

Imaging findings in diagnosis of mesenteric ischemia: how can we be sure?

Practice points

- Modern medicine relies on early, accurate diagnosis of the disease and prompt optimal treatment to reduce morbidity and mortality. Given the nonspecific clinical and laboratory findings, mesenteric ischemia (MI) continues to remain a diagnostic dilemma and challenge for the clinician.
- A high degree of clinical suspicion with imaging correlation remains the mainstay of prompt diagnosis and better clinical outcome.
- Computed tomography (CT) angiography (CTA) has replaced conventional catheter angiography in the evaluation and diagnosis of suspected MI, with higher sensitivity and specificity.
- Multidetector CT remains the modality of choice in acute MI due to faster scan time, higher resolution and comprehensive noninvasive modality to evaluate vascular and enteric system.
- With chronic MI, both CT and magnetic resonance angiography (MRA) helps in evaluating the major mesenteric vasculature and also in excluding other causes of abdominal pain, thereby directing the clinician to appropriate medical, surgical or vascular interventional therapy.
- While CT angiography and MRA can both be utilized in evaluation of patients with chronic MI, MRA provides an advantage of evaluating mesenteric vasculature without injecting intravenous contrast, beneficial in patients with contrast allergy or poor renal function.

Ashish P Wasnik¹,
Peter S Liu¹ & Joel F Platt^{*1}

¹University of Michigan Health System,
Department of Radiology 1500 E.
Medical Center Drive, Ann Arbor, MI
48109, USA

*Author for correspondence
jplatt@med.umich.edu

Mesenteric ischemia (MI) remains a complex disease entity characterized by acute or chronic perfusion abnormality to the GI tract. Because it presents with nonspecific symptoms and laboratory findings, MI remains a clinical diagnostic challenge. Given that MI is associated with high morbidity and mortality rates, early diagnosis remains critical for appropriate management and best clinical outcome. Over the past decade, significant technical improvements have been seen in computed tomography and MRI, particularly in their use for noninvasive angiographic imaging. These techniques are now the preferred test for initial evaluation of suspected MI. This article focuses on computed tomography and MRI features of MI in both the acute and chronic clinical settings, with a review of anatomy, etiopathogenesis and clinical features.

Keywords: bowel infarction • bowel ischemia • computed tomography • mesenteric ischemia • MRI

Mesenteric ischemia (MI) remains a complex disorder of the GI tract with high mortality rates ranging from 50 to 90% [1]. Although the exact incidence of MI is difficult to assess, there has been a general increase in incidence

over the past decade, which may be attributed to increased life expectancy/increased population age, improved clinical awareness, and better diagnostic imaging techniques [2]. MI occurs due to perfusion abnormality of

the bowel, depriving the tissue of oxygen and nutrients leading to cellular injury and necrosis. The clinical features remain nonspecific, including abdominal pain, nausea, vomiting, and diarrhea, all of which can overlap with other acute or chronic abdominal conditions. As a result, the diagnosis of MI remains a challenging clinical diagnosis. Imaging plays an important role in primary diagnosis of MI, and also aids in exclusion of other differential diagnostic considerations. While conventional catheter angiography has been the reference standard in evaluating acute MI, recent advances in cross sectional imaging have made these techniques very important for workup of suspected MI. In particular, modern computed tomography (CT) offers high spatial resolution, fast scan times, 3D data sets, and excellent evaluation of nonvascular findings. In fact, CT has demonstrated very high sensitivity and specificity for the diagnosis of MI, and has replaced catheter angiography as the primary imaging modality of choice [3]. This article reviews the mesenteric vascular anatomy, diagnostic technical considerations, etiopathogenesis, clinical presentation, and imaging findings in acute and chronic MI.

Anatomical consideration of mesenteric vasculature

Knowledge of normal and variant vascular anatomy forms the basis in understanding and diagnosing MI. There are three major branches of the abdominal aorta, which supply arterial blood to the small and large bowel. Most cephalad is the celiac artery/axis, arising approximately at the level of T12 vertebral body level, and supplying the foregut from the distal esophagus to the second portion of the duodenum. The superior mesenteric artery (SMA) arises approximately at the level of the L1 vertebral body, and supplies the midgut from the third portion of the duodenum to the distal transverse colon. Shortly after its origin, the SMA gives rise to multiple jejunal and ileal branches, as well branches to the proximal colon via the right and middle colic arteries, before terminating as ileocolic artery in the right iliac fossa. The inferior mesenteric artery (IMA) arises most caudally, approximately at the level of the L3/4 vertebral body, and supplies the hindgut from the transverse colon to the rectum (Figure 1).

There are a few collateral pathways between the three main mesenteric vessels, which can be important for preserved downstream perfusion in the setting of proximal vascular narrowing or occlusion. The primary collateral flow between the celiac and SMA distributions is through the pancreaticoduodenal arcade, which connects the celiac axis via the common hepatic artery and gastroduodenal artery, to branches of the proximal SMA through a vascular plexus surround-

ing the head of pancreas and duodenal C-loop. Two potential routes of collateral flow are present between the SMA and IMA, including the marginal artery of Drummond that courses peripherally along the mesenteric margin/reflection of the colon, and the Arc of Riolan, which is located in the more central portions of the mesentery [4]. In addition, there are connections from the mesenteric circulation to the deep pelvic arteries through a vascular plexus around the rectum. This allows communication of the IMA through the superior hemorrhoidal artery, to the branches of internal iliac arteries including the middle and inferior hemorrhoidal arteries.

Two watershed areas have been described in the colon, which relate to anatomic sites at the margins of different arterial perfusion zones. 'Griffith's point' refers to the splenic flexure of colon, which is located between the middle and left colic arterial perfusion zones. 'Sudeck' point' is an anatomic location in the IMA vascular territory between the last sigmoid colonic branch and the superior hemorrhoidal artery, which can be an important surgical landmark when considering sigmoid resection. Although there is some modern controversy about the continued surgical relevance of Sudeck's point, there are reported cases of postoperative ischemic strictures in patients for whom this anatomic landmark was ignored at surgery [2,5-6].

Venous drainage of the small bowel is through the superior mesenteric vein (SMV), while venous return from the colon is via tributaries to both the inferior and superior mesenteric veins. The inferior mesenteric vein variably drains into either splenic vein, SMV, or directly as a true trifurcation with the SMV and splenic vein. The union of the splenic vein and SMV becomes the extra hepatic portal vein (Figure 1C).

Technical consideration Abdominal radiograph

Even though abdominal radiograph remains the first imaging modality in patients with abdominal pain, the diagnostic yield for acute MI is very low and a normal abdominal radiograph does not exclude the diagnosis [7]. Several abdominal radiograph findings of acute MI are nonspecific and include gaseous distention of the bowel from the ileus and thumb printing from mural edema. Pneumatosis and portal venous gas both have higher specificity for diagnosis of MI, but are seen in the advanced stages of bowel infarction/necrosis. It is important to remember that pneumatosis in isolation does not fully implicate a diagnosis of MI, as it can be seen with variety of nonischemic or benign causes such as scleroderma and steroid use [8,9]. Nevertheless, presence of any of these features warrants further evaluation with cross-sectional imaging, particularly CT.

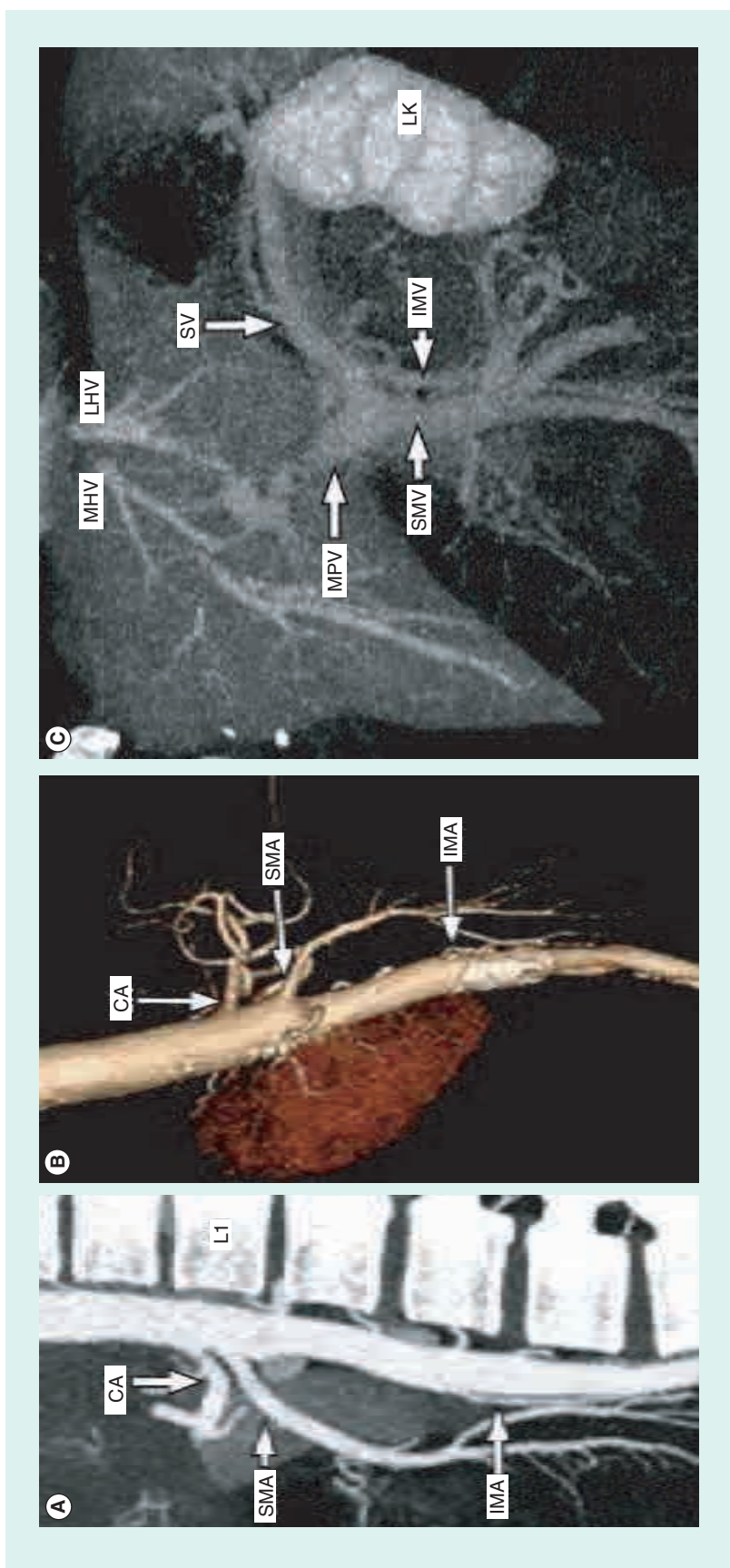


Figure 1. Normal mesenteric anatomy. (A) Coronal reformatted maximum intensity projection arterial phase computed tomography (CT) image. (B) Volume-rendered 3D reformatted CT image. (C) Coronal reformatted maximum intensity projection portal venous phase CT image.
 CA: Celiac artery; IMA: Inferior mesenteric artery; IMV: Inferior mesenteric vein; LHV: Left hepatic vein; MHV: Middle hepatic vein; MPV: Main portal vein; SMA: Superior mesenteric artery; SMV: Superior mesenteric vein.

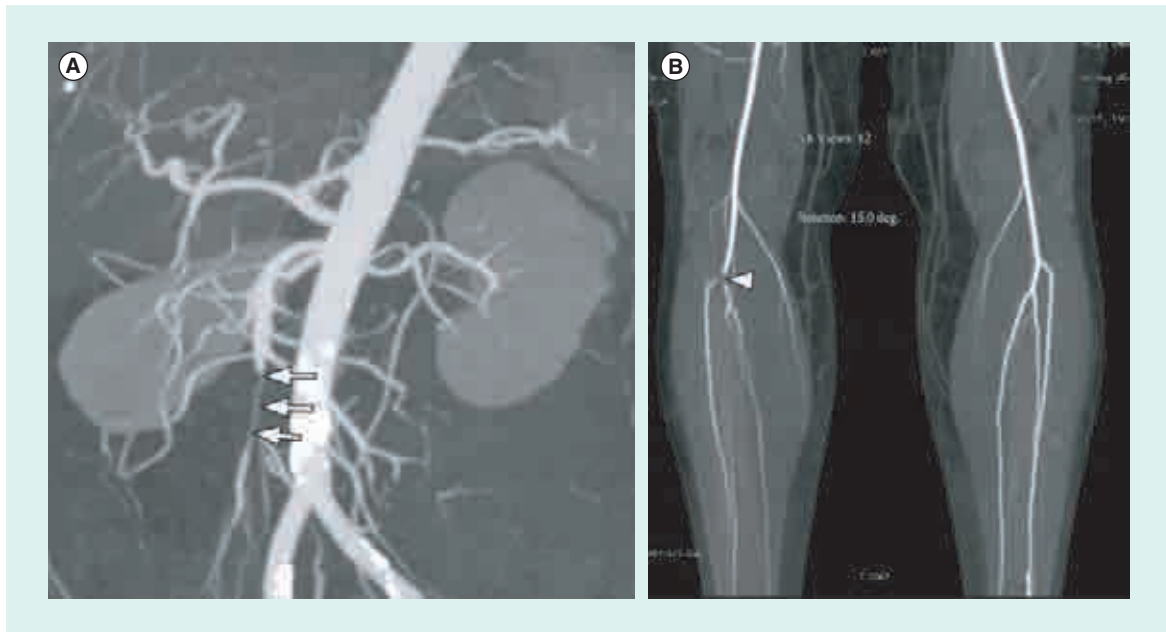


Figure 2. Superior mesenteric artery embolus. (A) Coronal maximum intensity projection reformatted contrast-enhanced arterial phase computed tomography of the abdomen in a 60-year-old man with heart disease and atrial fibrillation shows embolic occlusion of mid-distal superior mesenteric artery (arrows). (B) Coronal reformatted arterial phase computed tomography image through the lower extremity in the same patient, demonstrating a focal filling defect, consistent with embolus, in the right popliteal artery (arrowhead).

Doppler ultrasound

Ultrasound has a very limited role in evaluation of suspected MI, particularly due to poor acoustic transmission through overlying bowel gas and limited penetration in patients with large body habitus [10]. However,

Doppler ultrasound can be utilized for evaluation of large vein occlusion, such as the portal or splenic vein, but is rarely utilized for clinical suspected acute arterial MI [11]. Doppler ultrasound evaluation of the aorta and origins of celiac or SMA can be performed in selected patients with suspected chronic MI, such as those with diminished renal function [12].

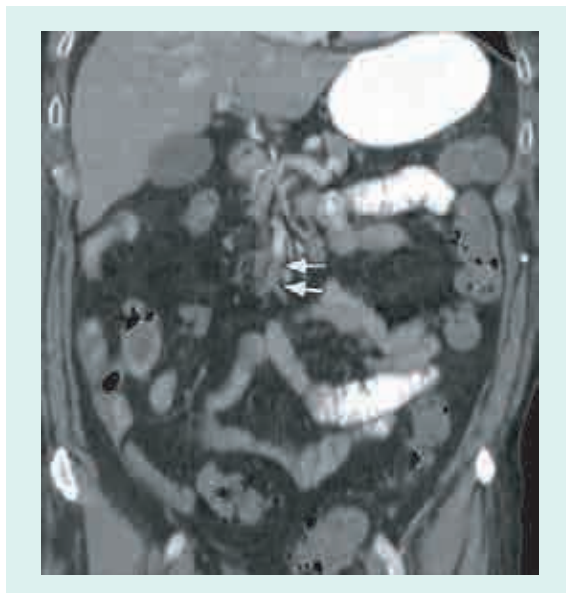


Figure 3. Superior mesenteric artery thrombus. Coronal reformat contrast-enhanced arterial phase computed tomography image in a 55-year-old man shows occlusion of the proximal superior mesenteric artery (arrows).

Multidetector CT

Recent advances in multidetector CT have led to improved accuracy and sensitivity in detecting bowel ischemia. A meta-analysis by Menke demonstrated a pooled sensitivity and specificity of 93 and 96%, respectively [13]. CT angiography is replacing catheter angiography in the acute clinical setting owing to several technical advantages, including faster imaging time, noninvasive technique and 3D volumetric data sets that facilitate multiplanar reformats, as well as the ability to detect pertinent nonvascular pathology [3]. A limitation to CTA remains in suboptimal evaluation of distal/peripheral branches.

In the acute clinical setting with suspected MI, specific protocols using neutral oral contrast media, such as water, milk or 'Low Hounsfield unit' contrast may allow better evaluation of the bowel wall itself, including depiction of mucosal enhancement and submucosal pathologies such as edema or hematoma. However, in many patients with acute abdomen, CT is usually performed following administration of positive oral

and intravenous contrast to evaluate for bowel-related pathologies such as bowel obstruction, perforation or abscess.

In patients who present with suspected MI at our institution, we perform CT angiography using a biphasic mesenteric protocol, with both arterial and venous phase imaging. This protocol utilizes administration of 1350 ml of per oral neutral contrast medium over 1–2 hours prior to CT. This can be tailored in acute and emergent cases and the contrast can be administered via nasogastric tube for more rapid distention or the study performed without oral contrast. The patient receives intravenous administration of 125 ml of non-ionic iodinated contrast material (using high iodine concentration contrast material, 370 mg iodine/ml) at rate of 3–4 ml/s, with image acquisition at 0.625 mm collimation in the arterial (30 s) and portal venous phase (60–70 s). Bolus-tracking or bolus-triggered method is used to optimize the phase of acquisition, with the enhancement threshold set at 150 Hounsfield units. The images are then reformatted on 3D workstation into maximum intensity projection, multiplanar reformatted and volume rendered images for detailed evaluation of the mesenteric vessels and bowel.

MRI

MRI in acute MI is generally not utilized given longer scan time, not suited for acutely ill patients and also from limited access. However, magnetic resonance angiography (MRA) can be effectively utilized in the nonemergent setting, and has demonstrated comparable results versus CTA in evaluation of suspected

chronic MI. A particular advantage of MRA relative to CTA is the ability to directly image vascular patency and flow without using injected contrast material. Noncontrast MRA techniques can be very useful for the evaluation of chronic MI in patients with impaired renal function. Some recent studies have shown the possible role of MRI in acute arterial MI and acute ischemia colitis, although this still remains in preliminary stages and need to be validated with larger studies [14,15].

At our institution, MRA of the mesenteric circulation is accomplished using either a 1.5 or 3.0 Tesla superconducting MRI system with surface receiver coils appropriate for the patient body habitus. The protocol begins with standard three orthogonal plane T2-weighted rapid T2-weighted sequences (such as single-shot fast spin echo), which allows general anatomic depiction of the abdomen and specific visualization of the bowel, including presence of wall thickening. Subsequently, a multiphase coronal 3D T1-weighted spoiled gradient echo sequence is performed prior to contrast administration and during the arterial, venous and delayed phases of vascular enhancement using intravenous gadolinium-based contrast material. In patients with renal insufficiency who cannot receive gadolinium-based contrast material, noncontrast MRA techniques may be substituted, including 3D phase-contrast MRA or 3D time-of-flight MRA. Novel noncontrast MRA sequences have also been developed to achieve high vascular signal and improved spatial resolution, including steady-state free precession techniques and cardiac-gated modi-

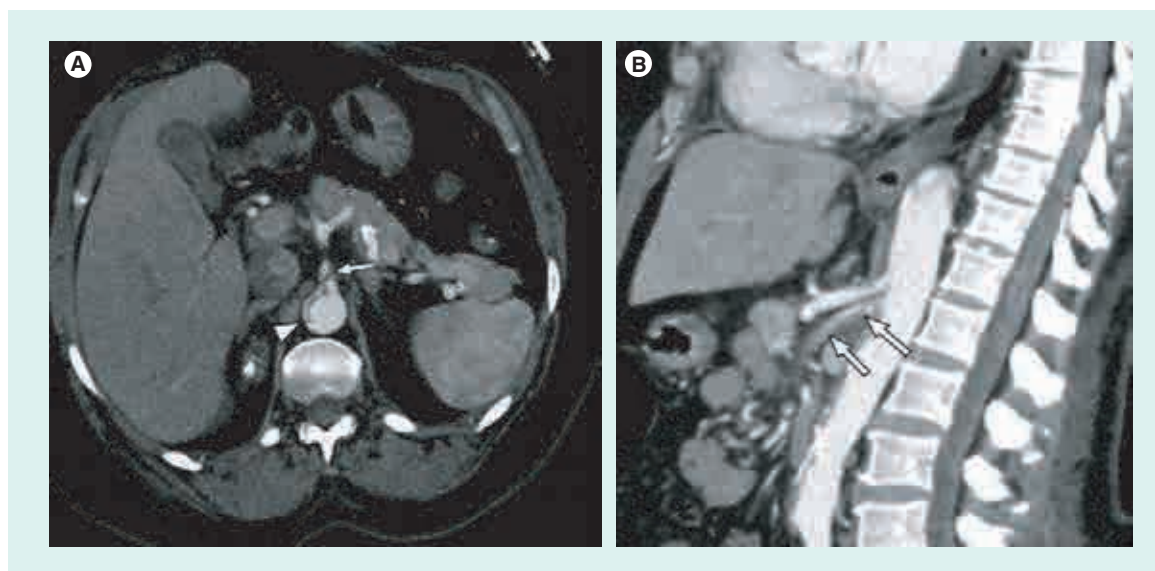


Figure 4. Aortic dissection extending into superior mesenteric artery. (A) Axial and (B) sagittal reformatted contrast-enhanced arterial phase computed tomography of the abdomen in a 49-year-old man shows aortic dissection (arrowhead in [A]) with dissection flap extending into the superior mesenteric artery (arrows in [A] and [B]).

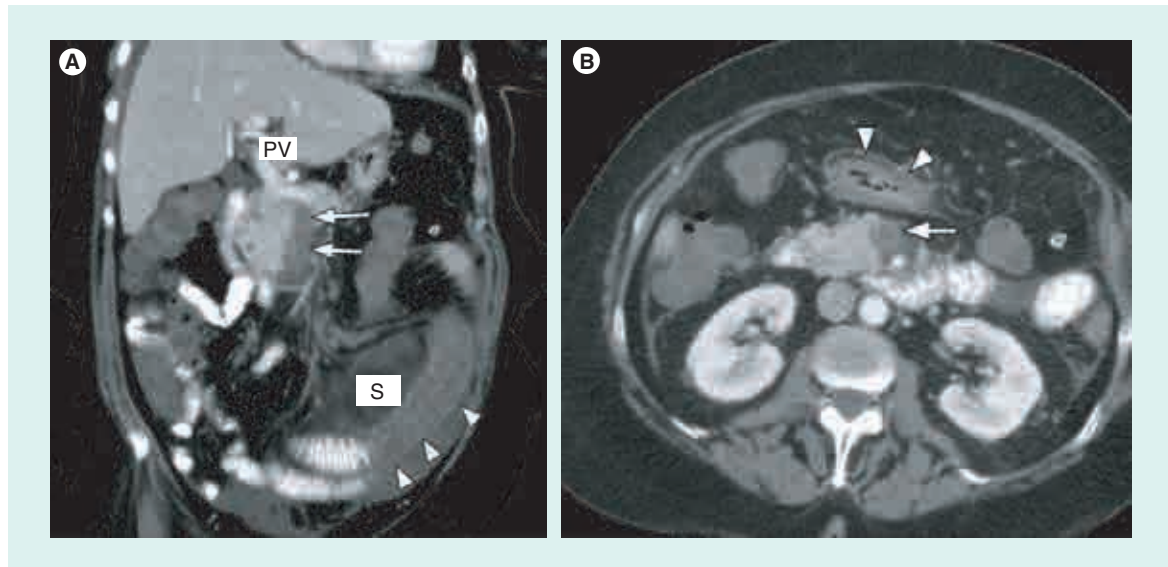


Figure 5. Superior mesenteric vein thrombosis. (A) Coronal and (B) axial contrast-enhanced computed tomography through the abdomen in a 55-year-old woman shows thrombus in the superior mesenteric vein (arrows in [A] and [B]), causing small bowel wall edema (arrowheads) and significant adjacent fluid and mesenteric fat stranding (S in [A]) consistent with bowel ischemia. PV: Portal vein; S: Mesenteric fat stranding.

fied fast-spin echo sequences [16]. Similar to CTA, 3D reformatted images are then generated from the 3D MRA sequence using a workstation, including maximum intensity projection, multiplanar reformatted and volume rendered techniques. For specific technical parameters, the reader is directed to reference articles for more detail [17].

Acute MI

Etiology & pathogenesis

Etiology of acute MI can be grossly divided as those due to arterial or venous occlusion or compromise, and from low flow states (nonocclusive MI).

Arterial emboli constitutes approximately 40–50% of cases of acute MI [1,7,18–19] and usually arise from the left atrium as a consequence of atrial fibrillation [19]. Emboli commonly lodge approximately 3–10 cm distal to the origin of the vessel, often at the level of middle colic artery, as the vessel tapers and decreases in caliber [1,7,18,20]. Other causes for emboli may include arrhythmias, cardiac valvular disease, prior myocardial infarction, atherosclerosis, aortic dissection or iatrogenic following therapeutic mesenteric embolization for gastrointestinal bleeding.

Arterial thrombosis accounts for 20–30% cases of acute MI, usually seen in patients with pre-existing atherosclerotic disease [1,19,21]. SMA thrombosis mostly occurs from rupture of an unstable atherosclerotic plaque at the origin of SMA from the aorta. Since these patients have a chronic diffuse atherosclerotic disease of the abdominal aorta and thus formation of collateral

pathways, they may present with symptoms of chronic MI, including postprandial pain, food aversion and weight loss [4].

Superior mesenteric vein thrombosis is implicated in 5–10% of cases [19,22]. The cause may be inherent from hypercoagulable status as with sickle cell disease, polycythemia vera, protein C and S deficiencies, antiphospholipid antibody syndrome, liver disease, carcinomatosis, use of oral contraceptive pills, pregnancy, sepsis causing thrombophlebitis, vasculitis and thrombotic microangiopathies. Alternatively, mesenteric venous thrombosis may occur secondary to mechanical vascular compression, such as from a mesenteric mass/tumor, bowel obstruction due to closed loop bowel obstruction, or an internal or external hernia leading to bowel strangulation [23–25].

Nonocclusive MI (NOMI) accounts for the approximately 20–30% of cases [18,22,26], and seen in patients with low flow states, such as sepsis, shock, hypotension, cardiac failure [1,27], or secondary to medications such as digitalis, ergot derivatives, norepinephrine and cocaine [28,29]. NOMI is associated with adverse outcomes, including mortality rates of 58–70% [1].

Histopathologically, bowel ischemia can broadly be divided into different stages of mural injury; with the early stage being reversible mucosal injury causing mucosal disruption and hemorrhage, the second or intermediate stage due to continued malperfusion causing submucosal and muscular injury and may constitute partial bowel wall necrosis. This stage may present with reperfusion injury and submucosal hem-

orrhage. The third stage represents irreversible damage with transmural bowel infarction/necrosis [1,30].

Clinical presentation

Acute MI constitutes the majority of cases versus chronic MI. Patients with embolic vascular etiology have an acute onset of abdominal pain compared with a more insidious onset of symptoms in thrombotic events [31]. Classically in acute MI, the degree of acute abdominal pain is out of proportion to the findings on physical examination.

However, many acute MI patients present with non-specific or vague abdominal pain associated with nausea, vomiting and diarrhea, which overlaps with many other gastrointestinal pathologies, including but not limited to inflammatory bowel disease, infectious enteritis, mechanical bowel obstruction, cholecystitis and pancreatitis. Therefore, the diagnosis of acute MI poses a diagnostic challenge clinically [4,32]. Absent bowel sound with peritoneal signs of abdominal guarding and rigidity may suggest irreversible bowel ischemia and infarction. NOMI has a similar nonspecific clinical presentation, although presence of volume depletion, hypotension, shock or predisposing etiological factors (e.g., underlying cardiac or renal disease, or medications) may be of certain help [26,33]. NOMI remains a difficult condition to diagnose and evaluation with CTA to exclude occlusive etiology and extent of damage, remains crucial in directing the clinician to medical or surgical management as patient without evidence of occlusive disease

and no significant bowel injury may well respond to medical therapy [26].

There are no definitive serum markers specific to diagnosis of acute MI. The several serum markers that have been described with MI includes serum amylase, lactate dehydrogenase, creatinine phosphokinase, inorganic phosphate and lactate, all of which remain non-specific and can be encountered with variety of other pathologies [4,32,34]. For example, elevated serum lactate level, which has been demonstrated to have a sensitivity of 100% for MI, has a specificity of only 42% and can be seen with other conditions such as bacterial peritonitis, pancreatitis and intestinal obstruction [35,36]. Elevated serum amylase can be seen late in the course of MI but is also elevated with other diseases such as acute pancreatitis. An elevated white blood cell count, although noted in 90% of patients with acute MI, can be seen in bowel infection or inflammation [32].

Imaging findings

The imaging findings can be grossly divided into vascular and nonvascular (bowel).

Vascular

Acute mesenteric artery occlusion occurs with thromboembolic phenomenon, predominantly involving the SMA and on CTA seen as filling defect and vessel cut-off sign. Embolic occlusion of SMA is usually seen as eccentric or central filling defect 3–10 cm from the origin (Figure 2) [1,4]. In these patients, syn-

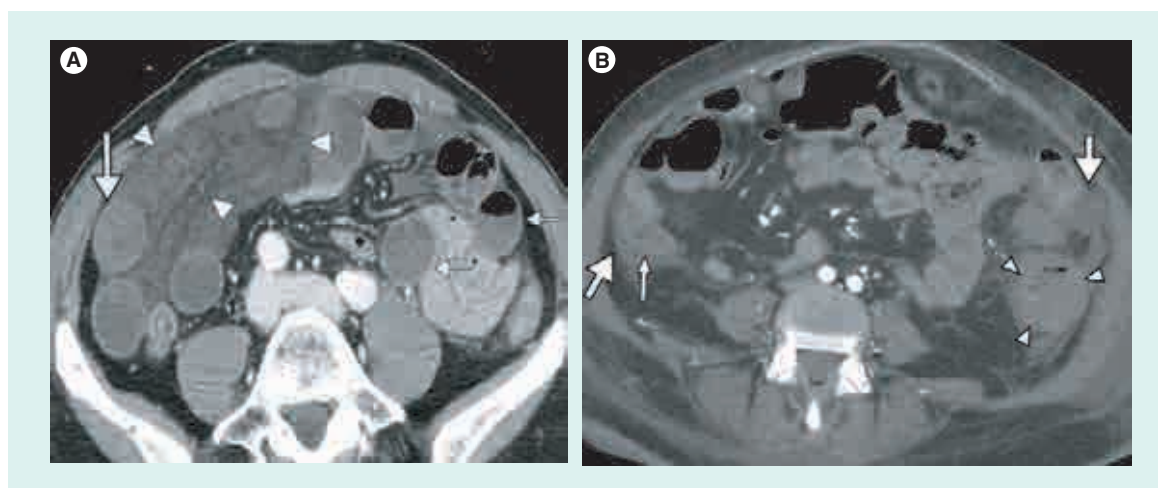


Figure 6. Bowel ischemia. (A) Axial contrast-enhanced arterial phase computed tomography image through the mid-abdomen in a 53-year-old man with acute abdominal pain shows circumferential small bowel wall thickening with target pattern of enhancement in the right abdomen (arrow) and significant adjacent mesenteric edema and fluid (arrowhead). On surgery this was confirmed as closed loop small bowel obstruction from an internal hernia with bowel necrosis. (B) Axial contrast-enhanced arterial phase computed tomography image through the mid-abdomen in a 64-year-old man with chronic intermittent postprandial abdominal pain shows circumferential wall thickening and decreased enhancement of the ascending colon (thin arrow) with lack of enhancement in the descending colon (arrowheads) and associated adjacent fluid and stranding (arrows). Colonic ischemia confirmed at surgery.

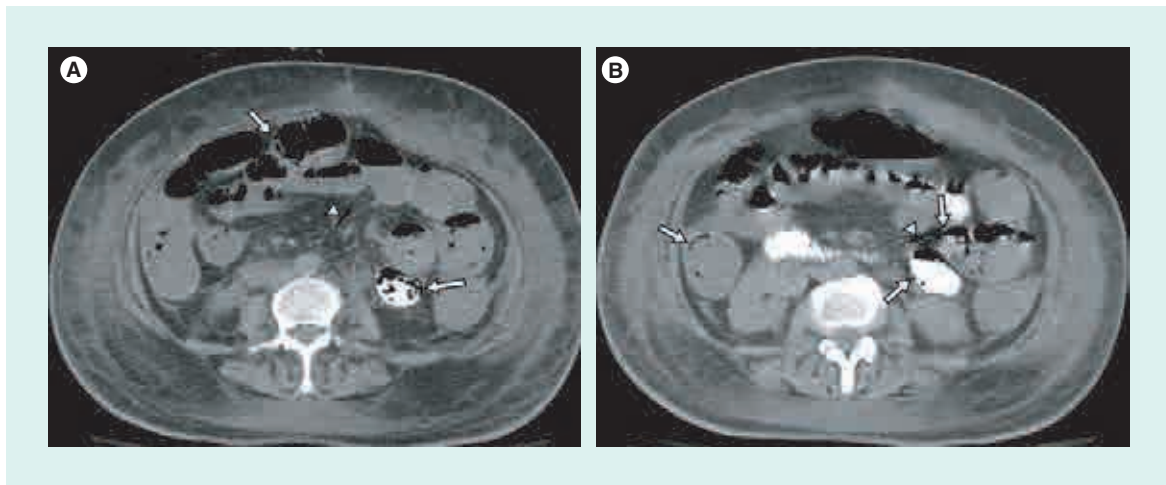


Figure 7. Nonocclusive mesenteric ischemia. Axial unenhanced computed tomography image through the mid abdomen (A & B) in this 73-year-old female with respiratory failure, cardiac arrest, hypotension, dropping hemoglobin and abdominal distension. Computed tomography shows pneumatosis involving small and large bowel (arrows), mesenteric venous gas (arrowheads) diffuse body wall and mesenteric edema most suggestive of bowel ischemia (nonocclusive mesenteric ischemia) in the given clinical setting. The study was performed without intravenous contrast given the patient's acute renal failure.

chronous embolic insult to other organs can be seen (Figure 2) [11].

In contrast with embolic occlusion, SMA thrombosis is seen as a filling defect within 2 cm of its origin and typically seen in the background of significant atherosclerotic disease of the aorta, including calcified and noncalcified atherosclerotic plaques, which predispose thrombus formation (Figure 3). SMA dissection either isolated or in continuity to aortic dissection is another cause of acute mesenteric vascular compromise that can lead to acute MI. SMA dissection is seen as thin hypodense or low signal-intensity flap with clear delineation of true and false lumen (Figure 4). In some instances the SMA can originate entirely from the false lumen of an aortic dissection, predisposing to higher risk for MI.

Mesenteric venous occlusion can be an acute or chronic presentation depending on presence of intraluminal thrombus and occlusion, or gradual compression and obstruction. Acute mesenteric venous occlusion can also occur in the setting of complicated or closed loop bowel obstruction. The occluded vein may be either distended from intraluminal thrombus or may be compressed from adjacent mass, causing sluggish flow and eventually thrombus (Figure 5). Dual-phase CTA with arterial and venous phases will best delineate the thrombus itself and the extent of disease as a filling defect in the vein itself.

In NOMI, which occurs due to splanchnic vasoconstriction causing hypoperfusion CTA, helps in excluding mesenteric occlusive etiology. On CT, the hypotensive or shock state can manifest as diminutive caliber of the SMA and mesenteric vascular arcade, and even the inferior vena cava [37].

Bowel (nonvascular)

There are various CT features of bowel abnormality in ischemia that in combination can be extremely helpful in prompt diagnosis.

Bowel wall thickening, attenuation & enhancement

While bowel wall thickening in itself is not a specific finding for MI, it remains a commonly encountered CT feature, seen in approximately 26–96% cases of bowel ischemia [38]. A small bowel is said to be thickened when it measures more than 3 mm in an optimally distended bowel, with underdistended loops sometimes posing difficulty in assessment. The norm for colonic wall thickness range from 3 to 5 mm. Bowel wall thickening can occur from mural edema, inflammation, hemorrhage, or even superinfection in the setting of bowel ischemia, with superinfection commonly seen in ischemic colitis [1,9,38–41]. The degree of bowel wall thickening in itself does not correspond to the severity of bowel injury [1,22]. However, the degree of wall thickening may vary with isolated arterial or venous occlusion. Arterial occlusive MI or infarction typically causes a paper thin wall, which has been attributed to the fact that lack of arterial flow causes loss of tissue volume, intestinal muscular tone and bowel dilation [1,22,41–42]. Bowel wall thickening of >8 mm and up to 15 mm is often observed with mesenteric venous occlusion, but also can be seen with complicated bowel obstruction/strangulation, ischemic colitis and following reperfusion in arterial occlusion implying intramural hemorrhage (Figures 5 & 6) [1,22,43]. Bowel wall attenuation may range from low attenuation related to

intramural edema or inflammation to high attenuation seen with intramural hemorrhage or hemorrhagic infarction, the latter best assessed on unenhanced CT images. In the absence of unenhanced CT, differentiating intramural hemorrhage and hyperemia or hyperperfusion may be difficult on contrast-enhanced CT. Hyperemia may be seen with mesenteric venous occlusion whereas hyperperfusion implies reperfusion in arterial occlusive or nonocclusive bowel ischemia or superinfection. While bowel wall thickening can be appreciated with both positive and neutral oral contrast, the latter is favored in assessing the bowel wall enhancement [40].

Postcontrast bowel wall/mucosal enhancement is an important feature in the evaluation of ischemia or infarction. The degree of enhancement can be best determined by comparison of the affected loop to the normal bowel. Diminished or absent postcontrast enhancement of the bowel mucosa (inner layer) is a specific but not sensitive finding for acute MI [1,22,44,45]. A stratified or 'target' enhancement of the bowel described with MI is seen as enhancing mucosal

and serosal layers, which have been offset by the edematous, hypodense submucosal layer (Figure 6) [40,46]. Hyperenhancement of the bowel wall on CT may be seen with hyperemia (in venous occlusive MI), hyperperfusion (reperfusion in arterial occlusive MI) or in NOMI (from reperfusion or due to slow flow secondary to splanchnic vasoconstriction from hypovolemia) (Figure 7) [45–47].

Bowel dilation and air–fluid level on CT has been described with acute bowel infarction and less often with reversible bowel ischemia. [1,48]. This is attributed to altered peristalsis secondary to transmural ischemia causing neural damage, resulting in increased exudation of fluid within the bowel lumen, thereby making the CT finding of bowel dilation and fluid–fluid level (rather than gas-filled bowel) more suggestive of acute bowel ischemia or infarction [1,18,42].

Pneumatosis intestinalis & portomesenteric venous gas

Pneumatosis intestinalis (presence of gas within the bowel wall) and portomesenteric venous gas

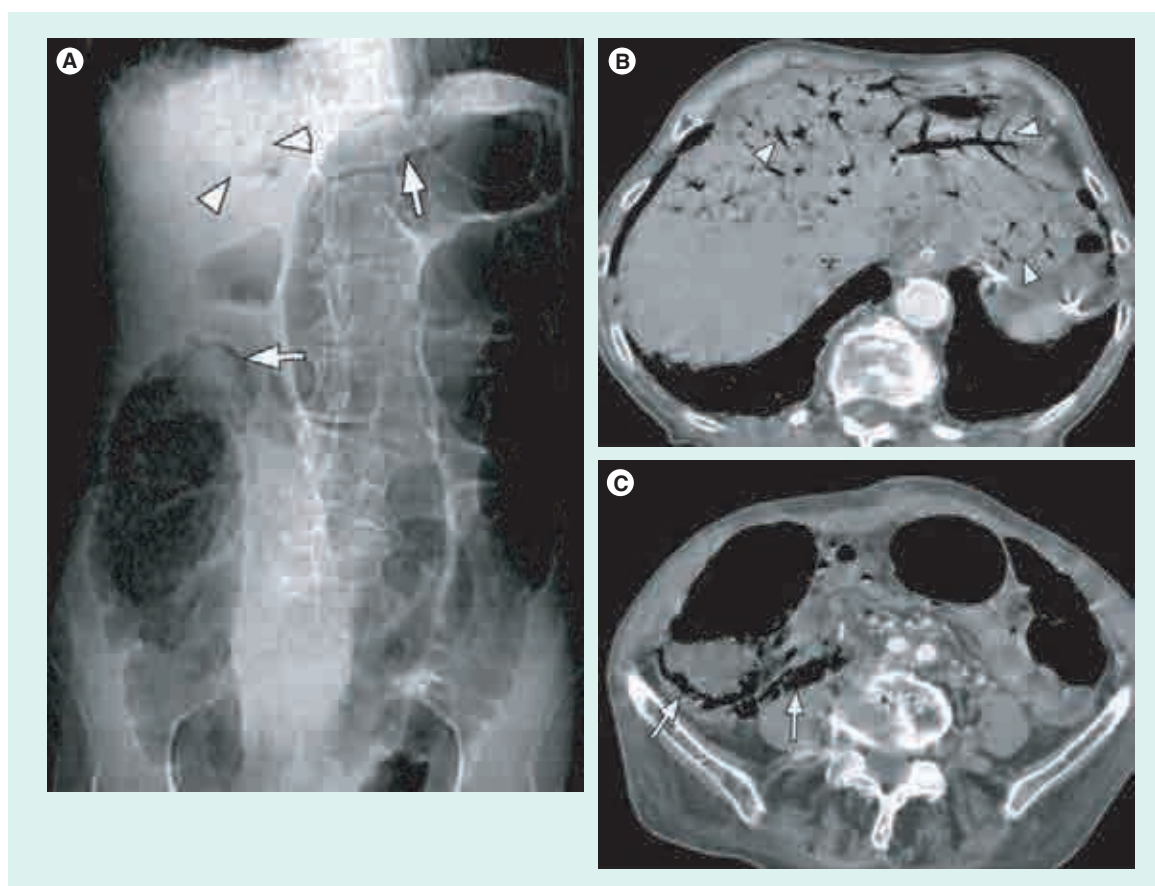


Figure 8. Bowel infarction with pneumatosis and portal venous gas. (A) Scout image from computed tomography scan on a 75-year-old man shows diffuse colonic dilation with pneumatosis (arrows) and intrahepatic portal venous gas (arrowheads). Axial contrast-enhanced computed tomography image through the (B) upper and (C) mid abdomen confirms the findings on scout view, consistent with transmural ischemia, confirmed on surgery.

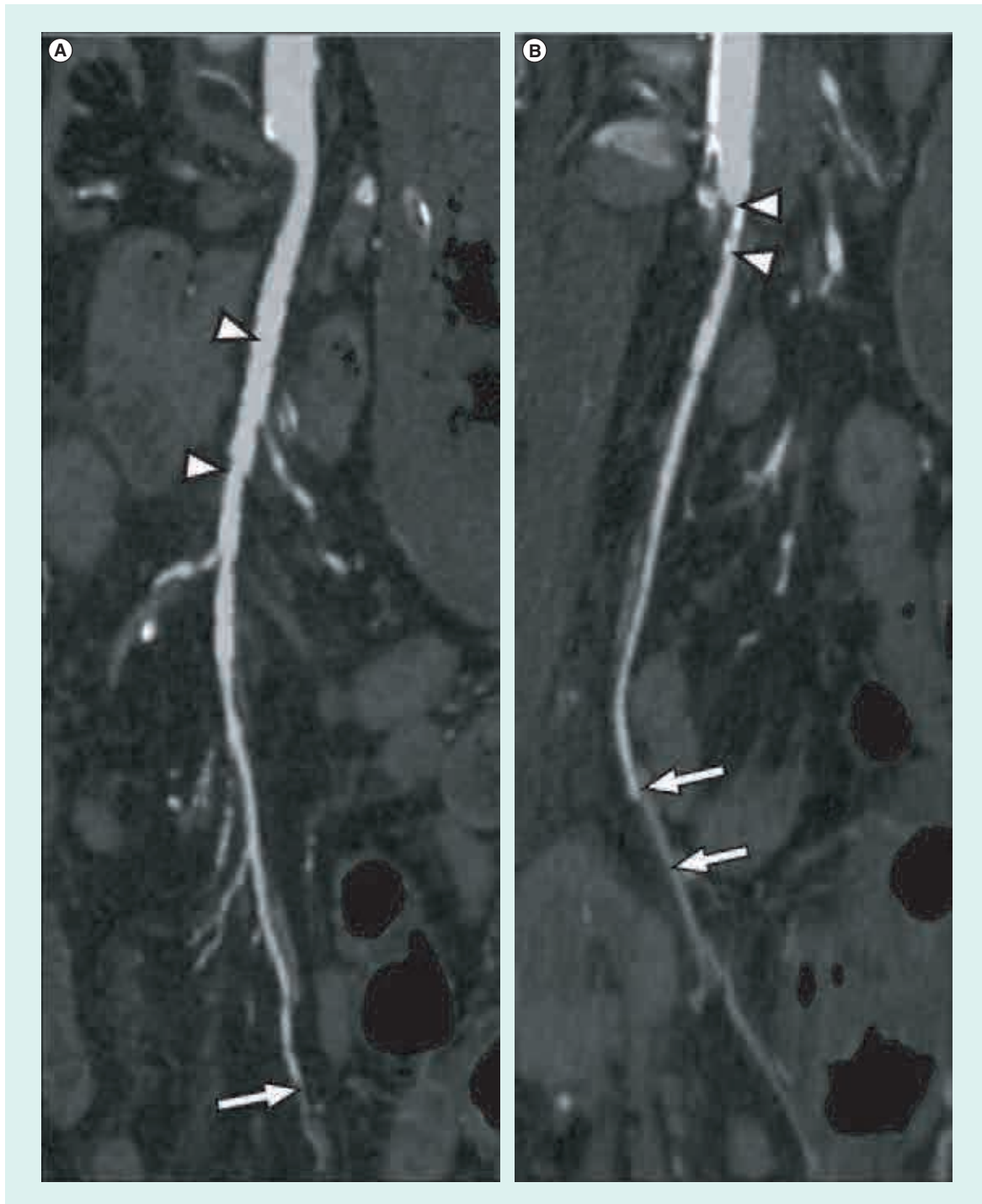


Figure 9. Chronic mesenteric ischemia. Curved reformatted arterial phase computed tomography images of the (A) superior mesenteric artery and (B) inferior mesenteric artery in a 64-year-old man with chronic intermittent postprandial abdominal pain. Demonstrates diffuse atherosclerotic narrowing of both the vessels (arrowheads) and near complete occlusion of more distal segmental branches (arrows).

is an imaging finding that is seen in the setting of bowel ischemia but also can be seen with nonischemic causes including chronic obstructive pulmonary disease, asthma, chronic steroids and chemotherapy. In the setting of ischemia, the presence of pneuma-

toxis and portomesenteric gas has been regarded as an ominous sign of irreversible injury and transmural necrosis (infarction) [1]. However, studies have shown that these two imaging findings do not always suggest transmural infarction but can be seen with

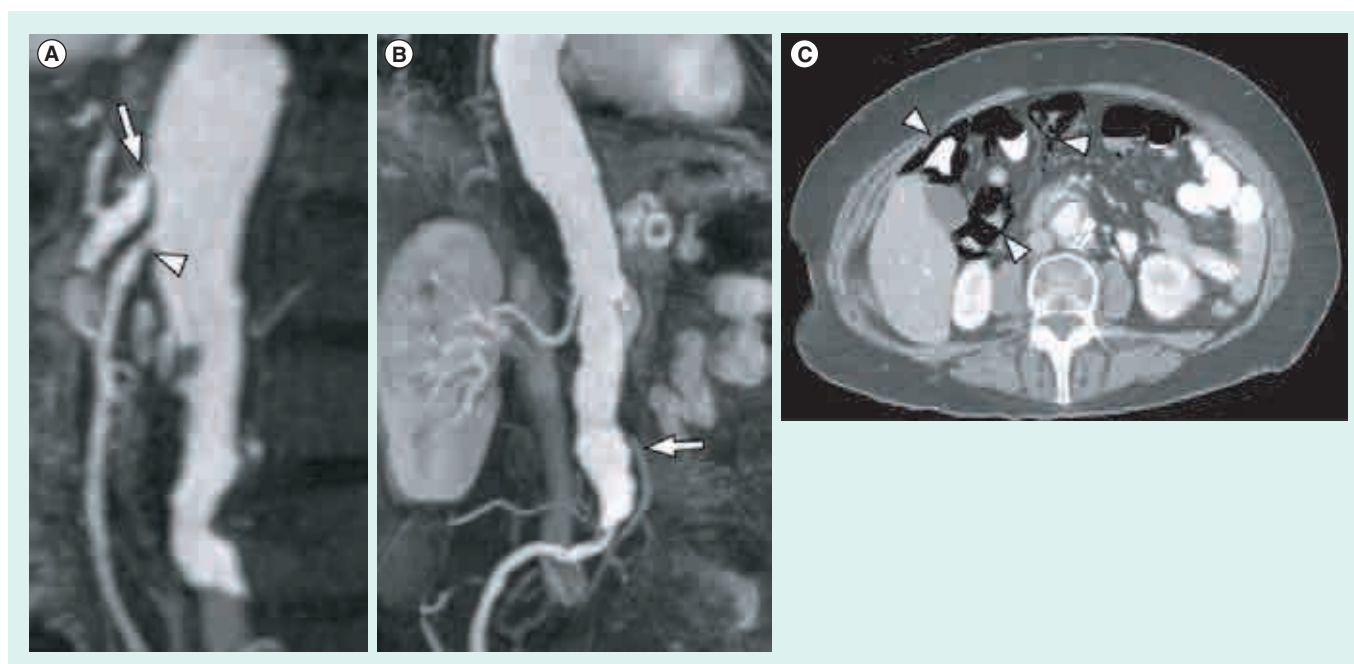


Figure 10. Acute episode of mesenteric ischemia in the setting of chronic mesenteric vascular disease. Contrast-enhanced T1-weighted images from magnetic resonance angiography in this 69-year-old woman with intermittent abdominal pain and bloating. (A) Sagittal and (B) oblique coronal views demonstrate diffuse atherosclerotic disease of the aorta with significant narrowing at the origin of the celiac artery (arrow) and superior mesenteric artery (arrowhead) in (A), and the inferior mesenteric artery (arrow) in (B). Subsequent contrast-enhanced computed tomography image in the same patient during another acute episode of abdominal pain shows wall thickening and band-like pneumatosis in the right colon (arrowheads).

partial mural bowel ischemia [49,50]. The patterns of pneumatosis on CT can vary from band-like to bubble-like gas collection. Band-like pneumatosis with portomesenteric venous gas is highly associated with transmural bowel infarction, while bubble-like pneumatosis and isolated portomesenteric venous gas may be related to partial mural bowel ischemia [49]. In the presence of these CT findings careful attention to other CT features of bowel ischemia (vascular occlusion, bowel wall thickening, altered wall enhancement and mesenteric features) remains crucial. The combination of absent bowel wall enhancement and presence of bowel wall pneumatosis has a sensitivity and specificity for MI of 42 and 97–100%, respectively (Figure 8) [51].

Erroneous CT diagnosis of pneumatosis can be made when intraluminal gas is trapped between feces, debris and adjacent mucosal fold or bowel wall – so called pseudopneumatosis. Pseudopneumatosis is often seen in the cecum and ascending colon, seen as punctate irregular gas column with gas collection against the bowel wall stopping at the free gas–fluid/debris interface with the bowel wall [52].

CT findings of mesenteric fat stranding, edema and fluid in bowel ischemia depends on etiology and severity. While it is commonly seen in mesenteric venous occlusion or complicated small bowel obstruction,

secondary to elevated mesenteric venous pressure, it can be seen with superinfection in ischemic colitis. However, presence of these CT features in arterial occlusive bowel ischemia would suggest transmural infarction as mesenteric changes are not evident in the early phase and thus help in assessing severity [1,43,53]. In NOMI, due to hypoperfusion and vasoconstriction there may not be significant mesenteric edema; however, with reperfusion mesenteric congestion and edema develops.

Chronic MI

Chronic MI accounts for less than 5% of all MI [32]. Chronic MI is seen in elderly patients with significant diffuse atherosclerotic disease and has a more indolent course over months to years. Clinical presentation includes long-standing symptoms of severe abdominal pain, often presenting immediately after eating (‘abdominal angina’) due to an inability of the splanchnic circulation to adequately respond to increased vascular demand by the small bowel, thereby leading to fear of eating (sitophobia) and subsequently weight loss [4].

Etiopathology

As mentioned previously, diffuse significant atherosclerotic disease is a predisposing factor for chronic

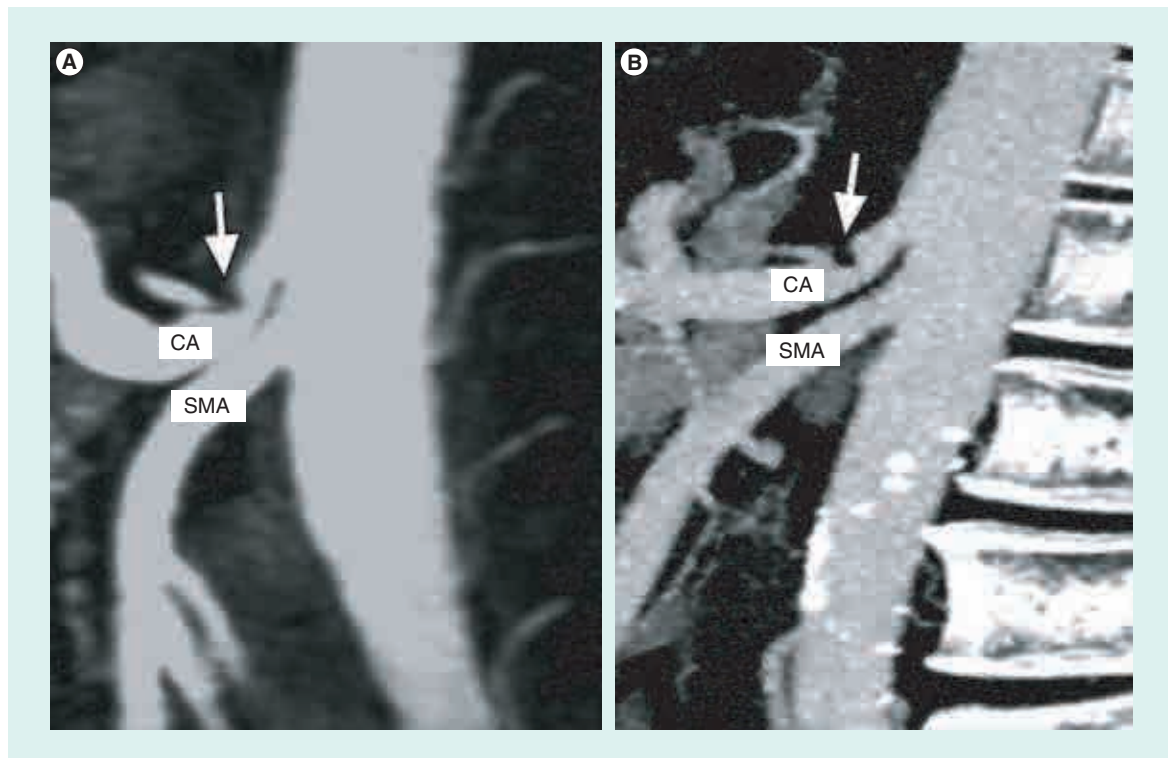


Figure 11. Hypertrophied median arcuate ligament. (A) Sagittal reformatted contrast-enhanced T1-weighted image from magnetic resonance angiography and (B) sagittal contrast-enhanced computed tomography in two different patients demonstrate sharp indentation on the superior aspect of the CA causing luminal narrowing and mild poststenotic dilation compatible with hypertrophic median arcuate ligament. Neither of the patients had bowel findings of ischemia. CA: Celiac artery; SMA: Superior mesenteric artery.

MI, with both calcified and noncalcified plaques contributing to narrowing/occlusion of the celiac axis, SMA and IMA. Occlusion of two of three major mesenteric vessels is generally required to consider the diagnosis of chronic MI [32]. Other vascular causes of chronic MI may include large or medium vessel vasculitis (Takayasu's arteritis and polyarteritis nodosa), median arcuate ligament syndrome, fibromuscular dysplasia and focal aneurysmal dilation. Vascular occlusion from direct invasion or encasement by mesenteric or retroperitoneal tumors and postradiation therapy changes are additional causes of chronic MI. Given the prolonged indolent course, there is generally sufficient time for development of collateral circulation, and thus bowel infarction is less commonly seen than acute MI [32].

Imaging

CTA with 3D reformation is highly sensitive in demonstrating the extent of atherosclerotic disease including stenosis at the origin of the major vessels, calcified or noncalcified plaque within the segmental vessel, and development of collateral circulation (Figure 9). Contrast-enhanced MRA is also helpful in evaluat-

ing atherosclerotic disease burden and assessing both luminal and vascular wall changes. MRA results are best in evaluation with proximal celiac and superior mesenteric artery, but has somewhat limited spatial and temporal resolution in evaluating inferior mesenteric artery and also segmental and peripheral mesenteric branches (Figure 10). Vasculitis can be seen on CTA and MRA as segmental narrowing of the involved vessels, often resulting in a beaded appearance of alternating strictures and focal dilations. 3D reformatted CT and MR images can also help to assess the degree of stenosis and oblique/tortuous vascular anatomy, and thereby assist in therapeutic planning for surgical revascularization or catheter-based intervention. In addition, CTA and MRA may demonstrate other causes of mesenteric vasculature narrowing, such as median arcuate ligament syndrome, which is caused by extrinsic compression of the celiac artery by the central tendon of the diaphragmatic crura (Figure 11); or direct invasion or encasement of the mesenteric vasculature by neoplastic process. While the bowel findings are often underwhelming in chronic MI, cases with superimposed acute episode on chronic mesenteric vascular changes will demon-

strate CT bowel findings similar to that described with acute MI.

Conclusion & future perspective

Given the nonspecific clinical presentation and laboratory markers for diagnosis of MI, further research in evaluating serum markers with higher accuracy may be beneficial.

CTA and MRA has undergone significant advancement in software and technology over the past decade improving spatial and temporal resolution with higher multiplanar capabilities thereby significantly increasing the sensitivity and specificity similar to conventional catheter angiography, long considered to be modality of choice in diagnosis of MI. Continued research and refinement of software, scanning techniques/parameters and also radiation dose-reduction techniques with CT continues to the current focus of radiology fratern-

nity. The role of MRI in imaging evaluation of acute MI is also underway.

While CTA has a high sensitivity and specificity in diagnosis, high clinical suspicion remains the key in directing the patient to timely imaging evaluation. Thus, more clinical awareness and suspicion of this entity is required among clinicians in both the emergent and nonemergent setting.

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