Imaging of Bowel Ischemia: An Update, From the *AJR* Special Series on Emergency Radiology

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Acute mesenteric ischemia is a life-threatening condition that results from abrupt reduction in or cessation of blood flow to the bowel. Characterized by nonspecific abdominal symptoms, mesenteric ischemia is infrequently encountered and commonly misdiagnosed, with potentially catastrophic consequences. Prompt clinical diagnosis and early implementation of therapeutic interventions are critical to improving patient outcomes. Because cross-sectional imaging plays a key role in the diagnosis of mesenteric ischemia, radiologists must be familiar with the varied imaging manifestations of intestinal ischemia. Thus, the objectives of this article are to review the various types and common causes of mesenteric ischemia and to describe its spectrum of multimodality imaging findings, with special attention to novel imaging techniques and emerging diagnoses.

Mesenteric ischemia (MI) occurs when an abrupt reduction in or cessation of blood flow to the bowel creates a hypoxic state in which oxygen delivery is insufficient to meet metabolic demand [1–3]. Left untreated, this intestinal hypoperfusion can rapidly progress to transmural infarction, necrosis, and perforation. Accounting for only 0.1% of acute surgical admissions and characterized by nonspecific abdominal symptoms [2, 4, 5], MI is infrequently encountered and often misdiagnosed, with potentially catastrophic consequences. Despite recent technologic advances in cross-sectional imaging and the introduction of novel treatment approaches, MI remains a highly morbid disease, with reported mortality rates ranging from 50% to 90% [2, 3, 5–7]. Prompt recognition of this condition and the early initiation of therapy are key to improving patient outcomes. Accordingly, the aims of this article are to review the various types and common causes of MI and to describe the spectrum of multimodality imaging findings, with special attention to novel imaging techniques and emerging diagnoses.

Anatomy

The celiac axis (CA), superior mesenteric artery (SMA), and inferior mesenteric artery (IMA) constitute the major arterial vessels of the splanchnic circulation [8] (Fig. 1). Arising at the level of the diaphragm, the CA gives rise to the common hepatic, splenic, and left gastric arteries and provides blood flow to the liver, spleen, stomach, and pancreas. The SMA serves as the predominant source of blood flow to the small bowel, giving rise to branches that supply the pancreas, duodenum, entirety of the jejunum and ileum, and right colon extending to the splenic flexure [9]. It anastomoses with the CA via pancreaticoduodenal branches and collateralizes with the IMA via the marginal artery of Drummond [10]. Supplying the left colon through the upper rectum, the IMA arises just above the aortic bifurcation and is the most diminutive of the mesenteric arteries. The middle and lower portions of the rectum are supplied by the anterior divisions of the iliac arteries [11]. Variable collateral pathways include the arc of Barkow, a communication between the left and right gastroepiploic arteries; the arc of Buhler, a communication between ventral embryonic segmental arteries; and the arc of Riolan, an anastomosis between the left and middle colic arteries [6, 10].

Classification and Imaging Findings

MI can be stratified, on the basis of symptom chronicity, into acute, chronic, and acute-on-chronic forms [1]. Defined as a sudden decrease in perfusion to the splanchnic circulation with the development of symptoms over the course of minutes to hours [1], acute MI

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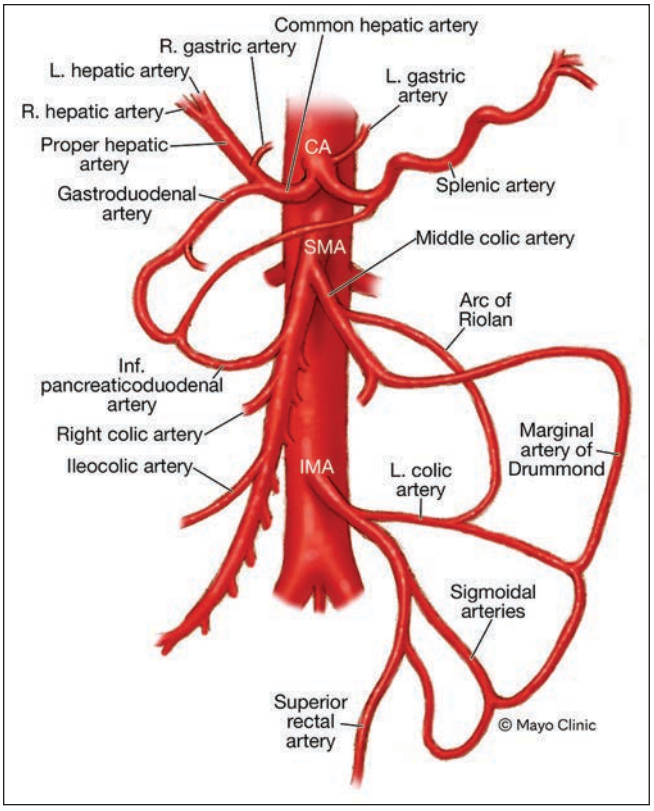

Fig. 1—Illustration shows normal mesenteric arterial circulation and common collateral pathways. R. = right, L. = left, CA = celiac artery, SMA = superior mesenteric artery, Inf. = inferior, IMA = inferior mesenteric artery. Used with permission of Mayo Foundation for Medical Education and Research, all rights reserved.

(AMI) is characterized by excruciating abdominal pain, frequently described as out of proportion to otherwise benign findings on examination [2, 3]. Physical signs are generic and include nausea, emesis, and diarrhea; the classically described triad of severe pain, bowel emptying (emesis and diarrhea), and a potential embolic source is observed in only 40–80% of patients [12]. The presence of peritonitis on examination heralds the development of irreversible intestinal ischemia and portends a poor prognosis [3].

HIGHLIGHTS

- *MI is a potentially life-threatening condition that is challenging to diagnose given its nonspecific clinical symptoms.*
- *Imaging plays a key role in accurate and timely diagnosis, and radiologists must be familiar with the varied imaging appearances of intestinal ischemia.*
- *CT is the first-line imaging modality for diagnosis of MI; emerging imaging techniques, such as dual-energy CT, may facilitate recognition of subtle signs of MI.*

Chronic MI (CMI) presents more insidiously over the course of months. CMI is twice as common in women compared with men, and characteristic symptoms include postprandial abdominal pain resulting in weight loss and a fear of eating (food fear) [13, 14]. CMI is typically caused by atherosclerotic occlusive disease at the origins of the mesenteric vessels. Owing to rich arterial collateralization, it frequently does not occur until hemodynamically significant stenoses have developed in at least two of the major splanchnic arteries [15].

Acute-on-chronic MI occurs when symptoms of AMI develop in patients previously diagnosed with CMI [1].

AMI can be further classified as either occlusive or nonocclusive, with occlusive MI subcategorized into embolic, thrombotic, and venous forms by the cause of vascular occlusion (Table 1).

Arterial Thromboembolism

Background

The most common cause of AMI, arterial embolic disease accounts for up to 50% of cases [1] and often results from underlying cardiovascular disease (e.g., atrial fibrillation or thrombi that form from a preceding myocardial infarction) [4]. The SMA is particularly susceptible to emboli because of its high flow rate and narrow

TABLE 1: Classification of Acute Mesenteric Ischemia

Cause	Frequency (%)	Imaging Features	Pathophysiology
Arterial embolism	≤ 50	Bowel wall thinning ("paper thin"), diminished or absent mural enhancement May see infarction or ischemia of solid abdominal viscera	Embolic vascular occlusion, frequently secondary to underlying cardiovascular disease (e.g., atrial fibrillation)
Arterial thrombosis	20–30	Bowel wall thinning ("paper thin"), diminished or absent mural enhancement	Atherosclerotic vascular occlusion in the absence of adequate collateral flow
Mesenteric venous thrombosis	5–20	Significant bowel wall thickening, mural stratification, mesenteric stranding, or edema	Impaired venous outflow Increased vascular resistance eventually obstructs arterial inflow
Nonocclusive mesenteric ischemia	5–20	Mesenteric vascular dilatation or narrowing ("string-of-sausages" sign) Features of CT hypoperfusion complex (e.g., bowel mural hyperenhancement, flattening of IVC, adrenal or renal hyperenhancement)	Poorly understood; likely a protective reflex in which mesenteric vessels constrict or spasm to preserve cardiac or CNS blood flow

Note—This table is based in part on information in Olson et al. [6], Fitzpatrick et al. [11], and Garzelli et al. [16]. IVC = inferior vena cava.

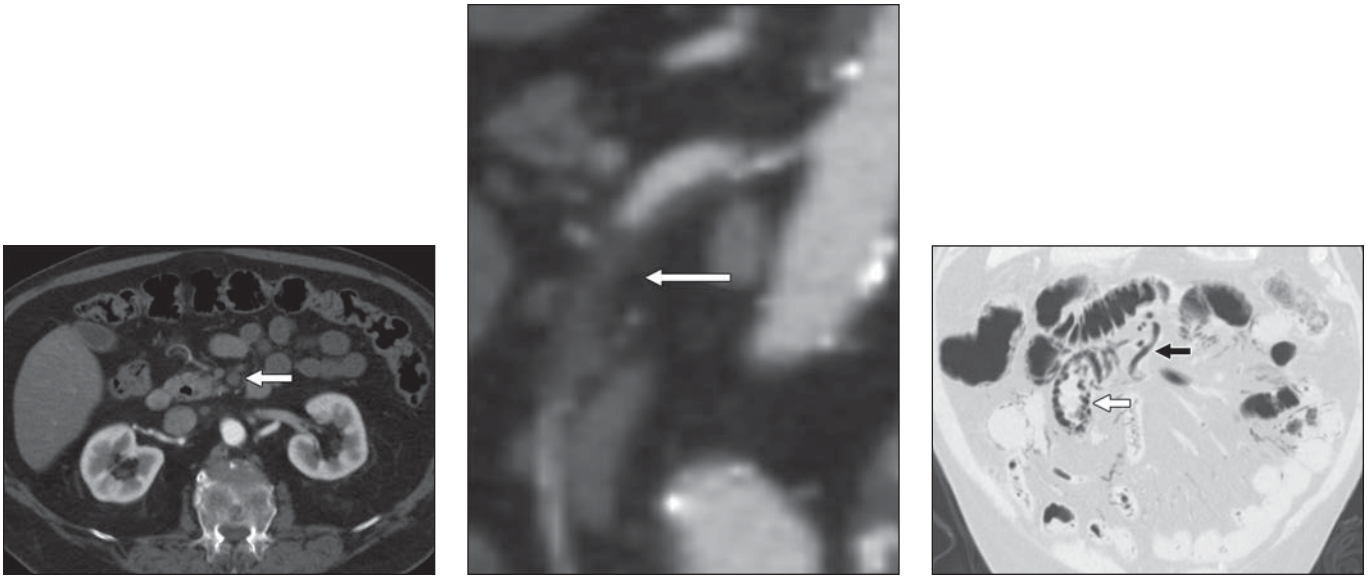

Fig. 2—67-year-old patient with acute abdominal pain who was diagnosed with embolic mesenteric ischemia.
A and **B,** Contrast-enhanced axial (**A**) and sagittal (**B**) CT images show embolic occlusion (*arrows*) of superior mesenteric artery.
C, Coronal CT image shows extensive pneumatosis (*white arrow*) and mesenteric venous gas (*black arrow*).

branching angle from the aorta (Fig. 2). Emboli often lodge 3–10 cm distal to the SMA origin, near the takeoff of the middle colic artery, which results in sparing of inferior pancreaticoduodenal branches and preservation of inflow to the proximal jejunum [8, 16]. A small proportion (≈ 15%) of emboli occur at the SMA orifice, which, in the absence of robust collateral circulation, may result in catastrophic small-bowel ischemia [8, 17].

Commonly arising in a background of systemic atherosclerotic disease, thrombotic occlusion is the second most common cause of AMI, responsible for 20–30% of cases [18, 19]. These patients often have a history of CMI and its attendant symptoms; the long time course of the disease facilitates formation of an extensive network of arterial collaterals, and symptomatic occlusions frequently involve at least two of the three major visceral arteries, usually the CA and SMA [4, 18, 20]. Critical vascular stenoses progress over time until complete thrombosis occurs during a period of low flow [1, 8]. Because thrombosis occurs at the ostia of the mesenteric vessels, ischemia can affect a long segment of bowel [11]. As a result, thrombotic AMI carries the worst prognosis of any subtype of MI [6, 7, 11].

Imaging Findings

Accurate and timely diagnosis of MI necessitates careful scrutiny of the mesenteric vasculature in both arterial and portal venous phases as well as the appearance of the small bowel and colon. Crucially, vascular abnormalities develop before bowel findings [6]. On contrast-enhanced images, low-attenuation filling defects in the mesenteric arteries or veins are specific indicators of thromboembolism; these may manifest as regions of high attenuation on noncontrast images [4]. Analysis of the mesenteric vasculature in multiple planes and on maximum-intensity-projection (MIP) images may facilitate detection of vascular abnormalities.

In arterial thromboembolic disease, reduction in or cessation of arterial blood flow results in absent or decreased bowel wall enhancement [4]. Restoration of intestinal perfusion may cause mural thickening, hyperenhancement, and stratification, similar to that seen with shock bowel [6] (Table 2). Progression of ischemia results in destruction of intramural nerves and loss of tissue volume and muscle tone, with a resultant "paper thin" appearance of the small-bowel wall, a highly specific but insensitive sign of AMI

TABLE 2: Imaging Features of Mesenteric Ischemia and Differential Considerations

Imaging Feature	Cause	Common Mimickers
Mesenteric edema or stranding	Mesenteric vascular engorgement; impaired venous outflow with extravasation of fluid into mesentery	Volume overload, third-spacing of fluid, portal hypertension
Bowel luminal dilatation	Hypocontractility and aperistalsis secondary to intestinal ischemia or infarction	Postoperative or nonspecific adynamic ileus, pseudoobstruction
Bowel mural hypoenhancement	Insufficient intestinal blood flow due to mesenteric arterial or venous occlusion	None—finding is specific for intestinal hypoperfusion
Bowel mural thickening	Venous outflow obstruction with continued arterial inflow, resulting in submucosal edema	Inflammatory bowel disease, portal hypertension, infectious or malignant processes
Pneumatosis or portomesenteric venous gas	Transmural infarction or necrosis with translocation of luminal gas across the mucosa	Benign causes of pneumatosis; pseudopneumatosis, or air trapped between fluid and the bowel wall

Note—This table is based in part on information in Olson et al. [6] and Fitzpatrick et al. [11].

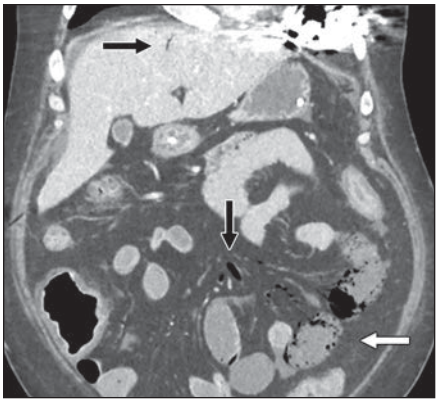

Fig. 3—59-year-old patient with acute nonocclusive mesenteric ischemia after repair of abdominal aortic aneurysm and placement of left ventricular assist device. Coronal CT image shows jejunal pneumatosis (*white arrow*) and portomesenteric venous gas (*black arrows*).

[16, 21]. Irreversible transmural infarction may result in the development of pneumatosis intestinalis, portomesenteric venous gas, and intraabdominal free air [4] (Fig. 3). Important clues to the diagnosis of embolic AMI include thrombus within the left-sided cardiac chambers or synchronous infarction or ischemia in solid abdominal viscera, particularly the kidneys, whereas thrombotic AMI more frequently develops in patients with significant underlying atherosclerotic disease [6].

Mesenteric Venous Thrombosis

Background

Approximately 5–20% of AMI cases are attributable to mesenteric venous thrombosis (MVT), which is more frequently encountered in younger patient populations [4, 16]. Risk factors include hypercoagulable states, such as those caused by factor V Leiden mutation or deficiencies in proteins S, protein C, and antithrombin; malignancy; infectious or inflammatory conditions; and venous stasis or obstruction secondary to immobility or conditions that cause extrinsic venous compression, such as bulky adenopathy [22]. The superior mesenteric vein (SMV) and portal vein are commonly involved, and symptoms are more likely to develop in patients with thrombosis of peripheral mesenteric venous branches [18]. Impaired venous outflow results in bowel mural edema and luminal distention (Fig. 4), with increased vascular resistance eventually obstructing arterial inflow and precipitating intestinal necrosis [4, 19].

Imaging Findings

A common finding in MVT is a filling defect in a mesenteric vein with peripheral rim enhancement of the venous wall. Central venous obstruction results in engorgement of collateral mesenteric venous pathways, and concurrent portal venous or splenic vein thrombus may be observed [4, 11]. Contrary to the marked bowel wall thinning that occurs with arterial occlusion, AMI due to MVT often presents with significant bowel mural thickening, sometimes up to 15 mm (normal thickness, 3–5 mm) [11]. Continued arterial inflow in the setting of obstructed venous outflow increases intramural hydrostatic pressure, and hypoattenuating edema within the bowel submucosa located between the enhancing mucosa and muscularis propria creates a halo or target appearance (Fig. 4). Perienteric mesenteric stranding and edema result from extravasation of fluid into the mesentery [6].

Nonocclusive Mesenteric Ischemia

Background

A lethal condition associated with high rates of in-hospital mortality [8, 16], nonocclusive MI (NOMI) is an umbrella term encompassing an array of disorders that cause intestinal hypoperfusion in the absence of arterial or venous thromboembolism [20, 23]. Believed to account for up to 20% of cases of AMI [2, 18], NOMI is likely underreported secondary to the significant challenges associated with its diagnosis [16]. Patients are often critically ill with significant predisposing conditions, including heart failure, major trauma, the use of vasopressors, and cardiogenic or septic shock [11] (Fig. 5). Pathophysiologically, NOMI is poorly understood but likely derives from a protective reflex in which the mesenteric vessels undergo constriction or spasm in an effort to preserve blood flow to the cardiac or central nervous systems [4, 11].

Imaging Findings

NOMI is challenging to diagnose on cross-sectional imaging. Constriction of the splanchnic vasculature occurs to preserve CNS and cardiac blood flow, and CTA may show narrowing of the

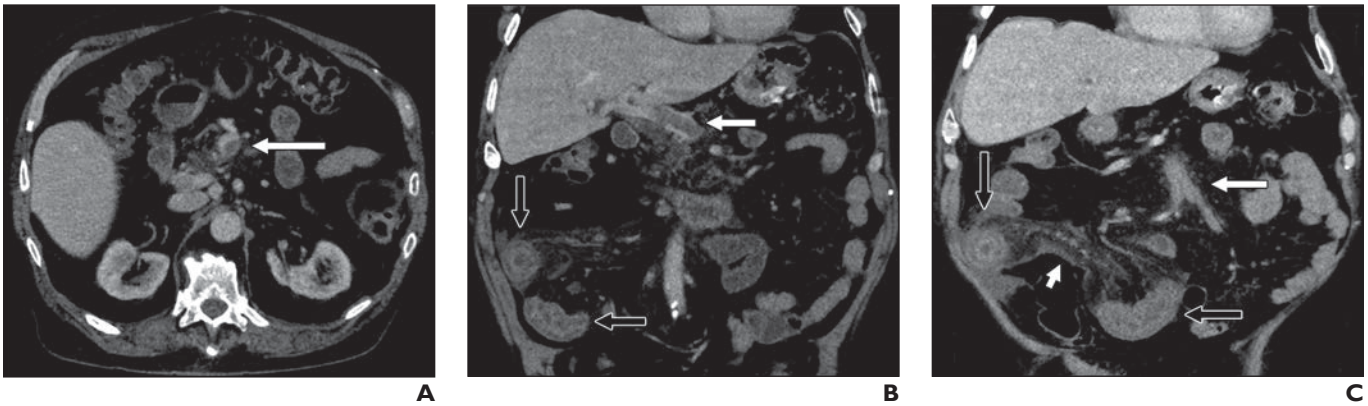

Fig. 4—75-year-old patient with history of orthotopic liver transplant who was diagnosed with mesenteric venous thrombosis.
A, Axial contrast-enhanced CT image shows partially occlusive thrombus (*arrow*) in superior mesenteric vein (SMV).
B, Coronal contrast-enhanced CT image shows thrombus within portal vein extending to portomesenteric confluence (*white arrow*). Loops of small bowel in right lower quadrant show mural thickening and stratification (*black arrows*).
C, Coronal contrast-enhanced CT image at more anterior location shows occlusive thrombus within SMV (*long white arrow*), small-bowel mural thickening and stratification (*black arrows*), and interloop fluid within mesentery (*short white arrow*).

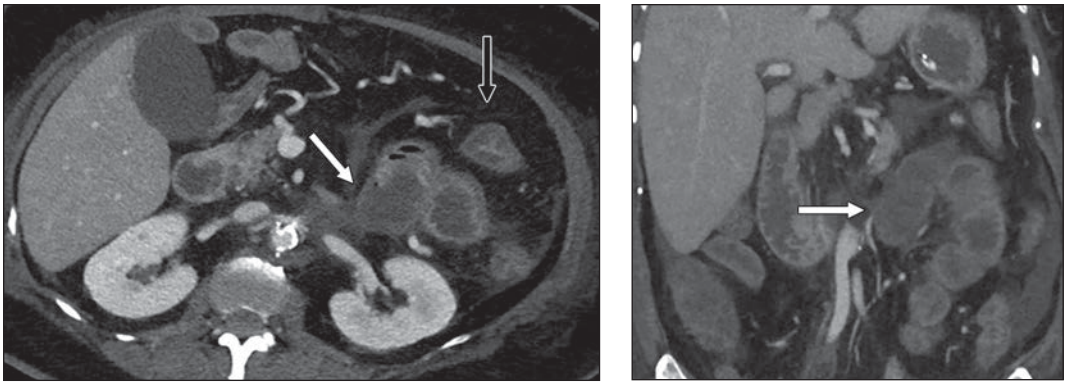

Fig. 5—65-year-old patient with nonocclusive small-bowel and colonic ischemia after aortofemoral bypass.
A, Axial contrast-enhanced CT image shows mural hypoenhancement of duodenum and jejunum (*white arrow*) near ligament of Treitz. Note colonic mural thickening and stratification (*black arrow*) involving descending colon.
B, Coronal contrast-enhanced CT image shows jejunal mural thickening and hypoenhancement with adjacent mesenteric fluid and stranding (*arrow*).

SMA and its first-order branches, beaded areas of arterial dilatation and narrowing (the so-called "string-of-sausages" sign) (Fig. 6), and features of the CT hypoperfusion complex, such as bowel mural hyperenhancement and hyperenhancement of the kidneys and adrenal glands [6, 11, 16]. There is often broad involvement of the small bowel and colon, and imaging findings are characteristically discontinuous and segmental, with intervening loops of normal bowel [4].

Special Considerations

Ileus, Obstruction, Ischemia, and Reperfusion

An important but frequently overlooked feature of AMI is bowel luminal dilatation. Intestinal infarction can result in hypocontractility and aperistalsis, rendering the bowel fluid filled and dilated (adynamic ileus), nonspecific findings with broad differential considerations. Absence of peristalsis has been reported as an early finding in AMI, and in the appropriate clinical context, this uniformly dilated bowel should be recognized as abnormal and heighten suspicion for ischemia [11].

A strangulating bowel obstruction is one resulting in either ischemia or infarction and most commonly results from a closed-loop obstruction, defined as obstruction of the bowel both proximally and distally at one point [6, 11]. Specific findings of strangulating obstruction include bowel mural hypoenhancement and the presence of mural hemorrhage, whereas the presence of mesenteric fluid has been described as highly sensitive for ischemia [6, 24].

The differentiation of reversible intestinal ischemia from irreversible transmural intestinal necrosis on imaging can be challenging. One previous analysis found that imaging features suggestive of necrosis include bowel wall thinning or thickening, luminal dilatation, and mural hypoenhancement; the presence of bowel loop dilatation, in conjunction with elevated serum lactate (> 2 mmol/L) and concomitant organ failure, was found to be highly specific for irreversible ischemia [25].

One study explored imaging features associated with intestinal reperfusion in patients with NOMI and found that mesenteric stranding, bowel mural thickening, and high attenuation of the bowel wall on noncontrast images were associated with reperfusion events. The recognition of potential spontaneous reperfusion in these cases may facilitate risk stratification in certain instances and enable prediction of which patients may progress to irreversible ischemia or require surgical intervention [26].

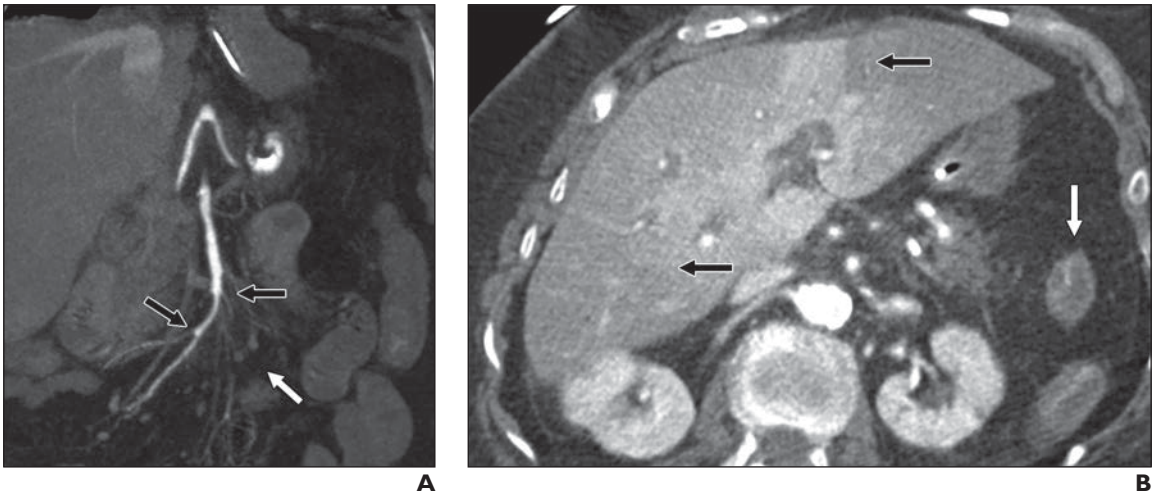

Fig. 6—88-year-old patient who presented to emergency department complaining of dizziness, abdominal pain, and hematochezia and was diagnosed with nonocclusive mesenteric ischemia.
A, Coronal CT image shows beaded appearance (*black arrows*) of superior mesenteric artery with pruning of peripheral branches (*white arrow*) supplying small bowel.
B, Axial CT image shows heterogeneous hepatic parenchymal enhancement (*black arrows*) and mural thickening involving descending colon (*white arrow*).

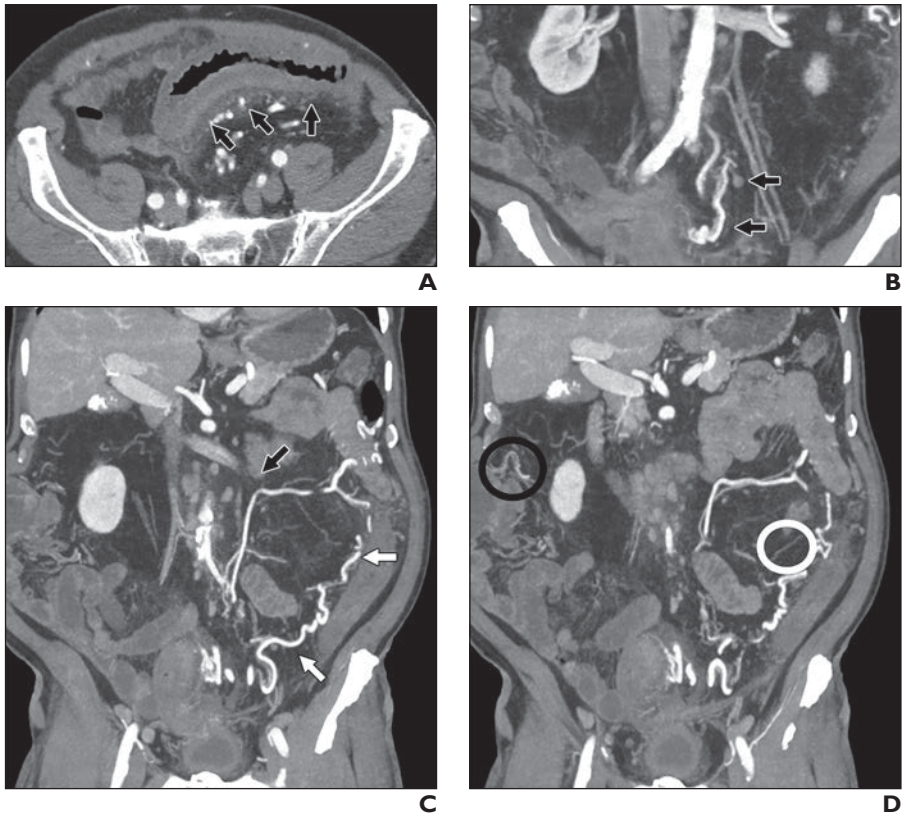

Fig. 7—82-year-old patient with idiopathic myointimal hyperplasia of mesenteric veins (IMHMV). Patient was initially diagnosed with ischemic colitis after endoscopic biopsy. Diagnosis of IMHMV was suggested on basis of preoperative imaging findings and confirmed after surgical resection.
A, Axial CT image shows marked mural thickening of descending and sigmoid colon (*arrows*).
B, Coronal maximum-intensity-projection (MIP) image shows early filling of dilated inferior mesenteric venous branches (*arrows*).
C and **D,** Coronal MIP images obtained at different levels show occlusion of inferior mesenteric vein (*black arrow*, **C**) with dilated pericolonic veins (*white arrows*, **C**) draining to splenic vein. Attenuation and tapering of peripheral mesenteric veins draining descending colon (*white circle*, **D**) are also shown, compared with normal peripheral mesenteric veins draining right colon (*black circle*, **D**).

Colonic Ischemia and Idiopathic Myointimal Hyperplasia of the Mesenteric Veins

Colonic ischemia is frequently classified by location as either right-sided or left-sided ischemic colitis. Left-sided colonic ischemia is believed to be due to self-limited microvascular occlusion, often occurring without obstruction of the splanchnic circulation [16]. Right-sided ischemia portends a poorer prognosis, is more commonly seen with occlusion of the SMA, and can occur in conjunction with small-bowel ischemia. For these reasons, right-sided colonic involvement is regarded as equivalent to AMI [27]. Most cases of colonic ischemia are transient, though 25% progress to chronic ischemia [28]. The proposed definition of chronic colonic ischemia includes the presence of histologic evidence of ischemia and a symptom duration ranging from 1 to 3 months [28, 29].

Imaging findings in colonic ischemia mirror those seen in the small bowel and include wall thickening, poor or absent enhancement, luminal dilatation (> 9 cm in the cecum and > 6 cm elsewhere within the colon), pneumatosis intestinalis, and frank perforation [27].

A rare cause of nonthrombotic chronic colonic ischemia, idiopathic myointimal hyperplasia of mesenteric veins (IMHMV) was first described in 1991 [30] but has received renewed attention in the literature in recent years. It represents a distinct cause of chronic colitis, with common clinical features including diarrhea, abdominal pain, and hematochezia [28]. IMHMV predominantly affects middle-aged men and typically involves the left colon, most frequently the rectosigmoid [31]. The true incidence of IMHMV is unknown, as it remains an underrecognized entity and is often misdiagnosed as inflammatory bowel disease or nonspecific ischemic colitis. Diagnosis is often delayed and challenging to establish histopathologically, and patients experience failure of medical therapies, including antibiotics, immunosuppressants, or anticoagulation, which ultimately leads to surgical resection. Surgery is often needed for definitive diagnosis and is usually curative, without recurrence after resection.

Contrast-enhanced CT features of IMHMV mimic those of ischemic colitis, with marked bowel wall thickening and bowel wall hypoenhancement [32]. Yun et al. [33] were the first, to our knowledge, to describe nonvisualization of the inferior mesenteric vein (IMV) at the level of the left renal vein on retrospective imaging review. Other CT features include pericolonic venous dilatation and occlusion, narrowing, or tapering of the peripheral mesenteric veins and/or IMV (Fig. 7). Occlusion of the IMV and peripheral mesenteric veins is often overlooked, and careful scrutiny of the venous system is necessary in patients with histologic findings suggesting colonic ischemia. Dilated pericolonic veins are often misinterpreted as engorged vasa recta, which are typically thinner and represent the comb sign commonly seen with inflammatory bowel disease. On CTA, early filling of dilated veins can be seen with IMHMV. The detection of these findings in a patient with histologic findings of colonic ischemia may allow the radiologist to first suggest the diagnosis given the nonspecific clinical and endoscopic findings and histopathologic challenges in diagnosis on mucosal biopsy.

Imaging Techniques

Radiography

Historically used as a screening tool in patients with acute abdominal pain, radiography is of limited utility in the diagnosis of MI. Imaging findings early in the disease course are nonspecif-

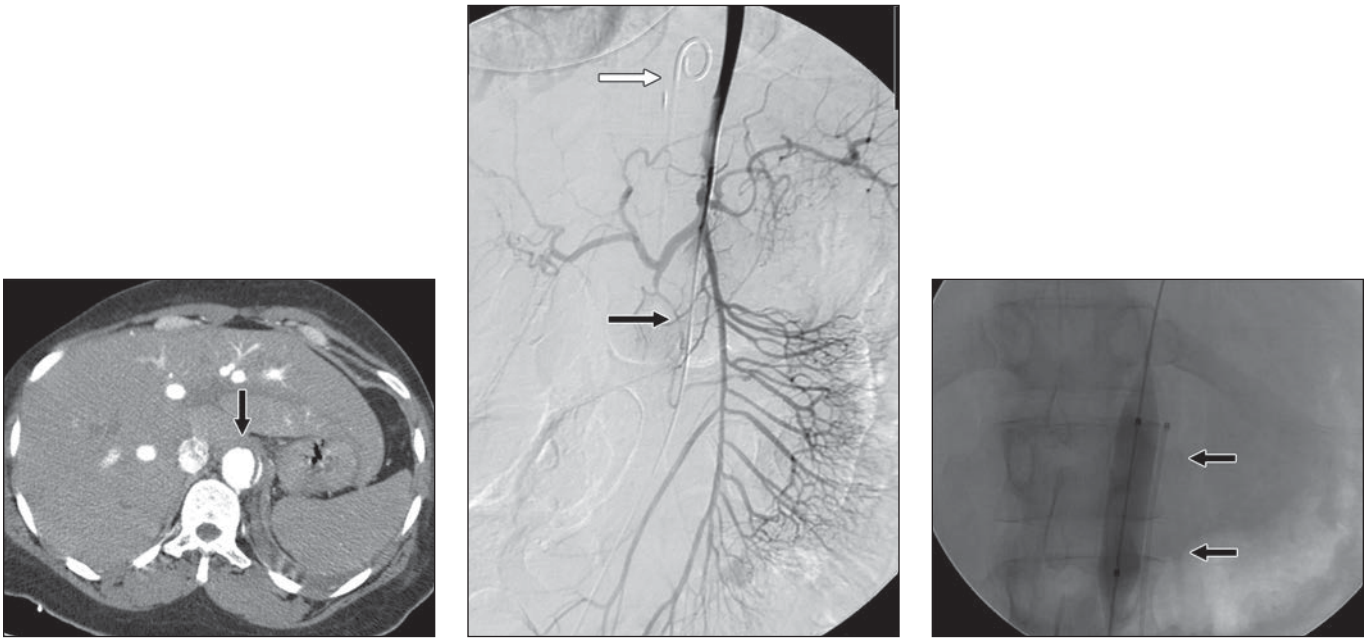

Fig. 8—72-year-old patient with abdominal pain secondary to type B aortic dissection.
A, Axial CT image shows type B aortic dissection and marked compression of true lumen by false lumen (*arrow*).
B, Catheter angiogram obtained after injection of contrast medium into true lumen shows diminutive size of true lumen, which supplies celiac axis and superior mesenteric artery (SMA). Gradient of 90 mm Hg was measured from false lumen to lumen of SMA. Note pigtail catheter in false lumen (*white arrow*) and straight flush catheter in true lumen (*black arrow*).
C, Fluoroscopic image shows 12-mm angioplasty balloon (*arrows*), which was used to perform fenestration between two lumens at level of SMA and reduced pressure gradient to 16 mm Hg. Patient reported rapid improvement in abdominal pain.

ic, ranging from a gasless abdomen to diffuse bowel dilatation characteristic of reflex ileus [19]. Late findings manifest as complications of transmural necrosis and bowel infarction and include portal venous gas, pneumatosis intestinalis, and free intraperitoneal air [2].

Ultrasound

Doppler ultrasound (US) has primarily been used for the diagnosis of CMI, with peak systolic flow velocities above 295 cm/s in the CA and above 240 cm/s in the SMA constituting hemodynamically significant (> 50%) stenosis [15, 19]. One small series reported that the detection of normal flow velocities in the CA and SMA on bedside US may be effective in excluding AMI, particularly the occlusive variety [34]. The use of contrast-enhanced US for evaluation of the mesenteric arteries remains an off-label application in the United States [35], though one investigation of 10 patients with surgically confirmed NOMI reported partial lack of bowel wall enhancement as a sensitive imaging feature [36]. Still, because US is highly operator dependent and may only be inconsistently capable of visualizing the mesenteric arteries secondary to factors such as shadowing from overlying bowel gas, dense vascular calcification, and patient body habitus, US remains a suboptimal imaging modality for the diagnosis of AMI [19].

Catheter Angiography

Historically, catheter angiography was used to diagnose the cause of AMI, but dramatic advances in CT have largely eliminated the need for these invasive procedures. Angiography is now reserved for potentially therapeutic procedures.

In select patients, either stent placement or catheter-directed thrombolytic therapy may be applied, but this should be done only in patients without signs of intestinal infarction [37].

MI can result from an aortic dissection extending into the proximal SMA or CA. However, hypoperfusion of the mesenteric vasculature may also result from compression of the lumen (usually the true lumen) from which patent mesenteric arteries arise. Cross-sectional imaging will show a slitlike true lumen perfusing the mesenteric vessel; if this finding is recognized, percutaneous intervention is a reasonable option. Catheter angiography is performed by catheterizing both lumens to obtain pressures in each. To improve perfusion to the bowel, a fenestration is performed at the level of the SMA and CA, equalizing the pressures in each lumen [38] (Fig. 8). This technique may stabilize patients with dissections and intestinal malperfusion, allowing elective repair of the aortic dissection or serving as definitive intervention [39].

Catheter-directed therapy is rarely indicated in patients with MVT. A patient may occasionally present with postprandial pain and have normal arterial inflow but also have hemodynamically significant narrowing of the SMV or portal vein. The bowel likely becomes congested in response to the increased arterial flow after eating, as the venous system cannot accommodate this increased flow. Transhepatic angioplasty and stent placement may be curative in this setting, although other causes for abdominal pain must be excluded (Fig. 9).

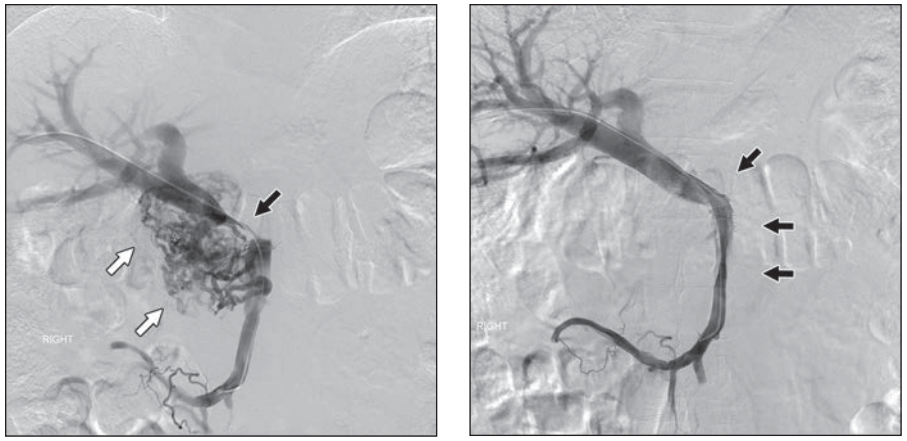

Fig. 9—63-year-old patient with locally advanced pancreatic adenocarcinoma and postprandial abdominal pain who was diagnosed with chronic mesenteric ischemia.
A, Venogram of superior mesenteric vein (SMV) shows severe stenosis near confluence (*black arrow*) with extensive collaterals (*white arrows*).
B, Venogram shows improved venous drainage of SMV (*arrows*) after self-expanding metal stent was dilated to 12 mm in diameter. Patient reported significant palliation of postprandial abdominal pain.

CT

Contrast-enhanced CTA has supplanted catheter-based angiography as the imaging modality of choice for the diagnosis of MI owing to its excellent spatial resolution and high sensitivity and specificity, both of which range from 90% to 100% [16]. Relative to catheter angiography, CTA has the added advantage of visualization of the entire abdomen and pelvis, enabling diagnosis of other potential causes of abdominal pain; one investigation encompassing nearly 1000 patients with a high clinical suspicion for MI reported a prevalence of AMI of just under 20% but noted that CTA was able to make a specific alternative diagnosis in over 60% of cases [40].

According to the American College of Radiology Appropriateness Criteria, the preferred protocol includes thin-section acquisitions in both arterial and venous phases. This facilitates production of multiplanar reformations, 3D renderings, and MIP images, all of which augment standard axial reconstructions in the diagnosis of MI [19]. Arterial phase images are obtained by bolus-tracking software when a standard aortic threshold is reached (typically 120–150 HU), with portal venous phase images subsequently acquired after a fixed 50- to 60-second delay [6, 16, 19] (Table 3). Historically, noncontrast sequences were attained before the injection of IV contrast medium, as early investigations reported that these images facilitated detection of high-attenuation intramural hemorrhage, atherosclerotic calcification, and assessment of bowel wall enhancement [41]. More recent series have questioned the necessity of this additional series, finding that its omission did not adversely affect diagnostic accuracy [19, 42]. Because AMI can rapidly prove fatal if left untreated, the benefits of IV contrast media outweigh any theoretic risk of contrast-induced nephropathy, and impaired renal function should not preclude its administration [16, 19].

In recent years, several groups have found that a split-bolus protocol, in which arterial and venous phase images are obtained concurrently, maintains high sensitivity and specificity for the diagnosis of MI while decreasing the contrast medium dose and generating fewer images for the radiologist to review [5, 43, 44]. In such protocols, an initial bolus of 100–150 mL of IV contrast medium is injected at a rate of 4–5 mL/s to generate portal venous phase images. This is followed by a second 50-mL contrast

TABLE 3: Sample CTA Protocol for Mesenteric Ischemia

Acquisition Parameter	Noncontrast Phase (Optional)	Arterial Phase	Portal Venous Phase
Patient position	Supine, arms above head	Supine, arms above head	Supine, arms above head
Anatomic coverage	Top of liver to pubic symphysis	Top of liver to pubic symphysis	Top of liver to pubic symphysis
Scan type	Helical	Helical	Helical
Rotation time (s)	0.5	0.5	0.5
Collimation	128 × 0.6	128 × 0.6	128 × 0.6
Pitch	0.6	0.6	0.6
Peak beam energy (kV)	120	120	120
Slice thickness (mm)	2	2	2
Increment (mm)	1.2	1.2	1.2
Acquisition timing	NA	Bolus-triggered (descending thoracic aorta, 2 cm above start location) at 150 HU	60 s after bolus injection
Contrast medium dose (mL)	NA	100 (140 if weight > 110 kg)	100 (140 if weight > 110 kg)
Contrast medium injection rate (mL/s)	NA	5 (6 if weight > 110 kg)	5 (6 if weight > 110 kg)

Note—NA = not applicable.

medium dose approximately 25–30 seconds later to optimize angiographic images [5]. This ultimately results in a single image stack in which both the arterial tree and mesenteric venous arcades are optimally opacified. Though multiphasic, compared with split-bolus, examinations were found to generate a higher mean attenuation in the SMA (≈ 337 vs 250 HU) [43], the two protocols showed no statistically significant difference in sensitivity, specificity, or diagnostic accuracy for AMI [5, 43]. Quantitative venous attenuation values were similar with both protocols, and though a split-bolus protocol increases contrast medium dose, no significant deleterious effects on renal function were observed [43]. The opacification of the arterial and venous systems in a single acquisition can reduce the number of images within the examination by 50% [43], lessening its complexity and potentially lowering the likelihood of significant interpretation errors [45, 46]. Reductions in patient dose may exceed 40% [43], a significant consideration considering AMI is suspected in younger patients [43, 47], without compromising the technical adequacy of the examination.

Dual-energy CT (DECT) has emerged over the past decade. In standard practice, most CT examinations are acquired at a tube voltage setting of 120 kV, resulting in a polychromatic beam with a mean energy of approximately 75 keV and a peak of 120 kVp. In comparison, DECT acquires data from two different energy levels simultaneously, resulting in two unique energy spectra. This enables creation of energy-specific datasets, including virtual noncontrast (VNC) images, iodine maps that underscore the accumulation of iodine in tissue, and monochromatic images that simulate a single beam energy [48, 49]. Monochromatic reconstructions at low energy (40–60 keV) approximate the k-edge of iodine and accentuate regions of differential enhancement [48], as the most specific indication of bowel ischemia is mural hypoenhancement, a frequently subtle finding that can be difficult to detect on conventional CT images [48]. After evaluation of conventional arterial or portal venous phase images, review of the monochromatic images and iodine maps generated as part of most DECT protocols may augment detection of differences in mural attenuation between closely adjacent loops of bowel.

Lourenco et al. [50] found that in patients with confirmed AMI, there was a 40% reduction in mural attenuation between ischemic and nonischemic segments on standard images, a difference that increased to 54% on reconstructed 40-keV images. On iodine maps, there was an even more marked difference in iodine concentration between ischemic segments and nonischemic segments, with the reduction in uptake measuring up to 65% in ischemic segments [50] (Fig. 10). Multiple additional investigations have found that the reconstructions provided by DECT increase the conspicuity of differences in enhancement between adjacent segments of bowel and facilitate detection of AMI [48, 50, 51].

VNC images can be reconstructed to determine whether increased mural attenuation within thickened segments of bowel on contrast-enhanced images is attributable to hemorrhage or enhancement [48]; high-attenuation material within the bowel wall that persists on VNC images is more likely to be hemorrhage (Fig. 10).

DECT can also be used to artificially increase IV contrast medium signal intensity on venous phase images, permitting adequate assessment of the arterial tree and potentially reducing the volume of IV contrast medium needed for an acquisition [48].

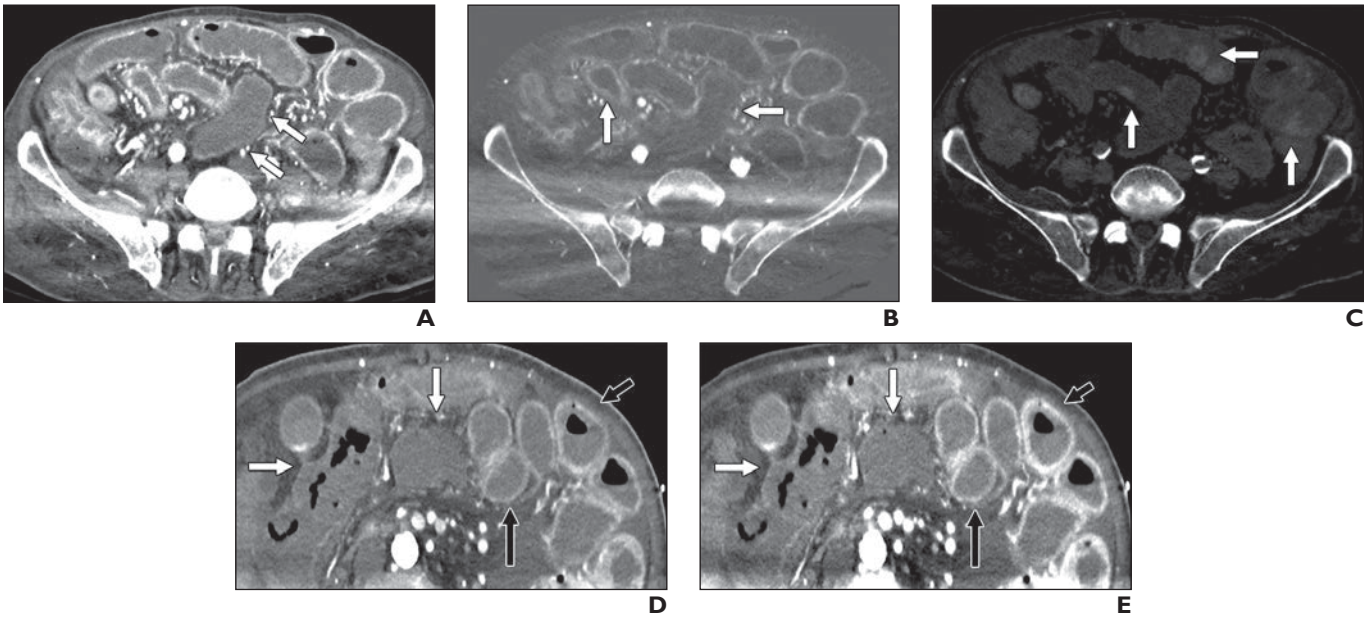

Fig. 10—82-year-old patient with nonocclusive mesenteric ischemia after cardiac surgery. Evaluation was performed by dual-energy CT.
A, Axial arterial phase CT image obtained at 50 keV shows hypoenhancing loop of small bowel in midabdomen (*arrows*).
B, Iodine-only arterial phase CT image obtained at 50 keV shows increased conspicuity of small-bowel hypoenhancement relative to adjacent loops of bowel (*arrows*).
C, Virtual noncontrast CT image obtained at same level as **A** and **B** shows intraluminal high-attenuation hemorrhage in multiple loops of small bowel (*arrows*).
D, Conventional linear blend arterial phase CT image shows small-bowel mural thickening, hypoenhancement, and ascites. Black arrows show enhancing, perfused loops of small bowel. White arrows highlight loops of poorly enhancing, ischemic bowel.
E, Dual-energy CT image obtained at 50 keV and at same level as **D** again shows small-bowel mural thickening and hypoenhancement. Images scanned at 50 keV, near k-edge of iodine, accentuate differences in bowel mural enhancement. Black arrows show enhancing, perfused loops of small bowel. White arrows highlight loops of poorly enhancing, ischemic bowel.

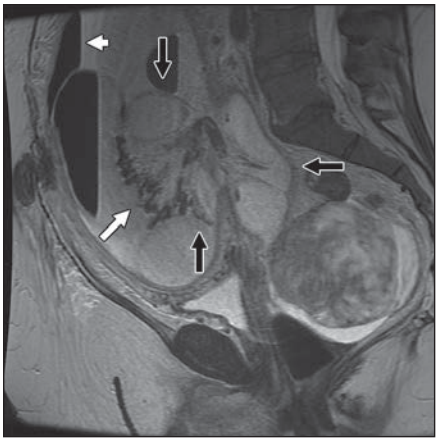

Fig. 11—47-year-old patient with abdominal pain and closed-loop small-bowel obstruction who was diagnosed with small-bowel ischemia. Sagittal MR image shows dilated fluid-filled small bowel radiating to central point (*black arrows*). Pneumatosis intestinalis (*long white arrow*), mesenteric venous gas, and pneumoperitoneum (*short white arrow*) are also shown.

Frequency-selective nonlinear blending, a postprocessing technique first described in 2016, allows separation of image information into low and high frequencies, with low frequencies containing information regarding image contrast and high frequencies encoding image noise [52]. As a result, a range of attenuations can be augmented within an image without increasing image noise [53]. One study found that the increased differences in attenuation between ischemic and nonischemic segments of bowel increased sensitivity for detection of MI [54]. This postprocessing can be applied independent of image acquisition technique, meaning that it may be useful in institutions in which DECT cannot be performed [53].

Furthermore, researchers have attempted to compare the quality of virtual monoenergetic images with those obtained at conventional single-energy CT. One group found that for abdominal CT, the iodine CNR of virtual monoenergetic images at 70 keV was comparable with that of single-energy scans performed at 120 kV but was less than that of single-energy scans performed at either 80 or 100 kV [55]. Thus, performing single-energy scans at lower kilovoltage in cases of suspected MI may result in better iodine CNR and enhance detection of subtle perfusion differences.

MRI

Because of its relatively low spatial resolution, longer image acquisition times, and difficulty visualizing secondary signs of bowel ischemia, MRI is primarily used for diagnosis of CMI, for which its sensitivity and specificity exceed 95% [14, 56]. In the late 1990s, pilot studies found that 2D time-resolved phase-contrast MRI (2D PC-MRI) showed promise as a potential diagnostic tool for the evaluation of CMI by quantifying rates of blood flow in the mesenteric vasculature before and after a meal challenge.

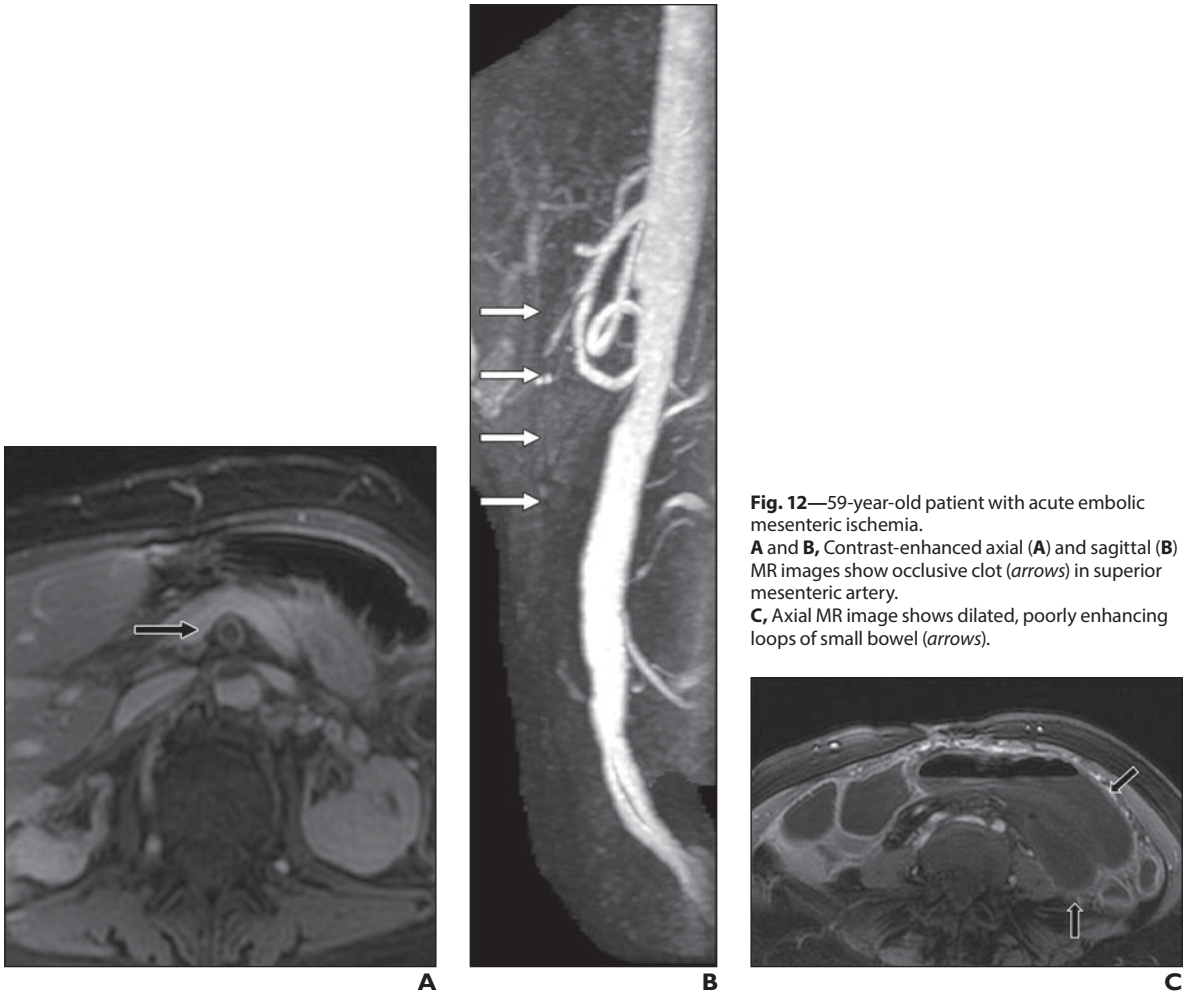

Fig. 12—59-year-old patient with acute embolic mesenteric ischemia.
A and **B,** Contrast-enhanced axial (**A**) and sagittal (**B**) MR images show occlusive clot (*arrows*) in superior mesenteric artery.
C, Axial MR image shows dilated, poorly enhancing loops of small bowel (*arrows*).

These investigations reported significant increases in flow within the SMA and portomesenteric venous system after a meal in healthy volunteers, with no or significantly reduced flow augmentation in patients with suspected CMI [57–59]. Because of long scan times, operator variability, and other implementation challenges, 2D PC-MRI was not widely adopted for assessment of MI. With recent advances in MRI technology, however, evidence is emerging that 4D flow MRI, which does not require the use of IV contrast medium and can be performed in under 10 minutes, may be a viable primary or adjunct diagnostic approach for CMI [56, 60]. This technique allows evaluation of both flow directionality and hemodynamic quantification. A recent investigation by Roberts et al. [61] showed that after a challenge meal, flow within the CA, SMA, SMV, and portal vein increased significantly in control patients and those without CMI, whereas no substantial flow changes occurred in those with CMI. Another emerging MRI technique in the imaging of MI is the use of ferumoxytol, a superparamagnetic iron oxide nanoparticle coated with a derivative of dextran. Administered IV, it serves as a blood pool contrast agent, enhancing image quality in 4D flow MRI. Its use allows high-resolution imaging of both the venous and arterial trees, permitting simultaneous assessment of venous and arterial causes of MI [60].

Though MRI is not commonly used for the diagnosis of MI, findings of ischemic bowel can sometimes be encountered on examinations performed for other purposes or in patients who cannot undergo CT, such as pregnant women (Fig. 11). Imaging findings are comparable with those seen on CT and include vascular filling defects (Fig. 12), bowel mural thickening and hypoenhancement, and evidence of transmural infarction or necrosis, such as pneumatosis, portomesenteric venous gas, and intraabdominal free air [62].

Conclusion

Because of the morbidity and mortality associated with MI, prompt clinical diagnosis and early implementation of therapeutic interventions are imperative to improve patient outcomes. CT has emerged as the first-line imaging modality for diagnosis, and novel imaging protocols and techniques have been developed to facilitate detection of MI's often insidious imaging features. A thorough understanding of the relevant vascular anatomy and the disease's characteristic imaging appearances may aid the radiologist in timely and accurate recognition of this often challenging condition.

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(Editorial Comment starts on next page)

Editorial Comment: Avoiding Pitfalls in the Diagnosis of Intestinal Ischemia

Diagnosing intestinal ischemia promptly is critical to initiate potentially life-saving intervention, and the radiologist plays a key role in making this diagnosis. Delay in the diagnosis is a source of patient morbidity, particularly when a CT scan is interpreted with inappropriate equivocation or when some of the supportive findings that would have prompted a timely intervention are missed. As emphasized in this work, CT is the first examination ordered in the evaluation of suspected bowel ischemia and in patients with acute abdominal symptoms even if bowel ischemia is not suspected.

When a CT arteriogram is ordered for suspected bowel ischemia, the addition of a portal venous phase is important. Of course, many times only routine contrast-enhanced CT is ordered, and only a venous phase image is obtained, which in fact can show all of the key findings. I also recommend trying the split-bolus protocol. When the findings are reported and a conclusion is rendered, comment can be made on which finding suggests a nonreversible infarction rather than just ischemia.

Of the myriad of findings in both acute and chronic bowel ischemia, those that I have seen often missed (or not mentioned in a report) include segmental absent bowel enhancement, near totally absent bowel enhancement, segmental diminished bowel enhancement, segmental hyperenhancement, small-volume free fluid in the mesentery (or a hernia sac), and partial superior mesenteric artery thrombus when distal to the origin or involving only jejunal branches. It is particularly important to develop a habit of interpreting the standard and maximum-intensity-projection coronal images carefully on every abdominal CT examination to assess bowel wall enhancement and the blood flow in the mesenteric arteries and veins. Dual-energy CT iodine maps can improve reader sensitivity for nonenhancing bowel wall.

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