of AMI
The management of acute mesenteric Ischemia is summarized in 3R’s: Resuscitation, rapid diagnosis and (Early) Revascularization. Surgical intervention plays the corner stone role in the management of AMI.

Medical management
All patients with mesenteric ischemia should be started on initial resuscitation with focusing on, hydration and analgesia. In cases of Non occlusive mesenteric ischemia the heparin infusion in combination with anticoagulants and minimizing the risk factors is essential.

Treatment of the predisposing factors of non-occlusive mesenteric ischemia is essential. In patients with acute mesenteric arterial embolism should be treated with heparin infusion.

In the situation of acute mesenteric thrombosis should be managed by heparin infusion, surgical by graft or even superior mesenteric artery implantation. In patients with mesenteric venous thrombosis, earlier anticoagulants with heparin alone or in combination with warfarin started.

Endovascular surgery
Patients with AMI with peritonitis an exploratory laparotomy with vascular intervention is the treatment of choice. Angioplasty and stenting is the modality of treatment mainly in patients with mesenteric ischemia due to atherosclerotic plaque rapture. Various studies have shown a comparison between open and endovascular surgery. Ryer in their single center retrospective study found no difference in mortality between the two groups. Whereas, Arthurs et al reported lower morbidity and mortality in the endovascular group. Other 3 studies revealed lower rates of bowel resection and lower mortality and morbidity were seen in these studies for the endovascular group of patients. Hence, the endovascular surgery looks to be promising in the absence of peritonitis. The endovascular procedure may be time-consuming, as the angioplasty is technically difficult because of the anatomy of the SMA, with re-stenosis rates are in the range of 20-50 %.
**Surgical intervention**

Elliot reported resection of bowel due to gangrene is reported as early as in 1895.\(^{22}\) As described above the surgical treatment of AMI consists of a vascular and abdominal surgical approach. Initial step is the reperfusion of the mesenteric arteries and evaluation and demarcation of potential resection of bowel segments. Open vascular repair remained the method of choice in cases of thromboembolic occlusion, with a success rate of 98-100\%.\(^ {23}\) In patients with the intra-abdominal infection such as bacterial translocation or gangrene, an autologous graft reconstruction is better to avoid an infected prosthesis. Other complications are vascular re-occlusions in (2\%) or bleeding (1\%).\(^ {23}\)

The 2\(^{\text{nd}}\) step is the control of the intra-abdominal septic foci; bowel segments with irreversible vascular damage should be located and removed. As the defects are extensive in the mucosa than the external lesions of the intestinal wall, bowel resections with a primary anastomosis should be undertaken only after careful consideration, to avoid the risk of anastomotic leak it is prefer the exteriorization of the healthy intestinal ends as a stoma.\(^ {24}\) The most favorable for the unstable patient is the resection of ischemic bowel segments with a stapler without anastomosis, followed by a re-evaluation after 24-48 h (damage control). Damage control laparotomy is the pivotal in AMI with bowel Involvement is essential, resection of gangrenous organ and leaving abdomen open without any anastomosis is a better approach, the laparotomy is closed by skin only. Repeated laparotomies may need further resection of bowel and finally abdomen can be closed in stages after complete gangrenous bowel resection and anastomosis. In the stage where abdomen is kept open by skin closer only the VAC (Vacuum Assisted Closure) dressing is a better choice.\(^ {25}\)

**Prevention**

The prevention and early management of arrhythmia may prevent embolic SMA occlusion. The non-occlusive AMI, proper management of risk factors and comprehensive regular follow-up by color Doppler may detect patients at the obstruction. The mesenteric venous Ischemia can be prevented up to some extent by preventing the triad of venous stasis, vascular injury and hence concentration. For all practical purposes apart from early timely diagnosis and treatment of at the risk of patient, there is no other option of preventing AMI.

**Conclusion**

Acute mesenteric ischemia (AMI) has remained an abdominal catastrophic clinical entity despite of recent advances in abdominal and vascular surgeries. AMI could be due to embolism, thrombosis, atherosclerotic plaque rapture or venous thrombosis. These patients have risk factors for thromboembolisms, such as coronary artery disease, atherosclerosis and thrombophilia. Patient will manifest with severe post-prandial abdominal pain with high lactate, metabolic acidosis and an early multi-detector CT will diagnose the entity with accuracy, vascular occlusion as well as bowel involvement. The initial AMI management is resuscitation, hydration and analgesia. If peritoneum and bowel are not involved and expertise are available the endovascular intervention, thrombectomy and stenting is increasingly used with variable success rate. If patient already has peritonitis and bowel ischemia the laparotomy with open thrombectomy is the time proven remedy, than one has to assess the extent of bowel involvement and resection should be performed earlier. As the bowel ischemia changes are much more in mucosal layer than the
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serosal one, it is advised that not to anastomose the bowel in initial laparotomy, have subsequent number of laparotomies and reassess the disease progress or control, and usually more than 2 laparotomies are required. Patient should be managed in the intensive care setup. Abdomen should be initially closed temporarily and anticoagulation should be commenced.

The AMI can be prevented up to some extent by preventing the arrhythmias, and regular intake of anticoagulation with diagnosed thrombophilia.

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