Imaging in Intestinal Ischemic Disorders

Richard M. Gore, MD\textsuperscript{a,}*\textsuperscript{a}, Vahid Yaghmai, MD\textsuperscript{b}, Kiran H. Thakrar, MD\textsuperscript{a}, Jonathan W. Berlin, MD\textsuperscript{b}, Uday K. Mehta, MD\textsuperscript{b}, Geraldine M. Newmark, MD\textsuperscript{b}, Frank H. Miller, MD\textsuperscript{b}

Gastrointestinal tract ischemia and infarction are a heterogeneous group of disorders (Tables 1 and 2) that have as their unifying theme hypoxia of the small bowel or colon. Ischemic bowel disease results from acute or chronic insufficiency of blood flow to the gut and includes acute and chronic small bowel and colonic ischemia (CI) in addition to infarction. Vascular compromise of the gut is a complex multifaceted condition that depends on the (1) state of the systemic circulation, (2) degree of functional or anatomic vascular compromise, (3) number and caliber of vessels affected, (4) response of the vascular bed to diminished perfusion, (5) nature and capacity of the collateral circulation, (6) duration of the ischemic insult, and (7) metabolic requirements of the involved segment of bowel.\textsuperscript{1–12}

Patients who have intestinal ischemic disorders most often present with abdominal pain and other nonspecific symptoms, such as nausea, vomiting, diarrhea, and bloating. The diagnosis of mesenteric ischemia (MI) is often one of exclusion after more common possibilities, including bowel obstruction, appendicitis, diverticulitis, cholecystitis, peptic ulcer disease, and gastroenteritis, have been excluded. Accordingly, a high index of clinical and radiologic suspicion is required to make a timely diagnosis of ischemia and infarction of the gut.\textsuperscript{1–12}

Gastrointestinal tract ischemia can threaten bowel viability with potentially catastrophic consequences, including intestinal necrosis and gangrene. Dramatic improvements in cross-sectional imaging have the potential to afford earlier and more precise diagnosis, which is key to the reducing the morbidity and mortality of this potentially fatal condition.\textsuperscript{1–12}

**EPIDEMIOLOGY**

Vascular compromise of the gut is responsible for approximately 0.1% of all hospital admissions and 1.0% of admissions for an acute abdomen. The diagnosis of this disorder is on the increase for several reasons. MI and infarction occur predominantly in the geriatric population with comorbid cardiovascular disease and other systemic dysfunction. The population is aging, and the number of cases of MI is expected to increase dramatically as the “Baby Boom” generation comes of age. Other factors include improved diagnostic techniques, heightened awareness of this diagnosis, and the efficacy of intensive care units to salvage critically ill patients.\textsuperscript{13–28}

The causes of MI are protean (Boxes 1–3). Bowel ischemia most commonly occurs within the sixth and seventh decades of life. The age of onset depends on patient gender and the etiology of the ischemia. Primary mesenteric venous thrombosis (MVT) and nonocclusive mesenteric ischemia (NOMI) present at the ages of 66.5 and 63 years, respectively.\textsuperscript{8} Superior mesenteric artery (SMA) occlusion and nonprimary MVT present nearly a decade later, at the ages of 77.5 and

\textsuperscript{a} Department of Radiology, Evanston Northwestern Healthcare, Northwestern University Medical School, 2650 Ridge Avenue, Evanston, IL 60201, USA

\textsuperscript{b} Department of Radiology, Northwestern Memorial Hospital, Northwestern University Medical School, 676 St. Clair Street, Chicago, IL 60611, USA

* Corresponding author.

E-mail address: rmgore1953@aol.com (R.M. Gore).

doi:10.1016/j.rcl.2008.05.004
0033-8389/08/$ – see front matter © 2008 Elsevier Inc. All rights reserved.
Chronic mesenteric ischemia (CMI) presents in younger patients than those with acute mesenteric ischemia (AMI), with a female prevalence as high as 4:1. This is in contrast to the overwhelming prevalence of men with peripheral vascular and aneurysmal disease. Generally, women present with ischemia at an earlier age than their male counterparts, which may contribute to the overall earlier age of onset of CMI.

Prompt diagnosis of MI is facilitated by recognizing the various risk factors and comorbidities. Although there is significant overlap among the risk factors for the various vascular disorders of the intestines, certain etiology-specific risk factors have been described.

AMI has been linked to congestive heart failure, valvular heart disease, cardiac arrhythmias, low cardiac output states, recent myocardial infarction, intra-abdominal malignancies, and emboli to the extremities. There is a greater than 50% association between CMI and coronary artery disease, peripheral vascular disease, hypertension, and smoking. Tobacco use is strongly associated with gut ischemia, with some 70% to 90% of patients admitting to significant use. Other comorbidities include diabetes, hypertension, renal disease, malignancy, gastrointestinal disease, and hypercoagulable states.

**INTESTINAL VASCULAR ANATOMY**

Knowledge of mesenteric vascular anatomy and physiology is key to an appreciation of the causes and consequences of intestinal ischemia and infarction. The anatomy of the mesenteric circulation is complicated by the almost endless variations of blood supply to the gut.

**Celiac Axis**

The celiac artery (Fig. 1) is the largest branch of the abdominal aorta, and it supplies the embryologic foregut. It leaves the abdominal aorta at an angle of 90° at the level of the T12 or L1 vertebral body. After coursing ventrally and inferiorly 1 to 2 cm, the celiac artery branches into the common hepatic, splenic, and left gastric arteries in 75% of the population. In 25%, there is a true trifurcation of these vessels, and in 1%, there is a common origin of the celiac and superior mesenteric branches—the celiacomesenteric trunk.

The common hepatic artery gives rise to the gastroduodenal artery, which then becomes the right gastroepiploic artery and the anterior and posterior superior pancreaticoduodenal arteries. Typically, the splenic artery gives off the left gastroepiploic artery, which joins the right gastroepiploic artery. The left gastric artery anastomoses with the right gastric artery along the lesser curvature of the stomach.

**Superior Mesenteric Artery**

The SMA (Fig. 2) is a large-caliber structure with a narrow takeoff from the aorta, making it the most susceptible of the major mesenteric vessels to embolic phenomena. It supplies the entire embryologic midgut and is the second largest intra-abdominal branch of the aorta. As a general rule, there are more SMA branches to the distal small bowel than the more proximal portions,
providing greater potential for distal anastomoses. The SMA originates 1 cm beneath the level of the celiac artery, usually at the level of L1, and courses inferiorly toward the right and terminates as the ileocolic artery at the level of the cecum. The major branches of the SMA are the inferior pancreaticoduodenal artery, the middle colic artery, the right colic artery, 4 to 6 jejunal branches, and 9 to 13 ileal branches.

The middle colic artery typically arises from the proximal SMA to supply the transverse colon and communicates with branches of the inferior mesenteric artery (IMA). The splenic flexure of the colon is a watershed region between these two mesenteric circulations. Accordingly, it is a frequent sight of ischemic colitis.

The right colic artery, which supplies the middle to distal ascending colon, usually arises from a common trunk with or just inferior to the middle colic artery.

Box 1: Causes of mesenteric ischemia

- AMI
- Emboli
  - Arrhythmias
  - Valvular disease
  - Myocardial infarction
  - Hypokinetic ventricular wall
  - Cardiac aneurysm
  - Aortic atherosclerotic disease
  - Iatrogenic
- Thrombosis
  - Atherosclerotic disease
- Nonocclusive
  - Heart failure
  - Cardiac bypass
  - Sepsis
  - Renal failure
  - Medications
  - Pancreatitis
  - Burns
- Venous occlusion
  - Hypercoagulable states
  - Sepsis
  - Malignancy
  - Portal hypertension
  - Compression
  - Pregnancy

Box 2: Disorders associated with mesenteric venous thrombosis

- Hypercoagulable states
  - Activated protein C resistance
  - Antithrombin resistance
  - Protein C deficiency
  - Protein S deficiency
  - Methylene tetrahydrofolate deficiency
  - Estrogen use (oral contraceptive, hormone replacement therapy)
  - Polycythemia vera
  - Thrombocytosis
  - Neoplasms
- Peripheral deep vein thrombosis
- Pregnancy
- Portal hypertension
  - Cirrhosis
  - Congestive splenomegaly
  - After sclerotherapy of esophageal varices
- Inflammation
  - Diverticulitis
  - Appendicitis
  - Pancreatitis
  - Perforated viscus
  - Inflammatory bowel disease
  - Pelvic or intra-abdominal abscess
- Postoperative state or trauma
  - Blunt abdominal trauma
  - Splenectomy and other postoperative states
- Decompression sickness
The IMA (Fig. 3) is the smallest of the mesenteric vessels and arises 6 to 7 cm below the SMA at the level of L3. It supplies the hindgut: the distal transverse colon, splenic flexure, descending colon, and rectosigmoid. The IMA is a narrow-caliber artery (0.5 cm) that has a relatively acute takeoff angle from the aorta, rendering it much less susceptible to embolic events. The major branches of the IMA include the left colic, sigmoid, and hemorrhoidal arteries. The ascending branches of the left colic artery reach the splenic flexure in 80% to 85% of patients and extend to the midtransverse colon in 15% to 20% of individuals. At this point, they anastomose with branches of the middle colic artery from the SMA. The sigmoidal branches form arcades that anastomose with the left colic artery and superior hemorrhoidal artery. The superior hemorrhoidal artery supplies blood to the wall of the upper two thirds of the rectum and to the mucosa of the lower third of the rectum.12,16

**Inferior Mesenteric Artery**

The IMA (Fig. 3) is the smallest of the mesenteric vessels and arises 6 to 7 cm below the SMA at the level of L3. It supplies the hindgut: the distal transverse colon, splenic flexure, descending colon, and rectosigmoid. The IMA is a narrow-caliber artery (0.5 cm) that has a relatively acute takeoff angle from the aorta, rendering it much less susceptible to embolic events. The major branches of the IMA include the left colic, sigmoid, and hemorrhoidal arteries. The ascending branches of the left colic artery reach the splenic flexure in 80% to 85% of patients and extend to the midtransverse colon in 15% to 20% of individuals. At this point, they anastomose with branches of the middle colic artery from the SMA. The sigmoidal branches form arcades that anastomose with the left colic artery and superior hemorrhoidal artery. The superior hemorrhoidal artery supplies blood to the wall of the upper two thirds of the rectum and to the mucosa of the lower third of the rectum.12,16

The middle hemorrhoidal artery arises from the anterior division of the internal iliac artery or from the vesical branch of this vessel. The middle hemorrhoidal artery traverses the infraperitoneal pelvis in the lateral ligaments and supplies the middle third of the rectum. The inferior hemorrhoidal artery is also a branch of the anterior division of the internal iliac artery. It is invested by endopelvic fascia as it exits the pelvis, below the piriformis muscle, through the greater sciatic foramen. It pursues a short course in the buttock and then

---

**Box 3**

**Causes of colonic ischemia**

- Inferior mesenteric artery thrombosis
- Arterial embolism
- Cholesterol emboli
- Cardiac arrhythmia
- Congestive heart failure
- Shock
- Volvulus
- Strangulated hernia
- Vasculitis
- Hematologic disorders
  - Sickle cell anemia
  - Protein C and S deficiencies
  - Antithrombin III deficiency
  - Factor V Leiden mutation (activated protein C resistance)
  - Factor I 2010A mutation (in combination with oral contraceptive use)
  - Polycythemia vera
- Infections
  - Parasites
    - *Angiostrongylus costaricensis*
    - *Entamoeba histolytica*
  - Viruses
  - Cytomegalovirus
  - Bacteria
    - *Escherichia coli* O157:H7
  - Trauma
  - Long-distance running
  - Pregnancy
- Surgical
  - Aneurysmectomy
  - Aortoiliac reconstruction
  - Gynecologic operations
  - Exchange transfusion
  - Colonic bypass
  - Lumbar aortography
  - Colectomy with IMA ligation
  - Colonoscopy
- Medications
  - Related to vasoconstriction or vasculitis
    - Digitalis
    - Vasopressin
  - Related to hypovolemia or constipation
    - Interferon-α
    - Saline laxatives
    - Estrogens
    - Progestins
    - Danazol
    - Psychotropic medications
    - Alosetron
  - Related to hypovolemia or constipation
    - Interferon-α
    - Saline laxatives
    - Estrogens
    - Progestins
    - Danazol
    - Psychotropic medications
    - Alosetron

**Gold**
- Pseudoephedrine
- Sumatriptan
- Cocaine
- Methamphetamine
- Nonsteroidal anti-inflammatory drugs
- Imipramine
re-enters the pelvis. It crosses the ischiorectal fossa, which may cause considerable bleeding if encountered during abdominoperineal resection of the rectum. This vessel supplies the levator ani and sphincters in addition to the lower rectum and anal canal. The mesenteric vascular anatomy is well depicted on cross-sectional imaging (Fig. 4).

**Mesenteric Collateral Flow Patterns**

There are numerous sources of collateral flow between the mesenteric vessels and nonmesenteric systemic vessels. This redundancy imparts substantial protection against intestinal ischemia and infarction after segmental vascular occlusion. As a result of these multiple potential sources of collateral flow, at least two of the three main vessels must be occluded or have critical stenoses for MI to develop.

**Celiac axis–superior mesenteric artery collaterals**

At autopsy, approximately 20% of individuals have greater than 50% stenosis of the celiac artery. Most of these patients are asymptomatic because of the rich collateral vessels from the SMA. The arc of Barkow forms potential communications between omental branches from the SMA and branches of the celiac axis.

**Superior mesenteric artery–inferior mesenteric artery collaterals**

At autopsy, it is not uncommon for the SMA (30%) and IMA (30%) to be stenotic. The marginal artery of Drummond, which lies in the subperitoneal space of the mesocolon of the descending colon, consists of branches from the ileocolic, right, middle, and left colic arteries. An anastomosis between the middle and left colic arteries is present in 95% of individuals and occurs at the splenic flexure—the so-called “Griffith’s point.” The arc of Riolan (meandering mesenteric artery) lies within the descending mesocolon as well but is more centrally located and usually joins the middle and left colic arteries.

**Inferior mesenteric artery–systemic circulation collaterals**

There are collateral vessels within the rectum by way of the anastomoses of the superior rectal...
(hemorrhoidal) arteries with the middle and inferior rectal arteries, which originate from the internal iliac blood vessels. The three hemorrhoidal vessels form a comprehensive anastomotic network in the submucosa of the anal canal and lower rectum. This complex blood supply to the rectum with its redundancies and collaterals explains why the rectum is often spared in patients who have bowel ischemia and infarction.

**Clinical correlates of mesenteric anatomy**
Chronic blockage of any single mesenteric artery is usually inconsequential if the collateral pathways are functional. Critical ischemia from acute occlusion can result from single-vessel disease in the absence of adequate collaterals or when multiple vessels are diseased.  

Certain operative procedures can disrupt key mesenteric collaterals. In a patient who has chronic celiac artery occlusion, a Whipple’s operation can cause hepatic ischemia by interrupting collateral arterial flow from the SMA through the pancreaticoduodenal arcades. A segmental colectomy may interrupt critical anastomotic networks between the SMA and IMA and result in acute ischemia in patients who have severe mesenteric occlusive disease.

The IMA is the most frequently occluded mesenteric vessel by chronic vascular disease. This artery is usually sacrificed during abdominal aortic aneurysm repair. If the SMA is severely diseased and the midgut is receiving a large proportion of blood from the arc of Riolan, sacrificing this vessel during aneurysm repair can cause small bowel and right colon infarction.  

**PHYSIOLOGY OF THE MESENTERIC CIRCULATION**
In the fasting state, the mesenteric vessels receive approximately 20% to 25% of the cardiac output and the splanchnic circulation contains approximately one third of the total blood volume, which makes it the circulatory system’s largest reservoir. Approximately 25% of the splanchnic circulation flows directly to the liver by way of the hepatic artery, and the remaining 75% of blood flow reaches the liver by way of the portal venous system.  

At rest, approximately 70% to 80% of the blood flow is directed to the mucosa, 15% to 25% is distributed to the muscularis propria and serosal layers, and 5% is distributed to the submucosal layer. The epithelial cells in the terminal villi receive 60% of the mucosal blood flow, with the crypts and goblet cells receiving the other 40%.  

**CLASSIFICATION OF ISCHEMIC BOWEL DISEASE**
Intestinal ischemic disorders have been classified into several major types:  

- AMI  
- SMAE  
- NOMI  
- Superior mesenteric artery thrombosis  
- Superior mesenteric vein thrombosis  
- CMI (intestinal angina)  
- CI  
- Reversible ischemic colopathy  
- Transient ulcerating ischemic colitis  
- Chronic ulcerating ischemic colitis  
- Colonic stricture  
- Colonic gangrene  
- Fulminant universal ischemic colitis  

CI is the most common vascular disorder of the gut, followed by AMI. AMI is associated with compromise of the blood flow in the SMA distribution.
Fig. 4. Normal mesenteric vascular anatomy depicted on cross-sectional imaging. (A) Sagittal maximum intensity projection (MIP) multidetector CT (MDCT) image demonstrates the origins of the celiac artery (solid white arrow), superior mesenteric artery (SMA) (broken white arrow), and IMA (black arrow). (B) Coronal reformatted MIP MDCT image nicely depicts the jejunal branches of the SMA (red arrow). The splenic artery (yellow arrow), common hepatic artery (white arrow), gastroduodenal artery (blue arrow), and proper hepatic artery (green arrow) are also shown. (C) Sagittal reformatted MDCT image shows atherosclerotic calcification (white arrow) in the SMA. (D) Sagittal reformatted MDCT image shows the IMA (white arrow) originating from an abdominal aortic aneurysm (A). (E) Coronal reformatted MDCT image demonstrates the superior mesenteric vein (SMV) (white arrow) and its major tributaries. (F) Gray-scale sagittal sonogram of the abdominal aorta reveals the origins of the celiac artery (broken arrow) and SMA (solid arrow). (G) MR imaging venogram shows the mesenteric circulation in a patient who has cirrhosis and portal hypertension. The white arrow indicates the portal vein, the yellow arrow indicates the SMV, and the red arrow indicates the splenic vein. (Courtesy of Dr. Jochen A. Gaa, Munich, Germany.)
affecting all or portions of the small bowel and right colon. In CMI, the splanchnic circulation is insufficient in meeting the functional demands of the gut but there is no loss of tissue viability. AMI, CMI, and CI have distinct clinical manifestations that require different management strategies. The differentiation among these various ischemic disorders can be made in some patients with the assistance of cross-sectional imaging.

PATHOLOGIC FINDINGS OF INTESTINAL ISCHEMIA

Small Bowel
Pathologic evidence of small bowel ischemia and infarction (Figs. 6–8) may be diffuse and confluent or patchy and multifocal. The serosal aspect of the affected small bowel often appears congested or blue and black. Perforations may be present but may not be accompanied by well-developed fibrinous exudates if the surgical resection occurs within a short time of presentation. The mesentery is usually pale in arterial occlusions and congested and hemorrhagic in venous thrombosis. The demarcation between normal and involved gut is usually abrupt. The intestinal lumen is invariably filled with blood, and the mucosal surface may appear beefy red, boggy, and ulcerated and may contain irregularly protruding mucosal islands. This mucosal appearance is responsible for the “thumbprinting” sign. Pseudomembranes and transmural hemorrhage may be seen. The wall of the involved segment is often friable and thin. In patients who have mesenteric vein thrombosis, thrombi can be seen in mesenteric veins on gross examination.14,15

Early histologic changes consist of hemorrhage, congestion, and edema of the submucosa, sometimes associated with preservation of the overlying mucosa. Submucosal changes can then lead to various degrees of mucosal necrosis with or without ulceration, luminal hemorrhage, and pseudomembrane formation. In the acute setting, there is absence of a chronic inflammatory response, although neutrophils may be seen if enough time has elapsed since the onset of occlusion.14,15

The mucosa shows a loss of epithelium, which occurs progressively from the tips of the villi to the base of the crypts and is associated with
various degrees of edema and congestion. Within hours of the injury, neutrophils influx into the damaged area. Depending on the extent and severity of the injury, the mucosal changes may reverse to normal if the ischemic insult stops. If the ischemia is persistent or severe, tissue healing may result in fibrosis and stricture formation.14,15

Mesenteric vessel evaluation may be confusing, because thrombi may form acutely as a response to stasis and congestion. Clinically significant thrombi show evidence of organization, implying their presence over a significant period. Fibrin thrombi may be present in small arterioles in areas of necrosis and do not, by themselves, indicate vasculitis or a hypercoagulable state.14,15

**Colon**

Acute ischemic lesions of the colon show necrosis of the superficial portion of the mucosa that often spares the deeper portions of the colonic crypts. The remaining crypts typically have an atrophic or withered appearance that reveals striking cytologic atypia, which may be mistaken for dysplasia. Pseudomembranes, hemorrhage into the lamina propria, and hyalinization of the lamina propria may also be seen. These lesions may regress on

![Fig. 5. Collateral vessels displayed on shaded-surface display multidetector CT (MDCT) image. The marginal artery of Drummond (arrowheads) in a patient who has SMA stenosis demonstrates the collateral pathways between the IMA and SMA. (From Horton KM, Fishman EK. Vascular disorders of the small bowel. In: Gore RM, Levine MS, editors. Textbook of gastrointestinal radiology. 3rd edition. Philadelphia: WB Saunders; 2008. p. 909.)](image)

![Fig. 6. Pathologic findings of intestinal ischemia and infarction: intraoperative images. (A) Short segment of small bowel infarction attributable to strangulation from closed-loop obstruction caused by an adhesive band. (B) Long segment of small bowel infarction caused by SMA occlusion. (C) Sigmoid colon infarction attributable to a volvulus.](image)
their own, or frank gangrene with perforation or stricture formation may occur.\textsuperscript{14,15}

The chronic phase of CI may be more difficult to diagnose, because the only histologic findings may be areas of submucosal fibrosis and stricture, which are nonspecific.\textsuperscript{14,15}

**CLINICAL FEATURES OF MESENTERIC ISCHEMIA**

To date, no reliable physiologic or biochemical means of detecting MI and predicting behavior have been established. Serum lactate is an established marker of cell hypoxia but lactic acidosis is often a late finding in the diagnostic pathway with
concomitant shock, bowel necrosis, and circulatory collapse.\textsuperscript{22,26} Recently, plasma D-dimer levels have been suggested as an early marker of acute ischemia. In animal studies, they have been shown to correlate with the onset of ischemia and function as a time-sensitive indicator of disease progression.\textsuperscript{20} The enzyme alcohol dehydrogenase has been identified as a time-sensitive indicator of bowel ischemia as opposed to generalized systemic hypoperfusion. Glutathione S-transferase is released with cell membrane damage, and several isoforms have been correlated with bowel specificity. This detoxifying cytosolic enzyme has been shown to exhibit a time- and duration-specific detectable increase with progressive tissue ischemia.\textsuperscript{4,20}

**Acute Mesenteric Ischemia**

The diagnosis of AMI can be difficult, because most patients have nonspecific symptoms of abdominal pain. Abdominal pain out of proportion to the findings on physical examination and...
persisting beyond 2 to 3 hours is the classic presentation. Nausea, diarrhea, vomiting, and anorexia can also be part of the initial symptom complex. An elevated white blood cell count is common; 15% of patients report melena or hematochezia, and occult fecal blood is found in 50%. With delay in diagnosis and progression to full-thickness mural injury, the patient develops peritoneal signs of distention, guarding, rigidity, and hypotension. Lactic acidosis is present in addition to elevations in serum amylase, aspartate aminotransferase, and creatine phosphokinase. If hyperkalemia and hyperphosphatemia are present, bowel infarction should be suspected.1,3,6,26

An oxygen supply that is insufficient to meet the demands of the gut results in increased production of lactate by the bowel as a result of anaerobic glycolysis within cells. MI also increases xanthinoxidase within small bowel mucosa, which converts hypoxanthine to uric acid. The free radicals that form with reperfusion damage the cytomembrane, and cell edema ensues with cellular decay (Fig. 9).

There are four clinical-radiologic stages that occur in patients who have ischemia and infarction of the gut.4,20,25,27,28

**First stage**
Immediately after arterial occlusion, there is rapid onset of severe abdominal pain associated with loose and sometimes bloody stools and vomiting. Typically, there is discordance in the severe, often excruciating, degree of abdominal pain and a relative paucity of physical abdominal findings. At this point, hyperperistalsis and hyperactive bowel sounds on auscultation are evident. The plain abdominal radiograph may show a gasless

---

**Fig. 13.** Pneumatosis intestinalis on plain abdominal radiograph. Multiple linear intramural lucencies (arrows) are present within the small bowel in this patient who has strangulated small bowel obstruction attributable to a ventral hernia. Necrotic bowel was resected at the time of surgery.

**Fig. 14.** Pneumatosis associated with mesenteric arterial compromise. MDCT scan of the pelvis displayed at lung windows demonstrates pneumatosis involving several ileal loops.

**Fig. 15.** Pneumatosis intestinalis attributable to small bowel infarction in an elderly patient who has cardiac arrhythmias. Coronal reformatted MDCT image shows intramural gas (red arrow) in several ileal segments. Mesenteric (yellow arrow) and intrahepatic (blue arrow) portal venous gas is also evident.
abdomen. Thrombus may be seen in cases of superior mesenteric artery thrombosis (SMAT) and superior mesenteric artery embolism (SMAE) but may be relatively normal in cases of NOMI.\textsuperscript{27,28} In patients who have acute MVT, plain abdominal radiographs may reveal a reflex ileus pattern without bowel distention. Sonography may show a homogeneously hypoechoic intestinal wall as a result of edema that occurs earlier in the course of disease when compared with SMA compromise.\textsuperscript{27,28}

Second stage
In this stage, the pain may be diminishing but becomes more continuous and diffuse. The abdomen becomes distended, and there is more generalized tenderness. Bowel sounds are absent. With persistent arterial occlusion, there is disruption of the microvascular integrity of the bowel wall. The capillary walls become damaged because they derive oxygen by direct diffusion from the blood pool. This increases mucosal permeability so that the remaining blood may extravasate, causing hemorrhagic foci in the thinned bowel wall (Fig. 10). Pari passu, the mucosa cannot produce the normal amount and quality of enteric secretions and the intestinal microflora can proliferate, producing gas.\textsuperscript{27,28}

Plain abdominal radiographs may show mild gaseous dilation of the affected loops, which have lost their tone. Multidetector CT (MDCT) demonstrates these findings in addition to a “paper-thin” bowel wall with decreased mural enhancement. If reperfusion does not occur, transmural bowel necrosis may ensue and intramural air may dissect into the necrotic mucosa; from there, it may dissect intramurally, subperitoneally, and into the abdomen. Thrombus may be seen in cases of superior mesenteric artery thrombosis (SMAT) and superior mesenteric artery embolism (SMAE) but may be relatively normal in cases of NOMI.\textsuperscript{27,28} In patients who have acute MVT, plain abdominal radiographs may reveal a reflex ileus pattern without bowel distention. Sonography may show a homogeneously hypoechoic intestinal wall as a result of edema that occurs earlier in the course of disease when compared with SMA compromise.\textsuperscript{27,28}

Second stage
In this stage, the pain may be diminishing but becomes more continuous and diffuse. The abdomen becomes distended, and there is more generalized tenderness. Bowel sounds are absent. With persistent arterial occlusion, there is disruption of the microvascular integrity of the bowel wall. The capillary walls become damaged because they derive oxygen by direct diffusion from the blood pool. This increases mucosal permeability so that the remaining blood may extravasate, causing hemorrhagic foci in the thinned bowel wall (Fig. 10). Pari passu, the mucosa cannot produce the normal amount and quality of enteric secretions and the intestinal microflora can proliferate, producing gas.\textsuperscript{27,28}

Plain abdominal radiographs may show mild gaseous dilation of the affected loops, which have lost their tone. Multidetector CT (MDCT) demonstrates these findings in addition to a “paper-thin” bowel wall with decreased mural enhancement. If reperfusion does not occur, transmural bowel necrosis may ensue and intramural air may dissect into the necrotic mucosa; from there, it may dissect intramurally, subperitoneally, and into the abdomen. Thrombus may be seen in cases of superior mesenteric artery thrombosis (SMAT) and superior mesenteric artery embolism (SMAE) but may be relatively normal in cases of NOMI.\textsuperscript{27,28} In patients who have acute MVT, plain abdominal radiographs may reveal a reflex ileus pattern without bowel distention. Sonography may show a homogeneously hypoechoic intestinal wall as a result of edema that occurs earlier in the course of disease when compared with SMA compromise.\textsuperscript{27,28}

Second stage
In this stage, the pain may be diminishing but becomes more continuous and diffuse. The abdomen becomes distended, and there is more generalized tenderness. Bowel sounds are absent. With persistent arterial occlusion, there is disruption of the microvascular integrity of the bowel wall. The capillary walls become damaged because they derive oxygen by direct diffusion from the blood pool. This increases mucosal permeability so that the remaining blood may extravasate, causing hemorrhagic foci in the thinned bowel wall (Fig. 10). Pari passu, the mucosa cannot produce the normal amount and quality of enteric secretions and the intestinal microflora can proliferate, producing gas.\textsuperscript{27,28}
peritoneal cavity and ultimately spread through the mesenteric and portal venous system.\textsuperscript{27,28} With persistent mesenteric venous occlusion, the intramural blood volume increases as arterial blood keeps flowing into the bowel wall in patients with venous compromise. This leads to increased intravascular hydrostatic pressure, which dilates the blood vessels and widens the fenestrations among the vascular endothelial cells. This leads to extravasation of plasma, contrast material, or red blood cells (Fig. 11) into the bowel wall or lumen. Tension in the submucosal extravascular compartment or prolonged stasis-induced thrombosis of the microvasculature may interrupt arterial blood flow. The imaging findings at this stage of disease are related to mural thickening, intramural hemorrhage, and submucosal edema.\textsuperscript{27,28}

Sonography may reveal thrombus at the origin of the superior mesenteric vein (SMV) and mural thickening with hyperechoic mucosal layers and hypoechoic submucosa attributable to edema of the affected loops.

On MDCT, the ischemic gut demonstrates a target appearance with an inner hyperdense ring as a result of surface mucosal hypervascularility, hemorrhage, and ulceration; a middle hypodense edematous submucosa; and a normal or slightly thickened muscularis propria. The damage to the gut may be reversible at this stage of impaired venous drainage, because the integrity of the deeper mural layers is preserved. If the vascular compromise persists, three possible outcomes may ensue: healing, chronic ischemia, or progression to intestinal infarction. Healing may lead to stricture formation because of circumferential granulation tissue formation and fibrosis in response to parietal layer damage.

Third stage
With progressive mural injury, fluid, protein, and electrolytes begin to leak into the lumen, the bowel becomes necrotic, and peritonitis develops. Fluid loss can be massive, and this stage of ischemia is clinically similar in its manifestations to other causes of generalized peritonitis.

If the arterial blood flow compromise is alleviated, reperfusion of the gut is associated with several radiographic findings best seen with MDCT. Blood, plasma, contrast material, or red blood cells may extravasate through the disrupted vascular wall into the mucosa and submucosa, causing mural thickening and bloody fluid filling the intestinal lumen. Plain abdominal radiographs may show mild dilation of the affected loops, with mural thickening, sparse and subtle valvulae conniventes, and some air-fluid levels. Ultrasound examination may reveal increased intraluminal secretions and decreased peristalsis.\textsuperscript{27,28}

On MDCT, the thickened submucosa may be hyperdense because of hemorrhage. After contrast administration, the mucosa may show hyperenhancement with submucosal edema and hypodensity. Mural stratification typically is preserved.

Persistent venous thrombosis leads to mesenteric vascular engorgement and edema, with the formation of venous collateral blood vessels. This stage produces imaging findings typical of patients with chronic venous impairment. CT shows mural thickening of the involved segments, peritoneal fluid, and mesenteric engorgement.

Fourth stage
Abdominal plain radiographs show markedly dilated loops with thickening of the valvulae conniventes and multiple air-fluid levels (Fig. 12). Extraluminal fluid and absent peristalsis are evident sonographically. CT may demonstrate SMA thrombosis or emboli; bowel enhancement is poor, and pneumatosis may be evident in cases of frank mural necrosis.\textsuperscript{27,28}

Frank intestinal infarction initially causes progressive submucosal hemorrhage and edema. The cyanosis leads to loss of integrity of the intestinal wall with necrosis and peritonitis. Intramural and mesenteric venous gas may be apparent, associated with subperitoneal or intraperitoneal serosanguineous or bloody fluid.

Conventional abdominal radiographs may show mural thickening, pneumatosis (Fig. 13), or pneumatoperitoneum. Sonography reveals mural
thickening of the involved segment, intramural or intraperitoneal gas, and peritoneal fluid. On CT, venous thrombosis, absence of mural enhancement, and the presence of fluid and gas may be evident in the mesenteric and portal veins, bowel wall, and subperitoneal or peritoneal space (Figs. 14 and 15).

Chronic Mesenteric Ischemia

This disorder, also known as abdominal angina (AA), is often insidious in its presentation (Fig. 16). The occlusive lesion compromises the increased blood flow that typically occurs after eating. This leads to postprandial pain that is maximal 30 to 90 minutes after food intake. The intensity of the pain leads to fear of food and weight loss, which is compensated for to some extent by frequent intake of small meals—the small meal syndrome. The appetite is usually not affected, which is an important distinguishing feature from patients who have cancer. Constipation is often present initially, but with the development of ischemic villous atrophy, malabsorption and diarrhea may develop.5

Colonic Ischemia and Infarction

Patients who have acute CI typically present with acute mild abdominal pain and tenderness over the affected bowel, most often on the left. Within 24 hours of the onset of abdominal pain, mild to

---

**Fig. 18.** Ischemic colitis: MDCT findings. (A) Axial image at the level of the splenic flexure demonstrates mural thickening and submucosal edema (white arrow). (B) More caudal scan shows that the proximal descending colon has a target appearance with higher density mucosa and muscularis mucosa framing the edematous submucosa (white arrow). A small amount of fluid is seen in the fascial trifurcation (red arrow) of the left subperitoneal space. (C) Coronal reformatted image reveals the full extent (red arrows) of colonic involvement.
moderate amounts of rectal bleeding or bloody diarrhea occur. Approximately one half of patients who have nongangrenous CI have complete resolution within 2 weeks, with recurrence developing in only 5% of individuals. Patients who have gangrenous colon ischemia and require surgical resection have greater than 50% mortality.

There are three clinical-radiologic stages that occur in patients who have ischemia and infarction of the gut.

First stage
With early colonic arterial compromise, there is mural thickening and extensive submucosal edema. The colon has a shaggy contour (Fig. 17), and there is a variable degree of pericolonic streakiness and peritoneal fluid. In this phase, there is mucosal hyperdensity as a result of hemorrhage. This is more commonly seen in the left colon.

In the early stages of colonic venous compromise, there is mural thickening and mucosal hyperdensity from hemorrhagic phenomena. Moderate peritoneal fluid may be present, and occlusion of mesenteric vessels is visible.

Second stage
In this phase of colonic arterial compromise, there is progression of the ischemic change without reperfusion. This causes symmetric and concentric mural thickening of the colon.

This phase of colonic venous compromise is associated with submucosal hypodensity attributable to mural edema (Fig. 18). The involved colon has a shaggy contour associated with pericolonic streakiness, indicating progression of the impaired venous drainage. Peritoneal fluid (Fig. 19) of varying amounts accompanies this stage of CI. Mesenteric vascular occlusion can be visualized.

Third stage
In this phase of arterial occlusion or hypoperfusion, there is frank infarction with intramural gas and mesenteric or portal venous gas. There is absence of a small amount of intraperitoneal fluid, absence of parietal enhancement, and pneumoperitoneum.

With progressive vascular compromise, the colon becomes homogeneously thickened with decreased enhancement. Pneumatosis (Fig. 20) and portal (Fig. 21) and mesenteric venous gas may be present, and occlusion of mesenteric vessels can be identified.

Differential Diagnosis
The recognition of bowel ischemia and infarction can be difficult, because most patients present with nonspecific symptoms and signs. This is particularly true in patients who have other pressing clinical issues. Appendicitis, diverticulitis, nonstrangulating bowel obstruction, peptic ulcer disease, gastritis, gastroenteritis, infectious ileocolitis, inflammatory bowel disease, pancreatitis, acute cholecystitis, and ruptured aortic aneurysm often enter the clinician’s mind as the most likely diagnosis, with intestinal ischemia not considered or placed at the bottom of the differential diagnosis. Clearly, the diagnosis hinges on having a high index of
suspicion. In one series evaluating gastrointestinal complications of cardiac surgery, 11.5% of these gastrointestinal complications were attributable to intestinal ischemia and 1.1% were attributable to mesenteric vascular occlusion. Other complications included upper gastrointestinal hemorrhage (28.6%), gastroesophagitis (12.2%), colitis (12.2%), pancreatitis (8.8%), cholecystitis (6.8%), perforated peptic ulcer (4.7%), diverticulitis (3.4%), and lower gastrointestinal hemorrhage (0.7%).

Prognosis

It is important to differentiate the cause of AMI because of variation in disease progression, response to treatment, and outcome. The prognosis is better after acute MVT than that after acute arterial MI. The prognosis after mesenteric arterial embolism is better than that after arterial thrombosis or nonocclusive ischemia. The mortality rate after surgical treatment of arterial embolism and venous thrombosis (54.1% and 32.1%, respectively) is less than that after surgery for arterial thrombosis and nonocclusive ischemia (77.4% and 72.7%, respectively).
DIAGNOSTIC IMAGING MODALITIES

Plain Abdominal Radiographs

Most patients who have intestinal ischemia demonstrate nonspecific findings, such as intestinal dilatation (see Fig. 12), gasless abdomen, a small bowel pseudo-obstruction pattern, or paralytic ileus. More specific but far less common findings include thumbprinting in which multiple, round, smooth soft tissue densities project into the intestinal lumen because of mucosal and submucosal edema and hemorrhage. Specific late signs indicating infarction include pneumatosis intestinalis (PI; see Fig. 13) and portal venous gas. In most cases of patients who have intestinal ischemia, however, plain radiographs are of limited value.27–39

Barium Studies

Barium studies have been replaced by MDCT and, to a lesser degree, MR imaging and ultrasound in the clinical setting of acute intestinal ischemia. Like plain abdominal radiographs, barium studies are too nonspecific and insensitive in evaluating the mural, mesenteric, and vascular manifestations of ischemia. Furthermore, dense barium interferes with subsequent MDCT examinations and transcatheter interventions. Colonoscopy is the most accurate means of establishing the diagnosis of ischemic colitis (Fig. 22).

Barium studies still are of some value in the setting of chronic intestinal angina and radiation enteritis. The hallmark of small bowel ischemia on conventional barium studies is thickening of the valvulae conniventes and “thumbprinting.” The intramural accumulation of blood may distend the submucosa to such a degree that the folds become focally dilated and flattened, especially along the mesenteric border of the bowel.19,40,41

Fig. 24. SMA emboli. (A) Sagittal reformatted maximum intensity projection (MIP) MDCT image demonstrates a small embolism (arrow) in the SMA. (B) In a different patient, a larger embolism (arrow) is seen in a more distal portion of the SMA.

Fig. 25. SMA embolism in an elderly woman in septic shock. Coronal reformatted MDCT shows diminished enhancement of the ileum (red arrows) in comparison to the jejunum (yellow arrows).
Angiography

Before the late 1990s and the introduction of MDCT, catheter angiography was the reference standard for the diagnosis of intestinal ischemia.\textsuperscript{23,42,43} This is an invasive procedure that has now been supplanted by CT angiography (CTA) and magnetic resonance angiography (MRA).\textsuperscript{44} Angiography is now primarily performed immediately before transcatheter intervention.

Transcatheter therapy is currently the intervention of choice in patients who have chronic intestinal angina. This involves the placement of a stent usually in the SMA (see Fig. 16). If there is high-grade stenosis (>70%) of the celiac trunk and SMA, both vessels are stented if technically feasible.\textsuperscript{45}

Doppler Ultrasound

Because of its dependence on patient factors, including body habitus, the presence of air-filled

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image}
\caption{NOMI secondary to systemic hypotension resulting from massive intraperitoneal rupture and hemorrhage of hepatocellular carcinoma of the left lobe of the liver. (A) Axial MDCT scan shows a hyperdense bowel (yellow arrows) secondary to intramural and mucosal hemorrhage. Note the patent vasa rectae (white arrow) and hemo-peritoneum. (B) Coronal reformatted MDCT image shows the massive hemoperitoneum (H), the hyperdense small bowel (yellow arrows), and active extravasation of contrast material in the left hepatic lobe neoplasm (red arrow). (C) Sagittal reformatted MDCT image shows a slit-like inferior vena cava (red arrows), attesting to the compromised cardiovascular status of the patient.}
\end{figure}
bowel loops, prior operations, and patient cooperation, ultrasound is not typically used in the initial evaluation of acutely ill patients who are suspected of having acute intestinal ischemia.46–50

In patients who have chronic intestinal angina, Doppler ultrasound can be useful in the detection of celiac artery and SMA stenoses. Peak systolic velocity of greater than 275 cm/s in the SMA and 200 cm/s in the celiac artery indicates greater than 70% stenosis of these vessels (see Fig. 16).34–36

**MR Imaging**

MR imaging and MRA can be used to image the gut, mesentery, and surrounding vasculature directly.51–54 MR imaging, however, is best used in the nonacute setting. The critically ill patient who is suspected of having intestinal ischemia typically has a life support apparatus incompatible with the MR imaging scanner. These patients are best scanned with MDCT, which also offers better spatial resolution.55,56 The theoretic advantage of using gadolinium-based intravascular contrast agents for MR imaging and MRA rather than the iodinated contrast used in CT in patients with renal insufficiency has been negated by the increasing incidence of nephrogenic systemic fibrosis related to the gadolinium-based MR imaging contrast agents.

In patients who have chronic intestinal angina, contrast enhanced three-dimensional MRA (Fig. 23) provides anatomic information similar to conventional angiography, and its effectiveness in evaluating the mesenteric circulation has been well documented.57

**Multidetector CT**

MDCT has become the preferred imaging technique for the evaluation of patients who are suspected of having acute and chronic intestinal ischemia. It can be performed quickly in critically ill patients and depends less on operator skill and patient factors than other imaging examinations.30,58–63

MDCT produces a volume data set that can be reformatted and viewed in any projection, affording exquisite visualization of the bowel wall, surrounding fat, mesenteries, and omenta, which may all show abnormalities with ischemia or infarction. CTA can visualize even tiny distal vascular segments and depict stenoses and their causes: atherosclerotic plaque, thrombus,
anatomic abnormalities (eg, obstruction), and tumor.18,25,59,64–78

ACUTE ARTERIAL AND VENOUS MESENTERIC ISCHEMIA

The clinical setting of AMI is characterized by the combination of a difficult diagnosis, high fatality rates, and the need for rapid and aggressive diagnostic and therapeutic interventions in patients who are often elderly and have multiple comorbidities.26

There are four main categories of AMI: SMAE (50%), NOMI (20%–30%), SMAT (15%–25%), and superior mesenteric vein thrombosis (SMVT) (5%).1–3

Superior Mesenteric Artery Embolism (SMAE)

The wide caliber and narrow takeoff angle of the SMA off the aorta make it particularly vulnerable to embolic events. The offending emboli (Figs. 24 and 25) usually originate from a left atrial or ventricular mural thrombus or vegetations on a heart valve. Nearly one half of patients who have SMAT have synchronous extramesenteric emboli, including peripheral artery emboli, and 20% have synchronous emboli to the spleen, kidneys, or other organs.79

The embolism usually lodges in the proximal SMA, 3 to 10 cm from its origin, in a tapered segment just distal to the middle colic artery branch, although some 15% of emboli cause occlusion at the origin of the SMA. Multiple emboli are present in 20% of cases.17 Emboli that occur proximal to the origin of the ileocolic artery are considered “major”; “minor” emboli are those that lodge in the SMA distal to the takeoff of the ileocolic artery or in the distal branches of the SMA.8

Nonocclusive Mesenteric Ischemia (NOMI)

The diagnosis of NOMI is made by excluding other causes of intestinal ischemia, such as atherosclerosis, arterial or venous thrombosis, embolism, or vasculitis. NOMI is seen in the setting of splanchnic vasoconstriction precipitated by hypoperfusion from acute myocardial infarction, congestive heart failure, arrhythmias, shock (Fig. 26), cirrhosis, sepsis, hypovolemia, chronic renal disease, medications, and the use of splanchnic vasoconstrictors.1,2

The angiographic features of NOMI include (1) narrowing of the origins of the SMA branches, (2) irregularities of these branches, (3) spasm of the mesenteric arcades, and (4) impaired filling of the intramural vessels.2,23

Fig. 28. Occlusive thrombus of the SMA. (A) Volume rendering shows a defect in the SMA (arrows). Note the thoracic aorta stent graft. (B) Sagittal reformatted image better demonstrates the thrombus (arrowheads).
Shock bowel is a subtype of NOMI and is caused by prolonged hypoperfusion because of hypovolemic shock.\textsuperscript{80,81} This is a transient phenomenon that resolves with restoration of normotension. Shock bowel causes increased permeability of the bowel wall to macromolecules, and the mural thickening and intraluminal fluid are attributable to failure of fluid resorption.

MDCT shows diffuse abnormalities of the small intestine, including mural and mucosal thickening, dilation, increased luminal fluid, lumen dilation, increased mural enhancement, and a normal-appearing colon.\textsuperscript{29} There is a decreased caliber of the abdominal aorta and inferior vena cava and moderate to large peritoneal fluid collections. The mural changes are attributable to increased mucosal permeability related to oxygen hypoperfusion, failed resorption capacity, slow flow, and interstitial leakage of contrast material. This appearance should be distinguished from the diffuse small bowel mural edema that accompanies aggressive volume resuscitation in the setting of trauma.

\textbf{Fig. 29.} SMV thrombosis in a woman on hormone replacement therapy. (A) Coronal reformatted MDCT images show thrombus (yellow arrow) in the first- and second-order tributaries of the SMV. (B) Thrombus (yellow arrow) is present in the main portal vein. There is mural thickening of the duodenum with some stranding of the adjacent fat, but most of the small bowel and right colon appear remarkably normal. Coronal MR imaging shows thrombus in the SMV (yellow arrow) (C) and main portal vein (yellow arrow) (D).
These patients show signs of elevated central venous pressure that include periportal edema, a dilated inferior vena cava, increased attenuation of mesenteric and retroperitoneal fat, and normal mural enhancement of the small bowel.31,80,81

Superior Mesenteric Artery Thrombosis (SMAT)

Because of previously developed collateral vessels, SMAT (Figs. 27 and 28) may have a somewhat more insidious onset than SMAE. The acute ischemic event is commonly superimposed on CMI and 20% to 50% of these patients have a history of postprandial abdominal pain, food aversion, and weight loss during the weeks to months before the seminal event. Because of the antecedent stenosing atherosclerosis, patients who have SMAT usually tend to develop symptoms more subacutely than those patients who have SMAE.17,18

Superior Mesenteric Vein Thrombosis (SMVT)

MVT (Figs. 29–31) occurs as an acute, subacute (weeks to months), or chronic disorder. The mean age of these patients is 48 to 60 years, which is younger than those with other forms of AMI. As many as 60% of patients have a history of peripheral vein thrombosis.2,8

The location of the primary thrombus within the mesenteric venous circulation depends on the cause. MVT attributable to cirrhosis, neoplasm, or operative injury begins at the site of obstruction and propagates peripherally. Thrombosis attributable to hypercoagulable states starts in smaller branches and propagates into the major trunks. MVT is associated with an extremely wide clinical spectrum ranging from a relatively asymptomatic patient in whom the thrombosis is diagnosed incidentally to an acute, severe, life-threatening disease.1–4,8

The degree of thrombosis may be large, but intestinal infarction is rare unless the branches of the peripheral arcades and the vasa recta are involved. If the collateral circulation is inadequate and venous drainage from the involved segment is compromised, the affected intestine becomes congested, edematous, cyanotic, and thickened with intramural hemorrhage.1 Serosanguineous peritoneal fluid heralds early hemorrhagic infarction.8

CHRONIC MESENTERIC ISCHEMIA

Atherosclerosis of the mesenteric circulation is quite common, particularly in the elderly population. Symptomatic CMI, however, is rare because of the development of extensive collaterals. Risk factors for the development of CMI include a positive family history, smoking, hypertension, and hypercholesterolemia, the same risk factors as for atherosclerosis. There is a female predominance of symptomatic disease.

Nonatherosclerotic causes of CMI are less frequent and include celiac artery compression (median arcuate ligament syndrome), chronic aortic dissection, inflammatory arterial disease, aortic coarctation, middle aortic syndrome, fibromuscular dysplasia, and neurofibromatosis.
COLONIC ISCHEMIA

CI is the most common vascular disorder of the gut in elderly patients. The colon is predisposed to ischemia because of the fact that it receives less blood flow per gram of tissue than does the remainder of the gastrointestinal tract. Indeed, there is an extensive network of intramural vessels arising from the vasa recta and vasa brevia in the mesenteric border of the gut that gives rise to a microvascular plexus in the muscularis propria and submucosal layer and is less well developed in the colon compared with the thinner walled small bowel.

CI encompasses a spectrum of injury that may be reversible or irreversible. CI can be further categorized as (1) reversible ischemic colonopathy (submucosal or intramural hemorrhage), (2) reversible or transient ischemic colitis, (3) chronic ulcerative ischemic colitis, (4) ischemic colonic stricture, (5) colonic gangrene, and (6) fulminant universal ischemic colitis. Many cases of transient or reversible ischemia of the colon are missed because the disease is self-limited.

PNEUMATOSIS INTESTINALIS

Intramural gas is associated with several disorders ranging from life-threatening to benign. PI pathogenically derives from four major categories: bowel necrosis, mucosal disruption, increased mucosal permeability, and pulmonary disease. The first three causes may be found in patients who have intestinal ischemia (see Figs. 13–15, 20, and 21). In patients who have intestinal ischemia, gas may dissect from the intestinal lumen because of an increase in intraluminal pressure (eg, from obstruction) or mucosal compromise.
(eg, ischemia, infarction). At the same time, gas-forming bacilli enter the submucosa through mucosal rents and produce intramural gas.\(^{82-84}\)

In the presence of PI in the setting of suspected intestinal ischemia, PI and the following elevated markers are associated with a poor prognosis: acidosis with a blood pH less than 7.3, hyperamylasemia of greater than 200 IU/L, a serum bicarbonate level of less than 20 mmol/L, and an elevated serum lactic acid level of greater than 2 mmol/L. Indeed, the presence of lactic acidosis greater than 2 mmol/L in patients who had PI was associated with a greater than 80% mortality rate.\(^{84}\)

PI is useful in differentiating early and nontransmural MI from full-thickness and irreversible transmural infarction. Linear PI is more often seen than bubbly PI in patients who have transmural bowel infarction. PI that accompanies portomesenteric venous gas correlates strongly with transmural bowel infarction, whereas PI without evidence of portomesenteric gas may have a more benign course.\(^{39,60,76}\)

**FOCAL SEGMENTAL MESENTERIC ISCHEMIA**

Short-segment ischemic disease may be caused by a large number of disorders, including vasculitis, medications, surgery, radiation, neoplasm, and, most importantly, bowel obstruction. Most cases of localized MI show similar radiologic features. It is important, however, to determine the underlying cause to guide diagnostic and therapeutic planning. The clinical presentation of localized MI depends on the length and distribution of the ischemia and the course of disease.\(^{30,33}\)

With the exception of strangulated bowel obstruction, there is usually adequate collateral circulation to prevent transmural hemorrhagic infarction; however, the affected bowel often becomes secondarily infected. Limited tissue necrosis may go on to complete healing, chronic enteritis, or stricture formation.\(^{30}\)

**Bowel Obstruction**

Strangulation and infarction (Figs. 32 and 33) are the most dreaded complications of bowel obstruction. Strangulation is usually seen in the setting of a closed-loop obstruction caused by volvulus, adhesions and bands, and internal or external hernias. This complication occurs in approximately 10% of patients who have small bowel obstruction and carries a high mortality rate of 20% to 37%.\(^{36}\)

The mural changes associated with closed-loop obstruction and strangulation include circumferential mural thickening (>3 mm), the target or halo sign (indicating submucosal edema), focal loss of mural enhancement (impaired arterial flow), persistent mural enhancement (impaired venous outflow), mural hemorrhage or haziness on noncontrast scans, and pneumatisis.\(^{30,36}\)

The mesenteric changes associated with closed-loop obstruction include radial configuration of bowel loops when vertically oriented; convergence of mesenteric vessels to a single point; close proximity of afferent and efferent limbs, often at the site of mesenteric convergence; a beak or whirl sign at the point of obstruction; C-shaped, U-shaped, or coffee bean configuration of the bowel loop with convergence toward the torsion; engorged mesenteric veins; mesenteric stranding or hemorrhage; ascites; portomesenteric venous gas; and perforation.\(^{20,30,36}\)

**Neoplasms**

Ischemic colitis is a well-recognized complication of obstructing colon carcinoma, developing in some 1% to 7% of cases.\(^{85}\) The bowel distention and elevated intraluminal pressure caused by the cancer produce vascular impairment in the mucosa and submucosa, leading to mucosal damage identical to ischemic colitis. The integrity of the colonic wall is further compromised by stagnation of fecal material above the tumor and by mechanical occlusion resulting from recurrent transient colonic twisting or torsion produced by the cancer. It is important to recognize ischemia proximal to colon cancer for two reasons. First, ischemia imperils a primary colonic anastomotic suture line, and postoperative complications occur in up to 25% of proximal ischemic colitis cases.\(^{30,86}\)
Second, ischemic colitis may lead to secondary congestive changes in the pericolic or perirectal fat that can simulate T3 invasion, leading to overstaging of tumor on CT. Similarly, the mural thickening that accompanies the ischemia may give a false impression of the length of tumor involvement, which can have important therapeutic implications in rectal cancers. One useful differentiating feature on CT is the fact that the mural thickening associated with ischemia causes submucosal edema, which leads to preservation of mural stratification and the so-called “target” sign. Colonic wall thickened by tumor is inhomogeneous in attenuation with loss of mural stratification. When a right-sided colon cancer obstructs the ileocecal valve, the resultant increased intraluminal pressure within the small bowel may lead to small intestinal ischemia.

**Drug Induced Enterocolitis**

A wide variety of orally and parenterally administered medications can cause small bowel ischemia and CI. Estrogens and oral contraceptives, which accelerate intravascular coagulation and inhibit fibrinolysis, are major risk factors for the development of thromboembolic events in the mesenteric, portal, and hepatic veins.

Several cardiac drugs can have a profound effect on the splanchic blood supply. Digitalis, inderal, dopamine, and vasopressin are commonly used drugs that can be powerful gut vasoconstrictors; they not only reduce mesenteric blood flow but cause contraction of precapillary sphincters in the intestinal mucosa.

Chemotherapeutic agents can lead to bowel ischemia and perforation, particularly in patients receiving long-term immunosuppression to prevent homograft rejection and in those patients receiving chemotherapy for leukemia (Fig. 34) and lymphoma.

**Radiation Enterocolitis**

Endarteritis obliterans is the microvascular lesion that develops in patients who receive abdominopelvic radiation therapy at doses of 45 to 60 Gy. The sigmoid colon, rectum, and terminal ileum are the sites most commonly affected by this progressive occlusive vasculitis that can lead to bowel ischemia, infarction, perforation, bleeding, and stricture and fistula formation.

Risk factors that predispose to the development of chronic radiation enteritis include hypertension, atherosclerosis, diabetes, prior abdominal surgery with adhesions, and a history of peritonitis.

The diagnosis of radiation enterocolitis is difficult, because the latent period between the radiation therapy and the development of radiation damage is usually 6 to 24 months but can be longer than 20 years. It may simulate clinically and radiologically recurrent cancer or adhesive disease.

**Trauma**

Trauma is an important cause of intestinal ischemia. Blunt trauma, especially attributable to motor vehicle collisions, can lead to seat-belt injury to the intestine and mesentery. The small bowel and its mesentery can be crushed between the seat belt and spine, causing a hematoma, transverse tear...
of the mesentery, and, ultimately, small bowel ischemia and infarction. The mesenteric tear interferes with local blood supply, and the resultant ischemia can be difficult to differentiate from non-ischemic mural thickening attributable to contusion. Additionally, the rapid deceleration can cause jejunal transection at a site within 20 cm of the ligament of Treitz, because the shearing force is directed between the relatively fixed proximal and more mobile distal jejunum. Penetrating trauma can directly injure the aorta, SMA, or IMA, leading to extensive hemoperitoneum or acute intestinal ischemia. If there is massive blood loss, the mesenteric blood supply to the gut is also compromised, leading to ischemic colitis or, less likely, enteritis, particularly in older individuals with advanced atherosclerotic change.

### Vasculitis

When larger vessels are involved, abdominal manifestations of vasculitis involving the mesenteric vessels may be indistinguishable from those of MI caused by emboli or thrombosis, except for associated evidence of systemic disease. Medium-sized arteritis, such as that found in polyarteritis nodosa, has a tendency to form aneurysms that can rupture and lead to intestinal and intraperitoneal hemorrhage. CT is helpful in differentiating MI caused by vasculitis from that caused by other conditions. Vasculitis preferentially tends to involve the small bowel (Fig. 36), whereas thromboembolic disease is more evenly distributed. Vascular thrombosis and atherosclerosis occur more frequently in those patients who have thromboembolic disease, whereas splenomegaly and genitourinary tract disease (eg, nephritis, cystitis, hydronephrosis) are more common in the vasculitis group. Accordingly, vasculitis should be suspected as the cause of MI in a younger patient; in those patients who have synchronous disease of the stomach, duodenum, or rectum; or when there is concomitant involvement of the small bowel and large bowel.

In patients who have polyarteritis nodosa, the pattern of bowel ischemia is usually multifocal and nonsegmental with long segments of bowel...
involvement. Primary duodenal involvement points to vasculitis as well.79,80

THERAPEUTIC OPTIONS

Patients who are suspected of having AMI require volume resuscitation; correction of hypotension, congestive heart failure, and cardiac arrhythmias; correction of acid-base and electrolyte abnormalities; and infusion of broad-spectrum antibiotics offering theoretic protection against the bacterial translocation that accompanies loss of mural integrity.26 Specific therapies are discussed next.

Superior Mesenteric Artery Embolism

Infusion of vasodilators (papaverine) and thrombolytic agents (eg, streptokinase, urokinase, recombinant tissue plasminogen activator) is useful in patients without signs of peritonitis.5 Embolism in the SMA induces a profound vasoconstriction of obstructed and nonobstructed branches of the SMA. If not promptly corrected, this vasoconstriction can become irreversible and persist even after the surgical removal of the embolus. Thrombolytic therapy is most likely to be successful when the thrombus is minor (distal to the origin of the ileocolic artery), when it is given within 12 hours of the onset of symptoms, and when the thrombus is only partially occluding.2 When signs of peritonitis are present, laparotomy is indicated with the intent to restore intestinal blood flow and resect necrotic bowel.7

Superior Mesenteric Artery Thrombosis

The treatment of choice is emergency surgical revascularization with continuous preoperative papaverine. Thrombolysis and angioplasty have also been used in this clinical setting.10

Nonocclusive Mesenteric Ischemia

In accordance26 with the pathophysiology of this disorder, there are two major principles of treatment: (1) correction of predisposing and precipitating factors and (2) treatment of the mesenteric vasoconstriction. In the absence of sepsis or peritoneal signs, vasodilator therapy is used. Patients are referred to laparotomy when there is no reaction to vasodilator infusion or if serum markers suggest necrosis or peritonitis.17 Infusional agents include papaverine, prostaglandin E1, phenoxybenzamine, tolazoline, and laevodosine.2,19

Chronic Mesenteric Ischemia (Intestinal Agina)

Percutaneous therapy is currently the therapy of choice and can also be used in critically ill patients. This involves the placement of a stent, usually in the SMA. If there is high-grade stenosis (>70%) of the celiac trunk and SMA, both vessels should be stented if technically feasible.12

Superior Mesenteric Venous Thrombosis

In asymptomatic patients in whom SMVT is diagnosed, a 3- to 6-month course of anticoagulation may be established. In patients who have

Fig. 36. Lupus vasculitis. Several proximal ileal loops demonstrate mural thickening and submucosal edema (arrows). These axial (A) and coronal (B) reformatted images also show edema in the mesentery.
symptomatic SMVT, anticoagulation with heparin for 7 to 10 days followed by an oral regimen of Coumadin for 3 to 6 months has been recommended. If peritoneal signs are present, laparotomy with embolectomy is indicated, followed by heparin and papaverine.¹,²,⁵

**SUMMARY**

Gastrointestinal tract ischemia can threaten bowel viability with potentially catastrophic consequences, including intestinal necrosis and gangrene. Because presenting symptoms and signs are relatively nonspecific and imaging findings may be confusing, the diagnosis of intestinal tract ischemia requires a high index of suspicion. It is important to attempt to determine the cause of the intestinal ischemia and differentiate between intestinal ischemia and infarction. The early inclusion of bowel ischemia and infarction in the differential diagnosis of patients with abdominal pain accompanied by an aggressive diagnostic and therapeutic approach may be the only way to improve patient survival in this potentially lethal disorder.

**REFERENCES**

28. Romano S, Romano L, Grassi R. Multidetector row computed tomography findings from ischemia to


