

Gastrointestinal Bleeding at CT Angiography and CT Enterography: Imaging Atlas and Glossary of Terms

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Abbreviations: CTA = CT angiography, CTE = CT enterography, GI = gastrointestinal, GIST = gastrointestinal stromal tumor, NET = neuroendocrine tumor, NSAID = nonsteroidal anti-inflammatory drug

RadioGraphics 2021; 41:1632-1656

<https://doi.org/10.1148/rg.2021210043>

Content Codes: CT ER GI

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Gastrointestinal (GI) bleeding is a common potentially life-threatening medical condition frequently requiring multidisciplinary collaboration to reach the proper diagnosis and guide management. GI bleeding can be *overt* (eg, visible hemorrhage such as hematemesis, hematochezia, or melena) or *occult* (eg, positive fecal occult blood test or iron deficiency anemia). *Upper GI bleeding*, which originates proximal to the ligament of Treitz, is more common than *lower GI bleeding*, which arises distal to the ligament of Treitz. *Small bowel bleeding* accounts for 5–10% of GI bleeding cases commonly manifesting as *obscure* GI bleeding, where the source remains unknown after complete GI tract endoscopic and imaging evaluation. CT can aid in identifying the location and cause of bleeding and is an important complementary tool to endoscopy, nuclear medicine, and angiography in evaluating patients with GI bleeding. For radiologists, interpreting CT scans in patients with GI bleeding can be challenging owing to the large number of images and the diverse potential causes of bleeding. The purpose of this pictorial review by the Society of Abdominal Radiology GI Bleeding Disease-Focused Panel is to provide a practical resource for radiologists interpreting GI bleeding CT studies that reviews the proper GI bleeding terminology, the most common causes of GI bleeding, key patient history and risk factors, the optimal CT imaging technique, and guidelines for case interpretation and illustrates many common causes of GI bleeding. A CT reporting template is included to help generate radiology reports that can add value to patient care.

*An invited commentary by Al Hawary is available online.
Online supplemental material is available for this article.*

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SA-CME LEARNING OBJECTIVES

After completing this journal-based SA-CME activity, participants will be able to:

- List the proper terminology for the types of GI bleeding and describe the imaging findings of GI bleeding used when reporting GI bleeding cases.
- List the most common causes of GI bleeding and the key patient history and risk factors for GI bleeding, and discuss the optimal CT imaging technique based on patient history.
- Identify the key diagnostic features of common causes of GI bleeding and case interpretation pearls and pitfalls.

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Introduction

Gastrointestinal (GI) bleeding is a common potentially life-threatening medical condition. Locating the source of bleeding can be challenging and often requires multidisciplinary coordination and evaluation with endoscopic and imaging techniques (1,2). Once the source is identified, bleeding that cannot be managed with conservative measures or

TEACHING POINTS

- Using a consistent search pattern can optimize detection and characterization of GI bleeding. The ideal way to review CT images in the picture archiving and communications system is to link the noncontrast, arterial, and venous phase images in the axial, coronal, and sagittal planes and review images in all three phases and planes in synchrony.
- The attenuation of usual intestinal contents in the bowel lumen generally approaches that of simple fluid (ie, 0–15 HU). Unclotted blood has attenuation of 30–45 HU and clotted blood has attenuation of 45–70 HU in the bowel lumen. Within the GI tract, content with attenuation of greater than 60 HU is considered a more robust sign of recent hemorrhage.
- Active GI bleeding is depicted by the accumulation of extravasated contrast material in the bowel lumen as a focus, jet, cloud, or blush of variable size, usually appearing during the arterial phase. Contrast extravasation generally changes in size, attenuation, shape, and location on later phase images usually moving downstream. An enhancing focus that changes in attenuation but not shape on later phase images may be a vascular lesion (eg, aneurysm, pseudoaneurysm, or angioectasia). The absence of hyperattenuating material on noncontrast images in the same location of possible contrast extravasation on postcontrast images helps to confirm active bleeding.
- Some causes of GI bleeding are not visible on CT images, in which case, identifying contrast extravasation or high-attenuation intraluminal blood can help to direct patient management.
- The most common CT pitfall that mimics active contrast extravasation is the presence of hyperattenuating material within bowel loops, most frequently hyperattenuating colonic fecal material, retained or inadvertently administered positive oral contrast material, or prior pill ingestion.

therapeutic endoscopy may require interventional radiology or surgical intervention.

Upper GI bleeding is more common than lower GI bleeding, with an annual incidence of 100–200 per 100 000 compared with 20.5–27 per 100 000 for lower GI bleeding (3,4). Small bowel bleeding accounts for 5%–10% of GI bleeding cases and is considered its own bleeding category, often manifesting as obscure GI bleeding, where the source remains unknown after complete GI tract endoscopic and imaging evaluation (5–8). CT can aid in identifying the location and cause of bleeding and is an important complementary tool to endoscopy, nuclear medicine, and conventional angiography in evaluating patients with GI bleeding.

The purpose of this pictorial review, authored by the Society of Abdominal Radiology GI Bleeding Disease-Focused Panel, is to provide a practical resource for radiologists interpreting CT in patients with GI bleeding to facilitate standardized examination interpretation and improve the clarity and clinical impact of radiology reports. This begins with a review of the proper terminology for the types of GI bleeding and the imaging findings of GI bleeding. Next, the most common

causes of GI bleeding and key patient history and risk factors are presented, followed by a description of the optimal CT imaging technique to evaluate GI bleeding on the basis of patient history. Interpretation pearls and pitfalls are provided, including considerations when using dual-energy CT. Many common causes of GI bleeding are illustrated, along with their key diagnostic features. Finally, a CT reporting template is provided that can be used to create a structured report for clarity and ease of communication with referring physicians.

Glossary of Terms for GI Bleeding

When interpreting the CT examinations of patients with GI bleeding, it is important for radiologists to understand the proper GI bleeding terminology to effectively communicate with referring clinicians and to provide clear and understandable radiology reports. The clinical terminology for the types of GI bleeding (4–6,9–16) and terms used when reporting the imaging findings of GI bleeding are included in Tables 1 and 2, respectively (2,4,13,15,17–21).

Most Common Causes of GI Bleeding

For patients with GI bleeding, there is a diverse list of potential causes of bleeding that are generally divided into upper GI, lower GI, and small bowel sources. Patients with upper GI bleeding commonly present with hematemesis and/or melena, although those with a brisk upper GI source can present with hemochezia (2). With suspected lower GI bleeding, the source may be located in the upper GI tract in 11%–15% of patients or in the small bowel in 2%–15% of patients (9,22). In patients with obscure GI bleeding, the small bowel is the most common source of bleeding, accounting for approximately 75% of cases (6,7).

The most common causes of upper and lower GI bleeding and their frequency are listed in Table 3 (1,4,23,24) and Table 4 (1,3,4,25), respectively. With small bowel bleeding, some causes are more common in younger versus older patients while neoplasms and Dieulafoy lesions have no age predilection (Table 5) (4,6,7,15).

Key Patient History and Risk Factors for GI Bleeding

Once the likely location of bleeding (ie, upper GI, lower GI, or small bowel) has been determined, the radiologist may be able to narrow the list of diagnostic possibilities on the basis of the patient's history and risk factors for GI bleeding. Important historical information includes the type of bleeding (eg, overt, occult, or massive), associated signs and symptoms (eg, abdominal pain, chest pain, or weight loss), contributing

Table 1: Clinical Terminology for the Types of GI Bleeding

Type of GI bleeding	Description
Upper GI bleeding	GI bleeding originating proximal to the ligament of Treitz
Lower GI bleeding	GI bleeding originating distal to the ligament of Treitz
Small bowel bleeding	GI bleeding that originates distal to the ampulla of Vater and proximal to the ileocecal valve. Also known as middle GI bleeding
Suspected small bowel bleeding	GI bleeding in which no bleeding source is identified after performing both upper and lower endoscopy
Obscure GI bleeding	GI bleeding in which no bleeding source is identified after the entire GI tract has been comprehensively evaluated with advanced endoscopic and imaging techniques. Obscure GI bleeding can be either overt or occult depending on whether clinically evident GI bleeding is present.
Overt GI bleeding	Visible GI bleeding such as hematemesis, hematochezia, or melena. The term overt GI bleeding is preferred rather than acute, because acute implies the rate of the patient's symptom onset or how they presented and does not necessarily describe the visibility of bleeding. Also, while patients with overt GI bleeding can present acutely, overt GI bleeding can also occur intermittently or over an extended period of time.
Occult GI bleeding	GI bleeding that is not clinically visible. Patients with occult GI bleeding present with a positive fecal occult blood test result or iron deficiency anemia when other causes of anemia are excluded.
Massive GI bleeding	GI bleeding associated with hemodynamic instability (eg, hypotension with systolic blood pressure <90 mm Hg, tachycardia, symptoms of preshock or shock) or bleeding requiring transfusion of more than 4 units of packed red blood cells per 24 hours.

Table 2: Terms Used When Reporting the Imaging Findings of GI Bleeding

Term	Definition
Extravasation	The presence of a focal area of high attenuation in the bowel lumen on any postcontrast phase image that was not present on noncontrast or earlier phase images. Active extravasation of contrast material into the bowel lumen on CT images is pathognomonic for active bleeding clinically (also called active hemorrhage).
Extravasation versus bleeding	Extravasation is an imaging finding that is synonymous with the clinical finding GI bleeding or active GI bleeding. Radiologists reporting GI bleeding can use the term extravasation or contrast extravasation to describe this imaging finding in the radiology report. However, the impression statement active bleeding for the final diagnosis in the radiology report may be more understandable for the referring clinician.
Arterial bleeding versus venous bleeding	Arterial bleeding is bleeding from an artery that may be visualized during the arterial phase of imaging, although a slow arterial bleed may only be visualized in the venous phase. Venous bleeding is bleeding from a vein that occurs during and may be visualized during the venous phase or later phase of imaging and not during earlier phases.
Intraluminal hemorrhage or blood and sentinel clot	When there is recent or active GI bleeding, because of its high protein content, blood in the bowel lumen in the region of bleeding has higher attenuation than the usual intestinal contents. The intraluminal blood that has the highest attenuation on CT images is known as the sentinel clot and may be located closest to the actual site of active or recent bleeding.
Pseudoaneurysm	A lobular focus of enhancement along an arterial branch initially visualized during the arterial phase of imaging that follows the attenuation of the arterial blood pool during later phases, but remains unchanged in size and shape on later phase images. Pseudoaneurysms communicate with the arterial lumen, and the walls are formed by the media, adventitia, or surrounding soft-tissue structures.

event (eg, trauma, vomiting, or hypotension), and recent procedures (eg, postpolypectomy or liver biopsy). Key patient history and risk factors for GI bleeding are listed in Table 6 (1,6,9,10,14,22,24,26–32).

CT Imaging Technique

When CT is performed for patients with GI bleeding, the goal of the examination depends on patient history. For patients with overt GI bleeding, the goal of CT is to identify intraluminal blood,

Table 3: Most Common Causes of Upper GI Bleeding

Cause of Bleeding	Frequency (%)
Peptic ulcer disease (including duodenal and gastric ulcers), mucosal erosive disease (involving the esophagus, stomach, or duodenum) or esophagitis, Zollinger-Ellison syndrome, or Cameron lesion	55–74
Varices*	5–14
Mallory-Weiss tear	2–7
Neoplasm	2–5
Vascular lesions (angioectasia, Dieulafoy lesion, arteriovenous malformation, or gastric antral vascular ectasia)	2–3
Portal hypertensive gastropathy	2.4
Undetermined cause	5
Hemobilia	Uncommon
Primary or secondary aortoenteric fistula [†]	Uncommon
Hemosuccus pancreaticus	Uncommon

*In patients with cirrhosis, esophageal and/or gastric varices are the most common cause of bleeding, accounting for 50–60% of cases.

[†]Primary aortoenteric fistulas are usually associated with an aortic aneurysm. Secondary aortoenteric fistulas typically occur after repair of an abdominal aortic aneurysm.

Table 4: Most Common Causes of Lower GI Bleeding

Cause of Bleeding	Frequency (%)
Diverticulosis	30–65
Colonic ischemia	5–20
Hemorrhoids	5–20
Colorectal neoplasm	2–15
Angioectasia	5–10
Polypectomy	2–7
Other causes of colitis (eg, inflammatory bowel disease or infections)	5–10
Rectal ulcer	0–8
Stercoral ulceration	0–5
Rectal varices	0–3
Radiation proctopathy	0–2
NSAID colopathy	0–2
Dieulafoy lesion	Uncommon
Upper GI source*	11–15
Small bowel source*	2–15

Note.—NSAID = nonsteroidal anti-inflammatory drug.

*With suspected lower GI bleeding, the source may be in the upper GI tract in 11–15% of patients and the small bowel in 2–15% of patients.

contrast extravasation, and/or the cause of bleeding. For occult GI or suspected small bowel bleeding, since there is a slower bleeding rate, the goal of CT is typically to identify the cause of bleeding rather than subtle contrast extravasation. Because of these different goals, many centers use two different CT

protocols, one for patients with overt GI bleeding and another for occult GI or suspected small bowel bleeding.

For the evaluation of overt GI bleeding, most centers use a multiphase CT technique including noncontrast or virtual noncontrast images, late arterial phase acquisition and portal venous phase (60–70 seconds after bolus initiation) or late venous phase (70–90 seconds after bolus initiation) acquisition. An overt GI bleeding CT angiography (CTA) protocol proposed by the Society of Abdominal Radiology GI Bleeding Disease-Focused Panel, on the basis of prior consensus opinion, is provided in Table 7 (33,34). For occult GI or suspected small bowel bleeding, protocols are more variable. Some centers use a multiphase technique, while others use a single-phase CT technique (eg, enteric phase, portal venous phase, or split-bolus). Options for an occult GI or suspected small bowel bleeding CT enterography (CTE) protocol are provided in Table 7 (4,13). GI bleeding CT protocols generally balance maximizing examination sensitivity and specificity, while minimizing radiation dose.

Interpretation Pearls and Pitfalls

Image Interpretation

Using a consistent search pattern can optimize detection and characterization of GI bleeding. The ideal way to review CT images in the picture archiving and communications system is to link the noncontrast, arterial, and venous phase images in the axial, coronal, and sagittal planes and review images in all three phases and planes in synchrony. The bowel should be systematically

reviewed on axial and coronal images acquired during all phases from proximal to distal (or distal to proximal), although clinical information can focus the search to a particular bowel segment. Identifying intraluminal hyperattenuating hemorrhage on noncontrast images can direct the search of arterial and venous phase images for a bleeding site. Subsequently, the arterial phase images should be scrutinized for signs of contrast extravasation, which should be confirmed on venous phase images, if the intraluminal contrast extravasation changes in size, attenuation, and/or shape. Venous phase images should also be reviewed to identify slower venous bleeds that may not appear during the arterial phase. Reviewing maximum intensity projection images can enhance delineation of vascular anatomy and pathology (2,4,35).

CT Signs of GI Bleeding

On noncontrast or virtual noncontrast CT images, high-attenuation intraluminal fluid may be a sign of active bleeding in this portion of the bowel. The attenuation of usual intestinal contents in the bowel lumen generally approaches that of simple fluid (ie, 0–15 HU). Unclotted blood has attenuation of 30–45 HU and clotted blood has attenuation of 45–70 HU in the bowel lumen. Within the GI tract, content with attenuation of greater than 60 HU is considered a more robust sign of recent hemorrhage (4,13). At CT, a *sentinel clot* refers to acutely clotted blood, which appears hyperattenuating and may be located closest to the bleeding site, whereas lower-attenuation unclotted blood may be located farther from the source (Fig 1) (2,13,35).

Active GI bleeding is depicted by the accumulation of extravasated contrast material in the bowel lumen as a focus, jet, cloud, or blush of variable size, usually appearing during the arterial phase. Contrast extravasation generally changes in size, attenuation, shape, and location on later phase images usually moving downstream (Figs 1–4). An enhancing focus that changes in attenuation but not shape on later phase images may be a vascular lesion (eg, aneurysm, pseudoaneurysm, or angioectasia) (13). The absence of hyperattenuating material on noncontrast images in the same location of possible contrast extravasation on postcontrast images helps to confirm active bleeding (12). Active extravasation may also be seen exclusively in the venous phase, resulting from venous bleeding, or less likely, slower or low-volume arterial bleeding (2,17,35). Finally, the absence of arterial phase extravasation does not imply a negative CTA examination until it is confirmed to be absent during the venous phase.

Table 5: Most Common and Uncommon Causes of Small Bowel Bleeding

Most common causes in patients younger than 40 years

- Inflammatory bowel disease
- Meckel diverticulum
- Neoplasm*
- Dieulafoy lesion*

Most common causes in patients 40 years or older

- Angioectasia
- NSAID enteropathy
- Neoplasm*
- Dieulafoy lesion*

Uncommon causes

- Small-bowel varices
- Portal hypertensive enteropathy
- Amyloidosis
- Blue rubber bleb nevus syndrome
- Osler-Weber-Rendu syndrome
- Non-small bowel sources (hemobilia, aortoenteric fistula, and hemosuccus pancreaticus)

Note.—Celiac disease is not listed, because when iron deficiency anemia occurs, the cause is usually malabsorption rather than GI bleeding.

*Neoplasms such as lymphomas, neuroendocrine tumors, adenocarcinomas, or polyposis syndrome and Dieulafoy lesions are common in both younger and older patient cohorts.

Even when no contrast extravasation is identified, CT may allow identification of the potential cause of GI bleeding. Analyzing the bowel wall, bowel and mesenteric vessels, and mesenteric attenuation changes can provide helpful information for the location and characterization of possible causes of GI bleeding (2,36). Some causes of GI bleeding are not visible on CT images, in which case, identifying contrast extravasation or high-attenuation intraluminal blood can help to direct patient management (4).

GI Bleeding CT Diagnostic Pitfalls

The most common CT pitfall that mimics active contrast extravasation is the presence of hyperattenuating material within bowel loops, most frequently hyperattenuating colonic fecal material, retained or inadvertently administered positive oral contrast material, or prior pill ingestion (Fig 5). Every hyperattenuating focus suspicious for active bleeding on contrast-enhanced images must be confirmed to be absent on noncontrast or virtual noncontrast images. Foreign bodies including feeding tubes, surgical drains, stents, embolic material, and surgical sutures or clips may not only be mistaken for active extravasation, but

Table 6: Key Patient History and Risk Factors for GI Bleeding

Cause	Key Patient History and Risk Factors for GI Bleeding
Angioectasia	Advanced age, aortic stenosis, end-stage renal disease, left ventricular assist device, presence of multiple angioectasias, anticoagulant or antiplatelet use
Aortoenteric fistula	Massive GI bleeding, infectious aortitis, prosthetic aortic graft, aortic aneurysm, radiation injury, tumor invasion, foreign body perforation
Cameron lesion	Large hiatal hernia
Colonic ischemia	Acute abdominal pain, hypotension, advanced age, embolic disease, chronic renal failure, trauma, recent high-risk surgery
Colorectal cancer	Advanced age, familial adenomatous polyposis, Lynch syndrome, Peutz-Jeghers syndrome, Crohn disease, ulcerative colitis, history of adenomatous polyps, family history of colorectal cancer
Crohn disease	Risk factors for acute lower GI bleeding include duration of Crohn disease, perianal disease, left colon involvement, steroid use
Delayed postpolypectomy bleeding	Polyp size >10 mm, thick stalk, immediate postpolypectomy bleeding, right-sided location, resumption of antithrombotic treatment, polyp pathology
Dieulafoy lesion	In the upper GI tract: antiplatelet agents, alcohol abuse, use of NSAIDs
Diverticular bleeding	Painless hematochezia, advanced age, hypertension, anticoagulant or NSAID use
Esophagitis	Gastroesophageal reflux disease, pill esophagitis medications (eg, erythromycin or NSAIDs), infections (eg, herpes simplex virus or cytomegalovirus)
GI malignancy	Unexplained weight loss, change in bowel habits, anemia
Hemobilia	Liver biopsy, cholecystectomy, endoscopic biliary biopsy or stenting, trauma, hepatic or bile duct tumors, hepatic artery aneurysm
Hemosuccus pancreaticus	Chronic pancreatitis, necrotizing pancreatitis, pancreatic pseudocysts, pancreatic neoplasm, pancreatic therapeutic endoscopy
Mallory-Weiss tear	Vomiting or retching, often related to heavy alcohol consumption
NSAID enteropathy or colopathy	Use of NSAIDs
Postsurgical anastomotic bleeding (marginal ulcers)	Gastric bypass surgery, Billroth II surgery, use of NSAIDs, <i>Helicobacter pylori</i> infection, cigarette smoking
Peptic ulcer disease or mucosal erosive disease	Epigastric pain; nausea; bloating and/or early satiety; <i>Helicobacter pylori</i> infection; NSAID, anticoagulant, or low-dose aspirin use; physiologic stress; Zollinger-Ellison syndrome
Small-bowel adenocarcinoma	Lynch syndrome, familial adenomatous polyposis, Peutz-Jeghers syndrome, Crohn disease, celiac disease
Small-bowel lymphoma	Celiac disease, Crohn disease, chronic immunosuppression, radiation therapy, nodular lymphoid hyperplasia
Small-bowel neuroendocrine tumor	Multiple endocrine neoplasia type I, neurofibromatosis type 1
Varices and portal hypertensive gastropathy	Massive GI bleeding, cirrhosis, portal hypertension, portal vein thrombosis

may also obscure areas of bleeding (35,37). Attention to subtle changes in attenuation surrounding foreign bodies and appropriate windowing to reduce beam-hardening artifact is needed. Cone beam artifacts can cause hyperattenuating foci at soft-tissue and air interfaces, especially in patients whose images show significant motion artifact, gaseous distention, or increased peristalsis during image acquisition and can result in a false-positive CTA examination (Fig 6) (2,37,38).

Other causes that may be mistaken for active hemorrhage include mucosal hyperenhancement of normal collapsed bowel segments, hyperatten-

uation of the diseased bowel wall, hypervascular masses, and vascular lesions (2,13).

The capability of CTA to allow detection of contrast extravasation is potentially decreased in fluid-filled bowel loops, because bleeding may become diluted. Thus, no neutral oral contrast material or water should be administered when performing CTA (4,35,37). A false-negative CTA examination can also result from slow GI bleeding below the threshold needed to produce a detectable focus of contrast extravasation, or from self-limited or intermittent GI bleeding, which may not be active at the time of CTA examination (2,13).

Table 7: Proposed Overt GI Bleeding CTA Protocol and Options for Occult GI or Suspected Small Bowel Bleeding CTE Protocol

Variable	Overt GI Bleeding CTA Protocol	Occult GI or Suspected Small-bowel Bleeding CTE Protocol
Intravenous contrast material and dose	125 mL of high-concentration contrast material (ie, 350–375 mgI/mL)*	125 mL of high-concentration contrast material (ie, 350–375 mgI/mL)*
Intravenous contrast administration rate	Highest rate possible (eg, 4–5 mL/sec)	Highest rate possible (eg, 4–5 mL/sec)
Saline solution flush	40–50 mL at 4–5 mL/sec	40–50 mL at 4–5 mL/sec
Oral contrast material	No oral contrast material is recommended [†]	1350–2000 mL of neutral enteric contrast material in divided aliquots, beginning 1 hour before the examination [‡]
Extent	Top of liver to femoral lesser trochanter	Top of liver to femoral lesser trochanter
Acquisition phases	Multiphase CT technique: Noncontrast- or virtual noncontrast [§] Late arterial phase (10 sec after aortic bolus trigger) [¶] Venous phase (70–90 sec after injection begins)	If multiphase CT technique is used: Late arterial phase (10 sec after aortic bolus trigger) [¶] Enteric phase (50 sec after injection begins) Late venous phase (90 sec after injection begins)
Alternative acquisition phases	NA	Alternative single-phase CT with enteric phase, portal venous phase, or split-bolus technique Alternative biphasic CT during arterial and enteric phases or arterial and portal venous phases
Image reconstruction	Axial 2.5–3 mm for each series (optional axial 1 mm) Coronal and sagittal 2.5–3 mm reconstructed images (50% overlap) Optional maximum intensity projection and volume-rendered reconstruction	Axial 2.5–3 mm for each series (optional axial 1 mm) Coronal and sagittal 2.5–3 mm reconstructed images (50% overlap) Optional maximum intensity projection and volume-rendered reconstruction
DECT postprocessing	40–60 keV (ie, virtual monoenergetic), iodine density, virtual noncontrast, and standard mixed (blended) series	40–60 keV (ie, virtual monoenergetic), iodine density, virtual noncontrast, and standard mixed (blended) series

Note.—For larger patients (>45 cm diameter), consider using single-energy CT rather than dual-energy CT (DECT). NA = not applicable.

*100 mL may be the minimum effective volume; however, the dose can be adjusted for patient size and contrast agent concentration. With DECT, intravenous contrast material volume can be reduced, depending on patient weight and kilovoltage.

[†]For the overt GI bleeding CTA protocol, oral contrast administration is discouraged for the following reasons: (a) scanning is delayed, during which time, transient or intermittent bleeding may cease; (b) oral contrast administration increases the aspiration risk during sedation or anesthesia, which can preclude or delay urgent interventional radiology or surgical interventions; (c) positive oral contrast material masks intraluminal contrast extravasation, which may have similar attenuation; and (d) neutral oral contrast material may dilute contrast extravasation, making it more difficult to identify active bleeding.

[‡]In the United States, common neutral enteric contrast agents include Breeza 500 mL (Beekley Medical) or NeuLumEX 450 mL (formerly VoLumen; Bracco Diagnostics); one bottle every 15–20 minutes for a total of three bottles. Optionally, an additional 250–500 mL of water can be administered right before scanning. In countries where these agents are unavailable, a solution of sorbitol or polyethylene glycol can be used for neutral enteric contrast material.

[§]True noncontrast series is recommended for single-energy CT and can be obtained with a low-radiation-dose technique. A virtual noncontrast series can be substituted when DECT is used.

[¶]Absolute bolus trigger threshold of 150 HU in the abdominal aorta at the mid liver level. Bolus tracking or a timing bolus is preferred to a fixed delay owing to variabilities in cardiac output, patient hydration, and comorbidities.

Considerations When Using Dual-Energy CT

Dual-energy CT techniques can be helpful in patients with acute GI bleeding. The ability to create virtual noncontrast reconstructions can eliminate the need for true noncontrast images

and reduce the total CT radiation dose (Fig 7) (39). In addition, low kiloelectron voltage (virtual monoenergetic), iodine density map, and color overlay images can increase the conspicuity of iodinated contrast material, thus facilitating detection of active extravasation (40). This can

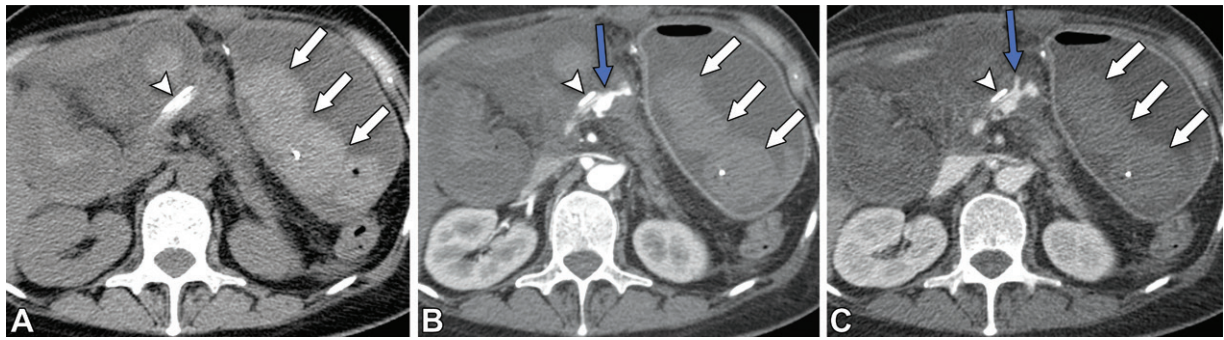


Figure 1. Sentinel clot related to active bleeding at the pancreaticojejunostomy anastomosis in a 65-year-old woman who presented with hematemesis and jaundice 12 days after she underwent the Whipple procedure for pancreatic adenocarcinoma. Axial noncontrast (A), arterial phase (B), and portal venous phase (C) CTA images show a sentinel clot in the stomach (white arrows) and contrast extravasation at the pancreaticojejunostomy site, which appears during the arterial phase (blue arrow in B) and changes in size, attenuation, and shape in the portal venous phase (blue arrow in C). There is a nasoenteric tube in place (arrowhead in all images).

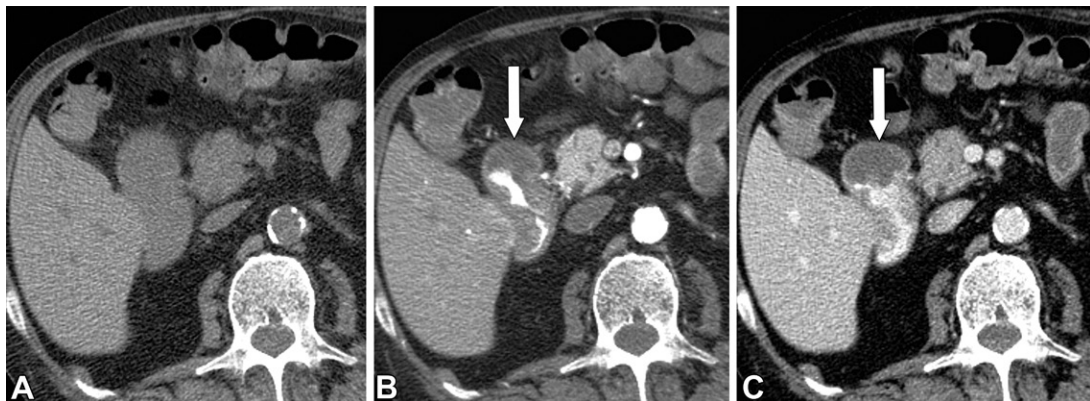


Figure 2. Active bleeding in the proximal duodenum, which was caused by two bleeding duodenal ulcers that were identified with endoscopy (not shown), in a 71-year-old man who presented with melena, hypotension, and shock. Axial noncontrast (A), arterial phase (B), and portal venous phase (C) CTA images show contrast extravasation in the proximal duodenum, which appears in the arterial phase (arrow in B) and changes in size, attenuation, and shape in the portal venous phase (arrow in C). The bleeding duodenal ulcers noted at endoscopy were not visualized on CT images.

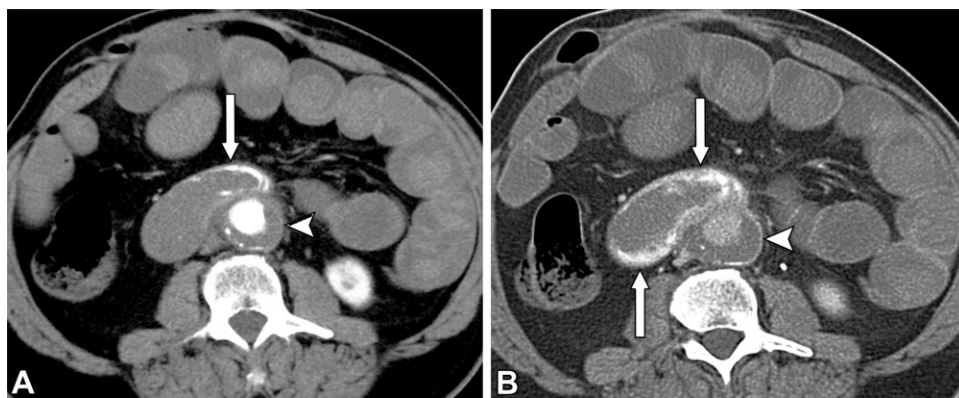


Figure 3. Aortic aneurysm with an aortoduodenal fistula and active bleeding in a 77-year-old man with a history of melena and anemia. Axial arterial phase (A) and late venous phase (B) CTA images show contrast extravasation in the distal duodenum in the arterial phase (arrow in A), which changes in size, attenuation, and shape in the late venous phase (arrows in B). Also noted is an abdominal aortic aneurysm containing eccentric thrombus (arrowhead in A and B).

be particularly helpful when subtle bleeding is partially obscured by adjacent hyperattenuating intraluminal hemorrhage (Fig 8). Metal suppression techniques can reduce beam hardening artifacts from dense materials such as surgical clips or ballistic fragments and improve detection of extravasated iodinated contrast material (39,40).

Causes of GI Bleeding

Upper GI Bleeding

Peptic Ulcer Disease.—Peptic ulcer disease includes stomach or duodenal ulcers. On CT images, direct signs of peptic ulcer disease include a focal outpouching and a mucosal enhancement defect. Indirect signs include gastric or duodenal wall thickening, submucosal edema, mucosal hyperenhancement, adjacent stranding, and signs of perforation (41–43). Patients with overt upper GI bleeding may have hemorrhagic intraluminal fluid on noncontrast CT images. An actively bleeding ulcer may show intraluminal contrast extravasation (Figs 9, 10, E1) (44).

Mucosal Erosive Disease and Esophagitis.—Esophagitis CT findings include circumferential esophageal wall thickening, mucosal hyperenhancement, and/or submucosal edema (Fig 11) (45). Reflux esophagitis is suggested when there is distal esophageal wall thickening, particularly when a hiatal hernia is present. Gastritis and duodenitis CT findings include mucosal fold thickening, submucosal edema, mucosal hyperenhancement, and adjacent stranding (46,47).

Cameron Lesions.—Cameron lesions are linear erosions or ulcers occurring with large hiatal hernias that are thought to result from mechanical trauma to the gastric mucosa as the stomach slides through the diaphragmatic hiatus or from pressure-induced mucosal ischemia at the hiatus. On CT images, Cameron lesions are largely radiographically occult, but the diagnosis could be suggested when overt upper GI bleeding occurs when a large hiatal hernia is present (48).

Variceal Bleeding.—Gastroesophageal varices usually result from portal hypertension, and overt upper GI variceal bleeding is a potentially lethal complication. Cirrhosis is the most common cause of portal hypertension. Noncirrhotic causes include portal vein, splenic vein, and hepatic veno-occlusive disease (49). On CT images, gastroesophageal varices appear as enhancing foci or tubular structures within the distal esophageal and/or stomach wall that follow blood pool attenuation during different

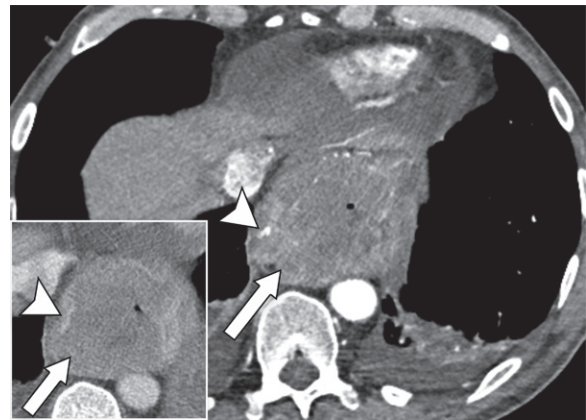


Figure 4. Esophageal neuroendocrine tumor (NET) with active bleeding in a 48-year-old man who presented with hematemesis. Axial arterial phase (main image) and 75-second venous phase (inset) CTA images show a large heterogeneously enhancing distal esophageal mass (arrows in both images), with a focus of contrast extravasation in the arterial phase (arrowhead in main image) that changes in size, attenuation, and shape in the venous phase (arrowhead in inset). Endoscopic biopsy performed 1 month earlier allowed confirmation of a poorly differentiated NET.

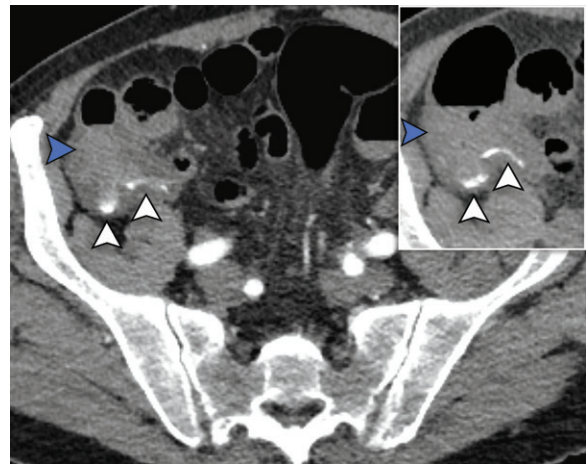


Figure 5. Hyperattenuating material in the cecum simulating contrast extravasation in a 90-year-old man with a history of chronic myelomonocytic leukemia and anemia who presented with bloody diarrhea. Arterial phase (main image) and non-contrast (inset) CTA images show hyperattenuating material in the posterior cecum, extending into the base of the appendix in the arterial phase image (white arrowheads in main image) which is also present on noncontrast images (white arrowheads in inset), confirming debris rather than contrast extravasation. There is also hyperattenuating fluid within the cecum, likely representing blood (blue arrowhead in both images). Active extravasation was noted in the distal ileum on CT images (not shown), although it was beyond the reach of subsequent lower endoscopy.

contrast-enhanced phases, and are best identified in the portal venous phase (Fig 12).

Mallory-Weiss Syndrome.—Mallory-Weiss syndrome relates to distal esophageal longitudinal mucosal tears or lacerations that extend into the esophagogastric junction and can cause

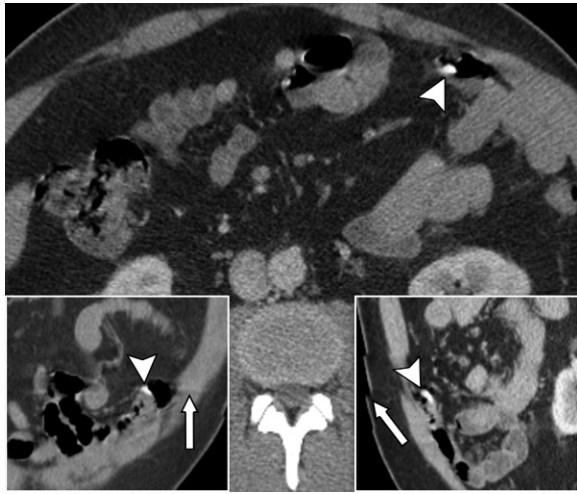


Figure 6. Cone beam artifact mimicking contrast extravasation. Axial (main image), coronal (left inset), and sagittal (right inset) portal venous phase CT images show a hyperattenuating focus at the interface between air and normal bowel fluid (arrowhead in all images), which is consistent with cone beam artifact. Additional findings to distinguish this artifact are the significantly higher attenuation of the artifact (271 HU) compared with that of the aorta (107 HU) and that of the inferior vena cava (90 HU), the adjacent streak artifact (arrow in left inset), and the adjacent abdominal wall stair-step artifact from patient motion (arrow in right inset).

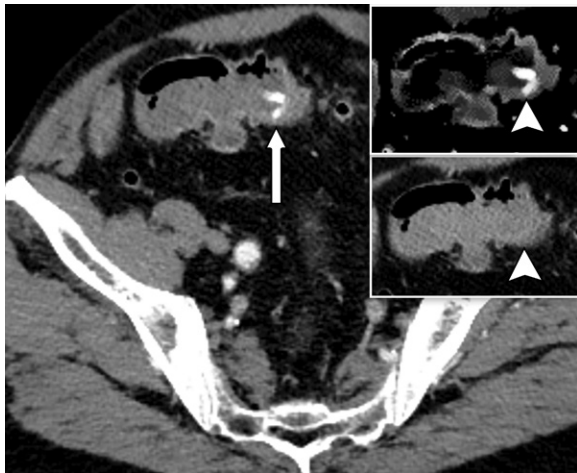


Figure 7. Active bleeding in the sigmoid colon from diverticulosis on a single-phase dual-energy CT image during the portal venous phase in an 83-year-old man with abdominal pain and blood in the stool. Axial mixed (blended) dual-energy CT image shows foci of increased attenuation within the sigmoid colon (arrow in main image), which is also present in the iodine map image (arrowhead in top inset), but not present in the virtual noncontrast image (arrowhead in bottom inset), confirming contrast extravasation. There is also sigmoid diverticulosis.

overt upper GI bleeding. Mallory-Weiss tears are usually associated with vomiting or retching that is often related to heavy alcohol consumption and occasionally occur as a complication of upper endoscopy (50). On CT images, Mallory-Weiss tears are typically radiographically occult, but intraluminal blood products or gas in the

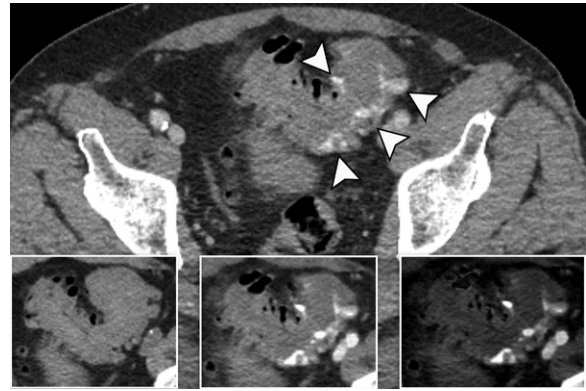


Figure 8. Acute diverticular bleed in the sigmoid colon on dual-energy CT images in a 72-year-old man who presented with bright red blood in the rectum. Axial portal venous phase mixed (blended) CT image (main image) shows multiple foci of contrast extravasation in the sigmoid colon (arrowheads) and diffuse sigmoid diverticulosis, some containing extravasated contrast material. Axial noncontrast CT image (left inset) shows hyperattenuating bowel contents in the sigmoid colon. Axial postprocessed monoenergetic 50-keV image (middle inset) and iodine map image (right inset) show contrast extravasation that is more conspicuous at the lower kiloelectron voltage setting and on the iodine map than on the mixed (blended) image.

esophageal wall at the site of mucosal injury can occur (51). Patients with overt upper GI bleeding can show contrast extravasation (Fig 13).

Neoplasms.—Esophageal, gastric, and duodenal cancers can all ulcerate and cause GI bleeding (1). Esophageal cancers can show asymmetric or marked focal wall thickening, often with paraesophageal lymph nodes (Figs 14, E2). Gastric cancer can produce focal or diffuse gastric wall thickening or manifest as an intraluminal polypoid lesion (Figs 15, E3) and can be associated with perigastric lymph nodes, liver and pulmonary metastases, and peritoneal disease. Gastric lymphoma can manifest as focal or diffuse wall thickening, an ulcerated mass, or polypoid or nodular fold thickening (52). Gastric metastatic disease can arise from melanoma, breast cancer, and lung cancer. Gastrointestinal stromal tumor (GIST), the most common GI tract mesenchymal tumor, most commonly arises in the stomach and small intestine. GISTs are commonly exophytic and can ulcerate, leading to GI bleeding (Figs 16, E4) (53). Benign neoplasms that can manifest with upper GI bleeding include leiomyomas, lipomas, and polyps.

Dieulafoy Lesions.—Dieulafoy lesions (also known as caliber-persistent arteries) are submucosal arteries that erode through a small GI tract mucosal defect (typically 2–5 mm) and can rupture, resulting in severe bleeding. Most occur in the proximal stomach, usually along

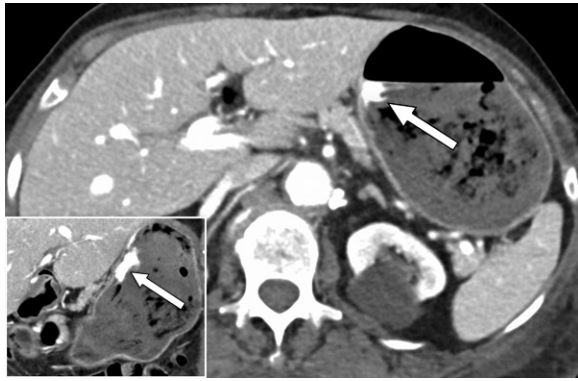


Figure 9. Gastric ulcer causing active bleeding in an 80-year-old woman receiving enoxaparin and warfarin for chronic atrial fibrillation and aortic valve replacement who underwent recent spinal fusion surgery. Axial (main image) and coronal (inset) portal venous phase CT images show contrast extravasation along the lesser curvature of the stomach (arrow in both images). Figure E1 is an upper endoscopic image in this patient that shows an actively bleeding ulcer in the lesser curvature of the stomach.

the lesser curvature, within 6 cm of the gastroesophageal junction, with one-third of them forming in places other than the stomach, most commonly the duodenum, followed by the colon (29,54,55). On CT images, Dieulafoy lesions show a nidus of arterial phase enhancement, and are not as well seen on subsequent enteric or venous phase images (Figs 17, E5). Importantly, a CT examination that does not include arterial phase images may cause these lesions to be missed.

Roux-en-Y Gastric Bypass.—GI bleeding can occur as an early or late (>30 days postoperatively) complication of bariatric Roux-en-Y gastric bypass surgery. Early bleeding can result from bleeding at the gastric pouch or gastric remnant staple line or the gastrojejunostomy or jejunojunction anastomoses. A common cause of late GI bleeding is marginal ulceration near the gastrojejunostomy site, occurring at a reported rate of 0.6%–16%. Marginal ulceration CT findings can include a definable ulcer, wall thickening at the gastrojejunostomy anastomosis, adjacent stranding, contrast extravasation if there is active bleeding (Fig 18), and free air if perforation has occurred (56).

Lower GI Bleeding

Diverticulosis.—Diverticulosis, the most common cause of lower GI bleeding, typically manifests with painless hematochezia (9). Diverticula most commonly arise in the sigmoid and left colon, although diverticulosis bleeding is more common in the right colon (1,10). The CTA hallmark of diverticular bleeding is contrast extravasation into

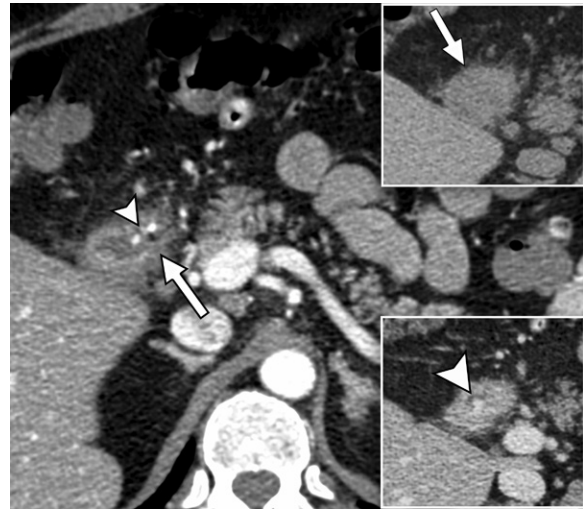


Figure 10. Duodenal ulcer with active bleeding in a 54-year-old man with anemia who presented with syncope and large-volume melena. Axial arterial phase (main image), noncontrast (top inset), and venous phase (bottom inset) CTA images show mild stranding adjacent to the proximal duodenum (arrow in top inset). On the arterial phase image there is discontinuous duodenal mucosal enhancement (arrow in main image), which is consistent with an ulcer crater, with associated duodenal wall thickening and adjacent stranding. There are foci of arterial enhancement (arrowhead in main image) that are not present on the noncontrast image and change in size, attenuation, and shape in the venous phase (arrowhead in bottom inset), which are consistent with active bleeding. After CT, the patient underwent angiography, which showed active hemorrhage from a gastroduodenal artery branch, and underwent gastroduodenal artery coil embolization. Subsequent endoscopy (not shown) demonstrated deeply cratered gastric and duodenal ulcers.

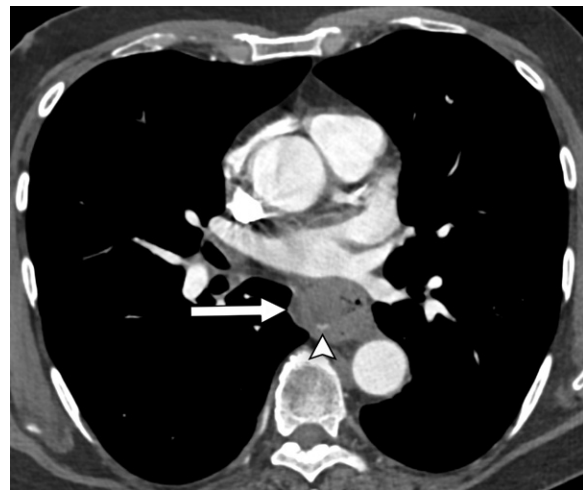


Figure 11. Esophagitis with possible active bleeding in a 62-year-old man with esophagitis, gastroesophageal reflux disease, and peptic ulcer disease who presented with chest pain and hematemesis. Axial arterial phase CT image shows diffuse esophageal wall thickening, fluid distending the esophagus, paraesophageal fluid (arrow), and a tiny focus of high attenuation in the posterior esophageal lumen that is concerning for active bleeding (arrowhead). However, contrast extravasation could not be confirmed on this single-phase CT image. At endoscopy (not shown), a bleeding deep mucosal tear was found in the esophagus, with an associated clot.

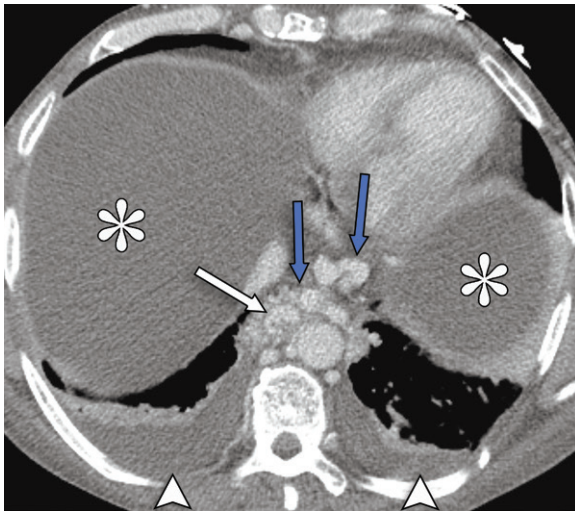


Figure 12. Esophageal and paraesophageal varices in a 63-year-old man with decompensated cirrhosis, portal hypertension, and anemia. Axial portal venous phase CT image shows submucosal esophageal varices (white arrow) and paraesophageal varices (blue arrows). Also noted is a large amount of ascites (*) and small bilateral pleural effusions (arrowheads).

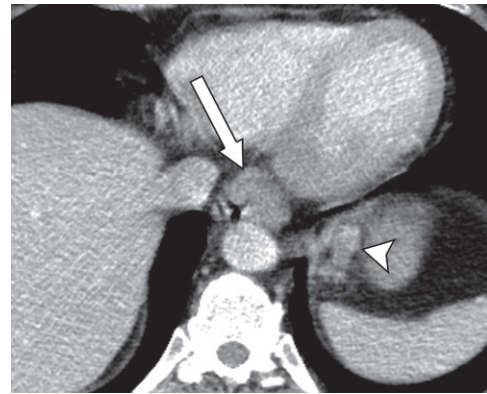


Figure 14. Esophageal carcinoma with adjacent lymphadenopathy in a 48-year-old man who presented with dysphagia, weight loss, and occult GI bleeding. Axial portal venous phase CT image shows moderate asymmetric distal esophageal wall thickening consistent with the biopsy-confirmed poorly differentiated adenocarcinoma (arrow), with adjacent lymphadenopathy (arrowhead). Figure E2 is an endoscopic image in this patient that shows the esophageal mass, with an area of hemorrhage.

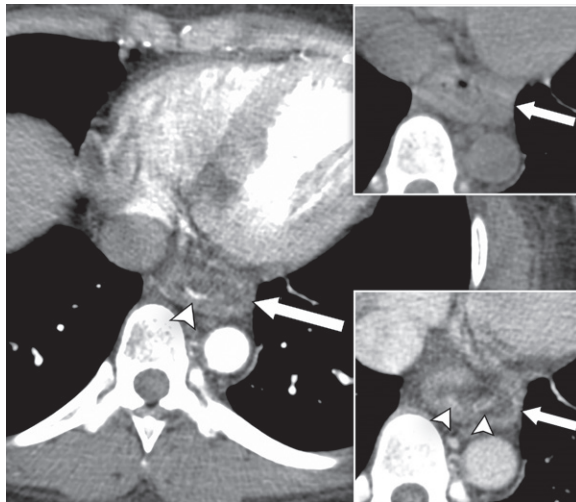


Figure 13. Mallory-Weiss tear with active bleeding in a 30-year-old woman who presented with chest pain, intractable retching, vomiting, hematemesis, and anemia. Axial arterial phase (main image), noncontrast (top inset), and portal venous phase (bottom inset) CTA images show circumferential distal esophageal wall thickening (arrows in all three images), periesophageal stranding and trace fluid, fluid in the esophageal lumen, and a linear focus of contrast extravasation in the posterior esophageal lumen (arrowhead in main image) that changes in size, attenuation, and shape in the portal venous phase (arrowheads, bottom inset), which is consistent with active bleeding.

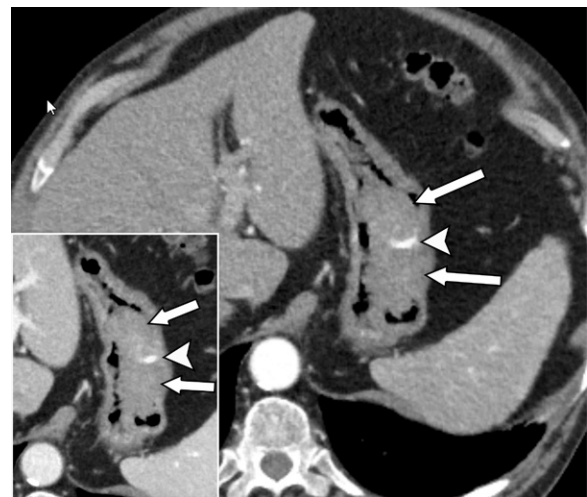


Figure 15. Gastric carcinoma in an 80-year-old man with weight loss and anemia. Axial arterial phase (main image) and portal venous phase (inset) CTA images show a large polypoid mass in the stomach (arrows), with adjacent contrast extravasation in the arterial phase (arrowhead in main image), which changes in size, attenuation, and shape in the portal venous phase (arrowhead, inset image). Figure E3 is an endoscopic image in this patient that shows a large gastric mass with an area of hemorrhage. Endoscopic biopsy results confirmed moderately differentiated adenocarcinoma.

a colonic diverticulum that appears on arterial phase images and changes in size, attenuation, shape, and location on delayed phase images (Figs 19, E6) (57).

Colonic Ischemia.—Colonic ischemia most commonly results from a sudden decrease in mesenteric perfusion caused by a “low flow” state (9).

Less commonly, colonic ischemia can result from arterial occlusion or mesenteric venous thrombosis. Most colonic ischemia cases occur in elderly patients. In younger patients, potential causes include hypercoagulable states, vasculitis, drugs and medication, posttraumatic shock, extreme exercise, and sickle cell disease. Colonic ischemia classically occurs in the colonic “watershed” areas (ie, splenic flexure and the rectosigmoid junction),



Figure 16. Gastric GIST with active bleeding in a 35-year-old woman with a history of paraganglioma who presented with abdominal pain, hypotension, tachycardia, and severe anemia. Axial portal venous phase (main image and top inset) and 7-minute delayed phase (bottom inset) CT images show a homogeneously enhancing lobulated partially exophytic and endophytic mass in the lesser curvature of the stomach (arrow in all images). There are foci of contrast extravasation in the portal venous phase (arrowhead in top inset) that increase in size and change in attenuation and shape in the delayed phase (arrowheads in bottom inset), which are consistent with active bleeding. Figure E4 is an endoscopic image in this patient that shows the endophytic portion of the mass, with an area of hemorrhage.

although any portion of the colon can be involved (9,22). Colonic involvement is typically segmental, most commonly affecting the left colon and sigmoid colon; pancolonic involvement is uncommon. The rectum is typically spared from ischemia owing to its dual splanchnic and systemic arterial blood supply (58). The typical clinical presentation is sudden-onset crampy abdominal pain, with hematochezia or bloody diarrhea occurring within 24 hours. Patients with isolated right colon ischemia may more commonly present with pain rather than rectal bleeding and generally have worse clinical outcomes compared with colonic ischemia in other parts of the colon. While the CT appearance of colonic ischemia overlaps with those of other causes of colitis, the pattern of involvement in conjunction with the clinical history helps with the diagnosis. The CT findings depend on disease severity and duration and if reperfusion has taken place and can include bowel wall thickening (which may have pronounced hypoattenuation), mucosal hyperattenuation, abnormal mural enhancement, pericolonic fat stranding, and free fluid (Figs 20, E7). Pneumatosis and/or portal venous gas may occur when there is bowel infarction (58,59).

Anorectal Disease.—Hemorrhoids are a dilated arteriovenous plexus arising from the superior and inferior hemorrhoidal veins. They are clas-

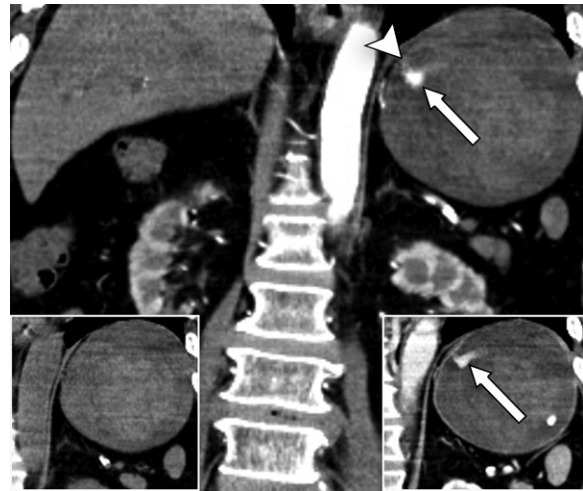


Figure 17. Active bleeding from a Dieulafoy lesion in the proximal stomach in a 62-year-old man with a history of massive hematemesis. Coronal arterial phase (main image), non-contrast (left inset), and portal venous phase (right inset) CTA images show a small hyperenhancing Dieulafoy lesion in the proximal stomach (arrowhead in main image) with a contiguous focus of contrast extravasation in the arterial phase (arrow in main image), which changes in size, attenuation, and shape in the portal venous phase (arrow in right inset). Figure E5 is an image during upper endoscopy that shows a small mucosal defect, containing a visible artery in the proximal stomach, which is consistent with a Dieulafoy lesion. There was no active bleeding at the time of endoscopy.

sified as internal or external on the basis of their location above or below the dentate line (9). Hemorrhoids are commonly present in patients with lower GI bleeding, although they are usually incidental findings, with only 2%–10% reported to cause acute lower GI bleeding (9). On CT images, hemorrhoids appear as enlarged serpiginous veins within the anus and lower rectum that are best defined on portal venous phase images (4).

In patients with portal hypertension, rectal varices are common, occurring in 40%–77% of patients, although significant bleeding has been reported to occur in less than 5%. They appear as serpiginous vessels within the rectal wall that are best defined in the portal venous phase. Although the imaging appearance is identical to hemorrhoids, they can be differentiated by location above the dentate line and the associated findings of portal hypertension in the abdomen and pelvis (Fig 21) (4,27).

Rectal ulcers can cause massive GI bleeding and arise from a variety of processes including stercoral ulceration, solitary rectal ulcer syndrome, neoplasm, trauma, ischemia, inflammatory bowel disease, and infection (9,22,60). When rectal ulcers cause overt lower GI bleeding, contrast extravasation may be visualized, although the actual ulcer may not be visible (Fig 22). Iatrogenic causes such as transrectal biopsy can also cause active GI bleeding.

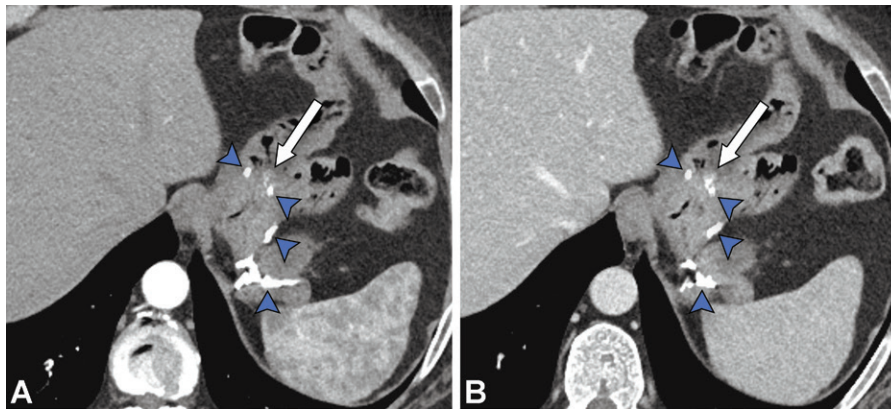


Figure 18. Roux-en-Y gastric bypass with active bleeding in a 49-year-old woman with a history of melena. Axial arterial phase (A) and portal venous phase (B) CTA images show contrast extravasation in the arterial phase at the gastrojejunostomy site (arrow in A), which changes in size, attenuation, and shape in the portal venous phase (arrow in B). There are multiple adjacent surgical clips related to Roux-en-Y gastric bypass surgery (arrowheads). The active bleeding was caused by a marginal ulcer adjacent to the gastrojejunostomy site, which was noted at upper endoscopy.

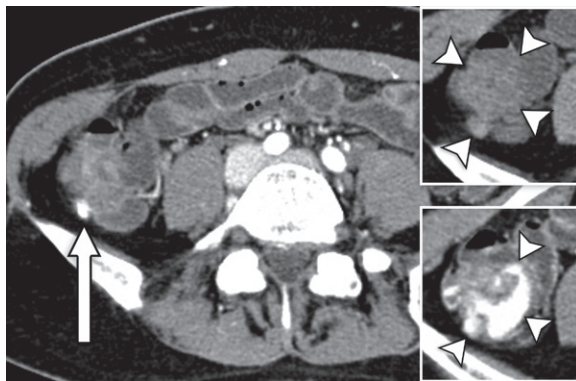


Figure 19. Active bleeding from a cecal diverticulum in a 46-year-old woman with hematochezia. Axial contrast-enhanced arterial phase (main image), noncontrast (top inset), and portal venous phase (bottom inset) CTA images show active extravasation within a cecal diverticulum in the arterial phase (arrow in main image) and further accumulation of contrast extravasation within the colonic lumen in the portal venous phase (arrowheads in bottom inset). The noncontrast image shows a sentinel clot within the cecum and cecal diverticulum (arrowheads in top inset). Figure E6 is a colonoscopic image that shows a blood clot at the orifice of the bleeding diverticulum.

In patients with a history of pelvic malignancy that was treated with radiation therapy, radiation proctopathy can cause GI bleeding owing to the relatively higher radiation doses used for pelvic tumors and the relative rectosigmoid colon immobility (27). When acute inflammation is present, CT findings can include rectal wall thickening, stratified mural hyperenhancement, and mesenteric vascular engorgement (Fig 23) (27,61).

Colorectal Neoplasm.—A colorectal neoplasm can manifest as a focal or circumferential mass (Fig 24), although owing to the lack of colonic preparation before CT, these lesions may not be visualized at the time of the examination. Thus, it is important

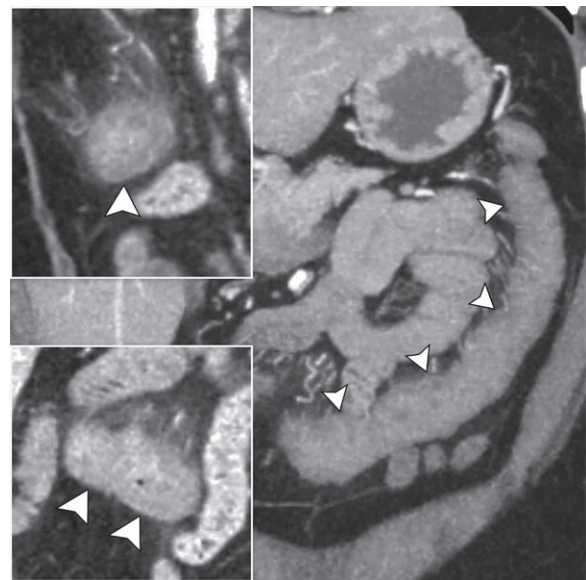


Figure 20. Colonic ischemia in a 48-year-old woman with factor V Leiden thrombophilia who presented with hematochezia. Coronal arterial phase maximum intensity projection (main image), sagittal arterial phase (top inset), and coronal portal venous phase (bottom inset) CT images show circumferential transverse colonic wall thickening with pericolonic fat stranding (arrowheads in all images). Figure E7 is an endoscopic image in this patient that shows diffuse colonic inflammation characterized by erosions, erythema, friability, and granularity. Tissue samples taken during colonoscopy showed ulcerated colonic mucosa, with fibrosis of the lamina propria, which are consistent with ischemic injury.

to “run the bowel” (evaluate the entire colon from proximal to distal) to maximize the likelihood of identifying these lesions. Patients with colon polyps larger than 1 cm may also present with GI bleeding (9). Postpolypectomy bleeding, the most common colonoscopy complication, occurs in 0.2%–1.8% of cases and can occur up to 2 weeks after polypectomy. Most patients with postpolypectomy bleeding

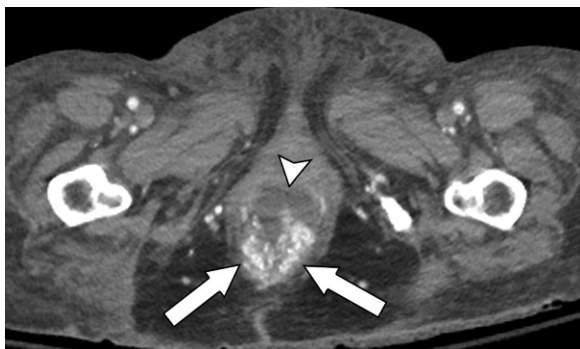


Figure 21. Rectal and perirectal varices in an 80-year-old woman with cirrhosis, portal hypertension, and ascites. Axial portal venous phase CT image shows multiple enhancing distended veins in the region of the rectum and the perirectal region that are consistent with varices (arrows). A small amount of ascites is also noted anterior to the rectum (arrowhead).

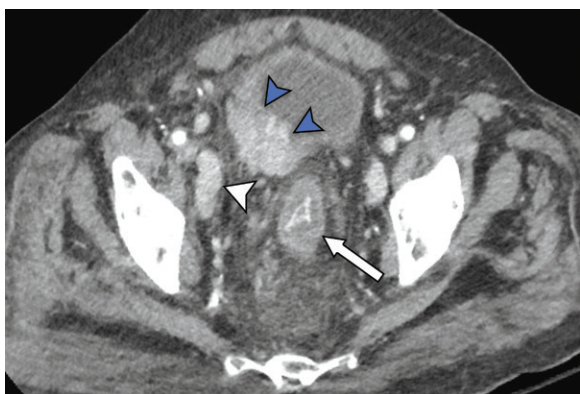


Figure 23. Radiation proctitis in a 67-year-old man with bladder and prostate carcinoma who was recently treated with radiation therapy and presented with lower abdominal pain and hematochezia. Axial portal venous phase CT image shows circumferential rectal wall thickening and edema (arrow), with perirectal edema that is consistent with the patient's history of radiation proctitis. Also noted is a mass representing bladder carcinoma in the right posterior bladder wall (blue arrowheads) and right external iliac lymphadenopathy (white arrowhead).

are immediately referred to colonoscopy for treatment without imaging (4).

Angioectasia.—While diverticulosis is the most common cause of lower GI bleeding, angioectasia is the most common vascular lesion causing lower GI bleeding and has an increased incidence with age. In the colon, angioectasia is more common in the cecum and ascending colon. On CT images, colonic angioectasia can appear as punctate or discoid foci of enhancement in the colon wall (Fig 25). As in the small bowel, angioectasias may be incidental findings unrelated to the cause of bleeding (9,62).

Other Causes of Colitis.—The CT appearance of the various causes of infectious colitis has considerable overlap, and diagnosis is generally based on the clinical findings and laboratory results. CT findings

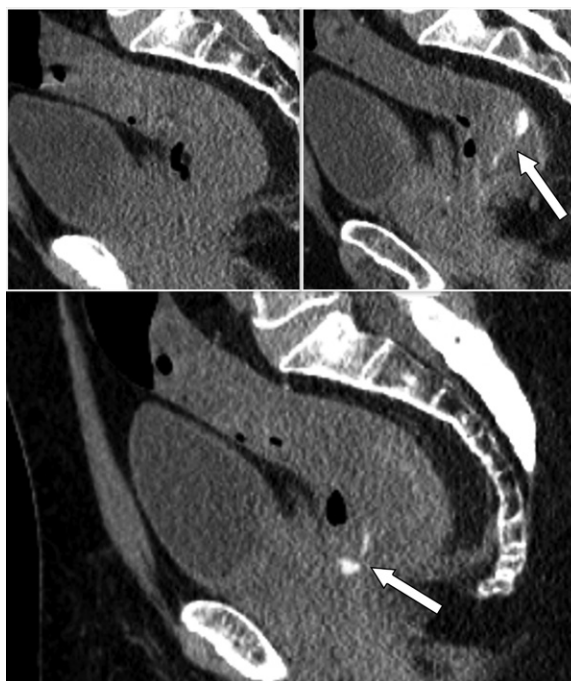


Figure 22. Rectal ulcer causing active GI bleeding in a 59-year-old woman who presented with hematochezia. Sagittal arterial phase (bottom image), noncontrast (top left image), and portal venous phase (top right image) CTA images show foci of contrast extravasation in the lower rectum (arrow in bottom image) which change in size, attenuation, and shape in the portal venous phase (arrow in top right image). A rectal wall ulcer was subsequently found at colonoscopy. (Reprinted, with permission, from reference 13.)

can include bowel wall thickening with hyperenhancement, pericolic stranding, and possibly ascites (Fig 26). Most infectious causes of colitis can involve the entire colon (eg, cytomegalovirus and *Escherichia coli*). However, some have a predilection for the right colon and may involve the ileum (eg, *Salmonella*, *Yersinia*, tuberculosis, and amebiasis). Others can predominantly involve the left colon (eg, schistosomiasis, shigellosis, herpes virus infection, and lymphogranuloma venereum) (58,63). Severe cases of *Clostridium difficile* infection have a characteristic appearance with marked wall edema and haustral fold thickening (58).

Similar to infectious colitis, the CT appearance of inflammatory bowel disease and nonsteroidal anti-inflammatory drug (NSAID)-induced colopathy is often nonspecific, requiring appropriate clinical history for diagnosis (64).

Small Bowel Bleeding

Inflammatory Causes

Small Bowel Ulcers.—Small bowel ulcers can be caused by Crohn disease, celiac disease, medications, infection, ischemia, cryptogenic multifocal ulcerous stenosing enteritis, or neoplasms. Small

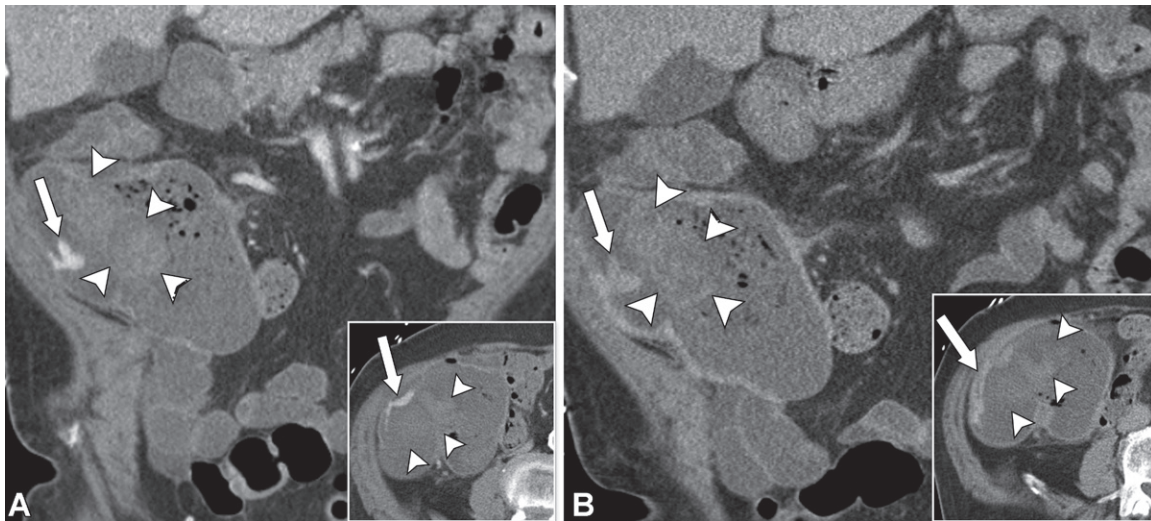


Figure 24. Colon carcinoma with active bleeding in an 84-year-old man with a supratherapeutic international normalized ratio who presented with bright red blood in the rectum and intermittent hypotension. Arterial phase coronal (main image) and axial (inset) (A) and delayed phase coronal (main image) and axial (inset) (B) CTA images show contrast extravasation in the arterial phase (arrows in A), which changes in size, attenuation, and shape in the delayed phase (arrows in B). Also noted is a heterogeneous mildly enhancing mass in the ascending colon (arrowheads in A and B). Subsequent colonoscopy showed an ascending colon mass that was confirmed to represent an adenocarcinoma at surgical pathologic examination. (Reprinted, with permission, from reference 4.)



Figure 25. Cecal angioectasia with active bleeding and jejunal GIST in a 73-year-old man with a history of occult GI bleeding. Coronal (main image) and axial (right inset) portal venous phase and axial arterial phase (left inset) CTE images show active extravasation in the cecum that appears in the arterial phase and changes in size, attenuation, and shape in the portal venous phase (arrow in all images) caused by cecal angioectasia, which was confirmed at colonoscopy. There is also a hyperenhancing exophytic mass in the jejunum (arrowheads in main image) that is consistent with a GIST, which was confirmed at surgical resection.

bowel ulcers and erosions are usually not visible on CT images. However, the involved bowel segment may show inflammatory findings.

Crohn Disease.—Severe lower GI bleeding with Crohn disease is an uncommon complication,

occurring in 0.6%–6% of patients (Figs 27, 28) (65). When patients with Crohn disease present with overt lower GI bleeding, diagnosing the site of bleeding and patient treatment can be challenging for the following reasons: (a) Crohn disease can involve multiple bowel segments, any of which can be the site of bleeding, and active bleeding may have stopped at the time of scanning; (b) the presence of strictures may preclude endoscopic evaluation; and (c) there is an increased risk of recurrent bleeding, occurring in 19%–41% of patients (66).

Nonsteroidal Anti-Inflammatory Drug Enteropathy.—NSAIDs may result in bowel injury with erosions, ulceration, and stricturing (67). This NSAID enteropathy is also referred to as *diaphragm disease* because the associated strictures are circumferential and short, resembling diaphragms. At cross-sectional imaging, the NSAID enteropathy hallmark is multiple short-segment, or diaphragm-like strictures that tend to be clustered within the same region or segment of the bowel, usually in the ileum. The wall associated with the strictures is typically thickened and hyperenhancing (Fig 29) (67). Potential mimics of NSAID enteropathy at imaging include Crohn disease, radiation-induced strictures, and cryptogenic multifocal ulcerous stenosing enteritis.

Vascular Lesions

Small bowel vascular lesions identified with endoscopy are classified according to the criteria proposed by Yano et al (62). In these criteria, type

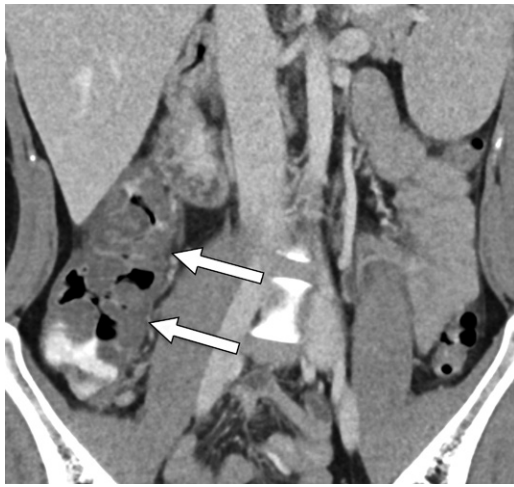


Figure 26. Hemorrhagic *Escherichia coli* colitis in a 49-year-old man who presented with abdominal cramps and blood in the stool. Coronal dual-energy mixed (blended) CT image shows marked cecal and ascending colonic wall thickening, with associated pericolic fat stranding (arrows). High-attenuation intraluminal fluid in the colon was likely ingested material, given its presence on virtual noncontrast images (not shown). Clinically, the patient was found to have hemorrhagic *E coli* colitis.

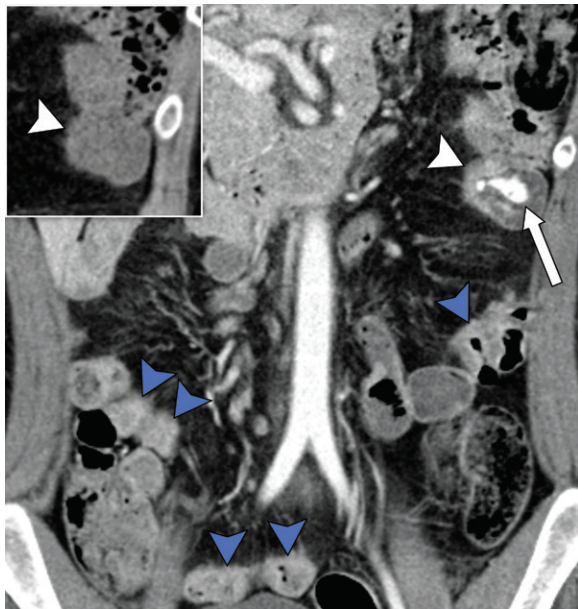


Figure 27. Active inflammatory small bowel Crohn disease with active bleeding in a 29-year-old man who presented with hematochezia and hemodynamic instability. Coronal portal venous phase (main image) and noncontrast (inset) CT images show asymmetric mural thickening and hyperenhancement involving the mesenteric wall of a distal jejunal bowel segment, which are consistent with active inflammatory Crohn disease (white arrowhead in both images) associated with contrast extravasation (arrow). Also noted are multiple additional segments of active inflammation in the jejunum and ileum (blue arrowheads).

1 lesions appear as punctulate or patchy regions of erythema, and represent angioectasia at pathologic examination. Yano type 2 or type 3 lesions



Figure 28. Active inflammatory small bowel Crohn disease with active bleeding in a 54-year-old man who presented with hematochezia and hemodynamic instability. Coronal portal venous phase (main image) and noncontrast (inset) CT images show asymmetric mural thickening and hyperenhancement involving the mesenteric wall of a segment of ileum, which are consistent with active inflammatory Crohn disease (arrowhead in both images) associated with contrast extravasation (arrows).

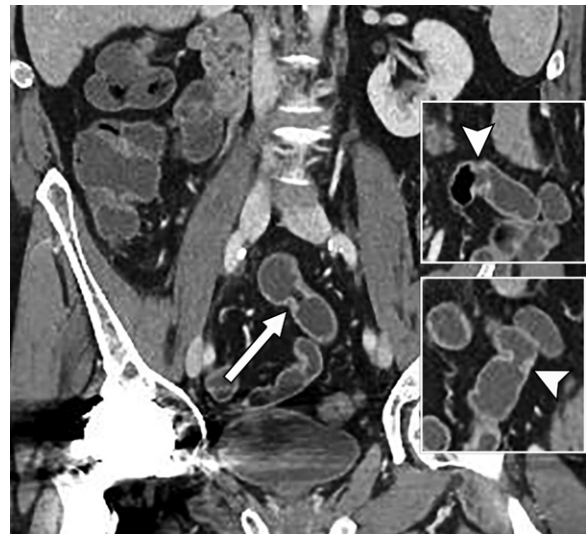


Figure 29. NSAID enteropathy in a 63-year-old man with irritable bowel syndrome and arthritis and chronic NSAID treatment who presented with occult GI bleeding and inconclusive findings at endoscopy (not shown). Coronal CTE images show three of the multiple diaphragm-like strictures that were present in the mid small bowel (arrow in main image, arrowhead in top and bottom inset), which are consistent with stricturing related to NSAID enteropathy.

have arterial pulsations and pathologically represent Dieulafoy lesions and arteriovenous malformations. All other lesions are classified as type 4. At multiphasic CT, vascular lesions are characterized according to their pattern of enhancement as arterial lesions, angioectasia, or venous lesions,

Table 8: Small Bowel Vascular Lesions Causing GI Bleeding: Lesion Enhancement during CT Acquisition Phases

Type	Pathology	Prevalence	Enhancement during CT Acquisition Phase		
			Arterial Phase	Enteric Phase	Delayed Phase
Type 1	Angioectasia	Common	–	++	+
Types 2 and 3 (high-flow lesions)*	Dieulafoy lesion, arteriovenous malformation	Uncommon	+++†	–	–
Type 4 (venous lesions)	Small bowel varices, venous malformation, hemangioma‡	Uncommon	–	+	+++

Note.— – = lesion has enhancement similar to the bowel wall, + = lesion has more enhancement than the bowel wall.

*Patients can present with life-threatening GI bleeding.

†The presence of an enlarged or early enhancing draining vein is diagnostic of an arteriovenous malformation.

‡Hemangioma peak enhancement may be lower and occur later than other venous lesions.

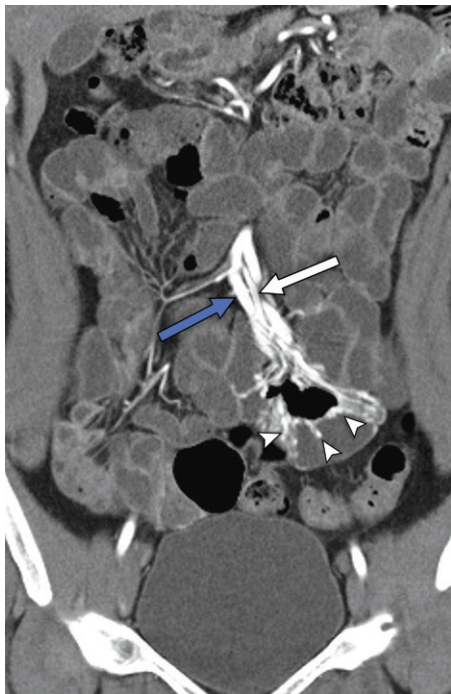


Figure 30. Arteriovenous malformation in the mid ileum in a 40-year-old woman with a history of postprandial abdominal pain and a positive stool occult blood test. Coronal arterial phase CTE image shows an enlarged ileal arterial branch (ie, feeding artery) (white arrow), enlarged tortuous vessels in the bowel wall (arrowheads), and early venous drainage (blue arrow), which are consistent with an arteriovenous malformation.

providing good correlation with the endoscopic classification (Table 8) (54).

Small bowel arterial lesions are uncommon high-flow lesions that are important to exclude, because they can lead to life-threatening GI bleeding. Arterial lesions include Dieulafoy lesions and arteriovenous malformations.

Dieulafoy Lesions.—As discussed in the upper GI bleeding section, Dieulafoy lesions are most com-

monly located in the proximal stomach. Sixteen percent of them occur in the small bowel, most commonly in the duodenum (29,54,55). On multiphase CT images, similar to arteriovenous malformations, Dieulafoy lesions have the highest attenuation during the arterial phase, although they are distinguished from arteriovenous malformations by the lack of an enlarged or early enhancing draining vein (54).

Arteriovenous Malformations.—The term *arteriovenous malformation* describes a vascular lesion with an abnormal communication between an artery and vein (54). On CT images, during the arterial phase, an arteriovenous malformation appears as a highly enhancing nidus, similar to a Dieulafoy lesion. However, the additional finding of an enlarged or early enhancing draining vein is diagnostic of an arteriovenous malformation (Fig 30).

Angioectasias.—Angioectasia (also known as *angiodyplasia* or *vascular ectasia*), the most common cause of small bowel bleeding, is more common in older patients (6). Angioectasia is thought to occur from chronic intermittent obstruction of veins as they pass through the contracting bowel wall muscular layers. On CT images, angioectasias appear as nodular or discoid foci of enhancement in the bowel wall, or focal ectasia of the intramural veins, which are usually not well seen in the arterial phase, show the highest enhancement in the enteric or portal venous phase, and decrease in conspicuity in the delayed phase (Fig 31). These lesions are usually multiple, most commonly occurring in the jejunum (13,54). Angioectasias may be incidental findings that are unrelated to GI bleeding, although since they can cause bleeding, this finding should generally be included in the radiology report. Angioectasias may appear more conspicuous on maximum intensity projection images (68).

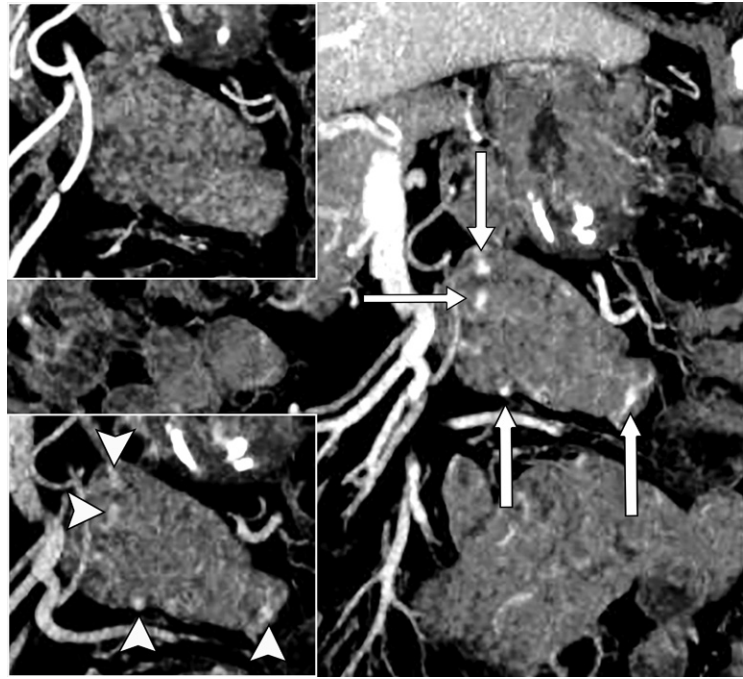


Figure 31. Small bowel angioectasias in a 76-year-old woman who was taking anticoagulation medication, experienced multiple episodes of melena that required hospitalization and transfusions, and underwent multiple upper and lower endoscopic examinations that were negative for the cause of bleeding. Coronal enteric phase (main image), arterial phase (top inset), and 90-second venous phase (bottom inset) maximum intensity projection CTE images show multiple nodular- and discoid-shaped hyperenhancing foci in the jejunum (arrowheads in bottom inset), which are most conspicuous in the enteric phase (arrows in main image) and are consistent with angioectasias.

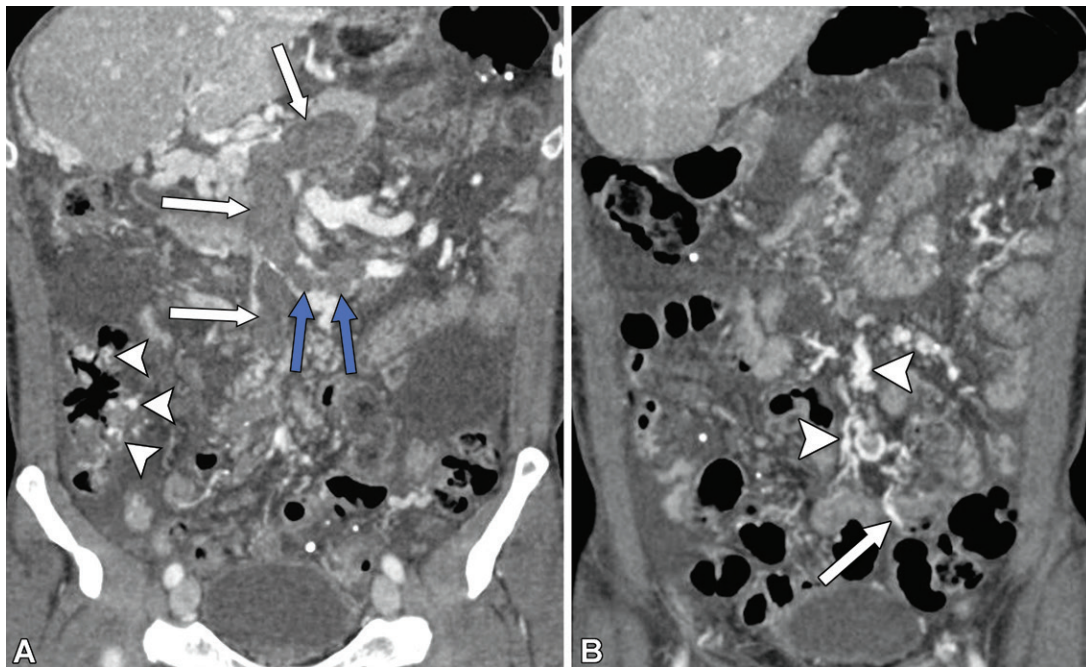


Figure 32. Ectopic varices in the small bowel and colon resulting from acute superimposed on chronic thrombus in the portal venous system in a 58-year-old man who presented with abdominal pain, melena, and a new hemoglobin level decrease. Coronal portal venous phase CT images show acute thrombus throughout the superior mesenteric vein and its tributaries (white arrows in A), extending into a surgically placed splenorenal shunt (blue arrows in A). Also noted are multiple varices in the right colon (arrowheads in A). There are multiple associated mesenteric venous collaterals (arrowheads in B) as well as small bowel varices (arrow in B).

Venous Lesions.—Small bowel vascular lesions, which have minimal or no enhancement during the arterial phase and become brighter on enteric and delayed phase images, may represent varices, a venous malformation, or a hemangioma (54). Varices can occur in patients with portal hypertension, those who have undergone surgery involving

the abdominal organs or vessels, or those with mesenteric venous obstruction. With portal hypertension, varices are usually located in the gastroesophageal region (Fig 12). Ectopic varices are portosystemic venous collateral vessels occurring outside the gastroesophageal region, usually in the GI tract, including the small bowel and umbilical

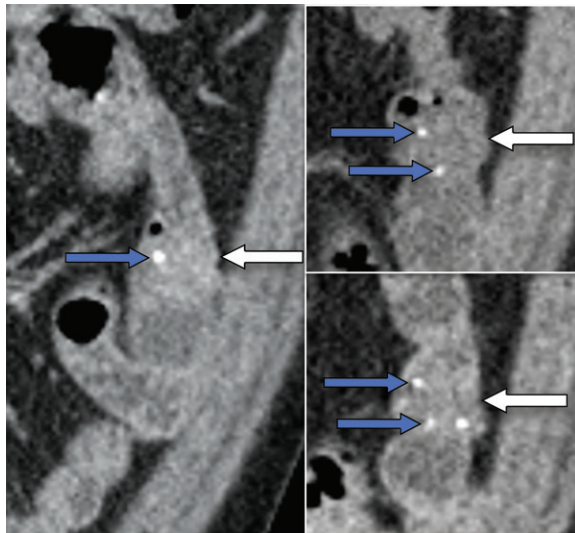


Figure 33. Small bowel hemangioma with calcified phleboliths in a 48-year-old man with iron deficiency anemia, a positive fecal occult blood test result, and a nodular erythematous lesion at capsule endoscopy. Coronal delayed phase (left image), arterial phase (top right image), and enteric phase (bottom right image) CTE images show a progressively enhancing small bowel mass (white arrow in all images), containing several calcified phleboliths (blue arrows in all images). On multiphasic CT images, phleboliths are best visualized in the earliest postcontrast phase, which in this case is the arterial phase. (Reprinted, with permission, from reference 13.)



Figure 34. Multiple plaque-like NETs in a 60-year-old woman with known lung and bone NET metastases. Prior abdominal MRI examination (not shown) showed mesenteric lymphadenopathy that was suspicious for a small bowel origin of metastatic NET. Enteric phase CTE images show two of numerous hyperenhancing plaque-like lesions in the mid small bowel (arrow in main image, arrowhead in inset), which are consistent with NETs. There was associated mesenteric lymphadenopathy (not shown). NETs were subsequently confirmed at surgery.

region (Fig 32) (69). Venous malformations are uncommon and are usually sporadic and solitary but can be multiple when associated with the *blue*



Figure 35. NET with serosal retraction in a 52-year-old woman who presented with anemia. Esophagogastroduodenoscopy, colonoscopy, and capsule endoscopy (not shown) did not show a source of bleeding. Coronal enteric phase CTE image demonstrates a hyperenhancing eccentric mass (arrow) in the ileum, with serosal retraction. NET was subsequently confirmed at surgical resection.

rubber bleb nevus syndrome. Venous malformation and hemangioma may manifest as a polypoid or infiltrative small bowel mass. These lesions generally have progressive enhancement that may follow a peripheral to central pattern or may be heterogeneous. Venous malformation and hemangiomas may contain calcified phleboliths (Fig 33) (70).

Small Bowel Tumors

Small bowel neoplasms are uncommon, representing 3% of GI neoplasms and 0.5% of all neoplasms. Small bowel neoplasms encompass benign entities such as adenomatous or hamartomatous polyps, and primary or secondary malignant masses.

Neuroendocrine Tumors.—Neuroendocrine tumors (NETs) represent the most common primary small bowel malignancy and most commonly occur in the distal ileum (26).

At cross-sectional imaging, small NETs have a characteristic flat, or plaque-like appearance (Fig 34). As the tumor enlarges, a desmoplastic reaction typically forms in the adjacent mesentery, resulting in the mass assuming a U-shaped or horseshoe appearance (Fig 35). NETs are characteristically hyperenhancing on arterial or enteric phase images. The tumors are frequently multiple, with two or more lesions identified in the same bowel segment. NETs frequently metastasize to nearby lymph nodes, which may appear hyperenhancing, clustered, or calcified (71). NETs' production

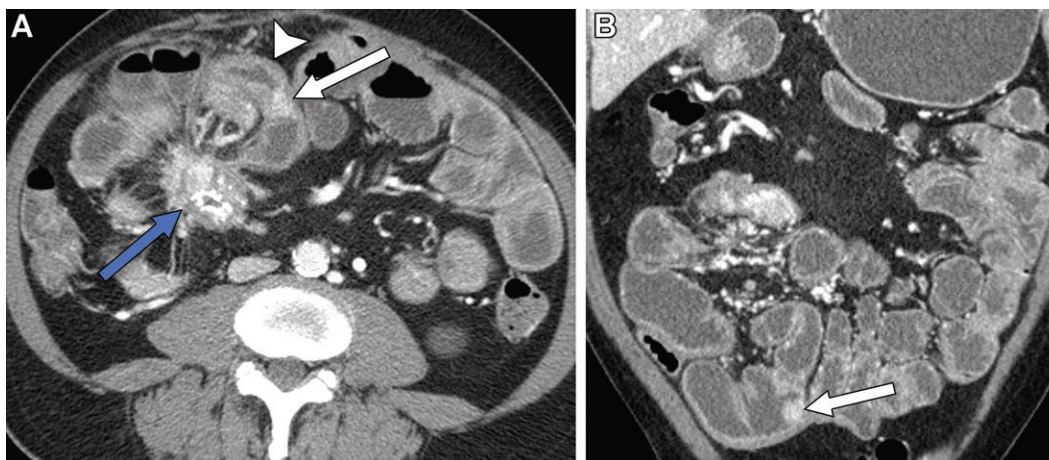


Figure 36. Multiple NETs, with an associated mesenteric metastasis and adjacent small bowel wall thickening, resulting from venous congestion in a 52-year-old woman with abdominal pain and overt GI bleeding. Axial (A) and coronal (B) portal venous phase CTE images show two of several hyperenhancing masses in the ileum that are consistent with NETs (white arrow in A and B). There is an associated spiculated mesenteric metastasis that contains coarse calcifications (blue arrow in A) and is tethered to adjacent small bowel loops, and adjacent small bowel wall thickening, resulting from venous congestion (arrowhead in A).

of serotonin and other vasoactive substances can cause a local desmoplastic reaction in the mesentery with spiculated or stellate soft-tissue thickening, commonly with calcification; bowel loop kinking, potentially causing bowel obstruction; serosal retraction; vascular encasement; and mural thickening secondary to ischemia (Fig 36) (26). Distant metastases are most commonly found in the liver, peritoneum, lungs, and bone.

Adenocarcinoma.—Adenocarcinoma is the second most common primary small bowel malignancy. Adenocarcinomas usually arise in the proximal small bowel, most commonly in the duodenum, followed by the jejunum, and thus, they are commonly identified with advanced endoscopic techniques such as video capsule endoscopy and push (extended) enteroscopy (13,26). However, in patients with Crohn disease, adenocarcinoma more commonly arises in the ileum. On CT images, adenocarcinoma commonly forms a single poorly enhancing mass that may grow with a circumferential or nodular pattern (Figs 37, E8). When growth is circumferential, the bowel lumen may have an “apple core” appearance and cause bowel obstruction. Adenocarcinoma can metastasize to local lymph nodes, the peritoneum, and the liver (26).

Gastrointestinal Stromal Tumors.—GISTs represent less than 10% of primary small bowel neoplasms and most commonly arise in the stomach, followed by the jejunum and the ileum (26,72). GISTs originate from the muscular layer of the bowel wall and can grow endophytically, exophytically, or both. Patients with endophytic tumors can present early with bowel obstruction, whereas

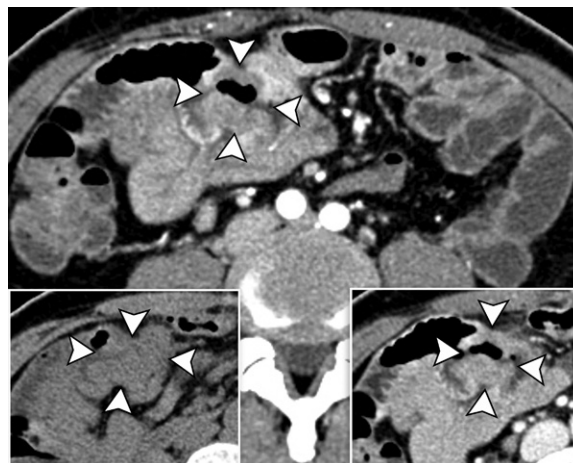


Figure 37. Jejunal adenocarcinoma in a 46-year-old woman with a positive fecal occult blood test. Axial arterial phase (main image), noncontrast (left inset), and portal venous phase (right inset) CTE images show an enhancing 3.2-cm luminal encircling mass with raised rolled edges in the proximal jejunum (arrowheads in all images). Figure E8 is an endoscopic image in this patient that shows the jejunal mass with an area of hemorrhage.

exophytic growth is associated with delayed diagnosis and larger size at patient presentation. On CT images, most GISTs form a well-circumscribed, heterogeneously hyperenhancing mass (Figs 38, E9). Imaging findings of ulceration, necrosis, and irregular or locally invasive growth are associated with high-grade or overtly malignant GISTs (72). Metastases are most commonly found in the liver and peritoneum. Local lymph node metastases are infrequent.

Lymphoma.—Lymphoma may involve the bowel as part of systemic disease or may arise from primary



Figure 38. Active bleeding from an exophytic small bowel GIST in a 50-year-old man with hematochezia. Coronal arterial phase (main image), noncontrast (left inset), and portal venous phase (right inset) CTE images show a hyperenhancing exophytic mass at the jejunioileal junction (arrow in all images), with an appearance consistent with a GIST. There is associated intraluminal contrast extravasation in the arterial phase (arrowheads in main image) that changes in size, attenuation, and shape in the portal venous phase (arrowheads in right inset). Figure E9 is a photograph of the gross specimen of the resected small intestine in this patient, showing a 3.4-cm exophytic hemorrhagic mass. A GIST was confirmed at surgical pathologic examination.



Figure 39. Small bowel lymphoma with aneurysmal dilatation in a 55-year-old man with iron deficiency anemia. Axial (main image) and coronal (inset) arterial phase CTE images show diffuse wall thickening involving a segment of the distal ileum with aneurysmal dilatation (arrow in both images). Findings are consistent with small bowel lymphoma. At surgical pathologic examination, a circumferential partially exophytic mass with central ulceration was noted. Diffuse large B-cell lymphoma was confirmed at surgical pathologic examination.



Figure 40. Multiple small bowel metastases from renal cell carcinoma in a 55-year-old man with a history of left renal cell carcinoma who underwent nephrectomy 5 years ago and now presents with occult GI bleeding. Axial arterial phase CT images show multiple hyperenhancing small bowel lesions (arrow in main image and arrowheads in inset image), which are consistent with metastases.

GI lymphoid tissue (26). On CT images, lymphoma may appear as a single mass or multiple small bowel masses. It may have a circumferential or nodular growth pattern, resembling adenocarcinoma. Aneurysmal dilatation of the lumen can be a helpful finding to differentiate lymphoma from adenocarcinoma, which tends to narrow the lumen (Fig 39). Lymphoma is commonly associated with bulky regional lymphadenopathy and splenomegaly, which can be helpful for diagnosis (26). Refractory celiac disease can be complicated by enteropathy-associated T-cell lymphoma and ulcerative jejunitis, both of which can cause GI bleeding, although when iron deficiency anemia occurs in patients with celiac disease, this is usually caused by malabsorption.

Metastases.—Metastases to the small bowel are the most common small bowel malignancy, representing approximately 50% of all small bowel neoplasms (73). A metastasis should be the primary consideration when identifying a new small bowel mass in a patient with a known malignancy. Hematogenous metastasis to the small bowel may arise from melanoma or lung or breast tumors, while direct peritoneal spread can occur from ovarian, gastric, or colonic tumors (26). Metastases may manifest as a single mass or multiple discrete masses or with peritoneal involvement. The appearance can range from small polypoid nodules to large lesions with aggressive features (Figs 40, E10) (74).

Other Causes

Meckel Diverticulum.—Meckel diverticulum represents an embryologic omphalomesenteric

duct remnant that communicates with the ileal lumen. At cross-sectional imaging, a Meckel diverticulum appears as a blind-ending bowel segment originating from the mid to distal ileum (Figs 41, E11). This structure can be difficult to identify, because the caliber, mural enhancement, and luminal contents may be similar to those of the adjacent ileum. Retrograde tracking of the small bowel from the ileocecal valve may help with identification. Meckel diverticulum can contain ectopic tissue, most commonly gastric or pancreatic, which can cause mural nodularity, thickening, or hyperenhancement (6,75). Several types of malignant masses can occur in a Meckel diverticulum, most commonly a NET (26,75).

Gastrointestinal Bleeding CT Reporting Template

When radiologists interpret GI bleeding CT examinations, using a CT reporting template such as the one available at <https://radreport.org/home/50829> may help in creating a structured report that is clear and concise for referring physicians.

Conclusion

GI bleeding is a potentially life-threatening medical condition that often requires multidisciplinary collaboration to make the proper diagnosis and select the appropriate treatment. CT is an important imaging technique for diagnosing the cause of GI bleeding and is usually complementary to conventional angiography and nuclear medicine. Interpretation of GI bleeding cases on CT images can be challenging owing to the large number of images to interpret and the wide variety of potential causes of bleeding. This pictorial review by the Society of Abdominal Radiology GI Bleeding Disease-Focused Panel provides a practical resource for radiologists interpreting GI bleeding CT studies so that their interpretation and radiology reports can be clear and concise and facilitate prompt clinical management.

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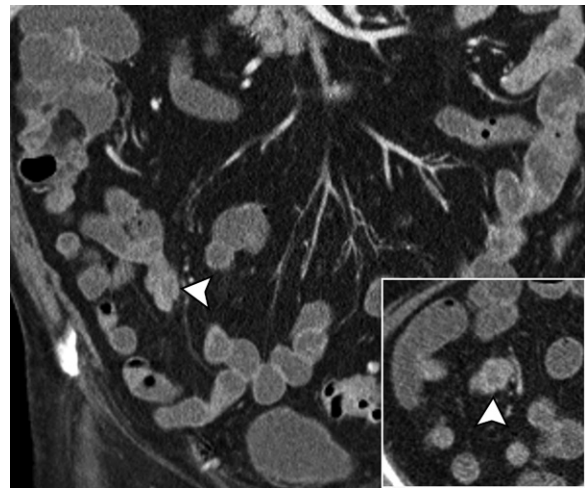


Figure 41. Meckel diverticulum in a 40-year-old man with occult GI bleeding. Coronal (main image) and axial (inset) portal venous phase CTE images show a hyperenhancing mildly thick-walled blind-ending tubular structure arising from the distal ileum, which is consistent with a Meckel diverticulum. At surgical resection, a Meckel diverticulum with associated inflammation was noted.

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Disclosures of Conflicts of Interest.—**O.L.B.** Activities related to the present article: editorial board member of *RadioGraphics* (not involved in the handling of this article). Activities not related to the present article: disclosed no relevant relationships. Other activities: disclosed no relevant relationships. **M.L.G.** Activities related to the present article: disclosed no relevant relationships. Activities not related to the present article: grants/grants pending from Annalise.ai and Philips Healthcare. Other activities: disclosed no relevant relationships.

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