Imaging of the Small Bowel Tumors

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KEYWORDS

• Imaging • Small bowel tumors • GIST • Neuroendocrine • Adenocarcinoma • Lymphoma

KEY POINTS

- Computed tomography and MR enterography are the imaging modalities of choice for evaluating small bowel tumors.
- Neuroendocrine neoplasms are small hyperenhancing lesions that are often multifocal within the small bowel.
- Small bowel adenocarcinomas are usually solitary annular/semi-annular masses with abrupt shouldering and luminal narrowing.
- Gastrointestinal stromal tumors are variably sized, smoothly marginated, lobulated tumors often with necrosis, hemorrhage, and/or degeneration.
- Primary small bowel lymphomas vary in imaging appearance and may be nodular/polypoid, infiltrative, or endoexoenteric with cavitation and fistulation common.

INTRODUCTION

Small bowel tumors account for only 5% of gastrointestinal (GI) tumors in the United States but have been rising in incidence over recent decades. The proportion of neuroendocrine tumors (NETs) has steadily increased since the 1970s,[1–4] partly due to the increased use of cross-sectional imaging and improved small bowel imaging techniques. Imaging plays a crucial role in detecting and diagnosing small bowel tumors, enhancing the ability to identify and treat various malignancies at earlier stages. Diagnosing small bowel tumors is challenging as many patients are asymptomatic or present with nonspecific symptoms such as abdominal pain, weight loss, nausea, vomiting, intestinal obstruction, and GI bleeding.[1,5] This article explores the current imaging techniques for small bowel evaluation, focusing on computed tomography (CT) and MR enterography, and examines their role in the 5 most common small bowel malignancies: neuroendocrine neoplasms (NENs), adenocarcinoma, gastrointestinal tumors (GISTs), small bowel lymphoma, and metastases.

IMAGING TECHNIQUES

Traditionally, fluoroscopic small bowel follow-through studies and enteroclysis were primary imaging modalities for the small bowel, but noninvasive CT and MR imaging have largely replaced these techniques. CT offers rapid image acquisition, wide availability, consistent image quality, high spatial resolution, and multi-planar reformatting capabilities. MR imaging provides multitimepoint imaging, superior contrast resolution, and lacks ionizing radiation. Both modalities assess the lumen, mural thickness, extramural extent, and extraintestinal findings. Routine abdominopelvic CT without oral contrast and sufficient bowel distension is not optimal for small bowel assessment, with studies showing routine single-phase portal venous CT frequently missing small bowel tumors compared to CT enterography (CTE). One study found a CTE small bowel tumor detection rate of 95% versus only 45% with routine single-phase CT.[6,7] Thus, CT and MR enterography (MRE) with optimal small bowel distension have become the mainstay for small bowel imaging.

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Radiol Clin N Am 63 (2025) 345–359
https://doi.org/10.1016/j.rcl.2024.11.001

For both CTE and MRE, patients fast for 4 to 6 hours before the examination to enhance compliance with drinking a large volume of fluid and to reduce intraluminal filling defects that can mimic masses.[1] Patients ingest approximately 1 L of oral contrast material in split doses 60 minutes prior to scanning to distend the small bowel lumen (**Table 1**). Commercially available contrast agents for enterography provide neutral contrast (similar to water) on CTE and biphasic characteristics on MRE (low T1 and high T2 signal intensity). For CTE, patients receive intravenous (IV) contrast material, and images are usually obtained during an enteric or early portal venous phase (50–60 seconds post-contrast injection). Images are acquired from the lung bases through the perineum with thin section acquisition and with multiplanar reformations created. Some small bowel tumors may be more conspicuous on different phases, although a single phase often suffices for most neoplasms.[1] A multiphase protocol should be employed, however, when GI bleeding is suspected.

The oral contrast protocol for MRE mirrors that of CTE. Administering spasmolytics (IV or intramuscular [IM] Glucagon or IV or sublingual hyoscine butylbromide) improves image quality for MRE, which is more sensitive to motion artifacts. MR images are obtained both before and after IV gadolinium contrast administration, with multi-planar imaging performed in both the axial and coronal planes. MR imaging may also be performed in the prone position to reduce motion artifact and improve small bowel distension. Motion-insensitive T2-weighted images (single shot fast spin echo [SSFSE], balanced steady-state free precession [bSSFP]) provide high contrast between the high T2 signal intensity oral contrast and relatively lower T2 signal intensity masses. The bSSFP sequences offer more homogenous intraluminal fluid compared to the SSFSE images, which suffer from intraluminal flow void artifacts. Diffusion-weighted images (DWIs) may help identify subtle lesions or confirm suspicious findings by demonstrating restricted diffusion. Three dimensional (3D) T1-weighted images performed before IV contrast administration identify high signal intensity intraluminal material or blood that can mimic enhancement. Post-contrast 3D T1-weighted images improve the detection of enhancing small bowel lesions, and multiphasic acquisition can mitigate equivocal findings secondary to bowel peristalsis and other imaging artifacts. **Table 1** provides CTE and MRE technique parameters.

ENDOSCOPIC EVALUATION

Conventional endoscopic techniques provide a detailed evaluation of the mucosal surface with the capability for biopsy but have limited reach into the small bowel. Balloon-assisted enteroscopy

Table 1
Computed tomography/MR enterography elements

Contrast	Protocol
Neutral enteric contrast—for example, Breeza, VoLumen, or CitraClear, allowing for adequate distension of small intestine	500 mL PO 60 min before scan 500 mL PO 45 min before scan 500 mL PO 30 min before scan 500 mL PO water 15 min before scan *NPO for 4–6 h prior to scan
Intravenous contrast	CT: Weight-based dosing of Omnipaque 300 followed by 50 mL of 0.9% NaCl MR imaging: Weight-based dosing of Gadavist: 1 mmol/mL
CT Phases	Single phase usually adequate for evaluation of small bowel tumors. However, multiple phases may be acquired, particularly if there is clinical presentation of GI bleeding • Arterial phase (bolus tracked) • Enteric phase (approximately 50 s post-contrast injection) • Delayed (90 s)
MR Sequences	Administration of IV/IM glucagon to reduce bowel peristalsis Sequences obtained include • Diffusion-weighted imaging • Half-Fourier Single Shot Fast Spin Echo • Balanced Steady-state-free Precession • 3D Fast Spoiled Gradient Echo—Pre-contrast and post-contrast

* The * is an additional note that patients need to be NPO 4-6 hours prior to scan as part of the enterography protocol.

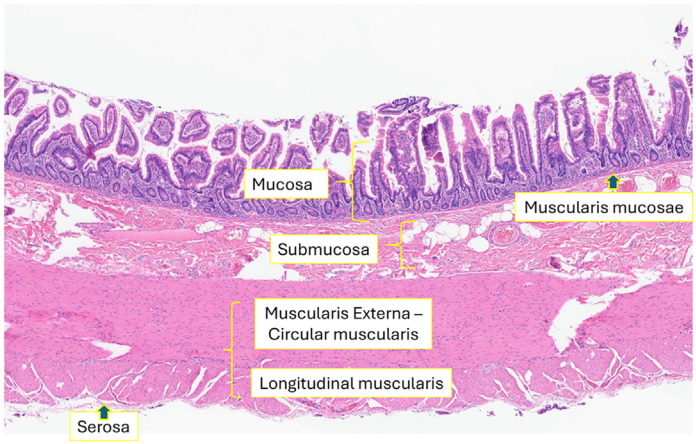

Fig. 1. Light microscopy of normal small intestine, stained with hematoxylin and eosin.

(antegrade or retrograde) allows access to a greater length of the small bowel but is time-consuming and requires specialized expertise. Video capsule endoscopy (VCE) offers a comprehensive view of the entire small bowel but presents challenges in localization and does not permit biopsy. Known or suspected GI obstruction is an absolute contraindication for VCE due to risk of retention, which may require surgical retrieval. Endoscopic techniques also have limitations in detecting submucosal tumors and cannot provide information about the extramural extent of small bowel tumors. Studies show that CTE is more sensitive (92.7%) than capsule endoscopy (29.6%) in detecting small bowel tumors.[8,9]

HISTOLOGY

The small bowel consists of 4 layers: the mucosa, submucosa, muscularis propria, and serosa (**Fig. 1**). The mucosa, the inner-most layer, contains the epithelium, lamina propria, and muscularis mucosa. The mucosa features villi, finger-like projections extending into the bowel lumen, lined by the epithelial layer consisting of absorptive enterocytes and secretory Goblet cells and with a core connective tissue layer called the lamina propria. The crypts of Lieberkuhn, the deepest part of the epithelium, contain enteroendocrine cells. The submucosa includes connective tissue, blood vessels, lymphatics, and the Meissner plexus. Peyer patches are lymphoid follicles located in the lamina propria and submucosa of the ileum. The muscularis propria is responsible for peristalsis. It contains the myenteric (Auerbach) nervous plexus between its 2 muscular layers and also houses the interstitial cells of Cajal, the pacemakers for peristalsis.[10] The serosa, the outermost layer present only in intraperitoneal segments, secretes serous fluid for lubrication.[11]

Table 2
World Health Organization classification of neuroendocrine neoplasms

WHO Category	Tumor Grade	Degree of Differentiation	Mitotic Rate (Mitoses/2 mm^2)	Ki-67 Index (%)
Neuroendocrine Tumors (NET)	Low (Gr 1)	Well	<2	<3
	Intermediate (Gr 2)	Well	2–20	3–12
	High (Gr 3)	Well	>20	>20
Neuroendocrine Carcinoma (NEC)	High, small cell type (SCNEC)	Poorly	>20	>20
	High, Large cell type (LCNEC)	Poorly	>20	>20
Mixed Neuroendocrine-Nonneuroendocrine Neoplasm	Variable	Well or Poorly	Variable	Variable

Adapted from Nagtegaal ID, Odze RD, Klimstra D, et al. (2020) The 2019 WHO classification of tumors of the digestive system. Histopathology 76:182-188. 10.1111/his.13975

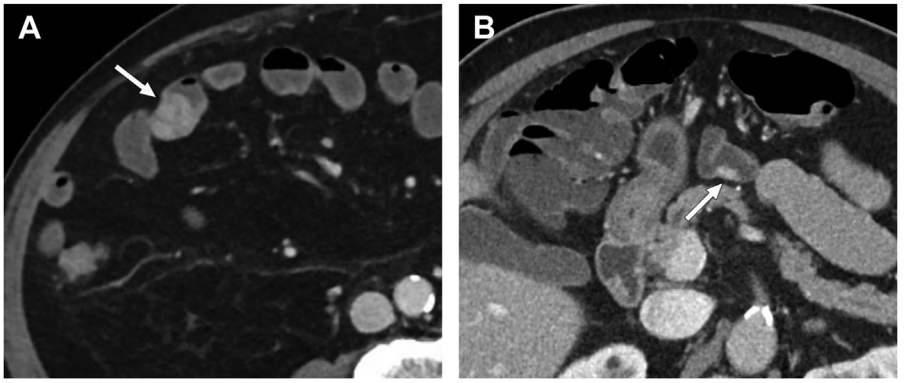

Fig. 2. Ileal neuroendocrine neoplasms (NEN). (*A*) Axial post-contrast CTE shows a hyperenhancing, eccentric plaque-like NET with crescentic morphology and serosal puckering (*arrow*). (*B*) Axial post-contrast CTE shows a small polypoid NEN in the proximal ileum (*arrow*). Ileal NENs are often multifocal, necessitating comprehensive small bowel survey.

SMALL BOWEL TUMORS

Neuroendocrine

NENs are the most common primary small bowel malignancy.[12] They originate from intraepithelial enteroendocrine cells and can occur throughout the small bowel.[6] Functional NENs secrete hormones causing specific syndromes, whereas nonfunctional NETs do not secrete clinically significant amounts of hormones. For this review, small bowel NENs are defined as those arising between the ligament of Treitz and the ileocecal valve, the majority of which occur in the distal ileum.[13] Most are nonfunctional, slow-growing, and often incidentally detected.[14,15] The World Health Organization classifies NENs into well-differentiated NETs (80%–90%), poorly differentiated neuroendocrine carcinomas (NECs, 10%–20%), and mixed neuroendocrine neoplasms (MiNENs)/non-NENs based on mitotic rate, Ki-67 index, and degree of differentiation.[16] NECs have distinct molecular mutations and are subdivided into small-cell and large-cell subtypes, both with high mitotic rate and Ki-67 expression (**Table 2**).[17] MiNENs, with histologic features of both neuroendocrine and non-NETs, carry a poor prognosis.[17]

Over half of small bowel NENs are detected by imaging, with smaller percentage identified at endoscopy or surgery.[6] Primary small bowel NENs are often small but hypervascular, appearing as hyperenhancing lesions on both CT and MR imaging during the arterial and/or enteric phase (**Fig. 2**A, B). They are multifocal in up to 33% to 54% of patients.[18] Most lesions are less than 2 cm and they can have a variety of morphologies: polypoid mucosal or submucosal lesion, eccentric plaque-like mural thickening (often with a crescentic appearance) with or without serosal puckering, and less commonly, carpet-like lesions with segmental submucosal spread.[18] MR imaging

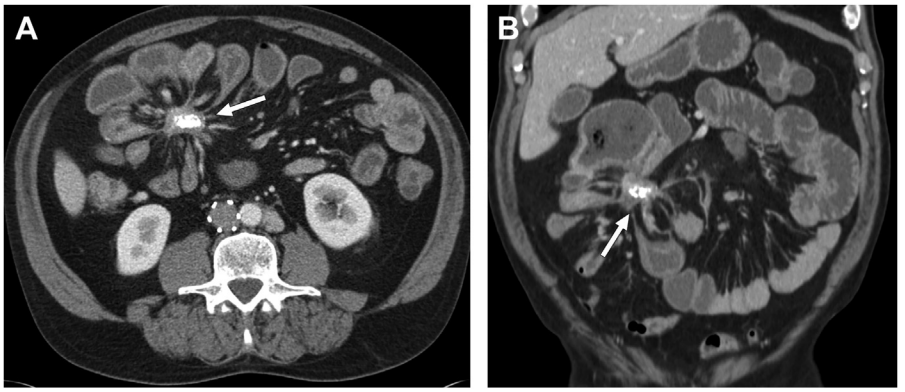

Fig. 3. Mesenteric nodal metastasis from small bowel NEN. (*A* and *B*) Axial and coronal post-contrast CTE images reveal a partially calcified nodal metastasis (*arrows*) with desmoplasia ("spoke wheel" appearance), causing multifocal small bowel tethering and low-grade obstruction.

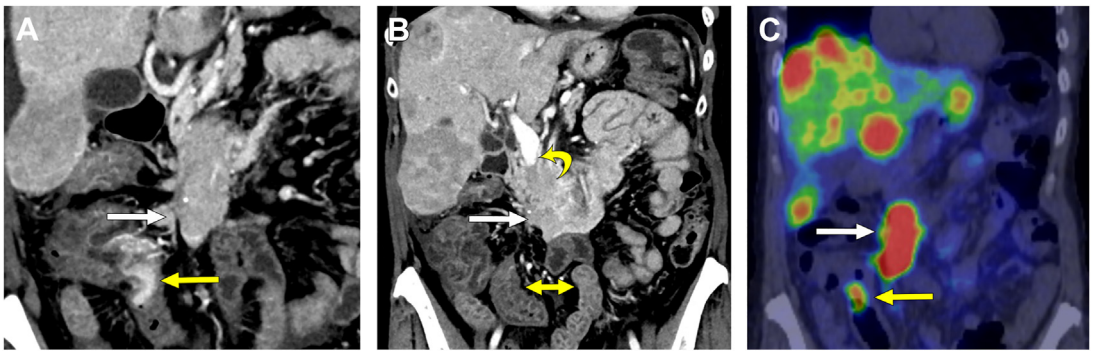

Fig. 4. Metastatic ileal NEN. (*A* and *B*) Coronal contrast-enhanced CT images demonstrate the crescentic-shaped primary tumor with serosal puckering (*A*, *yellow arrow*) and bulky hyperenhancing mesenteric nodal metastasis (*white arrows*). Nodal involvement may lead to vascular compromise (*B*, curved *arrow*), resulting in small bowel congestion (*B*, *double-head arrow*), ischemia, or varices. (*C*) Coronal fused image from Ga-DOTATATE PET/CT demonstrates radiotracer uptake within the primary neuroendocrine tumor (*yellow arrow*), nodal disease (*white arrow*), and liver metastases.

shows small bowel NENs as T1 isointense, T2 isointense or hyperintense, with avid arterial enhancement, and restricted diffusion.[6] Both CTE and MRE provide high per-patient sensitivity; however, per-lesion sensitivity suffers due to multifocality.[6] The desmoplastic reaction of these tumors often causes kinking or obstruction of the small bowel[6]

Despite their small size, NENs frequently metastasize, with nodal metastases common even in tumors less than 1 cm.[18] Imaging often shows hyperenhancing nodal metastases, which may appear round and smooth or stellate with spiculation, calcification, and mesenteric retraction due to hormone production and secondary fibrosis (**Fig. 3**A, B). Vascular encasement is common (**Fig. 4**A, B).[18] Extranodal metastases commonly occur in the liver but can also develop in the lungs, bones, ovaries, and peritoneum.[18] Sensitivity for liver metastases increases with biphasic contrast-enhanced liver CT or multiphasic MR imaging as they often hyperenhance.[6] Some radiologists advocate for hepatocyte-specific MR contrast agents for evaluating hepatic metastases.[6] Liver metastases are usually conspicuous and hyperintense on MR T2-weighted sequences and DWI.[6]

PET/CT or PET/MR imaging with somatostatin receptor analogs exploits the overexpression of somatostatin, present in 80% to 100% of small bowel NENs (**Fig. 4**C). They offer high per-patient sensitivity (but a much lower per-lesion sensitivity) and specificity for a primary tumor and metastases and can potentially help guide peptide receptor radionuclide therapy.[6] Somatostatin receptor PET/CT is less useful for well-differentiated NENs; however, it may have a role in poorly differentiated NENs.[6] Up to 10% of patients with stage IV NEN develop carcinoid syndrome, caused by

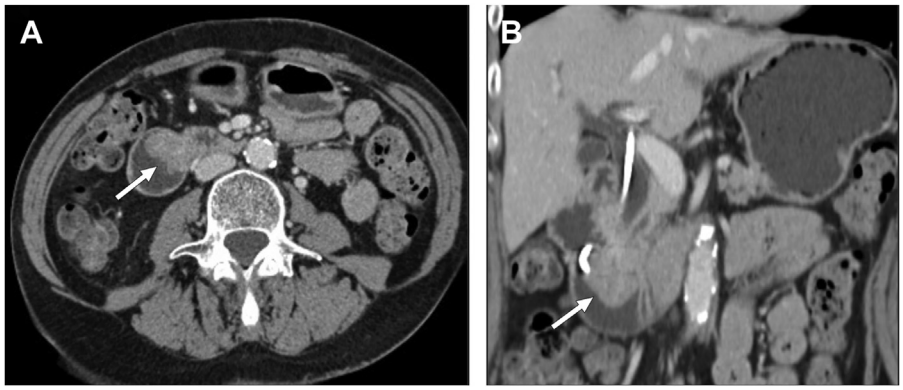

Fig. 5. Periampullary adenoma of the duodenum. (*A* and *B*) Axial and coronal contrast-enhanced CT images show a tubulovillous periampullary adenoma (*arrows*) causing obstruction and mild biliary dilatation (biliary stent present).

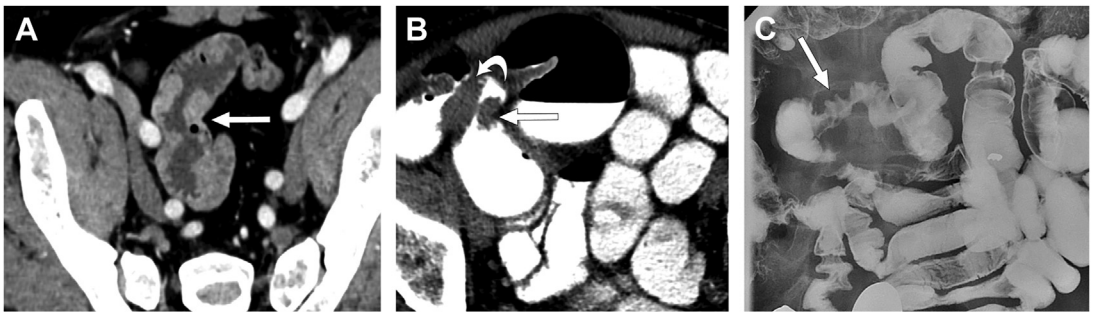

Fig. 6. Small bowel adenocarcinoma. (*A*) Axial contrast-enhanced CT demonstrates a semiannular, mildly enhancing mass with mild luminal narrowing (*arrow*). Given isoenhancement with bowel wall and lack of obstruction, this could be overlooked without adequate small bowel distension. (*B*, *C*) Axial contrast-enhanced CT and fluoroscopic small bowel follow-through in 2 patients with adenocarcinoma reveal circumferential bowel wall thickening with sharp shouldering margins, luminal narrowing, and upstream dilatation indicating small bowel obstruction. (*C*) The classic "apple-core" appearance. CT allows for assessment of perienteric mesenteric infiltration (*B*, curved *arrow*).

the release of serotonin and other vasoactive substances, leading to symptoms like flushing, diarrhea, wheezing, and, in severe cases, heart valve lesions.[14]

Adenocarcinoma

Small bowel adenocarcinomas make up about approximately 31% to 40% of small bowel malignancies. These primary adenocarcinomas, arising from the glandular epithelium, are most common in the duodenum (60%), followed by the jejunum (25%–29%), and ileum (10%–13%).[2] Patients often present with nonspecific symptoms, leading to delayed diagnoses and many patients present emergently due to obstruction or bleeding from advanced disease.[19] Most cases are sporadic, but associations with polyposis syndromes and inflammatory bowel diseases like Crohn's or celiac disease exist.[19]

Small bowel adenocarcinomas likely arise via an adenoma-to-carcinoma transformation, similar to colorectal carcinomas. The morphology (tubular/tubulovillous/villous), size, location, and multicentricity of the adenomas are significant risk factors for malignant transformation (**Fig. 5**A, B).[20,21] Imaging findings vary by location with periampullary and duodenal tumors, most common in the setting of polyposis syndromes, often appearing more polypoid and well-circumscribed.[22] However, many small bowel adenocarcinomas present as solitary annular or semi-annular masses with abrupt shouldering and luminal narrowing (apple-core morphology; **Fig. 6**A).[19,22] Ulceration is common, and luminal narrowing often results in small bowel obstruction (**Fig. 6**B). Most have mild homogeneous or heterogeneous contrast enhancement with increasing size.[23] MR imaging shows adenocarcinomas as mildly T2 hyperintense lesions with restricted diffusion. Locally advanced lesions may infiltrate into the adjacent mesenteric fat (**Fig. 6**C). Many cases present with regional lymph node metastases and distant metastases, commonly in liver and peritoneum.[19,23]

Surgery is the primary treatment of locoregional disease, but due to the rarity of this cancer,

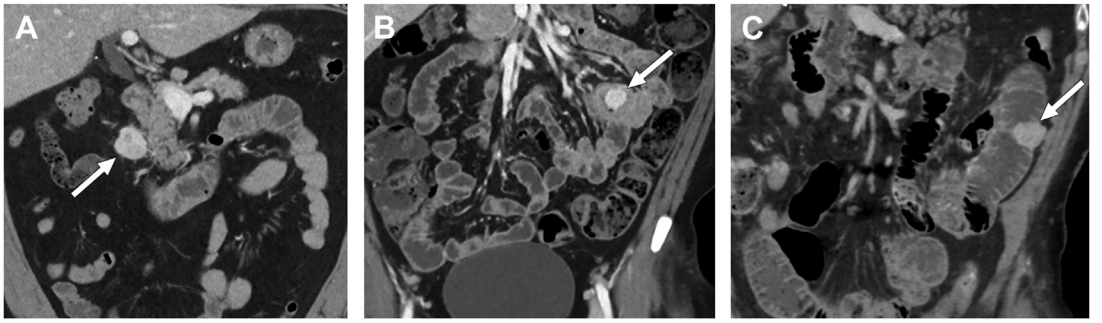

Fig. 7. Gastrointestinal stromal tumor (GIST). Coronal contrast-enhanced CT images demonstrate the exophytic (*A*), endophytic (*B*), and endoexophytic (*C*) growth patterns typical of small bowel GIST.

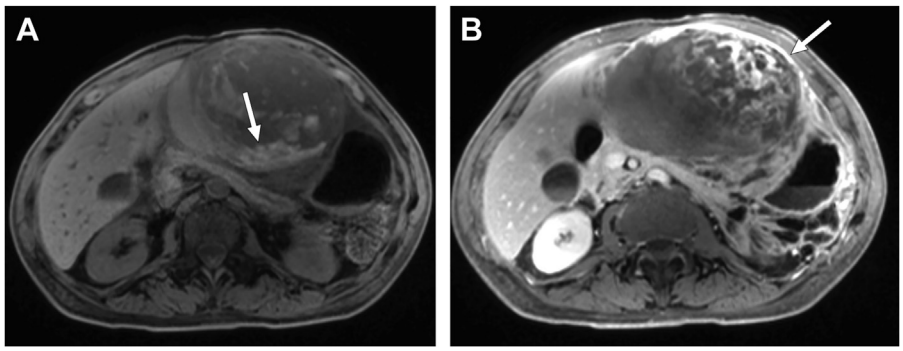

Fig. 8. Gastrointestinal stromal tumor (GIST). (*A*) Axial T1-weighted precontrast MR imaging shows a large heterogeneous mass with T1 hyperintense intratumoral blood products (*arrow*), consistent with GIST. (*B*) Axial T1-weighted postcontrast image depicts heterogeneous enhancement with areas of hemorrhage and cystic degeneration/necrosis.

treatment approaches are largely extrapolated from colorectal carcinoma protocols, despite small bowel adenocarcinoma being a distinct clinical and molecular entity with often worse prognosis.[24,25]

Gastrointestinal Stromal Tumor

GISTs can be found throughout the GI tract, with approximately 25% originating in the small intestine, the second most common location after the stomach.[22] These mesenchymal tumors are the fourth most common small bowel malignancy. While most small bowel GISTs arise sporadically, some cases involve inherited familial predisposition.[22,26] Up to 30% of GISTs exhibit overtly malignant features or high malignant potential[26] and even low-risk GISTs have up to a 20% recurrence risk.[26] Consequently, experts stratify these tumors

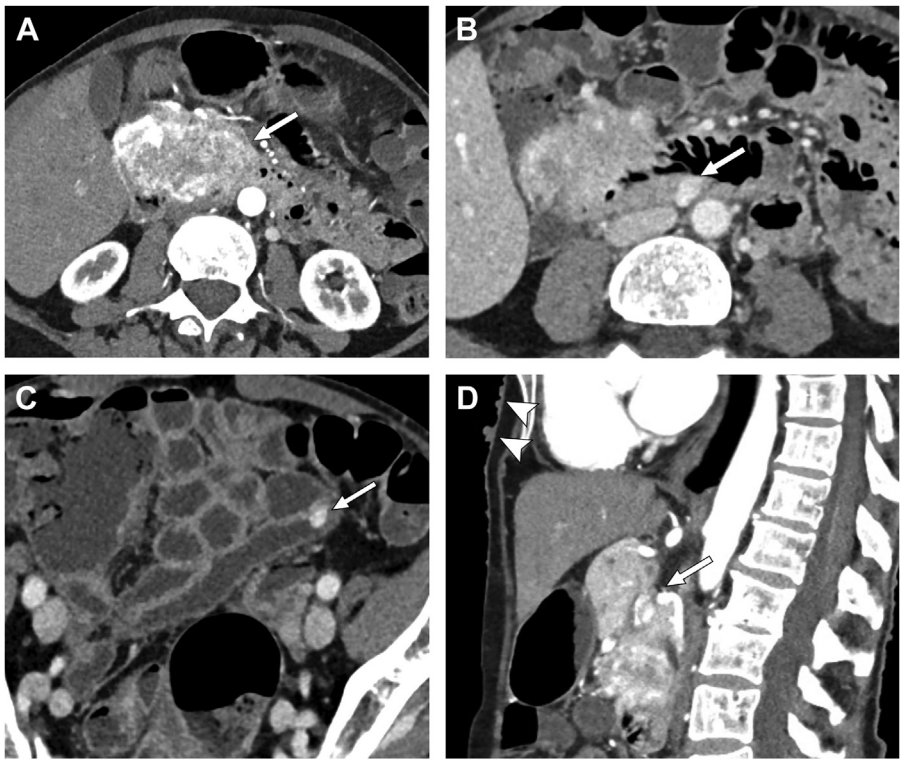

Fig. 9. Gastrointestinal stromal tumor (GIST). (*A–C*) Axial post-contrast CT images in a neurofibromatosis type 1 patient demonstrate multifocal small bowel GISTs (*arrows*). (*D*) Sagittal post-contrast CT shows a small bowel GIST (*arrow*) and multiple cutaneous neurofibromas (*arrowheads*).

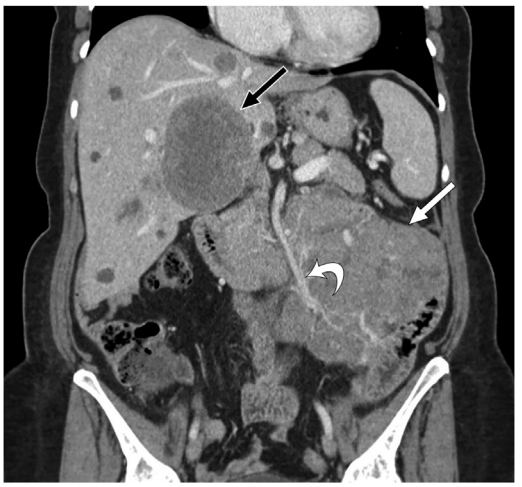

Fig. 10. Gastrointestinal stromal tumors (GIST). Coronal contrast-enhanced CT shows a large, predominantly exophytic mass (*arrow*), with prominent vein draining to the SMV ("tumor vessel sign", curved *arrow*) and multiple liver metastases (*black arrow*).

by malignant potential/behavior rather than classifying them as benign or malignant.[26] Tumor size and mitotic rate are independent prognostic factors used to predict aggressive behavior in small bowel GIST; smaller (especially <2 cm) tumors with low mitotic rate have the most favorable outcomes.[27]

Small bowel GISTs peak around at the age of 60 years.[26] They nearly universally overexpress the receptor tyrosine kinase protoncogene for tyrosine kinase receptor (KIT) (CD117), aiding diagnosis. Most KIT expressions result from *KIT* mutations,[28] though some GISTs that express KIT lack the *KIT* mutation, including those associated with neurofibromatosis type 1, platelet-derived growth factor receptor alpha (PDGFRA) mutations, and succinate dehydrogenase-deficient tumors. These *KIT* mutation-lacking tumors (approximately 15%) respond poorly to imatinib therapy.

GISTs originate in the submucosal muscular layer from the interstitial cells of Cajal and can exhibit endophytic, intramural, exophytic, or mixed endo/exophytic growth patterns (**Fig. 7**A–C). Mucosal ulceration can lead to early detection of small tumors, but up to 54% of small bowel GISTs are subserosal with a predominantly exophytic morphology.[21,22] Since these exophytic lesions do not cause obstruction, they are often asymptomatic and discovered incidentally. However, they can grow large, eventually causing symptoms due to their mass effect. Even large exophytic tumors may be missed at endoscopy if mucosal ulceration is not present.[27]

Imaging shows small GISTs as round, smoothly marginated, and homogeneously hyperenhancing tumors, while larger GISTs appear as lobulated lesions with less avid and more heterogeneous enhancement due to necrosis, hemorrhage, or degeneration (**Fig. 8**A, B).[21] GISTs of any size can ulcerate, and in larger tumors, this can result in cavitation, fistulization with the bowel lumen, or rupture.[21,27] Calcification is more common in larger tumors.[21] Genetically linked GISTs are more often multifocal with an increased incidence of lymph node metastases and unpredictable behavior (**Fig. 9**A–D).[28–30]

Determining the site of origin of large exophytic GISTs can be challenging, but the "tumor vessel sign" (**Fig. 10**), characterized by conspicuous vessels traceable from the tumor to a named vessel, and/or an enlarged, early draining vein visible during the arterial phase of enhancement, can assist in diagnosis and localization.[21]

GISTs primarily metastasize to the liver and omentum/peritoneum, with nodal metastases being rare.[21] The absence of lymphadenopathy despite hepatic or peritoneal metastases can be a diagnostic clue. Most liver metastases demonstrate

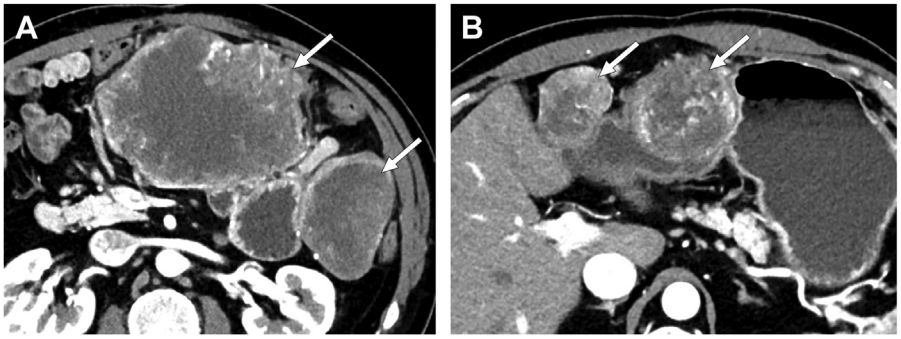

Fig. 11. Gastrointestinal stromal tumors (GIST). (*A* and *B*) Axial contrast-enhanced CT reveals large peritoneal metastases with round/lobulated morphology. Peritoneal disease can be extensive without ascites.

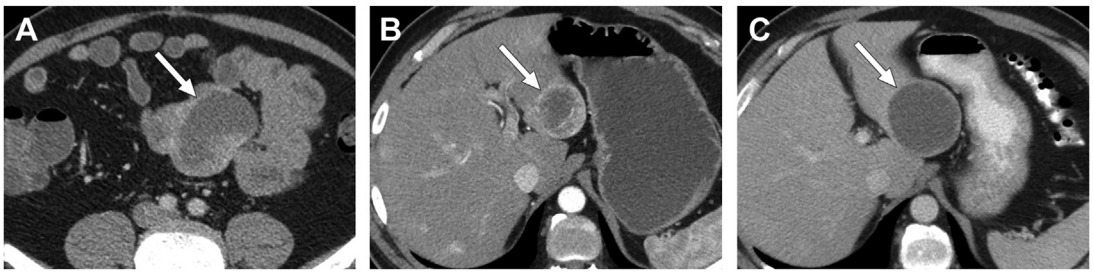

Fig. 12. Jejunal GIST and metastasis. (*A* and *B*) Axial contrast-enhanced CTs demonstrate a jejunal GIST (*A*, *arrow*) with liver metastasis (*B*, *arrow*). (*C*) Follow-up CT after imatinib treatment shows increased size but decreased enhancement ("pseudoprogression").

hypervascularity on arterial phase imaging with portal venous equilibration or washout, highlighting the need for multiphasic post-contrast imaging, and they can centrally necrose. On MR imaging, the liver metastases are T2 hyperintense and restrict diffusion.[21] In contrast to peritoneal carcinomatosis, peritoneal sarcomatosis from GIST presents as bulky rounded masses (**Fig. 11**A, B) without ascites or organ obstruction.[31] Oral contrast may increase the conspicuity peritoneal metastases.

Primary GISTs 2 cm or greater require surgical resection, while smaller GISTs can be resected or monitored for stability. Targeted molecular therapy with tyrosine-kinase inhibitors like imatinib has significantly improved outcomes, even with high-risk, metastatic, or recurrent disease.[32] GISTs often respond to molecularly targeted therapy with limited volume reduction but with decreased central enhancement (**Fig. 12**A–C). Some treated GISTs and their metastases can even be mistaken for cysts if not compared with pretreatment examinations. As such, response evaluation criteria in solid tumors (RECIST), which only considers tumor diameter, has limited utility in assessing response

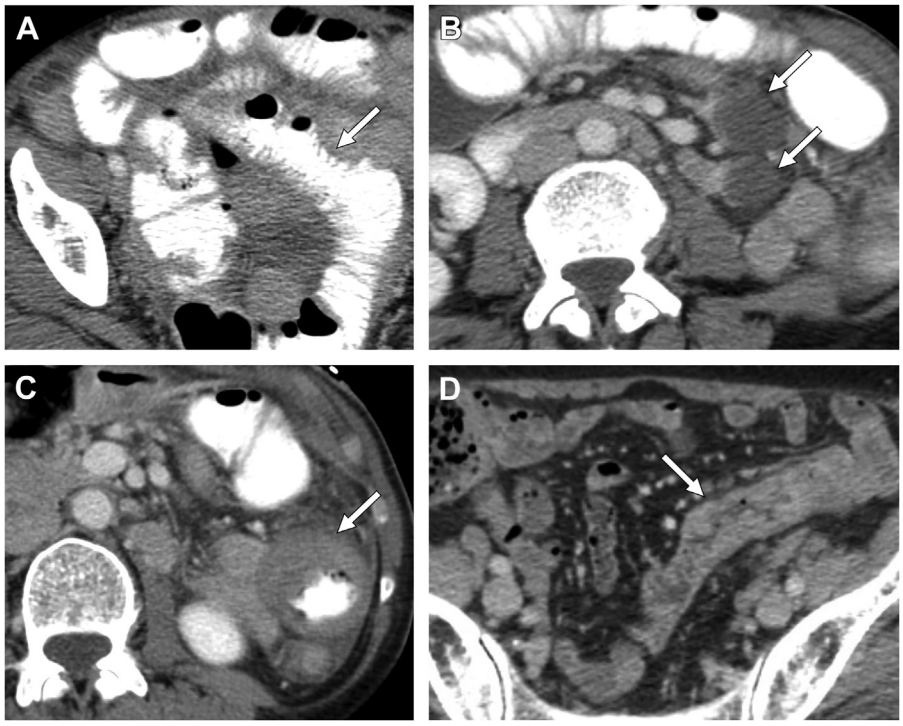

Fig. 13. Enteropathy-associated T-cell lymphoma (EATL) in celiac disease. (*A–C*) Axial contrast-enhanced CT images show jejunization of the ileum (*A*, *arrow*), cavitary mesenteric lymphadenopathy (*B*, *arrows*), and mildly enhancing circumferential jejunal mass indicative of EATL (*C*, *arrow*). (*D*) Axial contrast-enhanced CT in a different patient with EATL arising within ulcerative enteritis (*arrow*), which may be a precursor for EATL.

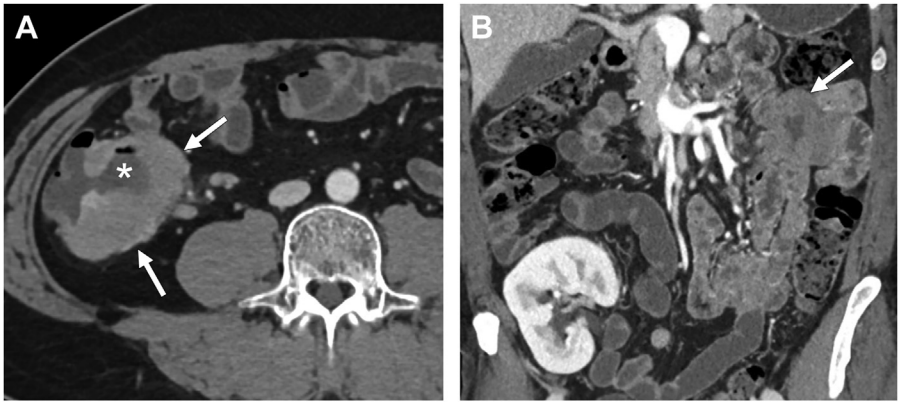

Fig. 14. Lymphoma in posttransplant lymphoproliferative disorder (PTLD). (*A*) Axial contrast-enhanced CT reveals a mildly enhancing, bulky ileal mass (*arrows*) with central ulceration (*asterisk*), consistent with B-cell lymphoma in a heart transplant recipient. (*B*) Coronal CT enterography shows a jejunal mass with central ulceration (*arrow*), consistent with diffuse large B-cell lymphoma secondary to PTLD (note right lower quadrant renal allograft).

for GISTs. The Choi criteria address this limitation by including tumor attenuation/enhancement as an additional measure of response. This system also helps avoid misdiagnosis of progression due to lesion enlargement from intra-tumoral hemorrhage, necrosis, edema, or myxoid degeneration ("pseudo-progression") and aids in the early diagnosis of recurrence within a treated lesion, even when there is no increase in tumor size (eg, when progression manifests as a "nodule in a mass").[33] Due to limitations in standard size-based criteria in CT and MR imaging, PET is crucial for assessing tumor response, as it can be more sensitive in detecting early treatment responses.[33–35] Combining CT for morphologic assessment and FDG-PET for functional assessment is optimal, with a 50% reduction in SUVmax and standardized uptake value (SUV) of less than 2.5 predicting sustained response.[33]

Lymphoma

The GI tract is the most common site for extranodal lymphoma, accounting for 5% to 20% of cases.[36] Primary lymphomas of the GI tract are rare, representing only 1% to 4% of GI malignancies, with most being secondary to disseminated nodal disease. The small bowel is the second most common site of extranodal lymphoma after the stomach, with 60% to 65% involving the ileum, 20% to 25% the jejunum, and 6% to 8% the duodenum.[36] These are a heterogeneous group, but most are non-Hodgkin variants. The most common small bowel lymphoma subtypes in the United States include diffuse large B-cell lymphoma, mantle cell lymphoma, Burkitt lymphoma, follicular lymphoma, and enteropathy-associated T-cell lymphoma (EATL).[37] Similar to other small bowel tumors, lymphoma often presents with nonspecific symptoms such as abdominal pain, nausea, diarrhea, GI

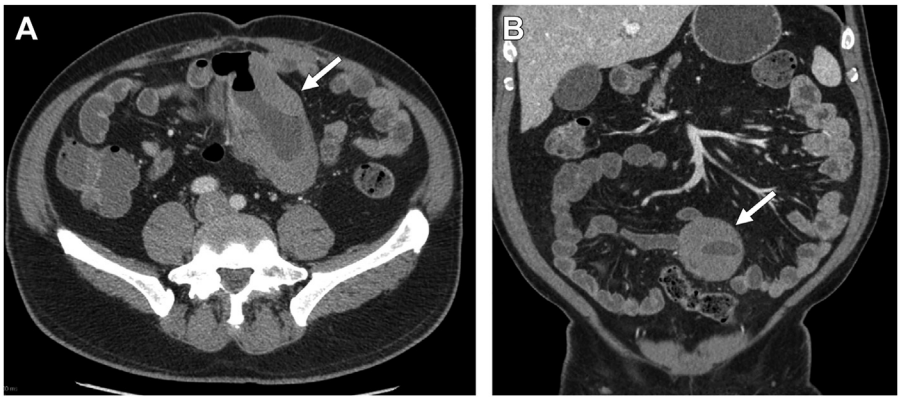

Fig. 15. Diffuse large B-cell lymphoma. (*A* and *B*) Axial and coronal CTE images demonstrate marked circumferential wall thickening and aneurysmal dilation in the ileum (*arrows*), characteristic of lymphoma.

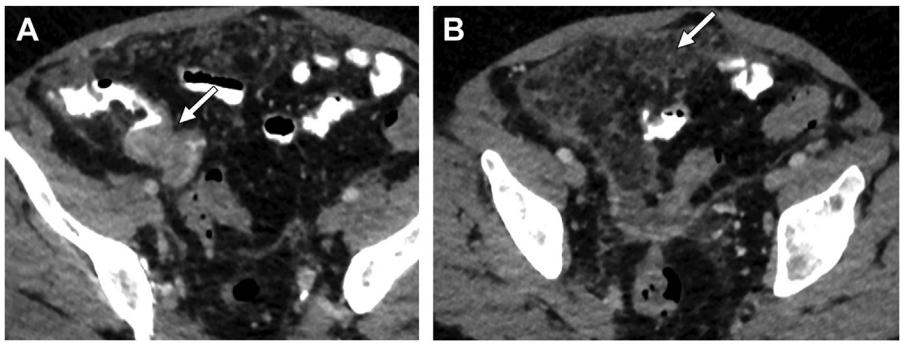

Fig. 16. Peritoneal lymphomatosis. Axial contrast-enhanced CT images demonstrate a soft tissue mass in the terminal ileum (*A*, *arrow*) and nodular infiltration of in the pelvic peritoneum (*B*, *arrow*), consistent with biopsy-proven small bowel lymphoma with peritoneal lymphomatosis. An identical appearance could be seen with small bowel adenocarcinoma and peritoneal carcinomatosis; tissue diagnosis is required.

bleeding, weight loss, and occasionally obstruction or perforation.[38]

Several conditions may predispose individuals to small bowel lymphoma. *Helicobacter pylori* infection is strongly associated with mucosa-associated lymphoid tissue (MALT) lymphoma of the stomach and, to a lesser extent, other GI tract sites. Immunodeficiency, both congenital and acquired, increases the risk of B-cell lymphoma, which tends to be aggressive and widespread at diagnosis.[36] Celiac disease, especially severe, refractory, or long-standing disease, is linked to an increased risk of EATL (**Fig. 13**A–D), which carries a poor prognosis, and possibly B-cell lymphoma.[39] Inflammatory bowel disease may be linked to Epstein–Barr virus-associated B-cell lymphoma, although data are conflicting. Posttransplant lymphoproliferative disorder is another risk factor for B-cell non-hodgkin's lymphoma (NHL) (**Fig. 14**A, B).[36]

Imaging features of primary and secondary GI lymphomas are identical. Cross-sectional imaging may show an infiltrative mass, polypoid lesions, ulcers, multiple nodules, or an endoexoenteric mass, which may be associated with cavitation and/or fistulization with the bowel lumen. The most characteristic appearance is the infiltrative phenotype, often with bulky mural thickening, mild homogeneous enhancement, and aneurysmal dilatation of the lumen due to infiltration of the muscularis propria and myenteric plexus (**Fig. 15**A, B). Bowel obstruction is uncommon due to the lack of desmoplastic reaction.[36]

Radiologic findings do not always correlate with pathologic subtypes, but some unique features are notable. Multifocal polypoid lesions or nodules, known as lymphomatous polyposis, are a rare presentation seen with mantle cell lymphoma, most prevalent in the jejunum and terminal ileum.[40] Follicular lymphoma may also present with multiple small polypoid lesions, especially in the proximal duodenum,[40] and MALT lymphoma can present similarly. Polypoid lesions can cause intussusception

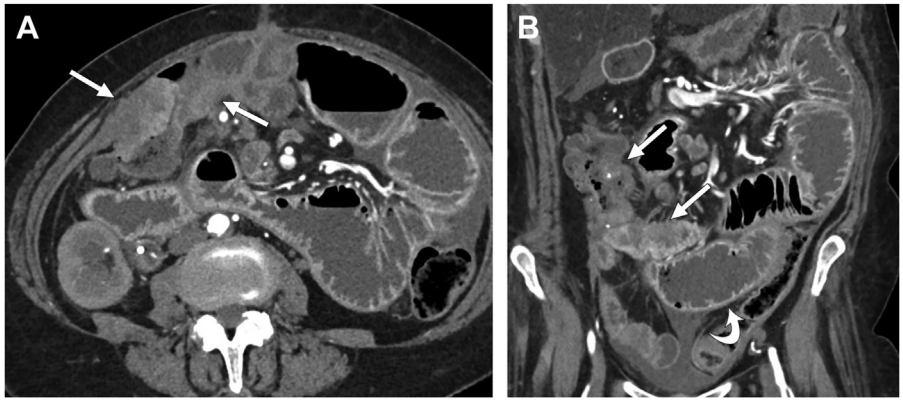

Fig. 17. Small bowel metastases, intraperitoneal spread. (*A* and *B*) Axial and coronal contrast-enhanced CT images reveal extensive implants on the small bowel surface (*arrows*) due to metastatic cecal adenocarcinoma, causing high-grade mechanical obstruction (*B*, curved *arrow*).

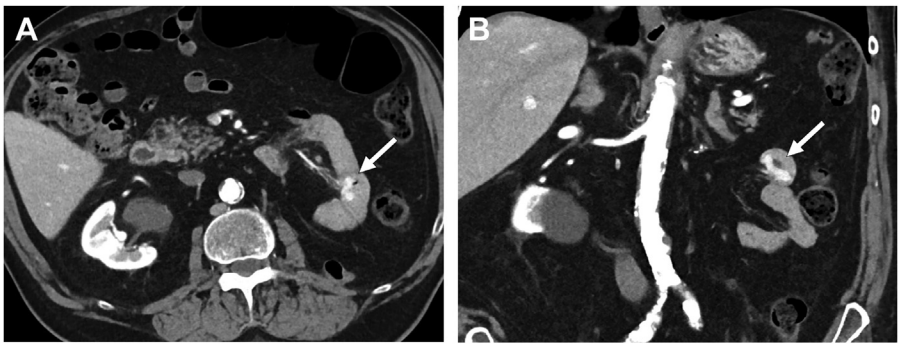

Fig. 18. Hematogenous metastasis to the small bowel. (*A* and *B*) Axial and coronal contrast-enhanced CT images show a hyperenhancing polypoid jejunal metastasis (*arrows*) in a patient with metastatic renal cell carcinoma. Small bowel metastases variably enhance but can parallel the enhancement of the primary tumor, as in this example.

and cause bowel obstruction. EATL typically affects the proximal jejunum or is diffuse, characterized by circumferential ulcers or strictures without bulky tumor mass (see **Fig. 13**D), and has a greater tendency for perforation due to angioinvasion.[40]

Regional lymph node enlargement is common in small bowel lymphoma and may be bulky, sometimes displaying the "hamburger sign," where nodal masses (bun) surround vessels (meat).[41] PET scans show variable FDG activity depending on the pathologic subtype.[42] Differentiating small bowel lymphoma from adenocarcinoma can be challenging. Adenocarcinomas are more commonly proximal, whereas lymphomas are often distal,

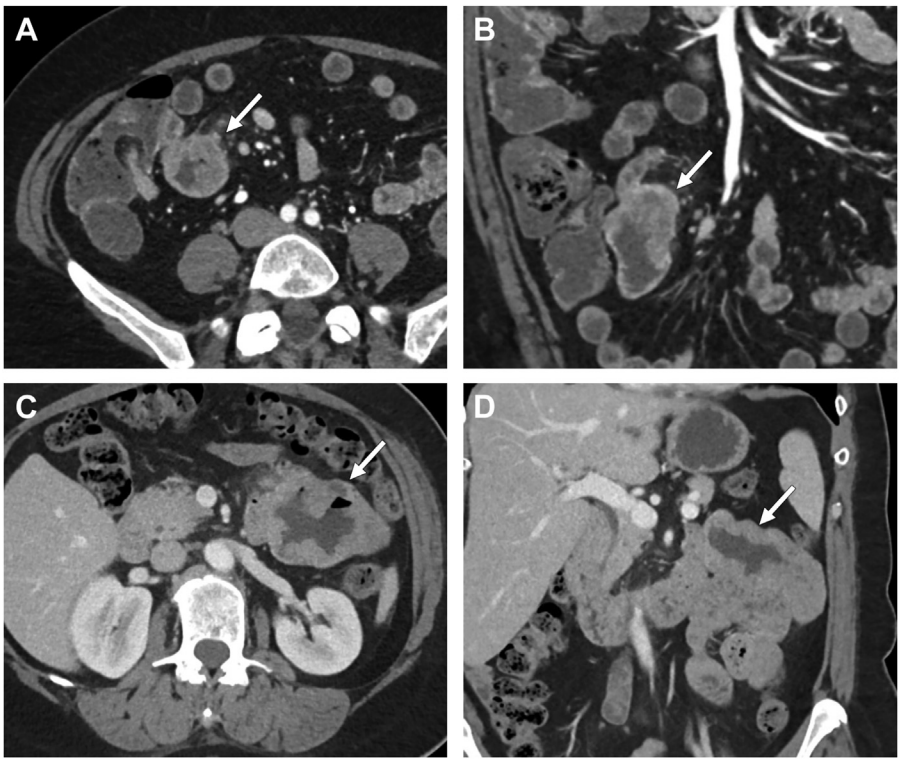

Fig. 19. Hematogenous metastases to the small bowel. (*A* and *B*) Axial and coronal contrast-enhanced CT images display a semi-annular ileal melanoma metastasis (*arrows*). (*C* and *D*) Axial and coronal contrast-enhanced CT images show irregular/nodular jejunal thickening and luminal dilatation without obstruction, similar to lymphoma (*arrows*).

though Crohn's-associated adenocarcinoma in the ileum and proximal small bowel EATL in patients with celiac (see **Fig. 13**) are notable exceptions.[43–45] Multifocality, bulky lymphadenopathy, especially extraperitoneal, and splenomegaly favor lymphoma over primary adenocarcinoma. Peritoneal lymphomatosis (**Fig. 16**A, B) occurs but is rare due to the peritoneum's relative lack of lymphoid tissue. Its imaging features resemble those of peritoneal carcinomatosis, but ascites is more common in peritoneal carcinomatosis. When present, peritoneal lymphomatosis usually accompanies enlarged mesenteric lymph nodes, bowel involvement, and other intra-abdominal organ involvement.[46] Ultimately, diagnosing lymphoma and distinguishing subtypes require tissue sampling for a definitive diagnosis.

Metastases

Metastases in the small bowel are more common than primary tumors and can occur through intraperitoneal seeding, hematogenous routes, lymphatic spread, or direct extension/invasion. Small bowel metastatic disease lacks distinct imaging features, with lesions varying in number, size, and morphology.[1,47]

Intraperitoneal spread, the most common mechanism, usually arises from GI and ovarian primary malignancies. It appears as nodular or plaque-like serosal deposits (**Fig. 17**A, B) or as eccentric or annular thickening of the bowel wall when mural invasion occurs. Additional synchronous peritoneal deposits typically appear in the dependent pelvis, paracolic gutters, small bowel mesentery, and/or omentum.[48] Carcinomatosis implants involving the small bowel can cause obstruction.[1] Hematogenously spread metastases primarily arise from lung cancer, breast cancer, melanoma, and renal cell carcinoma.[47] Enhancement of these lesions is variable but may mirror the primary tumor (**Fig. 18**A). Hematogenous metastases may present as unifocal or multifocal polypoid intraluminal lesions (**Fig. 18**B), intramural nodules, or short segments of circumferential or eccentric mural thickening (**Fig. 19**A–D). Initially intramural, these metastases can ulcerate the overlying mucosa, causing bleeding. They can also spread longitudinally through the submucosa, leading to luminal narrowing and obstruction. Intraluminal polypoid lesions may serve as a lead point for intussusception.[47] Direct invasion most often arises from ovarian or intra-abdominal GI malignancies.[1] Complications of small bowel metastases include obstruction, intussusception, and perforation. There is often evidence of metastatic disease elsewhere, and the primary tumor is frequently known at the time of diagnosis.[1]

SUMMARY

Advancements in imaging techniques, particularly CT and MRE, have significantly improved the evaluation of small bowel tumors. These modalities provide detailed evaluations that enhance detection, leading to earlier and more accurate diagnoses. Understanding the histopathology of the small bowel and its tumors is crucial for understanding their imaging appearance. Radiologists must be well versed in the imaging characteristics of the 5 most common small bowel malignancies—NEN, adenocarcinoma, GIST, small bowel lymphoma, and metastases—along with their associated ancillary features and clinical behaviors. This knowledge is essential for providing critical and accurate staging information for treatment planning and therapeutic monitoring, thereby offering the most value for clinicians.

ACKNOWLEDGMENTS

The authors acknowledge the assistance of Sonia Watson PhD, in preparation of the manuscript.

DISCLOSURES

The authors have no disclosures or financial interests to declare.

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