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Pictorial Review

Infections and infestations of the gastrointestinal tract. Part 1: Bacterial, viral and fungal infections

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The purpose of this article is to review the imaging findings of various infections affecting the gastrointestinal tract. Barium examinations, computed tomography (CT), magnetic resonance imaging (MRI), and ultrasonography all play an important role in the diagnostic workup of gastrointestinal tract infections. Knowledge of differential diagnosis, sites of involvement, and typical imaging features of different infections can help in accurate diagnosis and guide treatment.

Introduction

Gastrointestinal (GI) tract infections and infestations are relatively less common in the western world as compared to the developing countries, but may be encountered due to increased travel to endemic regions or in immigrant populations.[1] Furthermore GI tract infections may also be seen in immunocompromised patients. The presenting clinical findings may be similar to that of inflammatory bowel disease, and it is essential to distinguish these two as the treatment options are different. Imaging, particularly barium studies, ultrasonography (US), computed tomography (CT), and magnetic resonance imaging (MRI) play an important role in the diagnostic evaluation of infections and infestations of gastrointestinal tract, including the development of complications. Knowledge of the radiological patterns of involvement in different infectious diseases can be helpful in formulating an accurate diagnosis. This review illustrates the radiological findings in various gastrointestinal tract infections and infestations. Bacteria, fungi, or viruses may cause infections, whereas several parasitic species may cause infestations of the GI tract. The wide variety of GI tract infections and infestations is listed in Table 1. Imaging techniques, bacterial, viral, and fungal infections are reviewed in the first part of this review, whereas parasitic infestations and other miscellaneous infections are detailed in the second part.

Imaging techniques

Barium examinations

Barium examinations are often used in patients with suspected small bowel disease as they easily demonstrate irregularities of fold pattern and peristaltic abnormalities that are not readily appreciated using other imaging techniques.[2,3] Many infections and infestations may produce ulcerations and nodularity of the luminal surface, which are also best seen on these examinations. Certain parasites may often be seen as filling defects outlined by barium. Therefore, meticulously performed barium examinations still play a very important role in the diagnosis of intestinal infections and infestations as compared with other techniques.

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Table 1
Gastrointestinal tract infections and infestations.

Infections			Infestations		Miscellaneous
Bacterial	Viral	Fungal	Helmiths	Protozoans	
Mycobacterium tuberculosis	Herpes CMV	Candidiasis	Cestodes (tapeworm) - Taenia species	Amoebiasis	Tropical sprue Typhlitis *Closteridium difficile*
Yersinia enterocolitica			Nematodes (roundworm) Ascariasis, Strongyloides, Trichuriasis, Anisakiasis	Chagas disease	
Campylobacter jejuni			Trematodes (flukes) Schistosomiasis	Giardiasis	
Salmonella				LGV	
Actinomycosis					
Brucellosis					

CMV, cytomegalovirus; LGV, Lymphogranuloma venerum.

Many infections and infestations may produce a non-specific malabsorptive pattern in the small bowel. Although this pattern is not specific, it may provide a clue to an underlying GI tract abnormality.[4] Various radiological patterns are associated with this malabsorption pattern (Fig 1). Flocculation of barium is seen due to decreased intestinal pH caused by excess mucus production by goblet cells; segmentation can be seen as areas of stasis of with multiple to-and-fro peristalsis of the barium column; multiple fluid levels, smudging, and loss of mucosal detail may also be present as mucus prevents an adequate coating of the mucosa by the barium. A tubular, featureless bowel may be seen and this is known as the "moulage sign". This is due to barium being diluted and outlining large, fluid-containing hypotonic or atonic bowel segments.

Ultrasonography (US), CT and MRI

US is particularly useful for assessing the ileocaecal region, a region commonly affected by several bacterial infections. US can also be used to differentiate infections from inflammatory changes of Crohn's disease. Diagnostic or therapeutic lavage or drainage of abscesses secondary to infections can also be performed under US guidance.

CT and MRI are useful as they can provide detailed assessment of not only the bowel wall, but also extra-luminal disease such as lymphadenopathy, mesenteric and peritoneal inflammation, and other associated findings such as hepatosplenomegaly. CT and MRI are also useful in assessing extra-intestinal complications associated with GI tract infections.

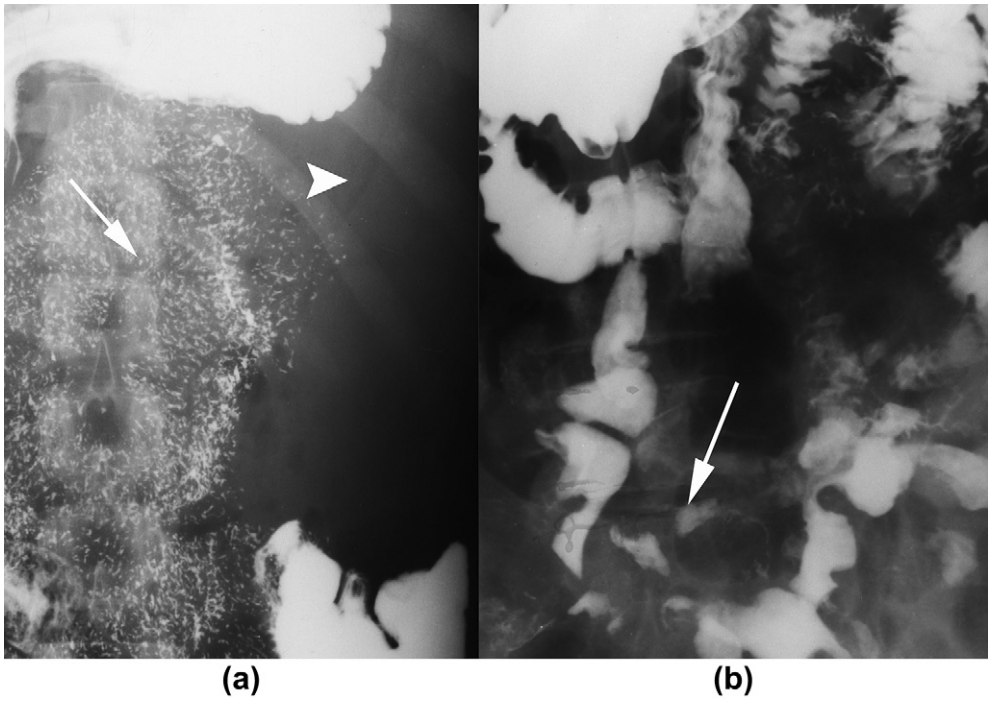
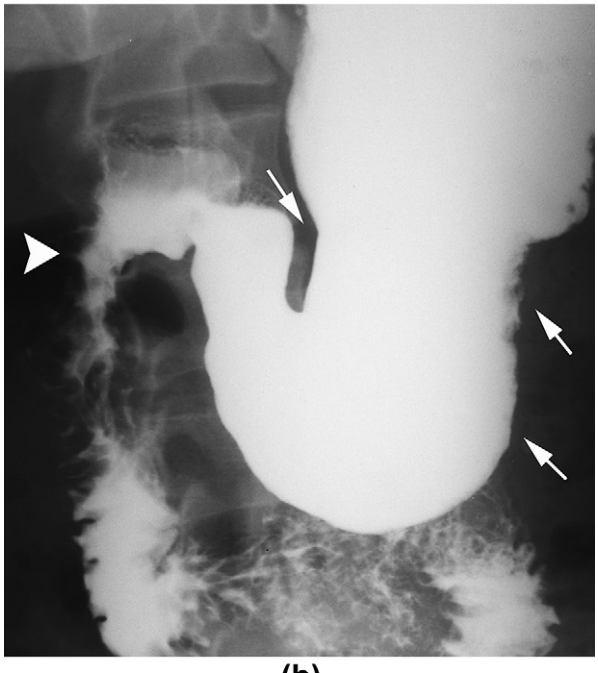


Figure 1 Malabsorptive patterns. (a) Barium examination shows marked flocculation of barium (arrow) within a short period of time after ingestion (barium is still present in the stomach) in a patient with tropical sprue. Note massive splenomegaly (arrowhead) displacing bowel loops. (b) Barium examination shows loss of normal small bowel pattern with areas of segmentation and break up of the barium column (arrow).

(a)



(b)

Infections and infestations (Table 1)

Bacterial infections

Tuberculosis (Figs 1–4)

Tuberculosis involves the GI tract by ingestion of infected sputum or bovine bacillus, via the haematological route, or by local spread. Although it can affect any part of the GI tract, the ileocaecal junction is the most commonly affected site due to the abundance of lymphoid follicles (Peyer patches). Pain, weight loss, fever, palpable masses, dysphagia, and ascites are the clinical presentations.[5]

Oesophageal involvement is typically in the upper third, most often by direct erosion from the adjacent mediastinal node.[6] Barium studies demonstrate mucosal irregularity, ulcers, and plaque formation in early stages, while fistulae and strictures are seen in chronic stages. Extrinsic indentation may be seen due to mediastinal lymphadenopathy, while traction diverticula may be seen post-cicatrization, especially at the level of carina.[7] Involvement of the stomach and duodenum is less common due to sparse lymphoid tissue, highly acidic peptic secretions, and rapid passage of ingested organisms into small bowel. If involved, gastric or duodenal tuberculosis may manifest as areas of deep ulcerations, most frequently on the lesser curvature of the antrum or in the pyloric region.[8] Ulcers near the pylorus may cause outlet obstruction and cicatrization due to ulceration can lead to an irregular or narrowed gastric contour, simulating a linitis plastica or primary scirrhous carcinoma of the stomach.

Most commonly, the ileocaecal region is involved and typically the caecal involvement exceeds ileal disease. The earliest findings of intestinal tuberculosis are spasm and oedema of the ileocaecal junction with abnormal peristalsis that is best observed on fluoroscopy.[6] Barium studies demonstrate nodular, thickened, and distorted folds at the ileocaecal junction in the early stages of the disease.[9,10] Small ulcerations and transaxial or “girdle ulcers” are seen that progress to hourglass strictures (Fig 2). Girdle ulcers are oval, perpendicular to the long axis of the bowel, and are typical of tuberculous infections.[6] Deep fissures, sinus tracts, enterocutaneous fistulae, and perforation can occur, although less commonly than in Crohn’s disease. In advanced disease, the caecum may be retraced superiorly with widening of the ileocaecal angle. The classical appearance of intestinal tuberculosis is that of a conical retracted caecum with a narrow ulcerated ileum.[8–10] Cross-sectional imaging demonstrates thickening of the bowel wall and mesenteric and peritoneal inflammation with necrotic lymphadenopathy.[11–13] Irregular, shaggy enhancement of the bowel wall may be seen on CT and

Figure 2 A 30-year-old woman with proven GI tract tuberculosis presenting with dysphagia, early satiety, and vomiting. (a) Axial CT image shows thickened oesophagus compressed by a necrotic mediastinal lymph node (arrow). (b) Barium study shows narrowed contour of the distal gastric body and antrum (arrows) as compared to the normally distended fundus, simulating linitis plastica. There is spasm and narrowing of the second part of the duodenum (arrowhead).

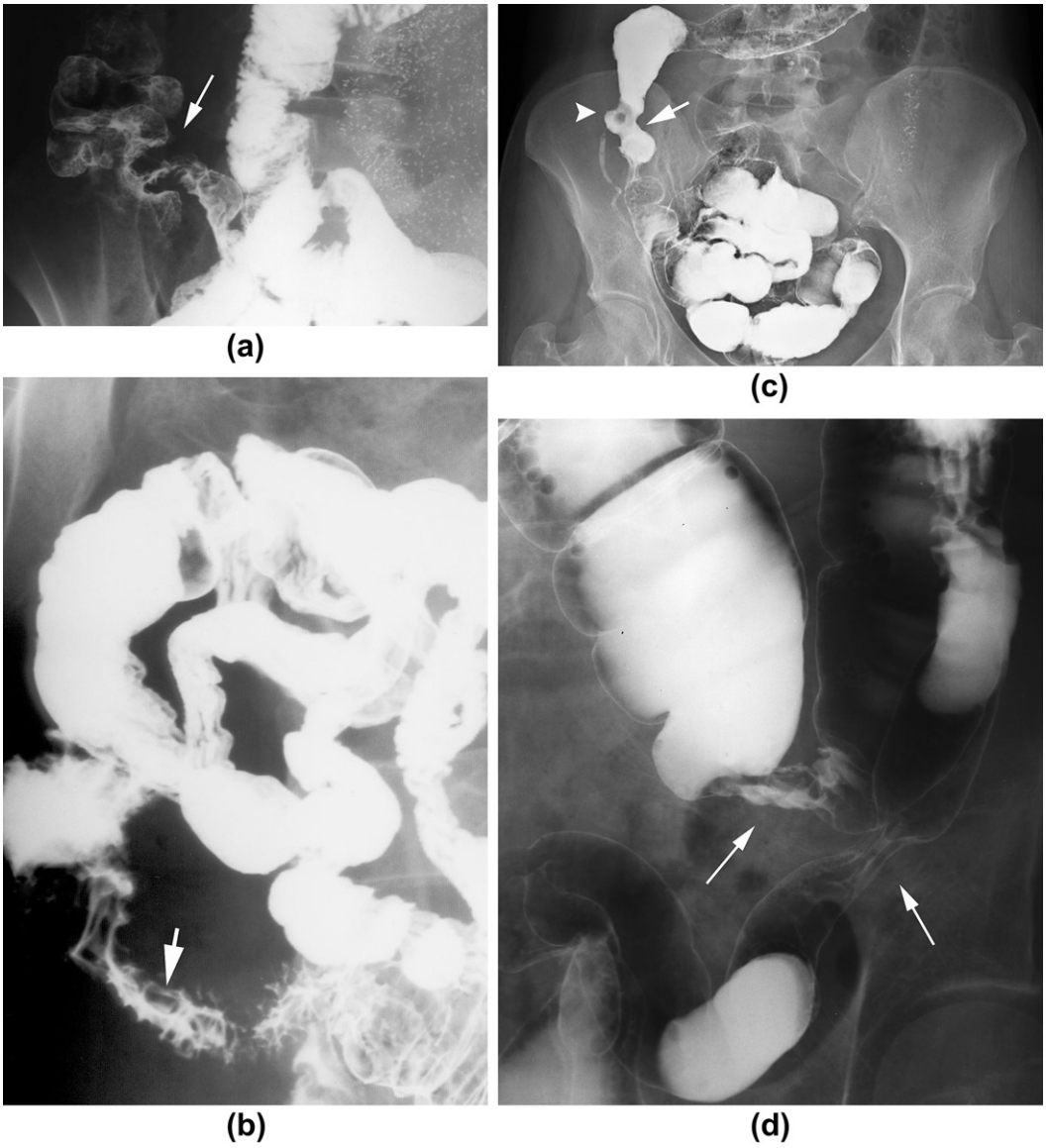


Figure 3 Barium examination findings in GI tract tuberculosis. (a) Thickening and oedema is present at the ileocaecal junction (arrow) and medial caecal wall in a patient with early ileocaecal tuberculosis. (b) Ileal involvement shows narrowing and irregularity of the terminal ileum with multiple, large, oval ("girdle") ulcers in the transaxial plane (arrows). (c) Advanced disease shows a conical retracted caecum (arrowhead) and patulous, widened ileocaecal junction (arrow). The appendix is seen attached to the contracted caecum. Note granular and polypoid changes in the mucosa of the transverse colon. (d) Chronic disease with tapered "hourglass" strictures in the colon (arrows).

MR examinations (Fig 4). Asymmetric thickening and enhancement of the terminal ileum and medial wall of the caecum may also be present. Peritoneal disease and ascites are often associated with ileocaecal tuberculosis.[10] The most common type of peritoneal disease is the so called "wet type" that manifests as large amounts of viscous ascitic fluid that shows high attenuation at CT due to its high protein and cellular content.[11] Peritoneal and omental thickening and enhancement may also be present (Fig 4). The fibrotic fixed type of peritoneal disease consists of large omental masses, which cause matted loops of bowel and tethering of bowel loops. In chronic cases, multiple strictures may be present that may lead to intestinal obstruction. Colonic involvement is more common on the right side (ascending and transverse colon) and is usually associated with ileal disease. Radiological findings include narrowing, ulceration, and mucosal granulation producing nodularity and polypoid changes.[14] Smoothly tapered, "hourglass" strictures may be seen. Tuberculosis is a well-recognized cause of rectal strictures in the Asian population.[15] Isolated rectal involvement is rare and may be mistaken for rectal malignancy.[6]

The main differential diagnosis for GI tract tuberculosis is Crohn's disease. Differentiating between these entities is important as corticosteroids are used for treating Crohn's disease that can provoke fulminant, catastrophic infection in patients with tuberculosis. Ulcers in tuberculosis tend to be axial (girdle), large, or oval. Linear, longitudinal ulcers of Crohn's disease are not seen. Contraction of the caecum and prominence of caecal over ileal changes suggest tuberculosis, whereas the ileum is predominantly involved in small

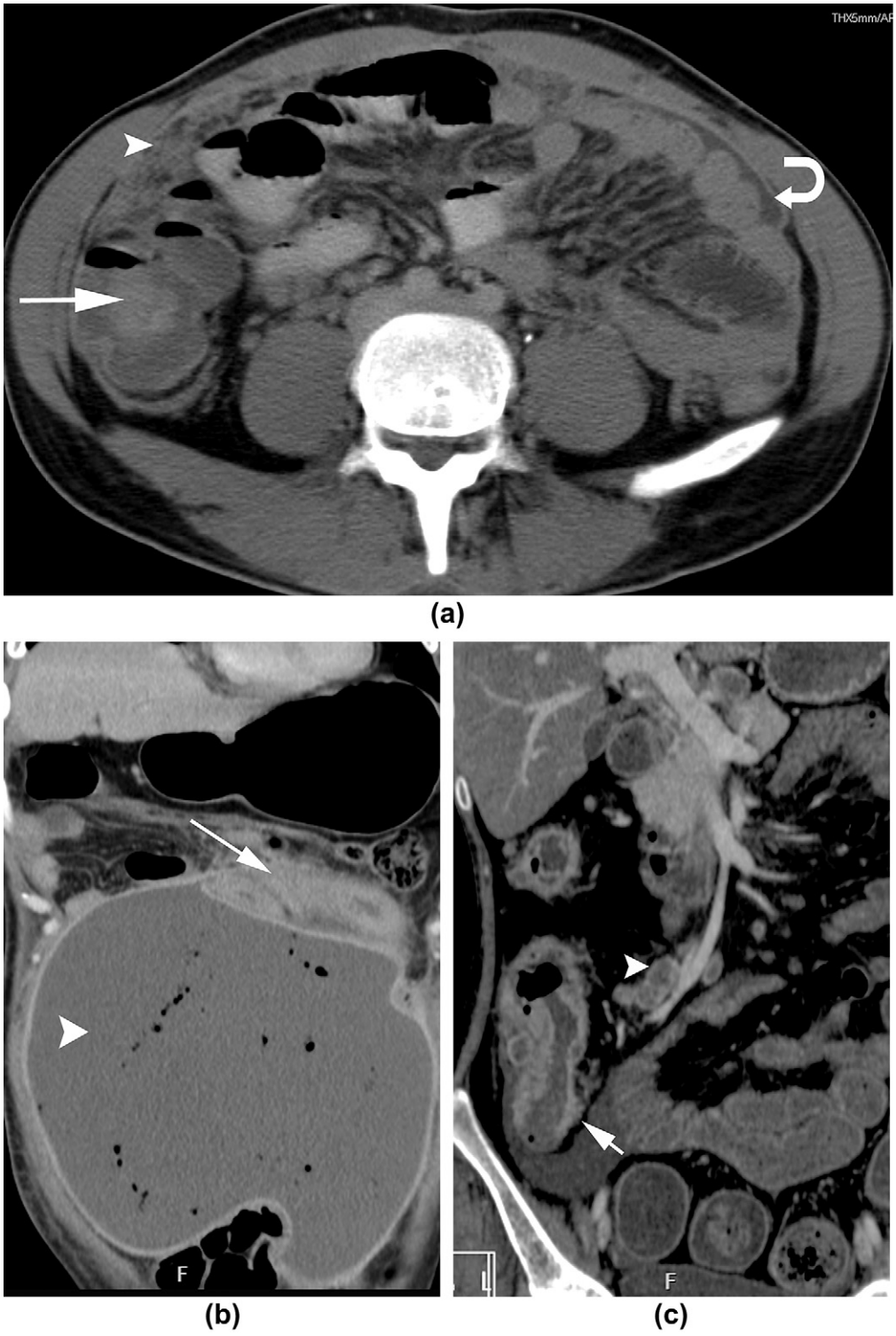


Figure 4 CT findings in GI tract tuberculosis. (a) Tuberculosis of the ileocaecal region with thickening and enhancement of the terminal ileum (arrow) omental and peritoneal thickening, nodularity and enhancement (arrowhead), and fluid collection with enhancing margins (curved arrow). (b) Intestinal tuberculosis showing enhancing thickened jejunal loops (arrow) and peritoneal "wet-type" disease manifesting as a large, high-attenuation fluid collection with enhancing rims. (Gas bubbles are present due to a recent percutaneous drain placement.) (c) Coronal CT image shows asymmetrical thickening and irregular enhancement of the terminal ileum (arrow) and necrotic lymph nodes (arrowhead) along the ileocolic blood vessels.

intestinal Crohn's disease. Ascites and necrotic lymph nodes are commonly seen in tuberculous infections, but are uncommon in Crohn's disease. Fat proliferation of the mesentery around the affected bowel is indicative of Crohn's disease rather than tuberculosis. If imaging findings are inconclusive, laparoscopy with a targeted biopsy can be considered as the most rapid and specific method for diagnosing GI tract tuberculosis.

Yersinia (Fig 5)

Yersiniosis is caused by *Yersinia enterocolitica* or *Yersinia pseudotuberculosis*, and typically affects the ileocaecal region. It is more common in children or young adults and may mimic acute appendicitis. In young patients *Yersinia* infection produces mild enteritis and results in small focal ulcerations in the small bowel. In adults it may present with acute or protracted fever, pain, and diarrhoea for periods lasting up to 4–6 weeks.[16]

Barium examinations show thickened intestinal folds with aphthous ulcers. Occasionally larger and deeper ulcers may also be present. US findings include increased prominence of submucosal layer of the terminal ileum.[17] The haustral folds of the ascending colon may appear prominent due to spasm and contractions leading to an "accordion" appearance. Typically the total bowel wall thickness is not significantly altered, although the submucosal layer may appear prominent. Associated lymphadenopathy may also be present. The lack of significant bowel thickening, mesenteric fatty proliferation, fistulae, or linear ulcers help to distinguish it from Crohn's disease.

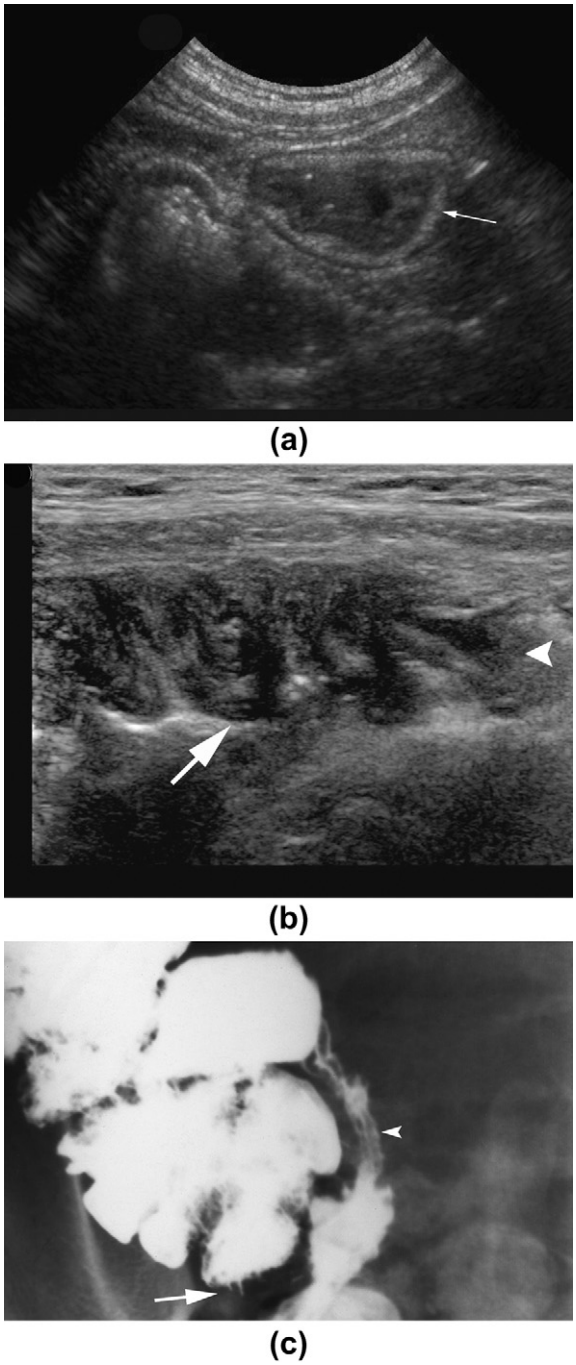


Figure 5 A 35-year-old woman with proven *Yersinia* infection. (a) Axial US image shows marked submucosal thickening and echogenicity (arrow) with lack of any significant mesenteric inflammatory changes. (b) Longitudinal US image shows prominent haustrations in the ascending colon (arrow) forming an accordion appearance. The arrowhead indicates the terminal ileum. (c) Barium study shows small aphthous ulcers in the terminal ileum (arrowhead) and deeper ulcers in the caecum (arrowhead). Note lack of any significant oedema, caecal retraction, or separation of bowel loops (mesenteric fatty proliferation).

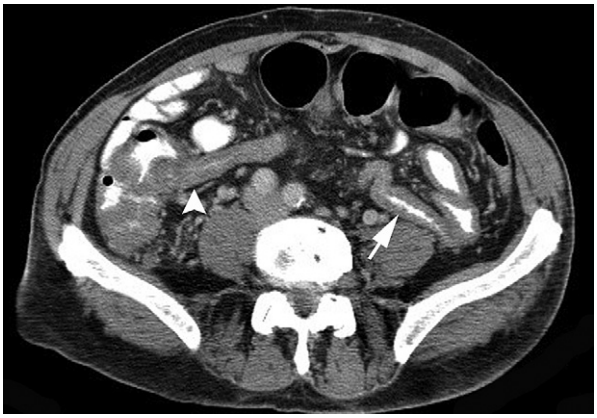


Figure 6 A 55-year-old man with *Campylobacter* enteritis. Axial CT image shows diffuse thickening of the small bowel (arrow) and the ileocaecal region (arrowhead). Nodularity is seen within the small bowel thickening (arrow). Note diffuse small bowel and caecal involvement with lack of mesenteric inflammation, fluid, or fibrofatty proliferation.

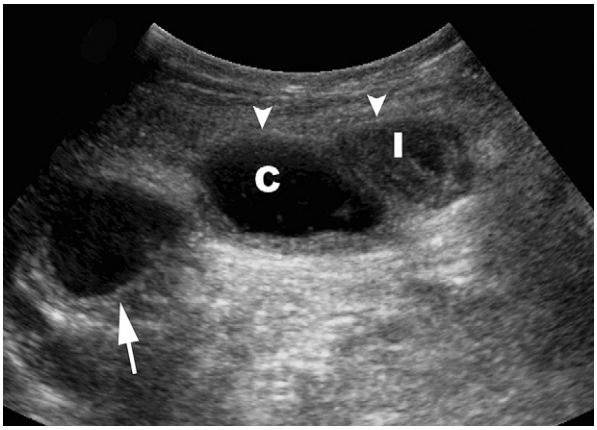


Figure 7 A 47-year-old man with typhoid fever. A distended, nontender appendix is seen (arrow) adjacent to ileocaecal junction (arrowheads). C, caecum; I, terminal ileum.

Campylobacter (Fig 6)

Campylobacter jejuni is a Gram-negative, micro-aerophilic bacteria. Ingestion of food contaminated with this bacteria results in diffuse, exudative enteritis with ulceration, oedema, and haemorrhage.[6] Clinical features include diarrhoea, fever, and pain and the symptoms may mimic acute appendicitis.[18] Barium examinations show ulceration and nodularity of the mucosal folds, most commonly in the distal ileum and the colon.[18–20] Pancolitis with diffuse granularity and loss of haustration simulating ulcerative colitis may also be seen. Cross-sectional imaging shows marked thickening of the bowel wall and submucosal

Figure 8 A 33-year-old woman with pyrexia and abdominal pain. (a) Axial CT shows multiple abscesses in the abdominal wall (arrowheads) adjacent to the descending colon (arrow). (b) Coronal CT image shows an infiltrative mass (arrow) ascending from the pelvis involving bowel loops with flank abscess (arrowhead). Note the radiodense IUD in the uterus and hydronephrosis due to ureteric involvement. (c) A sonogram shows the irregular appearance of the descending colon with a fistulation (arrow).

oedema.[19] Although the radiological findings may be indistinguishable from other colitides or *Yersinia* infection, the extensive involvement of the small bowel (jejunum and ileum) is not usually seen in *Yersinia*. Rarely there may be serious complications such as toxic megacolon and massive bleeding. *Campylobacter pylorus* is another organism that is known to cause gastric inflammation and marked thickening of gastric rugal folds in the paediatric population.[21]

Salmonella (Fig 7)

Salmonella infection is caused by ingestion of food contaminated with the Gram-negative bacteria, *Salmonella typhi* or *Salmonella paratyphi*. Diarrhoea, high-grade fever, and abdominal pain are the clinical presentations associated with typhoid fever.

The diagnosis of salmonella enteritis is confirmed by stool culture, with radiology playing a limited role.[6] Abdominal radiographs may show dilated bowel loops with fluid levels. Barium studies show mucosal ulceration in the small bowel, especially in the distal ileum along the anti-mesenteric border.[22] Pneumoperitoneum may be present in cases with perforated ulcers. US may reveal a non-tender, dilated, thickened appendix and gallbladder with associated mesenteric nodes and splenomegaly.[13]

Actinomycosis (Fig 8)

Actinomycosis is caused by *Actinomyces israeli*, an anaerobic bacterium that is part of the normal flora of the bowel but can cause infection following trauma, surgery (particularly appendicectomy), or placement of intrauterine devices (IUDs).[23] Vague abdominal pain, diarrhoea, and a palpable abdominal mass are the clinical findings. The ileocaecal junction region and appendix are most commonly involved, usually after appendicitis or surgery.[23] Actinomycosis secondary to pelvic inflammation most commonly ascends from a uterus that has had an intrauterine contraceptive device in place for several years. In these cases, the rectosigmoid is the most common site of involvement.[24] The disease typically presents as a slowly growing, infiltrative mass. Barium studies show distortion, and strictures with or without fistulae arising from the mass. Involvement of any abdominal organ and abdominal wall can occur by direct spread, with eventual formation of draining sinuses. CT usually reveals an infiltrative mass with focal areas of decreased attenuation that enhance with contrast medium with a tendency to invade surrounding tissues.[23,24] Bowel thickening is typically concentric and involves long segments and, therefore, there is considerable overlap of appearances with Crohn's disease, although surrounding lymphadenopathy and fat proliferation is uncommon. However, the most typical finding of actinomycosis is that of a cystic or solid mass adjacent to the bowel with intense enhancement due to abundant fibrous and granulation tissue.

Brucellosis (Fig 9)

Brucellosis is a zoonosis caused by Gram-negative *Brucella* species. It predominantly involves the musculoskeletal system with exuberant bony sclerosis and osteophyte formation.[25] Gastrointestinal tract is very rarely involved and imaging appearances mimic tuberculosis with involvement of the ileocaecal region and associated flank or paraspinal abscesses.

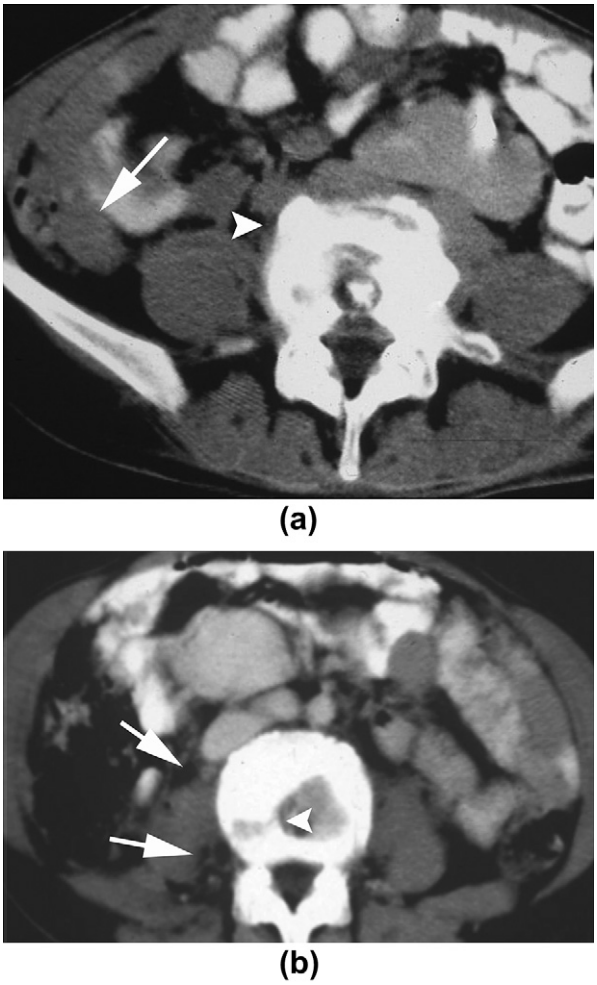


Figure 9 A 35-year-old male dairy worker with back pain and diarrhoea. Brucellosis was confirmed on follow-up. (a) Axial CT image shows non-specific thickening of the terminal ileum. Note exuberant bony sclerosis and osteophyte formation in the lumbar spine (arrowhead). (b) Axial CT image at higher level shows hypodense paraspinal abscesses (arrows) and vertebral body osteomyelitis (arrowhead).

Viral infections

Cytomegalovirus (Fig 10)

Cytomegalovirus (CMV) is a member of the Herpesviridae family, along with herpes simplex viruses 1 and 2, Epstein–Barr virus, and varicella-zoster virus. CMV infections usually occur in immunocompromised patients and results in vasculitis that may cause focal ischaemia, ulceration, bleeding, and perforation.[26,27] The oesophagus, stomach, and colon are the most commonly involved. Oesophageal infection presents with dysphagia and barium studies show discrete superficial ulcers in the mid or distal

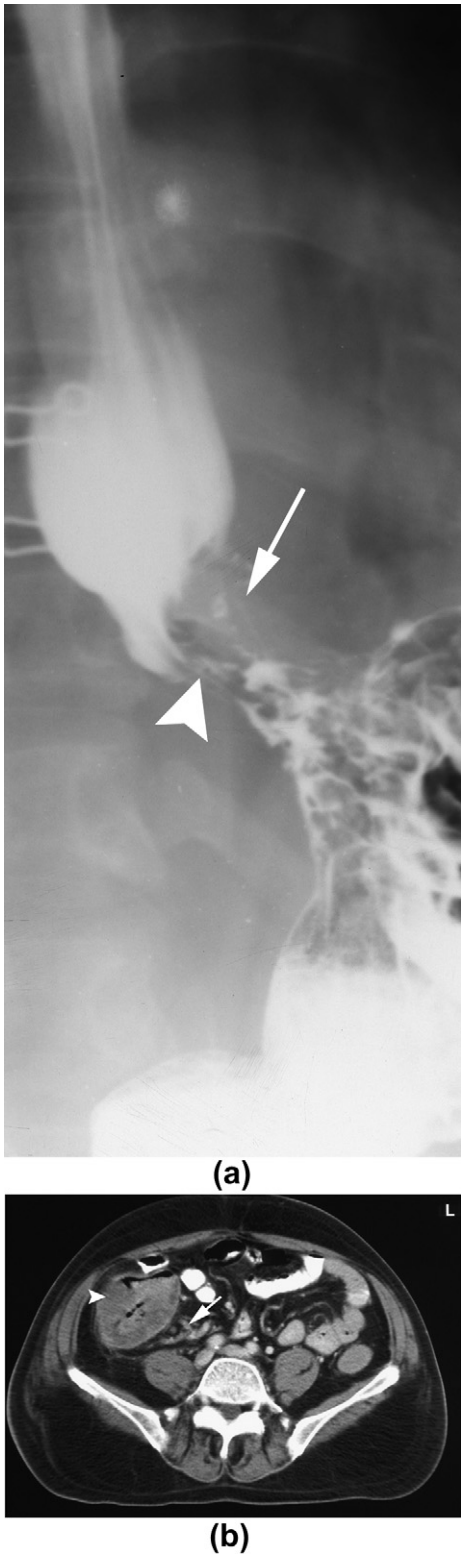


Figure 10 CMV infection. (a) Barium swallow examination shows an ulcer (arrow) in the distal oesophagus with surrounding plaque-like mound of oedema (arrowhead) (b). Axial CT image shows symmetrical thickening and submucosal oedema of the caecum (arrowhead) with engorged ileocolic blood vessels (arrow) in a patient with CMV ileocolitis.

oesophagus. Giant ulcers (>1 cm) may occur that are ovoid and elongated and show collection of barium surrounded by plaque-like oedema. Shallow ulcers or erosions can lead to gastritis and duodenitis, which results in mucosal nodularity, thickened folds, and irregular antral wall thickening. CMV colitis may mimic ulcerative colitis demonstrating mucosal granularity, submucosal oedema, and wall enhancement.[26] Diagnosis is confirmed by demonstrating inclusion bodies on the endoscopic biopsy specimen. The caecum and the ascending colon are most frequently affected by colitis, although a severe infection can result in pancolitis. CT findings include concentric thickening of the colonic wall, narrowing of the intestinal lumen and pericolic inflammatory changes.[27,28] Abdominal lymphadenopathy is uncommon in CMV infections.

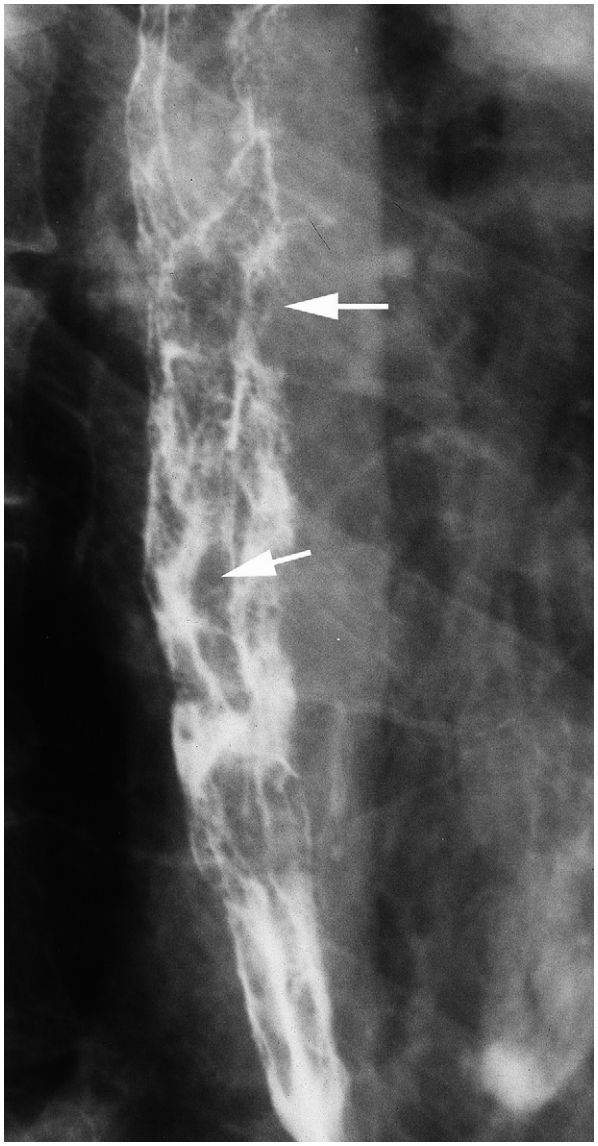


Figure 11 Herpes oesophagitis. Barium swallow examination shows multiple punctate ulcers in the proximal oesophagus (arrows) with surrounding mound of oedema.

Herpes (Fig 11)

Herpes simplex type 1 infections occur in immunocompromised patients but occasionally may occur as an acute self-limited disease in normal individuals. Lesions can be seen in oropharynx or oesophagus. Substernal pain and odynophagia are common clinical presentations. Barium studies demonstrate multiple, small (<1 cm) superficial ulcers in the upper or mid-oesophagus, which may be punctuate or linear with surrounding radiolucent mound of oedema.[29] Advanced disease results in extensive ulceration and plaque formation, which might be difficult to distinguish from candidiasis.

Fungal infections

Candidiasis (Fig 12)

Infections with *Candida albicans* is usually seen in chronically immunocompromised patients and presents with dysphagia and odynophagia. Barium studies demonstrate plaque-like lesions with longitudinal orientation in the upper and mid-oesophagus. In severe disease, large plaque-like filling defects may coalesce producing a "cobblestone" appearance with shaggy contours.[30] Fulminant candida oesophagitis may be seen in patients afflicted with human immunodeficiency virus (HIV) leading to complications such as obstruction, perforation, and fistula formation.

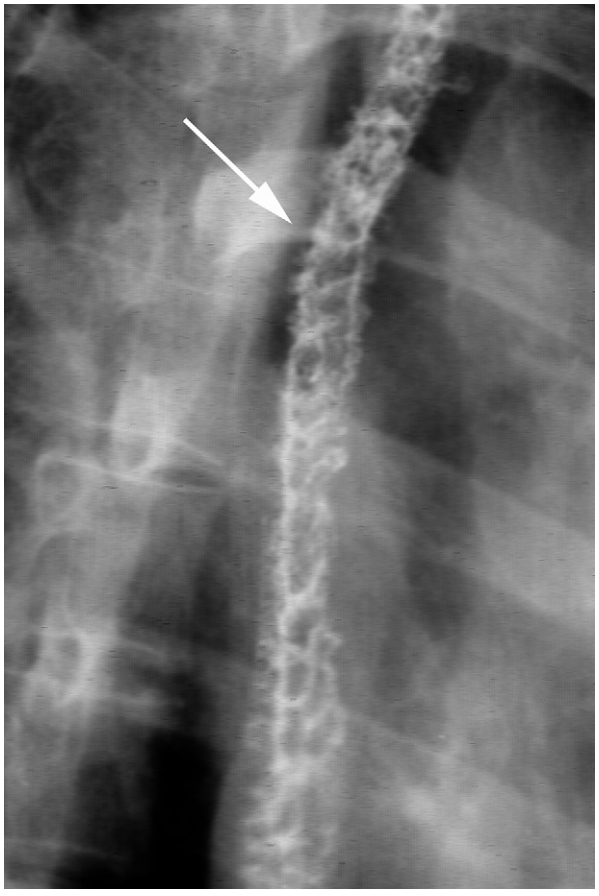


Figure 12 A 34-year-old immunocompromised patient with dysphagia. Barium swallow examination shows an irregular, shaggy oesophagus with plaque-like filling defects forming a cobblestone appearance due to ulcerating candidial oesophagitis.

Conclusion

Bowel infections and infestations produce a wide variety of radiological appearances. Imaging appearance depends on the location of bowel involvement and also the natural history of disease. Barium examinations play an important role in the diagnostic workup of GI tract infections. Knowledge of the location of intestinal infections, aetiology, and imaging appearances are crucial in making the right diagnosis.

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