Diagnosis of Acute Gastrointestinal Hemorrhage and Acute Mesenteric Ischemia in the Era of Multi-Detector Row CT

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KEYWORDS

• Gastrointestinal hemorrhage • Mesenteric ischemia • MDCT
• Bleeding • Bowel ischemia

ACUTE GASTROINTESTINAL HEMORRHAGE

Acute gastrointestinal (GI) hemorrhage is a commonly encountered symptom in both the primary care and emergency care settings. Acute GI hemorrhage is classified as upper or lower depending on the location of the source of the bleed in reference to the ligament of Treitz.[1] Upper GI hemorrhage is more common than lower GI hemorrhage. The annual incidence of upper GI hemorrhage ranges from 40 to 150 episodes per 100,000 persons, and the annual incidence of lower GI hemorrhage ranges from 20 to 27 episodes per 100,000 persons.[2]

Although most GI hemorrhages cease spontaneously, recurrent bleeding occurs in approximately 25% of patients.[3] Over the past 3 decades, the estimated overall mortality rate from acute GI hemorrhage decreased from 10% to 3%–5%.[4] Despite the improved survival for this common problem, mortality rates can reach up to 23% in the settings of massive hemorrhage or recurrent bleeding after hospital discharge.[5] A thorough understanding of the initial management of GI hemorrhage is important because prompt and aggressive diagnostic and therapeutic interventions are required. Managing acute GI hemorrhage involves 3 phases: resuscitation, diagnosis, and therapy. This section briefly discusses presentation, resuscitation, and therapy; a more detailed discussion of diagnosis ensues.

Presentation

The clinical presentation of GI hemorrhage depends on several factors, including the source of the hemorrhage, rate of hemorrhage, and amount of blood loss. Black or tarry stool or melena is associated with an upper GI hemorrhage. Often patients are unaware that this presentation reflects a GI source of bleeding. Consequently, patients may present later in the process with more severe symptoms, such as volume depletion. Although red rectal bleeding or hematochezia is associated with a lower GI hemorrhage and is more readily identified as alarming by patients, this symptom actually more accurately reflects the rate of blood loss and duration of blood within the GI tract. For example, a patient with a cecal hemorrhage and slow colonic transit may present with melena, whereas a patient with a brisk upper

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GI hemorrhage and rapid transit may present with hematochezia.[6,7]

Resuscitation

Volume depletion leads to decreased cardiac output that causes insufficient tissue oxygenation. In the acute setting, the complications from GI hemorrhage result form hypoperfusion rather than from the loss of hemoglobin. Thus, volume resuscitation is paramount. Intravenous (IV) access is essential. Clinicians should establish immediate IV access with large bore lines. Isotonic solutions are preferable. In most cases, the average adult can receive a bolus of 500 to 1000 mL without complications. Elderly patients or patients with heart disease may need more cautious hydration with a close evaluation of the fluid status. In the acute setting, fluid overload is less dangerous than volume depletion. Although transfusions are often necessary, hematocrit and hemoglobin levels are poor reflections of blood loss. Decisions to transfuse should be individualized. Ongoing monitoring is essential during the stabilizing period.[4]

Diagnosis

The differential diagnosis of acute GI hemorrhage is wide; **Box 1** outlines the more common causes. Multiple diagnostic modalities are used to confirm the presence of, and to establish the cause of, an acute GI hemorrhage.

Box 1
Differential diagnoses of GI hemorrhage

Common causes of upper GI hemorrhage
- Duodenal/gastric ulcers or erosions
- Esophageal varices
- Mallory-Weiss tears

Common causes of lower GI hemorrhage
- Diverticular disease
- Colonic neoplasm
- Ulcerative or ischemic colitis
- Angiodysplasia

Less common causes of GI hemorrhage
- Esophagitis
- Crohn's disease
- Radiation colitis
- Hemorrhoids

Data from Lee EW, Laberge JM. Differential diagnosis of gastrointestinal bleeding. Tech Vasc Interv Radiol 2004;7:112–22.

Endoscopy

Esophagogastroduodenoscopy (EGD) and colonoscopy remain the first-line diagnostic modalities for upper and lower GI hemorrhage.[8] Endoscopic evaluation is relatively safe. It allows direct visualization of hemorrhagic foci within the upper GI tract or colon and distal ileum. Many investigators report the sensitivity and specificity of EGD for GI hemorrhage between 92% and 98% and 30% and 100%, respectively.[9,10] Endoscopy can also serve as the means for therapeutic interventions in many patients with acute GI hemorrhage. The main limitation of EGD is the very poor visualization of the distal duodenum and the inability to evaluate the majority of the jejunum and the ileum. Complications are relatively low among experienced operators; however, perforation, aspiration pneumonia, and hemorrhage are known risks.[9] Endoscopy relays prognostic information as well. Variceal hemorrhage is associated with a mortality rate of 30%, much higher than other causes. The characteristics of ulcerations are used to estimate the likelihood of recurrent hemorrhage. A visible vessel, sentinel clot, or oozing ulcer is more likely to rebleed (**Fig. 1**). If the ulcer base is clean, only an estimated 10% rebleed.[4] Investigators have reported successful management of GI hemorrhage with injection therapy, thermal coagulation, and laser therapy.[11–13] An important limitation of emergency colonoscopy in the setting of acute GI hemorrhage is that blood clots and fecal material

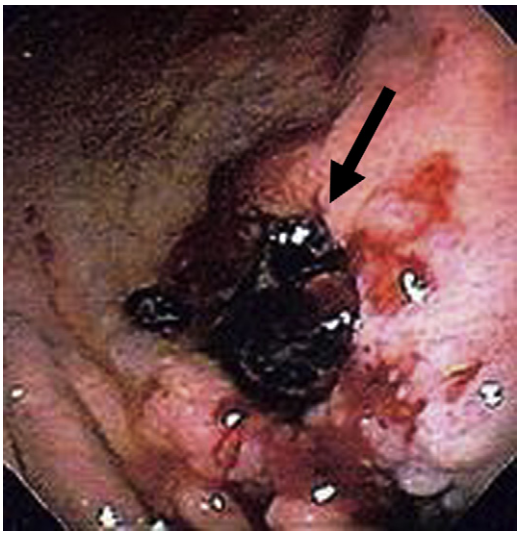

Fig. 1. Endoscopic view of GI mucosa demonstrates fresh blood at the base of the ulceration (*arrow*).

may obscure visualization of hemorrhagic sites in unprepared patients.

Capsule Endoscopy

Capsule endoscopy has a reported sensitivity varying between 42% and 80% among patients with obscure intestinal bleeding (negative upper and lower endoscopic examination results). Capsule endoscopy is more appropriately used in a select group of patients with obscure GI bleeding (no source identified on upper and lower endoscopic evaluation) assumed to originate from small bowel. This modality is time consuming and not appropriate in the acute care setting. In addition, it only visualizes mucosal surfaces. Capsule retention is the major known risk (**Fig. 2**), and up to 20% of examinations are incomplete.[14]

Diagnostic Imaging

In the setting of acute GI hemorrhage, abdominal radiographs seldom yield useful information and should be avoided because they delay diagnosis and treatment.

Nuclear Imaging

Technetium 99m (Tc 99m)-labeled red blood cell (RBC) or Tc 99m sulfur colloid scintigraphy can detect and localize GI hemorrhage (**Fig. 3**). Tc 99m RBC is 93% sensitive and 95% specific for detecting active GI hemorrhage at rates as low as 0.2 mL/min. Uptake of radiolabeled colloid by the liver and spleen minimizes the application in upper GI hemorrhage. Compared with Tc-99m sulfur colloid, labeled RBC scintigraphy allows patient evaluation for 24 hours after injection to detect rebleeding. However, scintigraphy is a time-consuming method with only limited sensitivity. The high false-positive localization rate (approximately 22%) limits the diagnostic value of this modality.[15,16]

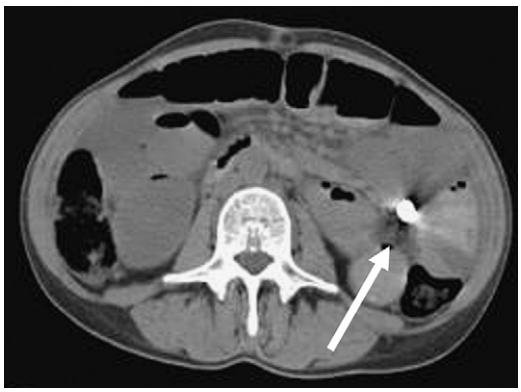

Fig. 2. Retained capsule. CT demonstrates a high-density metallic artifact (*arrow*) in the in the left upper quadrant in a patient who recently underwent capsule endoscopy. After 4 days, the capsule did not pass.

Mesenteric Angiography

Angiography detects GI hemorrhages greater than 0.5 mL/min (**Fig. 4**). Scintigraphy can help guide selective contrast injections. Therapeutic intervention via embolization is a potential advantage of this diagnostic tool. However, angiography is only 40% to 86% sensitive.[17] Complications include groin hematomas, vascular injuries at puncture sites, and distal embolization and occur in up to 2.2% of patients.[18]

Multidetector Computed Tomography

Over the past decade, multidetector computed tomography (MDCT) angiography emerged as a promising first-line modality for the time-efficient evaluation of GI hemorrhage. CT scanners are readily available in most acute care settings. The examination can be quickly performed with reproducible results and minimal invasiveness. MDCT is sensitive and accurately diagnoses or excludes active GI hemorrhage (**Figs. 5** and **6**). A meta-analysis published in 2010 demonstrated a pooled sensitivity of 89% and specificity of 85%. The initial experience of MDCT suggests the strong diagnostic capability of this modality.[19] Investigators report detection of hemorrhage with MDCT in animal models at a rate of 0.3 mL/min.[20] In addition, MDCT often diagnoses hemorrhagic foci when angiography fails to identify the source.[21,22] MDCT can detect hemorrhagic foci within the small bowel unlike upper or lower endoscopy. MDCT risks include ionizing radiation dose, IV contrast allergy, and contrast-induced nephropathy.

MDCT Techniques

The techniques and phases vary among institutions and individual radiologists. One example is provided. Images are acquired with the following parameters: detector configuration, 64 $\times$ 0.625 mm; section thickness, 0.9 mm; section increment, 0.45 mm; 120 kV; 405 mA; pitch, 0.923; and rotation time, 0.75 seconds. One hundred milliliters of IV contrast material is delivered at 4 mL/s. Automatic bolus-triggering software with use of a circular region of interest is placed on the abdominal aorta at the level of the diaphragm with a trigger threshold of 150 HU. Twenty-five seconds after the bolus trigger, data acquisition commences from the xyphoid process through the pubic symphysis. Continuous 3-mm axial, sagittal, and coronal sections are generated and

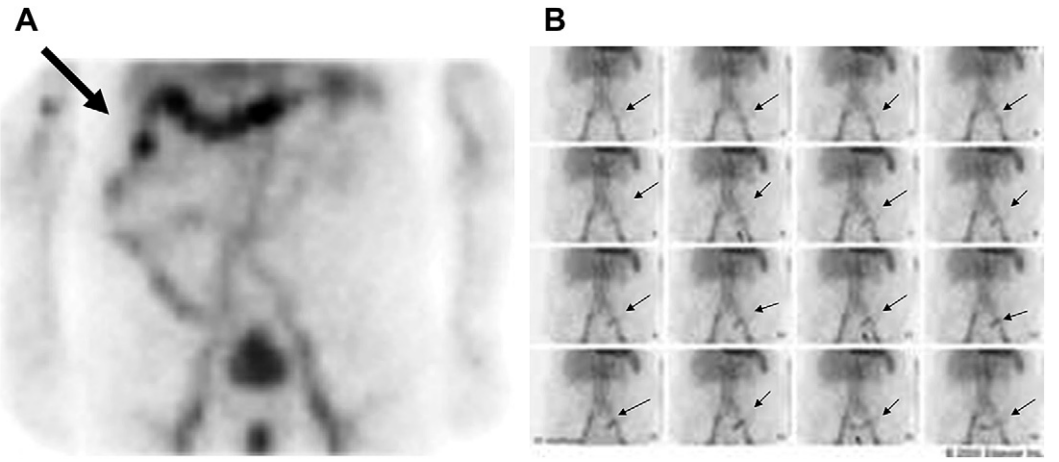

Fig. 3. (*A*) Tc-99m–labeled RBC image demonstrates increased radiotracer activity in the right and transverse colon consistent with acute GI hemorrhage (*arrow*). (*B*) Tc-99m–labeled bleeding scan demonstrates increased radiotracer activity in the sigmoid colon consistent with acute GI hemorrhage (*arrows*).

sent to picture archiving and communication system (PACS) for interpretation.

An unenhanced computed tomographic (CT) scan may be obtained immediately before CT angiography to identify any preexisting hyperattenuating areas within the bowel lumen that could be confused with hemorrhage at CT angiography. The technical parameters used to acquire the unenhanced data are as follows: detector configuration, 64 × 0.625 mm; section thickness, 3 mm; section increment, 3 mm; 120 kV; 150 mA; pitch, 0.891; and rotation time, 0.5 seconds. Contiguous 3-mm axial sections are then reconstructed and transferred to the PACS with CT angiographic data for interpretation.

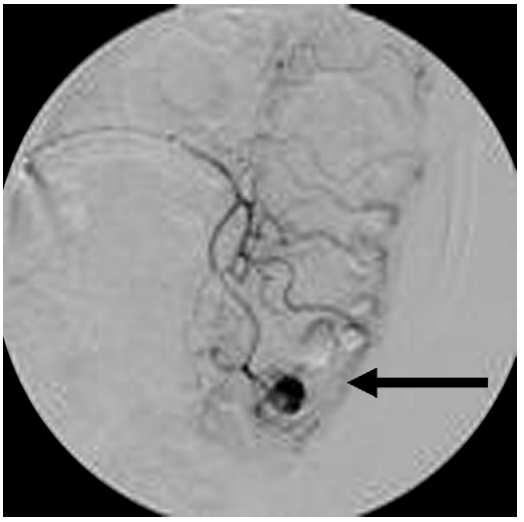

Fig. 4. Selective angiographic evaluation of the rectosigmoid region demonstrates pooling of contrast consistent with active colonic hemorrhage (*arrow*).

MDCT Findings

The increased spatial and temporal resolution provided by newer-generation MDCT technology, depicts active extravasation of IV contrast during the arterial and portal venous phases. The diagnosis of active GI bleed is made when hyperattenuating extravasated contrast material is identified within the bowel lumen. The extravasated contrast material may appear as jetlike, swirled, linear, ellipsoid, or pooled hyperattenuating foci or may fill the entire bowel lumen, resulting in a hyperattenuating loop. Varied approaches exist in evaluating patients with acute GI bleeds with MDCT. Some investigators apply attenuation thresholds as criteria for the diagnosis of acute bleeding.[23,24] Other investigators prefer to compare sequentially acquired unenhanced CT scans and CT angiograms without adherence to attenuation analysis. Most acutely extravasated contrast material into the bowel lumen will exceed 90 HU. It is conceivable that small amounts of bleeding may not reach this threshold because of volume averaging. Comparison with unenhanced images distinguishes active hemorrhage from other high-attenuation material within the GI tract. However, acquisition of both unenhanced and enhanced images increases the overall radiation dose in the patient. In either case, care must be taken to distinguish intraluminal contrast material extravasation from mucosal enhancement.

Therapy

As noted earlier, endoscopy and mesenteric angiography allow for both the detection and treatment of GI hemorrhage. Benefits, limitations, and risks are discussed.

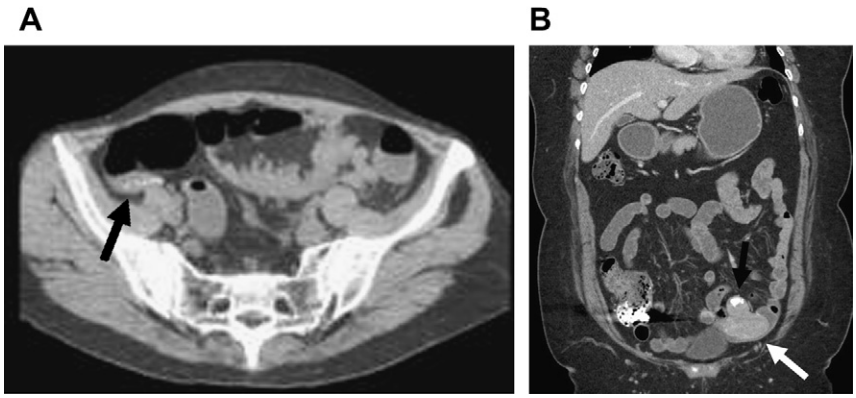

Fig. 5. (*A*) Delayed postcontrast image through the pelvis demonstrate pooling contrast in the right lower quadrant (*black arrow*) in a patient with bloody stools and anemia. Findings correlated to focus of hemorrhage at endoscopy. (*B*) Gastrointestinal hemorrhage. Coronal reformatted CT image through the bowel with focal high-attenuation focus in the sigmoid colon (*black arrow*). Intermediate-density hemorrhagic material pools in the colonic lumen (*white arrow*).

Summary

Acute GI hemorrhage commonly presents in the acute care setting. Potentially dire consequences necessitate rapid resuscitation, identification of the bleeding source, and treatment. Historically, the first-line approach used endoscopy. The advantages include the ability to both identify and treat the hemorrhagic source. The radiologic imaging choices in this setting include nuclear scintigraphy, angiography, and MDCT angiography. The previous sections outlined the strengths and weaknesses of these modalities. Although CT is not widely used as a first-line modality for the evaluation of acute GI hemorrhage, it is gaining popularity. CT is a noninvasive, rapid, readily available tool that accurately identifies GI hemorrhage and can provide useful information to guide subsequent therapeutic intervention.

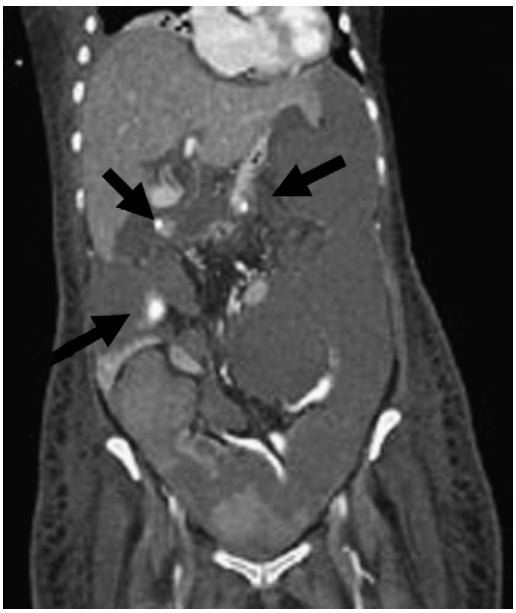

Fig. 6. Coronal reformatted image of the abdomen after IV contrast administration in a patient with clinical infectious (pseudomembranous) colitis. Focal high densities (*arrows*) in the right colon are consistent with active colonic hemorrhages.

ACUTE MESENTERIC ISCHEMIA

Although bowel ischemia is a complex disease with many manifestations, it is essentially caused by a significant reduction in the blood supply to the mesenteric circulation. Acute bowel ischemia represents one of the most dangerous abdominal conditions presenting in the acute care setting.[25,26] Investigators suggest that only 1% of acute abdomen hospitalizations are secondary to acute bowel ischemia.[27] Although acute bowel ischemia is an uncommonly encountered entity in the emergency department, the increase in average life expectancy of the population and the associated increase in vascular disease may lead to more frequent encounters in clinical practice.[28]

Despite the recognition and growing interest in this entity, identification and early diagnosis is challenging. Early diagnosis is essential because the consequences for acute bowel ischemia that progresses to bowel infarction are associated with extremely high mortality. In various case series, the mortality rate ranges from 60% to 90%.[29,30] In the mid-twentieth century, very few tools existed to diagnose this condition.[31] Patients were generally surgically explored, or the

diagnosis was made at autopsy. Over the past 60 years, the development of imaging technology expanded diagnostic possibilities.[32]

Radiographic evaluation of acute bowel ischemia in the initial phases is not optimal. At best, the findings in advanced stages are nonspecific and may represent several other entities with varying degrees of severity. Angiography provides the possibility to both identify the vascular lesion and to treat it; however, this service is often unavailable in the immediate acute care setting. In addition, angiography remains an invasive, labor-intensive, and expensive screening tool. CT is gaining popularity in evaluating patients suspected of acute bowel ischemia. Advances in CT imaging have improved the sensitivity and specificity of this modality. Investigators claim 92% specificity and 64% sensitivity.[28]

Etiology

The causes of mesenteric ischemia can be divided into 2 categories. In the overwhelming majority (approximately 80% of cases), the cause is secondary to occlusive or thromboembolic sources.[28] The remaining 20% are secondary to nonocclusive states (**Box 2**).

Box 2
Causes of acute mesenteric ischemia

Mesenteric arterial occlusion
- Thrombosis
- Embolism
- Dissection
- Stent placement
- Vasculitis
- Small vessel disease

Mesenteric venous occlusion
- Thrombosis
- Phlebitis
- Bowel obstruction or strangulation

Nonocclusive ischemia
- Shock
- Cardiac dysfunction
- Cardiac bypass
- Dehydration
- Vasoconstrictive drugs

Data from Wiesner W, Khurana B, Hoon J, et al. CT of acute bowel ischemia. Radiology 2003;226:635–50.

Occlusive Mesenteric Ischemia

Intestinal ischemia secondary to occlusive causes is due to blockage of either the arterial or venous system. The vast majorities of occlusions are arterial and may be either embolic or thrombotic in nature. Up to 50% of mesenteric infarctions are secondary to embolic obstruction of the superior mesenteric artery. These patients usually have cardiac arrhythmias such as atrial fibrillation or valvular vegetative disease that predisposes to arterial thrombi. In the remaining 50% of cases of bowel ischemia, arterial thrombosis causes ischemia. These patients usually suffer from atherosclerotic disease and develop atheromas at the level of the splanchnic arteries. Progressive narrowing of the lumen leads to mesenteric ischemia.

Venous thrombosis is responsible for about 10% of cases of mesenteric ischemia. Venous occlusion leads to blood stasis, decreased capillary circulation, and diminished cellular oxygen exchange. The ensuing cascade includes hemorrhagic distention of the intestinal wall, necrosis, and perforation. Venous ischemia and infarction are commonly associated with closed-loop obstruction. In rare cases, hypercoagulable states and vasculitis cause mesenteric ischemia.

Nonocclusive Mesenteric Ischemia

Nonocclusive causes account for approximately 20% of mesenteric ischemia. In this setting, the arterial and venous systems are patent, but the common pathway of drastically diminished perfusion exists for different reasons. Hypovolemic shock, cardiac failure, severe anemia, neurogenic vasodilatation, or splanchnic vasoconstriction can cause nonocclusive mesenteric ischemia and, if not corrected, lead to mesenteric infarction.

Anatomy, Physiology, and Pathology

Three main arteries supply the small bowel and colon. The celiac trunk, superior mesenteric artery, and inferior mesenteric artery arise from the abdominal aorta. The celiac trunk supplies the GI tract from the distal esophagus to the descending duodenum. The superior mesenteric artery supplies the distal duodenum, jejunum, ileum, and colon, generally to the splenic flexure. The inferior mesenteric artery supplies the colon from the splenic flexure to the rectum. Several rich vascular safety nets in the form of anastomoses exist in the mesenteric circulation. The gastroduodenal artery, usually the first branch of the common hepatic artery, connects the celiac trunk and the superior mesenteric artery. The marginal

artery of Drummond and the arcade of Riolan provide important collateral pathways between the superior mesenteric artery and the inferior mesenteric artery. The anastomotic connections between the inferior mesenteric artery and the lumbar branches of the aorta, sacral artery, and internal iliac arteries form up to 4 arcades. Several peripheral mesenteric vascular circuits run in series and in parallel. The 3 main parallel circuits supply the muscularis propria, submucosa, and mucosa. The 5 series vascular circuits include the arterioles, capillary complexes, and venous complexes. The superior mesenteric and inferior mesenteric veins run parallel to their similarly named arteries and drain the bowel in a similar manner. The inferior mesenteric vein flows into the splenic vein that joins the superior mesenteric vein to form the portal vein. A network of shared collateral pathways connects the mesenteric and systemic venous drainage allowing gastric and esophageal connections to communicate with renal, lumbar, and pelvic veins.[33,34] In cases of acute occlusions, collateral pathways provide essential means for perfusion and drainage. Distal vessels provide end organ perfusion and have fewer associated collateral pathways. Distal occlusions can be more damaging for this reason. Similarly, fewer patent collateral pathways exist in diffuse occlusive disease states.

Under normal circumstances, the mesenteric circulation receives approximately 25% of the cardiac output, of which two-thirds supplies the intestinal mucosa. These levels vary depending on physiologic needs. During periods of extreme stress, such as occurs with the fight-or-flight phenomenon, as little as 10% of the cardiac output may be diverted to the bowel. After a large carbohydrate meal, the mesentery may receive up to 35% of the cardiac output.[35] Under normal conditions, local and systemic autoregulation ensure adequate perfusion. In cases of hypovolemia, systemic autoregulation overrules the local mechanism in an attempt to protect the brain and heart.

The initial ischemic damage to the mesentery results in mild or superficial necrosis involving the mucosa. If ischemia is not corrected, the necrosis extends to submucosa. If ischemia continues, necrosis of the deep submucosal and muscular layers occurs. Fibrotic strictures secondary to local reparative changes may develop. In severe cases, partial mural bowel ischemia progresses to transmural bowel wall necrosis. This degree of infarction is a surgical emergency because it is associated with high mortality rates. Edema and hemorrhage follow partial-thickness necrosis. The inflammatory response that follows releases several mediators, such as cytokines and platelet-activating factor, into the mesenteric circulation. These mediators damage the already susceptible bowel. The mucosal barrier weakens. The bowel is more prone to bacterial invasion that can lead to bacterial enteritis or colitis that can lead to sepsis. Bleeding, intestinal perforation, abscess formation, and peritonitis can occur.[36]

CT of Mesenteric Ischemia

The changes at the cellular level contribute to the CT appearance of a mesentery during acute ischemic episodes.[32] **Box 3** outlines some of the common CT findings associated with mesenteric ischemia. Acute bowel ischemia may present as focal or diffuse, segmental or focal, and superficial or transmural changes. It can mimic many other mesenteric conditions encountered in the acute care setting. A high level of suspicion must exist.

Bowel Wall Thickening

Bowel wall thickening is the most commonly cited finding in acute mesenteric ischemia (**Fig. 7**). Investigations report bowel wall thickening in 26% to 96% of cases. Although the thickness of the normal bowel wall depends on the degree of distention and the presence of spasmodic contractions, ranges from 3 to 5 mm are generally accepted. Wall thickening is secondary to mural edema, hemorrhage, and/or superinfection of the bowel. However, bowel wall thickening is the least specific CT finding of mesenteric ischemia, as it is observed in a host of nonischemic entities affecting the bowel.

Dilatation

Bowel lumen dilation is associated with mesenteric ischemia in 56% to 91% of reported cases. Interruption of normal intestinal peristalsis is a direct result of ischemic damage to the bowel wall (**Fig. 8**).

Box 3
CT findings in mesenteric ischemia

- Bowel wall thickening
- Bowel dilation
- Abnormal or absent wall enhancement after IV contrast administration
- Mesenteric stranding/ascites
- Vascular engorgement
- Pneumatosis
- Portal venous gas

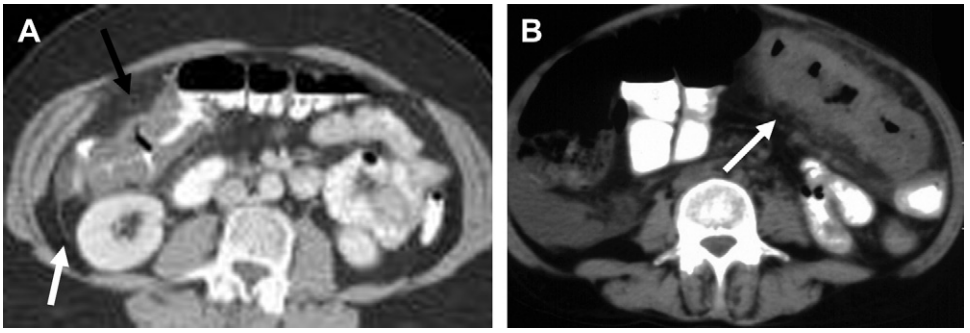

Fig. 7. Mesenteric ischemia. (*A*) Focal thickening of the ascending colon and a portion of the transverse colon (*black arrow*) spares the left colon. Minimal mesenteric stranding is noted along the right conal fascia (*white arrow*). (*B*) Focally thickened transverse colon involving the transverse colon and splenic flexure (*arrow*). Ischemic changes continued into descending colon.

Abnormal Enhancement

Ischemic bowel may demonstrate hypoenhancing or hyperenhancing walls. Poorly enhancing walls are usually homogenous secondary to wall edema. Hyperattenuating walls are associated with intramural hemorrhage or increased enhancement secondary to hyperemia or hyperperfusion.

Mesenteric Stranding/Ascites

Standing of the mesenteric fat, mesenteric fluid, and ascites are nonspecific CT findings of mesenteric ischemia. The presence of these findings depends on the cause of the ischemia, location, and severity. Investigators report the sensitivity and specificity of these 3 factors as 58%, 88%, and 75% and 79%, 90%, and 76%, respectively. If 2 of the 3 findings are present, the specificity increases to 94%.

Venous Engorgement

In cases of mesenteric venous occlusion and subsequent outflow obstruction, venous engorgement may be identified.

Pneumatosis and Portal Venous Gas

Pneumatosis and portomesenteric venous gas are less common but more specific findings of acute mesenteric ischemia. Investigators report these findings between 6% and 28% and 3% and 14%, respectively. Pneumatosis and portomesenteric venous gas may be focally or diffusely present (**Fig. 9**). When present in the liver, portomesenteric venous gas distributes to the periphery of the liver (**Fig. 10**).

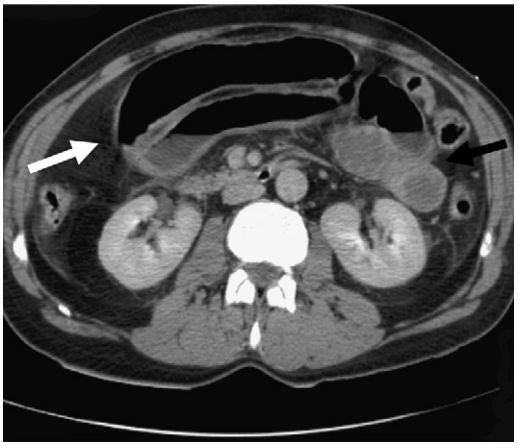

Fig. 8. Mesenteric ischemia. Ischemia affects normal peristalsis. Multiple loops of dilated small bowel (*white arrow*) and thickened loops of small bowel (*black arrow*) are noted. Mesenteric stranding is present.

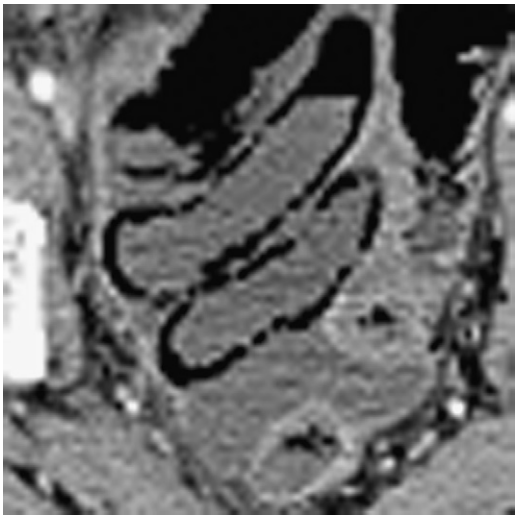

Fig. 9. Diffuse pneumatosis. Coronal reformatted post contrast image of the small bowel demonstrates diffuse pneumatosis.

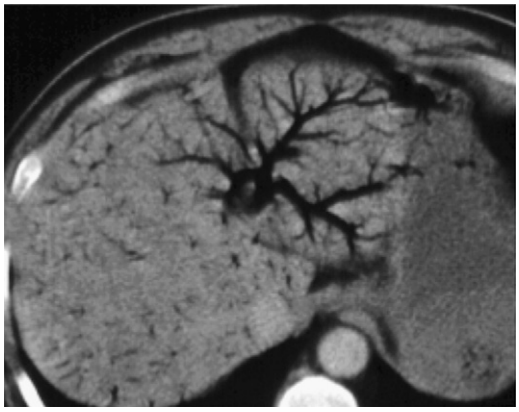

Fig. 10. Portal venous gas. Portal venous gas extends to the periphery of the liver.

SUMMARY

Although mesenteric ischemia is not one of the most commonly encountered entities in the acute care setting, it is associated with a high mortality rate. Thus, rapid and accurate diagnosis is imperative. The variable clinical and radiologic presentations and appearances of mesenteric ischemia pose a challenge to both clinicians and radiologists. Strong clinical-imaging communication and correlation is important to detect, diagnose, and treat this entity in the appropriate time course. CT is becoming an important tool in the evaluation of patients with mesenteric ischemia in the acute care setting. CT can not only aid in the rapid evaluation and diagnosis of mesenteric ischemia but also evaluate other abdominal conditions that may present in a similar manner. As the technology continues to mature, that is, higher speed, improved resolution, and multiplanar/multidimensional reformatting, CT is poised to play an increasingly important role in evaluating patients with suspected mesenteric ischemia.[37]

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