Acute Gut Ischemia

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KEYWORDS

• Acute mesenteric ischemia • Mesenteric arterial occlusion • Mesenteric thrombosis
• Mesenteric embolectomy • Mesenteric bypass • Catheter-directed thrombolysis
• Retrograde open mesenteric stenting

KEY POINTS

- Acute mesenteric ischemia is a surgical emergency with high mortalities. Common causes include emboli from a cardiac origin and thrombosis of a mesenteric plaque.
- Prompt diagnosis, fluid resuscitation, systemic anticoagulation, and mesenteric revascularization are critical for successful outcomes.
- Revascularization options include open embolectomy or mesenteric bypass, catheter-directed therapies or a hybrid approach of the 2, retrograde open mesenteric stenting.
- Despite technological advances, mortalities have not improved over the past decades, and, although endovascular therapies have improved outcomes, a selection bias exists. Revascularization modality should be chosen on a case-by-case basis.

INTRODUCTION

Acute mesenteric ischemia (AMI) remains a dreaded surgical emergency that continues to be fraught with elevated morbidity and mortality rates. Despite advances in imaging modalities and laboratory techniques, the diagnosis and management of AMI are difficult secondary to nonspecific symptoms at presentation and coexisting comorbidities. Although the advent of endovascular techniques has allowed for minimally invasive therapies, patient outcomes have only seen subtle improvements over the past few decades. This article aims to review the cause, clinical presentation and diagnosis, treatments, and outcomes for patients who present with AMI. The emphasis is aimed at AMI resulting from mesenteric arterial occlusion as a result of embolus and thrombosis, because these conditions most commonly require emergent surgical intervention and revascularization compared with mesenteric venous thrombosis or nonocclusive mesenteric ischemia.

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CAUSE

Insufficient perfusion that fails to meet the metabolic demands of the bowel results in the underlying pathophysiology in AMI.[1,2] Arterial obstruction is the underlying cause of AMI and can be the result of embolization from a more proximal source or thrombosis of a preexisting lesion. The incidence of embolization versus thrombosis varies depending on the series and institution; however, thrombosis is often found to be the most common cause, accounting for 50% to 70% of cases.[3–6]

Embolization

Emboli originate from a cardiac source in upwards of 90% of cases, with atrial fibrillation and ventricular thrombus following myocardial infarction being common pathologic conditions. Less likely origins include thoracic and abdominal aortic atheromas or aneurysms.[1,7–10] Emboli have a predilection to enter and obstruct the superior mesenteric artery (SMA) due to its size and the angle of origin as it comes off the aorta.[1,11] Furthermore, these emboli frequently become lodged distal to the jejunal branches and middle colic artery, subsequently sparing the proximal bowel and causing distal small bowel and colonic ischemia.[11] The remaining mesenteric arteries are unlikely to be affected by emboli, and involvement of the hypogastric arteries is unlikely to cause clinically significant ischemia given the robust pelvic collateral pathways.[11]

Thrombosis

Chronic proximal atherosclerotic plaques are the common underlying cause of SMA thrombosis, whereas other pathologic conditions, such as vasculitis, aneurysms, and dissections, are rare sources.[1,11] Patients incurring SMA thrombosis often present with a spectrum of symptoms and varying degrees of ischemia dependent on preexisting collateral pathways that have developed over time. This population will often have a history of prior chronic mesenteric ischemia symptoms with the triad of postprandial pain, food fear, and weight loss.[1] With the onset of acute on chronic ischemia, they not only may express symptoms of vague postprandial pain consistent with chronic mesenteric ischemia but also can exhibit sudden, intense pain equal to that of embolic phenomenon.[12] Furthermore, the distribution of intestinal ischemia will vary contingent on the collaterals that have been formed before complete thrombosis. Another distinction between embolus and thrombosis is the anatomic location, wherein thrombosis typically occurs flush with the SMA origin arising off the aorta.[1,11]

CLINICAL PRESENTATION AND DIAGNOSIS

Clinical Presentation

Patients presenting with AMI are usually in their 60s or 70s.[4,6,8,10,13] Women are up to 3 times more prone to suffer AMI compared with men.[14] Almost all patients present with abdominal pain as their chief complaint.[10,12] As mentioned previously, the characterization of the pain may vary depending on the cause of the arterial obstruction. In most cases, symptoms have been present for greater than 24 hours at the time of presentation.[5,10,15] A high index of suspicion is imperative when making the diagnosis, because AMI can be misdiagnosed as pancreatitis, hepatobiliary disease, diverticulitis, appendicitis, or bowel obstruction.[14] The classic description of AMI is severe, constant pain that is out of proportion to physical examination, and patients may experience emesis with the onset of pain.[12] The delay of physical examination findings such as rebound and guarding results from the progression of ischemia from the mucosal layer to the seromuscular layers, and then eventually full-thickness necrosis.