REVIEW ARTICLE

Acute mesenteric ischaemia : Part I

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Introduction

Acute mesenteric ischaemia (AMI) is a life-threatening emergency. Although uncommon, it remains a highly complex clinical problem with a mortality rate of 30 – 65 percent [1,2]. In under treated patients, the complex and progressive pathophysiology of AMI leads to the inevitable clinical outcome of extensive bowel necrosis, multi-organ failure and death. In spite of numerous advances in imaging modalities and treatment, there have been only modest improvements in mortality rates. Zettervall et al reported a decline in the in-hospital mortality rates (37-21%) of patients undergoing revascularization for AMI during the period 2000-2012 [3]. It has been suggested that while the major advances in imaging technology have resulted in earlier diagnosis, this has been counterbalanced by the contemporary AMI patient presenting at an advanced age with more severe underlying comorbidities [4]. The purpose of this article is to review the current approach to diagnosis and treatment of acute mesenteric ischaemia

Mesenteric vascular anatomy, etiology and pathophysiology of AMI

Vascular Anatomy

The celiac axis with its hepatic, splenic and left gastric arteries perfuse the foregut structures of the esophagus, stomach, and duodenum. The superior mesenteric artery (SMA) perfuses the structures of the mid-gut including the jejunum, ileum, and ascending colon and the first portion of the transverse colon. The inferior mesenteric artery (IMA) perfuses the hindgut structures of the distal transverse colon and descending and sigmoid colon. Gastroduodenal and pancreaticoduodenal arteries provide collateral pathways between the celiac axis and the SMA. The meandering mesenteric artery, sometimes referred to as the arc of Riolan, describes a collateral that forms between the SMA and IMA in the presence of occlusive disease. The marginal artery of Drummond lies along the mesenteric border of the bowel and also provides collateral flow between the SMA and IMA territory. The internal iliac arteries provide a collateral pathway to the IMA via the hemorrhoidal arteries in the setting of IMA occlusive disease [5] (Figure 1).

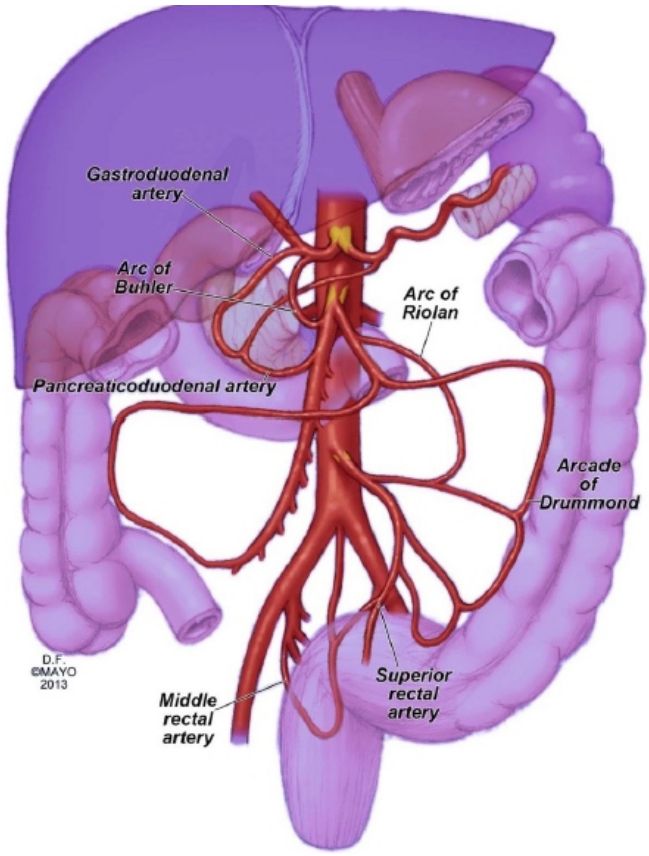

Figure 1. Mesenteric artery circulation and common collateral pathways in patients with occlusive mesenteric artery disease. Common collateral pathways include the pancreaticoduodenal artery between the celiac axis and SMA, and the arc of Riolan between the left colic artey (IMA) and middle colic artery (SMA).

(Courtesy: Rutherford's vascular surgery. 8th ed. Philadelphia: Elsievier Saunders, 2014; with permission)

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Etiology

Historically, superior mesenteric artery embolism has been the most common etiology of AMI, accounting for 40 – 50 % of cases [6] (Table 1). Most emboli are from a cardiac source secondary to atrial fibrillation, low-ejection fraction (congestive heart failure, cardiomyopathy), recent myocardial infarction with mural thrombus, or ventricular aneurysm. The overall incidence of thromboembolism may be declining secondary to improved treatment of atrial fibrillation with anticoagulation. Other embolic sources include arterial to arterial emboli (proximal aortic lesion), valvular heart disease, endocarditis, and complications from recent catheter-based angiography. The superior mesenteric artery (SMA) is the most commonly affected mesenteric vessel because of the oblique origin from the visceral aorta [6]. Emboli commonly lodge distal to the middle colic artery, just beyond the first few jejunal branches, creating a classic pattern of ischaemia that spares the first portion of the small intestine and the transverse colon [7]. Atheroemboli tend to lodge in the more distal mesenteric circulation due to their smaller size.

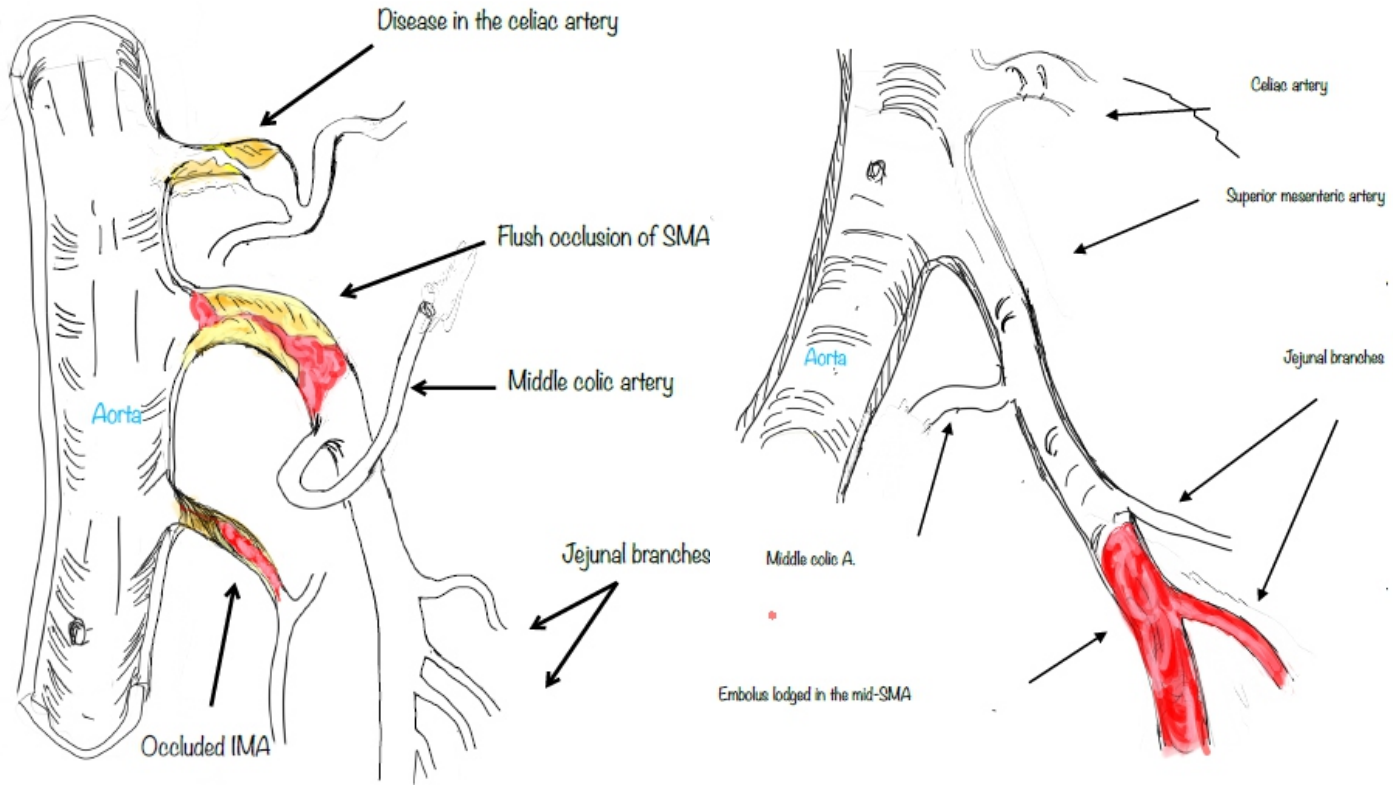
Acute arterial thrombosis, superimposed on pre-existing atherosclerotic disease, typically accounts for 25-30 % of AMI but recent data suggest that this may be the most common cause [1,8]. These patients typically have a history of weight loss and postprandial abdominal pain due to their progressive chronic occlusive disease. Occasionally, the patients may be asymptomatic prior to the development of AMI but present secondary to the acute thrombosis of an important collateral vessel. The onset of symptoms may therefore be insidious due to extensive collaterals maintaining bowel viability until the index event causes thrombotic occlusion of the vessel. The predominant arterial occlusive lesion responsible for AMI is nearly always at the SMA, in addition to accompanying occlusive disease of the celiac axis and IMA. The multi-vessel involvement explains the fact that in the case AMI due to SMA thrombosis, it is often the case that the entire GI tract is involved, in contrast to the sparing of the proximal jejunum and ascending colon seen in the setting of an embolic event (Figures 2A/2B).

Nonocclusive mesenteric ischemia (NOMI) most frequently occurs in the setting of severe systemic illness such as cardiogenic shock and multi-system organ failure and the bowel ischaemia occurs due to mesenteric arterial vasospasm and severe hypoperfusion. It is often under recognized in patients with reduced left-ventricular ejection fraction (LVEF), especially in those who require significant pressor support

Table 1. Etiology of acute mesenteric ischaemia

Causes of AMI		**Incidence**
Arterial embolism	Atrial fibrillation	40% - 50%
	Acute myocardial infarction	
	Ventricular aneurysm	
	Atheroemboli	
	Valvular heart disease/endocarditis	
Arterial thrombosis	Atherosclerotic mesenteric disease	25% - 30%
	Acute sequelae of aortic dissection (malperfusion syndrome)	
	Occlusion of bypass grafts/complications of angioplasty and stenting	
	Spontaneous mesenteric dissection	
Non-occlusive mesenteric ischemia (NOMI)	Multi-system organ failure	20%
	Shock	
Mesenteric venous thrombosis*	Intra-abdominal inflammatory states: pancreatitis, inflammatory bowel disease	10%
	Hypercoagulability	
	Local venous congestion (portal hypertension, congestive heat fail)	
	Direct injury (trauma or post-surgical)	

**mesenteric venous thrombosis will be discussed in detail in a separate article*



Figures 2 A-B. Location of proximal atherosclerotic thrombosis and embolic occlusion in the SMA.and middle colic artery (SMA).

with vasoactive medications such as epinephrine and norepinephrine [9]. It is claimed that NOMI may occur as often as 2.5-5% in a subset of patients with low ejection fraction and pressor support.

Mesenteric venous thrombosis (MVT) can lead to visceral ischaemia secondary to compromise in venous outflow leading to severe bowel congestion compromising arterial inflow. Various etiologies of AMI are compared and contrasted in Table 1.

Pathophysiology

The bowel can tolerate a remarkable degree of ischaemia without permanent cellular damage. Only one fifth of the mesenteric capillaries are open at any given time, and normal oxygen consumption can be maintained with only 20% of maximal blood flow [6, 10]. Below "critical blood flow" of 30mL/min/100g, oxygen delivery is limited leading to tissue injury [9]. Mucosal surfaces are affected first because the metabolic demand in the mucosa is much higher than that of the serosa. Clinically, this is manifested with mucosal infarction leading to bloody diarrhea and malabsorption.

Hagland described four clinical stages in the natural history of AMI [11]. The first stage of AMI, the hyperactive stage, is characterized by reflex intestinal contractions caused by ischaemia resulting in intermittent severe pain, loose stools (sometimes bloody) and vomiting. In the second paralytic stage, pain is more constant and diffuse, and the abdomen is distended and tender. Third stage of disarranged fluid balance occurs when there is leak of protein and electrolyte rich fluid from both the serosal and mucosal side of the gut. As the bowel becomes necrotic, peritonitis develops. In the final (fourth stage) of shock, the patient has frank peritonitis and massive volume loss.

Clinical presentation

The presence of bowel ischaemia causes the onset of severe pain which predates the infarction of the bowel, leading to the classic finding of pain out of proportion to physical examination findings. Until there is transmural necrosis and peritonitis, there is little peritoneal irritation and therefore minimal tenderness to palpation. However, depending on the timing and actual cause of AMI, this classic presentation may be absent in 20 – 25 % cases [6]. In patients with acute SMA embolism, the onset of symptoms is characteristically abrupt. Patients with SMA thrombosis may present with a more insidious onset of symptoms since many of these patients have well developed collaterals, which are more gradually compromised.

Often, the diagnosis is difficult to make on clinical grounds since the symptom complex of abdominal pain, distension, diarrhea, acidosis, sepsis, and gastro-intestinal bleeding may easily be mistaken for other common intra-abdominal pathology. NOMI patients may have an even more subtle

onset and a protracted course in a clinical picture dominated by their cardiac or septic shock. Due to their already critical status, no history is usually available.

Patients with mesenteric venous thrombosis typically have an insidious onset of pain, dissension, and hypovolemia in a setting of predisposing pathology such as portal hypertension or a hypercoagulable state. In general, clinical diagnosis of AMI needs a high-index of clinical suspicion and thorough analysis of all clinical data.

Table 2. Diagnostic criteria for bowel ischaemia: typical imaging findings associated with arterial and venous causes of ischaemia

Arterial embolus	**Arterial thrombosis**	**Non-occlusive mesenteric ischemia**	**Venous thrombosis**
Filling defect with a convex shape distal to the SMA origin.	Occlusive lesion flush with the ostia of the visceral vessels	Vasospasm throughout the mesenteric arterial circulation	Venous filling defect in delayed venous phase
No collaterals seen	Collaterals seen	Segmental or diffuse narrowing of the circulation. "Pruning" of jejunal and ileal branches	Diffuse arterial vasospasm
Bowel wall may be paper thin	Bowel wall may be paper thin	No change in bowel wall thickness	Marked bowel wall thickening
No mucosal enhancement	No mucosal enhancement	Asymmetric bowel perfusion	Increased mucosal enhancement
Bowel wall dilates only with infarction	Bowel wall dilates only with infarction	Bowel wall dilatation not apparent	Bowel wall may dilate without infarction
Little mesenteric stranding, hemorrhage or edema	Little mesenteric stranding, hemorrhage or edema	Not hazy until infarction occurs	Usually significant mesenteric inflammation with fluid and hemorrhage

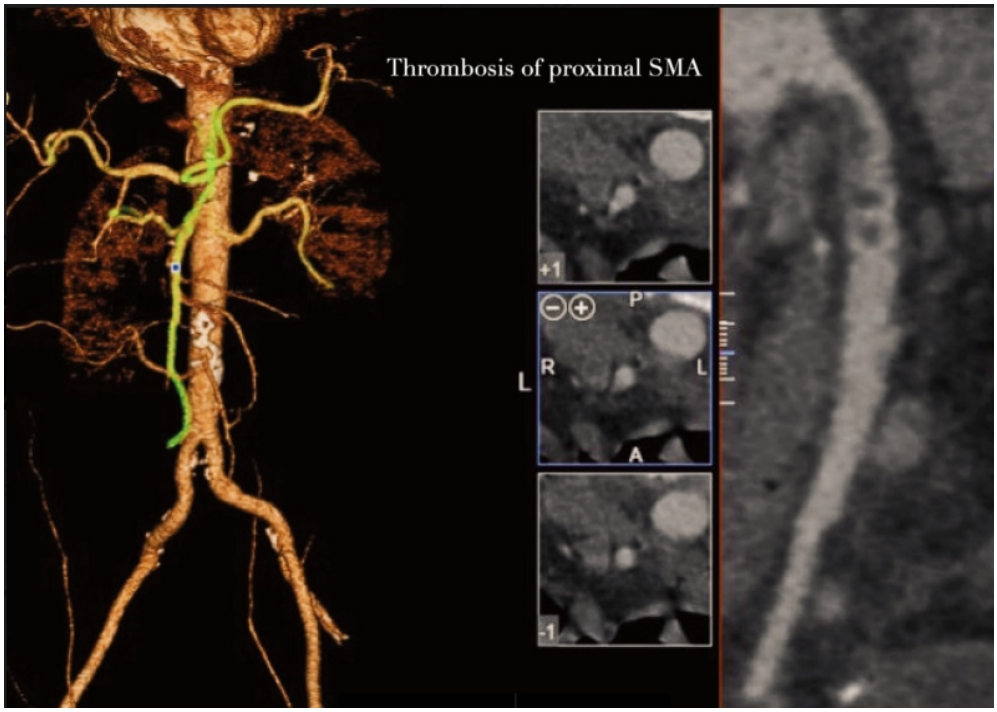


Figure 3. CT findings of thrombosis of SMA near the origin

Diagnostic studies

Leukocytosis and electrolyte abnormalities including high-anion gap are common findings. Lactic acidosis is seen in more advanced cases. High amylase, aspartate aminotransferase and lactate dehydrogenase can also be observed. However, all these serum markers are insensitive and non-specific. In a recent institutional review, most frequent laboratory findings included a leukocytosis (mean 17.9 +/- 7 x 10^9/L) and an elevated lactate (mean 3.4+/- 2 mmol/L) [12].

Plain abdominal radiographs are normal in up to 25% of patients with AMI. Ileus may be an early finding while more advanced cases may show evidence of bowel wall edema ("thumb printing") or pneumatosis.

Although duplex ultrasonography may be highly accurate in diagnosis of celiac and SMA stenosis in an elective setting, its role in AMI is limited because of several important reasons: it is highly operator dependent, accessibility may be poor during off-hours, and presence intestinal gas and abdominal tenderness can compromise the ability to image the relevant anatomy.

CT angiography is widely available and frequently able to provide the key diagnostic information. Using standardized protocols that include accurate timing of contrast and fine slices through the abdomen, CTA provides excellent imaging of the celiac artery, SMA and IMA arterial anatomy. CT imaging can also provide details such as pneumatosis, bowel wall edema and other findings such as solid-organ hypoperfusion. A compilation of CT angiographic features of acute mesenteric ischaemia are presented in Table 2 [13,14,15].

Henes et al, in a retrospective study of 959 patients, found that CT angiography had a sensitivity and specificity of 89.4% and 99.5% respectively for diagnosing AMI [16]. The presence of pneumatosis and portal venous gas is virtually diagnostic for transmural bowel infarction, with specificities approaching 100% for presence of ischaemia [12]. Numerous other studies have also corroborated accuracy of CTA in diagnosis of AMI.

Traditional catheter-based digital subtraction angiography (DSA), has been supplanted by CTA as the definitive study for occlusive forms of AMI because of its availability and non-invasive nature. However, DSA retains an important role in some situations such as in patients with suspected NOMI when catheter angiography may help establish the diagnosis and also allow for endovascular management of atherosclerotic disease of the proximal visceral vessels (Figure 4A-B).

Magnetic resonance angiography (MRA) has not found prominence in establishing the diagnosis of AMI. Its limited availability on emergency basis and slow image acquisition time are all compromises compared to CTA. In addition, secondary signs of AMI such as bowel wall thickening are difficult to assess with MRA.

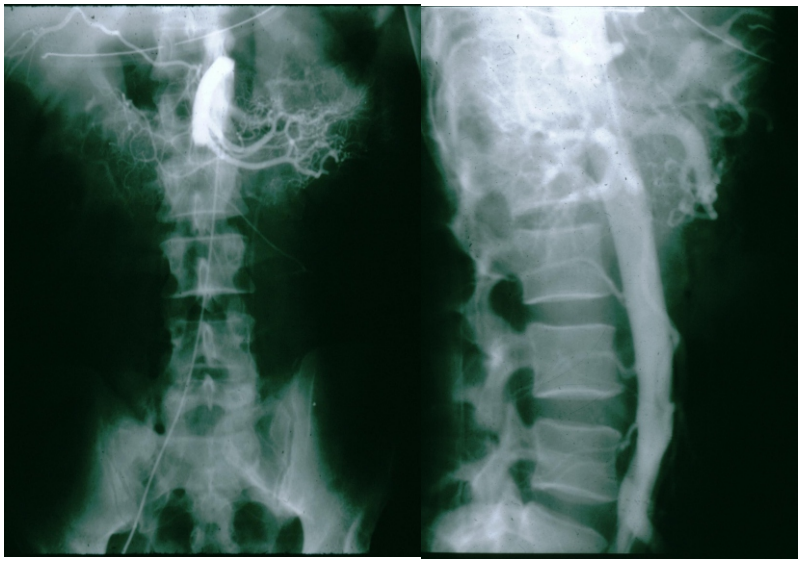


Figure 4 A-B. SMA embolus seen on selective mesenteric arteriogram (AP and lateral projections)

Diagnostic laparoscopy has a limited ability to assess bowel viability in the setting of AMI. Although bowel ischaemia may be inspected visually, there is no ability to manually palpate pulses in the mesentery. Furthermore, the use of intraperi-toneal CO2 insufflation is contraindicated in hypotensive, unstable patients precluding its use in most patients with AMI.

All authors disclose no conflict of interest. The study was conducted in accordance with the ethical standards of the relevant institutional or national ethics committee and the Helsinki Declaration of 1975, as revised in 2000.

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