Small Bowel: The Last Stronghold of Gastrointestinal Radiology

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Learning Objectives

- To become familiar with small bowel key imaging modalities and the clinical considerations in choosing the appropriate study
- To identify imaging signs of small bowel inflammation for diagnosis and characterization of the main causative entities
- To recognize radiological characteristics of the various benign and malignant small bowel neoplasms
- To learn about signs of mesenteric ischemia imaging and resulting ischemic bowel damage

18.1 Imaging Techniques

As direct endoscopic visualization of most of the midgut proximal to the ileocecal valve is impossible, radiological imaging plays a vital role in assessing and diagnosing pathology of the small bowel. The optimal choice of imaging technique depends on the clinical setting, the presumptive differential diagnosis, whether the patient presents as an outpatient or in the acute setting, and individual patient characteristics, such as age and ability to cooperate.

The two major cross-sectional imaging techniques for small bowel evaluation are computed tomography (CT) and magnetic resonance imaging (MRI), each utilizing various acquisition protocols. The appropriate selection between these techniques has been a subject of interest in multiple studies, as each has characteristic advantages and disadvantages. For example, a main concern of CT imaging is repeated exposure of patients to ionizing radiation, which is particularly a concern in young patients, as frequently occurs in inflammatory bowel disease (IBD). Additionally, CT lacks visualization of superficial lesions and thus, may be inferior in detecting subtle mucosal disease [1]. Conversely, MRI may be more sensitive for identifying mild inflammation, in differentiating inflammation from fibrosis and additionally, may be used to assess motility with cine sequences. Disadvantages of MRI include a higher prevalence of motion artifacts than in CT, which affect image quality and thus, sensitivity, especially with uncooperative patients and in the acute setting. Other advantages of contrast-enhanced CT over MRI in the setting of an acute abdomen include availability, rapid acquisition, superiority in the assessment of certain complications such as perforation or bowel obstruction and the ability to rapidly screen for a wide range of pathologies in and outside the small bowel [2]. In addition, in clinical practice CT is the imaging workhorse for abdominal symptomatology and frequently the first diagnostic test performed. Thus, familiarity with small bowel CT findings is, in many cases, the initial key to diagnosis and in suggesting more specific tests such as CT-E, MR-E, CT Angiography (CT-A) and MR Angiography (MR-A).

Indeed, the widespread use of cross-sectional imaging has obviated the need for radiographic enteroclysis, in which contrast material is injected through a naso-jejunal tube to distend the small bowel, which is then imaged fluoroscopically. The disadvantages of this technique include indirect visualization of the bowel wall, inability to assess extramural pathology and the bowel loop overlapping, limiting the study. Thus, CT and MR-enteroclysis have largely replaced radiographic enteroclysis. Currently, the prevalent imaging technique to depict the small bowel is either CT or MR enterography (CT-E, MR-E), which utilizes orally

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administered contrast rather than inserting a naso-jejunal tube to distend the bowel. Though enterography results in reduced luminal distention compared to enteroclysis, especially of the jejunum, this technique is less invasive than enteroclysis and thus, results in better patient acceptance [2]. Notwithstanding, the initial imaging study in the acute setting is often contrast-enhanced CT rather than CT-E, due to its rapid acquisition and reduced patient ability to ingest a large volume of contrast material. Unless contraindicated, intravenous contrast is always administered for characterization of mural and lesion enhancement patterns. Additionally, spasmolytic agents may be administered shortly before imaging to reduce motion artifact.

In some centers, intestinal ultrasound (IUS) is frequently used to image the small bowel in chronic and acute settings, with color Doppler being especially informative in assessing mural vascularity and inflammatory activity [3, 4]. Other available imaging modalities for the assessment of specific small bowel pathologies include capsule endoscopy, balloon-assisted enteroscopy, and elastography [5].

Key Point
The two major cross-sectional imaging modalities for the evaluation of the small bowel are MR-E and CT-E, with CT having a central role in the emergency setting.

18.2 Inflammatory and Infectious Diseases

18.2.1 Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) consists of two entities: Crohn's disease (CD), which can involve any part of the gastrointestinal tract though most commonly the terminal ileum, and ulcerative colitis (UC), which is limited to the colon, along with various extraintestinal manifestations in both conditions. CD is characterized as a transmural, granulomatous inflammatory disease of uncertain etiology. The diagnosis of CD is made based on combined clinical, radiologic, endoscopic, and histologic findings demonstrating discontinuous transmural inflammation of the gastrointestinal tract. Complications of CD include inflammatory and/or fibrotic strictures, which may manifest as complete or partial bowel obstruction as well as abscesses, perforation, fistulae and, after long-standing disease, an increased risk for malignancy. In Europe and North America, the diagnosis, staging, and management of CD constitute the most common clinical scenarios necessitating imaging of the small bowel [5].

Determining the extent of disease and surveillance of disease activity in CD is heavily reliant on cross-sectional imaging. Importantly, radiological response to treatment is associated with better long-term outcomes in patients and may be used as a target for treatment [6]. Imaging findings indicative of active small bowel inflammation include segmental mural hyperenhancement, which may be homogenously transmural, asymmetric, or involving only the inner wall resulting in a "halo sign". Other findings are bowel wall thickening above a normal 3 mm, intramural edema seen on fat-saturated T2-weighted MRI, ulcerations which appear as focal discontinuity in the intraluminal surface, diminished motility on cine MRI sequences, and inflammatory strictures with upstream luminal dilatation >3 cm (Fig. 18.1). Diffusion-weighted MR imaging may also be helpful in identifying areas of active inflammation, though diffusion restriction should not be used as a sole indicator of active disease. Penetrating disease may manifest as fistulas, which are categorized as simple or complex depending on whether there are single or multiple extra-enteric tracts. Complex fistulas often appear as a "clover leaf" or "star sign" involving multiple bowel loops or adjacent organs. Fibrofatty proliferation or "creeping fat" is seen as increased fat along the mesenteric border of abnormal bowel. The characteristic "comb sign" denotes engorged vasa recta that supply inflamed bowel loops, though it may also reflect past inflammation (Fig. 18.2). Inflammatory masses, abscesses, perienteric edema, acute or chronic mesenteric vein thrombosis, and mesenteric adenopathy are additional imaging findings consistent with penetrating disease [7]. It is important to remember that signs of bowel inflammation are nonspecific to CD and careful clinical correlation is needed to exclude other causes, including infectious etiologies such as yersiniosis.

Accurately determining disease activity based on imaging and reporting findings in a clinically useful manner is highly challenging. Consensus recommendations for the use and interpretation of CT-E and MR-E studies in small bowel CD have been established by the American Gastroenterological Association [7].

Key Point
Major imaging findings in active small bowel inflammation include mural hyperenhancement, engorged vasa recta, bowel wall thickening above 3 mm, intramural edema, ulcerations, diminished motility and inflammatory strictures with upstream luminal dilatation >3 cm.

18.2.2 Celiac Disease

Celiac disease is a maldigestion syndrome due to the aberrant recognition of gliadin, a component of gluten, as an immunogenic agent in genetically predisposed individuals. This trig-

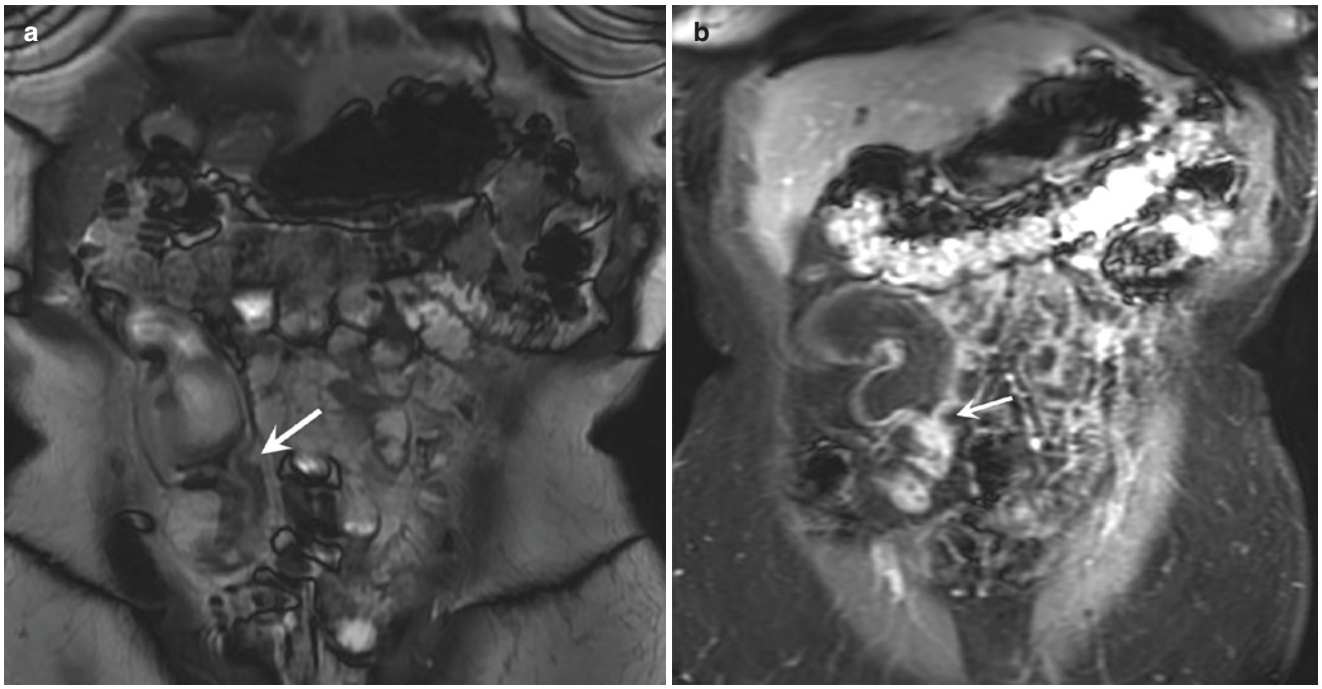

Fig. 18.1 MR-E in Crohn's disease. Coronal T1 pre- (**a**) and T1 post-contrast (**b**) MR-E images demonstrate an enhancing stricture (arrow) with upstream bowel dilatation in the terminal ileum

gers an antibody and cell-mediated response toward the villi and the intestinal mucosa, leading to severe mucosal deficit. Clinically, celiac disease is diverse, though typical symptoms include diarrhea, abdominal pain, steatorrhea, weight loss, vomiting, and manifestations of various nutritional deficits including iron-deficiency anemia. Hyposplenism, neuropsychiatric symptoms and dermatological manifestations, classically dermatitis herpetiformis, are also possible [5]. Symptoms usually abate with cessation of gluten ingestion. Long-standing, refractory disease may progress to enteropathy-associated T-cell lymphoma (EATL) or small bowel adenocarcinoma. Due to varying and atypical presentations, diagnosis may be challenging and is often delayed, as it primarily relies on obtaining biopsies of affected portions of the duodenum. These reveal characteristic histological findings of villous atrophy, crypt hyperplasia, and inflammatory cell infiltrate of the lamina propria. Medical imaging plays a supporting role in the timely diagnosis and management of this disease [5].

It is key to recognize radiological signs suspicious for celiac disease, so further diagnostic testing can be pursued. Fluoroscopic guided small bowel follow-through previously constituted the main imaging test for celiac disease by depicting small bowel malabsorption pattern (MABP) [8]. Features of MABP include excess fluid secretion resulting in multiple dilated, fluid-filled loops with reversal of the jejunal-ileal fold pattern ("moulage sign"), laminar flow of contrast due to decreased peristalsis, dilution, and flocculation of contrast material, and telescoping of bowel loops with transient intussusception. Of these signs, small intestinal fold pattern alterations, reflective of underlying villous atrophy, are the most specific sign for celiac disease and its depiction necessitates adequate distention of the jejunum [9]. As the use of fluoroscopic barium examinations has declined, cross-sectional imaging often constitutes the initial radiological assessment of abdominal complaints. CT-E and MR-E are both capable of depicting MABP of the small bowel and additional findings of active inflammation in the bowel wall and mesentery. These include mesenteric lymphadenopathy, proximal bowel wall thickening with or without submucosal fat deposition, and a hypervascular, engorged mesentery. Splenic atrophy is present in 30–60% of patients [10]. Refractory celiac disease (RCD), which develops in 2–10% of patients, can result in life-threatening complications including cavitary mesenteric lymph node syndrome, ulcerative jejunoileitis, EATL, adenocarcinoma and an increased risk for other gastrointestinal malignancies [9].

18.2.3 Graft Versus Host Disease

Graft versus host disease (GVHD) is an immune dysfunction most commonly following allogenic bone marrow transplants, though it may occur following transplantations of any organ rich in lymphocytes or after blood transfusions. In this disorder, competent donor lymphocytes attack recipient tis-

Fig. 18.2 Enhanced CT in Crohn's disease. Axial (**a**) and coronal (**b**) enhanced CT images of a long segment of distal ileum with bowel wall thickening, mesenteric fat stranding, and engorged vasa recta ("comb sign," arrow) in keeping with active inflammation

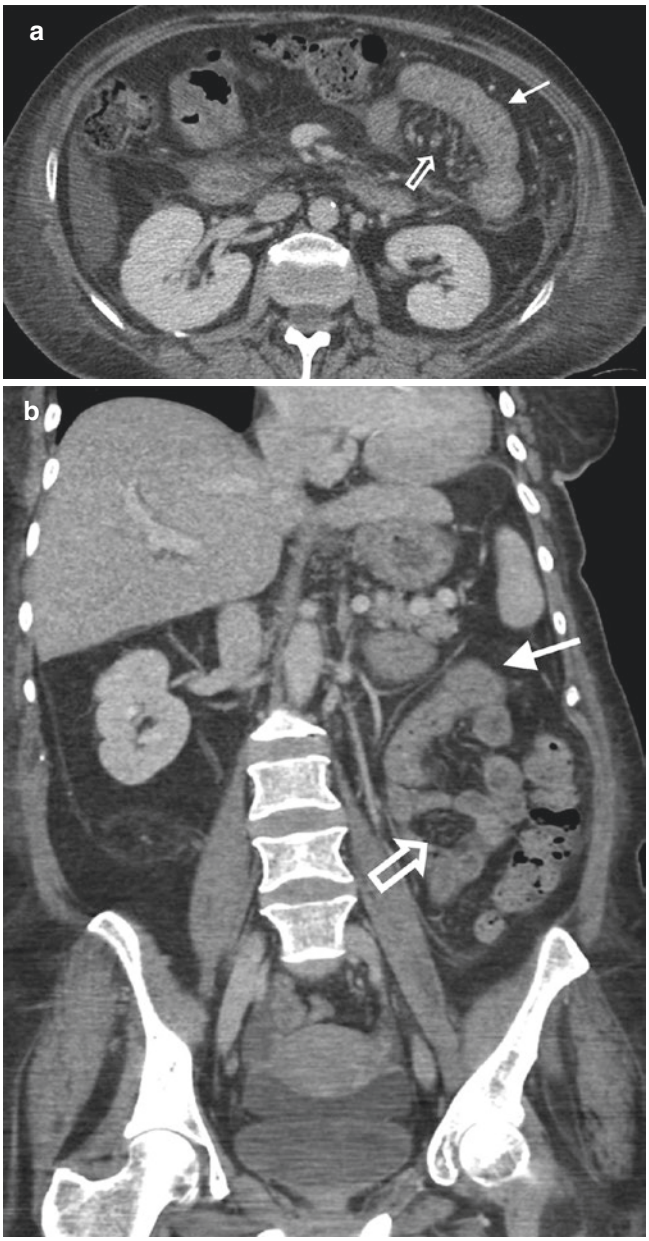

Fig. 18.3 Graft versus host disease. Axial (**a**) and coronal (**b**) enhanced CT images of a patient with GVHD, displaying thickened, edematous bowel wall (solid arrow) or normal caliber small bowel loops with engorged mesenteric vessels (hollow arrow)

sue, leading to inflammation and tissue destruction. Moderate to severe GVHD occurs in 30–50% of patients undergoing matched allogenic bone marrow transplants. Small bowel involvement is ubiquitous [11]. Symptoms include watery diarrhea, ileus, fever, and abdominal pain [5].

Typical imaging findings are nonspecific signs of bowel inflammation, including a thickened, enhancing bowel wall, engorged vasa recta, fluid-filled bowel loops, and mesenteric fat stranding (Fig. 18.3). The extent of involved bowel tends to be greater in GVHD than in IBD. It is critical to differentiate GVHD from infection, as treatment of GVHD involves immunosuppressive agents. As such, biopsies are often needed to confirm this diagnosis [5].

18.2.4 Infections

Infection remains an important etiology in the differential diagnosis in all cases of small bowel inflammation. Acute gastroenteritis, most frequently of viral etiology, is a common illness resulting in many emergency department visits [12]. While most cases are self-limiting, infectious enteritis occasionally requires targeted treatment and hospitalization. Moreover, it is often crucial to exclude infectious enteritis before treatment of other suspected inflammatory processes. Imaging findings are nonspecific and usually require additional testing, such as biopsy, to confirm the diagnosis (Fig. 18.4).

A variety of atypical pathogens can cause enteritis:

1. Tuberculous enteritis is found in Asia and Africa and most commonly involves distal ileal and ileocecal areas. Patients typically present with constitutional symptoms. Radiologically, it may appear as an ileocecal mass with deformity of the cecum and low attenuation mesenteric lymphadenopathy, though it cannot be reliably distinguished from CD or lymphoma.
2. Yersiniosis is an infectious disease presenting radiologically as a mild terminal ileitis with ulceration, lymphoid hyperplasia, wall thickening and increased, stratified enhancement. It is a cause of mesenteric adenitis in children and may mimic acute appendicitis. Yersiniosis may mimic radiologically mild CD.
3. Actinomycosis may cause clinical infection in predisposed individuals. This presents as a chronic, progressive, granulomatous infection with suppurative inflammation with sinus tracts and fistulation. The radiological appearance may mimic small bowel adenocarcinoma.
4. Whipple's disease is a rare bacterial infection caused by *Tropheryma whipplei*, which may infect any organ though always involves the small bowel. Imaging demonstrates thickening of the valvulae conniventes, nodularity of the duodenum and jejunum and possible bowel dilatation and characteristically low attenuation mesenteric lymphadenopathy (Fig. 18.5).
5. Infestations with helminths and other parasites are prevalent in tropical and subtropical areas. Strongyloidiasis and Giardiasis may demonstrate non-specific bowel wall thickening on imaging. Heavy infestations with Ascariasis lumbricoides may present with obstructive symptoms. Chagas' disease is a chronic condition caused by infection with *Trypanosoma cruzi* and can eventually damage the enteric nervous system resulting in dilatation and slow transit in any segment of the GI tract from the esophagus to the rectum. Small bowel involvement results in megaduodenum [5].
6. SARS-CoV-2, the causative virus of the COVID-19 pandemic, has been reported to cause gastrointestinal symptoms in a significant portion of patients [13]. Small bowel complications include ileus with persistent bowel dilatation, feeding intolerance and mesenteric ischemia displaying bowel wall thickening, pneumatosis, and even portal venous air on CT imaging. It is unclear whether these complications are SARS-CoV-2-specific or are indirect sequalae common in critically ill patients. Interestingly, the ACE2 receptor, through which the virus infects host cells, is heavily expressed on the brush border of the intestinal epithelium [14].

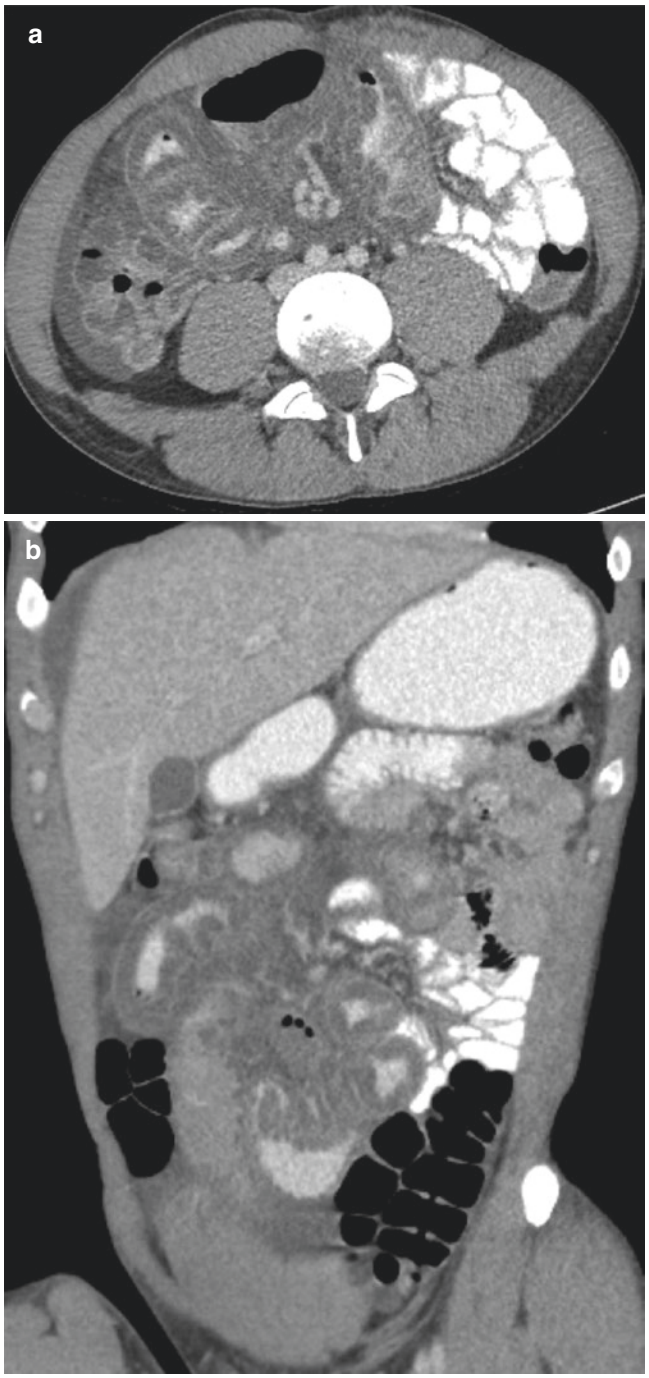

Fig. 18.4 Infectious gastroenteritis. Axial (**a**) and coronal (**b**) CT images of infectious gastroenteritis, with diffuse nonspecific markedly thickened, low attenuation small bowel loops with mesenteric fat inflammatory changes

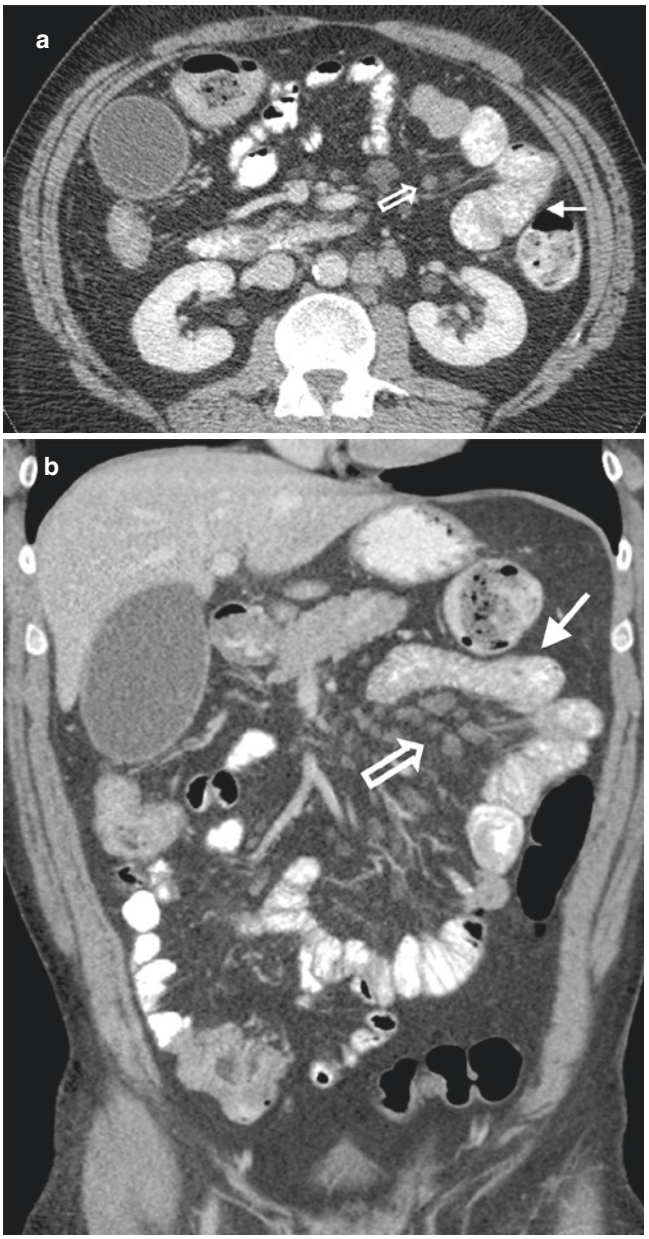

Fig. 18.5 Whipple's disease. Axial (**a**) and coronal (**b**) CT images in a patient with Whipple's disease, displaying characteristic nodularity of the duodenal and jejunal folds (solid arrows), along with multiple prominent low attenuation mesenteric lymph nodes (hollow arrows)

Key Point
It is critical to differentiate infection from other causes of bowel inflammation, as radiological signs may be similar and treatment as a non-infectious condition, for example with immunosuppressive agents, may result in exacerbation if the etiology is infectious.

18.3 Small Bowel Neoplasms

Despite constituting above 90% of the surface area of the gastrointestinal tract, small bowel neoplasms are relatively rare. Malignant tumors of the small intestine constitute only 3% of gastrointestinal cancers and 0.6% of all cancers in the USA [15]. For reasons unclear, the incidence of small bowel cancers is rising, particularly neuroendocrine tumors (NET) or carcinoid tumors.

Small bowel tumors are often clinically silent for long periods of time, and many are found incidentally during surgery or radiological exams performed for other reasons. Incidence is greatest in the proximal small bowel and decreases distally up to the terminal ileum. CT-E and MR-E have a central role in diagnosis and characterization of small bowel tumors [16].

18.3.1 Benign Neoplasms

Benign small bowel tumors are usually solitary, unless there is an underlying intestinal polyposis syndrome. Appearance on imaging is often that of a round and well-circumscribed intrinsic filling defect with smooth margins, single or multiple in polyposis. Adenomas and leiomyomas are the most common benign tumors of the small bowel and the only two with malignant predisposition. Rarer lesions include lipomas, vascular and neurogenic tumors, hamartomas, and heterotopias, which have no malignant predisposition [5].

Adenomas originate from glandular epithelium and may have a tubular, villous, or tubulovillous morphology, like in the colon. Tubular adenomas often appear on imaging as a solitary intrinsic filing defect with smooth margins, either sessile or pedunculated, while villous adenomas appear cauliflower-like and tend to be larger (>3 cm). Adenomas larger than 2 cm are routinely resected due to malignant potential. Adenomas are usually solitary unless there is an underlying polyposis syndrome. Multiple adenomas are typically of varying sizes and within a single bowel segment.

Lipomas are composed of mature adipose tissue arising from the submucosa and have no malignant potential. Less than 50% are symptomatic, and they may present with obstruction, bleeding, or manifest as the lead point for intussusception. Most small bowel lipomas arise in the ileum or in the duodenum. On imaging, they appear as a smooth, homogenous mass with fat attenuation on CT and no enhancement. On MRI they follow uniform macroscopic fat signals across all sequences. Symptomatic lesions may be resected.

Leiomyomas are the most common symptomatic benign small bowel neoplasm. These mesenchymal tumors, when in the small bowel, appear most frequently in the jejunum.

They are well circumscribed, homogenously enhancing soft tissue density masses on CT and MRI, which may calcify or ulcerate if large. Differentiation from GIST is not possible by imaging and requires histological analysis. Lesions larger than 6 cm, with irregular margins or associated lymphadenopathy, are suspicious for leiomyosarcoma. Symptomatic lesions, including those which present with obstruction, hemorrhage, and anemia, are surgically resected (Fig. 18.6).

Several non-neoplastic lesions present as mass-like on imaging, constituting a potential source of confusion. Small bowel diverticulitis, though rare, presents as a round, debris-filled mass-like structure with associated bowel wall thickening and mesenteric fat inflammatory changes. A Meckel diverticulum may appear as a mass-like blind-ended debris-filled pouch on the antimesenteric border of the distal ileum. An intramural hematoma appears as thickened, hyperattenuating bowel wall with possible luminal narrowing and obstruction and occurs in trauma or blood dyscrasias such as hemophilia.

18.3.2 Polyposis Syndromes

Polyposis syndromes include Familial Adenomatous Polyposis (FAP), and its variants Gardner and Turcot syndromes and Peutz-Jeghers syndrome, among others. Because many polypoid lesions are present, the risk of malignancy is greater. Patients may be symptomatic due to the polyps acting as lead points, causing intussusception. MR-E is the imaging exam of choice for the detection and characterization of multiple polyps.

FAP is an autosomal dominant condition manifesting with many premalignant colonic adenomas, with over 80% of patients also developing adenomas in the small bowel, most commonly in the periampullary region. Due to the near definite risk of developing colon cancer, patients undergo prophylactic proctocolectomy, though these patients are still at risk of developing small bowel malignancies. As it is not possible to remove all small intestine adenomas, only large ones are resected, and patients are monitored at regular intervals. Patients are also at risk of developing Gardner's syndrome, or mesenteric fibromatosis (locally aggressive desmoid tumors), which can infiltrate adjacent structures including the small bowel and cause obstruction.

Peutz-Jegher's syndrome is a rare, autosomal dominantly inherited condition with multiple hamartomatous polyps in the digestive tract, specifically the small bowel. In general, these lesions have much lower malignant potential than adenomas. Patients may suffer episodes of intermittent intussusception and bleeding. On CT or MRI, these lesions appear as smooth or lobulated, enhancing intrinsic filling defects in the small bowel lumen. This syndrome is also characterized by mucocutaneous perioral and genital melanin pigmentation, known as pluriorificial ectodermosis. Patients are also at increased risk of developing gynecological malignancies [5, 16].

Fig. 18.6 Leiomyoma. Axial (**a**) and coronal (**b**) CT images of a patient with a well-marginated, homogenously enhancing small bowel mass causing partial obstruction with mild proximal bowel dilatation (arrow). This lesion was ultimately diagnosed as leiomyoma on histology. Based on this imaging, the main differential diagnosis is GIST

18.3.3 Malignant Neoplasms

Imaging characteristics suggestive of a malignant lesion include irregular margins with heterogeneous enhancement, and invasion of adjacent structures. Due to nonspecific symptoms and low clinical suspicion, small bowel malignancies are often diagnosed at advanced stages and thus carry a poor prognosis [16]. Risk factors for primary malignancies include chronic inflammation, HIV infection and inherited conditions including hereditary nonpolyposis colorectal cancer (HNPCC), familial adenomatous polyposis (FAP) and Peutz-Jeghers syndrome. Metastatic lesions, most commonly from breast and lung cancer and melanoma, are more frequent than primary small bowel malignancy (Fig. 18.7).

For many years, adenocarcinoma constituted the most common histologic type of primary small bowel malignancy, though in recent years, it has been surpassed by neuroendocrine (carcinoid) tumors (NETs) [17].

Small bowel adenocarcinomas arise most commonly in the distal duodenum and proximal jejunum. On CT and MRI, they appear as an enhancing soft tissue density/intensity mass with possible luminal narrowing, either with eccentric or circumferential growth ("apple core" sign, like adenocarcinomas in the colon). They may present with vascular invasion, lymphadenopathy, peritoneal masses and obstruction. Adenocarcinomas, like other small bowel malignancies, often metastasize to the liver due to rich mesenteric venous drainage.

Gastrointestinal stromal tumor (GIST) originates from the interstitial cells of Cajal and is defined by its expression of KIT (CD117), a tyrosine kinase growth factor receptor. GISTs usually behave non-aggressively, though a minority display overtly malignant clinical behavior. GISTs often extend exophytically into the bowel lumen and the mesentery and may variably contain calcifications. On MRI, they display low T1 and high T2 signal intensity. It may be difficult to differentiate aggressive from non-aggressive GIST based on imaging alone. Non-aggressive lesions tend to be smaller than 5 cm, well circumscribed, have poor contrast enhancement and a low mitotic index on histology [5]. Aggressive lesions tend to be large and have a lobulated margin and heterogeneous enhancement, often with areas of necrosis and cavitation which may communicate with the bowel lumen. GIST may metastasize to the omentum, peritoneum, liver, or even extra-abdominally. Associated bulky adenopathy is rare with GIST. Since all GISTs are potentially malignant, they are considered for resection even if relatively small [16].

Gastrointestinal NETs arise from intraepithelial endocrine cells. 90% of small bowel NETs arise in the distal ileum and many are multifocal. Often the primary lesion is not visible on initial imaging, or it presents as a small intraluminal filling defect. More commonly, NET presents as a spiculated mesenteric mass eliciting a desmoplastic reaction, the result of regional metastasis. The tumor may have avid arterial contrast enhancement and contain calcifications. The tumor, along with the surrounding desmoplastic reaction, may cause small bowel obstruction or ischemia. Metastases to the liver may lead to carcinoid syndrome, manifesting with diarrhea, skin rash, sweating and, in severe cases, bronchospasm, flushing, and hypotension (Fig. 18.8).

Small bowel lymphoma may be primary (with no other lymphoma lesions) or part of systemic lymphoma at discov-

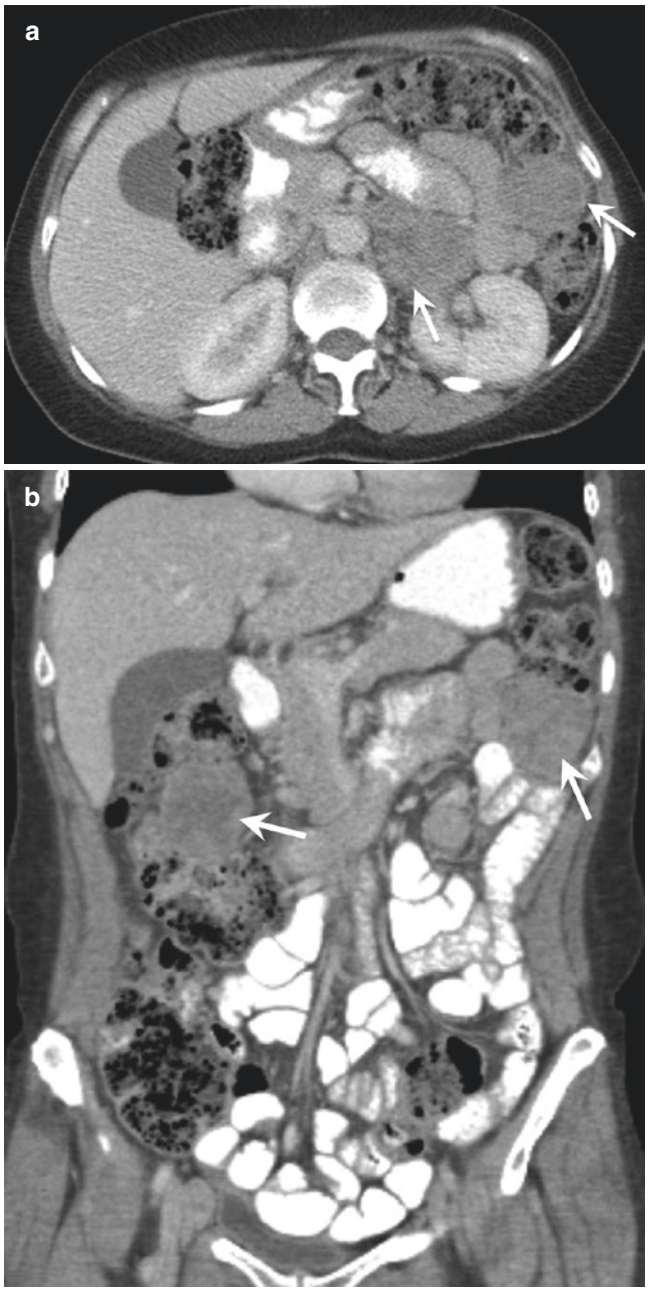

Fig. 18.7 Melanoma metastasis to small bowel. Axial (**a**) and coronal (**b**) CT images in a patient with metastatic melanoma, with multiple small bowel masses (arrows)

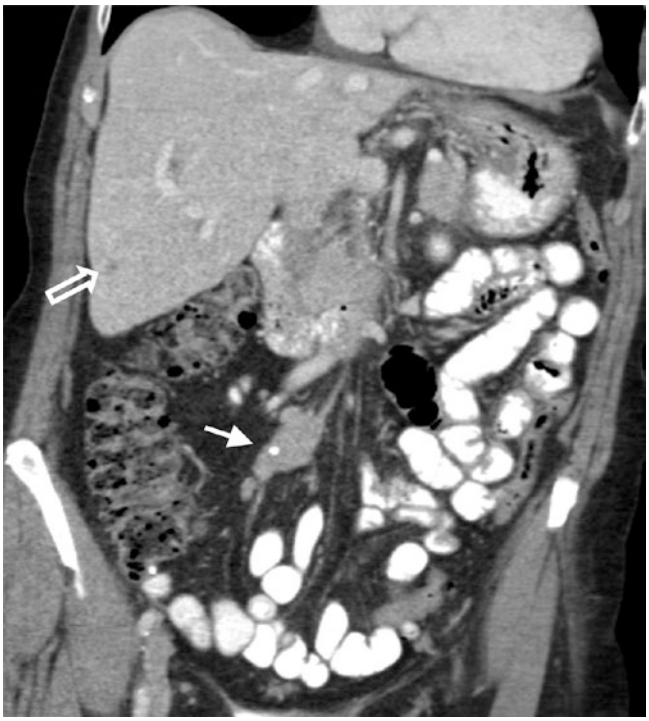

Fig. 18.8 NET. Coronal CT image of a patient with small bowel NET manifesting with a lobulated mesentery mass representing regional metastatic focus with internal calcifications (solid arrow). A liver metastasis at the inferior pole of the liver is partially visualized (hollow arrow)

Fig. 18.9 Coronal CT image of a patient with small bowel lymphoma after undergoing a kidney transplant. Note mass-like concentric thickening of the bowel wall with luminal narrowing

ery. When primary, the most common location is in the ileum due to abundant lymphoid tissue. There is often associated bulky adenopathy and multifocal disease. Lymphoma has multiple possible radiologic appearances, including mass-like wall thickening and dilatation (pseudoaneurysmal), a polypoid mass projecting into the lumen, a cavitary soft tissue mass (endoexoenteric) or with extension into the surrounding mesentery (Fig. 18.9). The stenosing form is rare and is frequently a complication of long-standing celiac disease. The most common form is pseudoaneurysmal. Due to its various presentations, lymphoma may be difficult to distinguish from other small bowel malignancies on imaging. A more distal location and multifocal involvement may assist in differentiating lymphoma from adenocarcinoma, while bulky adenopathy associated with lymphoma is not common in GIST.

Sarcoma is a relatively rare primary malignancy in the small bowel, constituting about 10% of small bowel cancers. The most common type is leiomyosarcoma, most frequently appearing in the jejunum. On CT and MRI, leiomyosarcoma appears as a large heterogeneously enhancing mass with central necrosis. The mass may cavitate and communicate with the bowel lumen. Radiological characteristics may overlap those of GIST [16].

Key Point

Benign characteristics of a small bowel tumor include a round and well-circumscribed intrinsic filling defect with smooth margins. Benign lesions include adenoma, lipoma, and leiomyoma. Malignant characteristics include irregular margins with heterogeneous enhancement and invasion of adjacent structures. Malignant tumors include adenocarcinoma, NET, malignant GIST, lymphoma, and sarcoma.

18.4 Mesenteric Ischemia

The superior mesenteric artery (SMA), which supplies the jejunum and ileum, is a large caliber vessel with a narrow origin, rendering it susceptible to embolic phenomena and occlusion. When collateral vascular pathways cannot compensate for SMA occlusion and the perfusion of the small bowel is compromised, mesenteric ischemia occurs [5]. Mesenteric ischemia may be acute or chronic. Prompt diagnosis and intervention are critical, particularly when the ischemia is acute, as delayed intervention often results in

catastrophic complications [18]. Since neither laboratory tests nor clinical examination is specific for mesenteric ischemia, imaging plays a critical role in its diagnosis. The gold standard imaging test for mesenteric ischemia is CT-A. Oral contrast material is usually not appropriate in acute cases, as it can interfere with detecting subtle changes in bowel wall enhancement, its administration can cause diagnostic delays and it is often not propagated well due to development of dynamic ileus and fluid-filled bowel loops.

Mesenteric ischemia manifests initially as mucosal injury, as the bowel mucosa is the most susceptible to vascular compromise, with progression in severity to transmural necrosis (bowel infarction), perforation, and peritonitis. Stricture formation and obstruction may occur with long-standing chronic ischemia [5].

The etiology of acute mesenteric ischemia is embolic occlusion in 40–50% of cases. Emboli may appear as high-attenuation findings in the SMA on non-contrast CT images or cause filling defects distally. In acute infarction, the diameter of the SMA is often enlarged with simultaneous reduction of the caliber of the SMV, causing reversal of the normal size relation between them. Contrast enhancement in the affected bowel wall is diminished or absent. As damage progresses, muscular tone is lost, and the bowel wall becomes progressively thinner as the bowel dilates as transmural infarction occurs ("paper thin wall"). If bowel thickening occurs, notably with a halo or target pattern on contrast images, it is usually due to reperfusion and is an encouraging sign. Conversely, intramural gas (pneumatosis intestinalis) and air in the mesenteric and portal venous system are highly suggestive of bowel infarction (Fig. 18.10). Visualization of extraintestinal gas is indicative of perforation, which also may be associated with mesenteric fat stranding and ascites. Hyperattenuation of bowel loops on non-contrast phases may be caused by hemorrhagic infarction.

Fig. 18.10 Axial enhanced CT image of a patient with mesenteric ischemia displaying distended, fluid-filled bowel loops and thin bowel walls, with pneumatosis intestinalis

Other causes of acute infarction include thrombus formation in a previously stenotic vessel, dissection, or arterial inflammation. Some cases, notably in thrombus formation within chronically diseased vessels, may have a more indolent course due to the development of vascular collaterals [18, 19]. An additional form of this condition is non-obstructive mesenteric ischemia (NOMI), in which systemic hypotension leads to vascular spasm of the mesenteric vessels. It is associated with low-flow states, such as cardiac insufficiency, severe trauma or, classically, patients undergoing hemodialysis [18]. Unlike in obstructive forms of mesenteric ischemia, in NOMI a main finding is narrowing origins of mesenteric branches and alternate dilatation and narrowing of intestinal branches, with discontinuous and segmental bowel involvement. Because distal branches of the SMA are difficult to visualize on CT, angiography has an important role in the diagnosis of this entity [19].

The vast majority (>90%) of chronic mesenteric ischemia is related to progressive atherosclerosis at the origins of mesenteric vessels [18]. The diagnosis of chronic mesenteric ischemia is based on clinical symptoms, classically postprandial abdominal pain and weight loss, and anatomic findings, including atherosclerotic occlusion of at least two of the three main splanchnic arteries without evidence of other gastrointestinal pathologies [5]. Although significant atherosclerotic calcifications of mesenteric vessels can also often be visualized in SMA acute thrombus formation, in chronic ischemia without acute thrombosis the appearance of the bowel is usually normal [19].

A minority of cases (5–15%) are due to mesenteric venous thrombosis and resultant bowel edema and diminished perfusion, usually in the setting of hypercoagulable states. Unlike acute arterial mesenteric ischemia, these are not surgical emergencies and often respond to anticoagulation [18]. On imaging, thrombi may be visualized in the mesenteric and portal veins as filling defects surrounded by rim-enhanced venous walls, along with accompanying engorgement of mesenteric veins and prominent bowel wall thickening with halo or target pattern enhancement. Mesenteric fat stranding and ascites are common findings and do not correlate with severity of bowel damage as in acute arterial ischemia. However, bowel wall enhancement may be diminished or absent with severe ischemia [19].

Ischemia may also develop due to secondary causes, such as bowel obstruction. In these cases, treatment of the primary cause is essential.

Key Point

Acute mesenteric ischemia is a surgical emergency and diagnosis must be made rapidly. Signs of irreversible ischemic bowel damage include a "paper-thin" bowel wall, pneumatosis intestinalis and portal venous air, prolonged absence of bowel wall enhancement and signs of perforation including extraintestinal air.

18.5 Concluding Remarks

The small bowel encompasses most of the surface area of the gastrointestinal tract and much of it is inaccessible to endoscopic study. As such, cross-sectional radiological imaging is an imperative component of assessing pathology in the small bowel. Numerous important clinical entities arise in the small intestine, with varying degrees of clinical urgency. In all the above pathologies, imaging constitutes an essential role in establishing a timely diagnosis and directing management. To this end, special care must be taken to choose the appropriate imaging study in any given clinical setting. Comprehensive knowledge of normal and pathologic bowel appearance on various studies is a prerequisite in delivering good care for patients.

Take-Home Messages

- CT and MRI have largely replaced conventional fluoroscopic-based methods of small bowel imaging, with CT-E and MR-E constituting the most specific techniques of small bowel imaging. While MR-E holds a greater role in outpatient settings, CT is often used in acute clinical scenarios.
- MR-E plays a vital role in the assessment of disease activity, complications, and response to treatment for Crohn's disease. Because of radiation concerns in a population of largely young patients, CT is utilized sparingly and reserved for identification of surgical complications in the emergency setting.
- Due to varied and nonspecific symptomology, celiac disease is often left undiagnosed for years. It is important to recognize characteristic features of small bowel malabsorption pattern (MABP) on abdominal imaging, which can indicate the need for further diagnostic testing, in order to minimize complications.
- Though rare, it is critical to identify and characterize small bowel tumors, including on non-targeted abdominal imaging when possible, as often symptoms are nonspecific and diagnosis is delayed. Small bowel malignancies include adenocarcinoma and NETs, and more rarely malignant GIST, lymphoma and sarcoma. MR-E serves as a primary tool for follow-up of multiple small bowel polyps in polyposis syndromes.
- Imaging plays a critical role in the diagnosis of mesenteric ischemia, with CT angiography being the gold standard imaging modality. Acute embolic mesenteric ischemia is a surgical emergency with high mortality rates and prompt intervention is vital.

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