Abdominal Radiology

Published online: 20 December 2016

Abdom Radiol (2017) 42:1319–1341
DOI: 10.1007/s00261-016-1015-1

Multidetector CT enterography of focal small bowel lesions: a radiological–pathological correlation

Farnoosh Sokhandon, Sayf Al-katib, Lawrence Bahoura, Alexander Copelan, Daniel George, Dominic Scola

Department of Radiology, Beaumont Health, Oakland University William Beaumont School of Medicine, Royal Oak, 3601 W 13 Mile Rd, Royal Oak, MI 48073, USA

Abstract

Focal small bowel lesions present a diagnostic challenge for both the radiologist and gastroenterologist. Both the detection and characterization of small bowel masses have greatly improved with the advent of multidetector CT enterography (MD-CTE). As such, MD-CTE is increasingly utilized in the workup of occult gastrointestinal bleeding. In this article, we review the spectrum of focal small bowel masses with pathologic correlation. Adenocarcinoma, the most common primary small bowel malignancy, presents as a focal irregular mass occasionally with circumferential extension leading to obstruction. Small bowel carcinoid tumors most commonly arise in the ileum and are characterized by avid enhancement and marked desmoplastic response of metastatic lesions. Aneurysmal dilatation of small bowel is pathognomonic for lymphoma and secondary findings of lymphadenopathy and splenomegaly should be sought. Benign small bowel masses such as leiomyoma and adenoma may be responsible for occult gastrointestinal bleeding. However, primary vascular lesions of the small bowel remain the most common cause for occult small bowel gastrointestinal bleeding. The arterial phase of contrast obtained with CTE aids in recognition of the vascular nature of these lesions. Systemic conditions such as Peutz–Jeghers syndrome and Crohn's disease may be suggested by the presence of multiple small bowel lesions. Lastly, potential pitfalls such as ingested material should be considered when faced with focal small bowel masses.

Key words: CT enterography—Focal small bowel

Review of current literature reveals few studies and published data dedicated to the imaging of focal small bowel lesions with multidetector computed tomography enterography (MD-CTE). It is well accepted that traditional barium exams, endoscopy, and routine abdominal and pelvic CT scans have a low yield for diagnosis of focal enteric pathology.

The clinical presentation of focal small bowel lesions varies widely. Patients may present with pain, obstruction, bleeding, anorexia, weight loss, perforation, or jaundice [1]. The non-specific nature of these symptoms and the lack of reliable clinical findings may result in a significant delay in diagnosis [2].

MD-CTE is most commonly performed for evaluation of new onset, recurrence, or complications of Crohn's disease and other enteric inflammatory processes. Other indications include evaluation for infectious enteritis, mesenteric ischemia, malignant or benign small bowel neoplasms, and occult GI bleeding.

Capsule endoscopy is considered the preeminent imaging modality for patients with suspected small bowel blood masses, but the improved temporal resolution of late-generation multidetector row CT scanners and improved bolus tracking techniques enhance the radiologic conspicuity of hyperenhancing small bowel lesions [3]. There is a paradigm shift in the evaluation of patients with small bowel bleeding due to MD-CTE, capsule endoscopy (CE), and other endoscopic developments [4]. MD-CTE is increasingly being used for the identification and determination of etiology of occult GI bleeding [5].

In this review, we present a variety of focal small bowel lesions and their appearance on MD-CTE. Images are correlated with gross and microscopic pictures when applicable.

Correspondence to: Sayf Al-katib; *email:* Sayf.Al-Katib@beaumont.org

Table 1. Summary of imaging and pathological features of focal small bowel lesions

Diagnosis	Background information	Location	Imaging features	Pathology
Adenocarcinoma	Most common small bowel malignancy. Most common presenting symptoms is abdominal pain	Most common in duodenum. Often in ileum in setting of Crohn's disease	Focal circumferential soft tissue mass resulting in irregular luminal narrowing ± regional lymphadenopathy	May arise from adenomatous polyps
Carcinoid tumor	Second most common small bowel malignancy. Rarely presents with carcinoid syndrome (watery diarrhea, flushing and endocardial fibrosis) when disease spreads to the liver	Appendix most common site followed by ileum	Avidly enhancing polypoid mass. Primary tumor usually small. Tumor spread with calcified mesenteric mass with desmoplastic response and tethering of adjacent small bowel loops. Liver metastasis usually hypervascular	Well-differentiated neuroendocrine tumor. Incite fibrosis which results in desmoplastic response on imaging
Lymphoma	Third most common small bowel malignancy. Inflammatory bowel disease and history of solid organ transplantation are known risk factors	Stomach is the most common site of gastrointestinal tract involvement followed by ileum	Infiltrating mildly enhancing mass ± regional lymphadenopathy and splenomegaly. Classic "aneurysmal bowel dilatation" appearance of affected segment. May show ulceration, perforation and sterile abscesses	Non-Hodgkin B-cell lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma) is the most common subtype. T-cell lymphoma has high association with celiac disease. Disruption of autonomic nerve plexus in muscularis propria of bowel results in aneurysmal dilatation
Gastrointestinal stromal tumor	Most common mesenchymal tumor of the gastrointestinal tract. Part of Carney's triad. Increased incidence in NF-1 patients	Most common in the stomach followed by small bowel	Often large, hypervascular and exophytic. May have areas of necrosis and intratumoral hemorrhage	Larger tumors correlate with higher pathologic grade nd predict poor clinical outcome
Melanoma	Most often due to metastasis although rarely primary small bowel melanoma may occur	Small bowel is the most common site of gastrointestinal tract metastasis of melanoma	Solitary enhancing small bowel mass	Primary small bowel melanoma can be suspected when no other organs including skin are affected
Metastasis	Metastasis is overall more common than primary small bowel tumors		Often have similar imaging features to primary tumor	Routes of spread to small bowel include intraperitoneal seeding, hematogenous and direct invasion
Leiomyoma	Most common benign small bowel tumor	Most common mesenchymal tumor of esophagus. Less common in small bowel	May have overlap of imaging features with GIST. Solitary mass with homogenous enhancement	Well-differential smooth muscle cells without mitosis helps to distinguish from leiomyosarcoma
Adenoma	Second most common benign tumor of the small bowel. Patients with familial adenomatous polyposis syndrome are at increased risk	Duodenum most common site	Sessile or pedunculated masses with homogenous enhancement	Sessile type has highest risk for malignant degeneration into adenocarcinoma
Lipoma	Third most common benign small bowel tumor. Multiple subserosal lipomas are associated with Crohn's disease	Colon is the most common site of gastrointestinal tract involvement followed by the small bowel. Ileum is the most common segment of small bowel	Fat attenuation well-circumscribed mass. Solid, non-lipomatous component may be due to ulceration as liposarcoma is rare	Originate from submucosal layer of bowel. Outward extension is limited by muscularis propria
Angiodysplasia	Most common cause of small bowel bleeding. Associated with end-stage renal disease and aortic stenosis	Most common in the right colon but can occur throughout the small bowel	Tuft -like hypervascular enhancing focus less than 5 mm which fades on delayed phases. Multifocal in 40% to 75%	Composed of abnormally dilated thin-walled vessels with high propensity for bleeding
Arteriovenous malformation (AVM)	Most common in the colon. Jejunum is most common site of small bowel involvement followed by ileum	Most common in the right colon but can occur throughout the small bowel	Enlarged feeding artery and early draining vein with hypervascular focus which fades on delayed phases of imaging	Thought to evolve from angiodysplasia in the small bowel
Ectopic pancreas	Usually incidental however may because symptomatic in cases of pancreatitis or development of neoplasm	Duodenum most common site followed by stomach and jejunum	Extramucosal circumscribed enhancing mass which may have a central umbilication of the rudimentary duct.	Similar histologic appearance to normal pancreas. Overlying mucosa may have marked inflammation

Summary statement

In this article, we review the MD-CTE appearance of focal small bowel lesions including neoplastic (both malignant and benign masses), vascular lesions, and non-neoplastic lesions with pathologic correlation and review potential imaging pitfalls encountered on MD-CTE. Table 1 summarizes some of the imaging and pathological features of focal small bowel lesions presented in this review article.

Technique

MD-CTE utilizes negative oral contrast agent composed of low density barium sulfate suspension, VoLumen® (Bracco, Milan, Italy) which results in uniform low attenuation distention of the small bowel lumen. Polyethylene glycol (PEG) can also be used as a negative enteric contrast. When combined with arterial phase imaging after intravenous contrast administration, MD-CTE allows for optimal detection of enhancing small bowel lesions. At our institution, patients ingest a total of

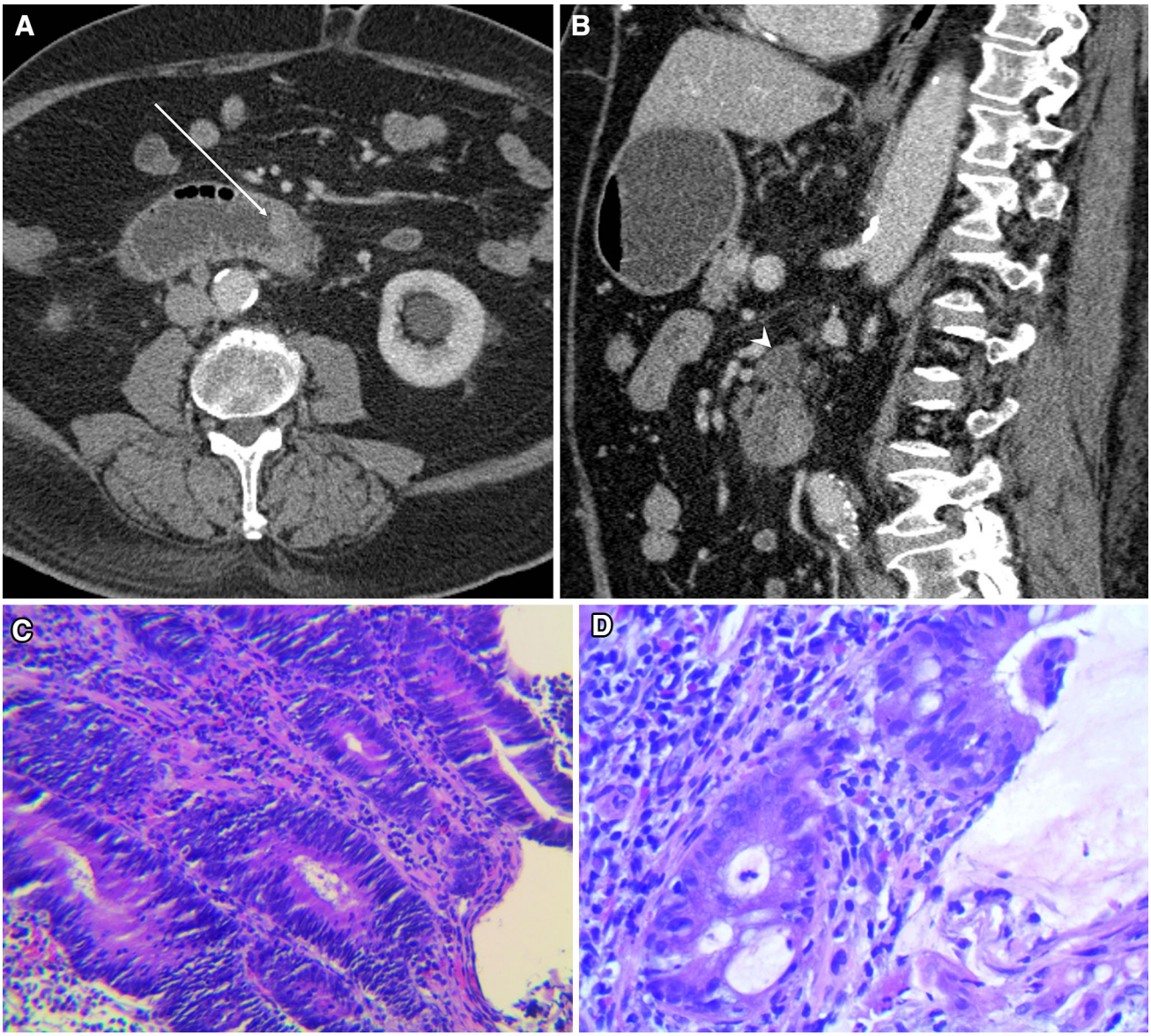


Fig. 1. Duodenal adenocarcinoma in a 76-year-old male who presented with epigastric pain, nausea, and vomiting for 3 days. Axial image from CTE (**A**) shows focal circumferential mass in the duodenum (*arrow*). There is shouldering with significant narrowing of the lumen and dilatation of the duodenum proximal to the mass. Sagittal image (**B**) shows extension of the mass to adjacent mesenteric lymph nodes (*arrowhead*). Hematoxylin and eosin photomicrograph ×200 magnification (**C**) shows a villous adenoma with high-grade dysplasia. Additional section reveals invasive adenocarcinoma in the duodenal submucosa with mucin production (**D**).

three 450 ml bottles of VoLumen spaced 15 min apart with a glass of water just prior to the scan to distend the stomach. A dose of 80–120 cc of intravenous contrast is injected intravenously at a rate of 5 cc/s with a scan delay of 50 s. Contrast dose is adjusted based on patient body mass index and renal function. Images are obtained with a slice thickness of 1 mm × 0.5 mm with 1 mm × 1 mm reconstructions. Source images are routinely processed at

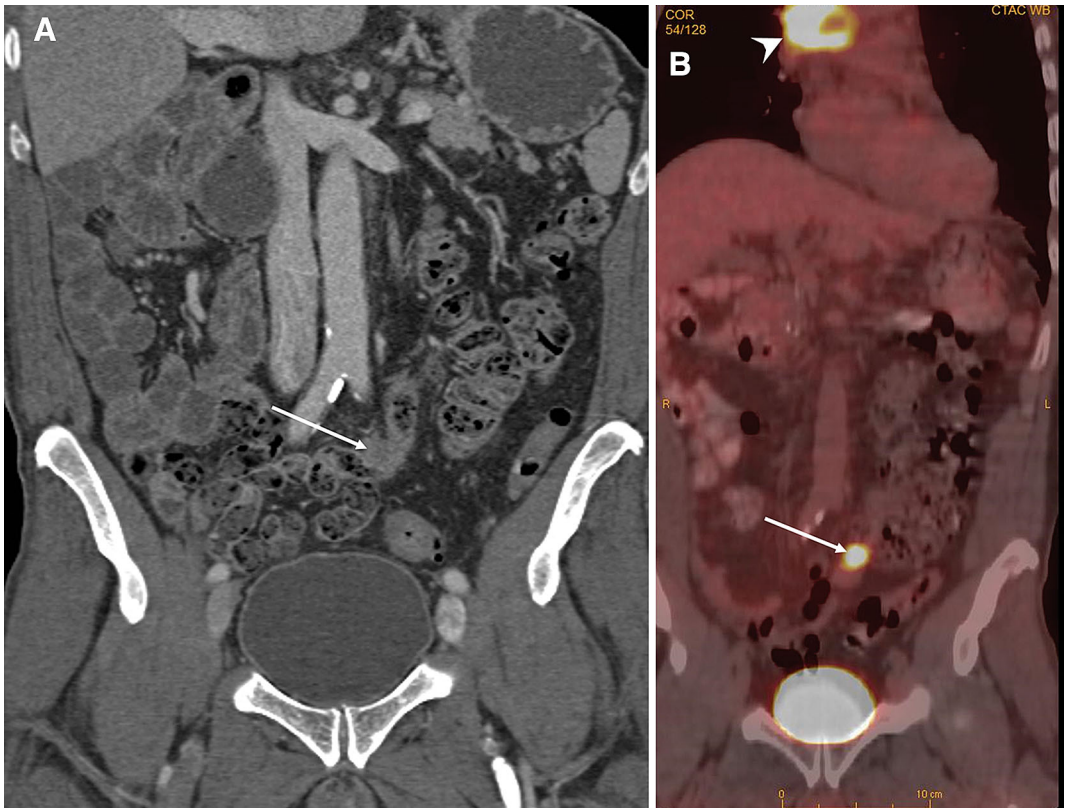


Fig. 2. Adenocarcinoma of the ileum in a 56-year-old male who presented with SVC syndrome secondary to mediastinal metastasis. Coronal image from CTE (**A**) shows focal moderately enhancing mass in the ileum (*arrow*). Coronal fusion image from PET/CT (**B**) shows intense uptake in the ileum corresponding to the small bowel mass seen on CTE (*arrow*) and bulky mediastinal adenopathy accounting for the patients presenting symptoms (*arrowhead*). Endoscopic biopsy of the ileal mass revealed poorly differentiated primary small bowel adenocarcinoma. Mediastinal biopsy was concordant with metastatic adenocarcinoma from small bowel primary.

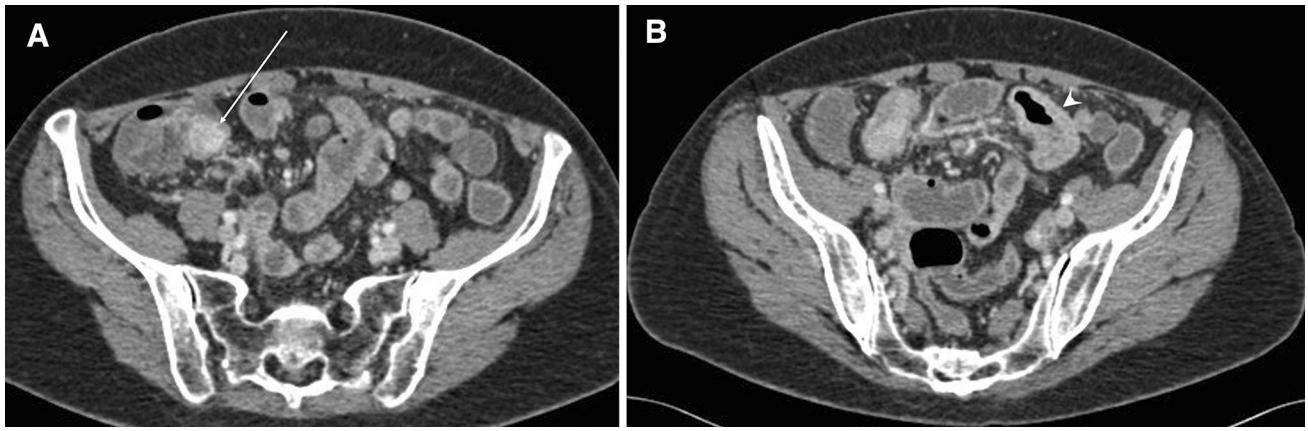


Fig. 3. Multifocal small bowel carcinoid tumor in a 59-year-old female with recurrent abdominal pain. Axial image from CTE (**A**) shows polypoid arterially enhancing mass protruding into the lumen of the ileum (*arrow*). Additional image (**B**) shows a segmental carpet lesion in an ileal segment secondary to submucosal spread of carcinoid tumor, an appearance which may mimic Crohn's disease (*arrowhead*).

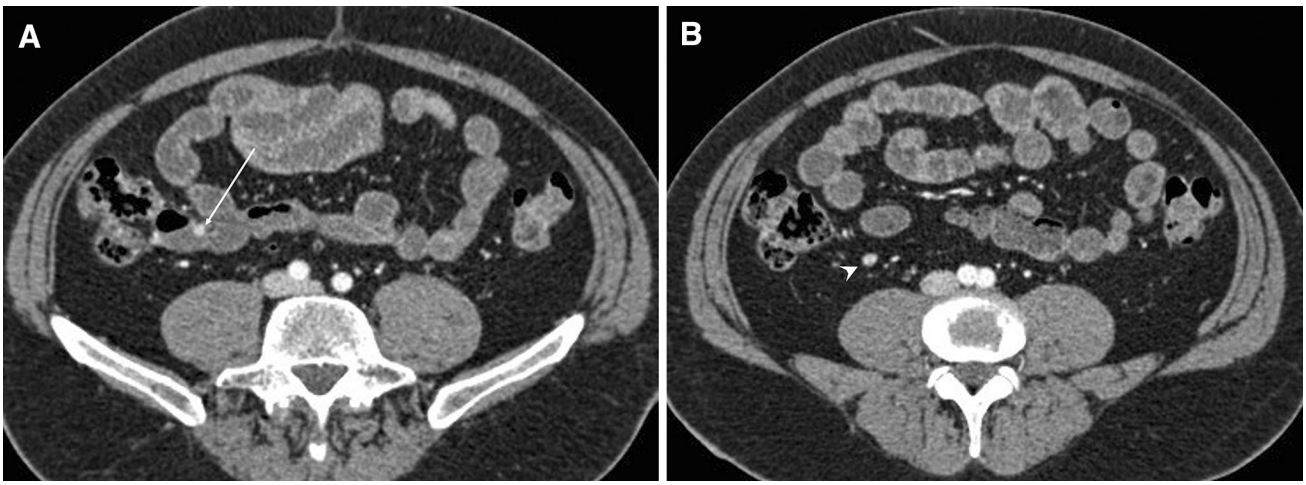


Fig. 4. Small primary small bowel carcinoid tumor in a 52-year-old female with abdominal pain and anemia. Axial images from CTE (**A**) and (**B**) show a small 5 mm hypervascular ileal polypoid mass (*arrow*). A small regional lymph node is also present in the right lower quadrant (*arrowhead* in **B**).

an independent three-dimensional workstation, where coronal and oblique coronal maximum intensity projection (MIP) images are obtained. MIP images are useful to detect enhancing lesions, although inspection of source axial images is necessary to avoid potential pitfalls. Multiphase CT enterography is especially useful to assess suspected vascular small bowel lesions in patients with obscure gastrointestinal bleeding and consists of a delayed and an enteric phase. Enteric phase is typically acquired 50 s after the injection of intravenous contrast and the delayed phase at 85 s. However, given the added radiation exposure, multiphase studies are not routinely acquired at our institution.

Malignant neoplasms of small bowel

Adenocarcinoma

Adenocarcinoma is the most common primary malignancy of the small bowel. More than half of primary small bowel adenocarcinomas arise in the duodenum followed by the jejunum and ileum [6]. A notable exception to this rule occurs in patients with Crohn's disease in which greater than 66% of cases arise from the ileum [7]. This is hypothesized to be secondary to chronic inflammation in the terminal ileum. Other known risk factors include celiac disease, Peutz–Jeghers syndrome, familial polyposis syndromes, ileostomy, and duodenal or jejunal bypass surgery [8]. The clinical presentation of small bowel adenocarcinoma is non-specific with abdominal pain being the most common presentation [6]. Obstruction due to adenocarcinoma occurs more often when the primary site of malignancy is distal small bowel rather than in the duodenum. Even less commonly, patients may present with obscure GI bleeding and anemia.

The imaging features of small bowel adenocarcinoma on MD-CTE include a short segment circumferential soft tissue mass with abrupt overhanging edges resulting in irregular luminal narrowing [9] (Fig. 1). This corresponds to the fluoroscopic finding of the "apple-core" appearance. Adenocarcinoma may also present as a polypoid mass which may act as a lead point for intussusception. A focal small bowel mass with moderate heterogeneous enhancement, ulceration, and adjacent lymphadenopathy should elevate concern for adenocarcinoma. Luminal narrowing with proximal dilatation may also be encountered. When such a lesion is discovered additional search for perivascular invasion, liver and omental metastasis are warranted as these are common sites of disease spread. Rarely, small bowel adenocarcinoma has been the cause of Krukenberg tumor of the ovary [10]. PET/CT is useful for staging as it may detect unsuspected sites of metastasis which would preclude surgical cure [11] (Fig. 2).

As in the colon, small bowel adenocarcinoma may arise from adenomatous polyps [12]. Thus, small bowel adenomas should be resected due to the risk of malignant degeneration. Small bowel adenocarcinoma typically presents after invasion of the lamina propria and bowel wall. The degree of differentiation and histologic subset has little prognostic implication. Prognosis is poor with 5-year survival rate of approximately 30% [12].

Carcinoid tumor

Carcinoid tumors are a group of neuroendocrine tumors which most commonly affect the gastrointestinal system [13]. They are the second most common type of primary small bowel malignancy. After the appendix, the small bowel is the most common location of carcinoid tumors

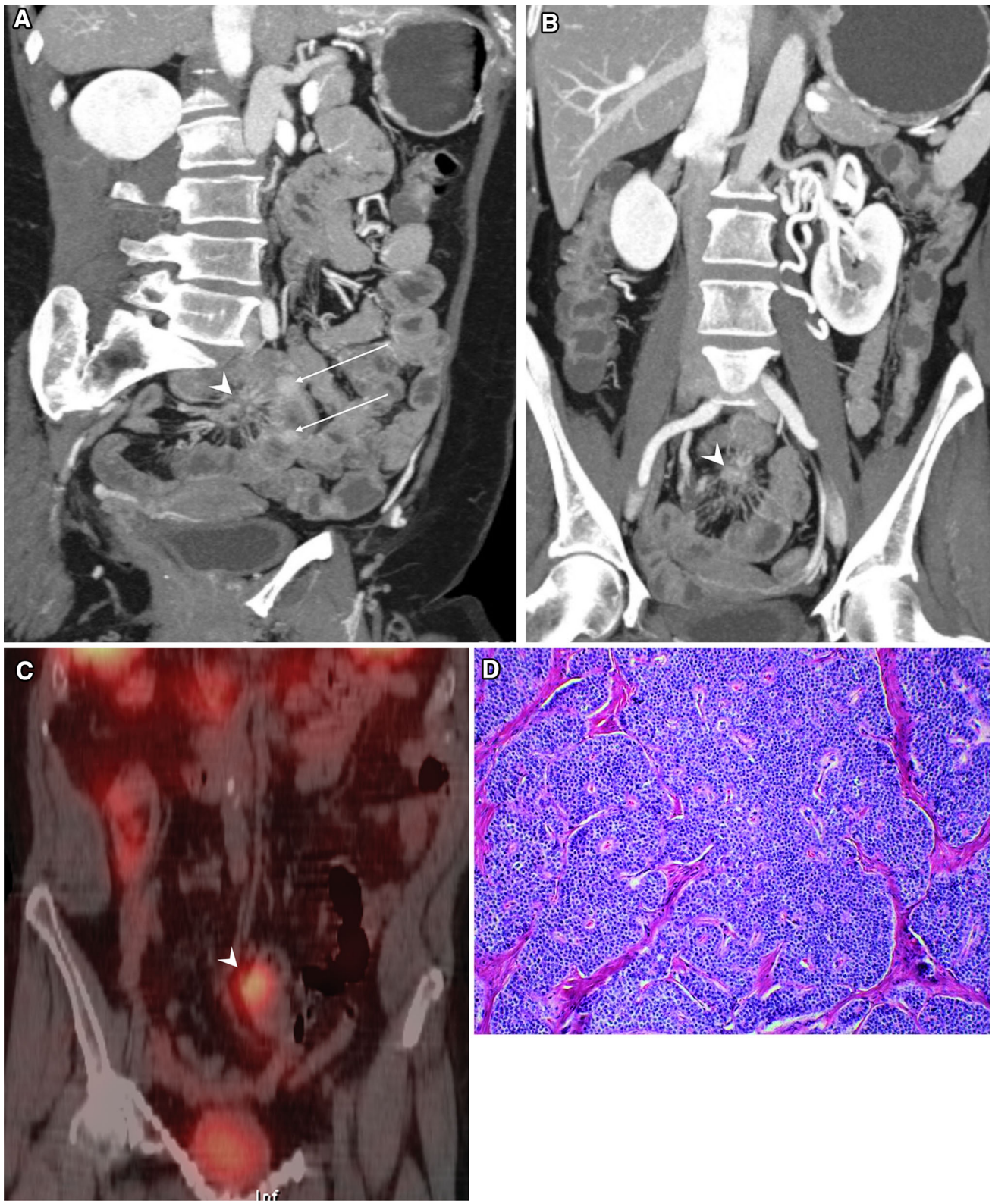


Fig. 5. Malignant carcinoid tumor in a 53-year-old female with abdominal pain and diarrhea. Oblique sagittal (**A**) and coronal MIP (**B**) images from CTE show multiple enhancing small bowel masses (*arrows*). In addition, there is a mesenteric mass with stellate appearance tethering adjacent small bowel loops (*arrowhead*). Coronal image from octreotide SPECT/CT (**C**) shows uptake at the root of the small bowel mesentery corresponding to the desmoplastic reaction on CTE. The primary small bowel lesions do not show uptake on octreotide scan due to their small size. Photomicrograph (×200 magnification; hematoxylin and eosin stain) (**D**) shows nests of monotonous cellular proliferation with round nuclei and submucosal infiltration.

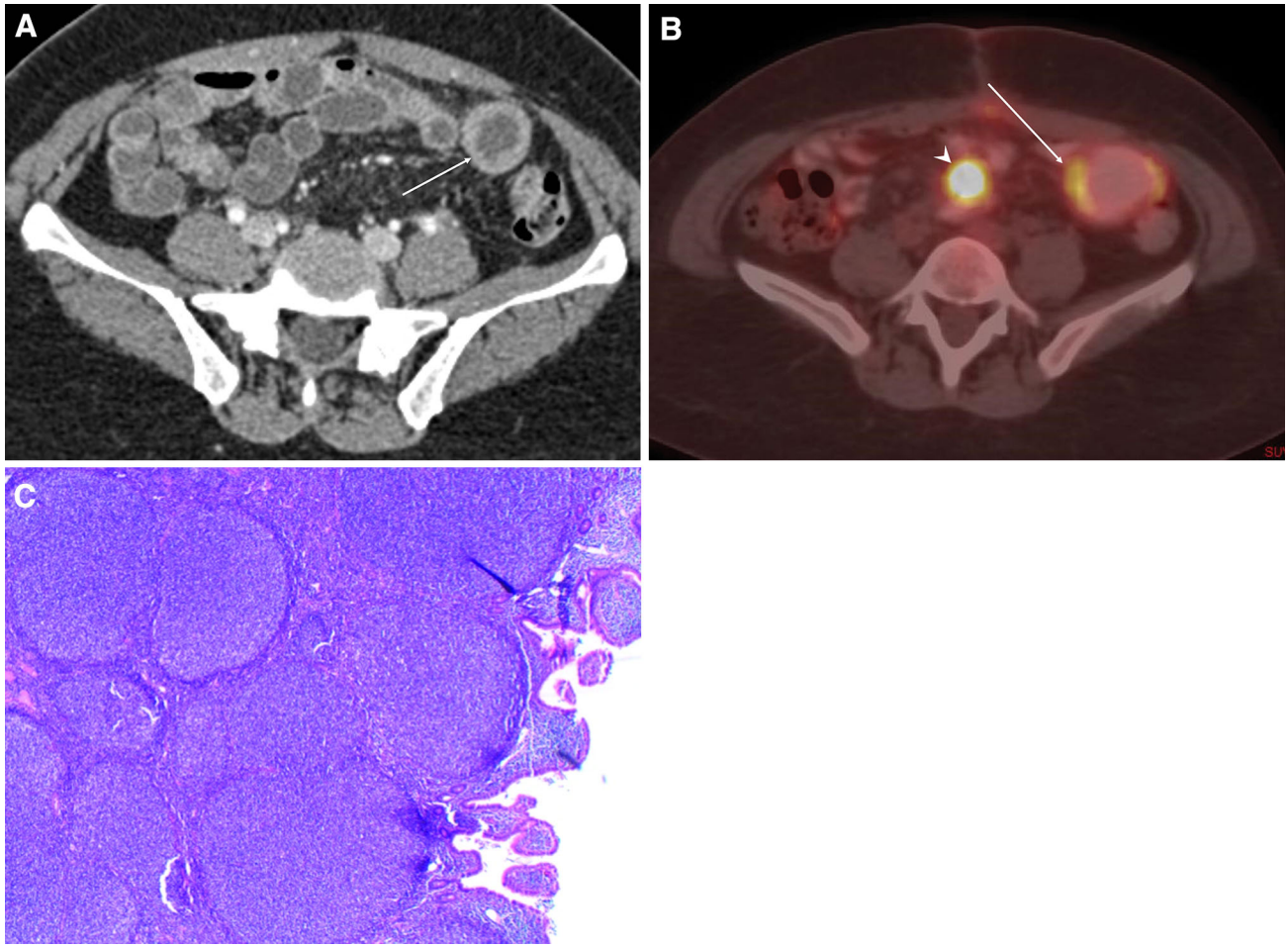


Fig. 6. Primary small bowel lymphoma in a 47-year-old female with abdominal pain and vomiting for three months. Axial image from CTE (**A**) shows an abnormal loop of small bowel with irregular nodular wall thickening and aneurysmal dilatation (*arrow*). Axial fusion image from PET/CT (**B**) shows FDG activity localized to the small bowel wall (arrows). An FDG avid lymph node is also present (*arrowhead*). Photomicrograph (×200 magnification; hematoxylin and eosin stain) (**C**) shows extensive transmural involvement of low-grade follicular lymphoma.

[14, 15]. Approximately, 90% of small bowel carcinoid tumors arise in the ileum. This tumor incites a desmoplastic response which accounts for much of its imaging appearance. The clinical presentation is non-specific with abdominal pain being the most common. Carcinoid syndrome (watery diarrhea, flushing, and endocardial fibrosis) occurs only in a minority of patients and implies disease spread to the liver. Carcinoid tumors of the duodenum are most commonly gastrinomas which are functional in one-third of cases [12].

MD-CTE shows a smoothly marginated, avidly enhancing polypoid small bowel mass which may be multiple in 15% to 35% of cases [12] (Fig. 3). Other imaging patterns on MD-CTE include small submucosal masses, large intraluminal ulcerating masses, and enhancing carpet lesions which may mimic Crohn's disease (Fig. 3b). Early arterial phase imaging and negative intraluminal contrast make MD-CTE an optimal examination to detect primary tumors. The primary tumor is typically smaller than 2 cm, and the high spatial resolution of MD-CTE is useful to detect primary lesions as small as 5 mm (Fig. 4). However, it is the metastatic disease which often dominates the imaging findings in cases of carcinoid tumor. The classic appearance is that of a calcified mesenteric mass with radiating bands emanating from it, tethering adjacent bowel loops due to desmoplastic response (Fig. 5). The incidence of metastasis increases greatly for primary tumors larger than 1 cm [14]. Liver metastases show early arterial enhancement which is optimally displayed on MD-CTE examinations. Indium-111 octreotide, a somatostatin analogue, can be used to detect the primary site of carcinoid tumor as well as distant metastasis [14] (Fig. 5e).

Carcinoid tumors are a well-differentiated neuroendocrine tumor arising from gastrointestinal submucosa [12]. They are typically poorly circumscribed with infiltration to the submucosa; however, the overlying mucosa is typically intact. They have usually deeply invaded the

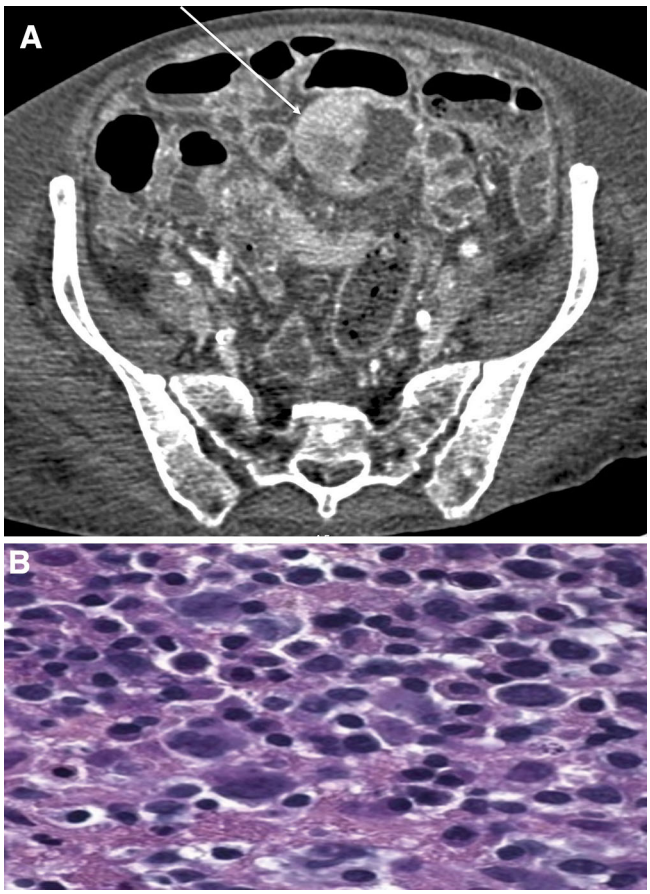


Fig. 7. Primary large B-cell lymphoma of small bowel in a 70-year-old female who presented to the emergency department with weakness, fatigue, anemia, and hypercalcemia. Axial image from CTE (**A**) shows a partially exophytic transmural hyperenhancing mass arising from the wall of the distal jejunum (*arrow*). No enlarged lymph nodes are present. Photomicrograph (×200 magnification; hematoxylin and eosin stain) (**B**) shows involvement of small bowel wall with large B-cell lymphoma.

bowel wall and mesentery by the time of discovery. These tumors are associated with significant fibrosis, especially when there is mesenteric nodal involvement. The distinction between benign and malignant carcinoid tumors is based on the presence of metastasis as morphologic features alone cannot make this distinction [12].

Lymphoma

The third most common small bowel malignancy, lymphoma, can arise primarily from small bowel or be a site of secondary involvement [16, 17]. The stomach is the most commonly affected portion of the gastrointestinal tract followed by the small bowel. Given that the distal ileum has the greatest amount of lymphoid tissue, it is no surprise that it is the most commonly affected segment of small bowel. Most cases involving the small bowel are non-Hodgkin B-cell lymphoma. T-cell lymphoma has a

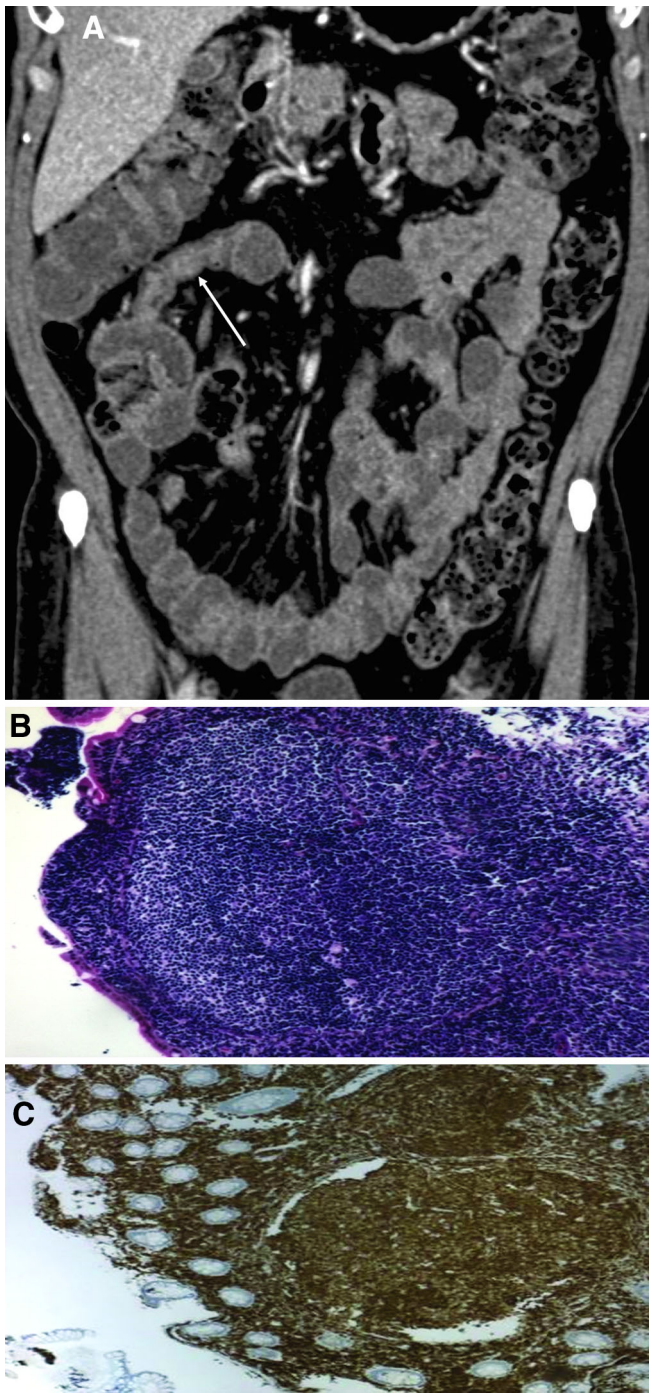


Fig. 8. Primary small bowel lymphoma in a 63-year-old male with abnormal screening colonoscopy. Coronal image from CTE (**A**) shows nodular and irregular wall thickening involving the terminal ileum mimicking inflammatory bowel disease (*arrow*). However, notice the lack of perienteric inflammatory changes and engorgement of vasa recta typically seen in active transmural Crohn's disease. Photomicrograph (×20 magnification; hematoxylin and eosin stain) (**B**) and BCL-2 immunostaining (×100 magnification) (**C**) show atypical lymphoid infiltrate with follicles and surrounding halo of histiocytes compatible with low-grade follicular lymphoma.

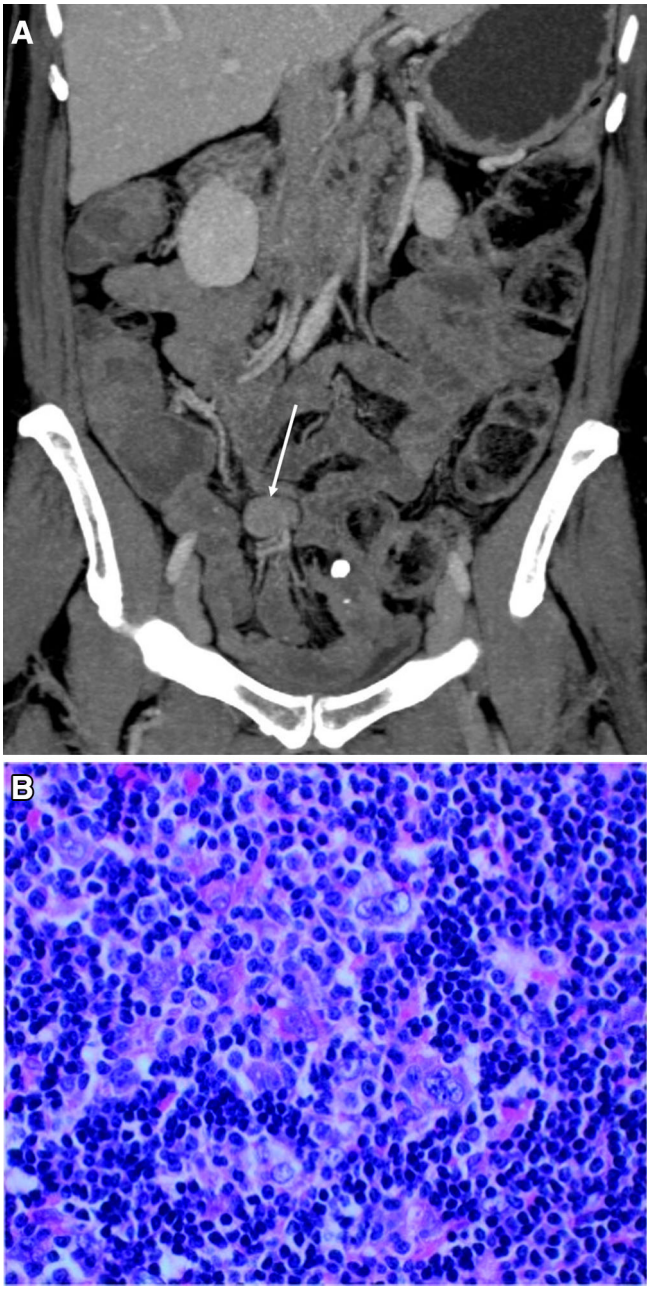


Fig. 9. Hodgkin's Lymphoma in a 65-year-old previously healthy female with abdominal pain and weight loss. Coronal MIP image from CTE (**A**) shows an enhancing mass which was believed to be arising from the small bowel in an exophytic manner (*arrow*). Photomicrograph (×200 magnification; hematoxylin and eosin stain) (**B**) shows large atypical cells with enlarged nucleoli and multinucleated "popcorn" cells consistent with nodular lymphocytic predominant Hodgkin lymphoma in an enlarged lymph node. The initial presentation of Hodgkin's lymphoma with disease isolated below the diaphragm in a middle-aged patient is quite unusual.

high association with celiac disease and occurs most commonly in the jejunum [18]. Other risk factors for development of small bowel lymphoma include acquired

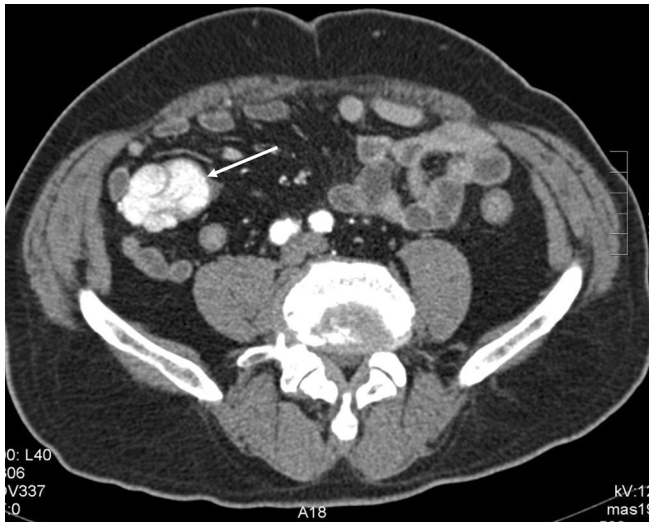
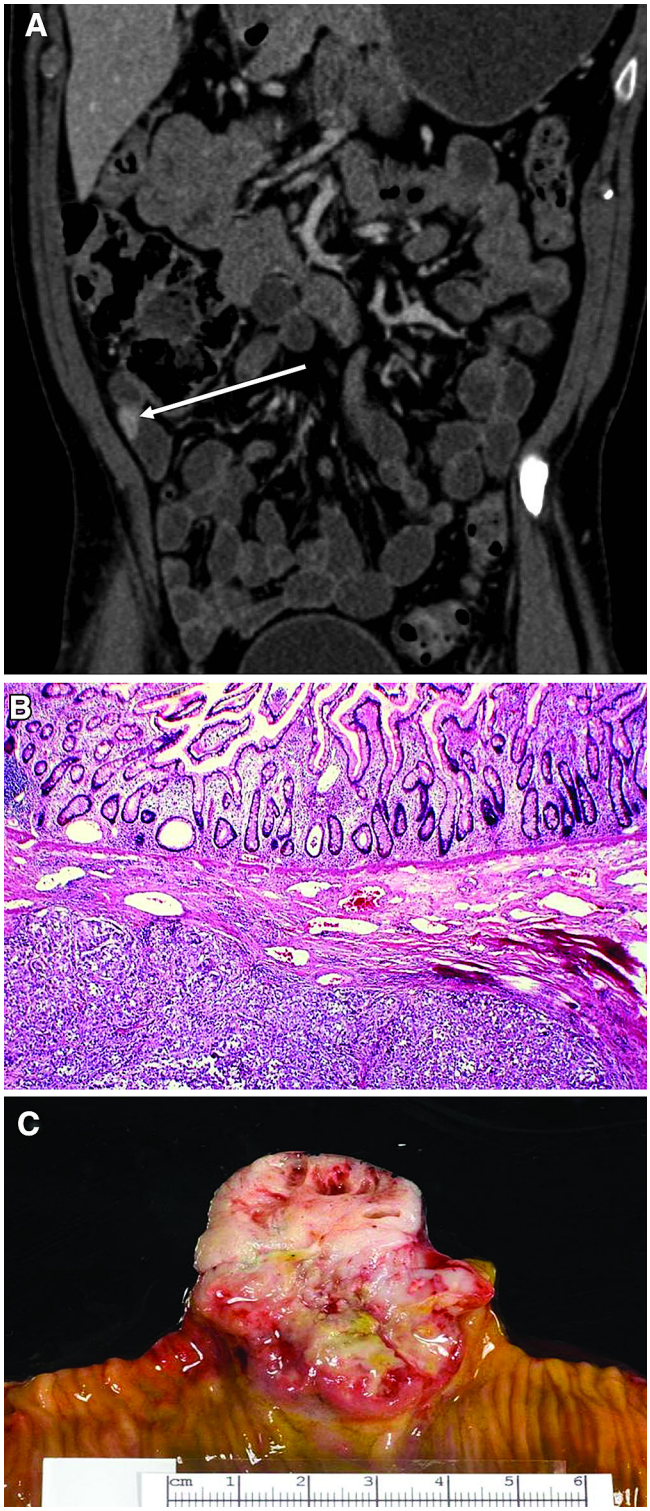


Fig. 10. Hyperenhancing ileal GIST in a 47-year-old male presenting with chronic abdominal pain and anemia. Axial image from a CTE demonstrate an arterially hyperenhancing exophytic mass (*arrow*) arising from an ileal loop in the right lower quadrant.

immunodeficiency syndrome, inflammatory bowel disease, immunosuppression after solid organ transplantation, systemic lupus erythematosus, and chemotherapy. Epstein-Barr virus has a known association with Burkitt lymphoma which commonly occurs in the ileocecal region in pediatric patients [19]. Patients were present with non-specific symptoms such as abdominal pain, nausea, vomiting, and anorexia.

MD-CTE may show an infiltrating circumferential mildly enhancing small bowel mass with or without regional lymphadenopathy. Bowel lumen may be narrowed or enlarged; if lymphomatous cells invade the muscularis propria and disrupt the autonomic nerve plexus, this results in aneurysmal bowel dilatation (Fig. 6). Other patterns of disease on MD-CTE include mucosal nodular thickening and large exocentric mass with extension into adjacent soft tissues (Fig. 7). Tumors may extend over a long segment of bowel and may show ulceration and perforation; resultant fistulous tracts and sterile abscesses have been reported [19]. Involvement of the terminal ileum may mimic Crohn's disease, although lack of active inflammatory changes may help to make the distinction (Fig. 8). Lymphoma may also present as a focal mass acting as a lead point for intussusception; however, obstruction is an uncommon feature of small bowel lymphoma. Splenomegaly and lymphadenopathy in other areas help prospectively raise the diagnosis of lymphoma.

In Western countries, B-cell lymphoma of mucosa-associated lymphoid tissue (MALT lymphoma) is the most common subtype of primary small bowel lymphoma. T-cell lymphomas are much less common and primarily occur in the setting of celiac disease [12].



◀**Fig. 11.** Small bowel Melanoma in a 26-year-old male presenting with vague abdominal symptoms. Coronal image from a CTE (**A**) demonstrates a small hyperenhancing lesion associated with an ileal loop in the right lower quadrant (*arrow*). Photomicrograph (×20 magnification; hematoxylin and eosin stain) (**B**) demonstrates a high-grade malignant neoplasm involving the submucosa and muscularis propria. Gross specimen (**C**) of small bowel with cross section of a 4.5 cm mass with central necrosis involving the submucosa and muscularis propria. This lesion was presumed to represent a primary small bowel melanoma, as no additional lesions were identified elsewhere in the body, including the skin.

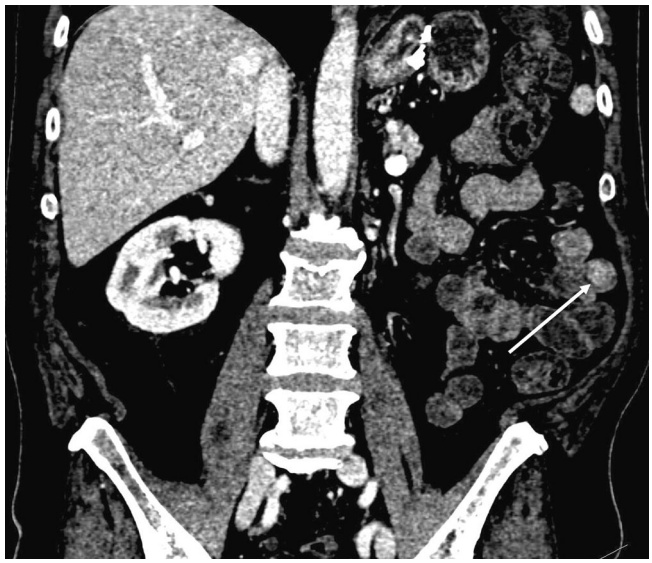
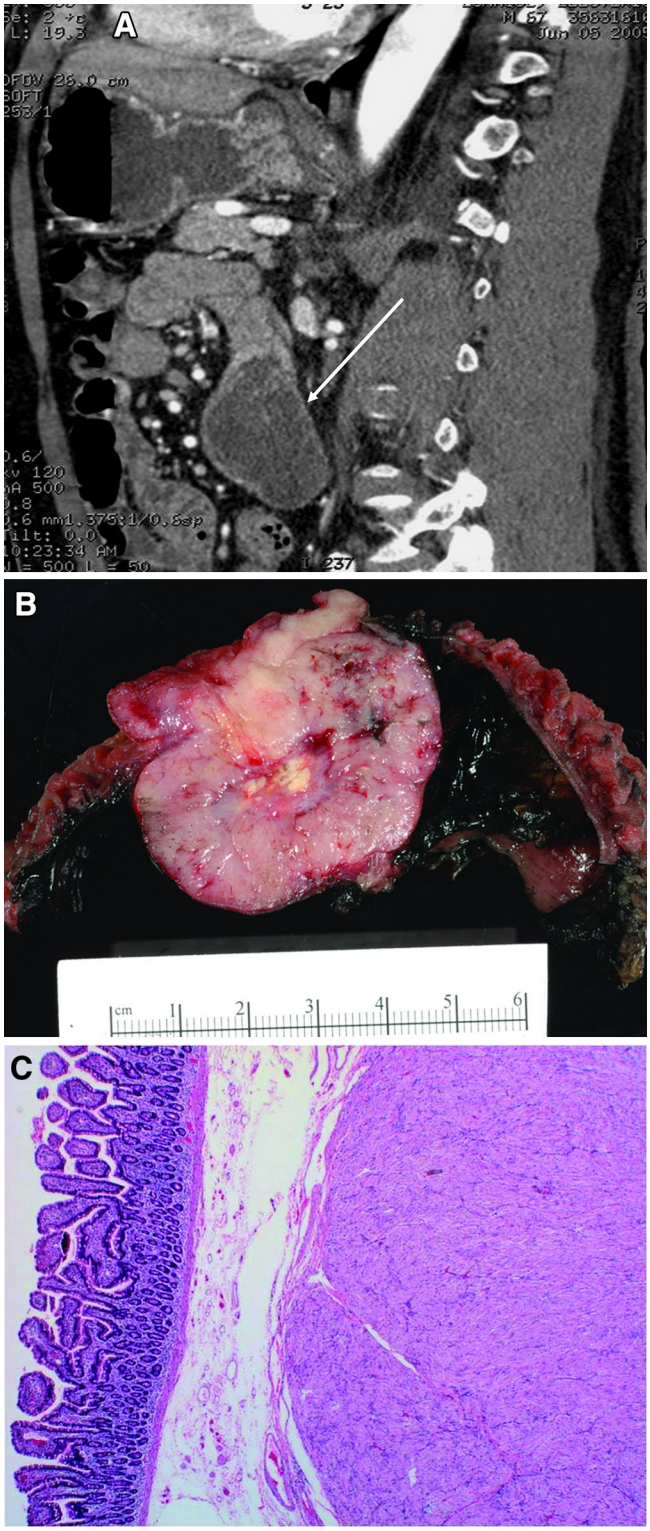


Fig. 12. Metastatic small bowel lesions in a 64-year-old male with history of renal cell carcinoma and left nephrectomy, presenting with GI bleeding. Coronal image from CTE reveals a small relatively iso-attenuating lesion within a jejunal loop in the left abdomen. Upon excision, this was found to represent a metastatic focus of the patient's primary renal cell carcinoma.

Hodgkin's lymphoma of the small bowel is rare with few case reports in the literature [20]. In fact, should pathologic evaluation yield Hodgkin's lymphoma from a small bowel biopsy specimen, a thorough search for involved lymph nodes is recommended as isolated small bowel Hodgkin's lymphoma is exceedingly rare (Fig. 9).

Gastrointestinal Stromal Tumors

Gastrointestinal stromal tumor (GIST) is the most common mesenchymal tumor of the gastrointestinal tract. Most exhibit a mutated tyrosine kinase receptor gene and in some capacity, are treated by tyrosine kinase inhibitors. A relatively recently described entity, GISTs tend to occur after the age of 40, but have also been described in the pediatric population. Approximately, 5000 cases are diagnosed annually in the United States [21], and the latest annual age-adjusted incidence is estimated at 0.78 in 100,000, according to a recently published study that analyzed data following the implementation of GIST-specific histology coding [22]. There is an increased incidence of GIST in patients with neu-



◀**Fig. 13.** Duodenal leiomyoma in a 45-year-old male with upper GI bleeding. Sagittal image from CTE (**A**) demonstrates a hypo-attenuating exophytic mass (*arrow*) arising from the duodenum. Gross pathology specimen (**B**) reveals the duodenum with a well-circumscribed exophytic mass involving the submucosa and muscularis propria. Note the ulceration on the mucosal surface responsible for active hemorrhage. Photomicrograph (×20 magnification; hematoxylin and eosin stain) (**D**) reveals a well-circumscribed lesion involving the submucosa, comprised of uniform fibrous spindle cells.

rofibromatosis type I, and along with paragangliomas and pulmonary chondromas, GISTs are part of the Carney's triad. GISTs occur most commonly in the stomach followed by small bowel, but can much less commonly present at any location throughout the gastrointestinal tract, from the esophagus to the anus. They can also rarely occur primarily within the liver, gallbladder, peritoneal cavity, or retroperitoneum [23]. Patients with GISTs most commonly present with nonspecific abdominal symptoms (pain and nausea). However, they can be found incidentally without accompanying symptoms or exhibit a wide array of clinical presentations, including bowel obstruction, intraluminal GI bleeding (occult or frank) from mucosal ulceration, or intraperitoneal bleeding secondary to rupture.

The appearance of small bowel GISTs can vary greatly on MD-CTE, depending on the size and aggressiveness of the tumor. GISTs are often described as large, hyperenhancing, and exophytic, though this is not always the case (Fig. 10). They can exhibit hypo- or iso-enhancement and can be endoluminal in location as well. They often have a heterogeneous appearance secondary to necrosis or intra-tumoral hemorrhage. They can ulcerate, cavitate, or fistulize with adjacent structures [24]. GISTs can exhibit various combinations of the described imaging findings and should be considered in the differential diagnosis when encountering small bowel lesions with any of these features. Metastatic lesions are often present in the liver or peritoneal cavity [24]. MD-CTE is useful in identifying smaller lesions that may be obscured in collapsed or unopacified bowel loops. Features such as the presence of metastases, local invasion, and larger lesions often suggest a high-grade GIST and predict poor outcome [25].

Similar to the imaging and clinical presentations, GISTs exhibit a broad spectrum of microscopic characteristics. There are three main subtypes that are widely accepted: spindle cell type, which is the most common (70%), epithelioid type, and mixed spindle cell and epithelioid types. They can range from fairly hypocellular to densely cellular with high mitotic rates [23].

Melanoma

Primary intestinal melanoma is an extremely rare malignancy to affect the small bowel. Far and away, most melanomas occurring in the gastrointestinal tract are metastatic lesions from a cutaneous primary, and the small bowel is the most common location of gastrointestinal tract metastasis [26]. It has been postulated that primary intestinal melanoma may arise from Schwan-

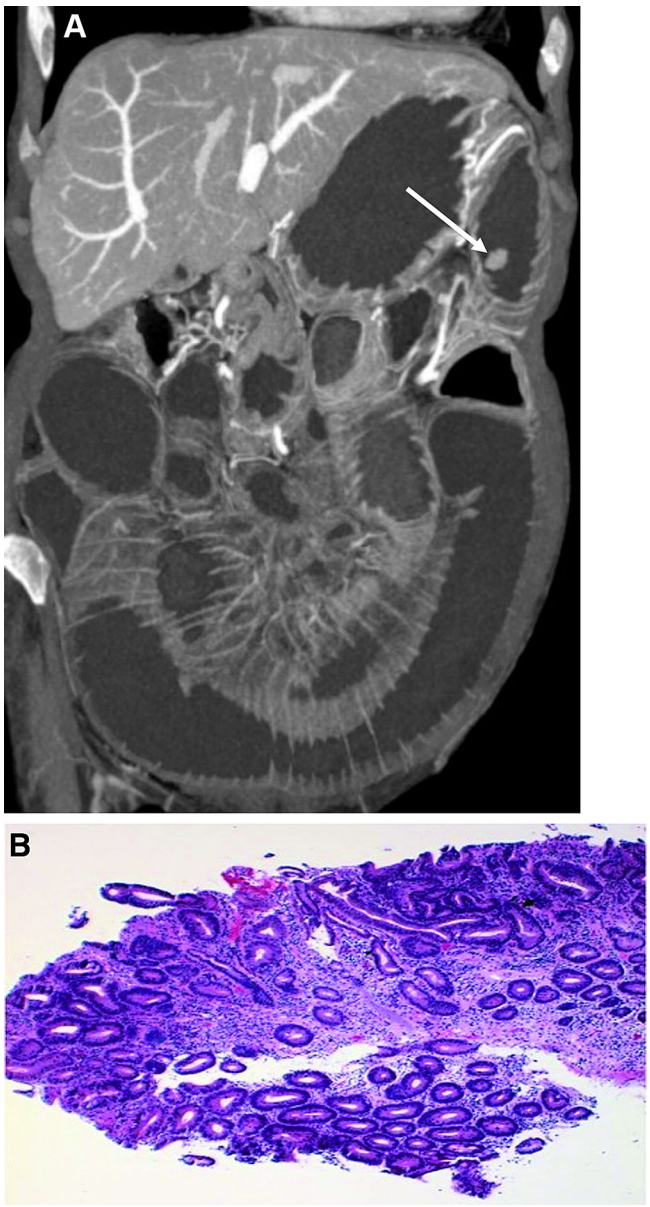


Fig. 14. Tubular adenoma in a 45-year-old male with scleroderma who presented with a GI bleed. Coronal image from CTE (**A**) demonstrate an enhancing polypoid lesion within the jejunum (*arrow*). Small bowel loops are dilated with hidebound appearance. Photomicrograph (×20 magnification; hematoxylin and eosin stain) (**B**) confirms tubular adenoma, with more crowded, disorganized glands than the normal underlying mucosa and the cells lining the glands of the adenoma having hyperchromatic nuclei.

nian neuroblast cells of the intestine's autonomic innervations [27]; others believe that they arise from melanocytes which can be rarely found in non-cutaneous sites such as the gastrointestinal tract [28]. Some authors believe that primary small bowel melanoma does not exist as a separate entity and that such lesions are metastases from either unidentifiable cutaneous lesions or cutaneous lesions that have regressed [27]. Sachs and colleagues proposed criteria for diagnosing primary small bowel melanoma: (1) biopsy-proven melanoma from the small intestine at a single focus, (2) no evidence of disease in any other organ, including the skin (no evidence of regressed melanoma lesion), eye, and lymph nodes outside the regional drainage of the lesion in question, and (3) disease-free survival of at least 12 months after diagnosis [28].

Patients typically present with similar non-specific signs and symptoms seen with any small bowel tumor, including vague abdominal pain, nausea, vomiting, GI bleeding, palpable mass, bowel obstruction, or intussusception. Similarly, findings on imaging are non-specific. It can present as a solitary enhancing small bowel lesion (Fig. 11). MD-CTE can also aid in defining the mural and transmural extent of disease, which is important for surgical planning. Given that melanoma lesions typically demonstrate increased FDG uptake, FDG-PET CT is also helpful in identifying metastases to other regions of the small bowel or regional lymph nodes [26]. The use of FDG-PET can also reveal an occult cutaneous or extraintestinal lesion that would likely be regarded as the primary site of melanoma.

Histopathologic examination demonstrates a melanocytic precursor lesion with melanoblastic cells or melanosis in the intestinal mucosa. The lesion typically demonstrates positivity to Melan A staining [27].

Metastases

Metastatic lesions are more common in the small bowel than are primary tumors. The incidence and prevalence vary with various malignancies. It has been reported that up to 5% to 10% of patients with certain malignancies such as breast cancer or melanoma will develop intestinal metastases. They are characterized by means of spread, including intraperitoneal seeding, hematogenous spread, or direct invasion from adjacent organs or colon [29].

Metastatic lesions often exhibit similar findings and characteristics on imaging, particularly MD-CTE, as the primary tumor. Metastases can be solitary or multiple. Intraperitoneal seeding, frequently occurring secondary to primary mucinous tumors of the ovary, appendix, or colon, usually appears on CT as multiple small, nodular lesions along the small bowel serosa, mesentery and omentum [8]. Common malignancies that spread hematogenously to the small bowel include bronchogenic carcinoma, breast carcinoma, malignant melanoma, and renal cell carcinoma. MD-CTE may be necessary to identify small lesions not easily identifiable on conventional CT (Fig. 12). Local invasion from primary pancreatic, biliary, or colonic malignancies is also seen involving the small bowel [8].

Benign small bowel neoplasms

Leiomyoma

Leiomyomas comprise the majority of benign tumors of the small bowel. While they are the most common mesenchymal tumors in the esophagus, they are less likely to occur in the small bowel. The reported incidence is approximately between 20% and 45%. These lesions may appear anywhere throughout the ileum or jejunum, demonstrating a slight predilection for the jejunum [30]. Leiomyomas are typically an incidental finding as patients are asymptomatic, though given that they can be highly vascular and ulcerate, bleeding is the most frequently reported symptom. Obstruction from intussusception or intraluminal growth is the second most common presentation among symptomatic patients [30, 31].

The imaging features of leiomyoma are not specific or distinguishable from those of gastrointestinal stromal tumor. They typically present as a solitary lesion and can originate from subserosal, intramural, or submucosal tissues. Within the small bowel, the majority appears as polypoid or intramural masses [32]. MD-CTE findings of a homogenous, round, smoothly marginated, contrast enhancing mass in the absence of metastases or mesenteric changes, are compatible with benign leiomyomas (Fig. 13).

Pathologically, they consist of well-differentiated smooth muscle, appearing as perpendicular fascicles of spindle cells with no evidence of mitosis, which is a critical finding in ruling out a leiomyosarcoma [12, 31].

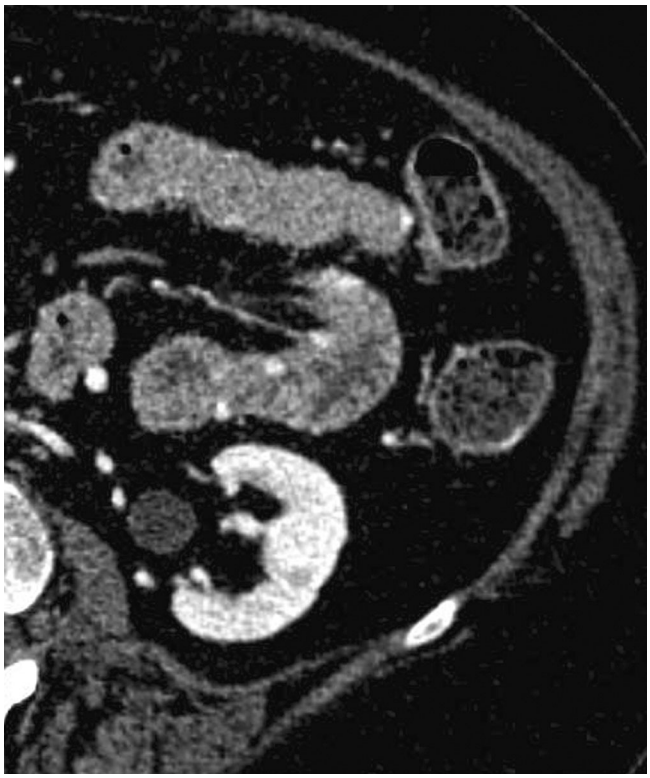


Fig. 15. Multifocal jejunal angiodysplasia in a 65-year-old male presenting with OGIB. Multiple punctate areas of avid enhancement are seen on axial CTE image throughout a segment of jejunum characteristic of angiodysplasia.

Adenoma

Adenomas are the second most common benign tumor of the small bowel with a reported incidence of 15% to 20%. Patients with familial adenomatous polyposis syndrome have an increased risk of small bowel adenoma which may be multiple. They are typically classified as tubular, tubulovillous, and villous. Villous adenomas comprise approximately 40% of small bowel adenomas, usually appear as sessile and lobulated, have a predilection for the duodenum, and carry the highest risk of malignant transformation [31]. Patients with adenomas are usually asymptomatic, only occasionally presenting with GI bleeding or obstruction secondary to intussusception. Patients with small bowel adenomas are at increased risk for colorectal neoplasms warranting dedicated screening. MD-CTE can identify these sessile and pedunculated masses, but can also identify small mural-based nodules in the mucosa, with most adenomas demonstrating homogeneous enhancement (Fig. 14).

On pathologic evaluation, adenomatous epithelium with pseudostratified enlarged hyperchromatic nuclei is present; however, there must be no evidence of high-grade dysplasia or lamina propria invasion, otherwise malignant transformation is suspected [12].

Lipoma

Lipomas represent the third most common benign small bowel tumor with a reported incidence of 8% to 20%. The small bowel is the second most common location of gastrointestinal lipomas, with colon being the most common. Most small bowel lipomas are found in the ileum, are solitary, and originate in the submucosal layer with intraluminal projection as outward projection is prevented by the muscularis propria. When lipomas are multiple and subserosal, there is usually an association with Crohn's disease [33]. As with adenomas, lipomas are usually an incidental finding as patients are asymptomatic, though they may present with intermittent obstruction from intussusception.

MD-CTE is often diagnostic as the attenuation of the tumor will measure equal to fat, typically −80 to −120 Hounsfield units [33, 34]. If a solid, non-fat component is identified, a liposarcoma should be considered; however, ulceration overlying the lipoma can result in non-fat density or stranding in the lesion. Lipomas can show ulceration especially when larger than 2 cm. Pathologic features generally include mature white adipose tissue without atypia or mitotic figures.

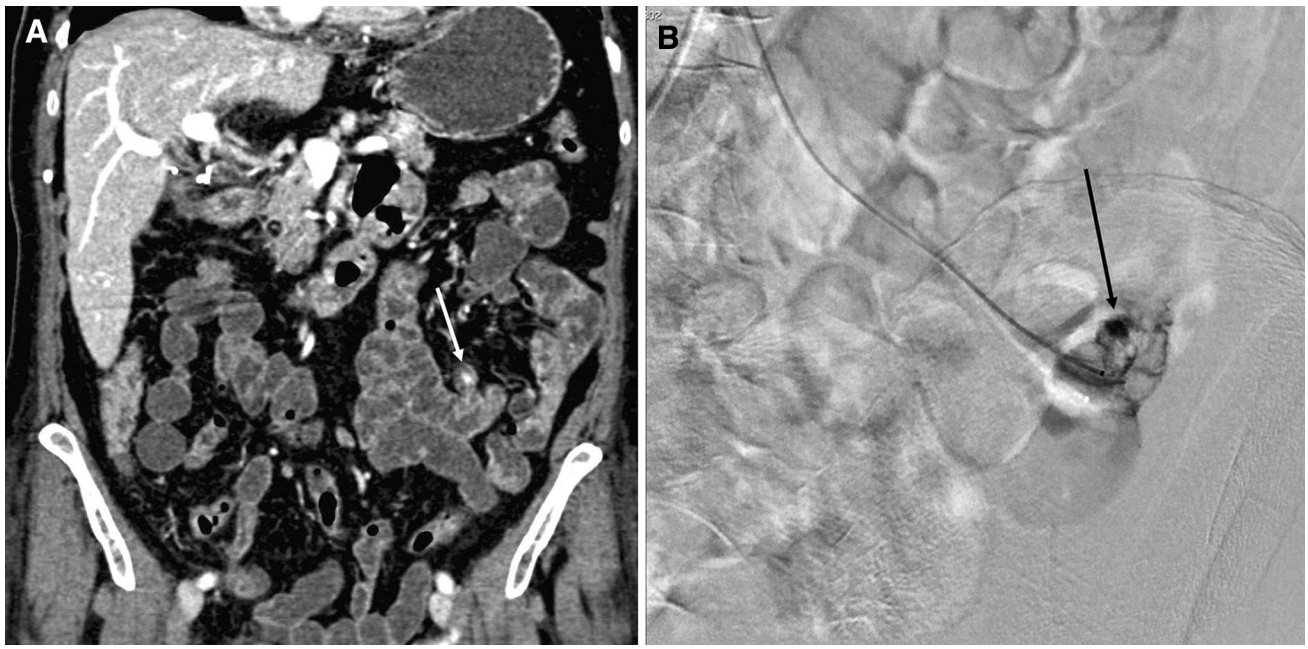


Fig. 16. Multifocal angiodysplasia in a 55-year-old female with OGIB. Coronal image from CTE (**A**) shows a focal small tuft-like avidly enhancing lesion (*arrow*). Multiple additional punctate foci of submucosal enhancement ranging in size between 2 and 10 mm are also present (not shown). Subsequent superior mesenteric artery selective angiogram (**B**) obtained at the time of sclerosant therapy shows active intraluminal contrast accumulation (*arrow*) corresponding to the area of enhancement on CTE.

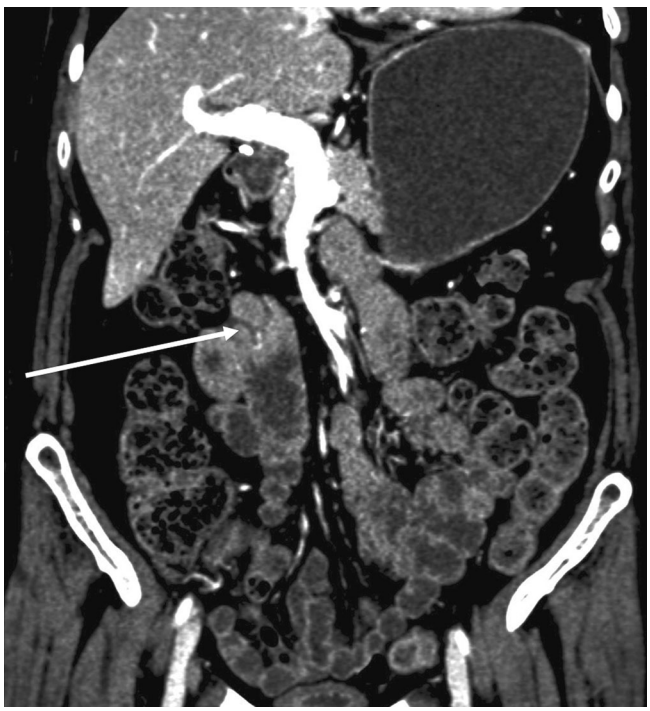


Fig. 17. Jejunojejunal intussusception caused by a small focal angiodysplasia as a lead point in a 69-year-old male with abdominal pain. Coronal image from CTE reveals a small angiodysplastic enhancing focus, at the tip of intussusceptum lead point (*arrow*). Capsule endoscopy confirmed a single small angiodysplastic vessel without bleeding in the region of distal jejunum.

Vascular Lesions of Small Bowel:

Vascular lesions are the most common lesions responsible for small bowel GI bleeding [4]. Occult gastrointestinal bleed (OGIB) accounts for 10% of all GI bleeding [35]. Approximately, 5% of OGIB originate in the small bowel for which vascular lesions account for 40% [36]. Many of the vascular lesions detected by endoscopy or multiphase MD-CTE are incidental and may not be associated with bleeding [37]. Fewer than 10% of angiodysplastic lesions are associated with bleeding [38]. Recurrent bleeding after endoscopic treatment is not uncommon due to the presence of multiple lesions in up to 50% of cases [39].

It was previously proposed that MD-CTE could not detect superficial lesions, but only angiodysplasias and AVMs could be diagnosed, whereas CE can detect superficial lesions [40, 41]. However, a more recent study demonstrated that MD-CTE has a sensitivity of 88% for the detection of vascular lesions compared with 36% for CE [42]. Even though capsule endoscopy is the initial study of choice in many institutions, MD-CTE is a reasonable alternative for first evaluation in some cases.

Small bowel vascular lesions associated with occult gastrointestinal bleeding can be classified on the basis of multiphase MD-CTE findings distinguishing each type of lesion by a common morphology and enhancement pattern [37]. Vascular lesions of the small bowel can be

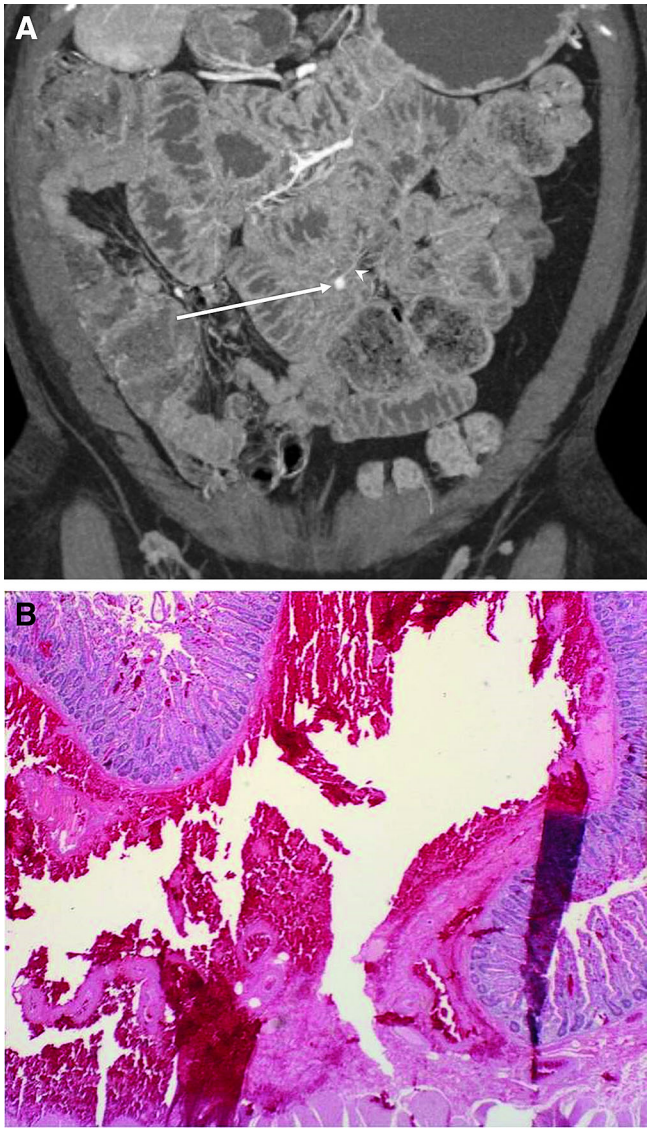


Fig. 18. Jejunal AVM in a 47-year-old patient with occult GI bleed. Coronal MIP image from CTE (**A**) reveals an early arterially enhancing vascular tuft (*arrow*) and an early draining vein (*arrowhead*) during arterial phase characteristic of a jejunal AVM. Video capsule endoscopy was positive suggesting vascular lesion with active bleeding, and photomicrograph (×20 magnification; hematoxylin and eosin stain) revealed a submucosal AVM (**B**).

classified into angioectasias/angiodysplasias, arterial, and venous lesions.

Angiodysplasia

Angiodysplasia is also commonly referred to as angioectasias. Angiodysplasia, the most common cause of small bowel bleeding, is characterized by abnormally dilated, tortuous, thin-walled vessels, involving small capillaries, veins, and arteries [43]. These thin tortuous vessels lacking an internal elastic layer most commonly

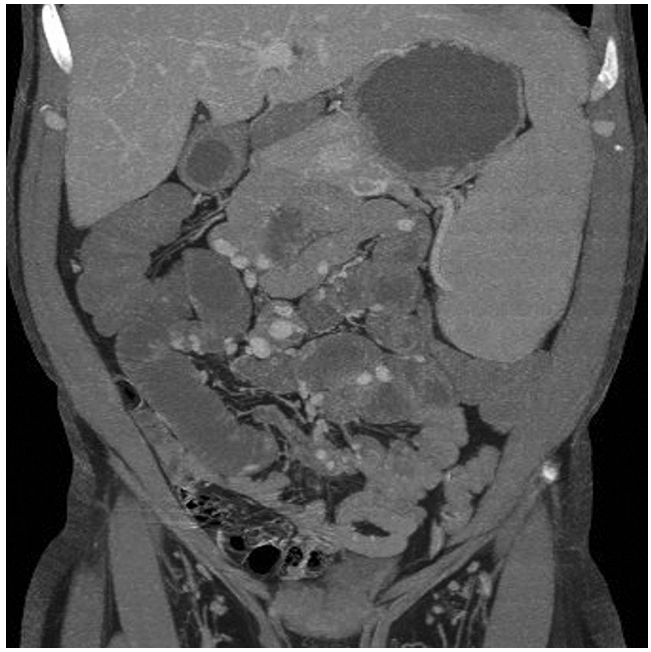


Fig. 19. Small bowel varices in a 57-year-old male with history of sarcoidosis who presents with unintentional weight loss, chills, and lymphadenopathy. Coronal MIP image from CTE demonstrates multiple enhancing nodular varices throughout the small bowel.

cause intermittent bleeding [36]. Boley suggested that angioectasias are the result of a degenerative process associated with aging, caused by chronic intermittent low-grade obstruction to venules, capillaries, and arteries of the mucosal vascular unit. Ultimately, pre-capillary sphincters lose their competency, producing small arteriovenous communications [44]. Angiodysplasias are visualized within the mucosal/submucosal layers of the gut, are lined by endothelium with little or no smooth muscle, and lack inflammatory or fibrotic changes. [45, 46] Angiodysplasia appears endoscopically as an arborizing patchy erythematous 2-10 mm area that bleeds easily with contact. The incidence of angiodysplasia peaks in the 8th to 9th decades of life and can be multifocal in 40% to 75% of cases. They are associated with end-stage renal disease and aortic stenosis and can be found throughout the small bowel. Angiodysplasia is reported 2.6% to 6.2% of patients incidentally, but in 50% of OGIB patients undergoing capsule endoscopy [37].

Angiodysplasia is characterized by a tuft-like area of enhancement best seen on enteric phase of intravenous contrast, fading on delayed imaging [29]. On MD-CTE angiodysplasia consists of focal punctate or discoid areas of enhancement less than 5 mm in size or bulbous swelling of the intramural vessels (Fig. 15). Uncommonly, angiodysplasia manifests as a focal area of enhancement with early draining vein during arterial phase as opposed to a prominent artery with early draining vein as in

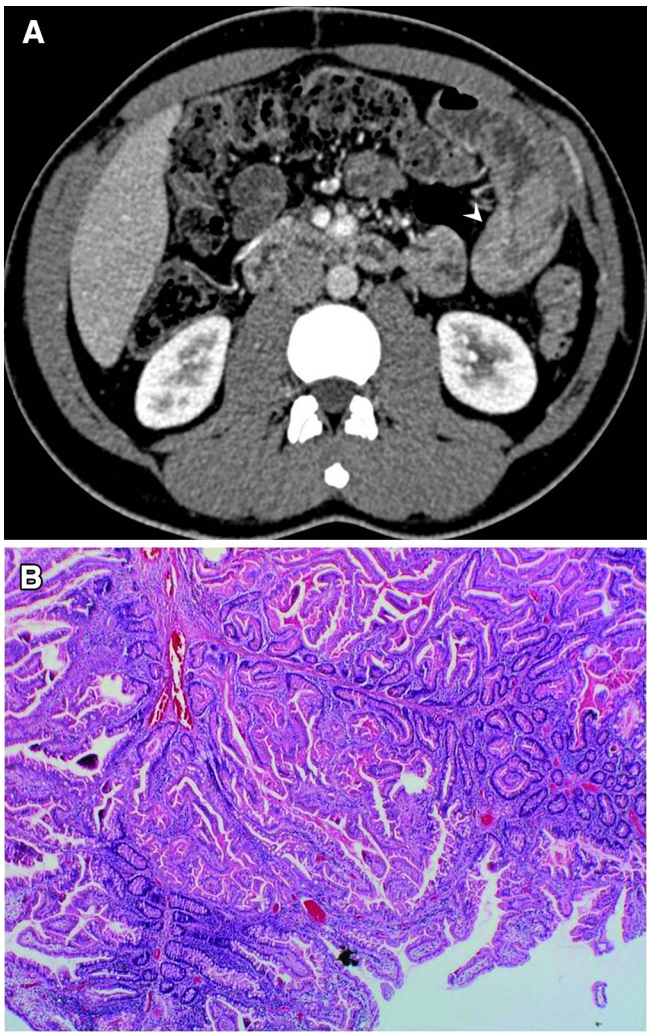


Fig. 20. Small bowel polyps in a 27-year-old female with Peutz–Jeghers syndrome presenting with abdominal pain. Axial image from CTE (**A**) demonstrates a jejunojejunal intussusception with a polypoid lesion as the leading point (*arrowhead*). Multiple additional enhancing small bowel polypoid masses are also present (not shown). Photomicrograph (×20 magnification; hematoxylin and eosin stain) (**B**) demonstrates crypts and villi of varying lengths divided by arborizing muscle bundles compatible with a hamartomatous polyp.

AVMs [47]. When multiple small bowel AVMs are encountered, the largest lesion is typically considered the offending lesion responsible for gastrointestinal bleeding (Fig. 16). Although small, foci of angiodysplasia may serve as a lead point causing intussusception, a clinically relevant abnormality in the absence of gastrointestinal bleeding (Fig. 17).

Arterial lesions

Arterial lesions include arteriovenous fistulas/malformations (AVF/AVM) and less commonly Dieulafoy lesions. All arterial lesions enhance most brightly during arterial phase and fade or become invisible on enteric and delayed phases. Because these lesions are exposed to high arterial pressure, there is a significant potential for life-threatening bleeding. [37] AVMs are most common in the colon but can rarely be seen in the jejunum (10.5%), ileum (8.5%), and duodenum (2.3%) [4]. AVMs are abnormal communications between artery and vein [37]. Therefore, MD-CTE appearance shows an enlarged feeding artery that is accompanied by an early draining vein (Fig. 18). These are thought to be congenital in the colon and spinal cord but could actually evolve from angioectasias in small bowel [48].

Dieulafoy lesions are essentially normal vessels in an abnormal location within the submucosa. They are non-tapering submucosal arteries which can cause life-threatening bleeding so are unlikely to present with OGIB [49]. They can also be referred to as "caliber persistent arteries." The presence of a draining vein distinguishes AVM from Dieulafoy's lesion [37]. The often life-threatening Dieulafoy's lesion is uncommon (2% to 3.5%) in the small bowel and common in the stomach [50, 51]. A more recent study showed 16% occurring in the small bowel, most in the duodenum [37].

Venous lesions

The most common venous lesions of small bowel are venous angiomas and small bowel varices, with the former being exceedingly rare (as in Klippel–Trenaunay syndrome). Venous angiomas are pathologically hamartomas lined by endothelial cells filled with blood [52]. Venous lesions do not enhance on arterial phase, and progressively enhance during enteric and delayed phases in multiphasic MD-CTE. Examining arterial phases does allow for the detection of phleboliths, which are commonly present in venous lesions of the small bowel. Small venous angiomas look like hepatic hemangiomas with slow progressive globular enhancement [37].

Unlike venous angiomas, small bowel varices are an acquired pathology. In patients with prior history of abdominal surgeries small bowel varices may form within adhesion bands [53]. This warrants higher index of suspicion for varices not only in patients with known portal hypertension, but also in any post-abdominal surgery patient. If multiphasic CT enterography is performed, small bowel varices may be visible on the enteric phase and become more intense on the delayed phase with progressive filling of the mesenteric-systemic venous collateral veins (Fig. 19). Small bowel varices are more commonly seen in patients with portal hypertension than in those with normal portal venous pressure. In patients with reported small bowel varices, 17% are seen in jejunal or ileal, and 26% are seen in peristomal locations [54]. A focused study in patients with portal hypertension showed associated varices, in

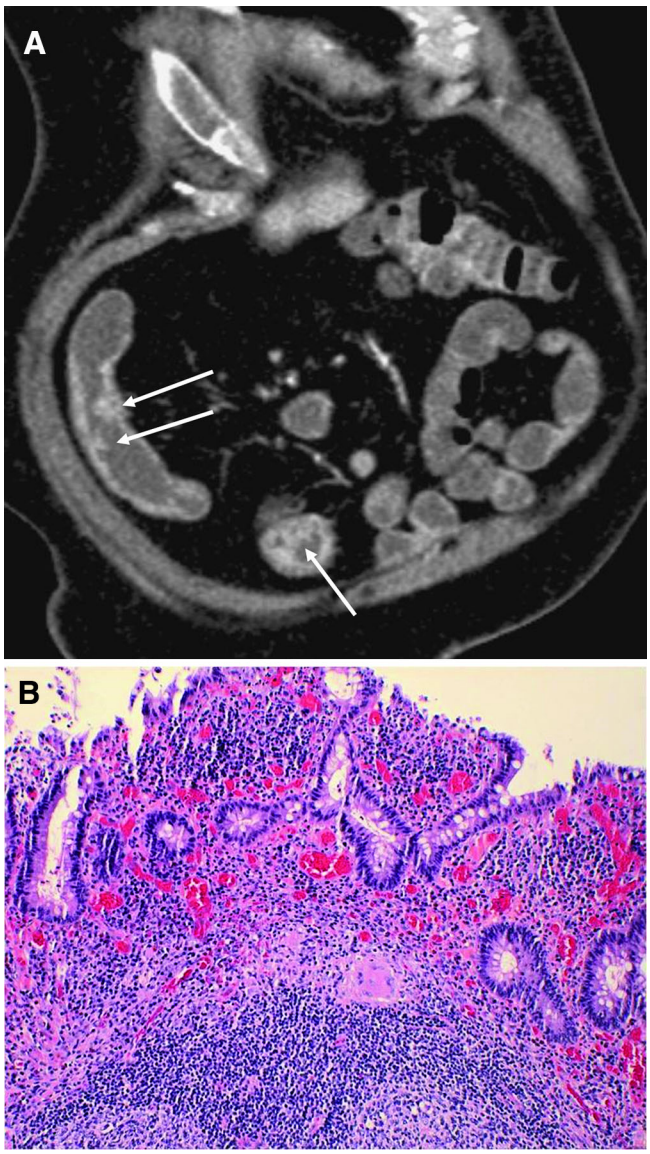


Fig. 21. Inflammatory polyps in a 60-year-old male with a history of Crohn's disease. Coronal image from CTE (**A**) demonstrates multiple abnormal focal hyperenhancing lesions (*arrows*) within the distal ileum with background bowel mucosal enhancement suggestive of active disease. Photomicrograph (×20 magnification; hematoxylin and eosin stain) (**B**) reveals crypt architectural distortion, full thickness inflammatory infiltrate with prominent lymphoid aggregates, small granuloma, and cryptitis seen within an inflammatory polyp.

duodenal, jejunal, ileal, and peristomal locations in 32.9%, 4%, 1.2% and 5.8% of patients, respectively [55]. In patients with portal hypertension who underwent CE and/or ileocolonoscopy, small bowel varices were observed in 8% to 21% [56, 57].

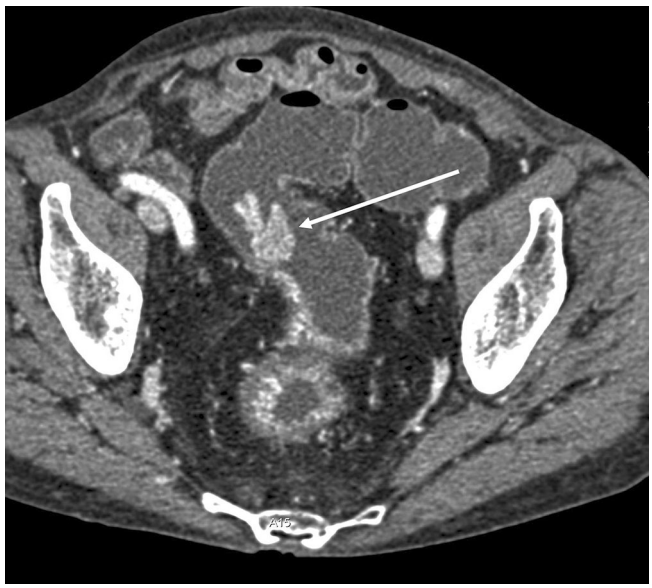


Fig. 22. Inflammatory polyp in a 54-year-old patient with known history of Crohn's Disease. Axial image CTE image demonstrates a polypoid, pedunculated hyperenhancing mass arising within the rectosigmoid junction (*arrow*). Note the hyperenhancing background mucosa, representing active disease.

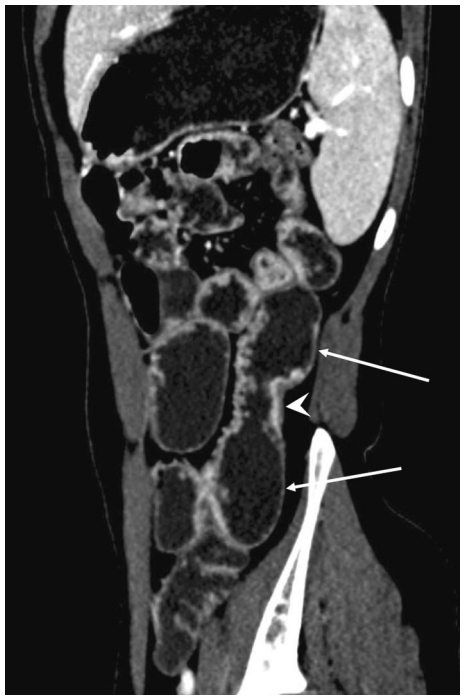


Fig. 23. Pseudopolyps in a 27-year-old male with a history of Crohn's disease. Sagittal oblique image from CTE demonstrates focally thickened and irregular jejunal folds (*arrowhead*), known as pseudopolyps of Crohn's disease. Additionally, pseudosacculation of the proximal and distal segments (*arrows*) is also shown.

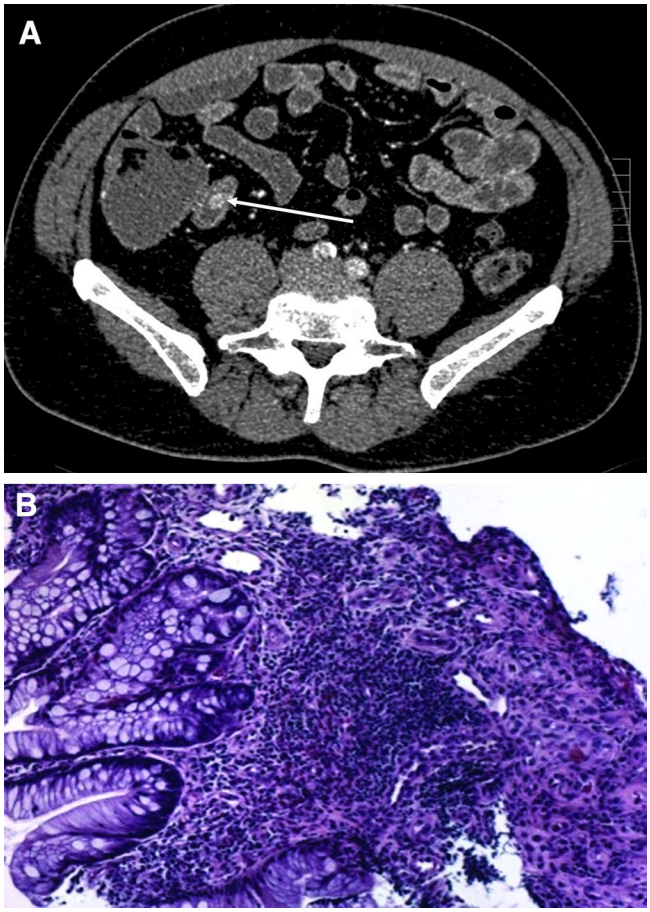


Fig. 24. Post-inflammatory polyp in a 58-year-old male with a known history of Crohn's disease, currently without symptoms. Axial image from CTE (**A**) demonstrates a 1.1 cm focal enhancing nodule (*arrow*) within the terminal ileum without radiologic evidence to suggest active disease. Photomicrograph (×100 magnification; hematoxylin and eosin stain) (**B**) reveals chronic active enteritis with focal granulation tissue formation, consistent with post-inflammatory polyp.

Other focal lesions of small bowel

Peutz–Jeghers syndrome

Peutz–Jeghers syndrome (PJS) is an autosomal dominant condition, linked to a mutation of the STK11 gene, and is characterized by gastrointestinal hamartomatous polyps and mucocutaneous pigmentation, particularly the vermilion border of the lips. PJS is a rare entity with a reported incidence between 1 in 50,000 to 1 in 200,000 live births [58]. Hamartomatous polyps may be found throughout the gastrointestinal tract but are most commonly present in the small bowel (60% to 90%) and colon (50% to 64%). The most frequent locations, in order of prevalence, are the jejunum, ileum, duodenum, colon, and stomach. The polyps may result in gastrointestinal hemorrhage as well as abdominal pain secondary to intussusception, obstruction, or infarction [59]. Additionally, extraintestinal polyps may be found in the gallbladder, bronchi, bladder, and ureter. [60] The mucocutaneous pigmented lesions are present in approximately 95% of individuals with PJS. These lesions most commonly arise in infancy and typically occur in the regions of the mouth, fingers and toes, nostrils, and volar aspects of the hands and feet and may regress after puberty; however, the lesions tend to persist in the buccal mucosa. PJS may be diagnosed clinically when any number of Peutz–Jeghers (PJ) hamartomas are present plus at least two of the following three signs: family history of PJS, polyposis of the small bowel, or mucocutaneous lentiginosis [58].

Imaging surveillance in PJS is recommended to observe for potential malignant transformation of these polyps and mechanical complications [61]. There is, however, no consensus as to the frequency of surveillance imaging, the age of commencement of imaging, nor which organs should be monitored. MD-CTE will typically demonstrate numerous polyps within the small bowel and colon and occasionally the stomach (Fig. 20). While the imaging appearance of the individual hamartomatous polyps is often non-specific, the multiplicity and location of the polyps may clue the radiologist into the diagnosis. The jejunum and ileum are the most frequently involved and the polyps tend to occur in clusters rather than carpeting the bowel [59, 62]. The individual polyps typically vary in shape and size and may be pedunculated or sessile. Larger lesions may demonstrate a lobulated surface [62].

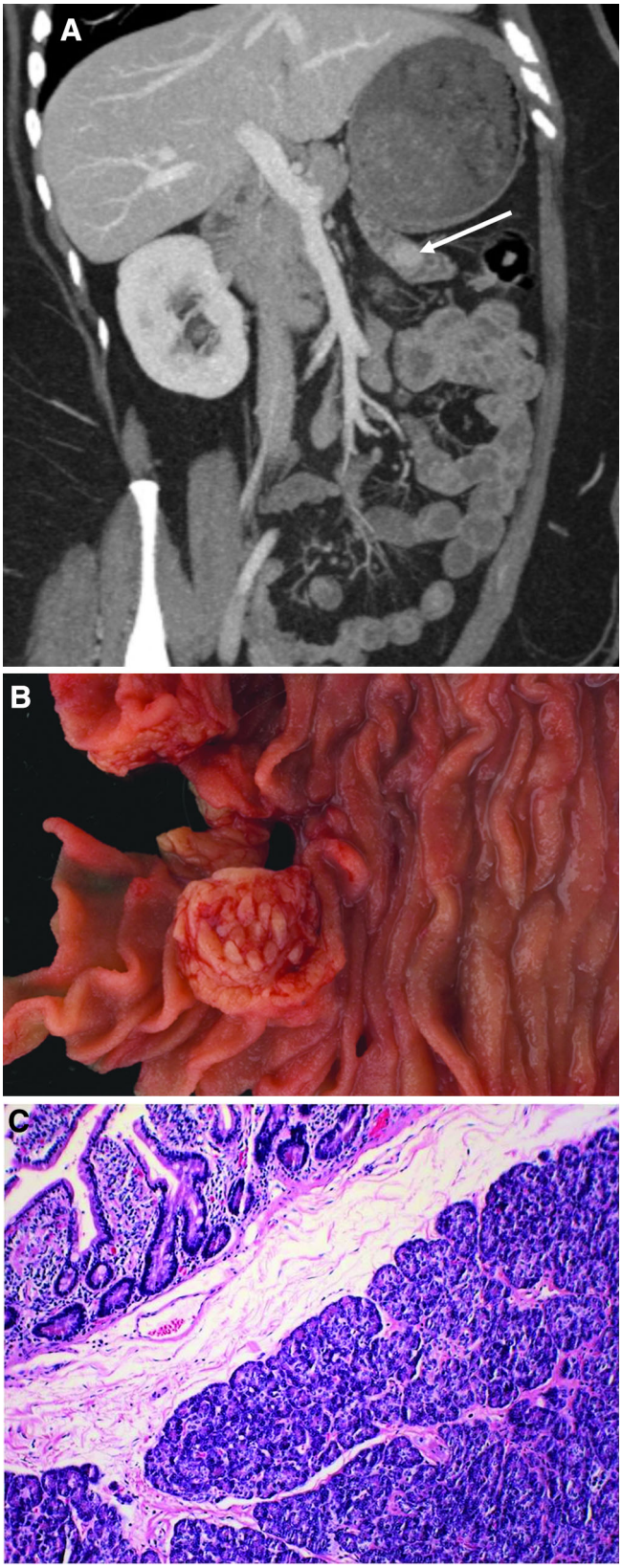
The detection of multiple polyps within the stomach, small bowel, and colon is not limited to PJS. Alternative polyposis syndromes including juvenile polyposis, familial adenomatous polyposis, Cowden syndrome, and Cronkhite–Canada syndrome must be excluded. In most cases, the clinical presentation of the patient will suggest the diagnosis of PJS; however, histologic examination of the polyps is necessary for definitive diagnosis.

Intraluminal polyps may result in intussusception of the small bowel. Since the intussusception is often not associated with significant bowel obstruction, the radiologic appearance can be subtle. A bowel-within-bowel pattern may result, demonstrating a target-like or sausage-shaped mass depending on the orientation of the intussusception.

The characteristic pathologic features of PJ polyps include a frond-like elongated epithelial component with cystic gland dilatation extending to the submucosa or muscularis propria with arborising smooth muscle extending into polyp fronds [63].

Crohn's related polypoid lesions

There are a variety of polypoid manifestations of Crohn's disease including inflammatory polyps, post-inflammatory polyps, and pseudopolyps. While these le-



◀**Fig. 25.** Ectopic pancreas in a 52-year-old male with anemia and suspected gastrointestinal bleed. Oblique sagittal image from CTE (**A**) demonstrate a hyperenhancing lesion within the jejunum. Gross specimen (**B**) demonstrates a polypoid mass within the jejunum. Photomicrograph (×20 magnification; hematoxylin and eosin stain) (**C**) demonstrates normal pancreatic tissue with mixed pancreatic acini and ducts confirming diagnosis of ectopic pancreas.

sions may incite confusion upon the radiologist, there are discriminating factors which may aid in differentiating these pathologies [64].

In active Crohn's disease, inflammatory polyps and pseudopolyps often coexist. An inflammatory polyp is produced when an inflammatory infiltrate, edema, or granulation tissue involves a focal area of mucosa/submucosa within the bowel. This focal area projects above the level of surrounding mucosa and is therefore a true polyp. Pseudopolyps arise in the setting of linear ulcerations that typically crisscross in transverse and longitudinal directions. When extensive ulcerations are present with only scattered foci of normal intervening mucosa, the ulcerated areas may have the appearance of the mucosa, while the true mucosal foci appear as filing defects or polyps. In Crohn's disease, this appearance is known as "cobblestoning."

Post-inflammatory polyps arise as a result of the healing process of the extensive ulcerations created during active Crohn's inflammation. Epithelium regenerates along the undersurface of the remaining foci of normal mucosa as well as along the base of the ulcerated regions, resulting in fingerlike projections of submucosa surrounded by mucosa. Given the typical long and thin geometry of these lesions, they are often referred to as filiform polyps.

Although inflammatory polyps, pseudopolyps, and post-inflammatory polyps may be seen on multiple imaging modalities, these lesions are often identified on MD-CTE as this imaging modality is frequently utilized for inflammatory bowel disease. Determining whether active inflammation is present, is of critical importance in discriminating between these lesions as inflammatory polyps and pseudopolyps are seen in active Crohn's, whereas post-inflammatory polyps are not.

Inflammatory polyps are seen during active Crohn's and are usually present in a region of bowel with secondary signs of active inflammation including mucosal hyperenhancement and mural stratification. These polyps typically appear as hyperenhancing lesions and are most often located within the ileum (Figs. 21 and 22). In contradistinction to pseudopolyps, these lesions are more often solitary or focal.

Pseudopolyps, or cobblestoning, is seen in active Crohn's and may produce the radiographic appearance of an area matted with multiple filling defects, similar to the intestinal polyposis syndromes (Fig. 23). Secondary inflammation and edema extending into the extraintestinal space are suggestive of this diagnosis.

Post-inflammatory polyps, or filiform polyps, most often appear as thin filling defects within the lumen of the bowel and tend to be multiple in nature (Fig. 24).

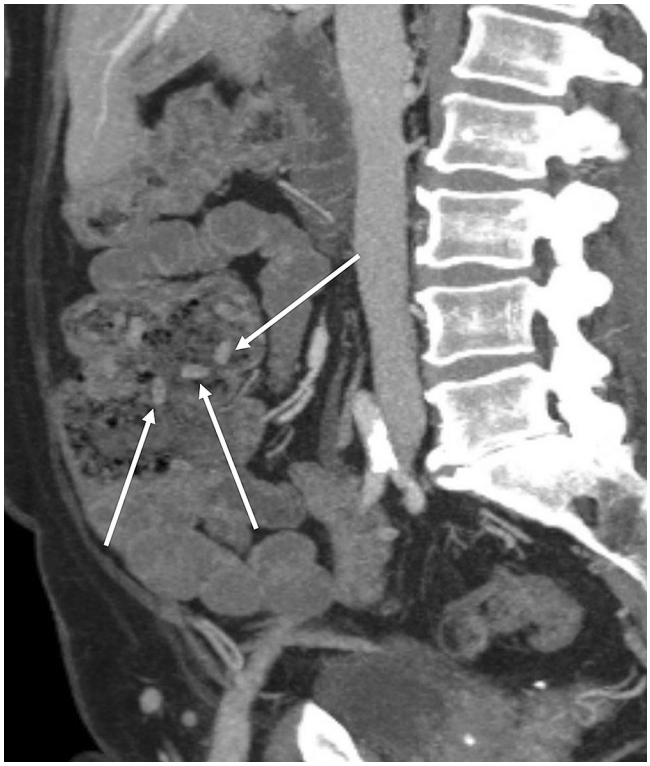


Fig. 26. MD-CTE in a 65-year-old with occult GI bleed. Sagittal MIP image from CTE shows oval-shaped hyperdense intraluminal abnormalities (*arrows*) mimicking polypoid lesions. The hyperdense foci appear to be separate from the mucosa and represent ingested pills.

There may be luminal narrowing, but there is usually no evidence of acute inflammatory disease. Thus, post-inflammatory polyps do not typically demonstrate associated mural edema or hyperemia [65].

Inflammatory polyps, as the name indicates, typically demonstrate a full thickness inflammatory infiltrate with prominent lymphoid aggregates and granulomas at histology. Post-inflammatory polyps often demonstrate a fingerlike geometry with evidence of focal granulation tissue formation without active inflammation.

Ectopic pancreas

Ectopic pancreas, also commonly referred to as heterotopic or aberrant pancreas, is a focus of pancreatic tissue discrete from and lacking anatomic and vascular continuity with the main body of the pancreatic gland. The incidence of ectopic pancreas in autopsy studies is approximately 0.6% to 15%, while the clinical incidence is 1 in 500 laparotomies [66].

While most often discovered incidentally at surgery or autopsy, some patients may develop pancreatitis, pseudocysts, pancreatic cancer, and insulinoma, presenting with abdominal pain, gastrointestinal bleeding, and obstruction [67].

Ectopic pancreas may present on imaging as a submucosal lesion within the stomach or duodenum. Given that GISTs comprise approximately 90% of gastric submucosal tumors, ectopic pancreas may frequently be mistaken for a submucosal GIST or leiomyoma on imaging studies or at endoscopic examination.

While ectopic pancreas may be seen anywhere within the gastrointestinal tract, it is most commonly present within the duodenum (28%), stomach (26%), and proximal jejunum (16%) (Fig. 25) [68–70]. Ectopic pancreas

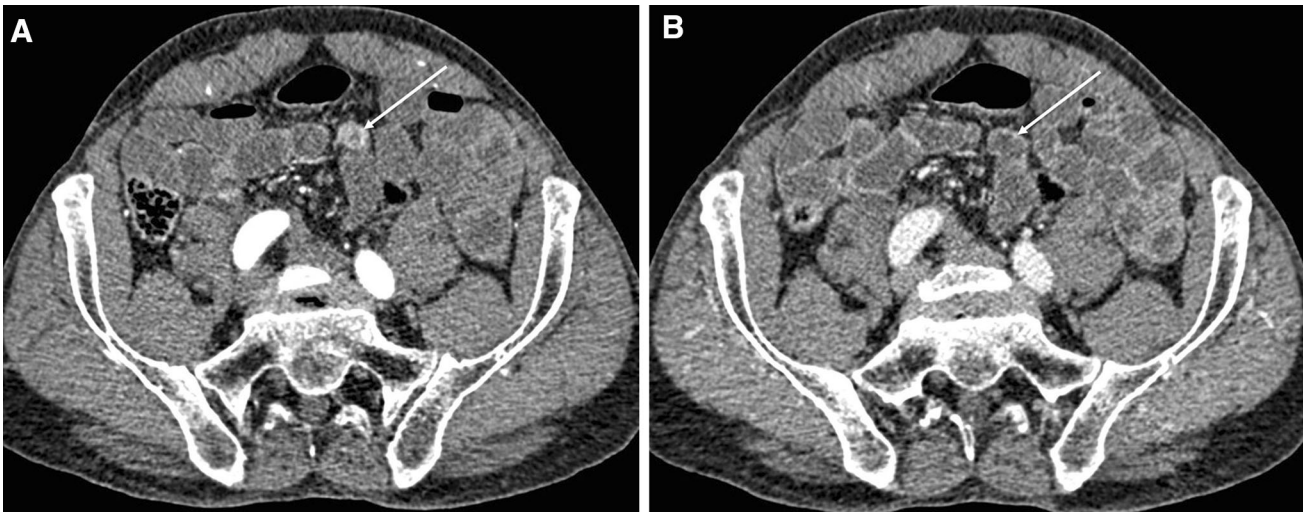


Fig. 27. MD-CTE in a 55-year-old patient with occult GI bleed. Axial arterial phase image from CTE (**A**) suggest a focal hyperenhancing lesion in a non-distended segment of ilium which resolves in delayed images as shown on axial venous phase image (**B**) consistent with peristalsis.

has also been reported within jejunal and ileal diverticula, Meckel's diverticula, gallbladder and bile ducts, fallopian tubes, and the umbilicus.

Kim et al described several radiologic findings of which may help to differentiate ectopic pancreas from GISTs and leiomyomas [71]. First, ectopic pancreas is more likely to demonstrate prominent enhancement of the overlying mucosa which is likely related to the repeated inflammatory changes associated with the lesion. As far as location, ectopic pancreas is more likely to occur within the gastric antrum or duodenum, whereas GISTs are more commonly seen in the gastric body or fundus. A long-axis:short-axis diameter ratio greater than 1.4 was found to be statistically significant in distinguishing ectopic pancreas from GIST or leiomyoma. In addition, endoluminal growth favors ectopic pancreas, whereas exophytic growth suggests a GIST. Ectopic pancreas more often demonstrates an ill-defined margin at MD-CTE.

Endoscopic ultrasound may reveal a submucosal lesion with indistinct borders, heterogeneous echogenicity, and the presence of an anechoic area.

Histologic examination is similar to normal pancreatic tissue and subtypes include predominantly ducts, predominantly pancreatic acini, and mixed. The overlying mucosa may reveal marked inflammation.

Potential pitfalls in diagnosis of focal small bowel lesions

As all imaging tests, MD-CTE has diagnostic pitfalls and limitations. Sensitivity of MD-CTE for evaluation of diffuse small bowel disease largely depends on the severity and extent of disease. As for focal small bowel lesions, sensitivity depends on the size and contrast enhancement of the lesion as well as the degree of small bowel distention. One should confirm the reproducibility of a suspected focal lesion in multiple planes and different reconstruction algorithms such as MIPS and source images. A suspected small fatty lesion on MIP images may relate to a small intraluminal gas bubble on source images. Ingested pills not dissolved within the small bowel lumen can be mistaken for hyperenhancing polypoid lesions (Fig. 26). True polypoid lesions arise from the bowel mucosa and are usually not perfectly oval or round as ingested capsules or pills are. Focal short segment area of peristalsis may be mistaken for isoenhancing or mildly hyperenhancing focal lesion (Fig. 27). With multiphasic MD-CTE, a true focal lesion should be persistent on all phases, whereas peristalsis usually is seen on a single phase and resolves on other phases. With single-phase examinations, one should assure the presence of a suspected lesion on all planes.

Conclusion

Multidetector CT enterography has rapidly gained acceptance as a method of choice for evaluation of diffuse small bowel pathologies specifically Crohn's disease. In our opinion, MD-CTE is the diagnostic test of choice for evaluation of suspected focal small bowel lesions.

Compliance with ethical standards

Funding No funding was received for this study.

Conflicts of interest The authors declare that they have no conflict of interest.

Ethical approval This article does not contain any studies with human participants or animals performed by any of the authors.

Informed consent Statement of informed consent was not applicable since the manuscript does not contain any patient data.

References

1. Maglinte DD, O'Connor K, Bessette J, Chernish SM, Kelvin FM (1991) The role of the physician in the late diagnosis of primary malignant tumors of the small intestine. Am J Gastroenterol 86:304–308
2. Ciresi DL, Scholten DJ (1995) The continuing clinical dilemma of primary tumors of the small intestine. Am Surg 61:698–703
3. Paulsen RS, Huprich J, Fletcher J, et al. (2006) Diagnostic tool in evaluating small bowel disorders: review of clinical experiences with over 700 cases. RadioGraphics 26:641–662
4. Deepak G, Sharma V, Rana S, Bhasin D (2014) Small bowel bleeding: a comprehensive review. Gastroenterol Rep 2:262–275
5. Huprich J, Fletcher J, Fidler J (2011) Obscure GI bleeding: the role of multiphase CT enterography. Appl Radiol. Accessed at 4 Feb 2015 from http://appliedradiology.com/articles/obscure-gi-bleeding-the-role-of-multiphase-ct-enterography#sthash.IKrG1APQ.dpuf.
6. Dabaja BS, Suki D, Pro B, Bonnen M, Ajani J (2004) Adenocarcinoma of the small bowel: presentation, prognostic factors, and outcome of 217 patients. Cancer 101(3):518–526 (PubMed PMID: 15274064)
7. Weber NK, Fletcher JG, Fidler JL, et al. (2014) Clinical characteristics and imaging features of small bowel adenocarcinomas in Crohn's disease. Abdom Imaging 40(5):1060–1067 ([Epub ahead of print])
8. Buckley JA, Fishman EK (1998) CT evaluation of small bowel neoplasms: spectrum of disease. Radiographics 18(2):379–392
9. Soyer P, Boudiaf M, Fishman EK, et al. (2011) Imaging of malignant neoplasms of the mesenteric small bowel: new trends and perspectives. Crit Rev Oncol Hematol 80(1):10–30. doi: 10.1016/j.critrevonc.2010.09.010
10. Loke TK, Lo SS, Chan CS (1997) Case report: Krukenberg tumors arising from a primary duodenojejunal adenocarcinoma. Clin Radiol 52(2):154–155
11. Cronin CG, Scott J, Kambadakone A, et al. (2012) Utility of positron emission tomography/CT in the evaluation of small bowel pathology. Br J Radiol 85(1017):1211–1221. doi: 10.1259/bjr/64534573
12. Iacobuzio-Donahue, CA. II, Montgomery, E. Gastrointestinal and liver pathology. Copyright © 2012, 2005 by Saunders, an imprint of Elsevier Inc.
13. Pelage JP, Soyer P, Boudiaf M, et al. (1999) Carcinoid tumors of the abdomen: CT features. Abdom Imaging 24(3):240–245
14. Horton KM, Kamel I, Hofmann L, Fishman EK (2004) Carcinoid tumors of the small bowel: a multitechnique imaging approach. AJR Am J Roentgenol 182(3):559–567
15. Soyer P, Dohan A, Eveno C, et al. (2013) Carcinoid tumors of the small-bowel: evaluation with 64-section CT-enteroclysis. Eur J Radiol 82(6):943–950. doi:10.1016/j.ejrad.2013.02.013

16. Horton KM, Fishman EK (2004) Multidetector-row computed tomography and 3-dimensional computed tomography imaging of small bowel neoplasms: current concept in diagnosis. J Comput Assist Tomogr 28(1):106–116
17. Fernandes DD, Galwa RP, Fasih N, Fraser-Hill M (2012) Cross-sectional imaging of small bowel malignancies. Can Assoc Radiol J 63(3):215–221. doi:10.1016/j.carj.2010.10.001
18. Mendelson RM, Fermoyle S (2006) Primary gastrointestinal lymphomas: a radiological-pathological review. Part 2: Small intestine. Australas Radiol 50(2):102–113
19. Ghai S, Pattison J, Ghai S, et al. (2007) Primary gastrointestinal lymphoma: spectrum of imaging findings with pathologic correlation. Radiographics 27(5):1371–1388
20. Morgan PB, Kessel IL, Xiao SY, Colman M (2004) Uncommon presentations of Hodgkin's disease. Case 1. Hodgkin's disease of the jejunum. J Clin Oncol 22(1):193–195
21. Pisters PWT, Blanke CD, von Mehren M, et al. (2011) A USA registry of gastrointestinal stromal tumor patients: changes in practice over time and differences between community and academic practices. Ann Oncol 22(11):2523–2529
22. Ma GL, Murphy JD, Martinez ME, Sicklick JK (2015) Epidemiology of gastrointestinal stromal tumors in the era of histology codes: results of a population-based study. Cancer Epidemiol Biomarkers Prev 24:298
23. Zhao X, Yue C (2012) Gastrointestinal stromal tumor. J Gastrointest Oncol 3(3):189–208
24. Hong X, Choi H, Loyer EM, et al. (2006) Gastrointestinal stromal tumor: role of CT in diagnosis and in response evaluation and surveillance after treatment with imatinib. Radiographics 26(2):481–495
25. Tateishi U, Hasegawa T, Satake M, et al. (2003) Gastrointestinal stromal tumor. Correlation of computed tomography findings with tumor grade and mortality. J Comput Assist Tomogr 27:792–798
26. Lens M, Bataille V, Krivokapic Z (2009) Melanoma of the small intestine. Lancet Oncol 10(5):516–521
27. Kouladouros K, Gartner D, Munch S, et al. (2015) Recurrent intussusception as initial manifestation of primary intestinal melanoma: case report and literature review. World J Gastroenterol 21(10):3114–3120
28. Sachs DL, Lowe L, Chang AE, et al. (1999) Do primary small intestinal melanomas exist? Report of a case. J Am Acad Dermatol 41(6):1042–1044
29. Elsayes KM, Al-Hawary MM, Jagdish J, et al. (2010) CT enterography: principles, trends, and interpretation of findings. Radiographics 30(7):1955–1970
30. Gong F, Swain P, Mills TN (2000) Wireless endoscopy. Gastrointest Endosc 51:725–729
31. Xynopoulos D, Mihas A, Paraskevas E, Dimitroulopoulos D, et al. (2002) Small bowel tumors. Ann Gastroenterol 15:18–35
32. Gourtsoyiannis N, Mako E (1997) Imaging of primary small intestinal tumors by enteroclysis and CT with pathological correlation. Eur Radiol 7:625–642
33. Thompson W (2005) Imaging and findings of lipomas of the gastrointestinal tract. Am J Roentgenol 184(4):1163–1171
34. Heiken JP, Forde KA, Gold RP (1982) Computed tomography as a definitive method for diagnosing gastrointestinal lipomas. Radiology 142:409–414
35. Savides TJ, Jensen DM (2010) Gastrointestinal bleeding. In: Feldman M, Frience S, Brandt LJ (eds) *Sleisenger and Fordtran's gastrointestinal and liver disease*, 9th edn. Philadelphia: Elsevier Medicine
36. Raju GS, Gerson L, Das A, Lewis B, American Gastroenterological A (2007) American Gastroenterological Association (AGA) Institute technical review on obscure gastrointestinal bleeding. Gastroenterology 133:1697–1717
37. Huprich JE, Barlow JM, Hansel SL, Alexander JA, Fidler JL (2013) Multiphase CT enterography evaluation of small-bowel vascular lesions. AJR Am J Roentgenol. 201(1):65–72
38. Imdahl A (2001) Genesis and pathophysiology of lower gastrointestinal bleeding. Langenbecks Arch Surg 386:1
39. Singh AK, Federman DD (2015) *Scientific American medicine*. Ontario/Philadelphia: Decker Electronic Publishing, Inc.
40. Hara AK, Walker FB, Silva AC, Leighton JA (2009) Preliminary estimate of triphasic CT enterography performance in hemodynamically stable patients with suspected gastrointestinal bleeding. AJR Am J Roentgenol 193(5):1252–1260
41. Soyer P (2012) Obscure gastrointestinal bleeding: difficulties in comparing CT enterography and video capsule endoscopy. Eur Radiol 22:1167–1171
42. Huprich JE, Fletcher JG, Fidler JL, et al. (2011) Prospective blinded comparison of wireless capsule endoscopy and multiphase CT enterography in obscure gastrointestinal bleeding. Radiology 260:744–751
43. Liao Z, Gao R, Xu C, Li ZS (2010) Indications and detection, completion and retention rates of small bowel capsule endoscopy: a systemic review. Gastrointest Endosc 71:280–286
44. Boley SJ, Sammartano R, Adams A, et al. (1977) On the nature and etiology of vascular ectasias of the colon: degenerative lesions of aging. Gastroenterology 72:650–660
45. Poralla T (1998) Angiodysplasia in the renal patient: how to diagnose and how to treat? Nephrol Dial Transplant 13:2188–2191
46. Regula J, Wronska E, Pachlewski J (2008) Vascular lesions of the gastrointestinal tract. Best Pract Res Clin Gastroenterol 22:313–328
47. Huprich JE (2009) Multi-phase CT enterography in obscure GI bleeding. Abdom Imaging 34(3):303–309
48. Boley SJ, Sprayregen S, Sammartano RJ, Adams A, Kleinhaus S (1977) The pathophysiologic basis for the angiographic signs of vascular ectasias of the colon. Radiology 125:615–621
49. Baxter M, Aly EH (2010) Dieulafoy's lesion: current trends in diagnosis and management. Ann R Coll Surg Engl 92:548–554
50. Dulic-Lakovic E, Dulic M, Hubner D, et al. (2011) Bleeding Dieulafoy lesions of the small bowel: a systematic study on the epidemiology and efficacy of enteroscopic treatment. Gastrointest Endosc 74:573–580
51. Norton ID, Petersen BT, Sorbi D, et al. (1999) Management and long-term prognosis of Dieulafoy lesion. Gastrointest Endosc 50:762–767
52. Boyle L, Lack EE (1993) Solitary cavernous hemangioma of the small intestine: case report and literature review. Arch Pathol Lab Med 117:939–941
53. Tang SJ, Jutabha R, Jensen DM (2002) Push enteroscopy for recurrent gastrointestinal hemorrhage due to jejunal anastomotic varices: a case report and review of the literature. Endoscopy 34:735–737
54. Norton ID, Andrews JC, Kamath PS (1998) Management of ectopic varices. Hepatology 28:1154–1158
55. Watanabe N, Toyonaga A, Kojima S, et al. (2010) Current status of ectopic varices in Japan: results of a survey by the Japan Society for Portal Hypertension. Hepatol Res 40:763–776
56. De Palma GD, Rega M, Masone S, et al. (2005) Mucosal abnormalities of the small bowel in patients with cirrhosis and portal hypertension: a capsule endoscopy study. Gastrointest Endosc 62:529–534
57. Rana SS, Bhasin DK, Jahagirdar S, et al. (2006) Is there ileopathy in portal hypertension? J Gastroenterol Hepatol 21:392–397
58. Giardiello FM, Trimbath JD (2006) Peutz–Jeghers syndrome and management recommendations. Clin Gastroenterol Hepatol 4:408–415
59. Utsunomiya J, Gocho H, Miyanaga T, et al. (1975) Peutz–Jeghers syndrome: its natural course and management. Johns Hopkins Med J 136:71–82
60. Vogel T, Schumacher V, Saleh A, et al. (2000) Extraintestinal polyps in Peutz–Jeghers syndrome: presentation of four cases and review of the literature. Deutsche Peutz–Jeghers–Studiengruppe. Int J Colorectal Dis 15:118–123
61. Betts AD, Latchford AR, Vasen AR, et al. (2010) Peutz–Jeghers syndrome: a systematic review and recommendations for management. Gut 59(7):975–986
62. Buck JL, Hamed RK, Lichenstein JE, Sobin LH (1992) Peutz–Jeghers syndrome. RadioGraphics 12:365–378
63. Jass JR, Williams CB, Bussey HJ, et al. (1988) Juvenile polyposis—a precancerous condition. Histopathology 13:619–630
64. Buck J, Dachman A, Sobin L (1991) Polypoid and pseudopolypoid manifestations of inflammatory bowel disease. RadioGraphics 11:293–304
65. Sinha R, Rajiah P, Murphy P, Hawker P, Sanders S (2009) Utility of high-resolution mr imaging in demonstrating transmural

pathologic changes in Crohn disease. RadioGraphics 29(6):1847–1867
66. Elpek GO, Bozova S, Kupesiz GY, Ogus M (2007) An unusual cause of cholecystitis: heterotopic pancreatic tissue in the gallbladder. World J Gastroenterol 13(2):313–315
67. Rooney DR (1959) Aberrant pancreatic tissue in the stomach. Radiology 73:241–244
68. Thoeni RF, Gedgaudas RK (1980) Ectopic pancreas: usual and unusual features. Gastrointest Radiol 5:37–42
69. Grendell JH, Ermak TH (1998) Anatomy, histology, embryology, and developmental anomalies of the pancreas. In: Felman M, Friedman L, Brandt L (eds) *Sleisenger & Fordtran's gastrointestinal and liver disease*. Philadelphia, PA: Saunders, pp 761–771
70. Wei R, Wang WB, Chen QH, Liu JS, Zhang B (2011) Upper gastrointestinal tract heterotopic pancreas: findings from CT and endoscopic imaging with histopathologic correlation. Clin Imaging 35(5):353–359
71. Kim JY, Lee JM, Kim KW, et al. (2009) Ectopic pancreas: CT findings with emphasis on differentiation from small gastrointestinal stromal tumor and leiomyoma. Radiology 252(1):92–100