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

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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. 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. ¹⁵,¹⁶ 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.
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               | • Nonneiminomatous germ cell cancer |
|                                  | Enlarged hypoechoic nodes (± matted appearance) or hyperechoic nodes with posterior shadowing (due to calcifications) | • Lymphoma |
|                                  | CT               | • Metastatic nodes     |
| Gastrointestinal tuberculosis    | Esophagus US     | • Fungal infections    |
|                                  | Asymmetric wall thickening with loss of wall stratification, mucosal ulceration and adjacent LAD | • Celiac disease |
| Stomach & duodenum CT           | Irregular diffuse wall thickening, fistulous tracts, diverticula, or strictures with LAD | • Esophageal cancer |
| Small intestine & colon CT      | Diffuse wall thickening of affected segment (predominantly in the terminal ileum and cecum) with surrounding fat stranding and LAD | • Gastric scirrhous adenocarcinoma |
| Hepatosplenic tuberculosis      | US               | • Pancreatic cancer of the head |
|                                  | Multiple variable size hypoechoic lesions | • Metastatic disease |
|                                  | Late stage: calcifications | • Chron's disease |
|                                  | CT               | • Infectious causes (syphilis) |
|                                  | Multiple variable size hypodense lesions with central or peripheral post-contrast enhancement, LAD, ascites and peritonitis | • Primary cecal malignancy |
|                                  | Late stage: Calcifications | • Chron's disease |
|                                  | MRI              | • Backwash ileitis (from ulcerative colitis) |
|                                  | 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 | • Infectious causes (Yersinia and amebiasis) |
| Genitourinary tuberculosis      | Kidney CT        | • Primary malignancy |
|                                  | Early: Focal hypo-perfusion on contrast-imaging | • Pyogenic abscess |
|                                  | Late: Multiple strictures resulting in uneven calciestasis | • Metastasis |
|                                  | Healed/chronic: Renal atrophy, progressive hydronephrosis, and dystrophic calcifications ("putty kidney") | • Fungal infections (Candida, Aspergillus) |
| Ureter                           | CT               | • Transitional cell/squamous cell carcinoma |
|                                  | Early: Irregular thickening of the ureteric wall with luminal narrowing and periureteric inflammatory changes | • Medullary sponge kidney |
|                                  | Late: Strictures, ulceration and fibrosis leading to a corkscrew ureter and proximal hydroureteronephrosis | • Papillary necrosis |
|                                  | Chronic: Pipe-stem ureter and foreshortening of ureter | • Chronic pyelonephritis |
| Bladder                          | CT               | • Ureteral stones       |
|                                  | Early: Shrunken bladder with wall thickening and surrounding fat stranding | • Infectious causes (schistosomiasis) |
|                                  | Late: Thimble bladder due to chronic scarring, calcifications (rare) | • Transitional cell carcinoma |
| Genitourinary tuberculosis       | Ovaries US, CT and MRI | • Post-radiation cystitis |
|                                  | Enlarged heterogeneous ovars with associated ascites and regional LAD | • Chemotherapy (cyclophosphamide) |
| Fallopian tubes                  | US, CT and MRI   | • Infectious causes (schistosomiasis) |
|                                  | Multifoical constrictions and scarring resulting in a “beaded” appearance with hydro/pyosalpinx | • Ovarian carcinoma |
| Testis & epididymis US           | Diffuse enlargement with heterogenous echotexture with increased vascularity or heterogenous ill-defined hypoechoic testicular lesion | • Salpingitis isthmica nodosa |
|                                  | MRI              | • Fallopian tube endometriosis |
| Prostate                         | US               | • Bacterial epididymitis |
|                                  | Hypoechoogenicity and increased vascularity | • Prostate carcinoma |

(continued on next page)
involved abdominal group of lymph nodes are omental, mesenteric, and peripancreatic lymph nodes. 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.

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). Lymph node enlargement in tuberculous lymphadenitis often demonstrates self-limited growth, with a range of 12–40 mm. TB lymphadenitis can also be seen on ultrasonography as round or ovoid enlarged lymph nodes with a central hypoechoic area that may be matted.

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. In addition, fungal infections and celiac disease can also have a similar appearance.

### 3. Gastrointestinal tuberculosis

Gastrointestinal (GI) TB is a rare manifestation of abdominal TB and is the sixth most common form of extrapulmonary TB. It can affect any part of the GI tract starting from the esophagus to the rectum. The main forms of GI TB are (i) ulcerative type (60%), which is characterized by single or multiple mucosal ulcers commonly affecting the jejunum and ileum, (ii) ulcer-cystic 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. Complications of the three forms are similar and may include intestinal perforation, bleeding and fistula formation. Bowel obstruction may also occur as a result of mechanical obstruction secondary to stricture formation or intestinal hypertrophy.

#### 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. It occurs due to the spread of infection from either the lungs, spine or mediastinal nodes at the level of the carina. Esophageal involvement often manifests due to extrinsic compression from the lymphadenopathy resulting in either compression or narrowing of the esophagus. In addition to constitutional symptoms, dysphagia and retrosternal pain may be present.

Common radiologic findings include irregular wall thickening, mucosal ulceration, fistula formation, and traction diverticulum and strictures as a result of chronic fibrotic changes. CT imaging is more helpful in delineating tuberculous lymphadenopathy from the displaced esophagus and the extent of fistulous tracts. Endoscopic ultrasound may show asymmetric esophageal wall thickening with loss of wall stratification.

#### 3.2. Stomach

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

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.

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. 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. 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).
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.

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.

3.4. Jejunum and ileum

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

Early stages of ileocecal TB are marked by mild mural thickening involving the medial cecal wall and the valve, with regional lymphadenopathy and inflammatory changes. 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).

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). 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.

3.5. Appendix

Isolated involvement of TB in the appendix is rare and accounts for only 0.08% of all appendectomy specimens. 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. An appendiceal mass mimicking malignancy may also be seen.

3.6. Colon

Colorectal TB is seen in about 10% of gastrointestinal TB cases without small intestine involvement. The cecum is the most commonly involved site in colorectal TB, with concomitant involvement of the ileocecal valve and terminal ileum. Aside from the cecum, the transverse colon, rectum, and ascending colon have been reported to be the commonest sites of involvement in colorectal TB. 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.

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 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.

4. Hepatosplenic tuberculosis

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

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.

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).
dissemination from a primary site of infection or by local spread from the GI tract. The two main forms of hepatosplenic TB are (i) the more common micronodular-miliary type and (ii) the rarer macro-nodular type.

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. The tuberculous bacilli reach the liver via the hepatic artery in miliary TB, and via the portal vein in macro-nodular TB. It is described to occur more frequently in males with no specified age range. Hepatic tuberculosis commonly presents with non-specific symptoms such as hepatomegaly, fever, respiratory symptoms, abdominal pain, weight loss, and ascites leading to delayed diagnosis. 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. Gallbladder involvement is extremely rare and is only seen in extensive and severe abdominal TB. Findings can include a large gall-bladder with a thickened wall with a possible intraluminal soft tissue mass.

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. However, it is the third most common organ to be affected in immunocompromised people with TB infection. It is described to occur more frequently in males 19–53 years old. Splenic tuberculosis commonly presents with fever, hepatosplenomegaly, pallor, GI bleed, and weight loss.

A micronodular or macronodular pattern can be seen in both hepatic and gallbladder tuberculosis.
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.52 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.54

The radiological differential for the appearance of hepatosplenic tuberculosis of the micronodular-miliary type includes lymphoma, metastasis, sarcomiosis 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 extrapoluminary 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.50,56

The onset and progression of genitourinary TB is usually insidious, with an approximate 20-year latency period between infection and expression of genitourinary TB.7,12 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 calycyeal 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 hydrourephrosis, 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.61 The distal third of the ureter is the most common site of involvement in ureteric TB.5,56 In the early stages, intravenous urography may demonstrate dilatation and irregular appearance. CT imaging can show thickening of the ureteric wall and periureteral inflammatory changes. As the disease progresses, ulceration and chronic fibrotic changes lead to a corkscrew appearance of the ureters and proximal hydroureronephrosis. 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. 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.
anatomic points of narrowing, namely the ureteropelvic junction, pelvic brim, and vesicoureteric junction. 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. Fig. 12. Tubo-ovarian tuberculosis in a patient with a history of pulmonary tuberculosis. A) US of right adnexa demonstrates a heterogeneous 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 (asterisk) 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.

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.  

With the progression of the infection to involve the bladder, urinary urgency, increased frequency, dysuria, hematuria and lumbar pain occur.  

Imaging reveals a shrunken bladder with wall thickening that mimics transitional cell carcinoma.  

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.  

Isolated ovarian tuberculosis involvement is rare, usually affects those in endemic areas, and can mimic ovarian carcinoma.  

Ovarian TB usually develops as a consequence of fallopian tuberculosis, with a resulting development of a tubo-ovarian abscess extending throughout the peritoneum.  

The fallopian tubes are involved in around 94% of those with genital tuberculosis, most commonly with bilateral involvement.  

If left untreated, the infection may spread to the peritoneum (causing peritonitis) and/or uterus cavity (causing endometritis).  

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.  

Multifocal constrictions and scarring of the fallopian tubes with hydro/pyosalpinx eventually result in a “beaded” or “rigid pipe stem” appearance.  

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.  

Urethral tuberculosis is very rare but may occur with prostate involvement, and results in urethral strictures or fistulas.  

Urethroperineal fistulas, which result in a “watering can perineum”, can also be seen with schistosomiasis infections.  

Penile tuberculosis is also rare and can manifest as an ulcerative lesion or cause penis deformity.  

6. Tuberculous peritonitis

Peritoneal spread of tuberculosis is the second most common presentation of abdominal TB, affecting between 31 and 58% of cases.  

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.  

It equally affects both sexes and is mostly seen in the age ranges of 35–45 years old.  

Three forms of tuberculous peritonitis have been described: 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 ascites.
**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).
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.\(^{87,89}\) Intraperitoneal abscesses are often multiloculated, peripherally enhancing and may have foci of calcification (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 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-fast 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.\(^{80,86,97}\)

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.\(^{80}\) 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\(^{9,97}\) 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. 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.\(^{100}\) While not having enough sensitivity (74%), IGRA tests have good specificity (87%) which can help differentiate intestinal TB from Crohn’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

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**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).
isoniazid). Delay in the initiation of anti-tuberculous therapy is associated with worse prognosis and higher mortality, 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. I. and S. F.: nothing to disclose.

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