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TB or not TB: A comprehensive review of imaging manifestations of abdominal tuberculosis and its mimics

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ABSTRACT

The ever-growing prevalence of tuberculosis is a cause for concern among both developing and developed countries. Abdominal tuberculosis is the most common site of extrapulmonary tuberculosis and involves almost all of the visceral organs. Clinical presentation of abdominal tuberculosis is often non-specific. Thus, having a high index of clinical suspicion is necessary to aide early diagnosis and guide prompt initiation of appropriate treatment. In this review, we focus on the entire spectrum of abdominal tuberculosis and other diseases mimicking it with an emphasis on their imaging findings.

1. Introduction

In 2018, tuberculosis (TB) affected an estimated 10 million people worldwide, the majority of which were from developing countries.[1] Tuberculosis poses a major health concern in not only developing countries but also developed countries due to the impact of globalization and emigration, and the association of TB with human immunodeficiency virus (HIV) infection.[2] Abdominal involvement is the most common extrapulmonary manifestation of TB, accounting for about 5% of all tuberculosis cases worldwide.[3–5]

Abdominal tuberculosis may involve the lymph nodes, gastrointestinal system, peritoneum, and solid organs. It can occur as a consequence of reactivation of latent tuberculosis, ingestion of *Mycobacterium tuberculosis* (via infected lung secretions, unpasteurized dairy products or undercooked meats), hematogenous spread from an adjacent focus, or through infected lymphatic channels and nodes.[6] Risk factors for developing abdominal TB include underlying medical conditions such as cirrhosis, diabetes mellitus, HIV infection, renal insufficiency, and malignancy; medical treatment with steroids and anti-tumor necrosis factor (TNF) agents; and others such as malnutrition, tobacco smoking, intravenous drug use and alcoholism.[4,7–12] In general, clinical features are non-specific and may include fever, weight loss, abdominal/back pain, ascites, diarrhea, abdominal mass, bowel obstruction, and hematuria. A high index of suspicion is required to allow for early diagnosis and prompt initiation of therapy. In this review, we discuss and illustrate the clinical and radiologic features of abdominal tuberculosis and other diseases mimicking it (Table 1), and briefly discuss the management and treatment of these patients.

2. Tuberculous lymphadenitis

Tuberculous infection and inflammation of the lymph nodes, called tuberculous lymphadenitis, is the most common presentation of abdominal TB presenting in 55–66% of cases.[8,13,14] Tuberculous lymphadenitis occurs more commonly in females than males and immigrants from endemic countries with an age range of 30–40 years old.[14,15] The cervical lymph nodes are the most commonly involved group due to its proximity to the lung parenchyma.[9,16] Approximately 5.7–17.2% of cases with peripheral tuberculous lymphadenopathy also have involvement of abdominal lymph nodes.[8,13,14] The most frequently

Abbreviations: TB, tuberculosis; HIV, human immunodeficiency virus; TNF, tumor necrosis factor; GI, gastrointestinal; GU, genitourinary; ADA, adenosine deaminase; AFB, acid-fast bacillus; NAAT, nucleic acid amplification test; SAAG, serum-ascites albumin gap; TST, tuberculin skin test; IGRA, interferon-gamma release assays; PCR, polymerase chain reaction.

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Table 1
Summary of clinical imaging findings and key differential diagnoses of the spectrum of abdominal tuberculosis.

Diagnosis		Imaging findings	Differential diagnosis
Tuberculous lymphadenitis		US Enlarged hypoechoic nodes (± matted appearance) or hyperechoic nodes with posterior shadowing (due to calcifications) CT First stage: Homogenous enhancement Second stage: Central caseous necrosis and peripheral rim enhancement Final stage: Fibrosis and calcifications	• Nonseminomatous germ cell cancer • Lymphoma • Metastatic nodes • Fungal infections • Celiac disease
Gastrointestinal tuberculosis	Esophagus	Endoscopic US Asymmetric wall thickening with loss of wall stratification, mucosal ulceration and adjacent LAD CT Irregular diffuse wall thickening, fistulous tracts, diverticula, or strictures with LAD	• Esophageal cancer
	Stomach & duodenum	CT Irregular diffuse wall thickening (most commonly at the antrum and distal body) with ulceration, fibrosis, sinus and fistula tracts (rare) and LAD	• Gastric scirrhous adenocarcinoma • Pancreatic cancer of the head • Metastatic disease • Chron's disease • Infectious causes (syphilis)
	Small intestine & colon	CT Diffuse wall thickening of affected segment (predominantly in the terminal ileum and cecum) with surrounding fat stranding and LAD	• Primary cecal malignancy • Chron's disease • Backwash ileitis (from ulcerative colitis) • Infectious causes (*Yesinia* and amebiasis)
Hepatosplenic tuberculosis		US Multiple variable size hypoechoic lesions Late stage: calcifications CT Multiple variable size hypodense lesions with central or peripheral post-contrast enhancement, LAD, ascites and peritonitis Late stage: Calcifications MRI Multiple variable size lesions with T1-weighted hypointense and T2-weighted isointense to hyperintense signal showing diffusion restriction with central or peripheral post-contrast enhancement, LAD, ascites and peritonitis	• Primary malignancy • Pyogenic abscess • Metastasis • Fungal infections (*Candida*, *Aspergillus*)
Genitourinary tuberculosis	Kidney	CT Early: Focal hypo-perfusion on contrast-imaging Late: Multiple strictures resulting in uneven caliectasis Healed/chronic: Renal atrophy, progressive hydronephrosis, and dystrophic calcifications ("putty kidney")	• Transitional cell/squamous cell carcinoma • Medullary sponge kidney • Papillary necrosis • Chronic pyelonephritis
	Ureter	CT Early: Irregular thickening of the ureteric wall with luminal narrowing and periureteric inflammatory changes Late: Strictures, ulceration and fibrosis leading to a corkscrew ureter and proximal hydroureteronephrosis Chronic: Pipe-stem ureter and foreshortening of ureter	• Ureteral stones • Infectious causes (schistosomiasis)
	Bladder	CT Early: Shrunken bladder with wall thickening and surrounding fat stranding Late: Thimble bladder due to chronic scarring, calcifications (rare)	• Transitional cell carcinoma • Post-radiation cystitis • Chemotherapy (cyclophosphamide) • Infectious causes (schistosomiasis)
Genitourinary tuberculosis	Ovaries	US, CT and MRI Enlarged heterogenous ovaries with associated ascites and regional LAD	• Ovarian carcinoma
	Fallopian tubes	US, CT and MRI Multifocal constrictions and scarring resulting in a "beaded" appearance with hydro/pyosalpinx	• Salpingitis isthmica nodosa • Fallopian tube endometriosis
	Testis & epididymis	US Diffuse enlargement with heterogenous echotexture with increased vascularity or heterogenous ill-defined hypoechoic testicular lesion MRI Low signal intensity in T2-weighted sequence	• Bacterial epididymitis
	Prostate	US Hypoechogenicity and increased vascularity MRI	• Prostate carcinoma

(*continued on next page*)

Table 1 (*continued*)

Diagnosis	Imaging findings	Differential diagnosis
	Diffuse, radiating area with streaky low signal intensity on T2-weighted MRI (watermelon skin) or an enlarged heterogeneous prostate with small abscesses	
	Chronic: Diffuse dystrophic calcifications	
Tuberculous peritonitis	CT	• Disseminated peritoneal carcinomatosis
	Wet: Free or loculated ascites with nodular peritoneal enhancement and omental caking	• Ovarian carcinoma
	Fibrotic: Omental thickening and omental caking, fixed bowel loops, matted bowel and mesentery ± loculated ascites, abdominal cocoon/encapsulating peritoneal sclerosis (late stage)	• Peritoneal mesothelioma
	Dry: Fibrous adhesions of bowel loops, mesenteric thickening, and caseous mesenteric lymphadenopathy	• Non-tuberculous peritonitis
	CT and MRI	• Soft tissue tumors
Tuberculous abscess	Multiloculated peripherally enhancing abscess with surrounding fat stranding and peripherally enhancing necrotic LAD and foci of calcifications	• Tuberculoma
		• Fungal infections

Abbreviations: LAD = lymphadenopathy.

involved abdominal group of lymph nodes are omental, mesenteric, and peripancreatic lymph nodes.[17–19] Clinical presentation of abdominal tuberculous lymphadenitis in additional to constitutional symptoms are based on the site of lymphadenopathy and include abdominal pain, acute abdomen, jaundice, portal vein thrombosis and portal hypertension, renovascular hypertension, fistula formation, duodenal stricture and stenosis.[17,20–22]

Tuberculous lymphadenitis progresses through different stages which can be reflected on CT imaging. The first stage of lymphoid proliferation is marked by lymph node enlargement with homogenous enhancement. With disease progression, the central part of the lymph node undergoes caseous necrosis resulting in a centrally non-enhancing node with peripheral capsular rim-enhancement (Fig. 1). Capsular degeneration results in the fusion of adjacent lymph nodes which appear as a multilocular enhancement. In the final stage, after treatment or healing, the lymph nodes undergo fibrosis, and calcifications can be seen (refer to Fig. 5).[23] Lymph node enlargement in tuberculous lymphadenitis often demonstrates self-limited growth, with a range of 12–40 mm.[24] TB lymphadenitis can also be seen on ultrasonography as round or ovoid enlarged lymph nodes with a central hypoechoic area that may be matted.[24]

The radiological differential for the appearance of abdominal tuberculous lymphadenitis includes malignancies such as non-seminomatous germ cell tumor, pancreatic cancer, lymphoma, and metastatic lymph nodes.[19,25] In addition, fungal infections and celiac disease can also have a similar appearance.[26]

3. Gastrointestinal tuberculosis

Gastrointestinal (GI) TB is a rare manifestation of abdominal TB and is the sixth most common form of extrapulmonary TB.[27] It can affect any part of the GI tract starting from the esophagus to the rectum. The three main forms of GI TB are (i) ulcerative type (60%), which is characterized by single or multiple mucosal ulcerations commonly affecting the jejunum and ileum, (ii) ulcero-hypertrophic type (30%), which is characterized by thickening and ulceration of intestinal wall, and (iii) hypertrophic type (10%), which is characterized by scarring and fibrosis commonly affecting the ileum and cecum.[8] Complications of the three forms are similar and may include intestinal perforation, bleeding and fistula formation.[6,27] Bowel obstruction may also occur as a result of mechanical obstruction secondary to stricture formation or intestinal hypertrophy.[28]

3.1. Esophagus

Esophageal TB is very rare and is the least common site of TB involvement in the gastrointestinal tract seen in only 0.2–1% of cases.[5,6] It occurs due to the spread of infection from either the lungs, spine or infected mediastinal nodes at the level of the carina.[3,8] Esophageal involvement often manifests due to extrinsic compression from the lymphadenopathy resulting in either compression or narrowing of the esophagus.[24] In addition to constitutional symptoms, dysphagia and retrosternal pain may be present.[12,29,30]

Common radiologic findings include irregular wall thickening, mucosal ulceration, fistula formation, and traction diverticula and strictures as a result of chronic fibrotic changes (Fig. 2).[24] This ulceration can mimic esophageal malignancy with mucosal nodularity on barium imaging. CT imaging is more helpful in delineating tuberculous lymphadenopathy from the displaced esophagus and the extent of fistulous tracts.[31] Endoscopic ultrasound may show asymmetric esophageal wall thickening with loss of wall stratification.[30]

3.2. Stomach

Gastric involvement with TB is rare and seen in 0.4%–2% of cases[6] generally affecting the antrum and distal body.[3,24] Symptoms of gastric

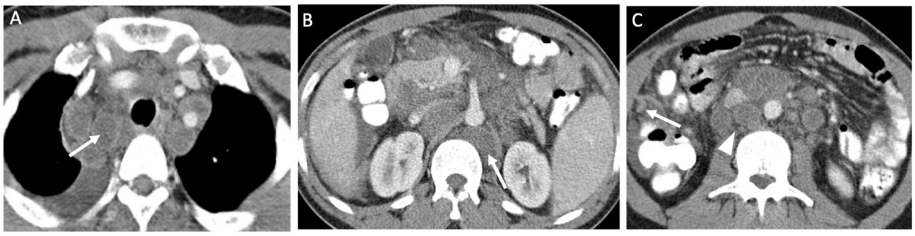

Fig. 1. Tuberculous lymphadenitis. Contrast-enhanced CT demonstrates multiple A) mediastinal, B) retrocrural, C) mesenteric/omental (arrow) and retroperitoneal (arrowhead) necrotic lymph nodes with peripheral capsular rim-enhancement and low central density. A small right-sided pleural effusion is also seen in image A.

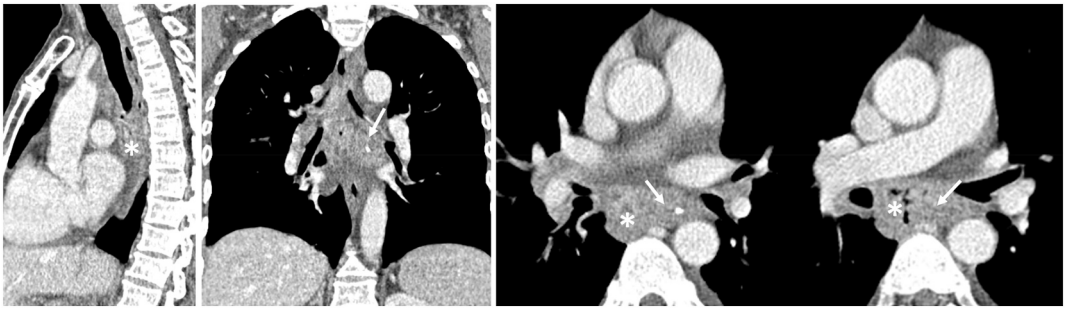

Fig. 2. Esophageal tuberculosis. A low-density heterogenous soft tissue mass (arrowhead) involving the mid-thoracic esophagus is seen, which was formed possibly from the conglomeration of irregular esophageal wall thickening and paraesophageal lymphadenopathy. Multiple enlarged and matted medistinal lymph nodes with central necrosis are seen (asterisk).

involvement include vague epigastric discomfort and upper GI bleeding. Nausea and vomiting may occur in the presence of gastric outlet obstruction secondary to antral narrowing. CT imaging can show the late-stage hypertrophic features of tuberculous pyloric stenosis.[24] The presence of sinus tract and fistula is rare but suggestive of tuberculosis. The most commonly involved parts are the gastric antrum and distal body. On imaging, the hypertrophic form can show severe and diffuse wall thickening (Fig. 3). Ulceration and fibrosis can lead to antral narrowing. Rarely, fistula and sinus tracts also occur in gastric TB.[24]

The radiological differential for the appearance of gastric TB includes malignancies such as gastric scirrhous adenocarcinoma, gastrointestinal stromal tumor, lymphoma and metastatic disease (lung, breast, pancreas), and other causes such as syphilis, sarcoidosis, peptic ulcer disease and Chron's disease.[32–35]

3.3. Duodenum

Duodenal TB is uncommon and seen in 2–2.5% of cases of TB with GI involvement. The involvement of the C-loop of the duodenum occurs due to contiguous invasion or extrinsic compression from adjacent lymph nodes.[24] Patients may present with obstructive or dyspeptic symptoms, with the former being more common. The duodenum is most commonly affected by extrinsic compression from adjacent lymphadenopathy resulting in obstruction and can be easily demonstrated on CT

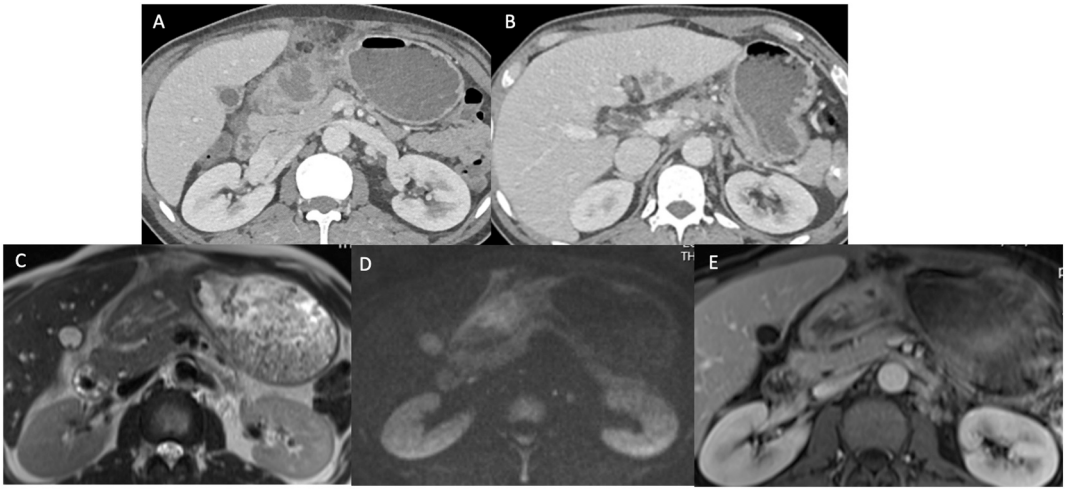

Fig. 3. Gastric tuberculosis. A and B) Contrast-enhanced CT demonstrates diffuse irregular thickening of the antrum of the stomach with surrounding fat stranding. Mild proximal dilation of stomach could be suggestive for gastric outlet obstruction. Small multi-loculated abscess is identified in the left lobe of the liver. C) T1-weighted MRI redemonstrates the diffuse irregular thickening of the gastric wall with surrounding inflammatory changes, D) diffusion restriction and E) post-contrast enhancement.

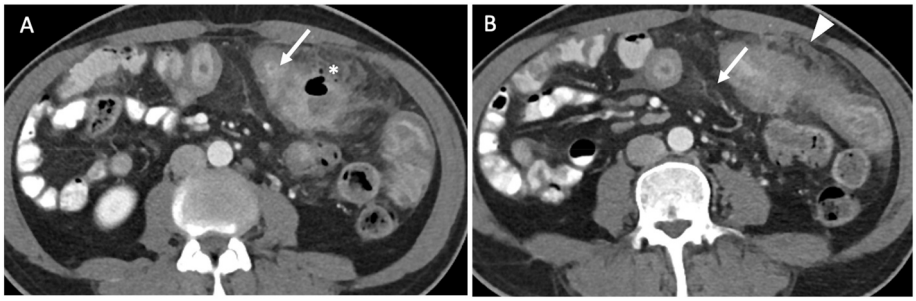

Fig. 4. Jejunal and ileal tuberculosis. A) Contrast-enhanced CT demonstrates diffuse jejunal and ileal thickening (arrow) as well as a few tiny extraluminal air bubbles (asterisk) which indicate micro perforation. Omental nodularity (arrowhead) and mesenteric fat stranding (arrow) are also evident in image B.

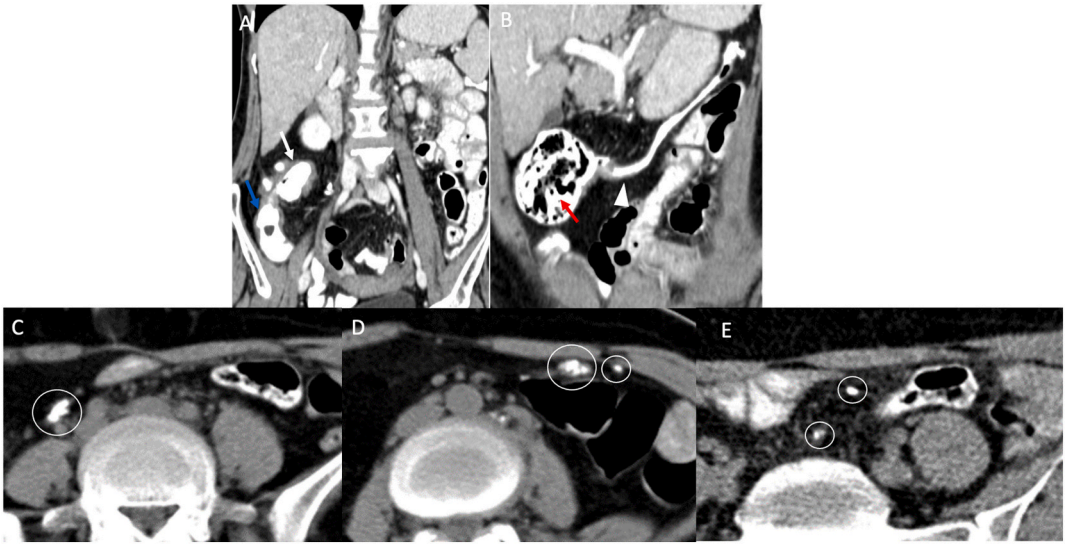

Fig. 5. Ileocecal tuberculosis (late stage). A and B) Contrast-enhanced CT demonstrates a deformed ileocecal junction with a conical cecum (white arrow) and a shrunken right colon with a stricture (arrowhead) at the hepatic flexure and proximal dilation (red arrow). Terminal ileum is marked by the blue arrow. C, D and E) Multiple calcified mesenteric and omental lymph nodes are also seen (circles). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

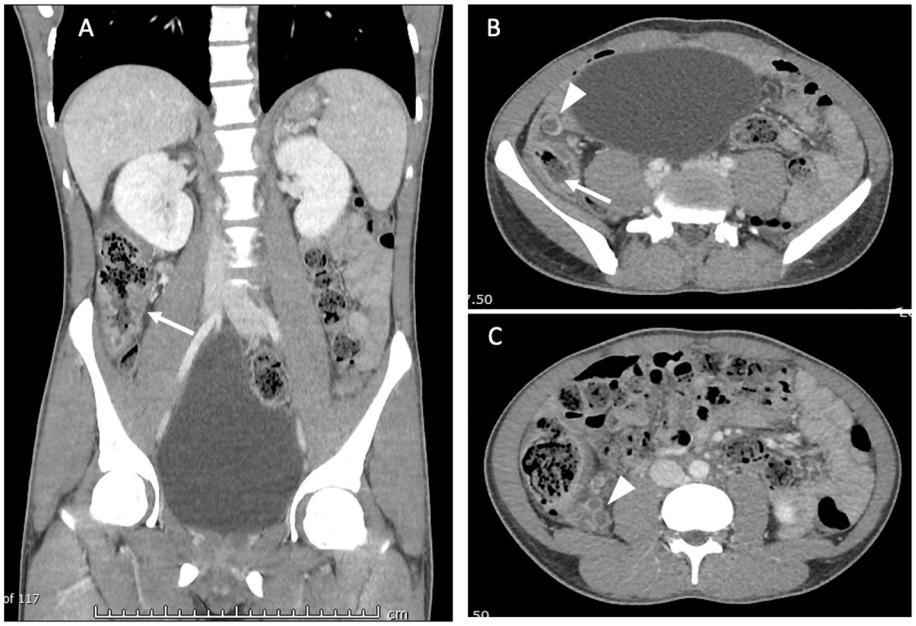

Fig. 6. Cecal and colonic tuberculosis. A) Contrast-enhanced CT demonstrates mural thickening and enhancement of the cecum and ascending colon with surrounding inflammatory changes (arrow). B and C) Multiple regional necrotic mesenteric lymph nodes are also evident (arrowhead).

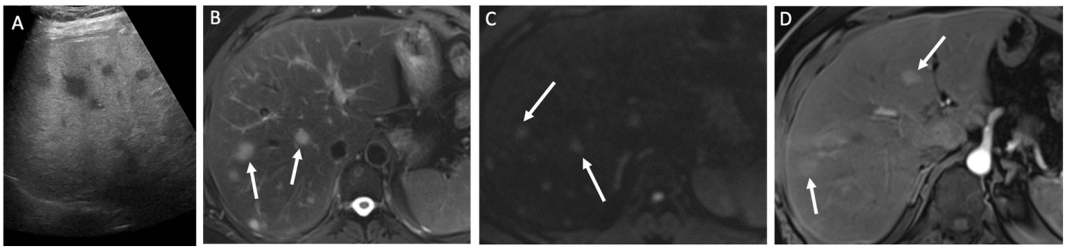

Fig. 7. Hepatic tuberculosis. A) US of liver shows multiple hypoechoic liver lesions. MRI of the same patient reveals B) multiple T2 fat sat hyperintense liver lesions with C) associated diffusion restriction and D) post-contrast enhancement.

scan. Duodenum may also demonstrate intrinsic hypertrophic involvement which can be seen on CT as thickening of the duodenal walls. Ulceration can lead to strictures and fistulae, which are seen clearly on barium studies.[36]

The radiological differential for the appearance of duodenal TB includes superior mesenteric artery syndrome, atypical peptic ulcer disease, Chron's disease and malignancies such as lymphoma and pancreatic carcinoma of the head.[6]

3.4. Jejunum and ileum

The ileocecal region is the most common site of involvement in GI TB seen in 80–90% of cases.[3,8] An abundance of lymphoid tissue and an increased rate of absorption may predispose this site for TB involvement.[37] The involvement of the rest of the small bowel is infrequent and usually occurs together with peritonitis.[24] Clinical symptoms include colicky abdominal pain, weight loss and anemia, and complications due to intestinal obstruction, perforation and hemorrhage may also occur.[19]

Early stages of ileocecal TB are marked by mild mural thickening of the ileum and cecum. Late stages can show eccentric mural thickening involving the medial cecal wall and the valve, with regional lymphadenopathy and inflammatory changes.[37] These findings are easily demonstrated on CT imaging (Fig. 4). The earliest sign on barium studies is hypermobility and spasm of the ileocecal valve with associated edema. Barium studies can also show a narrowed terminal ileum with a gaping, incompetent ileocecal valve (Fleischner sign) or rapid emptying of the diseased segment through the incompetent valve from a narrowed terminal ileum to a rigid and shortened cecum (Stierlin sign).[19] More advanced stages can show a conical cecum, with a shrunken and retracted cecum out of the right iliac fossa due to fibrosis of the mesocolon (Fig. 5).[37]

The radiological differential for the appearance of jejunal and ileal TB includes infectious causes such as amebiasis and infection caused by *Salmonella* and *Yersinia*; inflammatory causes such as Crohn's disease and ulcerative colitis causing backwash ileitis, and malignancy such as primary cecal malignancy.[19,38,39]

3.5. Appendix

Isolated involvement of TB in the appendix is rare and accounts for only 0.08% of all appendectomy specimens.[40] Clinical presentation can be similar to that of acute appendicitis. However, contiguous involvement of the ileocecal region on imaging would be more suggestive of TB involvement.[37] An appendiceal mass mimicking malignancy may also be seen.[37]

3.6. Colon

Colorectal TB is seen in about 10% of gastrointestinal TB cases without small intestine involvement.[11,41] The cecum is the most commonly involved site in colorectal TB, with concomitant involvement of the ileocecal valve and terminal ileum.[6] Aside from the cecum, the transverse colon, rectum, and ascending colon have been reported to be the commonest sites of involvement in colorectal TB.[41] Clinical features include abdominal pain, weight loss and change in bowel habits. Bowel wall perforations and fistulas are seen in up to 18.9% of cases. Localized abscess formation may also be seen with GI tract involvement.[37]

Colonoscopy in the setting of colorectal TB shows linear or fissured ulcers (which may be transverse or circumferential) covered with exudates. These ulcers are surrounded by abnormal mucosa (erythema, edema, and/or nodularity), which is in contrast to normal-appearing mucosa surrounding the ulcers in Crohn's disease. The most common findings on CT scans in colonic TB in decreasing order of frequency are strictures, features of colitis, and polypoid lesions[37] in addition to those seen with jejunal and ileal TB (Fig. 6).

The radiologic differential for the appearance of colorectal TB includes amebic colitis, Crohn's disease, pseudomembranous and ischemic colitis, and malignancy.[19]

4. Hepatosplenic tuberculosis

Hepatosplenic involvement is a rare manifestation of extrapulmonary TB and occurs more frequently as a result of hematogenous

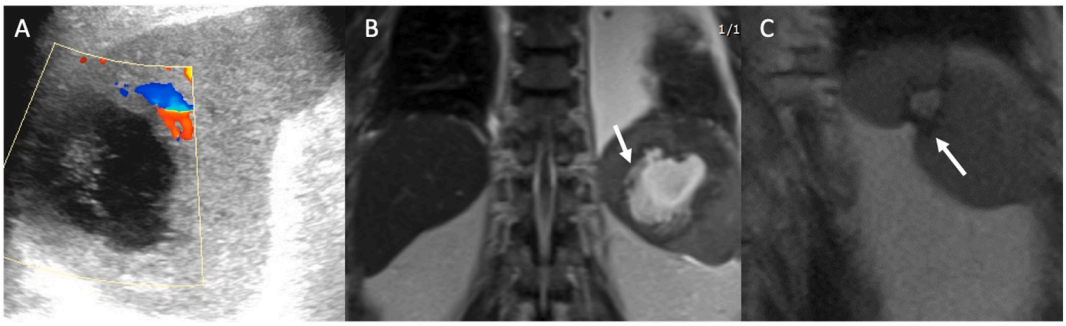

Fig. 8. Splenic tuberculosis. A) US of spleen demonstrates a heterogenous hypoechoic structure (4.9 cm × 4.3 cm) without internal vascularity, suggestive of an abscess. B) T2-weighted image shows a hyperintense lobulated mass (arrow), suggestive of an abscess. Left pleural effusion is also identified. C) MRI 7 months post anti-TB treatment reveals near complete resolution of the splenic lesion with a small residual and scarring (arrow).

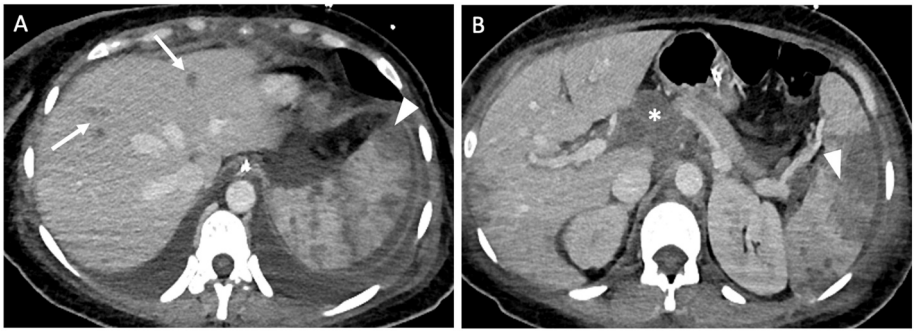

Fig. 9. Hepatosplenic tuberculosis in a young female with newly diagnosed HIV. A and B) Contrast-enhanced CT demonstrates hypo-enhancing nodules of variable size in the liver (arrows) and spleen (arrowheads). Periportal necrotic lymph nodes are also evident (asterisk). Bilateral pleural effusion and small volume ascites are present.

dissemination from a primary site of infection or by local spread from the GI tract.[19,42] The two main forms of hepatosplenic TB are (i) the more common micronodular-miliary type and (ii) the rarer macronodular type.[8]

4.1. Liver

Hepatic tuberculosis has been estimated to occur in 1% of active TB cases, with 79% due to micronodular-miliary TB and 21% due to macronodular-local hepatic TB.[42] The tuberculous bacilli reach the liver via the hepatic artery in miliary TB, and via the portal vein in macronodular TB.[43] It is described to occur more frequently in males with no specified age range.[44] Hepatic tuberculosis commonly presents with non-specific symptoms such as hepatomegaly, fever, respiratory symptoms, abdominal pain, weight loss, and ascites leading to delayed diagnosis.[42,45,46] Jaundice may also be evident, indicating the extension of infection into the biliary system leading to the even more rare gallbladder tuberculosis. The gallbladder wall and mucosa are resistant to *M. tuberculosis*, and thus infection denotes a severe diffuse abdominal TB.[47,48] Gallbladder involvement is extremely rare and is only seen in extensive and severe abdominal TB. Findings can include a large gallbladder with a thickened wall with a possible intraluminal soft tissue mass.[48]

4.2. Spleen

The spleen is an unusual organ to be affected by TB with several isolated literature cases being reported and is the cause of 4% of splenectomies in patients.[49] However, it is the third most common organ to be affected in immunocompromised people with TB infection.[50] It is described to occur more frequently in males 19–53 years old.[51] Splenic tuberculosis commonly presents with fever, hepatosplenomegaly, pallor, GI bleed, and weight loss.[51,52]

A micronodular or macronodular pattern can be seen in both hepatic

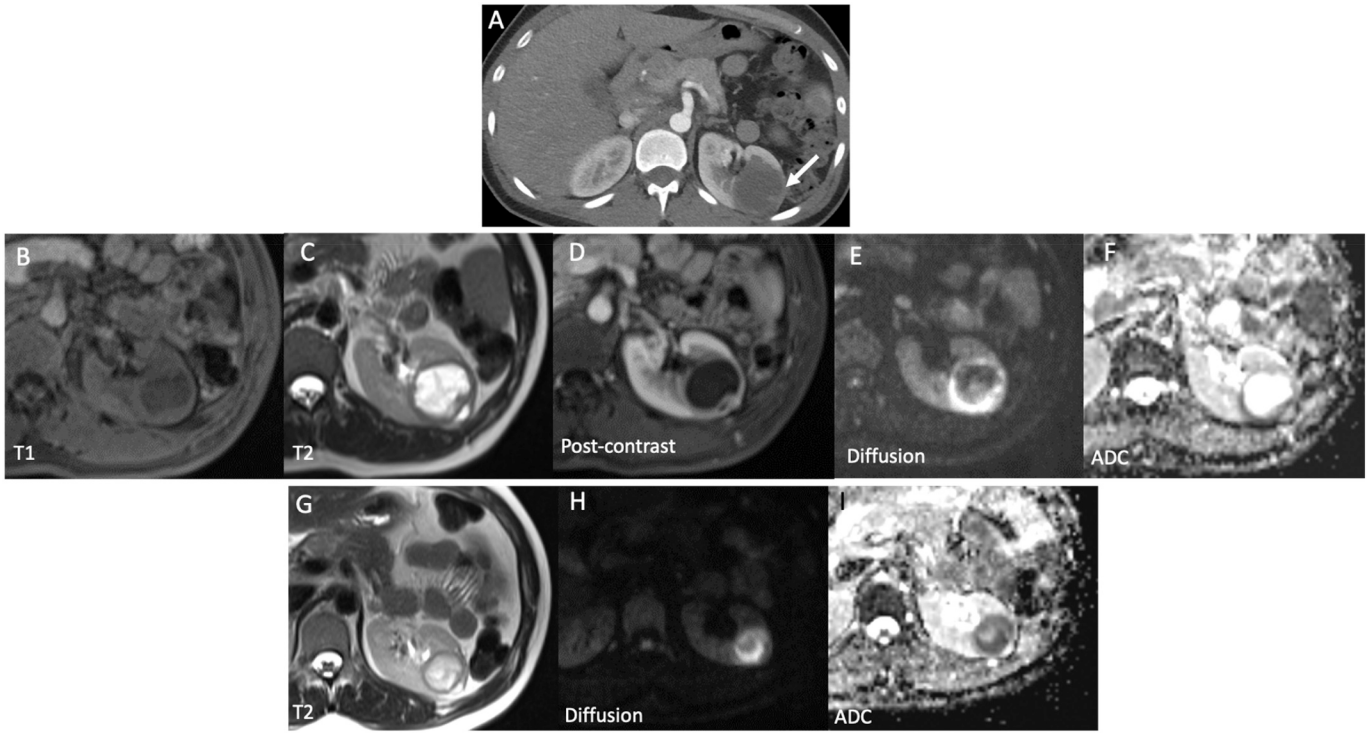

Fig. 10. Renal tuberculosis. A) Contrast-enhanced CT shows a hypo-enhancing cystic lesion in the left kidney (arrow). MRI demonstrates a well-defined, septate cystic lesion which is B) T1 hypointense and C) T2 hyperintense in the interpolar region. The lesion also has a thick T2 hypointense wall which shows D) post-contrast enhancement and E and F) diffusion restriction. G, H and I) MRI 3-months post anti-TB treatment shows a reduction in size of the lesion along with presence of caseating material within the lesion as apparent by increased diffusion restriction within the fluid content.

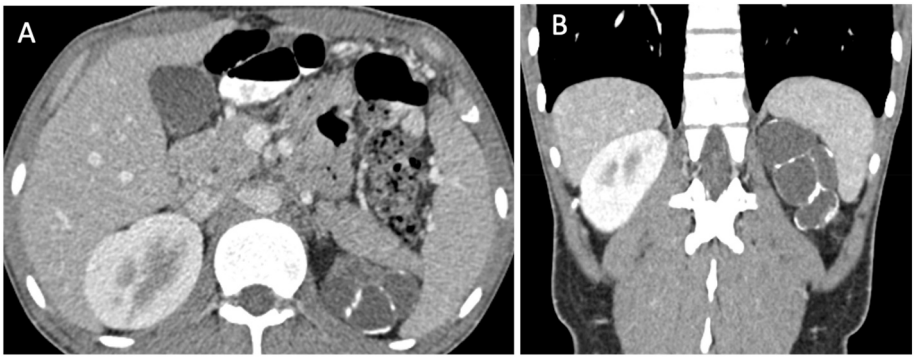

Fig. 11. Pelvicalyceal tuberculosis in a patient with known miliary tuberculosis. Contrast-enhanced CT demonstrates diffuse caliectasis with extensive thinning and calcification of the left renal parenchyma.

and splenic tuberculosis. Ultrasound shows a diffusely hyperechoic liver or spleen.[24] Micronodular or miliary TB appears as numerous tiny hypodense lesions on CT and is most commonly seen in patients with hematogenous spread of primary miliary pulmonary disease. These lesions may demonstrate minimal peripheral enhancement with IV contrast administration.[53] These tiny lesions are often too small to be seen on CT, in which case the only apparent finding may be hepatosplenomegaly.[24] In more advanced stages, the lesions undergo calcification which makes them easier to identify. The tiny nodules are sometimes better characterized by high-resolution ultrasound as hypoechoic lesions.[37]

Macronodular TB is less common and occurs secondary to the spread of tuberculous bacteria through the portal vein.[37] These are seen on CT as single or multiple low attenuating lesions in a background of hepatosplenomegaly (Figs. 7, 8 and 9). Post-contrast images can show early central enhancement, and calcifications can be seen in more chronic cases.[19] The lesions also exhibit peripheral rim enhancement due to the presence of granulation tissue, thereby making the diagnosis and differentiation of TB from primary tumors and metastasis challenging. On MR imaging, the lesions are hypointense on T1-weighted imaging and isointense to hyperintense on T2-weighted imaging with a possible hypointense rim. Post-contrast studies usually show peripheral enhancement.[53]

The radiological differential for the appearance of hepatosplenic tuberculosis of the micronodular-miliary type includes lymphoma, metastasis, sarcoidosis and fungal infections (*Candida*, *Aspergillus*, *Cryptococcus*, histoplasmosis), while the differential for macronodular type tuberculosis includes pyogenic abscesses, metastasis, and primary malignancies. The additional differential for gallbladder involvement includes gallbladder carcinoma and adenomyomatosis.[19,48,54]

5. Genitourinary tuberculosis

The genitourinary (GU) tract is the second most common site for extrapulmonary tuberculosis after lymph node involvement, with a vast majority of cases (90%) occurring in developing countries.[7] Genitourinary TB occurs more commonly in older adults and predominantly affects males.[7,12] Genitourinary TB occurs more frequently in HIV-infected individuals compared to those without.[55,56]

The onset and progression of genitourinary TB is usually insidious, with an approximate 20-year latency period between infection and expression of genitourinary TB.[57] Patients with GU tuberculosis start with a pulmonary focus which through hematogenous seeding leads to infection of the kidneys, prostate and epididymis; bacilluria spreading the infection to the ureter, bladder, and prostate, with subsequent spread to the ejaculatory ducts, seminal vesicles, vas deferens and epididymis.[58]

5.1. Kidney

The kidneys are the most common site of GU tuberculous infection. Nearly 75% of renal TB cases have unilateral involvement.[59] Presentation of renal TB is often non-specific – pyuria and microscopic hematuria may be found incidentally. In the early stages, edema and vasoconstriction in the renal parenchyma can lead to focal hypoperfusion on contrast-enhanced studies, giving an appearance similar to pyelonephritis.[60] Low attenuation parenchymal tumor-like lesions without urinary tract involvement can also occur rarely.[19]

The collecting system is most commonly involved in renal tuberculosis. Initially, only a few calyces are involved, with calyceal deformity or papillary necrosis. Eventual healing and fibrosis lead to multiple strictures resulting in uneven caliectasis, the most common feature seen on cross-sectional imaging (Figs. 10 and 11). The caliectasis is not always seen on excretory urography due to poor opacification due to infundibular stenosis, giving the appearance of the "phantom calyx".[19,60] Urography can also show a "moth-eaten" calyx due to erosions leading to irregular collections of pools of contrast.[19] In healed or chronic renal TB, atrophy, progressive hydronephrosis, and calcifications can occur. This leads to changes in morphology, with the kidneys appearing to have multiple cysts. Eventually, a "putty kidney" appearance occurs when dystrophic calcifications develop in the entire kidney.[60]

The radiological differential for the appearance of renal tuberculosis includes papillary necrosis, medullary sponge kidney, transitional cell, and squamous cell carcinoma, chronic pyelonephritis, and xanthogranulomatous pyelonephritis.[19,61]

5.2. Ureter

Infection from the kidneys can spread down to involve the ureter. Ureteric tuberculosis is present in 50% of patients with genitourinary TB.[8] The distal third of the ureter is the most common site of involvement in ureteric TB.[3,59] In the early stages, intravenous urography may demonstrate dilatation and irregular appearance. CT imaging can show thickening of the ureteric wall and periureteric inflammatory changes. As the disease progresses, ulceration and chronic fibrotic changes lead to a corkscrew appearance of the ureters and proximal hydroureteronephrosis. Pipe-stem appearance of the ureter due to chronic thickening of the ureteric wall resulting and foreshortening can also occur.[19] The most common locations for strictures are at the normal

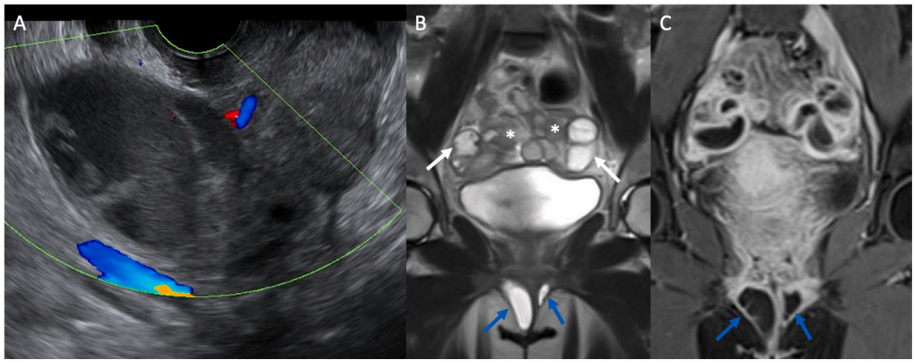

Fig. 12. Tubo-ovarian tuberculosis in a patient with a history of pulmonary tuberculosis. A) US of right adnexa demonstrates a heterogenous hypoechoic lesion. Right ovary is not seen separately. B and C) Coronal T2 and post-contrast T1 sequences demonstrate bilateral hydrosalpinx (white arrows) and pyosalpinx (asterisks) with thick enhancing walls and surrounding fat stranding, suggestive of pelvic inflammatory disease. Bilateral vaginal cysts with thick enhancing walls are also noted (blue arrows). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

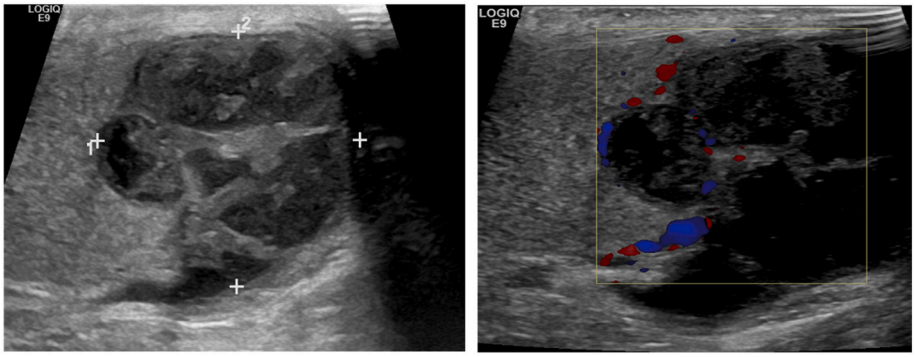

Fig. 13. Tuberculous epididymo-orchitis in a patient with miliary tuberculosis. US of the testes demonstrates heterogenous collection with peripheral vascularity in the lower testicle and epididymal tail region.

anatomic points of narrowing, namely the ureteropelvic junction, pelvic brim, and vesicoureteric junction.[3]

The radiological differential for the appearance of ureteral tuberculosis includes ureteral stones and calcifications caused by schistosomiasis. Ureteral wall calcifications due to tuberculosis are infrequent but its presence in association with renal calcifications is more suggestive of tuberculous infection.[62]

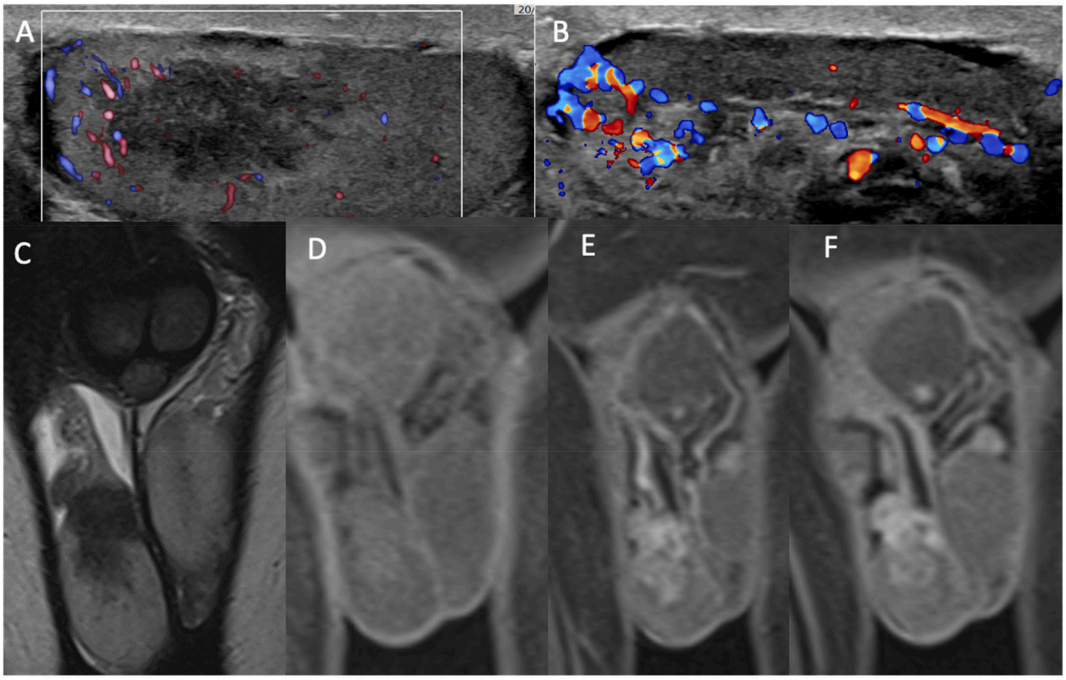

Fig. 14. Tuberculous epididymo-orchitis in a patient with known pulmonary tuberculosis. A and B) US of the right testicle shows an ill-defined hypoechoic lesion and an enlarged epididymis with some peripheral vascularity. MRI demonstrates an ill-defined C) T2 hypointense lesion and D) T1 intermediate signal lesion with E and F) multiloculated peripheral enhancement involving the epididymal head and thick enhancing spermatic cord on the right side.

5.3. Bladder

Infection of the bladder with tuberculosis can occur either by way of hematogenous dissemination or through the passage of infected urine.[12] With the progression of the infection to involve the bladder, urinary urgency, increased frequency, dysuria, hematuria and lumbar pain occur.[57,58]

Imaging reveals a shrunken bladder with wall thickening that mimics transitional cell carcinoma.[19] Filling defects due to bladder wall granulomas can also be seen. Advanced disease with chronic scarring eventually results in a thimble bladder, which is a small, contracted and irregular bladder. Calcifications in the bladder wall are rare.[19] Complications include vesicoureteric reflux resulting from ureteric orifice fibrosis.[60]

The radiological differential for the appearance of tuberculous bladder calcification includes iatrogenic causes such as cystitis post-radiation, chemotherapy (cyclophosphamide) and Bacillus Calmette-Guérin treatment (for bladder cancer); infectious causes such as schistosomiasis; and malignancy such as transitional cell carcinoma.[19,60]

5.4. Genital TB

5.4.1. Ovaries, fallopian tubes, and uterus

Genital TB in women may mimic pelvic inflammatory disease and is accountable for nearly 10% of infertility cases in women globally, with a greater incidence in developing countries.[7] Isolated ovarian tuberculosis involvement is rare, usually affects those in endemic areas, and can mimic ovarian carcinoma.[63,64] Ovarian TB usually develops as a consequence of fallopian tuberculosis, with a resulting development of a tubo-ovarian abscess extending throughout the peritoneum.[65] Imaging has low specificity for differentiating between ovarian tuberculosis and malignancy due to similar appearances on ultrasound, CT, and MRI – heterogeneous masses with the potential to infiltrate the omentum and associated ascites and/or lymphadenopathy.[63]

The fallopian tubes are involved in around 94% of those with genital tuberculosis, most commonly with bilateral involvement.[66–68] If left untreated, the infection may spread to the peritoneum (causing peritonitis) and/or uterine cavity (causing endometritis).[12,66] Clinical symptoms include chronic lower abdominal pain, irregular vaginal bleeding, and infertility. Hysterosalpingogram shows features of tubal occlusion, most prominent in the isthmus and ampulla due to its rich blood supply.[69] Multifocal constrictions and scarring of the fallopian tubes with hydro/pyosalpinx eventually result in a "beaded" or "rigid pipe stem" appearance (Fig. 12). The radiological differential for the appearance of fallopian tube tuberculosis includes salpingitis isthmica nodosa, which shows several diverticula filled with contrast on the hysterosalpingogram, and fallopian tube intraluminal endometriosis.[70]

Infection from fallopian tubes spreads to involve the endometrium in 50–70% of patients, although the myometrium is rarely affected.[60,71] Severe endometrial adhesions in uterine tuberculosis may mimic Asherman syndrome. Vaginal and vulvar tuberculosis is extremely rare.

5.4.2. Testis, epididymis, seminal vesicles and prostate

Tuberculous involvement of male genital organs has been reported in 30%–90% of patients. Physical findings in male genital TB consist of a scrotal lump, epididymal hardening, or fistula.[7] The epididymis is the most commonly affected site (10–55%) in genital TB in males, owing to its rich blood supply.[12,58] The infection is generally unilateral and begins at the tail of the epididymis and thereby spreads to the entire duct.[12] Pain, swelling and an enlarged epididymis and testis may be found on examination. In tuberculous epididymo-orchitis, the US shows swelling and heterogeneous echotexture of the involved segment (Fig. 13) or a heterogenous ill-defined hypoechoic testicular lesion; MRI shows a relatively low signal intensity of T2-weighted images, indicating inflammation or fibrosis (Fig. 14). The differential diagnosis of tuberculous epididymo-orchitis includes bacteria epididymitis (due to *N. gonorrhea*, and *C. trachomatis*).

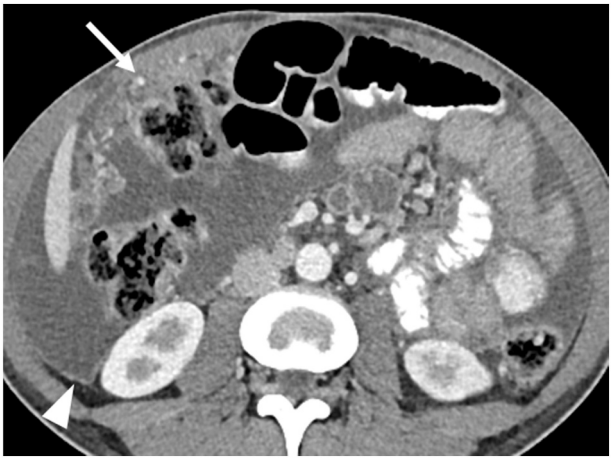

Fig. 15. Tuberculous peritonitis. Contrast-enhanced CT demonstrates ascites with omental thickening and caking (arrow) along with peritoneal thickening and enhancement (arrowhead).

Tuberculosis of the seminal vesicles appears as wall thickening, contraction, and intraluminal or wall calcifications.[60] Tuberculosis of the prostate is rare and may manifest as either prostatitis or an abscess.[66] Nodularity or enlargement may be appreciated on rectal examination. Tuberculosis of the seminal vesicles or prostate may result in necrosis, calcification, caseation, and cavitation.[19] The US of tuberculous prostatitis reveals hypoechogenicity and increased vascularity, similar to that seen in prostate cancer. Prostate tuberculosis appears as a diffuse, radiating area with streaky low signal intensity on T2-weighted MRI as referred to as the "watermelon skin" sign.[65,72,73] It can also appear as an enlarged heterogeneous prostate with small abscesses in advanced stages. Diffuse dystrophic calcifications may be seen with long-standing prostatic tuberculosis. The radiological differential for the appearance of prostatic tuberculosis is prostate carcinoma, which is usually seen as an area of low signal intensity within the normal high signal intensity of the peripheral zone of the prostate, with enhancement on post-contrast images.[74]

Urethral tuberculosis is very rare but may occur with prostate involvement, and results in urethral strictures or fistulas.[75] Urethroperineal fistulas, which result in a "watering can perineum", can also be seen with schistosomiasis infections.[76] Penile tuberculosis is also rare and can manifest as an ulcerative lesion or cause penis deformity.[77,78]

6. Tuberculous peritonitis

Peritoneal spread of tuberculosis the second most common presentation of abdominal TB, affecting between 31 and 58% of cases.[79–81] It mainly occurs secondary to hematogenous spread from a pulmonary focus, but may also occur from lesions in adjacent organs or rupture of an infected lymph node or fallopian tubes.[8,82] It equally affects both sexes and is mostly seen in the age ranges of 35–45 years old.[80]

Three forms of tuberculous peritonitis have been described[3,8,83]: wet, fibrotic, and dry. The three forms have overlapping clinical manifestations of abdominal pain and tenderness, ascites, and fever, except for the dry-plastic type which does not present with abdominal distention. The wet type is the most common, occurring in 90% of cases, and is characterized by a significant amount of free or loculated ascites which is usually hyperdense (20–45 HU) due to high protein content (Fig. 15). The fibrotic fixed type occurs in 60% of cases and is characterized by omental thickening and enhancement, omental "cake-like" mass, fixed and matted bowel loops and mesentery, sometimes with loculated ascites and may progress to an abdominal cocoon or encapsulating

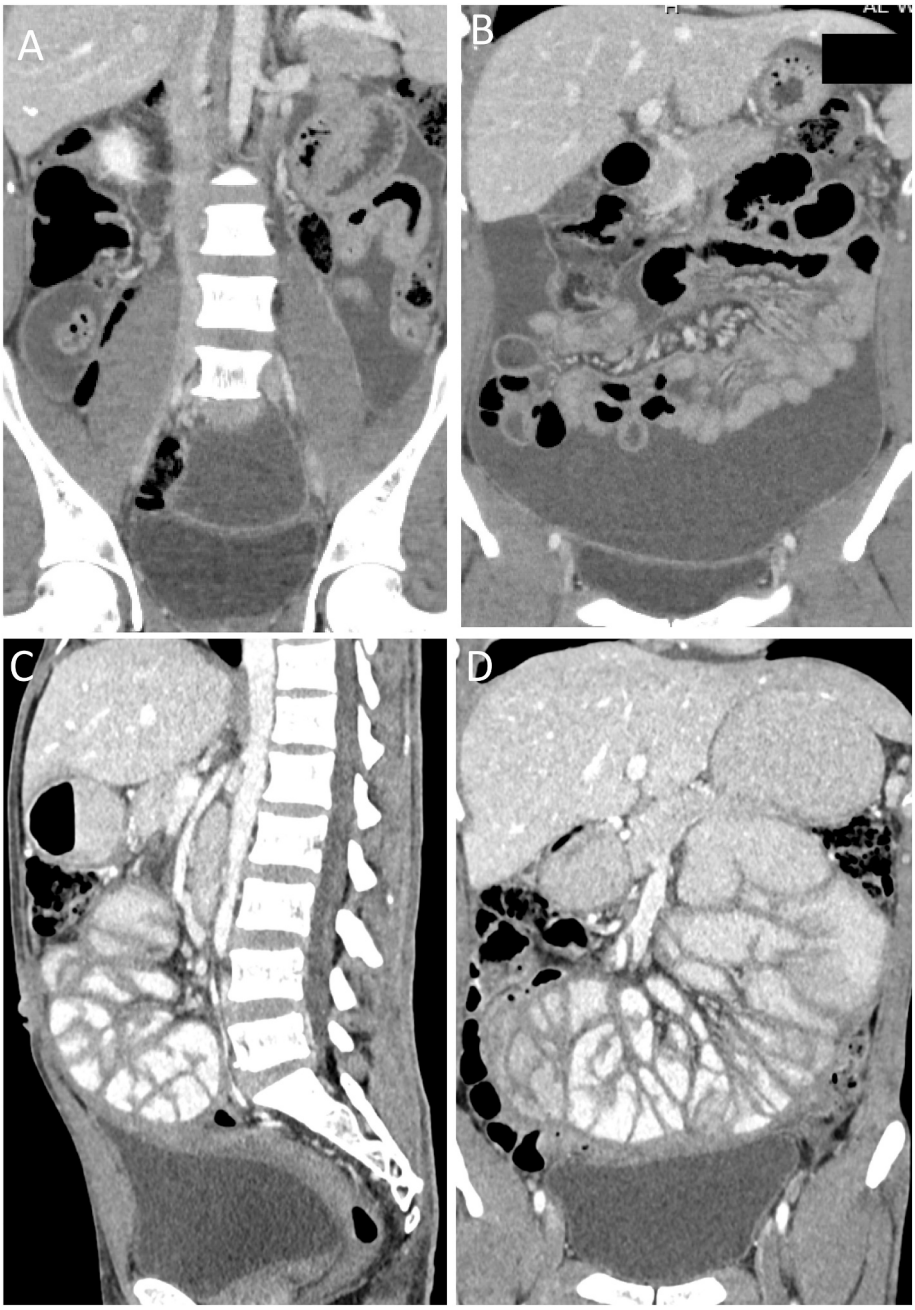

Fig. 16. Tuberculous peritonitis. A and B) Contrast-enhanced CT demonstrates diffuse thickening of proximal bowel with matted loops and loculated ascites. C and D) Contrast-enhanced CT 6 months post anti-TB treatment shows a thick enhancing sac-like structure with matted bowel loops in the center of the abdomen and proximal bowel dilation, indicating encapsulating peritoneal sclerosis (abdominal cocoon).

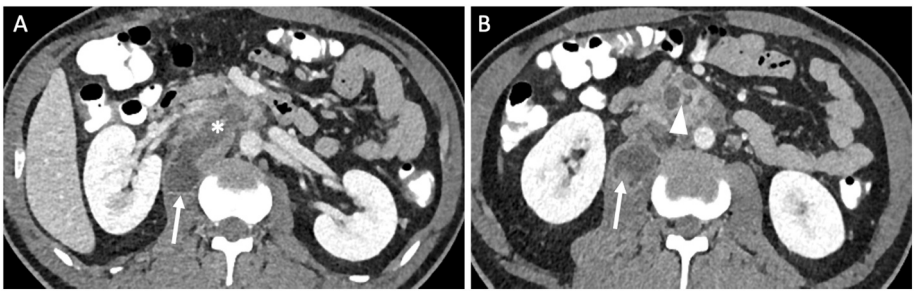

Fig. 17. Tuberculous abscess. Contrast-enhanced CT demonstrates a peripherally enhancing collection in the enlarged right psoas muscle (arrow) which appears connected (asterisk) to adjacent multiple necrotic lymph nodes (arrowhead).

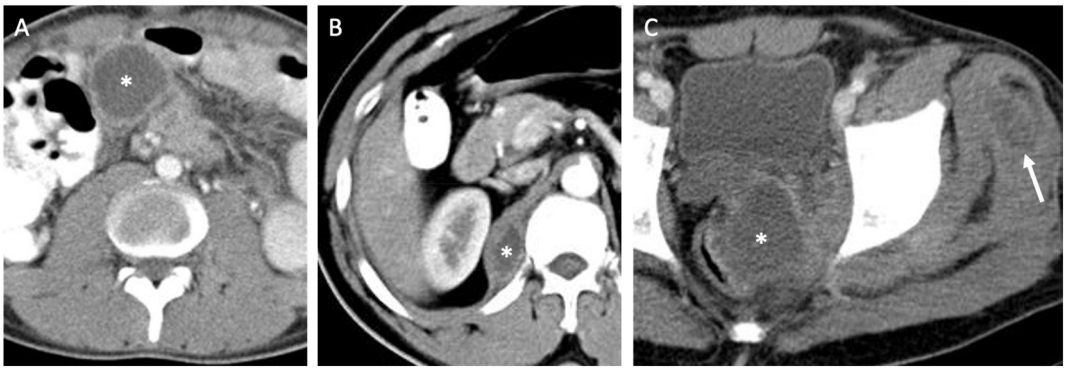

Fig. 18. Tuberculous abscess in multiple different patients. Contrast-enhanced CT demonstrates a peripherally enhancing A) mesenteric, B) left diaphragmatic crus, C) pericolic abscess (asterisk). A small left gluteal abscess is also seen (arrow).

peritoneal sclerosis with intestinal obstruction (Fig. 16). Dry or plastic-type is the least common type, occurring in 10% of cases and is characterized by fibrous adhesions of bowel loops, mesenteric thickening, and caseous mesenteric lymphadenopathy. Radiological features of different types can be seen in the same patient, with varying degrees of mesenteric and omental involvement.[80,84]

The radiological differential for the appearance of tuberculous peritonitis includes non-tuberculous peritonitis, disseminated peritoneal carcinomatosis, ovarian carcinoma, and peritoneal mesothelioma.[80,83,85,86]

7. Tuberculous abscess

A tuberculous abscess is a rare manifestation of tuberculosis, usually presenting as a complication of primary tuberculous infection but can also uncommonly present as an isolated form of primary tuberculosis.[87–89] Abdominal tubercular abscesses can involve the paraspinal or psoas muscles, the anterior abdominal wall, or be intra-peritoneal. Paraspinal or psoas muscle abscesses are often related to TB infection involving the vertebral bodies (Pott's disease) with subsequent extension to the adjacent muscle. The infection is usually restricted to one muscle, and clinical manifestation includes constitutional symptoms in addition to progressive pain and swelling of the affected site, and limitation of hip joint movement if the psoas muscle is affected.[87,89]

An isolated paraspinal cold abscess can also be seen, with more chronic cases showing calcific foci.[19] Intraperitoneal abscesses are often multiloculated, peripherally enhancing and may have foci of calcifications (Figs. 17 and 18). A high index of suspicion for tuberculous abscess should be present when other changes such as peripherally enhancing lymphadenopathy, and peritoneal, mesenteric, and omental changes are seen.[90]

The radiological differential for the appearance of abdominal tuberculous abscess includes other bacterial and fungal infections, tuberculoma, necrotizing fasciitis, hematoma, and soft-tissue tumors.[87,91,92]

8. Management and treatment

The approach to establishing the diagnosis of abdominal TB requires a high index of suspicion as often the clinical and radiologic findings are non-specific. Abdominal TB should be suspected in those patients with clinical manifestations (such as fever, weight loss, diarrhea, abdominal pain, distension and/or mass, hepatomegaly, hematuria) along with relevant epidemiological factors (such as past or present residence in/travel to an endemic country, history of prior TB infection or possible exposure). Laboratory findings may reveal normocytic anemic, thrombocytosis and/or an elevated erythrocyte sedimentation rate and C-reactive protein.[80,93,94]

The definitive diagnosis of abdominal TB may be established through bacteriologic examination of clinical specimens (e.g., ascitic fluid, urine, pus or biopsy specimens) via acid-fact bacilli (AFB) smear, culture, and nucleic acid amplification test (NAAT).[95] AFB smear and culture sensitivities are low (<50%). Nevertheless, culture is particularly useful to conduct drug-susceptibility testing and genotyping. Culture results can take up to 12 weeks (depending on the broth culture system used) compared to the rapid identification in hours through NAAT which has high sensitivity (95% in smear-positive cases) and specificity (98%) for detecting extrapulmonary TB.[80,96,97]

Workup for abdominal TB should also include radiologic imaging; CT would be preferred for evaluation as it allows for the assessment of lymphadenopathy, ascites, and peritoneal and solid organ involvement.[98] Other imaging modalities can also be helpful – ultrasound is useful for identifying lymphadenopathy, ascites and peritoneal and intestinal thickening, and barium enema can demonstrate mucosal ulcerations, strictures and ileocecal valve incompetence.[6] An abdominal X-ray can be useful for identifying air-fluid levels (during the presentation of intestinal obstruction), and calcifications present in the liver and/or ureter.[42]

In the presence of ascites, patients should undergo diagnostic paracentesis with the fluid analyzed for adenosine deaminase (ADA) level and in addition to acid-fast bacillus (AFB) smear, culture, nucleic acid amplification test (NAAT), and routine tests (cell count with differential, albumin and protein levels, Gram stain). Patients with peritoneal TB have a lymphocytic predominance with a low serum-ascites albumin gap (SAAG, <1.1 g/dL).[80] An elevated ascites ADA level (30 to 39 international units/L) in non-cirrhotic patients helps support the diagnosis of peritoneal TB[5,99] but does not establish it with certainty. In the case of non-diagnostic ascitic fluid or absence of ascites, tissue biopsy should be pursued as guided by the extent of anatomic involvement seen on imaging.[5] Histology may demonstrate caseating granulomas with necrosis, which is not pathognomonic by itself but helps support the diagnosis of TB in the setting of other clinical, epidemiologic and imaging findings.[42] The tuberculin skin test (TST) and interferon-gamma release assays (IGRAs) may be positive in patients with abdominal TB, however, they do not differentiate between active and latent TB infection.[94] While not having enough sensitivity (74%), IGRA tests have good specificity (87%) which can help differentiate intestinal TB from Chron's disease.[100] Data on stool polymerase chain reaction (PCR) utility is limited, but small studies have reported good sensitivity (79–100%) and specificity (88–100%) in smear-positive cases, and so this may be used as an adjunct for the diagnosis of intestinal TB.[101,102]

The mainstay of treatment for abdominal TB is with anti-tuberculous therapy. The current INDEX-TB guidelines recommend standard treatment for all forms of abdominal TB which consists of a two-month regimen of four drugs (rifampicin, isoniazid, pyrazinamide and ethambutol) followed by a four-month regimen of two drug (rifampicin and

isoniazid).[103–105] Surgery may be warranted in cases of complications such as abscess and/or fistula formation, bleeding, bowel perforation and/or obstruction and stricture formation, and in the absence of response to anti-tuberculous treatment within two weeks to evaluate for alternative diagnoses such as Chron's disease and lymphoma.[94] Delay in the initiation of anti-tuberculous therapy is associated with worse prognosis and higher mortality,[46,106] which further emphasizes the need for timely evaluation and initiation of therapy.

9. Conclusion

Tuberculosis is a major source of public health concern worldwide affecting virtually every organ system. A high index of clinical suspicion and knowledge about the appearance of TB infection within various organ systems, and its mimics, is central to guiding appropriate timely treatment, especially in high-risk populations.

Declaration of competing interest

H. L., S. A., F. S. A., S. L and S. F.: nothing to disclose.

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References

1. Geneva: World Health Organization. Global tuberculosis report 2019. 2019.
[2] Stojkovic M, et al. Radiological diagnoses in the context of emigration: infectious diseases. Rofo 2018;190(2):121–33.
[3] Burrill J, et al. Tuberculosis: a radiologic review. Radiographics 2007;27(5):1255–73.
[4] Sharma SK, Mohan A. Extrapulmonary tuberculosis. Indian J Med Res 2004;120(4):316–53.
5. Rathi P, Gambhire P. Abdominal Tuberculosis. J Assoc Physicians India 2016;64(2):38–47.
[6] Debi U, et al. Abdominal tuberculosis of the gastrointestinal tract: revisited. World J Gastroenterol 2014;20(40):14831–40.
[7] Figueiredo AA, et al. Epidemiology of urogenital tuberculosis worldwide. Int J Urol 2008;15(9):827–32.
8. Leder RA, Low VH. Tuberculosis of the abdomen. Radiol Clin North Am 1995;33(4):691–705.
[9] Peto HM, et al. Epidemiology of extrapulmonary tuberculosis in the United States, 1993-2006. Clin Infect Dis 2009;49(9):1350–7.
[10] Shafer RW, et al. Extrapulmonary tuberculosis in patients with human immunodeficiency virus infection. Medicine (Baltimore) 1991;70(6):384–97.
[11] Thoeni RF, Margulis AR. Gastrointestinal tuberculosis. Semin Roentgenol 1979;14(4):283–94.
[12] Thwaites G. 40 - tuberculosis. In: Farrar J, et al., editors. Manson's tropical infectious diseases. Twenty-third ed. London: W.B. Saunders; 2014. p. 468–505 [e3].
[13] Mert A, et al. Tuberculous lymphadenopathy in adults: a review of 35 cases. Acta Chir Belg 2002;102(2):118–21.
14. Salvador F, et al. Epidemiology and diagnosis of tuberculous lymphadenitis in a tuberculosis low-burden country. Medicine 2015;94(4):e509.
[15] Fontanilla JM, Barnes A, von Reyn CF. Current diagnosis and management of peripheral tuberculous lymphadenitis. Clin Infect Dis 2011;53(6):555–62.
[16] Mathiasen VD, et al. Epidemiology of tuberculous lymphadenitis in Denmark: a nationwide register-based study. PLoS One 2019;14(8):e0221232.
[17] De Backer AI, et al. Abdominal tuberculous lymphadenopathy: MRI features. Eur Radiol 2005;15(10):2104–9.
[18] Yang ZG, et al. Tuberculosis versus lymphomas in the abdominal lymph nodes: evaluation with contrast-enhanced CT. AJR Am J Roentgenol 1999;172(3):619–23.
[19] Harisinghani MG, et al. Tuberculosis from head to toe. Radiographics 2000;20(2):449–70 [quiz 528-9, 532].
[20] Konishi H, et al. A case of tuberculous mesenteric lymphadenitis and peritonitis with symptoms of acute abdomen. Kekkaku 1995;70(2):117–20.
[21] Caroli-Bosc FX, et al. Abdominal tuberculosis involving hepatic hilar lymph nodes. A cause of portal vein thrombosis and portal hypertension. J Clin Gastroenterol 1997;25(3):541–3.
[22] Puri S, Khurana SB, Malhotra S. Tuberculous abdominal lymphadenopathy causing reversible renovascular hypertension. J Assoc Physicians India 2000;48(5):530–2.
23. Zhang G, et al. Differentiation between tuberculosis and leukemia in abdominal and pelvic lymph nodes: evaluation with contrast-enhanced multidetector computed tomography. Clinics (Sao Paulo, Brazil) 2015;70(3):162–8.
[24] Pereira JM, et al. Abdominal tuberculosis: imaging features. Eur J Radiol 2005;55(2):173–80.
[25] Koh DM, Hughes M, Husband JE. Cross-sectional imaging of nodal metastases in the abdomen and pelvis. Abdom Imaging 2006;31(6):632–43.
[26] Paño B, et al. Pathways of lymphatic spread in gynecologic malignancies. Radiographics 2015;35(3):916–45.
27. Malikowski T, et al. Tuberculosis of the gastrointestinal tract and associated viscera. J Clin Tuberc Other Mycobact Dis 2018;12:1–8.
[28] Ha HK, et al. Intestinal tuberculosis with abdominal complications: radiologic and pathologic features. Abdom Imaging 1999;24(1):32–8.
[29] Nagi B, et al. Imaging of esophageal tuberculosis: a review of 23 cases. Acta Radiol 2003;44(3):329–33.
[30] Sharma V, et al. Primary esophageal tuberculosis mimicking esophageal cancer with vascular involvement. Endosc Ultrasound 2016;5(1):61–2.
[31] Williford ME, et al. Esophageal tuberculosis: findings on barium swallow and computed tomography. Gastrointest Radiol 1983;8(2):119–22.
32. Chaudhary P, et al. Gastric tuberculosis. Indian J Tuberc 2019;66(3):411–7.
[33] Kim S-E, et al. A case of gastric tuberculosis mimicking advanced gastric cancer. Korean J Intern Med 2006;21(1):62–7.
[34] Park JJ, Cheon JH. Differential diagnosis: intestinal tuberculosis. In: Kim WH, Cheon JH, editors. Atlas of inflammatory bowel diseases. Berlin, Heidelberg: Springer Berlin Heidelberg; 2015. p. 71–85.
[35] Baldwin M, et al. Part 1-classic signs in gastrointestinal radiology. Appl Radiol 2011;40(12):22.
[36] Nagi B, et al. Radiological findings in duodenal tuberculosis: a 15-year experience. Abdom Imaging 2015;40(5):1104–9.
[37] Gupta P, et al. Common and uncommon imaging features of abdominal tuberculosis. J Med Imaging Radiat Oncol 2019;63(3):329–39.
[38] Thoeni RF, Cello JP. CT imaging of colitis. Radiology 2006;240(3):623–38.
[39] Elsayes KM, et al. CT enterography: principles, trends, and interpretation of findings. RadioGraphics 2010;30(7):1955–70.
[40] Chong VH, et al. Tuberculous appendix: a review of clinical presentations and outcomes. Singapore Med J 2011;52(2):90–3.
[41] Nagi B, et al. Colorectal tuberculosis. Eur Radiol 2003;13(8):1907–12.
42. Hickey AJ, et al. A systematic review of hepatic tuberculosis with considerations in human immunodeficiency virus co-infection. BMC Infect Dis 2015;15:209.
43. Chien RN, Lin PY, Liaw YF. Hepatic tuberculosis: comparison of miliary and local form. Infection 1995;23(1):5–8.
[44] Chaudhary P. Hepatobiliary tuberculosis. Ann Gastroenterol 2014;27(3):207–11.
[45] Tai W-C, et al. Liver tuberculosis in southern Taiwan: 15-years clinical experience. 內科學誌 2008;19(5):410–7.
46. Wang JY, et al. Disseminated tuberculosis: a 10-year experience in a medical center. Medicine (Baltimore) 2007;86(1):39–46.
47. Xu XF, et al. Gallbladder tuberculosis: CT findings with histopathologic correlation. Korean J Radiol 2011;12(2):196–202.
[48] Vanhoenacker FM, et al. Imaging of gastrointestinal and abdominal tuberculosis. Eur Radiol 2004;14(suppl 3):E103–15.
[49] Simu G, et al. Microscopic patterns in surgically removed spleens. Rom J Morphol Embryol 1991;37(1–2):81.
50. Gupta A. Splenic tuberculosis: a comprehensive review of literature. Pol Przegl Chir 2018;90(5):49–51.
[51] Pottakkat B, et al. Tuberculosis of the spleen as a cause of fever of unknown origin and splenomegaly. Gut Liver 2010;4(1):94.
[52] Ho P-L, Chim C-S, Yuen K-Y. Isolated splenic tuberculosis presenting with pyrexia of unknown origin. Scand J Infect Dis 2000;32(6):700–1.
[53] Kakkar C, et al. Hepatic tuberculosis: a multimodality imaging review. Insights Imaging 2015;6(6):647–58.
[54] Ahmed S, Horton KM, Fishman EK. Splenic incidentalomas. Radiol Clin North Am 2011;49(2):323–47.
55. Nzerue C, et al. Genitourinary tuberculosis in patients with HIV infection: clinical features in an inner-city hospital population. Am J Med Sci 2000;320(5):299–303.
[56] Marques LP, et al. AIDS-associated renal tuberculosis. Nephron 1996;74(4):701–4.
[57] Christensen WI. Genitourinary tuberculosis: review of 102 cases. Medicine (Baltimore) 1974;53(5):377–90.
58. Figueiredo AA, Lucon AM, Srougi M. Urogenital tuberculosis. Microbiol Spectr 2017;5(1).
[59] Zissin R, et al. Computerized tomography findings of abdominal tuberculosis: report of 19 cases. Isr Med Assoc J 2001;3(6):414–8.
[60] Jung YY, Kim JK, Cho KS. Genitourinary tuberculosis: comprehensive cross-sectional imaging. AJR Am J Roentgenol 2005;184(1):143–50.
[61] Gibson MS, Puckett ML, Shelly ME. Renal tuberculosis. Radiographics 2004;24(1):251–6.
62. Prakash J, et al. Extensive renal and ureteral calcification due to tuberculosis: rare images for an uncommon condition. BMJ Case Rep 2013:2013.
[63] Nebhani M, et al. Tuberculose pelvienne simulant une tumeur ovarienne bilatérale: À propos d'un cas. J Gynecol Obstet Biol Reprod 2004;33(2):145–7.
64. Rabesalama S, et al. Isolated ovarian tuberculosis mimicking ovarian carcinoma: case report and literature review. Afr J Infect Dis 2011;5(1):7–10.
[65] Engin G, et al. Imaging of extrapulmonary tuberculosis. Radiographics 2000;20(2):471–88 [quiz 529-30, 532].
[66] Wang L-J, et al. Pictorial essay. CT features of genitourinary tuberculosis. J Comput Assist Tomogr 1997;21(2).
[67] Agarwal J, Gupta JK. Female genital tuberculosis—a retrospective clinico-pathologic study of 501 cases. Indian J Pathol Microbiol 1993;36(4):389–97.

[68] Abbara A, Davidson RN, Medscape. Etiology and management of genitourinary tuberculosis. Nat Rev Urol 2011;8(12):678–88.
[69] Chavhan GB, et al. Female genital tuberculosis: hysterosalpingographic appearances. Br J Radiol 2004;77(914):164–9.
[70] Revzin MV, et al. Imaging evaluation of fallopian tubes and related disease: a primer for radiologists. Radiographics 2020;40(5):1473–501.
[71] Mondal SK, Dutta TK. A ten year clinicopathological study of female genital tuberculosis and impact on fertility. JNMA J Nepal Med Assoc 2009;48(173):52–7.
[72] Cheng Y, et al. Multiparametric magnetic resonance imaging characteristics of prostate tuberculosis. Korean J Radiol 2015;16(4):846–52.
[73] Wang JH, Sheu MH, Lee RC. Tuberculosis of the prostate: MR appearance. J Comput Assist Tomogr 1997;21(4):639–40.
[74] Lovegrove CE, et al. Prostate imaging features that indicate benign or malignant pathology on biopsy. Transl Androl Urol 2018;7(suppl 4):S420–35.
[75] Indudhara R, Vaidyanathan S, Radotra BD. Urethral tuberculosis. Urol Int 1992;48(4):436–8.
[76] Kawashima A, et al. Imaging of urethral disease: a pictorial review. Radiographics 2004;24(suppl 1):S195–216.
77. Singal A, et al. Tuberculosis of the glans penis: an important differential diagnosis of genital ulcer disease. Int J STD AIDS 2017;28(14):1453–5.
[78] Vasanthi R, Ramesh V. Tuberculous infection of the male genitalia. Australas J Dermatol 1991;32(2):81–3.
[79] Hanson RD, Hunter TB. Tuberculous peritonitis: CT appearance. AJR Am J Roentgenol 1985;144(5):931–2.
80. Sanai FM, Bzeizi KI. Systematic review: tuberculous peritonitis–presenting features, diagnostic strategies and treatment. Aliment Pharmacol Ther 2005;22(8):685–700.
[81] Sheldon CD, et al. Incidence of abdominal tuberculosis in Bangladeshi migrants in East London. Tuber Lung Dis 1993;74(1):12–5.
[82] Singh MM, Bhargava AN, Jain KP. Tuberculous peritonitis. An evaluation of pathogenetic mechanisms, diagnostic procedures and therapeutic measures. N Engl J Med 1969;281(20):1091–4.
[83] Ha HK, et al. CT differentiation of tuberculous peritonitis and peritoneal carcinomatosis. AJR Am J Roentgenol 1996;167(3):743–8.
84. Srivastava U, et al. Tuberculous peritonitis. Radiol Case Rep 2014;9(3):971.
[85] Bilgin T, et al. Peritoneal tuberculosis with pelvic abdominal mass, ascites and elevated CA 125 mimicking advanced ovarian carcinoma: a series of 10 cases. Int J Gynecol Cancer 2001;11(4):290–4.
[86] Rodríguez E, Pombo F. Peritoneal tuberculosis versus peritoneal carcinomatosis: distinction based on CT findings. J Comput Assist Tomogr 1996;20(2):269–72.
[87] Alawad AAM, Gismalla MD. Tuberculous abscess of the anterior abdominal wall: an unusual site of presentation. Glob J Med Clin Case Rep 2015;2(3):030 [31: p. 030].
[88] Malhotra MK. Cold abscess of the anterior abdominal wall: an unusual primary presentation. Niger J Surg 2012;18(1):22–3.
[89] Thakur SKS, Sapatnekar V, Pariyani R. Unusual presentation of abdominal Koch - cold abscess. Int J Contemp Med Res 2015;2(1):132–5.
[90] Dong P, et al. Intraperitoneal tuberculous abscess: computed tomography features. World J Radiol 2015;7(9):286–93.
91. Pacheco C, et al. Cutaneous tuberculosis as metastatic tuberculous abscess. J Brasil Pneumol 2015;41(2):200–2.
[92] Sezgin B, et al. Concomitant cutaneous metastatic tuberculous abscesses and multifocal skeletal tuberculosis. Indian J Dermatol 2008;53(3):149–53.
[93] Rai S, Thomas WM. Diagnosis of abdominal tuberculosis: the importance of laparoscopy. J R Soc Med 2003;96(12):586–8.
[94] Kapoor VK. Abdominal tuberculosis. Postgrad Med J 1998;74(874):459–67.
[95] Centers for Disease Control and Prevention. Chapter 4: diagnosis of tuberculosis disease. In: Core curriculum on tuberculosis: what the clinician should know; 2013.
96. Maynard-Smith L, et al. Diagnostic accuracy of the Xpert MTB/RIF assay for extrapulmonary and pulmonary tuberculosis when testing non-respiratory samples: a systematic review. BMC Infect Dis 2014;14:709.
[97] Diaz ML, et al. Polymerase chain reaction for the detection of Mycobacterium tuberculosis DNA in tissue and assessment of its utility in the diagnosis of hepatic granulomas. J Lab Clin Med 1996;127(4):359–63.
[98] Suri S, Gupta S, Suri R. Computed tomography in abdominal tuberculosis. Br J Radiol 1999;72(853):92–8.
99. Lewinsohn DM, et al. Official American Thoracic Society/Infectious Diseases Society of America/Centers for Disease Control and Prevention Clinical Practice Guidelines: Diagnosis of Tuberculosis in Adults and Children. Clin Infect Dis 2017;64(2):e1–33.
[100] Chen W, et al. Effectiveness of interferon-gamma release assays for differentiating intestinal tuberculosis from Crohn's disease: a meta-analysis. World J Gastroenterol 2013;19(44):8133–40.
[101] Kokuto H, et al. Detection of Mycobacterium tuberculosis (MTB) in fecal specimens from adults diagnosed with pulmonary tuberculosis using the Xpert MTB/rifampicin test. Open Forum Infect Dis 2015;2(2):ofv074.
[102] Ramadass B, et al. Fecal polymerase chain reaction for Mycobacterium tuberculosis IS6110 to distinguish Crohn's disease from intestinal tuberculosis. Indian J Gastroenterol 2010;29(4):152–6.
[103] Sharma SK, et al. Index-TB guidelines: guidelines on extrapulmonary tuberculosis for India. Indian J Med Res 2017;145(4):448–63.
104. Jullien S, et al. Six-month therapy for abdominal tuberculosis. Cochrane Database Syst Rev 2016;11(11):Cd012163.
[105] Makharia GK, et al. Intermittent directly observed therapy for abdominal tuberculosis: a multicenter randomized controlled trial comparing 6 months versus 9 months of therapy. Clin Infect Dis 2015;61(5):750–7.
[106] Chow KM, et al. Tuberculous peritonitis-associated mortality is high among patients waiting for the results of mycobacterial cultures of ascitic fluid samples. Clin Infect Dis 2002;35(4):409–13.