SA–CME Information

An Overview of Acute Mesenteric Ischemia

Description

Acute mesenteric ischemia (AMI) is a true surgical emergency and a rare life-threatening condition, accounting for 0.01% of hospital admissions, with extremely high mortality rates (up to 69%). Poor outcomes remain prevalent despite advances in both diagnostic and treatment options over the last two decades

Early diagnosis and management are particularly important given that the highest incidence of AMI occurs in the elderly population, who often have multiple comorbidities leading to a worse prognosis. Biphasic contrast enhanced multidetector computed tomography (MDCT) images have become the mainstay and standard of care for investigation and timely diagnosis of AMI.

As such, the importance of recognizing imaging features of AMI and timely communication of findings with the referring physicians is of utmost importance for diagnostic radiologists and always a worthwhile topic for review. We have therefore endeavored to provide a brief summary of the presentation of AMI, its causes, relevant anatomy, and most importantly, illustrated review of CT findings that delineate ischemic changes of the bowel and mesentery.

Learning Objectives

Upon completing this activity, the reader should be able to:

- Explain the different pathophysiological processes that cause mesenteric ischemia;
- Identify the specific image findings related to mesenteric ischemia; and,
- Describe and explain the clinical management options for mesenteric ischemia.

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Disclosures: None.

Target Audience

- Radiologists
- Related Imaging Professionals

System Requirements

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Expiration date: **December 31, 2023**
Date of review: **December 13, 2023**
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An Overview of Acute Mesenteric Ischemia

Arash Mirrahimi, MD, MSc; Charlotte Gallienne, MD; Hournaz Ghandehari, MD, FRCPC

Acute mesenteric ischemia (AMI), a surgical emergency, is an uncommon but serious condition that accounts for 1 in 1000 hospital admissions in the United States and Europe[1] and has extremely high mortality rates, ranging between 50 and 69%.[2] Mortality remains high despite advances in diagnostic and treatment options, including the advent and widespread use of multidetector computed tomography (MDCT). This is thought to be the result of the vague nature of AMI's clinical presentation, poor correlation with biochemical markers, and resultant associated delays in diagnosis and management.[3]

Acute insufficiency of mesenteric blood flow accounts for 60-70% of cases of mesenteric ischemia[4], with the remainder related to chronic mesenteric ischemia, which is not a focus of this review. The major causes of AMI include mesenteric arterial occlusion (embolism or thrombosis), non-occlusive mesenteric ischemia due to intestinal hypoperfusion, and mesenteric venous occlusion (thrombosis).[5-7] Vasculitis is a rare risk factor for AMI, but this review will focus on the aforementioned etiologies.

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The incidence of AMI is increasing, owing partly to increased recognition and diagnosis, but more so to the increase in the aging population, with its cardiovascular and/or systemic diseases that further accentuate AMI risk. Early diagnosis and management are of utmost importance given AMI's prevalence in the elderly population, who often have worse prognoses due to comorbidities.

Biphasic MDCT has become the standard of care for investigating and diagnosing AMI, particularly given its easy availability in the emergency setting.[3] With its capacity to identify mesenteric ischemia, its severity, and potential causes, MDCT can have very high sensitivity and specificity.[8] The following review provides a summary of AMI, its causes and relevant anatomy, and an illustrated review of imaging findings.

Pathophysiology, Causes, and Anatomy of AMI

Development of intestinal ischemia depends on the following factors: systemic perfusion, number and caliber of affected vessels, available collateral circulation, and duration of the ischemic insult. Ischemic injury develops with prolonged mismatch between cellular metabolism and delivery of oxygen and nutrients. This mismatch can result from mesenteric arterial occlusion (MAO), mesenteric venous congestion/occlusion (MVO), or non-occlusive mesenteric ischemia (NOMI), as discussed below. In the gastrointestinal (GI) tract, mismatch with a 75% reduction in mesenteric blood flow can be tolerated for up to 12 hours without significant injury.[9]

It is also important to recognize that intestinal injury is caused by both tissue hypoxia and reperfusion. The latter occurs following blood flow restoration, with further damage caused by the release of free oxygen radicals, toxic ischemic injury byproducts, and neutrophil activation, which may lead to multisystem organ failure.[10,11] Alternatively, after a prolonged period of ischemia, progressive vasoconstriction is established in the obstructed vascular territory, which can persist even after blood flow returns, leading to persistent ischemia, full-thickness necrosis of the bowel wall, and perforation.[4,6]

Based on AMI pathophysiology; ie, MAO, NOMI, or MVO, three subtypes of ischemic change can be identified that are relevant to imaging findings (Figure 1):

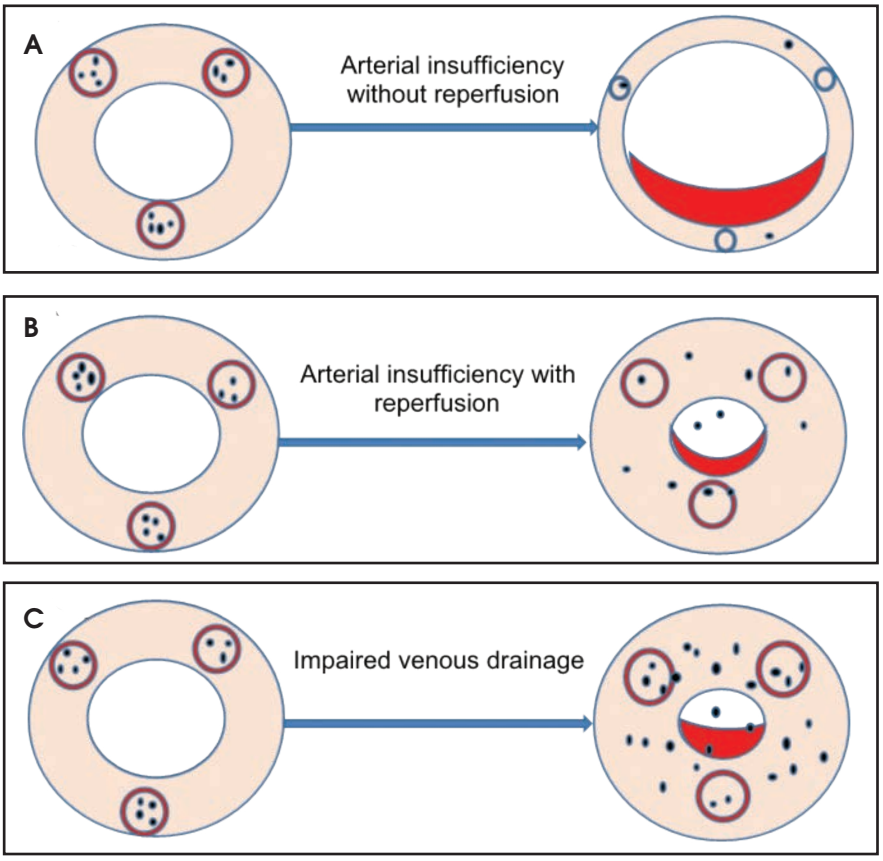

FIGURE 1. *Simplified sketch demonstrating cross-sectional bowel-wall changes in ischemia. The small ovoid circles represent red blood cells, and the red circles represent mural vessels. (A) Pale Ischemic Type due to persistent arterial insufficiency without reperfusion. The bowel wall gradually becomes thinned with depletion of intravascular blood with ensuing disruption of microvasculature and mucosa, leading to minimal residual blood extravasation into the bowel wall and the lumen. The lumen becomes distended with gas due to bacterial proliferations. (B) Hemorrhagic Type with transient arterial insufficiency with reperfusion. The bowel initially has low attenuation due to blood loss (not shown), and then once the blood supply is re-established, the re-perfused blood extravasates through the damaged vasculature and mucosa, causing mucosal and submucosal edema, enhancement or hemorrhage, and bloody intraluminal fluid. (C) Venous Type due to impaired venous drainage. The bowel wall becomes gradually thickened due to continuing leakage of the blood components (plasma, contrast materials, red blood cells) through the distended and increasingly permeable vascular wall into the bowel wall and lumen. Appearance is similar to (B), despite difference in mechanisms.*

1. Pale ischemic type resulting from persistent arterial insufficiency without reperfusion;
2. Hemorrhagic type resulting from transient arterial insufficiency with subsequent reperfusion and associated injury; and,
3. Venous type resulting from impaired venous drainage (most commonly seen as the mechanism for ischemia in setting of mesenteric strangulation or venous thrombosis).

Ischemic damage can range from reversible ischemia, including mucosal infarction and mural infarction involving the mucosa and submucosa, to irreversible transmural infarction and perforation. The bowel demonstrates spastic contraction in the early phase of the disease, followed by adynamic ileus caused by ischemic tissue damage, including muscular and neurologic infarction.

Mesenteric artery occlusions account for most AMI cases, which can be related to cardioembolism, arterial thrombosis, occlusive atherosclerosis, arterial dissection, post-vascular interventions (eg, iatrogenic embolic showers),[12] and, rarely, to vasculitides or fibromuscular dysplasia (Table).

Given this common etiology, knowledge of arterial anatomy and territorial supply to the GI tract is key to AMI diagnosis, causality, and treatment planning. The GI tract is supplied by three major arteries: the celiac artery, which provides blood to the stomach and duodenum; the superior mesenteric artery (SMA), which supplies the jejunum, ileum, and proximal colon to the splenic flexure; and the inferior mesenteric artery (IMA), which supplies the distal colon, from the splenic flexure to the rectum (Figure 2).

The most common site of MAOs from thromboembolic events is the SMA, owing to its large diameter and narrow takeoff angle that lead to anatomical susceptibility to lodging of emboli. This phenomenon often happens 3 to 10 cm distal to the origin of SMA, in a segment that tapers distal to the origin of the middle colic artery, thereby sparing the first few tributary branches to the jejunum. It is noteworthy, however, that in 15% of thromboembolic events the embolus is lodged at the origin of the SMA.[4] The middle segment of the jejunum is most often involved in the ischemic process as it is the farthest from the collateral circulation of the celiac axis and the inferior mesenteric artery, while the proximal jejunum is usually spared. The IMA is rarely affected by embolus due to its small caliber.[5]

The second most common etiology of AMI is NOMI. In this condition, the mesenteric vessels are patent but blood flow is very low, causing splanchnic vasoconstriction with subsequent multifocal infarction of solid and visceral organs. NOMI can therefore be challenging to diagnose and may result from shock (hemorrhagic, cardiogenic, or septic), heart failure (due to acute myocardial infarction or congestive heart failure), dehydration, stress (high-endurance athletes), chronic renal failure

Table. Causes of Mesenteric Arterial Occlusion

Causes	Source/etiology
Thromboembolism	Left atrium thrombus (in setting of atrial fibrillation)
	Left ventricle (in setting of myocardial infarction)
	Cardiac valves (due either to underlying atherosclerotic changes/valvular disease or, more commonly, to infective endocarditis)
	Proximal aorta (dislodged atherosclerotic plaque)
Arterial Thrombosis	Due to thrombosis of atherosclerotic plaque at stenotic sites
Occlusive	Usually chronic and secondary to underlying atherosclerotic disease
Dissection	Post-traumatic injury
	Related to vascular risk factors; eg, hypertension and atherosclerotic disease
Iatrogenic/ Intervention related	Embolic shower from manipulation of atherosclerotic vessels
	Dislodged stents
Other rare causes	Vasculitides or fibromuscular dysplasia

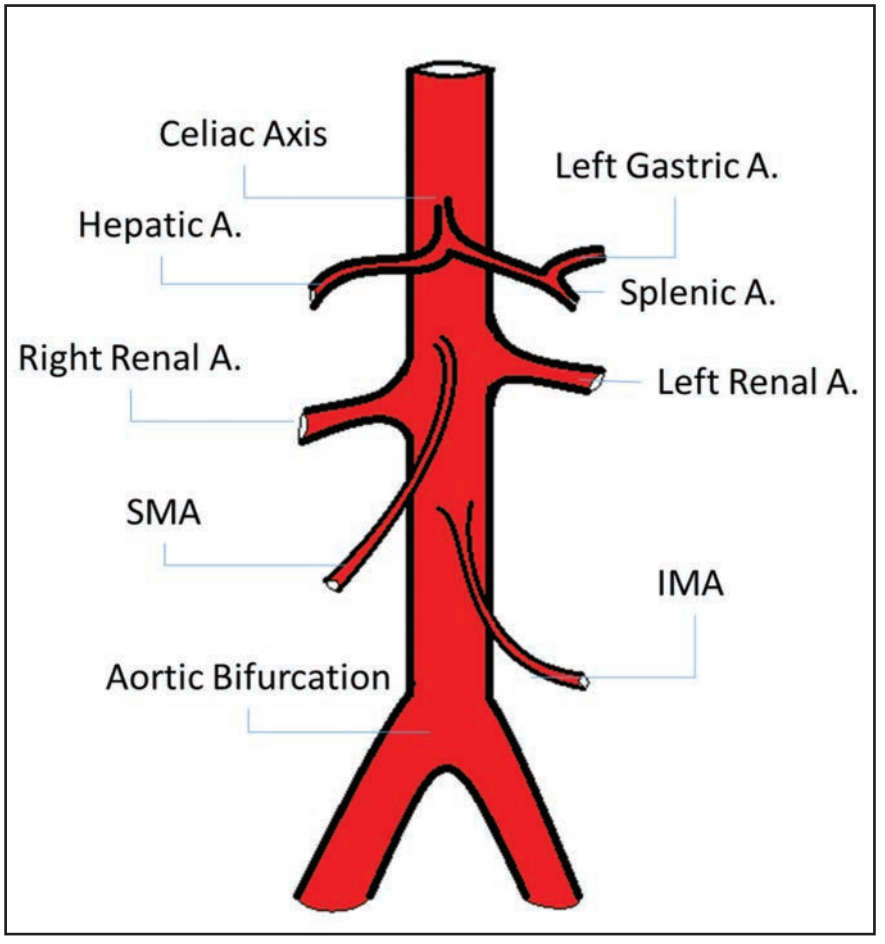

FIGURE 2. *Abdominal aorta anatomy.*

requiring hemodialysis,[13] or drugs, illicit and therapeutic; eg, cocaine, digitalis, ergot derivatives, and norepinephrine.[14] NOMI is often associated with the worst outcomes, reaching mortality rates of 58-70%.

The third-most prevalent cause of AMI is MVO, which results from venous congestion caused by mesenteric strangulation, venous thrombosis,[15] phlebitis of intramural veins (rarely), or overdistention.

Imaging Technique

Prior to widespread availability of MDCT, catheter angiographic studies were used in suspected cases of AMI to identify and treat the underlying etiology.[4] Today, MDCT is capable of diagnosing AMI, as well as potential other causes of "acute abdomen", with excellent sensitivity and specificity.[8] In a recent meta-analysis of six studies (3 prospective and 3 retrospective) with 142 positive AMI cases out of 619, MDCT was shown to have a pooled sensitivity and specificity of 93.3% and 95.9%, respectively, for AMI when analyzed by experienced radiologists.[8]

The optimal protocol for AMI detection is MDCT with unenhanced

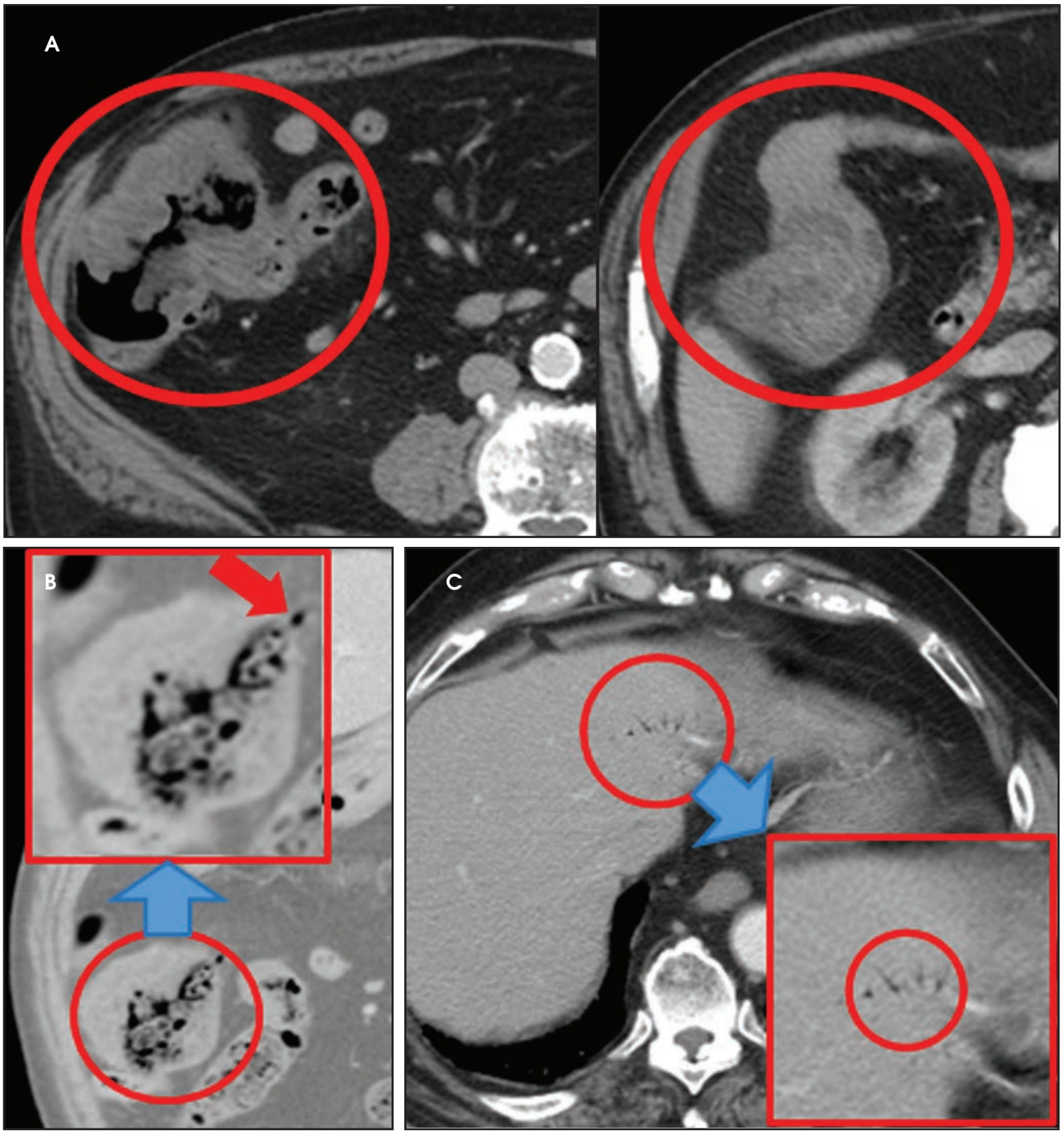

FIGURE 3. *83-year-old with right-sided abdominal pain. (A) Thick-walled hypoenhancing proximal colon, normalizing at hepatic flexure; (B) Extraluminal air suggestive of at least a microperforation; (C) Portal venous gas. Intraoperatively, there was a gangrenous cecum and proximal ascending colon.*

images followed by arterial and venous phases without oral contrast.[16] Between 100 and 150 mL of nonionic iodinated contrast material is administered at a rate of 2.5–4 mL per second, with a scanning delay of 30 seconds for the arterial phase and 60–70 seconds for the venous phase. Axial sections of 5-mm thickness are acquired with coronal and sagittal reformations. Bolus tracking is preferred, as follows: arterial phase scanning commencing 2 seconds after trigger threshold of 150 Hounsfield units (HU) reaches the supraceliac aorta, with the portal venous

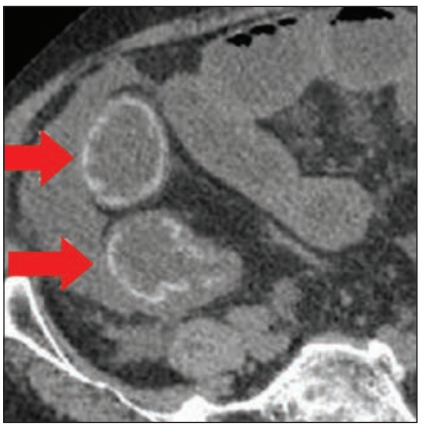

FIGURE 4. *Hyperattenuating bowel wall in keeping with hemorrhage (arrows) on unenhanced images*

phase scan acquired at a fixed delay of 45 seconds thereafter.

Imaging Findings and Management

Many MDCT findings can be suggestive of AMI. They differ on the basis of pathogenesis (veno-occlusive or arterio-occlusive), severity and stage, location (small bowel or large bowel), and presence of hemorrhage. The features with the highest positive predictive value are decreased or absent bowel-wall enhancement (Figure 3A), increased attenuation (intramural hemorrhage) of the bowel wall on unenhanced CT (Figure 4); and filling defect in vessels representing thrombus (Figure 5B). Filling defects may be hyper-attenuating on pre-contrast images. Particular attention should be paid to filling defects in the SMA (Figure 5B), IMA, SMV, and small mesenteric arteries, as well as to ancillary findings of ischemia in the liver, spleen, and kidneys (Figure 6) which are commonly overlooked.[17]

Other important but less specific MDCT findings include bowel-wall thickening (Figure 3A), the most common and least specific sign; dilated lumen with paper-thin wall, due to interruption of peristalsis reflex due to

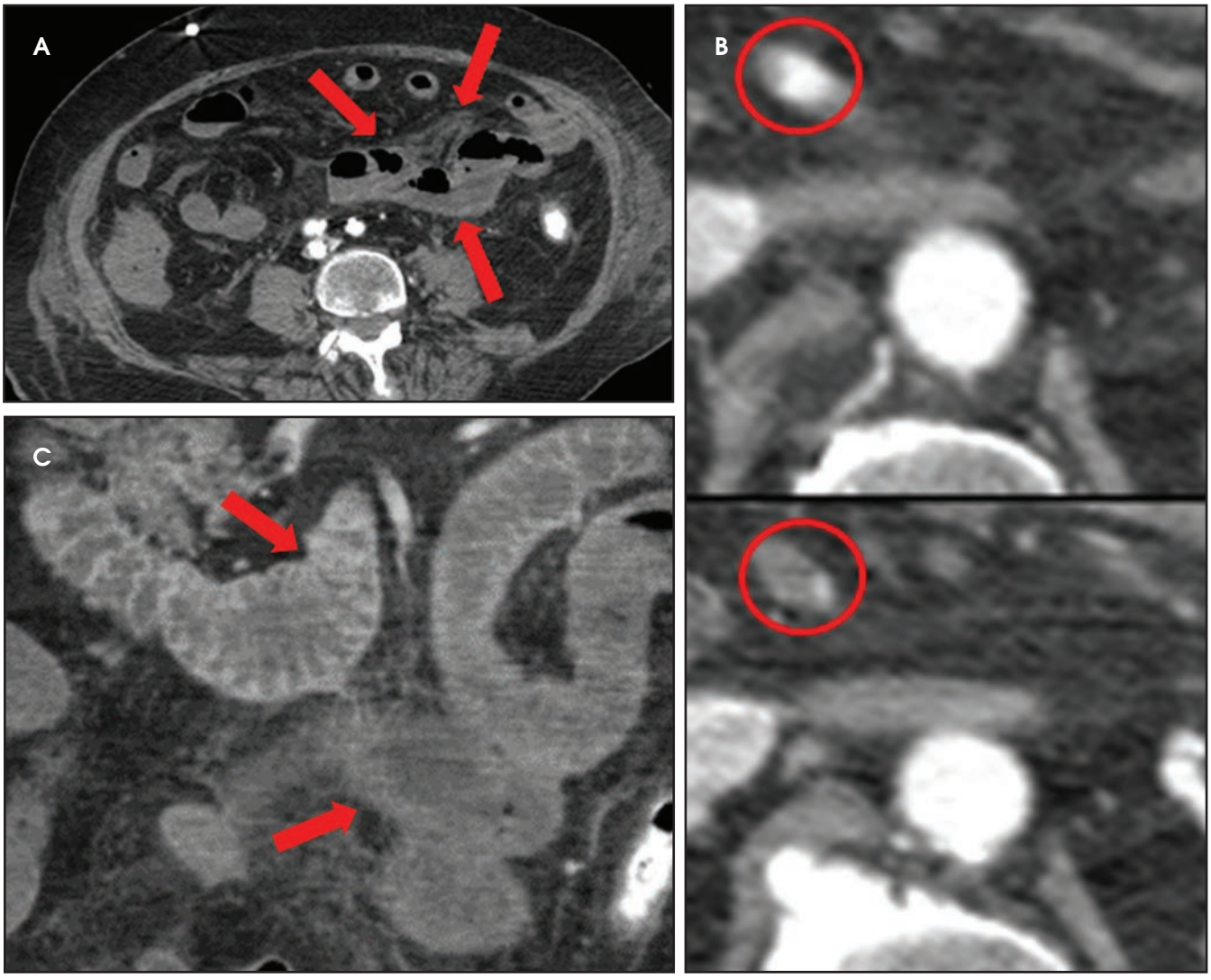

FIGURE 5. *80-year-old with diffuse abdominal pain 8 days post-cardiac intervention, with lactate level of 3. (A) Featureless and poorly enhancing bowel (arrows); (B) SMA thrombus; (C) differential enhancement of two segments of bowel (arrows) with associated surrounding mesenteric haziness/stranding. Intraoperative findings: nonviable bowel with extensive small- and large-bowel ischemia distal to ligament of Treitz.*

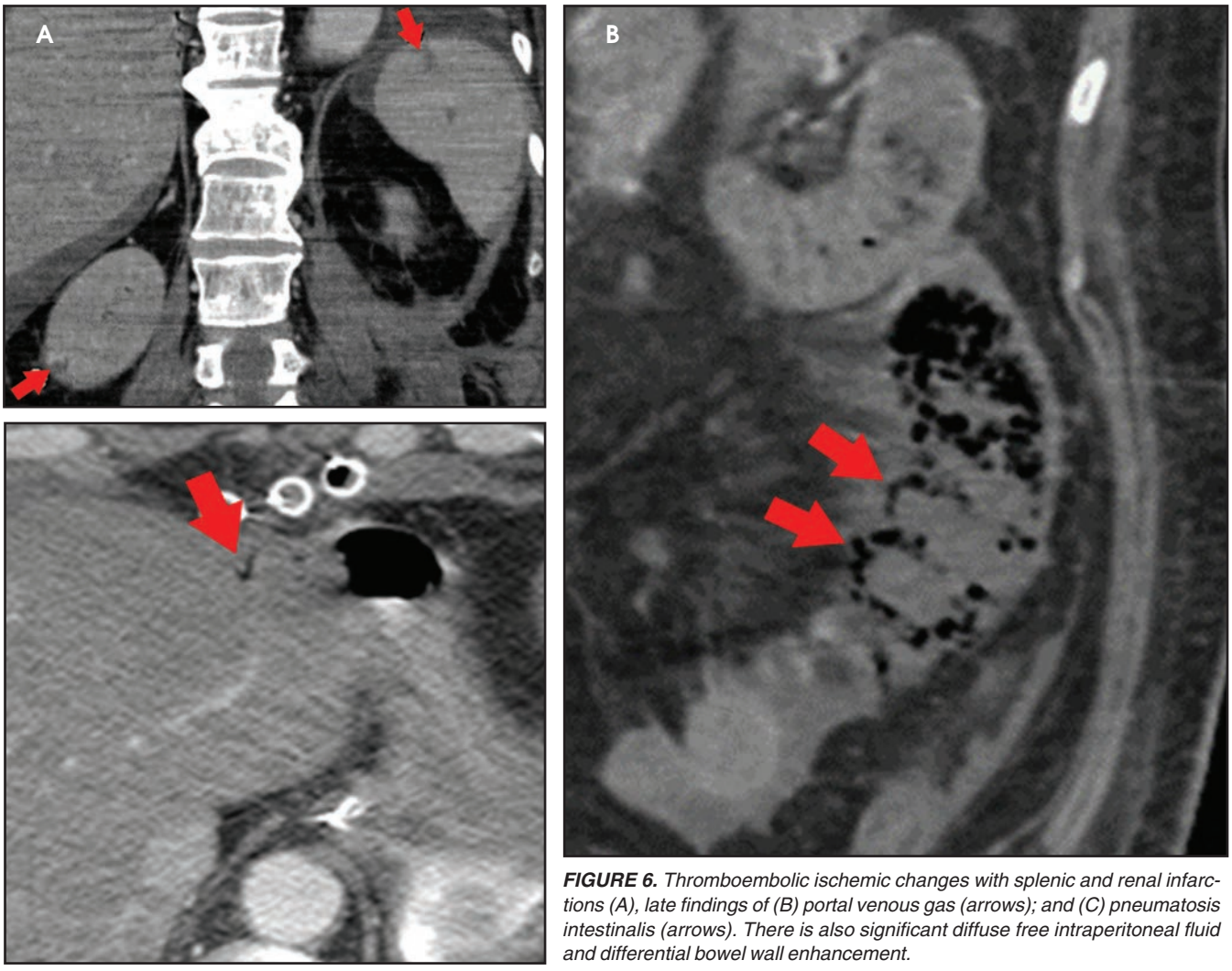

FIGURE 6. *Thromboembolic ischemic changes with splenic and renal infarctions (A), late findings of (B) portal venous gas (arrows); and (C) pneumatosis intestinalis (arrows). There is also significant diffuse free intraperitoneal fluid and differential bowel wall enhancement.*

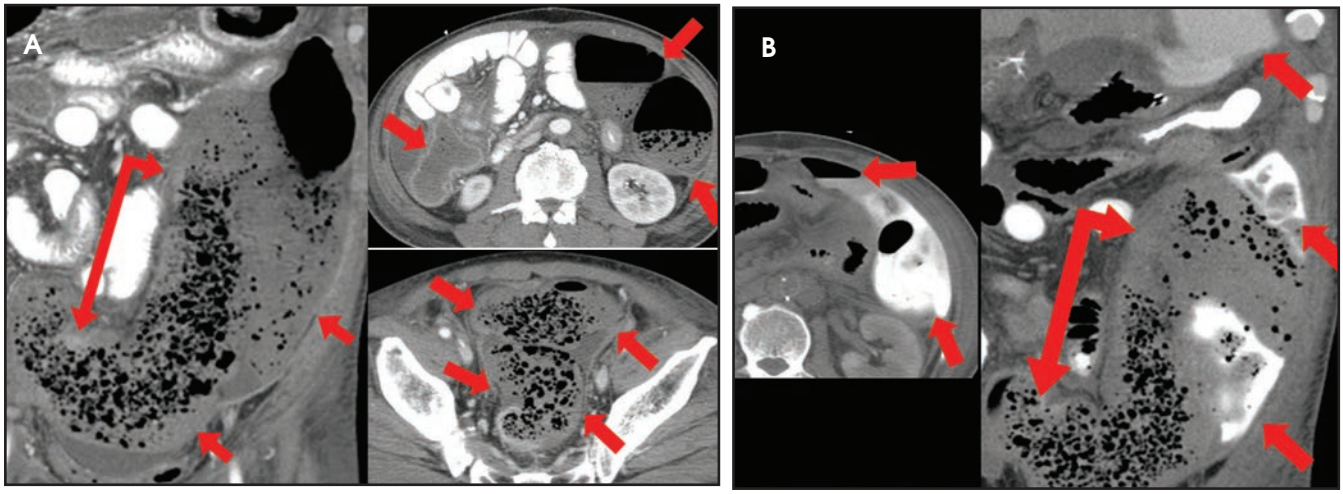

FIGURE 7. *69-year-old, one week post-aortobifemoral bypass graft and a lactate level of 5.2. (A) Dilated, featureless left colon with thin imperceptible wall and differential enhancement compared to right colon (arrows). (B) 24 hours later, repeat CT shows perforation with free air (arrow), extraluminal spillage of enteric contrast (arrows), and dilated, featureless left colon with non-enhancing wall (double arrow). Intraoperative findings: severely necrotic descending colon and sigmoid with bowel-wall liquefaction and area of abnormality involving the mid-transverse colon to rectum.*

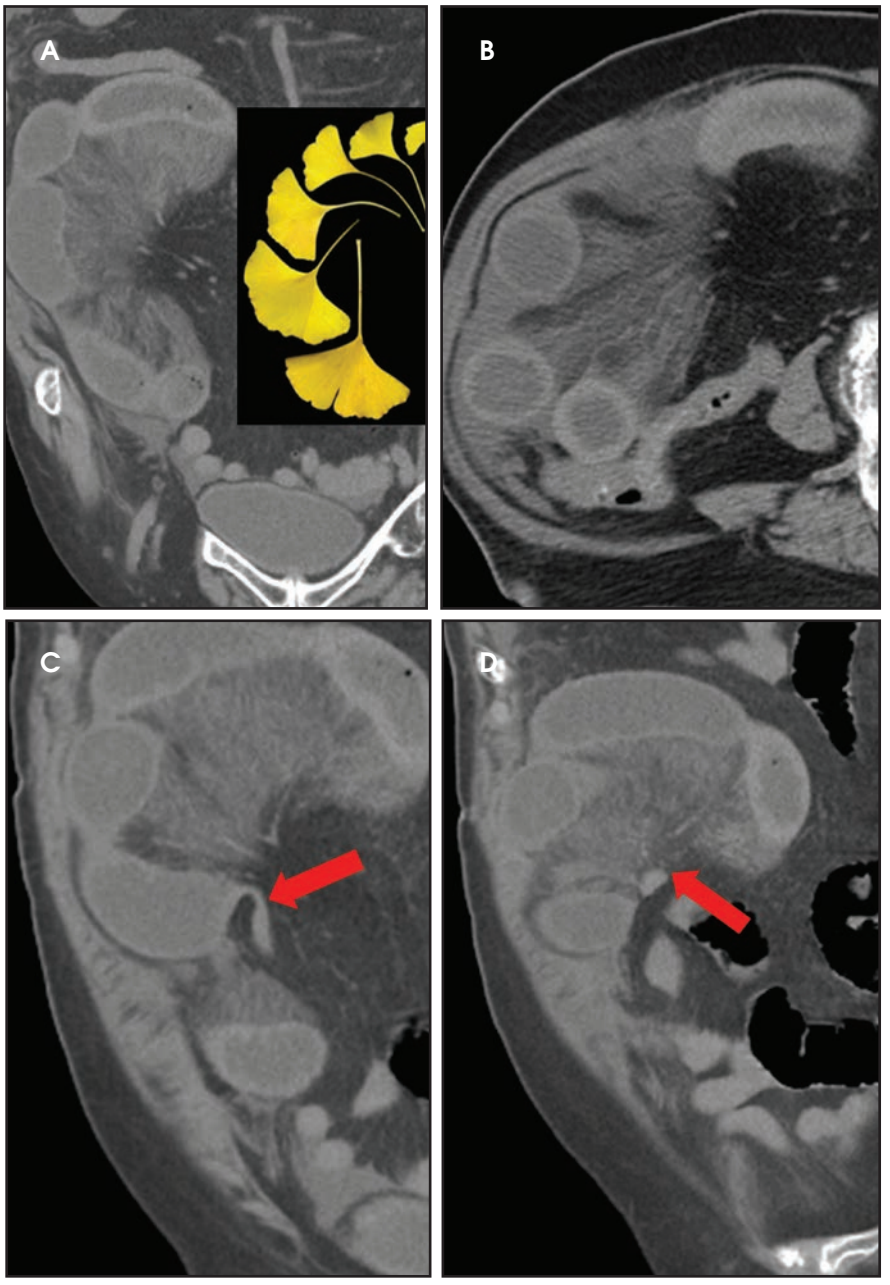

FIGURE 8. *89-year-old with abdominal pain, vomiting, and lactate level of 3.4. Small-bowel obstruction with "fan shaped segment" (A), and two adjacent transitions ("entering (C, white arrow) and exiting (D, red arrow) loops," in keeping with closed-loop obstruction. (B) Wall thickening, mesenteric congestion, and stranding concerning for ischemia. At surgery an ischemic segment with full-thickness necrosis was resected.*

ischemic injury and/or irreversible and transmural ischemic damage to bowel wall; mesenteric stranding, and free fluid. Late findings of AMI are ominous and often associated with worse outcomes and irreversible ischemia; these include extraluminal (free or focal), intravenous (mesenteric or portal venous), or mural (pneumatosis intestinalis) gas, which all indicate transmural injury with or without perforation (Figures 3,7). The etiologies of pneumatosis intestinalis (Figure 8) and portal venous gas in ischemic colitis are debated, but a popular theory suggests transmural ischemia and subsequent bacterial translocation. In cases of intestinal pneumatosis associated with suspected bowel ischemia, detection of hepatic portal or portomesenteric venous gas increases the likelihood of transmural bowel infarction (Figures 3,7, 8).

Characteristic Imaging Findings and Management for the Three Main Causes of AMI:

Imaging findings in arterial occlusion bowel ischemia include:

- A bowel wall that may be thinned and/or featureless (Figures 5,7), unchanged, or thickened (specifically in reperfusion) (Figure 3).
- Proximal occlusions, which cause greater injury (Figure 5).
- Increased bowel-wall attenuation in hemorrhagic infarction on unenhanced CT (Figure 4).
- Absent or diminished attenuation on contrast-enhanced CT (CECT, Figure 5), target appearance, or increased attenuation with reperfusion.
- Fat stranding and hazy mesentery in the setting of established ischemia (Figure 5).

Management of AMI resulting from arterial occlusion bowel includes anticoagulation, endovascular treatment (EVT) by an interventional radiologist, and surgical intervention, such as bowel resection.[3,18,19]

Veno-occlusive causes of AMI can appear similar to arterial occlusion, with bowel-wall thickening and variable attenuation on unenhanced CT (Figures 1,4, 9), absent or diminished attenuation on the contrast-enhanced phase or a target-sign appearance with reperfusion. Additional imaging findings include:

- Moderate or prominent dilatation of bowel with fluid retention.
- Bowel strangulation and obstruction (Figure 8).
- Portomesenteric venous thromboses with venous engorgement (Figure 9).
- Hazy mesentery and ascites resulting from increased back-pressure (Figure 8).

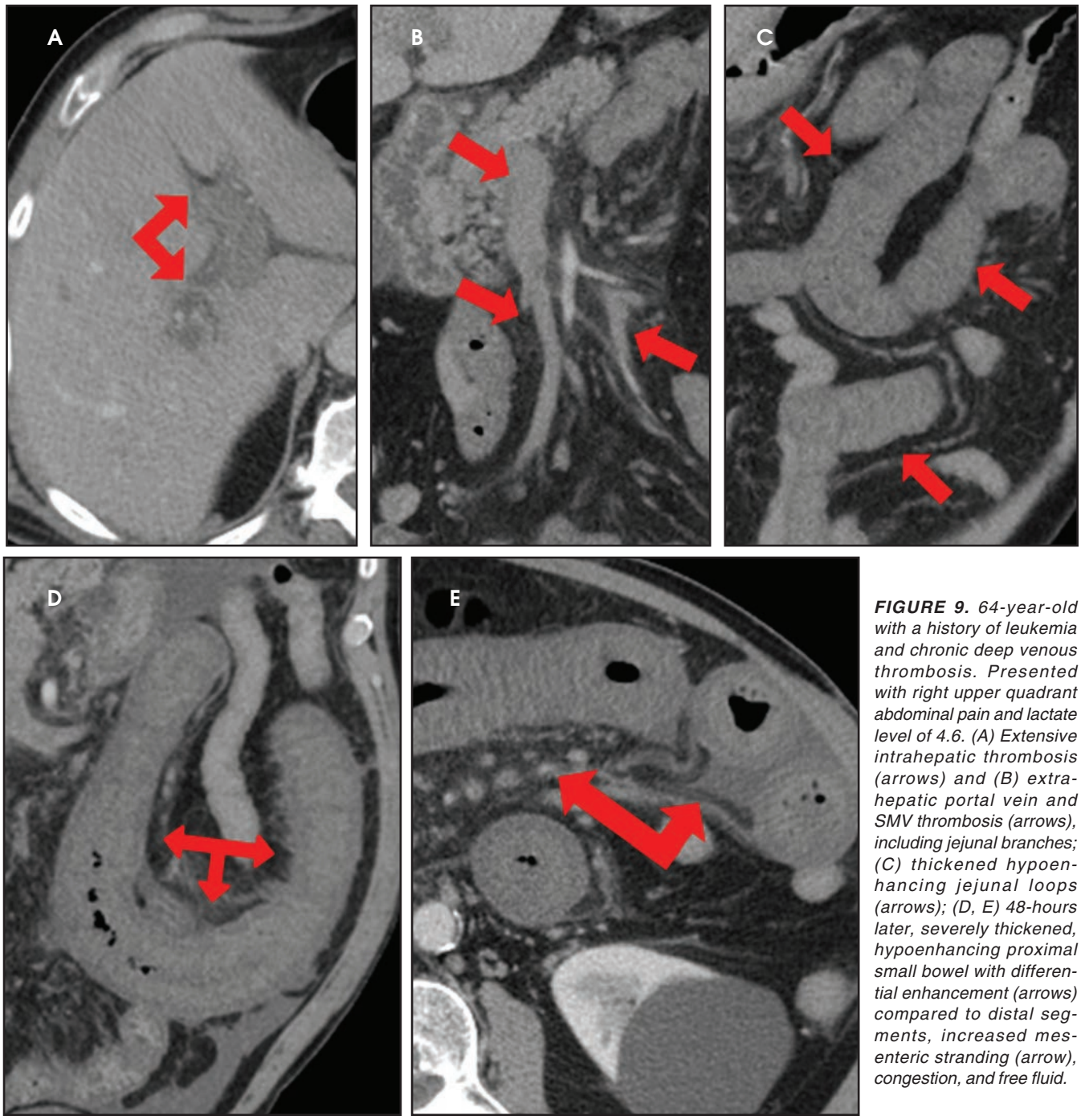

FIGURE 9. *64-year-old with a history of leukemia and chronic deep venous thrombosis. Presented with right upper quadrant abdominal pain and lactate level of 4.6. (A) Extensive intrahepatic thrombosis (arrows) and (B) extrahepatic portal vein and SMV thrombosis (arrows), including jejunal branches; (C) thickened hypoenhancing jejunal loops (arrows); (D, E) 48-hours later, severely thickened, hypoenhancing proximal small bowel with differential enhancement (arrows) compared to distal segments, increased mesenteric stranding (arrow), congestion, and free fluid.*

Systemic anticoagulation is the first-line therapy for veno-occlusive AMI;[20] however, rapid recanalization with systemic thrombolytic therapy and EVT may help improve outcomes in patients with large thrombotic burden and clinical deterioration.[21]

NOMI, the most difficult entity to identify early, is characterized by segmental and discontinuous involvement unrelated to a specific arterial territory. The most reliable early finding is differential enhancement, which is difficult to recognize owing to its discontinuous and segmental distribution. Treatment is often supportive, with attempts to reverse the insulting factors; eg, reversal of shock by addressing myocardial injury or hypovolemia. However, mesenteric contraction and vasospasm often persist, even after addressing precipitating events, and lead to worse prognosis.

Developing Technologies and Future Directions

Although CECT angiography has shown high sensitivity to acute bowel

ischemia,[8] the most common and readily recognized signs detailed above, including bowel-wall thickening, bowel dilatation, mesenteric stranding, and fluid, remain nonspecific. Conversely, bowel-wall hypoenhancement, the CT sign with the highest specificity (93–100%), is not as easily recognized.[22-26] Difficulty recognizing hypo-enhancement may be technique related; eg, the use of low energy levels, low volume/concentration of contrast agent, inadequate delay time or case related, as in the presence of intramural hemorrhage with high attenuation of the bowel wall on unenhanced CT or luminal distention with thinned bowel wall.

In recent years, strides made by dual-energy CT (DECT) technology toward improved recognition of differential bowel-wall enhancement have garnered interest.[16]DECT has been shown to help identify such differences more readily than conventional MDCT.[16,27] However, DECT is not widely available and remains in its infancy with respect to clinical application. Further studies are required to consolidate the degree of improved performance and specificity for bowel-wall hypoenhancement detection with DECT.

Conclusion

Acute mesenteric ischemia is a rare but life-threatening condition that requires timely recognition and intervention. The role of radiologists is pivotal to early identification of AMI; hence, considering AMI as a differential diagnosis when protocolling and identifying the subtle image findings detailed in this review remains vital to delivering the best patient care and outcomes.

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