



Review Article

Acute Mesenteric Ischemia: Review of Etiologies and Treatment Options

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Abstract

Acute mesenteric ischemia is a life-threatening condition that can be caused by several different pathologies. Numerous treatment options are available including endovascular intervention and open surgical revascularization. Here we review the most common etiologies of acute mesenteric ischemia and modern treatment options.

Introduction

Acute Mesenteric Ischemia (AMI) is a potentially fatal interruption in blood flow to the small intestine. A typical cascade ensues starting with ischemia, then cellular damage followed by bowel necrosis and eventually death if not treated promptly [1]. AMI is typically considered a surgical emergency [2]. While the small intestine receives blood flow from the celiac and superior mesenteric arteries the majority of perfusion comes from the superior mesenteric

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artery (SMA) [3]. The splanchnic circulation receives between 15-35% of cardiac output. However, the oxygen extraction ratio of the small intestine is quite low allowing it to usually avoid ischemia until the blood supply reduces by over 50% [3].

There are four main causes that account for nearly all cases of acute mesenteric ischemia. Approximately half of the cases of AMI are due to arterial embolism [4,5]. Thrombosis of the SMA accounts for about 25% of the cases [6]. Non-occlusive mesenteric ischemia contributes another 20% of cases [7]. Mesenteric venous thrombosis accounts for most of the remaining 5-10% of cases [8]. Aortic dissection involving the thoracic or abdominal aorta may also lead to acute visceral, renal or limb ischemia. That topic has been extensively discussed in the aortic dissection literature [9-13]. Chronic mesenteric ischemia is a distinct pathology with different diagnostic and treatment strategies that will not be detailed here.

Arterial Embolism

The SMA is especially vulnerable to arterial embolism due to its large caliber and shallow take-off angle from the aorta. Emboli tend to be large enough to lodge in the proximal portion of the SMA; with 50% coming to rest several centimeters beyond the origin where the vessel starts to taper. The proximal jejunal and middle colic branches are typically spared, preserving flow to these territories [14]. In around 15% of cases the emboli may lodge in the more proximal SMA. The most common source of emboli is the heart, although the some emboli may originate from the thoracic aorta. Atheroemboli from the aorta tend to be smaller and lodge more distally in the SMA branches, leading to more diffuse ischemia of the bowel [15]. Autopsy studies have confirmed such patterns of emboli [16]. Conditions such as arrhythmia, recent myocardial infarction, severe left ventricular dysfunction and endocarditis may predispose to heart-related emboli [15]. Half of patients with emboli to the SMA have atrial fibrillation and roughly one-third of patients will have suffered a previous embolic event [17]. Synchronous solid organ infarction has been seen in 15-35% of patients with SMA emboli, demonstrating that the SMA is not the sole target of embolism [7,18,19].

Acute ischemia of the SMA typically presents with severe abdominal pain and unimpressive abdominal exam findings-the 'pain out of proportion to exam' that surgical textbooks often describe [20]. As ischemia progresses the patient may develop peritonitis and all the accompanying exam findings. Patients presenting late in the disease process may present in extremis with septic shock [8].

Rapid diagnosis of AMI is critical. Mortality rates rise steadily with time from onset. Mortality rates are 0-10% with immediate treatment, 50-60% for delays of 6-12 hours, and 80-100% with treatment delays of 24 hours [21]. Computed Tomography Angiography (CTA) has become the diagnostic modality of choice for suspected mesenteric ischemia [22-25]. CTA allows for complete visualization of the abdominal vasculature and can be tailored to allow bowel wall enhancement or visualize solid organ infarct [26]. In addition to the arterial phase images typically acquired in CTA, the addition of pre-contrast and

delayed venous phases allow characterization of arterial thrombus, calcifications and bowel wall enhancement. Areas of hypo-perfusion of the bowel can be identified during the venous phase. The addition of a negative oral contrast agent (water) helps to distend the bowel to increase the accuracy of assessing bowel wall viability [27]. However not all patients will be able to tolerate anything by mouth and this step adds time to the CT procedure. CT is also useful in the diagnosis on non-vascular abdominal pain or other vascular pathologies unrelated to intestinal ischemia.

Patients with AMI often have laboratory abnormalities as well. Leukocytosis is common but non-specific and there may be a left-shift toward immature leukocytes [4]. Acidosis is also common and can be triggered by dehydration, malnutrition or elevated lactate. Electrolyte abnormalities are common and should be corrected if possible before intervention. Specific biomarkers for intestinal ischemia have not yet been identified [28].

Duplex ultrasound has long been used to evaluate blood flow, including in the viscera. In the setting of acute ischemia, ultrasound can demonstrate segmental SMA occlusion and diminished flow distally [29]. However, the time required for the exams, the dependence on technician skill and the inability for patients with abdominal pain to tolerate the procedure make ultrasound less useful in AMI [4]. Ultrasound remains a useful procedure in vessel surveillance after intervention.

The diagnosis of AMI can be made definitively with CTA imaging and clinical findings. Then the first major dilemma is differentiating between an embolic source, mesenteric arterial thrombosis or non-occlusive mesenteric ischemia (discussed below). The clinical scenario can generally differentiate between emboli and other causes of acute ischemia [30]. Factors predisposing to emboli and prior episodes of embolization are the most common clues to an acute embolism. A low probability of emboli and strong evidence of other etiologies should guide the diagnosis accordingly.

Once the diagnosis is made, fluid resuscitation should begin and broad-spectrum antibiotics should be administered as there is a significant risk of bacterial translocation across the intestinal membrane. The goal of fluid resuscitation is to augment perfusion. Significant fluid sequestration is common [31,32]. Vasopressors may be needed to maintain blood pressure, but improved pressure must be balanced against the mesenteric vasoconstriction caused by vasoactive agents such as norepinephrine, epinephrine and vasopressin. Dobutamine, milrinone and dopamine may have a less detrimental effect on the splanchnic circulation [2,33,34]. The bowel mucosa is the first component affected by ischemia and its barrier function may suffer, hastening bacterial translocation. This reinforces the use of broad spectrum antibiotics in patients with mesenteric ischemia [35]. The patients should be systemically anticoagulated by bolus, typically with unfractionated heparin, to reduce the risk of thrombus propagation and further emboli.

When arterial embolization is diagnosed and resuscitation has begun the next step is deciding if the patient already has ischemic bowel. Although endovascular interventions are available and widely accepted for the treatment of arterial embolism, there is nearly universal agreement that patients presenting with frank ischemia of the bowel should undergo immediate laparotomy [36,37]. Patients with peritoneal free air, hemodynamic instability or peritonitis should

undergo prompt exploration. Patients, who have developed considerable pain on palpitation rather than pain out of proportion to exam are already demonstrating compromise of the bowel and should be explored. Other findings pointing toward bowel ischemia are leukocytosis, metabolic or lactic acidosis and such CT findings as bowel thickening, lack of bowel wall enhancement, mesenteric stranding, free fluid, pneumatosis intestinalis or portal venous gas [18]. Patients with one or more of these signs should proceed to surgery. There is a 3-part strategy for surgery in these cases: re-establishing blood flow, resection of non-viable bowel while preserving viable bowel and re-examining the bowel at a second look operation [8].

Options for revascularization include the following:

- Open embolectomy of the SMA, which is typically performed through the small bowel mesentery
- Bypass to a distal, patent portion of the SMA from either the aorta or iliac artery
- A hybrid approach utilizing retrograde endovascular techniques through the distal SMA, which can be used to performed angioplasty or stenting of ostial or proximal SMA stenosis [2,4,38]

Once blood flow is restored, the bowel is examined after 10-15 minutes and all grossly ischemic areas are resected. The bowel is usually left in discontinuity since a second look operation in 24-48 hours is almost always warranted. Attempting an anastomosis in marginally viable bowel may actually worsen outcomes [39]. A second-look operation allows time for marginal bowel to either declare itself ischemic or recover after reperfusion. Any bowel that is not grossly ischemic should be left *in situ* and examined at the second-look operation. The goal is to preserve bowel length. A temporary closure is often placed on the abdomen in expectation of the second-look operation. Patients with SMA emboli tend to require care in the intensive care unit for hemodynamic monitoring and ongoing fluid resuscitation. Systemic anticoagulation should be achieved with heparin to a goal Partial Thromboplastin Time (PTT) of 60-90 seconds.

Endovascular techniques are also available for treating acute emboli to the SMA. If there is no evidence of frank bowel ischemia and the patient is hemodynamically stable, it is reasonable to attempt an endovascular approach. Femoral, brachial and radial approaches are all viable options. The downward angle of the SMA makes upper extremity access especially appealing. Endovascular techniques include mechanical thrombectomy with devices such as the angiojet (Boston Scientific, Massachusetts) or suction thrombectomy through a large catheter or sheath. Recombinant Tissue Plasminogen Activator (rTPA) may be administered to the embolus using an infusion catheter. The clot may be macerated using balloons or wires, but this technique risks embolization into the more distal branch vessels. Stents (bare metal or covered) may be placed over the embolus to exclude it and restore a patent lumen. Care should be taken that the stent does not impede flow into significant visceral branches. For an acute embolus, the goal of endovascular intervention should be restoration of normal SMA perfusion during the index procedure. Prolonged treatment, such as an infusion of rTPA or papaverine overnight, is not advised as there is a significant risk of progressing to frank bowel ischemia during the treatment.

During and after endovascular intervention the patient should be closely monitored for any signs of deterioration. Signs such as increased abdominal pain, worsening tachycardia or hypotension or the development of acidosis should prompt conversion to surgical exploration. Significant harm occurs when definitive treatment of bowel ischemia is delayed. Frank bowel ischemia must always be considered, especially while endovascular intervention is in-process because the bowel cannot be directly inspected [21]. If bowel ischemia is suspected, the bowel can be evaluated by laparoscopy, but this must be done meticulously since patchy ischemia may be easily missed and accurately assessing the color of the bowel can be difficult by laparoscopy. If any doubt remains regarding the bowel viability, laparotomy should be performed.

Because emboli to the SMA are often well-organized, firm and rubbery they are less amenable to dissolution by rTPA. At our institution we have elected to proceed directly with laparotomy and open revascularization when embolism is the suspected etiology, such as in patients with atrial fibrillation and no evidence of chronic SMA stenosis. Our experience with endovascular intervention for SMA emboli has been unsatisfying and we have seen delays in definitive treatment, increased lengths of stay and inferior outcomes by first attempting endovascular intervention.

SMA Thrombosis

Thrombosis of the SMA may occur in low-flow states and there is almost always pre-existing evidence of stenosis or calcification in the SMA [2]. A careful history may reveal chronic post-prandial pain, unintentional weight loss or other signs of peripheral vascular disease. Patients may have had prior intervention, such as angioplasty or stenting. Patients with SMA thrombosis are more likely to have had time to develop collaterals that prevent the severe acute ischemia in SMA embolism [6].

CTA is still the imaging modality of choice when SMA thrombus is suspected [18]. A three-phase scan can reveal the area of thrombus, the presence of collateral flow and evidence of any frank bowel ischemia. Just as with SMA embolism any signs pointing toward frank bowel ischemia should prompt surgical exploration. Those without evidence of bowel ischemia are good candidates for endovascular intervention.

The same treatment options are available for SMA thrombosis as with embolism. The thrombus that forms within a stenotic SMA tends to be softer and less-organized than in SMA embolism. Thus it is amenable to treatment by rTPA infusion [40]. Because these patients are often better collateralized than in cases of SMA embolism, a prolonged rTPA infusion, either for several hours or overnight, is often feasible without risking progression to frank bowel ischemia. Of course the patient should be closely monitored for any signs of bowel ischemia and if they develop the patient should proceed to laparotomy [41].

Once the thrombus has been lysed or extracted (such as with the Angiojet device), the underlying stenosis can be evaluated and treated [42]. Stenosis tends to be present in the proximal portion of the vessel. For densely calcified lesions and those near the ostium, a balloon expandable stent should be considered [43]. As with treating SMA emboli, care should be taken not to cover any significant arterial branches, although exclusion of a single branch at its origin rarely causes complication.

Patient suffering SMA thrombosis should be treated initially with systemic anticoagulation but rarely require it long-term. Almost all patients with SMA thrombosis and underlying vascular disease should receive low-dose aspirin therapy life-long [44]. If a stent is placed, dual anti-platelet therapy (clopidogrel) for 30-90 days may be considered, but the evidence for improved stent patency with such therapy is scant [45].

Mesenteric Venous Thrombosis

Thrombosis of one of the mesenteric veins or the portal vein can occasionally lead to bowel ischemia [4]. More common outcomes include mild intestinal edema and vague abdominal pain. Venous thrombosis can progress to arterial insufficiency over a period of days-to-weeks leading to transmural bowel ischemia [6]. There is often an underlying serious abdominal pathology.

Unless there is significant contraindication all patients with mesenteric venous thrombosis should receive systemic anticoagulation and then transition to long-term anticoagulation. There is little data to support any specific anticoagulation [46]. Some authors recommend low-molecular weight heparin for mild cases of mesenteric venous thrombosis and unfractionated heparin for more severe cases such as when bowel edema is present [38].

Generally anticoagulation is sufficient treatment, however on occasion additional intervention may be necessary. Such situations include when bowel edema progresses, leukocytosis develops or abdominal pain increases. Options include tTPA infusion through the SMA or retrograde in a transhepatic fashion to access the thrombus directly [47]. Another option is transhepatic mechanical thrombectomy with devices such as the Angiojet [48].

Patients presenting with evidence of frank bowel ischemia should proceed with surgical exploration. Open thrombectomy of the portal or mesenteric veins can be performed through laparotomy although this approach is rarely indicated. The bowel typically becomes ischemic secondary to venous obstruction so arterial intervention is rarely indicated. After addressing the mesenteric thrombus, resection of any ischemic bowel should be performed. A second-look operation may be useful if the bowel is of questionable viability.

Non-Occlusive Mesenteric Ischemia

Non-Occlusive Mesenteric Ischemia (NOMI) occurs during systemic circulatory failure when blood flow is redistributed to vital organs. Vasoconstriction of the mesenteric arteries leads to ischemia [6]. There is typically a severe underlying illness such as heart failure, sepsis or hypovolemia. Treatment should be aimed at the underlying cause and in restoring hemodynamic stability. The administration of vasoconstrictive agents may help systemic blood pressure but worsen mesenteric ischemia [4,33]. When intervention is necessary, the most common treatment is an intra-arterial infusion of papaverine through the superior mesenteric artery [49]. Evidence of frank bowel ischemia should be treated with surgical exploration and resection. NOMI remains a lethal condition, with mortality rates of 50-83%, often secondary to the underlying illness [50].

Conclusion

Acute mesenteric ischemia is a life-threatening condition that requires prompt diagnosis and treatment. There is a role for endovascular intervention in selective patients, especially those with SMA

thrombosis. Open surgical intervention is still often necessary and should be strongly considered as first line therapy for SMA embolism.

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