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Title Page

Imaging Of Ischemia, Obstruction And Infection In The Abdomen

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Keywords

Intestinal obstruction; Intestinal ischemia; computed tomography; acute abdomen; bowel; colon.

Synopsis

Intestinal ischemia is a serious condition that continues to be associated with mortality rates in excess of 70%. Intestinal obstruction and gastrointestinal tract sepsis are common conditions, accounting for a large proportion of patients admitted with acute abdominal symptoms to emergency departments. This article discusses the imaging methods and key findings of these entities in the emergency radiology department. The review includes imaging examples, diagnostic options, protocol selections, diagnostic criteria and differential diagnoses.

Key Points

1. CT is the modality of choice in investigation of the acute abdomen in the non-pregnant adult but ultrasound and MRI in particular are being increasingly used.
2. In suspected bowel ischemia, multiphase CT imaging with the use of negative oral contrast in place of positive oral contrast, where possible, is recommended.
3. Complete mesenteric arterial occlusion without reperfusion will result in bowel thinning and minimal mural enhancement whereas other causes of intestinal ischemia will lead to bowel wall thickening, intramural hemorrhage and mural hyper-enhancement.

4. Key findings to decipher regarding bowel obstruction are the location and etiology of transition, grade of obstruction, the presence of close-loop obstruction and the presence of ischemia.

Paragraph Tagging

Intestinal obstruction; intestinal ischemia; intra-abdominal infection; CT; computed tomography; radiography; MRI; magnetic resonance imaging; ultrasound; acute abdomen; emergency department; emergency radiology.

Celiac trunk; superior mesenteric artery; SMA; inferior mesenteric artery; IMA; marginal artery of Drummond; small bowel; large bowel; colon; intestinal malrotation; Meckel's diverticulum.

CT protocol; multidetector CT; intravenous contrast; oral contrast.

Acute intestinal ischemia; mesenteric arterial occlusion; mesenteric venous thrombosis; bowel obstruction; non-occlusive mesenteric ischemia; NOMI; pneumatosis; bowel wall thickening; bowel wall enhancement.

Small bowel obstruction; large bowel obstruction; adhesions; abdominal hernia; volvulus; gallstone ileus; bezoar; colon cancer; adenocarcinoma; diverticulitis; closed loop; beak sign; string of pearls sign; bowel distention; high grade; small bowel feces.

Enteritis; infectious enteritis; intestinal TB; Meckel's diverticulitis; appendicitis; colitis; cecitis; infectious colitis; pseudomembranous colitis; clostridium difficile; amebic colitis; accordion sign; fat halo sign; typhlitis; neutropenic enterocolitis; toxic megacolon; bowel perforation; diverticulitis; diverticular abscess; centipede sign; comma sign; bowel perforation; pneumoperitoneum; omental infarct; epiploic appendagitis; peptic ulcer disease.

Introduction

Intestinal obstruction and intra-abdominal infection associated with the gastrointestinal tract account for a huge proportion of emergency surgical admissions with abdominal symptoms. The former alone accounts for approximately 20% of admissions in this category¹⁻³. Intestinal ischemia is uncommon but still carries a mortality in excess of 70%⁴. As a result of the frequency and gravity of the conditions, timely diagnosis of these entities by emergency radiologists is of key importance. We examine the imaging approach in patients suspected of having the aforementioned conditions, demonstrate key findings and portray potential complications.

Normal Anatomy and Imaging Techniques

Important anatomical considerations

Knowledge of the vascular supply and drainage of the gastrointestinal tract is of key importance in understanding intestinal ischemia. The celiac trunk, superior mesenteric artery (SMA) and smaller infraduodenal inferior mesenteric artery (IMA) must be scrutinized on abdominal imaging studies as a routine. The celiac trunk supplies the foregut, from the distal esophagus to the mid part of the descending duodenum. The SMA, the artery of the mid-gut, supplies the gastrointestinal tract from the mid second part of the duodenum to the junction of the middle and distal thirds of the transverse colon with the IMA, the hindgut artery, supplying the remainder. Collateralization between the SMA and IMA via the marginal artery of Drummond is variable. In contradiction, there is a rich plexus of collaterals between the celiac trunk and SMA. The bowel receives approximately 20% of cardiac output with the mucosa receiving two-thirds of

this ^{5,6}, thus explaining why the mucosa and submucosa are most sensitive to changes in supply in the setting of ischemia.

The relative locations of small and large bowel loops are of significance in assessing for internal hernia, a challenging diagnosis that is frequently a closed loop obstruction. It is worth noting on normal studies that the small bowel loops should not lie lateral or anterior to the ascending/descending colon or transverse colon respectively. In addition, the paraduodenal spaces should be devoid of extra bowel loops. Intestinal malrotation, which is associated with midgut volvulus and internal herniation, results in failure of the third part of the duodenum to cross the midline, in association with small bowel being located on the right side of the abdomen and the colon being principally left sided. The SMA and SMV (superior mesenteric vein) relationship may be reversed in addition ⁷. Malrotation with non-rotation will also result in an appendix that lies to the left of midline, a finding that will also be present in situs inversus.

The vermiform appendix measures a mean of 11 cm and the tip is most commonly retrocecal (74%) though the remainder have a variable location ⁸.

Meckel's diverticulum is an anatomical variant that results from persistence of part of the omphalomesenteric duct, is present in 2% of the population and is located approximately 2 feet from the ileo-cecal valve on the antimesenteric border ⁹.

Normal bowel diameter may measure 2.5 cm for the small intestine, 9 cm for the cecum and 6 cm for the remainder of the colon ^{10,11}.

Imaging Techniques

Radiography

Abdominal radiography is frequently used in assessment of the acute abdomen. It has a reported sensitivity of 69-80% for bowel obstruction ¹²⁻¹⁵ but is insensitive in assessing for complications or etiology. In addition, abdominal radiographs are neither sensitive nor specific for detection of intestinal ischemia or infectious/inflammatory conditions such as diverticulitis, colitis or appendicitis. The erect chest radiograph remains an essential part of assessment and detection of pneumoperitoneum in suspected hollow viscus perforation.

Computed Tomography

Multidetector CT (MD CT) is the main modality for the diagnosis of bowel obstruction and ischemia due to the availability, speed, sensitivity and specificity ^{1,2,5,16-24}. MD CT has a sensitivity in excess of 80% for intestinal ischemia detection ⁵. MD CT accuracy of 95% is reported for detection of high-grade small bowel obstruction, though accuracy is decreased for low-grade obstruction ²⁵. A sensitivity in excess of 90% is reported for large bowel obstruction ^{26,27}. Sensitivity and specificity for appendicitis is also in excess of 90% ^{20,28}.

Magnetic Resonance Imaging

MRI is being increasingly used in detection of bowel obstruction, ischemia and infection. MR imaging has a sensitivity that is similar to CT in assessing bowel obstruction ^{29,30} and ischemia ^{31,32}. In addition, MRI is comparable to CT in infectious/inflammatory conditions such as appendicitis and diverticulitis ^{20,33,34}.

Ultrasound

In children and pregnancy, US is the first choice modality in assessment of suspected appendicitis. Ultrasound has a complimentary role to CT and MRI in assessing small bowel obstruction and suspected ischemia; however, its main strengths include ready availability in most hospitals, avoidance of exposure to ionizing radiation and the portable nature of the modality, which means that it can be performed at the bedside in critically ill patients ^{30,35,36}.

Fluoroscopy

Angiography is rarely used currently for diagnosis in intestinal ischemia but is used during endovascular therapy. In the past, contrast enemas and barium follow through studies were utilized as part of large and small bowel obstruction evaluation but current practice means that use has been superseded.

Imaging Protocols

	Suspected Ischemia	Suspected obstruction or bowel infection
CT phases	Unenhanced	
	Angiographic phase (30-35 seconds)	
	Portovenous phase (60-65 seconds)	Portovenous phase (60-65 seconds)
Positive oral contrast	500-600 ml water preferred	Optional in obstruction Preferred in infection

Intravenous contrast	300-350 mgI/ml 3-4 ml/second	300-350 mgI/ml 3-4 ml/second
Recon slice	1.5-3.0 mm	1.5-3.0 mm

Many authors would advocate a 3-phase protocol for assessing suspected acute mesenteric ischemia. The unenhanced phase may not be required for the diagnosis of bowel ischemia, however³⁷. The absence of positive oral contrast is essential for detection of bowel wall enhancement in suspected ischemia.

Intraluminal fluid acts as a good negative oral contrast agent in cases of bowel obstruction. Positive oral contrast in the setting of suspected appendicitis has a marginal accuracy benefit^{28,38}, particularly in patients of low BMI, but the use of same may result in some delay in imaging of the patient. Multidetector isometric thin section imaging with multiplanar reconstructions is essential in bowel wall and vasculature assessment; review of reconstructions in the coronal plane are especially useful for investigating cases of bowel obstruction.

Imaging Findings/Pathology

Ischemia

Acute intestinal ischemia is an uncommon condition, accounting for approximately 0.1% of hospital admissions³⁹. All cause mortality remains approximately 70% however^{4,39}, a figure that increases to closer to 90% when infarction has become established.

The causes of acute mesenteric ischemia are as follows:

- Arterial occlusion (50-60% of cases ^{5,32})
- Venous thrombosis (5-10% of cases ⁵)
- Small vessel disease – vasculitides, radiation, chemotherapy-induced
- Mechanical bowel obstruction
- Non-occlusive mesenteric ischemia (shock bowel)

Key imaging findings include:

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

Bowel wall thickening

Normal bowel wall thickness ranges from 3 to 5 mm ^{40,41}. The degree of bowel wall distention is a factor in determining if the bowel wall is truly thickened and caution should be exhibited when the bowel is collapsed or over distended. Wall thickening from mucosal/submucosal edema or hemorrhage is the commonest finding in intestinal ischemia, and it is present in most cases that

are not due to acute arterial occlusion, however, bowel wall thickening is not specific for intestinal ischemia ^{16,19,21,22,42,43} (Fig. 1). The degree of wall thickening does not correlate with the severity of ischemic wall damage. In the setting of acute arterial occlusion however, the wall is frequently thinned ('paper thin wall') (Fig. 2) and this would appear to be explained by the absence of vascular flow required to produce edema or hemorrhage.

Bowel wall attenuation on control images

Increased bowel wall attenuation from submucosal hemorrhage on unenhanced CT images is a specific sign for bowel ischemia ^{37,44}. The sign is rarely seen in isolation ^{37,45}. Like bowel wall thickening, submucosal hemorrhage is rarely present in the setting of arterial occlusion.

Abnormal wall enhancement

Reduced or absent bowel wall enhancement, which is an insensitive but specific sign, may be seen in arterial occlusion without reperfusion ^{5,16,43} (Fig. 1). Reduced enhancement is also associated with infarction ⁴⁶. In cases of incomplete arterial occlusion, arterial occlusion with reperfusion and other causes of intestinal ischemia, increased bowel wall enhancement is more commonly evident.

Increased luminal diameter

Bowel distention is seen in up to 90% of bowel infarction cases but its presence is variable in cases of reversible ischemia ^{5,16,43}. The distention is

predominantly due to increased intraluminal fluid and ileus from neuro-enteric plexus dysfunction.

Mesenteric arterial or venous occlusion

The finding of arterial (Fig. 2 & 3) or venous occlusion (Fig. 4) in the mesenteric vessels is indicative of the cause rather than an effect of mesenteric ischemia. Embolus rather than thrombosis is more common in the superior mesenteric artery in the acute setting. Infarcts may also be seen in the other viscera such as the kidneys or spleen as a result of emboli. In the chronic setting, atherosclerosis dominates in terms of etiology but it is worth noting that incidental mesenteric atherosclerotic disease is common in asymptomatic elderly patients⁴⁷. On the venous side, thrombosis may be due to a local trigger such as intra-abdominal infection, neoplasm or inflammation, portal hypertension, or a systemic tendency for clot formation such as antiphospholipid syndrome or oral contraceptive pill use^{5,16,48,49}.

Ascites and mesenteric fluid or stranding

As with many other intra-abdominal pathologies, the presence of free fluid is a non-specific sign in the setting of suspected intestinal ischemia. The presence of free fluid in the mesentery (Fig. 4) is reportedly more sensitive (88% vs. 75%) and specific (90% vs. 76%) than the finding of free ascites in the setting of ischemia from small bowel obstruction^{50,51}. The presence of ascites and/or mesenteric stranding may increase the likelihood of infarction in the setting of arterial occlusion⁵. The ascites may be of high density from hemorrhage.

Pneumatosis intestinalis, pneumatosis portalis and pneumoperitoneum

Detection of pneumatosis intestinalis (gas in the bowel wall) at CT is best performed by utilizing 'lung window' settings (Fig. 3). The dependent part of the wall is the best location to detect this finding where the dependent luminal contents can be utilized to improve conspicuity and localization. Gas may appear as bubbles or rims of gas separating the wall layers. Images have to be closely scrutinized to assess for pneumatosis portalis (gas in the mesenteric or portal veins) (Fig. 5), in addition. Distinguishing intrahepatic porto-venous gas (Fig. 6) from pneumobilia can be achieved by following the course of the vessel or channel involved. In addition, pneumobilia is more frequently central and does not involve the subcapsular ~2 cm of parenchyma, unlike porto-venous gas which is frequently peripheral and extends much closer to the capsule. In the correct clinical context, the presence of pneumatosis intestinalis or pneumatosis portalis have specificities approaching 100% for ischemia detection, particularly when seen with other findings such as abnormal bowel wall enhancement ^{5,16}. The presence of both entities has a reported specificity of 83% for infarction though the sensitivity is only 17% ⁵². There are multiple other causes of these entities, however. Alternate causes of pneumatosis intestinalis or portalis include systemic lupus erythematosus, scleroderma, barotrauma, asthma, chronic obstructive pulmonary disease, intra-abdominal infection, bowel obstruction, iatrogenic bowel distention and inflammatory bowel disease ^{53,54}. The presence of free air in the setting of intestinal ischemia is highly suggestive of infarction.

Obstruction

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

Small bowel obstruction (SBO)

Adhesions from prior abdominal surgery represent the commonest cause of small bowel obstruction accounting for up to 75% of cases ^{1,24,25} with hernias being the next commonest cause. Further causes are best summarized by congenital (e.g. ileal atresia, midgut volvulus), extrinsic (e.g. volvulus, compression from mass), bowel wall (e.g. stricture, intussusception, tumor) and intraluminal (e.g. meconium ileus, gallstones, foreign body, bezoar) categories.

Large bowel obstruction (LBO)

Mechanical obstruction of the colon is most commonly due to colon cancer, accounting for 50-60% ^{10,27,55}. Other causes include volvulus, diverticulitis, inflammatory bowel disease, radiation, ischemia, fecal impaction or hernia.

Imaging findings

Considerations include:

- Proximal bowel distention
- Distal collapsed bowel
- 'String of pearls sign'

- Transition point 'beak sign'
- Grading
- Small bowel feces
- Potential closed loop
- Visible cause SBO
- Visible cause LBO
- Possible mesenteric ischemia

Bowel distention with distal collapse

The key finding in small (Fig. 7) or large bowel obstruction (Fig. 8) is dilatation of the loops proximal and collapse of loops distal to a point of obstruction. Small bowel diameter in excess of 2.5 cm and large bowel diameter in excess of 6 cm (9 cm for cecum) are considered dilated. On plain radiography, small bowel loops are dilated centrally with visible transverse valvulae conniventes whereas the large bowel is peripheral in location with haustral folds that are seen to incompletely traverse the wall. The location of distal bowel collapse helps decide whether the obstruction is in the small or large bowel. If the obstruction is in the colon but the ileo-cecal valve is competent, the small bowel will not be dilated. It is important to differentiate mechanical obstruction from paralytic ileus and pseudoobstruction. Paralytic ileus typically occurs in the post-operative patient, is often painless with absent bowel sounds and demonstrates small and large bowel distention. Colonic pseudoobstruction, known as Ogilvie's syndrome in the acute setting, on the other hand, is similar in presentation to large bowel obstruction in that the patients often have pain and distention but frequently have a history of chronic constipation^{56,57}. Imaging in

pseudoobstruction demonstrates a dilated proximal colon, distal collapse and classically, a caliber change at the splenic flexure, without a lesion at the site of transition ⁵⁶. The site of transition, however, can be anywhere in the colon ⁵⁷. In this syndrome, the small intestine may be dilated if the ileo-cecal valve is incompetent.

'String of pearls sign'

The 'string of pearls sign' is seen on lateral decubitus or erect abdominal radiographs, in addition to CT, when small bubbles of gas are trapped between the valvulae conniventes. It is reported to be specific for small bowel obstruction and absent in the setting of adynamic ileus or gastro-enteritis ⁵⁸.

Transition point 'beak sign'

Typically, transition from dilated proximal bowel to collapsed distal bowel occurs at the site of obstruction, though this isn't always the case. When an abrupt transition occurs, the bowel forms a "beak sign" whereby the dilated proximal bowel quickly tapers to the transition point resulting in the shape of a beak (Figs. 7 & 9). In a closed loop obstruction, 2 transition point beak signs may be seen. The absence of a 'beak sign' is associated with successful non-operative management in the setting of adhesion related SBO, along with the presence of a small bowel feces sign and an anterior parietal adhesion ⁵⁹. On the other hand, two 'beak signs' or more, a whirl sign, a C- or U-shaped bowel loop and high-grade obstruction are more likely to require surgical management in the setting of adhesional SBO ^{59,60}.

Grading

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

Small bowel feces

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

Potential closed-loop

Closed-loop obstruction occurs when the bowel is obstructed at 2 different points along its length, usually at a single location and from a single cause. It is an important diagnosis to make as it carries a higher risk of bowel ischemia and usually requires prompt surgical intervention ^{2,10,25,63}. Two 'beak signs' may be seen from the 2 points of obstruction, along with a radial or U-shaped configuration of the bowel loops. A whirl (or whirlpool) sign may also be

seen, where the mesenteric vessels have a swirling appearance from torsion of these structures around themselves⁶⁴. Closed-loop obstruction is associated with hernias (particularly internal hernias) (Fig. 11), midgut volvulus, cecal volvulus (Fig. 12) and sigmoid volvulus (Fig. 13). A proximal colonic obstruction with a competent ileo-cecal valve may also behave like a closed-loop obstruction.

Visible cause SBO

Differentiation of SBO from LBO is fundamental in assessing the etiology with the location of the transition point being key to same. Intraluminal causes of SBO such as bezoar, inspissated contents (distal intestinal obstruction syndrome, seen in cystic fibrosis) (Fig. 14) or gallstone ileus (Fig. 15) appear as intraluminal filling defects. Mural causes have the appearance of focal or segmental wall thickening that may be circumferential (Fig. 16) or eccentric. Extrinsic causes of SBO are the most commonly encountered etiologies. An adhesional band is rarely evident in the setting of SBO hence it is a diagnosis of exclusion. Abdominal wall hernias such as incisional (Fig. 17), epigastric, umbilical, para-umbilical, inguinal (Fig. 18) and femoral (Fig. 19) hernias may be seen in addition to diaphragmatic, pelvic and other internal hernias⁶³. Internal hernias are increasing in frequency, due to the increased volume of surgery, particularly bariatric surgery^{63,65}, and their diagnosis can be challenging. Regarding internal herniation, in addition to features of closed-loop obstruction, the herniated bowel loops, most commonly small bowel, have an abnormal location relative to the colon or duodenum.

Visible cause LBO

With regard to large bowel obstruction, the etiology of exclusion is a colonic neoplasm, given that it is the cause in 50-60% of cases ^{10,27,55}. A circumferential or eccentric short segment enhancing mass is usually evident in the setting of colon cancer, classically located in the sigmoid colon (Fig. 8). Diverticulitis or diverticular stricture involves a longer segment with a greater degree of peri-colonic fat stranding and diverticulae. If CT findings are equivocal, MRI is reported to have a superior sensitivity and specificity for differentiating colonic tumor from diverticulitis ³⁴. If LBO is due to cecal (Fig. 12) or sigmoid volvulus (Fig. 13), the diagnosis may be made on abdominal radiography. In cecal volvulus, the cecal pole will have an ectopic location, pointing towards or located in the left upper quadrant. Sigmoid volvulus classically has an abnormal loop of large bowel extending out of the pelvis forming a coffee bean shape ⁶⁶. The CT findings are in keeping with those on plain radiographs in addition to closed-loop obstruction.

Possible mesenteric ischemia

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

Gastrointestinal tract infection

Small bowel

Infectious enteritis

Acute infectious enteritis (or gastro-enteritis) is usually self-limiting and typically does not require imaging. Imaging may be performed, however, in atypical presentations, with chronic symptoms and in the immunosuppressed patient. Causative organisms include bacteria (*Salmonella*, *Yersinia*, *E Coli*, *Campylobacter*, *Mycobacterium tuberculosis (TB)*), protozoa (*Giardia lamblia*, *Cryptosporidium*) and viruses (*Cytomegalovirus*, *Norovirus*, *Rotavirus*). *Giardia* has a preponderance for the proximal small bowel, the CT findings of which are non-specific, with circumferential wall thickening and lymphadenopathy being the commonest⁶⁷. The majority of pathogens most commonly affect the ileum^{22,68}. Ninety percent of TB occurs in the ileum or cecum due to the stasis and lymphatic abundance⁶⁹. Ileal TB usually results in asymmetric wall thickening in the ileum and multiple large low density/necrotic nodes on CT^{68,70}. Crohn's disease on the other hand, gives symmetrical circumferential wall thickening, small nodes, fibrofatty mesenteric proliferation ('creeping fat') and potential for fistula formation as a result of the transmural inflammation²¹. *Salmonella* gives a similar appearance to Crohn's disease in terms of segmental circumferential wall thickening^{68,71}. *Yersinia* typically results in mild symmetrical bowel wall thickening, aphthous ulceration and mucosal nodularity²². Typhlitis (neutropenic enterocolitis) will be discussed in the large bowel section.

Meckel's diverticulitis

Approximately 2% of the population has a Meckel's diverticulum. Many of these true diverticulae are incidental findings but complications or symptoms develop in 4-40%^{9,72,73}. Diverticular infection/inflammation or diverticulitis (Fig. 20) accounts for up to 30% of complications^{9,73}. Gastrointestinal bleeding,

intussusception, obstruction, perforation and band strangulation are other common presentations or complications. The right lower quadrant or close to midline are the most likely locations of a Meckel's diverticulum at CT examination in the setting of diverticulitis ⁷⁴. It appears as a blind ending pouch of approximately 2.5cm in (outer wall) diameter and approximately 3.3cm in length with a mean wall thickness of almost 4 mm. In the setting of Meckel's diverticulitis, inflammatory changes in the surrounding fat and some free fluid are evident. Other complications such as bowel obstruction or perforation may also be present, making the identification of a Meckel's difficult.

Colon

Appendicitis

Acute appendicitis is a very common cause of acute abdominal pain, particularly in children and adolescents, with a lifetime risk of developing acute appendicitis of 6-8% ⁷⁵. 'Graded compression' ultrasound ⁷⁶ is usually the first line imaging investigation in children, young adults and pregnancy. The user dependent nature of the modality likely contributes to variable reported sensitivities of 12-90% ⁷⁷⁻⁷⁹ and commonly encountered lack of confidence in sonographic diagnosis ⁸⁰, though high specificities in excess of 85% are routinely reported. MRI has reported accuracies, in excess of 90%, that are similar to CT in diagnosis of acute appendicitis ^{20,33,77,78,81}. As a result, the use of MRI in this setting continues to increase. Regardless of the imaging modality, typical findings include an appendix diameter of >6 mm, luminal obliteration, non-compressible appendix, appendicolith, peri-appendiceal inflammatory fat

changes, free fluid, inflammatory phlegmon and abscess^{75,78,79,81-83} (Figs. 21 & 22).

Colitis and cecitis

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

In Western countries, bacteria most commonly cause infectious colitis. In particular, the number of cases of pseudomembranous colitis has markedly increased in recent times, as a result of increased usage of broad spectrum and prophylactic antibiotics^{84,85}. *Clostridium difficile* is not a normal bowel commensal organism but colonizes the colon after antibiotics or chemotherapy have disrupted the normal colonic biology^{85,86}. The bacteria produce 2 toxins (A & B) that result in the colonic inflammation. Typical CT findings of pseudomembranous colitis (Fig. 23) are wall thickening of 11-14 mm, submucosal low attenuation from edema and an irregular mucosal contour^{85,87,88}. The “accordion sign”, where positive oral contrast interspersed between thickened haustral folds is said to look like an accordion, may also be seen, though it can be seen in any severe colitis⁸⁹ (Fig. 24). In addition, the “target sign”, where post contrast enhancement of the mucosa and muscularis propria with intervening submucosal edema has the appearance of a target, may also be evident (Fig. 23). The “fat halo sign” is similar but occurs when the submucosa

demonstrates fat deposition, though it predominantly occurs in ulcerative colitis⁹⁰.

Amebic colitis occurs as a result of colon invasion from the trophozoite form of the organism. The inflammation usually starts in the cecum, often involves the rectum and may result in diffuse colitis⁸⁷. The most distal part of the ileum may be involved. Amebic colitis results in wall thickening, ulceration, pseudopolyps and skip lesions that are not dissimilar to Crohn's colitis. The classic coned cecum, where the cecum resembles an inverted cone pointing at the appendix base, develops after subacute infection⁸⁷. The cone-shaped cecum can be seen in multiple inflammatory, infectious or neoplastic conditions, however. Amebomas, focal areas of granulation, can closely mimic colonic neoplasms^{87,91,92}, particularly if hepatic amebic disease is present^{93,94}. Residual colonic scarring is not infrequent post infection.

There is huge overlap in the location and appearances of most bacterial and viral infectious colitides, hence stool culture and history are of key importance in identifying the offending organism. Certain patterns of involvement are associated with certain organisms, however. *Cytomegalovirus* and *Escherichia coli* often result in diffuse colitis, *Shigella* and *Salmonella* colitides are frequently confined to the right colon, schistosomiasis is usually confined to the left colon, and *Chlamydia trachomatis*, *Neisseria gonorrhoeae* and *Herpes* species are confined to the rectosigmoid region.

Typhlitis, also known as neutropenic enterocolitis, has an incidence of up to 26% in certain patient groups undergoing chemotherapy⁹⁵, but can occur in any immunosuppressed patient. The mortality remains in excess of 50% despite increased awareness and aggressive management with bowel rest, fluids, broad

spectrum antimicrobial therapy and blood products where necessary ⁹⁵. It is likely multifactorial in etiology with neutropenia, bowel wall injury from treatment or neoplasm, and intramural hemorrhage from thrombocytopenia resulting in invasive polymicrobial infection from commensal bowel organisms ⁹⁵⁻⁹⁷. Clinical symptoms and signs include of abdominal pain, localized tenderness, distention, nausea, vomiting, bloody diarrhea and fever. CT, the imaging modality of choice, demonstrates increased wall thickening (typically ~7 mm) involving the cecum, and often ileum and remaining colon ^{87,96,98} (Fig. 25). The disease is reportedly limited to the cecum in 28% ⁹⁶. Pneumatosis, mucosal enhancement, free fluid and fat stranding may also be seen ^{87,96}. It is worth remembering that neutropenic patients are also of increased risk of other colitides, including *CMV* colitis and pseudomembranous colitis ⁹⁶.

For any infectious colitis, the main differential diagnoses includes non-infectious etiologies of colitis such as inflammatory bowel disease with radiation colitis and ischemic colitis being less common. Diverticulitis, neoplasm or graft-versus-host disease are additional alternate diagnoses to consider. Amebic, TB and pseudomembranous colitis most closely mimic inflammatory bowel disease. Radiation colitis will likely be suspected from the history and ischemic colitis will be found in the older age group, with a vascular distribution and features mentioned in the 'mesenteric ischemia' section. Bowel wall thickness, bowel wall attenuation, degree of wall thickening, symmetry of wall thickening, length of segment involved and associated findings such as stranding, lymphadenopathy and abscess are key factors which aid in delineating etiology ^{41,90}.

Complications of colitis are readily assessed using CT. They include perforation, abscess, ischemia or toxic megacolon. The latter complication is amongst the most feared and is more commonly caused by ulcerative colitis or increasingly, pseudomembranous colitis ⁹⁹, but may be associated with any severe form of colitis. Nitric oxide and interleukins appear to be important in the pathogenesis ⁹⁹. The key imaging findings are dilatation of the colon, typically the transverse colon, to greater than 6 cm diameter with loss of the haustral folds and wall thinning ^{100,101}, findings that help distinguish it from acute severe colitis. Radiographs are useful in assessing bowel diameter in the setting of toxic megacolon ¹⁰², but CT is the superior investigation as it can detect small perforations and abscesses in addition to making a more accurate diagnosis ⁹⁹.

Diverticulitis

Colonic diverticula are acquired pseudo-diverticula that may result from increased intraluminal pressure and occur at weak points in the circular muscle where mucosal vasculature penetrates on the mesenteric wall ¹⁰³. They predominantly occur in the descending and sigmoid colon but can be found anywhere in the colon apart from the rectum. Diverticulosis is very common in the Western world, is predominantly a disease of older adults, is found in >70% of those over 80 years and approximately 5% of people at 40 years of age ^{104,105}. Traditional thinking about etiology was that it occurred in patients lacking in dietary fiber ¹⁰⁶, but studies to confirm this are conflicting ^{107,108}. Diverticulitis occurs when one or more of these diverticula become obstructed at the neck resulting in stasis, inflammation, infection and perforation. Diverticulitis occurs in between 1 and 25% in patients with diverticular disease, though recent

studies suggest that rates of development of acute diverticulitis is at the lower end of that range ¹⁰⁵. It is amongst the commonest inpatient gastrointestinal diagnoses ¹⁰⁴. Typical presentations include symptoms and signs of left iliac fossa pain and tenderness, altered bowel habit, nausea, pyrexia, possibly a palpable mass and raised inflammatory markers. CT is the investigation of choice in suspected acute diverticulitis. Findings include mesenteric fat stranding that is disproportionate to the degree of colonic wall thickening, mural hyperemia, segmental involvement, fluid accumulation in the mesenteric root (the “comma sign”), mesenteric hypervascularity (the “centipede sign”) and the presence of diverticula ^{87,109,110} (Fig. 26). Associated findings may include an abscess (present in up to 30%) (Figs. 26 & 27), localized perforation, generalized perforation or fistula. Fistulae may be from the involved colon to: colon, small bowel, vagina, bladder or skin. Classification of the severity of acute diverticulitis is advantageous and a number of scales exist. One such surgical scale is the *Hinchey* classification ¹¹¹: stage Ia – phlegmon; stage Ib – with pericolic or mesenteric abscess; stage II – with walled off pelvic abscess; stage III – with generalized purulent peritonitis; stage IV – with generalized feculent peritonitis. Though the described classification system has a surgical basis and classification may not be used commonly in practice, the elements should be mentioned in the radiology report to indicate severity.

Differentiating diverticulitis from colonic neoplasm is challenging. The presence of diverticula, disproportionate fat stranding, “comma” or “centipede” signs, mesenteric root fluid, mild smooth concentric wall thickening, gradual transition from normal to abnormal bowel thickness, long (5-10 cm) affected segment and minimal or no lymphadenopathy are associated with acute

diverticulitis. On the other hand, the absence of diverticula, fat stranding, “comma” or “centipede” sign and mesenteric fluid along with the presence of lymphadenopathy, bowel wall shouldering, increased density of the focal bowel wall thickening or mass post contrast administration, and irregular eccentric short segment (<5 cm) wall thickening is associated with colonic neoplasm^{110,112-114}. MRI may have an advantage over CT in differentiating tumor from diverticulitis³⁴. Epiploic appendagitis is another important differential diagnosis in patients with suspected diverticulitis. The key finding is an ovoid fat-density lesion with surrounding inflammation adjacent to the colon with little if any wall thickening¹¹⁵(Fig. 28).

All patients should undergo endoscopy after an episode of diverticulitis, predominantly to ensure no underlying neoplasm¹⁰⁵.

Sepsis from gastrointestinal perforation

Hollow viscus perforation and associated abdominal sepsis remains a common problem but the radiological diagnosis of pneumoperitoneum can be readily made on chest and/or abdominal radiographs^{116,117}. Subdiaphragmatic free air on erect chest radiography, or the double wall (“Rigler’s sign”), ‘lucent liver sign’ or ‘football sign’ on abdominal radiography indicate pneumoperitoneum (Fig. 29). The lateral decubitus abdominal radiograph is rarely required but useful if the erect chest radiograph is inclusive, CT is unavailable or the patient is too unstable for transfer (Fig. 30). Perforated peptic ulcer disease (PUD) is much less common than in the era before proton pump inhibitor therapy but it is still a leading cause of pneumoperitoneum. In one recent US series, PUD (16%), diverticulitis (16%), trauma (14%), malignancy

(14%) and bowel ischemia (10%), appendicitis (6%) and endoscopy (4%) were the leading causes of pneumoperitoneum when postoperative pneumoperitoneum was excluded ¹¹⁸. Further imaging is often not required if the clinical picture clarifies the likely cause and if further imaging would delay surgical management. CT imaging has a role in planning the type of operative treatment, prognosis and assessing those patients that have clinical symptoms of peritonitis but no radiographic signs of perforation. One scenario where the latter is true is in the setting of PUD perforation into the lesser sac (Fig. 31). In this scenario, a gas and fluid containing collection is often seen in the lesser sac, posterior to the stomach. When CT imaging is performed in the setting of known pneumoperitoneum, a careful search for the likely etiologies above, should be completed. The presence of fat stranding, abscess, wall thickening and potential mural defect will help localize the site of perforation (Fig. 32).

Diagnostic Criteria

Intestinal ischemia

1. Abnormal bowel wall thickness
 - a) Paper thin in acute complete arterial occlusion without re-perfusion
 - b) Diffusely thickened, possibly with submucosal hemorrhage in other causes of ischemia
2. Abnormal wall enhancement
 - a) Absent or hypoenhancement in acute complete arterial occlusion without re-perfusion
 - b) Hyperenhancement in other causes of ischemia

3. Increased luminal diameter
4. Arterial or venous occlusion
5. Mesenteric fluid or stranding
6. Pneumatosis intestinalis
7. Pneumatosis portalis

Obstruction

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

Infection

1. Enteritis
 - a) Circumferential wall thickening
 - b) Asymmetric nodular wall thickening in TB ileitis
 - c) Mural hyperenhancement
 - d) Fat stranding
 - e) Lymphadenopathy (particularly in TB ileitis)
2. Meckel's diverticulitis
 - a) Blind ending tubular structure approximately 2 feet from the ileocecal valve
 - b) Wall thickening
 - c) Surrounding fat stranding

3. Appendicitis
 - a) Appendix diameter of >6mm
 - b) Luminal obliteration
 - c) Non-compressible appendix at ultrasound
 - d) Appendicolith
 - e) Peri-appendiceal inflammatory fat changes
4. Colitis
 - a) Circumferential segmental wall thickening
 - b) Mucosal hyperenhancement
 - c) 'Accordion sign'
 - d) 'Halo sign'
 - e) Peri-colonic fat stranding
 - f) Submucosal edema
5. Typhlitis
 - a) As for colitis but may be confined to cecum or involve the ileum
6. Diverticulitis
 - a) Diverticula
 - b) Fat stranding that is disproportionate to the mild smooth concentric wall thickening
 - c) 'Comma' or 'centipede' signs
 - d) Mesenteric root fluid
 - e) Gradual transition from normal to abnormal bowel thickness
 - f) Relatively long (5-10 cm) affected segment
 - g) Minimal or no lymphadenopathy

Differential Diagnosis

Intestinal ischemia differentials

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

Bowel obstruction differentials

1. Paralytic ileus or pseudoobstruction
 - Recent surgery or trauma
 - Shock
 - Medications
 - Electrolyte abnormalities
2. Celiac disease
3. Scleroderma
4. Mesenteric ischemia

Bowel infection differentials

1. Appendicitis or Meckel's Diverticulitis

- Omental infarct
 - Epiploic appendagitis
 - Ileitis
 - Right-sided colonic diverticulitis
 - Pelvic inflammatory disease
2. Enteritis/ileitis
- Crohn's disease
 - Intestinal ischemia
 - Spondyloarthropathy associated ileitis
 - Vasculitis
 - Neoplasm (e.g. lymphoma)
 - Radiation
3. Infectious colitis
- Inflammatory bowel disease
 - Typhlitis
 - Radiation colitis
 - Diverticulitis
 - Neoplasm
 - Graft-versus-host disease
4. Diverticulitis
- Colonic neoplasm
 - Colitis
 - Epiploic appendagitis
 - Omental infarct

5. Perforation/pneumoperitoneum

- Residual post-operative air
- Peptic ulcer disease
- Diverticulitis
- Trauma
- Bowel malignancy
- Bowel ischemia
- Appendicitis
- Endoscopy

Pearls, Pitfalls, Variants

Ischemia

- Bowel wall thickness is difficult to assess in under-distended or over-distended bowel, hence the level of distention should be taken into account. Oral intake of water or per-rectal administration of water or air will facilitate adequate distention.
- Ischemic bowel, particularly ischemic colon, may contract as a result of ischemia. Again, adequate distention with oral water or per-rectal water or air is important.
- Bowel distention with intraluminal fluid and mural thickening as a result of ischemia may be mistaken for mechanical obstruction.

Clinical findings, transition point appearance and ancillary findings are key in this regard.

Obstruction

- Bowel distention with intraluminal fluid and mural thickening as a result of ischemia may be mistaken for mechanical obstruction. Clinical findings, transition point appearance and ancillary findings are key in this regard.
- Paralytic ileus and colonic pseudoobstruction can be mistaken for adhesional obstruction. The presence of collapsed distal bowel, a transition point 'beak sign' and visible cause are important differentiating factors.

Infection

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

What the Referring Physician Needs to Know

Ischemia

- Location and length of involved segment
- Etiology
- Features raising concern for bowel infarction – pneumatosis intestinalis, pneumatosis portalis, pneumoperitoneum

Obstruction

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

Infection

1. Enteritis

- Extent of involvement
- Possible etiology
- Complications

2. Appendicitis

- Simple or complicated (phlegmon, abscess)

3. Meckel's diverticulitis

- Suspected diagnosis
- Presence of complications (abscess, bowel obstruction)

4. Colitis, cecitis and typhlitis

- Segment and extent of bowel involved
- Likely etiologies
- Presence of complications (toxic megacolon, ischemia, abscess, perforation)

5. Diverticulitis

- Location and length of involved segment
- Presence of peri-colic phlegmon or abscess
- Presence of pelvic abscess
- Presence of localized 'walled-off' perforation
- Presence of generalized pneumoperitoneum
- Presence of feculent peritoneal contamination

Conclusions

Ischemia, obstruction and gastro-intestinal infection are serious conditions frequently associated with significant morbidity and mortality in addition to being frequently encountered entities by the emergency radiologist. CT remains the investigation of choice though other modalities, MRI in particular, are going to play a greater role in the future. It is of key importance to ensure that the CT protocol is set up correctly in suspected intestinal ischemia,

as multiphase imaging is required for optimal assessment. Measures should also be taken to ensure adequate bowel distention.

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Figure Legends:

Figure 1. A-B

Axial contrast enhanced CT scan in a 47-year-old female patient with acute abdominal pain. A hypervascular mesenteric carcinoid mass (**A**, arrowheads) is seen in the mesentery with a marked surrounding desmoplastic reaction. The adjacent small bowel shows signs of venous ischemia (**A&B**, arrows) with wall thickening, submucosal edema and mucosal hyperenhancement.

Figure 2. A-D.

Axial and sagittal CT images, from an 81-year-old male with a history of atrial fibrillation, who attended the emergency department with acute severe constant abdominal pain. Acute complete occlusion of the superior mesenteric artery (**A**, white arrows) is identified along with poorly enhancing small bowel walls (**B**, arrowheads). Distal small bowel loops show hyperenhancement (**C**, curved arrows) suggesting some reperfusion but ongoing ischemia. Pneumatosis portalis (portovenous gas) is seen in the peri-enteric veins anteriorly (**D**, black arrows).

Figure 3. A-B.

Axial and sagittal arterial phase CT images on a 48-year-old female patient with long term cognitive impairment who attended with abdominal pain and distention. Multiple dilated bowel loops from stercoral large bowel obstruction are seen along with pneumatosis intestinalis (**A**, arrowheads) and pneumatosis portalis (portovenous gas) (**A**, curved arrows). The stercoral obstruction led to

severe abdominal compartment syndrome resulting in obstruction of the abdominal aorta (**B**, arrows) with resultant ischemia.

Figure 4. A-B.

Coronal reformatted portovenous phase CT images on a 46-year-old female patient with acute abdominal pain and vomiting. Thrombus is identified in the superior mesenteric vein (**A**, arrows). There is resultant ischemia of the small bowel with stranding and fluid in the mesentery (**B**, curved arrow) and mural thickening and hyperenhancement in the mildly dilated small bowel loops (**B**, arrows).

Figure 5.

Axial contrast enhanced CT image from a 58-year-old male with mesenteric ischemia and perforation related to mechanical obstruction. Intrahepatic pneumatosis portalis (portovenous gas) is seen within the liver (arrows), extending almost to the capsule of the liver.

Figure 6. A-B.

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

Figure 7.

Coronal reformatted CT image in a 52-year-old male with high-grade adhesional small bowel obstruction demonstrating dilated proximal small bowel loops (arrowheads) and a 'beak sign' (arrows) at the site of obstruction.

Figure 8.

Axial CT image in a 61-year-old female patient with an obstructing sigmoid tumor (arrows) that demonstrated shouldering and asymmetrical bowel wall thickening and proximal colonic dilatation (arrowhead).

Figure 9.

Coronal T2-weighted fat saturated MRI image in a 36-year-old male patient with high-grade small bowel obstruction from a stricture secondary to underlying Crohn's disease (arrows). The proximal loops are dilated (arrowheads) and there is a benign stricture at the site of transition.

Figure 10. A-B.

Axial T2-weighted fat saturated MRI image in a 39-year-old male patient demonstrating a small bowel feces sign (**A**, arrows), proximal to a strictured segment of distal ileum (**B**, arrowhead) that has an adjacent phlegmon (**B**, curved arrows).

Figure 11.

Axial CT image from a 46-year-old woman with a history of prior bowel surgery who presented with suspected bowel obstruction. An internal hernia through a defect in the transverse mesocolon is demonstrated (arrows), where there is a

visible 'beak sign'. The closed-loop obstruction involves the dilated loops of small bowel (arrow).

Figure 12. A-B.

Abdominal radiograph and axial CT image in a 40-year-old patient with suspected bowel obstruction. The cecal pole has flipped and is now located and pointing to the left upper quadrant (**A**, arrows). A 'beak sign' is demonstrated from one of the transition points from the resultant closed-loop obstruction (arrowheads).

Figure 13. A-B.

Abdominal radiograph and coronal reformatted image in an 81-year-old patient with sigmoid volvulus. A dilated loop of colon is seen extending from the pelvis towards the left upper quadrant (**A**, arrows) with a resultant coffee bean sign (**A**, arrows and arrowheads). The subsequent CT demonstrates a 'beak sign' (**B**, arrow), 'whirl sign' (**B**, arrowhead) and collapse of the proximal loop (**B**, curved arrow).

Figure 14.

Coronal reformatted CT image in a 26-year-old cystic fibrosis patient with distal intestinal obstruction syndrome. Inspissated fecal-type contents are seen in the distal ileum (arrows) resulting in proximal bowel obstruction and dilated loops. High-density contents are seen in the non-dilated small bowel distal to the obstruction from a prior investigation (arrowhead).

Figure 15. A-C

Abdominal radiograph and axial CT images on a 59-year-old patient with gallstone ileus. Dilated small bowel loops are seen (**A**, arrows) along with pneumobilia (**A&B**, curved arrows) on the radiograph and CT. The non-calcified gallstone was identified in the lower abdomen (**B**, arrowhead).

Figure 16.

Coronal T2-weighted fat saturated MRI image in a 65-year-old male patient with small bowel obstruction. Dilated small bowel (arrows) is seen superior to a shouldered circumferential lesion (arrowheads) with collapsed bowel distally (curved arrow). Histology following surgical resection confirmed adenocarcinoma.

Figure 17.

Axial CT image from a 67-year-old patient with small bowel obstruction from an incisional hernia. Dilated bowel loops (arrowheads) are seen proximal to a transition point 'beak sign' (arrow) within the hernia neck (curved arrows).

Figure 18.

Coronal reformatted CT image on a 49-year-old female patient with small bowel obstruction from a left inguinal hernia. Dilated small bowel loops (arrows) are seen proximal to the direct left inguinal hernia (arrowhead) that lies medial to the inferior epigastric vessels (curved arrow).

Figure 19.

Axial CT image from a 71-year-old female patient with small bowel obstruction from a right femoral hernia. The hernia sac (arrow) is seen medial to the femoral artery and vein (arrowhead).

Figure 20.

Sagittal reformatted CT image from a 47-year-old patient with acute abdominal pain from a Meckel's diverticulitis. The blind ending dilated bowel diverticulum is evident (arrows) with surrounding fat stranding. The afferent and efferent small bowel loops are seen (arrowheads) along with the normal high-lying appendix (curved arrow).

Figure 21.

Ultrasound image on an 18-year-old male with acute appendicitis. The appendix (arrows) measures almost 10 mm in diameter and was non-compressible with surrounding echogenic fat.

Figure 22.

Axial CT image on a 41-year-old patient with acute appendicitis. The dilated appendix, seen in cross section (arrowhead), is enlarged, has luminal obliteration and demonstrates mural enhancement. Surrounding inflammatory fat stranding is also evident (arrows).

Figure 23.

Coronal reformatted CT image from a 41-year-old oncology patient with pseudomembranous colitis involving the entire colon. Diffuse wall thickening, with submucosal edema (arrows), an elongated target sign in the left iliac fossa, mesenteric hypervascularity (arrowheads) and ascites (curved arrows) are shown.

Figure 24.

Coronal reformatted CT image on a 64-year-old patient with acute infectious (coliform) colitis demonstrating the 'accordion sign' (arrowheads) where the positive oral contrast is interspersed between the enlarged haustral folds.

Figure 25.

Coronal reformatted CT image from a 67-year-old oncology patient with typhlitis. Wall thickening involving the terminal ileum (arrowheads), cecum and ascending colon (arrows) is demonstrated.

Figure 26.

Axial CT image in a 49-year-old female with acute diverticulitis. Multiple diverticula are visible (arrows) along with surrounding fat stranding (curved arrow) and a peri-colic abscess (arrowheads).

Figure 27. A-B.

Axial CT images on 75-year-old male patient with severe complex pelvic sepsis from acute diverticulitis. Large abscesses are seen in the pelvis (**B**, arrows), extending into the buttock via the sciatic notch. In addition, gas is seen in the

spinal canal and right paraspinal muscles (**A**, arrowheads) as a result of local spreading of infection.

Figure 28.

Axial CT image on a 29-year-old male with right sided epiploic appendagitis. The typical central ovoid fat density is seen with a surrounding halo of inflammation (arrows).

Figure 29. A-B.

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

Figure 30.

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

Figure 31.

Axial CT image from a 41-year-old patient with a perforated ulcer in the first part of the duodenum. A defect is seen in the medial duodenum (arrows), a large

collection is seen in the lesser sac posterior to the stomach (arrowheads) and further pneumoperitoneum is also seen anteriorly (curved arrows).

Figure 32.

Axial CT image from a 51-year-old male with a sigmoid perforation secondary to an ingested chicken bone (arrow). Considerable stranding is seen adjacent to the foreign body as well as an anterior pneumoperitoneum (arrowhead).