Imaging of Ischemia, Obstruction and Infection in the Abdomen

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INTRODUCTION

Intestinal obstruction and intra-abdominal infection associated with the gastrointestinal tract account for a huge proportion of emergency surgical admissions with abdominal symptoms. The former alone accounts for approximately 20% of admissions in this category.\textsuperscript{1–3} Intestinal ischemia is uncommon but still carries a mortality in excess of 70%.\textsuperscript{4} As a result of the frequency and gravity of the conditions, timely diagnosis of these entities by emergency radiologists is of key importance. This article examines the imaging approach in patients suspected of having the aforementioned conditions, shows key findings, and discusses potential complications.

NORMAL ANATOMY AND IMAGING TECHNIQUES

Knowledge of the vascular supply and drainage of the gastrointestinal tract is of key importance in understanding intestinal ischemia. The celiac trunk, superior mesenteric artery, (SMA) and smaller infraduodenal inferior mesenteric artery (IMA) must be scrutinized routinely on abdominal imaging studies. The celiac trunk supplies the foregut, from the distal esophagus to the midpart of the descending duodenum. The SMA, the artery of the midgut, supplies the gastrointestinal tract from the middle of the second part of the duodenum to the junction of the middle and distal

KEYWORDS

- Intestinal obstruction
- Intestinal ischemia
- Computed tomography
- Acute abdomen
- Bowel
- Colon

KEY POINTS

- Computed tomography (CT) is the modality of choice in investigation of the acute abdomen in nonpregnant adults but ultrasonography and MR imaging in particular are increasing being used.
- In suspected bowel ischemia, multiphase CT imaging with the use of negative oral contrast in place of positive oral contrast, where possible, is recommended.
- Complete mesenteric arterial occlusion without reperfusion results in bowel thinning and minimal mural enhancement, whereas other causes of intestinal ischemia lead to bowel wall thickening, intramural hemorrhage, and mural hyperenhancement.
- Key findings to interpret regarding bowel obstruction are the location and cause of transition, grade of obstruction, the presence of closed-loop obstruction, and the presence of ischemia.

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thirds of the transverse colon, with the IMA, the hindgut artery, supplying the remainder. Collater-
alization between the SMA and IMA via the mar-
ginal artery of Drummond is variable. In contrast,
there is a rich plexus of collaterals between the ce-
liac trunk and SMA. The bowel receives approxi-
mately 20% of cardiac output with the mucosa
receiving two-thirds of this,5,6 thus explaining
why the mucosa and submucosa are most sensi-
tive to changes in supply in the setting of ischemia.
The relative locations of small and large bowel
loops are of significance in assessing for internal
hernia, a challenging diagnosis that is frequently
a closed-loop obstruction. It is important to care-
fully evaluate the anatomic relationships of the
small bowel loops in relation to the ascending/de-
scending colon or transverse colon respectively. In
addition, the paraduodenal spaces should be
devoid of extra bowel loops. Intestinal malrotation,
which is associated with midgut volvulus and inter-
nal herniation, may occur when the small bowel
mesentery is short, as signified by finding that
the third part of the duodenum does not cross
the midline. In such cases the small bowel is char-
acteristically located on the right side of the
abdomen and the colon principally left sided. In
addition, the SMA and superior mesenteric vein
relationship may be reversed.7
Malrotation with nonrotation also results in an
appendix that lies to the left of midline, a finding
that is also present in situs inversus.
The vermiform appendix measures a mean of
11 cm and the tip is most commonly retrocecal
(74%), although the remainder have a variable
location.8 Meckel diverticulum is an anatomic
variant that results from persistence of part of the
omphalomesenteric duct, is present in 2% of the
population, and is located approximately 60 cm
(2 feet) from the ileocecal valve on the antimesen-
teric border.9
Normal bowel diameter may measure 2.5 cm for
the small intestine, 9 cm for the cecum, and 6 cm
for the remainder of the colon.10,11

**Imaging Techniques**

**Radiography**
Abdominal radiography is frequently used in
assessment of the acute abdomen. It has a re-
ported sensitivity of 69% to 80% for bowel
obstruction12–15 but is insensitive in assessing for complications or cause. In addition, abdominal ra-
diographs are neither sensitive nor specific for
detection of intestinal ischemia or infectious/in-
flammatory conditions such as diverticulitis, col-
tis, or appendicitis. The erect chest radiograph
remains an essential part of assessment and
detection of pneumoperitoneum in suspected hol-
low viscus perforation.

**Computed tomography**
Multidetector (MD) computed tomography (CT) is
the main modality for the diagnosis of bowel
obstruction and ischemia because of its availabil-
ity, speed, sensitivity, and specificity.1,2,5,16–24
MD CT has a sensitivity in excess of 80% for intesti-
al ischemia detection.1 MD CT accuracy of 95%
is reported for detection of high-grade small bowel
obstruction, although accuracy is decreased for
low-grade obstruction.25 Sensitivity in excess of
90% is reported for large bowel obstruction.26,27
Sensitivity and specificity for appendicitis are
also in excess of 90%.20,28

**MR imaging**
MR imaging is being increasingly used in detection
of bowel obstruction, ischemia, and infection. MR
imaging has a sensitivity that is similar to CT in as-
sessing bowel obstruction29,30 and ischemia.31,32
In addition, MR imaging is comparable with CT in
infectious/inflammatory conditions such as
appendicitis and diverticulitis.20,33,34

**Ultrasonography**
In children and pregnancy, ultrasonography is the
first-choice modality in assessment of suspected
appendicitis. Ultrasonography has a complimen-
tary role to CT and MR imaging in assessing small
bowel obstruction and suspected ischemia; how-
ever, its main strengths include ready availability
in most hospitals, avoidance of exposure to
ionizing radiation, and the portable nature of the
modality, which means that it can be performed
at the bedside in critically ill patients.30,35,36

**Fluoroscopy**
Angiography is rarely used currently for diagnosis in
intestinal ischemia but is used during endovascular
therapy. In the past, contrast enemas and barium
follow-through studies were used as part of large
and small bowel obstruction evaluation but current
practice means that use has been superseded.
Many investigators advocate a 3-phase protocol
for assessing suspected acute mesenteric
ischemia. The unenhanced phase can be valuable
if mural hyperdensity related to intramural hemor-
rhage is identified. This finding is a specific indica-
tor of ischemia but is not required in all cases for
the diagnosis of bowel ischemia.37 The absence
of positive oral contrast is essential for detection
of bowel wall enhancement in suspected
ischemia. Intraluminal fluid acts as a good nega-
tive oral contrast agent in cases of bowel obstruc-
tion. Positive oral contrast in the setting of
suspected appendicitis has a marginal accuracy benefit, particularly in patients of low body mass index, but its use may result in some delay in imaging of the patient. MD isometric thin-section imaging with multiplanar reconstructions is essential in bowel wall and vasculature assessment; review of reconstructions in the coronal plane is especially useful for investigating cases of bowel obstruction.

### IMAGING FINDINGS/PATHOLOGY

#### Ischemia

Acute intestinal ischemia is an uncommon condition, accounting for approximately 0.1% of hospital admissions. However, all-cause mortality remains approximately 70%, a figure that increases to closer to 90% when infarction has become established.

The causes of acute mesenteric ischemia are as follows:

- Arterial occlusion (50%–60% of cases)
- Venous thrombosis (5%–10% of cases)
- Small vessel disease: vasculitides, radiation, chemotherapy induced
- Mechanical bowel obstruction
- Nonocclusive mesenteric ischemia (shock bowel)

Key imaging findings include:

- Abnormal bowel wall thickness
- Increased bowel wall attenuation from submucosal hemorrhage on the unenhanced CT phase
- Abnormal wall enhancement
- Increased luminal diameter
- Mesenteric arterial or venous occlusion
- Ascites and mesenteric fluid or stranding
- Pneumatosis intestinalis (gas in the bowel wall)
- Pneumatosis portalis (gas in the portovenous system)
- Pneumoperitoneum

#### Bowel wall thickening

Normal bowel wall thickness ranges from 3 to 5 mm. The degree of bowel wall distention is a factor in determining whether the bowel wall is truly thickened and caution is needed when the bowel is collapsed or overdistended. Wall thickening from mucosal/submucosal edema or hemorrhage is the commonest finding in intestinal ischemia, and it is present in most cases that are not caused by acute arterial occlusion; however, bowel wall thickening is not specific for intestinal ischemia (Fig. 1). The degree of

![Fig. 1.](image-url) Axial contrast-enhanced CT scan in a 47-year-old female patient with acute abdominal pain. A hypervascular mesenteric carcinoid mass (A, arrowheads) is seen in the mesentery with a marked surrounding desmoplastic reaction. The adjacent small bowel shows signs of venous ischemia (A and B, arrows) with wall thickening, submucosal edema, and mucosal hyperenhancement.
wall thickening does not correlate with the severity of ischemic wall damage. However, in the setting of acute arterial occlusion, the wall is frequently thinned (paper-thin wall) (Fig. 2) and this seems to be explained by the absence of vascular flow required to produce edema or hemorrhage.

**Bowel wall attenuation on control images**

Increased bowel wall attenuation from submucosal hemorrhage on unenhanced CT images is a specific sign for bowel ischemia. The sign is rarely seen in isolation. Like bowel wall thickening, submucosal hemorrhage is rarely present in the setting of arterial occlusion.

**Abnormal wall enhancement**

Reduced or absent bowel wall enhancement, which is an insensitive but specific sign, may be seen in arterial occlusion without reperfusion (see Fig. 1). Reduced enhancement is also associated with infarction. In cases of incomplete arterial occlusion, arterial occlusion with reperfusion, and other causes of intestinal ischemia, increased bowel wall enhancement is more commonly evident.

**Increased luminal diameter**

Bowel distention is seen in up to 90% of bowel infarction cases but its presence is variable in cases of reversible ischemia. The distention is predominantly caused by increased intraluminal fluid and ileus from neuroenteric plexus dysfunction.

**Mesenteric arterial or venous occlusion**

The finding of arterial (see Fig. 2; Fig. 3) or venous occlusion (Fig. 4) in the mesenteric vessels indicates the cause rather than an effect of mesenteric ischemia. Embolus rather than thrombosis is more common in the SMA in the acute setting. Infarcts may also be seen in the other viscera, such as the kidneys or spleen, as a result of emboli. In the chronic setting, atherosclerosis dominates in terms of cause but incidental mesenteric atherosclerotic disease is common in asymptomatic elderly patients. On the venous side, thrombosis may be caused by a local trigger such as intra-abdominal infection; neoplasm or inflammation; portal hypertension; or a systemic tendency for clot formation, such as antiphospholipid syndrome or oral contraceptive pill use.

![Fig. 2](image-url)

**Fig. 2.** Axial and sagittal CT images, from an 81-year-old man with a history of atrial fibrillation, who attended the emergency department with acute severe constant abdominal pain. Acute complete occlusion of the SMA (A, white arrows) is identified along with poorly enhancing small bowel walls (B, arrowheads). Distal small bowel loops show hyperenhancement (C, curved arrows) suggesting some reperfusion but ongoing ischemia. Pneumatosis portalis (portovenous gas) is seen in the perienteric veins anteriorly (D, black arrow).
Ascites and mesenteric fluid or stranding
As with many other intra-abdominal disorders, the presence of free fluid is a nonspecific sign in the setting of suspected intestinal ischemia. The presence of free fluid in the mesentery (see Fig. 4) is reportedly more sensitive (88% vs 75%) and specific (90% vs 76%) than the finding of free ascites in the setting of ischemia from small bowel obstruction. The presence of ascites and/or mesenteric stranding may increase the likelihood of infarction in the setting of arterial occlusion. There should be increased suspicion for ischemia.

Fig. 3. Axial and sagittal arterial phase CT images of a 48-year-old female patient with long-term cognitive impairment who attended with abdominal pain and distention. Multiple dilated bowel loops from stercoral large bowel obstruction are seen along with pneumatosis intestinalis (A, arrowheads) and pneumatosis portalis (portovenous gas) (A, curved arrows). The stercoral obstruction led to severe abdominal compartment syndrome resulting in obstruction of the abdominal aorta (B, arrow) with resultant ischemia.

Fig. 4. Coronal reformatted portovenous phase CT images of a 46-year-old female patient with acute abdominal pain and vomiting. Thrombus is identified in the superior mesenteric vein (A, arrows). There is resultant ischemia of the small bowel with stranding and fluid in the mesentery (B, curved arrow) and mural thickening and hyper-enhancement in the mildly dilated small bowel loops (B, arrowheads).
in the presence of high-density ascites related to bowel wall compromise and subsequent hemorrhage.

**Pneumatosis intestinalis, pneumatosis portalis, and pneumoperitoneum**

Detection of pneumatosis intestinalis (gas in the bowel wall) at CT is best performed by using lung-window settings (see Fig. 3). The dependent part of the wall is the best location to detect this finding because the dependent luminal contents can be used to improve conspicuity and localization. Gas may appear as bubbles or rims of gas separating the wall layers. In addition, images have to be scrutinized closely to assess for pneumatosis portalis (gas in the mesenteric or portal veins) (Fig. 5). Distinguishing intrahepatic portovenous gas (Fig. 6) from pneumobilia can be achieved by following the course of the vessel or channel involved. In addition, pneumobilia is more frequently central and does not involve the subcapsular ~2 cm of parenchyma, unlike portovenous gas, which is frequently peripheral and extends much closer to the capsule. In the correct clinical context, the presence of pneumatosis intestinalis or pneumatosis portalis has specificities approaching 100% for ischemia detection, particularly when seen with other findings such as abnormal bowel wall enhancement. The presence of both entities has a reported specificity of 83% for infarction, although the sensitivity is only 17%. However, there are multiple other causes of these entities. Alternate, more benign causes of pneumatosis intestinalis or portalis include systemic lupus erythematosus, scleroderma, barotrauma, asthma, chronic obstructive pulmonary disease, intra-abdominal infection, bowel obstruction, iatrogenic bowel distention, and inflammatory bowel disease. The presence of free air in the setting of intestinal ischemia strongly suggests transmural infarction.

**Obstruction**

Obstruction of the small intestine accounts for 80% of mechanical small bowel obstructions, with the remaining 20% being caused by colonic obstruction. The typical presentation is with abdominal pain, distention, vomiting, and absolute constipation.

**Small bowel obstruction**

Adhesions from prior abdominal surgery represent the commonest cause of small bowel obstruction (SBO), accounting for up to 75% of cases, with hernias being the next commonest cause. Further causes can be broadly categorized by cause: congenital (eg, ileal atresia, midgut volvulus), extrinsic (eg, volvulus, compression from mass), bowel wall disorders (eg, stricture, intussusception, tumor), and intraluminal disorders (eg, meconium ileus, gallstones, foreign body, bezoar).

**Large bowel obstruction**

Mechanical obstruction of the colon is most commonly caused by colon cancer, which accounts for 50% to 60%. Other causes include volvulus, diverticulitis, inflammatory bowel disease, radiation, ischemia, fecal impaction, or hernia.

**Imaging findings**

Considerations include:

- Proximal bowel distention
- Distal collapsed bowel
- String-of-pearls sign
- Transition point beak sign
- Grading
- Small bowel feces
- Potential closed loop
- Visible-cause SBO
- Visible-cause large bowel obstruction
- Possible mesenteric ischemia

**Bowel distention with distal collapse**

The key finding in SBO (Fig. 7) or large bowel obstruction (Fig. 8) is dilatation of the loops proximal to, and collapse of loops distal to, a point of obstruction. Small bowel diameter in excess of 2.5 cm and large bowel diameter in excess of 6 cm (9 cm for cecum) are considered dilated. On plain radiography, small bowel loops are dilated centrally with visible transverse valvulae conniventes, whereas the large bowel is peripheral.
in location with haustral folds that are seen to incompletely traverse the wall. The location of distal bowel collapse helps decide whether the obstruction is in the small or large bowel. If the obstruction is in the colon but the ileocecal valve is competent, the small bowel is not dilated. It is important to differentiate mechanical obstruction from paralytic ileus and pseudo-obstruction. Paralytic ileus typically occurs in postoperative patients, is often painless with absent bowel sounds, and shows small and large bowel distention. In contrast, colonic pseudo-obstruction, known as Ogilvie syndrome in the acute setting, is similar in presentation to large bowel obstruction in that the patients often have pain and distention but frequently have a history of chronic constipation.\textsuperscript{56,57} Imaging in pseudo-obstruction shows a dilated proximal colon; distal collapse; and, classically, a caliber change at the splenic flexure, without a lesion at the site of transition.\textsuperscript{56} However, the site of transition can be anywhere in the colon.\textsuperscript{57} In this syndrome, the small intestine may be dilated if the ileocecal valve is incompetent.

**String-of-pearls sign**

The string-of-pearls sign is seen on lateral decubitus or erect abdominal radiographs, in addition to CT, when small bubbles of gas are trapped between the valvulae conniventes. It is reported

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**Fig. 6.** Axial CT images from a 74-year-old man with an obstructing perforated gastric tumor (B, arrows), resulting in mesenteric ischemia with intrahepatic portovenous gas (A, arrowheads). Pneumoperitoneum is also evident (curved arrows).

**Fig. 7.** Coronal reformatted CT image in a 52-year-old man with high-grade adhesional SBO showing dilated proximal small bowel loops (arrowheads) and a beak sign (arrows) at the site of obstruction.

**Fig. 8.** Axial CT image in a 61-year-old female patient with an obstructing sigmoid tumor (arrows) that showed shouldering and asymmetrical bowel wall thickening and proximal colonic dilatation (arrowhead).
to be specific for SBO and absent in the setting of adynamic ileus or gastroenteritis.\textsuperscript{58}

**Transition point beak sign**

Typically, transition from dilated proximal bowel to collapsed distal bowel occurs at the site of obstruction, although this is not always the case. When an abrupt transition occurs, the bowel forms a beak sign whereby the dilated proximal bowel quickly tapers to the transition point, resulting in the shape of a beak (see Fig. 7; Fig. 9). In a closed-loop obstruction, 2 transition point beak signs may be seen. The absence of a beak sign is associated with successful nonoperative management in the setting of adhesion-related SBO, along with the presence of a small bowel feces sign and an anterior parietal adhesion.\textsuperscript{59} In contrast, 2 or more beak signs, a whirl sign, a C-shaped or U-shaped bowel loop, and high-grade obstruction are more likely to require surgical management in the setting of adhesional SBO.\textsuperscript{59,60}

**Grading**

Bowel obstruction is typically graded either into complete or high-grade obstruction, or partial or low-grade obstruction. Incomplete obstruction is suggested when the transition is not abrupt, the distal bowel is not fully collapsed, the proximal bowel is only mildly distended, and ingested contents such as positive oral contrast traverse the obstruction.\textsuperscript{1,10,25} In contrast, complete obstruction is the opposite of these entities. High-grade obstruction is more likely to require operative management in the setting of adhesion-related SBO.\textsuperscript{59}

**Small bowel feces**

The presence of solid heterogeneous feces-like particulate matter mixed with gas in the small bowel proximal to the site of obstruction is a useful sign in the setting of bowel obstruction (Fig. 10). The main usefulness seems to be in locating the site and cause of obstruction.\textsuperscript{1,61} It is associated with successful nonoperative management in the setting of adhesion-related SBO.\textsuperscript{59} However, it is not pathognomonic for obstruction because it is reported to be seen in up to 6% of asymptomatic studies.\textsuperscript{62}

**Potential closed loop**

Closed-loop obstruction occurs when the bowel is obstructed at 2 different points along its length, usually at a single location and from a single cause. It is an important diagnosis to make because it carries a higher risk of bowel ischemia and usually requires prompt surgical

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**Fig. 9.** Coronal T2-weighted fat-saturated MR imaging in a 36-year-old male patient with high-grade SBO from a stricture secondary to underlying Crohn disease (arrows). The proximal loops are dilated (arrowheads) and there is a benign stricture at the site of transition.

**Fig. 10.** Axial T2-weighted fat-saturated MR imaging in a 39-year-old male patient showing a small bowel feces sign (A, arrows), proximal to a strictured segment of distal ileum (B, arrowhead) that has an adjacent phlegmon (B, curved arrows).
Two beak signs may be seen from the 2 points of obstruction, along with a radial or U-shaped configuration of the bowel loops. A whirl (or whirlpool) sign may also be seen, in which the mesenteric vessels have a swirling appearance from torsion of these structures around themselves.\textsuperscript{64} Closed-loop obstruction is associated with hernias (particularly internal hernias) (Fig. 11), midgut volvulus, cecal volvulus (Fig. 12), and sigmoid volvulus (Fig. 13). A proximal colonic obstruction with a competent ileocecal valve may also behave like a closed-loop obstruction.

**Visible-cause small bowel obstruction**

Differentiation of SBO from large bowel obstruction is fundamental in assessing the cause, with the location of the transition point being the key.

**Visible-cause large bowel obstruction**

With regard to large bowel obstruction, the cause of exclusion is a colonic neoplasm, given that it is the cause in 50% to 60% of cases.\textsuperscript{10,27} A circumferential or eccentric short segment enhancing mass is usually evident in the setting of colon cancer, and is most frequently located in the sigmoid colon (see Fig. 8). Diverticulitis or diverticular stricture involves a longer segment with a greater degree of pericolonic fat stranding and diverticula. If CT findings are equivocal, MR imaging is reported to have a superior sensitivity and specificity for differentiating colonic tumor from diverticulitis.\textsuperscript{34} If large bowel obstruction is caused by cecal (see Fig. 12) or sigmoid volvulus...
In cecal volvulus, the cecal pole has an ectopic location, pointing toward or located in the left upper quadrant. Sigmoid volvulus classically has an abnormal loop of large bowel extending out of the pelvis, forming a coffee bean shape. The CT findings are in keeping with those on plain radiographs in addition to closed-loop obstruction.

Possible mesenteric ischemia
Signs of bowel ischemia, as outlined earlier, may be seen in association with bowel obstruction. Ischemia is more likely to occur in the setting of closed-loop and high-grade obstruction.

**GASTROINTESTINAL TRACT INFECTION**

**Small Bowel**

**Infectious enteritis**
Acute infectious enteritis (or gastroenteritis) is usually self-limiting and typically does not require imaging. However, imaging may be performed in atypical presentations, with chronic symptoms and in immunosuppressed patients. Causative organisms include bacteria (*Salmonella*, *Yersinia*, *Escherichia coli*, *Campylobacter*, *Mycobacterium tuberculosis* [tuberculosis (TB)], protozoa (*Giardia lamblia*, *Cryptosporidium*), and viruses (*Cytomegalovirus*, *Norovirus*, *Rotavirus*). *Giardia* has a...
preponderance for the proximal small bowel, the CT findings of which are nonspecific, with circumferential wall thickening and lymphadenopathy being the commonest. Most pathogens usually affect the ileum. Ninety percent of TB occurs in the ileum or cecum because of the stasis and lymphatic abundance. Ileal TB usually results in asymmetric wall thickening in the ileum and multiple large low-density/necrotic nodes on CT. In contrast, Crohn disease is more frequently associated with small nodes, fibrofatty mesenteric proliferation (so-called creeping fat), and potential for fistula formation as a result of the transmural inflammation. Salmonella gives a similar appearance to Crohn disease in terms of segmental circumferential wall thickening.

![Fig. 15. Abdominal radiograph and axial CT images on a 59-year-old patient with gallstone ileus. Dilated small bowel loops are seen (A, arrows) along with pneumobilia (A and B, curved arrows) on the radiograph and CT images. The noncalcified gallstone was identified in the lower abdomen (C, arrowheads).]

![Fig. 16. Coronal T2-weighted fat-saturated MR imaging in a 65-year-old male patient with SBO. Dilated small bowel (arrows) is seen superior to a shouldered circumferential lesion (arrowheads) with collapsed bowel distally (curved arrow). Histology following surgical resection confirmed adenocarcinoma.]

![Fig. 17. Axial CT image from a 67-year-old patient with SBO from an incisional hernia. Dilated bowel loops (arrowheads) are seen proximal to a transition point beak sign (arrow) within the hernia neck (curved arrows).]
which often presents with acute pain and tenderness, typically results in mild symmetric bowel wall thickening, aphthous ulceration, and mucosal nodularity. Typhlitis (neutropenic enterocolitis) is discussed later.

**Meckel diverticulitis**
Approximately 2% of the population has a Meckel diverticulum. Many of these true diverticula are incidental findings but complications or symptoms develop in 4% to 40%. Diverticular infection/inflammation or diverticulitis (Fig. 20) accounts for up to 30% of complications. Gastrointestinal bleeding, intussusception, obstruction, perforation, and band strangulation are other common presentations or complications. The right lower quadrant or close to midline are the most likely locations of a Meckel diverticulum at CT examination in the setting of diverticulitis. It appears as a blind-ending pouch of approximately 2.5 cm in (outer wall) diameter and approximately 3.3 cm in length with a mean wall thickness of almost 4 mm. In the setting of Meckel diverticulitis, inflammatory changes in the surrounding fat and some free fluid are evident. Other complications, such as bowel obstruction or perforation, may also be present, making the identification of a Meckel difficult.

**Colon**

**Appendicitis**
Acute appendicitis is a common cause of acute abdominal pain, particularly in children and
adolescents, with a lifetime risk of developing acute appendicitis of 6% to 8%. Graded compression ultrasonography is usually the first-line imaging investigation in children, young adults, and pregnancy. The user-dependent nature of the modality likely contributes to variable reported sensitivities of 12% to 90% and the commonly encountered lack of confidence in sonographic diagnosis, although high specificities in excess of 85% are routinely reported. MR imaging has reported accuracies, in excess of 90%, that are similar to those of CT in diagnosis of acute appendicitis. As a result, the use of MR imaging in this setting continues to increase. Regardless of the imaging modality, typical findings include an appendix diameter of greater than 6 mm, luminal obliteration, noncompressible appendix, appendicolith, periappendiceal inflammatory fat changes, free fluid, inflammatory phlegmon, and abscess (Figs. 21 and 22).

Colitis and cecitis
Like infectious enteritis, patients with infectious colitis often do not require imaging, unless there is doubt about the diagnosis or complications are suspected. The causes are similar to those of infectious enteritis with the addition of Clostridium difficile (pseudomembranous colitis), the protozoan infection amebiasis (Entamoeba histolytica), the parasitic infection schistosomiasis, Chlamydia trachomatis, Neisseria gonorrhoeae, and Herpes species.

In Western countries, bacteria most commonly cause infectious colitis. In particular, the number of cases of pseudomembranous colitis has markedly increased in recent times as a result of increased usage of broad-spectrum and prophylactic antibiotics. C difficile is not a normal bowel commensal organism but colonizes the colon after antibiotics or chemotherapy have disrupted the normal colonic biology. The bacteria produce 2 toxins (A and B) that result in the colonic inflammation. Typical CT findings of pseudomembranous colitis (Fig. 23) are wall thickening...
of 11 to 14 mm, submucosal low attenuation from edema, and an irregular mucosal contour. The so-called accordion sign, in which positive oral contrast interspersed between thickened haustral folds is said to look like an accordion, may also be seen, although it can be seen in any severe colitis (Fig. 24). In addition, the target sign, in which postcontrast enhancement of the mucosa and muscularis propria with intervening submucosal edema has the appearance of a target, may also be evident (see Fig. 23). The fat-halo sign is similar but occurs when the submucosa shows fat deposition, although it predominantly occurs in ulcerative colitis.

Amebic colitis occurs as a result of colon invasion from the trophozoite form of the organism. The inflammation usually starts in the cecum, often involves the rectum, and may result in diffuse colitis. The most distal part of the ileum may be involved. Amebic colitis results in wall thickening, ulceration, pseudopolyps, and skip lesions that are similar to Crohn colitis. The classic coned cecum, in which the cecum resembles an inverted cone pointing at the appendix base, develops after subacute infection. However, the cone-shaped cecum can be seen in multiple inflammatory, infectious, or neoplastic conditions. Amebomas are focal areas of granulation that can closely mimic colonic neoplasms, particularly if hepatic amebic disease is present. Residual colonic scarring is common after infection.

There is huge overlap in the location and appearances of most bacterial and viral infectious colitides, hence stool culture and history are of key importance in identifying the causative organism. However, certain patterns of involvement are associated with certain organisms. Cytomegalovirus and E. coli often result in diffuse colitis; shigella and salmonella colitides are frequently confined to the right colon; schistosomiasis is usually confined to the left colon; and C. trachomatis, N. gonorrhoeae, and Herpes species are confined to the rectosigmoid region.

Typhilitis, also known as neutropenic enterocolitis, has an incidence of up to 26% in certain patient groups undergoing chemotherapy, but can occur in any immunosuppressed patient. The mortality remains in excess of 50% despite increased awareness and aggressive management with bowel rest, fluids, broad-spectrum antimicrobial therapy, and blood products if necessary. It is likely multifactorial in cause with neutropenia, bowel wall injury from treatment or neoplasm, and intramural hemorrhage from thrombocytopenia resulting in invasive polymicrobial infection from commensal bowel organisms. Clinical symptoms and signs include abdominal pain, localized tenderness, distention, nausea, vomiting, bloody diarrhea, and fever. CT is the imaging modality of choice and shows increased wall thickening (typically ~7 mm) involving the cecum, and often the ileum and remaining colon (Fig. 25).
Pneumatosis, mucosal enhancement, free fluid, and fat stranding may also be seen. Neutropenic patients are also at increased risk of other colitides, including cytomegalovirus colitis and pseudomembranous colitis. For any infectious colitis, the main differential diagnoses include noninfectious causes of colitis, such as inflammatory bowel disease, with radiation colitis and ischemic colitis being less common. Diverticulitis, neoplasm, or graft-versus-host disease are additional alternate diagnoses to consider. Amebic, TB, and pseudomembranous colitis most closely mimic inflammatory bowel disease. Radiation colitis is likely to be suspected from the history and ischemic colitis is found in the older age group, with a vascular distribution and features mentioned earlier. Bowel wall thickness, bowel wall attenuation, degree of wall thickening, symmetry of wall thickening, length of segment involved, and associated findings such as stranding, lymphadenopathy, and abscess are key factors that aid in delineating the cause.

Complications of colitis are readily assessed using CT. They include perforation, abscess, ischemia, and toxic megacolon. Toxic megacolon is among the most serious and is most commonly caused by ulcerative colitis or, increasingly, pseudomembranous colitis, but may be associated with any severe form of colitis. Nitric oxide and interleukins seem to be important in the pathogenesis. The key imaging findings are dilatation of the colon, typically the transverse colon, to greater than 6 cm in diameter with loss of the haustral folds and wall thinning, findings that help distinguish it from acute severe colitis. Radiographs are useful in assessing bowel diameter in the setting of toxic megacolon, but CT is the superior investigation because it can detect small perforations and abscesses in addition to making a more accurate diagnosis.

Diverticulitis

Colonic diverticula are acquired pseudodiverticula that may result from increased intraluminal pressure and occur at weak points in the circular muscle where mucosal vasculature penetrates on the mesenteric wall. They predominantly occur in the descending and sigmoid colon but can be found anywhere in the colon apart from the rectum. Diverticulosis is common in the Western world, is predominantly a disease of older adults, and is found in greater than 70% of people more than 80 years of age and approximately 5% of people at 40 years of age. Traditional thinking about cause was that it occurred in patients lacking in dietary fiber, but studies to confirm this are conflicting. Diverticulitis occurs when one or more of these diverticula become obstructed at the neck, resulting in stasis, inflammation, infection, and perforation. Diverticulitis occurs in between 1% and 25% of patients with diverticular disease, although recent studies suggest that rates of development of acute diverticulitis are at the lower end of that range. It is among the commonest inpatient gastrointestinal diagnoses. Typical presentations include symptoms and signs of left iliac fossa pain and tenderness, altered bowel habit, nausea, pyrexia, possibly a palpable mass, and increased inflammatory markers. CT is the investigation of choice in suspected acute diverticulitis. Findings include mesenteric fat stranding that is disproportionate to the degree of colonic wall thickening, mural hyperemia, segmental involvement, fluid accumulation in the mesenteric root (the comma sign), mesenteric hypervascularity (the centipede sign), and the presence of diverticula. Associated findings may include an abscess (present in up to 30% of cases) (see Fig. 26; Fig. 27), localized perforation, generalized perforation, or fistula. Fistulae may be from the involved colon to colon, small bowel, vagina, bladder, or skin. Classification of the severity of acute diverticulitis is advantageous and several scales exist. One such surgical scale is the Hinchey classification: stage Ia, phlegmon; stage Ib, with pericolic or mesenteric abscess; stage II, with walled-off pelvic abscess; stage III, with generalized purulent peritonitis; stage IV, with generalized feculent peritonitis. Although the described classification system has a surgical basis and classification may not be used commonly in practice, the elements should be mentioned in the radiology report to indicate severity.

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Differentiating diverticulitis from colonic neoplasm is challenging. The presence of diverticula, disproportionate fat stranding, comma or centipede signs, mesenteric root fluid, mild smooth concentric wall thickening, gradual transition from normal to abnormal bowel thickness, long (5–10 cm) affected segment, and minimal or no lymphadenopathy are associated with acute diverticulitis. In contrast, the absence of diverticula, fat stranding, comma or centipede signs, and mesenteric fluid along with the presence of lymphadenopathy, bowel wall shouldering, increased density of the focal bowel wall thickening or mass after contrast administration, and irregular eccentric short segment (<5 cm) wall thickening is associated with colonic neoplasm. MR imaging may have an advantage compared with CT in differentiating tumor from diverticulitis. Epiploic appendagitis is another important differential diagnosis in patients with suspected diverticulitis. The key finding is an ovoid fat-density lesion with surrounding inflammation adjacent to the colon with little if any wall thickening (Fig. 28).

All patients should undergo endoscopy after an episode of diverticulitis, predominantly to ensure that there is no underlying neoplasm.

Sepsis from gastrointestinal perforation
Hollow viscus perforation and associated abdominal sepsis remains a common problem but the radiological diagnosis of pneumoperitoneum can readily be made on chest and/or abdominal radiographs. Subdiaphragmatic free air on erect chest radiography, or the double wall (Rigler sign), lucent liver sign, or football sign on abdominal radiography indicates pneumoperitoneum (Fig. 29). The lateral decubitus abdominal radiograph is rarely required but useful if the erect chest radiograph is inclusive, CT is unavailable, or the patient is too unstable for transfer (Fig. 30). Perforated peptic ulcer disease (PUD) is less common than in the era before proton pump inhibitor therapy but it is still a leading cause of pneumoperitoneum. In one recent US series, PUD (16%), diverticulitis (16%), trauma (14%), malignancy (14%), bowel ischemia (10%), appendicitis (6%), and endoscopy (4%) were the leading causes of pneumoperitoneum when postoperative pneumoperitoneum was excluded. Further imaging is often not required if the clinical picture clarifies the likely cause and if further imaging would delay surgical management. CT imaging has a role in planning the type of operative treatment, the prognosis, and in assessing those patients who have clinical symptoms of peritonitis but no radiographic signs of perforation. One scenario in which there may be symptoms of peritonitis but no radiographic signs of perforation, is in the setting of PUD perforation into the lesser sac (Fig. 31). In this scenario, a collection of gas and fluid is often seen in the lesser sac, posterior to the stomach. When CT imaging is performed in the setting of known pneumoperitoneum, a careful search for the likely causes should be completed. The

Fig. 27. Axial CT images of 75-year-old male patient with severe complex pelvic sepsis from acute diverticulitis. Large abscesses are seen in the pelvis (B, arrows), extending into the buttock via the sciatic notch. In addition, gas is seen in the spinal canal and right paraspinal muscles (A, arrowheads) as a result of local spreading of infection.

Fig. 28. Axial CT image of a 29-year-old man with right-sided epiploic appendagitis. The typical central ovoid fat density is seen with a surrounding halo of inflammation (arrows).
presence of fat stranding, abscess, wall thickening, and potential mural defect helps to localize the site of perforation (Fig. 32).

**DIAGNOSTIC CRITERIA**

**Intestinal ischemia**
1. Abnormal bowel wall thickness
   a. Paper-thin in acute complete arterial occlusion without reperfusion
   b. Diffusely thickened, possibly with submucosal hemorrhage in other causes of ischemia
2. Abnormal wall enhancement
   a. Absent or hypoenhancement in acute complete arterial occlusion without reperfusion
   b. Hyperenhancement in other causes of ischemia
3. Increased luminal diameter
4. Arterial or venous occlusion
5. Mesenteric fluid or stranding
6. Pneumatosis intestinalis
7. Pneumatosis portalis

**Obstruction**
1. Dilated proximal bowel (>2.5 cm for small bowel, >9 cm for cecum, >6 cm for remaining colon)
2. Collapsed or nondistended bowel distally
3. Transition point with or without beak sign
4. Possible visible cause at transition point

**Infection**
1. Enteritis
   a. Circumferential wall thickening

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Fig. 29. Erect frontal chest radiograph and frontal abdominal radiograph in a 61-year-old man with pneumoperitoneum from colonic perforation. A large volume of subdiaphragmatic free air is seen (A, arrowheads). Multiple double wall (Rigler) signs are seen (B, arrows) along with pneumatosisis intestinalis and portalis (B, curved arrows).

Fig. 30. Lateral decubitus abdominal radiograph in an 87-year-old patient with pneumoperitoneum from perforated peptic ulcer disease. A large volume of free air (arrowheads) is seen adjacent to the liver (arrows) in the right upper quadrant.

Fig. 31. Axial CT image from a 41-year-old patient with a perforated ulcer in the first part of the duodenum. A defect is seen in the medial duodenum (arrows), a large collection is seen in the lesser sac posterior to the stomach (arrowheads), and further pneumoperitoneum is also seen anteriorly (curved arrows).
b. Asymmetric nodular wall thickening in TB ileitis
c. Mural hyperenhancement
d. Fat stranding
e. Lymphadenopathy (particularly in TB ileitis)

2. Meckel diverticulitis
   a. Blind-ending tubular structure approximately 60 cm (2 feet) from the ileocecal valve
   b. Wall thickening
c. Surrounding fat stranding

3. Appendicitis
   a. Appendix diameter of greater than 6 mm
   b. Luminal obliteration
c. Noncompressible appendix at ultrasonography
d. Appendicolith
e. Periappendiceal inflammatory fat changes

4. Colitis
   a. Circumferential segmental wall thickening
   b. Mucosal hyperenhancement
c. Accordion sign
d. Halo sign
e. Pericolonic fat stranding
f. Submucosal edema

5. Typhilitis
   a. As for colitis but may be confined to cecum or involve the ileum

6. Diverticulitis
   a. Diverticula
   b. Fat stranding that is disproportionate to the mild, smooth, concentric wall thickening
c. Comma or centipede signs
d. Mesenteric root fluid
e. Gradual transition from normal to abnormal bowel thickness
f. Long (5–10 cm) affected segment
g. Minimal or no lymphadenopathy

PEARLS, PITFALLS, VARIANTS

Ischemia
- Bowl wall thickness is difficult to assess in underdistended or overdistended bowel, hence the level of distention should be taken into account. Oral intake of water or perrectal administration of water or air facilitates adequate distention.
- Ischemic bowel, particularly ischemic colon, may contract as a result of ischemia. Again, adequate distention with oral water or perrectal water or air is important.
- Bowl distention with intraluminal fluid and mural thickening as a result of ischemia may be mistaken for mechanical obstruction. Clinical findings, transition point appearance, and ancillary findings are key in this regard.

Obstruction
- As mentioned in the final point regarding ischemia, it is important to differentiate bowel that is dilated as a result of ischemia, from obstructed bowel.
- Paralytic ileus and colonic pseudoobstruction can be mistaken for adhesional obstruction. The presence of collapsed distal bowel, a transition point beak sign, and visible cause are important differentiating factors.

Infection
- As with mesenteric ischemia, bowel wall thickness in the context of underdistention or overdistention needs careful assessment in patients with colitis or enteritis. Adequate distention with oral or rectal contrast or air is important in optimizing bowel distention.
- Regarding diverticulitis, the findings described earlier are useful in differentiating diverticulitis from colon adenocarcinoma.
- The retrogastric lesser sac needs to be scrutinized in patients with upper abdominal peritonitis to assess for a PUD perforation into the lesser sac.

What the Referring Physician Needs to Know

Ischemia
- Location and length of involved segment
- Cause
• Features raising concern for bowel infarction: pneumatosis intestinalis, pneumatosis portalis, pneumoperitoneum

Obstruction
• Small or large bowel obstruction
• Closed loop or not
• High-grade (complete) or low-grade (incomplete) obstruction
• Cause
• Presence of ischemia
• Presence of perforation

Infection
1. Enteritis
   a. Extent of involvement
   b. Possible cause
   c. Complications
2. Appendicitis
   a. Simple or complicated (phlegmon, abscess)
3. Meckel diverticulitis
   a. Suspected diagnosis
   b. Presence of complications (abscess, bowel obstruction)
4. Colitis, cecitis, and typhilitis
   a. Segment and extent of bowel involved
   b. Likely causes
   c. Presence of complications (toxic megacolon, ischemia, abscess, perforation)
5. Diverticulitis
   a. Location and length of involved segment
   b. Presence of pericolic phlegmon or abscess
   c. Presence of pelvic abscess
   d. Presence of localized walled-off perforation
   e. Presence of generalized pneumoperitoneum
   f. Presence of feculent peritoneal contamination

SUMMARY
Ischemia, obstruction, and gastrointestinal infection are serious conditions frequently associated with significant morbidity and mortality in addition to being entities frequently encountered by emergency radiologists. CT remains the investigation of choice, although other modalities, MR imaging in particular, are going to play a greater role in the future. It is of key importance to ensure that the CT protocol is set up correctly in suspected intestinal ischemia, because multiphase imaging is required for optimal assessment. Measures should also be taken to ensure adequate bowel distention.

Differential Diagnosis

Intestinal ischemia differentials
1. Bowel obstruction
2. Dilated bowel after endoscopy or contrast enema with pneumatosis
3. Infectious or inflammatory enteritis or colitis
4. Scleroderma, vasculitis with pneumatosis
5. Bowel wall hematoma
6. Gastrointestinal lymphoma
7. Graft-versus-host disease
8. Angioedema

Bowel obstruction differentials
1. Paralytic ileus or pseudo-obstruction
   a. Recent surgery or trauma
   b. Shock
   c. Medications
   d. Electrolyte abnormalities
2. Celiac disease
3. Scleroderma
4. Mesenteric ischemia

Bowel infection differentials
1. Appendicitis or Meckel diverticulitis
   a. Omental infarct
   b. Epiploic appendagitis
   c. Ileitis
   d. Right-sided colonic diverticulitis
   e. Pelvic inflammatory disease
2. Enteritis/ileitis
   a. Crohn disease
   b. Intestinal ischemia
   c. Spondyloarthritis associated ileitis
   d. Vasculitis
   e. Neoplasm (eg, lymphoma)
   f. Radiation
3. Infectious colitis
   a. Inflammatory bowel disease
   b. Typhilitis
   c. Radiation colitis
4. Diverticulitis
   a. Colonic neoplasm
   b. Colitis
   c. Epiploic appendagitis
   d. Omental infarct

5. Perforation/pneumoperitoneum
   a. Residual postoperative air
   b. PUD
   c. Diverticulitis
   d. Trauma
   e. Bowel malignancy
   f. Bowel ischemia
   g. Appendicitis
   h. Endoscopy

REFERENCES


