



Abdominal visceral tuberculosis: a malignancy mimic

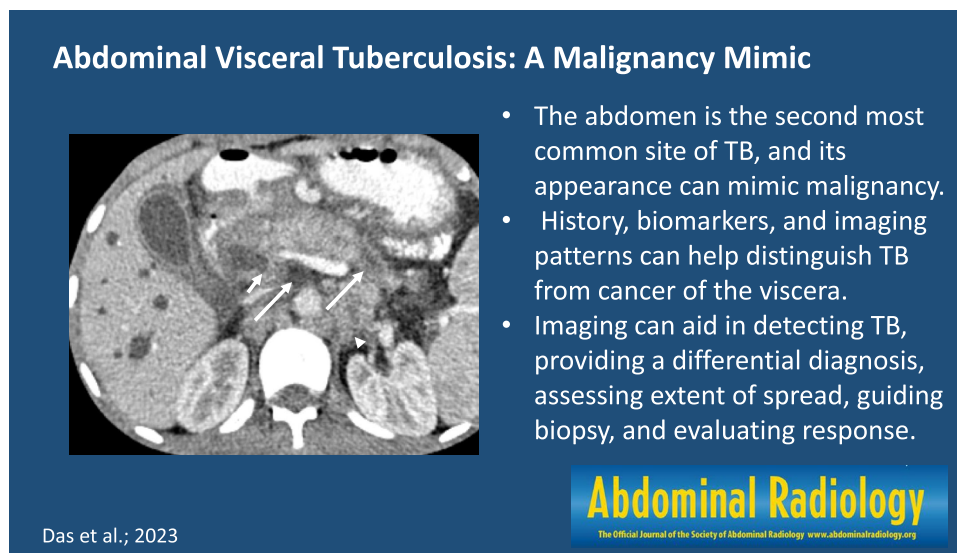
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Abstract

The purpose is to discuss abdominal tuberculosis mimicking malignancy involving the abdominal viscera. TB of the abdominal viscera is common, especially in countries where tuberculosis is endemic and in pockets of non-endemic countries. Diagnosis is challenging as clinical presentations are often non-specific. Tissue sampling may be necessary for definitive diagnosis. Awareness of the early and late disease imaging appearances of abdominal tuberculosis involving the viscera that can mimic malignancy can aid detecting TB, providing a differential diagnosis, assessing extent of spread, guiding biopsy, and evaluating response.

Graphical abstract



Keywords Tuberculosis · Cancer · Ultrasonography · CT · MRI · Abdominal viscera

Introduction

Tuberculosis (TB) remains endemic in many countries and pockets in various non-endemic countries still experience TB or are seeing a resurgence. The latter may be

related to socioeconomic conditions such as congregated living, homelessness, and addiction; immigration from endemic areas; and delayed diagnosis due to the COVID-19 pandemic [1]. It is a global health challenge with increasing prevalence due to secondary immunosuppression and development of multi-drug-resistant strains. It often presents a conundrum to radiologists because it has an extensive spectrum of imaging manifestations and frequently mimics malignancy. TB most commonly infects the lungs and can spread to any organ

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in the body via the bloodstream, lymphatics from infected nodes, direct contiguous spread from infected foci, and ingestion of infected sputum. The abdomen is the most common extrapulmonary site for tuberculosis resulting in infection of the gastrointestinal tract, peritoneum, viscera and nodes by *Mycobacterium tuberculosis* (*M. tuberculosis*) [2, 3]. Isolated visceral tuberculosis is less common, seen in 15%-20% of cases [4]. Route of spread is usually haematogenous and, less often, by direct extension from infected foci. The genitourinary system is often involved. Fifteen percent of liver, spleen, and pancreas TB is found with concomitant lung involvement [5].

Diagnosis of visceral TB poses a challenge because it usually presents without any specific symptoms. Moreover, it is difficult to make a prospective diagnosis because TB can mimic neoplastic conditions, such as lymphoma and malignancy of various abdominal organs. Distinguishing these is clinically significant because the therapy for one may negatively impact or make the other worse. For example, resulting in dissemination of TB or metastases or resulting in inappropriate upstaging of malignancy. Moreover, TB and malignancy may coexist, and patients with cancer have a higher incidence of TB due to immunosuppression [6, 7]. A detailed medical history, potential exposure to TB, classic radiologic features, serum markers, and acid-fast bacilli (AFB) on biopsy specimens with, or sometimes without, successful mycobacterial culture can help differentiate this disease [8]. A high index of suspicion is necessary in the appropriate clinical setting supplemented by cross sectional imaging and tissue sampling for diagnosis of abdominal visceral TB.

Generally, abdominal TB is rare, particularly if isolated, and is more commonly due to hematologic spread in the setting of disseminated disease. It often presents with coexisting lymphadenopathy and its appearance depends on chronicity. Specific organ-based features are described below. Abdominal TB is usually diagnosed by a combination of radiologic and histopathologic studies. Radiology can aid detecting the disease, providing a differential diagnosis, assessing extent of spread, guiding biopsy, and evaluating response to therapy. At biopsy, caseating necrosis in granulomas is the histologic hallmark of TB [9, 10]. Staining for AFB is often negative [10]. Polymerase chain reaction (PCR)-based assays can be more sensitive and specific for diagnosing TB [11, 12]. If there is a high degree of suspicion, patients may be treated empirically with anti-tuberculosis therapy and imaging used to assess response. A paper on TB and malignancies it may mimic in lymph nodes, gastrointestinal tract, and peritoneum has been recently published [7]. In this article, we discuss the various cross-sectional imaging features of visceral abdominal tuberculosis and malignancy mimics.

Hepatic tuberculosis

The prevalence of hepatic TB is variable in the reported literature. An estimate of the incidence is around 1% of all active TB cases [13, 14]. The incidence of extrapulmonary TB, including hepatic TB, is higher in HIV-infected populations. In a study of 164 patients with HIV infected with disseminated TB, hepatic TB was seen in 17.4%. Hepatic TB is typically seen with other features of disseminated abdominal disease. Isolated hepatic TB represents less than 1% of all cases [8]. Clinical features of hepatic TB are nonspecific, resulting in late diagnosis. The most common signs of hepatic TB include hepatomegaly, abdominal pain, fever, and weight loss. Rare manifestations include splenomegaly and jaundice [15–18]. Liver function tests can show elevated transaminases, alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) whereas these tend to be preserved in low burden metastatic disease. An inverted albumin/globulin ratio favors the diagnosis of hepatic TB, particularly in endemic areas. [19].

Morphologically, hepatic TB can manifest as parenchymal involvement with focal or diffuse, micronodular (miliary) and/or macronodular involvement. A very rare variant is the serohepatic type that involves the connective tissue beneath the serosa of liver. It manifests as a thickened liver capsule with subcapsular nodular lesions and is referred to as “sugar-coated” or “frosted” liver [20]. Haematogenous dissemination from lungs is the most common mode of spread to the liver [21] and occurs via the hepatic artery. Local spread from gastrointestinal tract or abdominal lymph nodes is uncommon.

Micronodular hepatic TB refers to diffuse, randomly arranged nodules 0.5–2 mm in diameter [22]. It occurs due to haematogenous spread of the disease, usually in the setting of disseminated disease. On ultrasound, these appear as multiple discrete echogenic lesions. Sometimes, if the nodules are too small to be detected, the liver can show a diffusely echogenic appearance with hepatomegaly. On CT, these nodules typically appear hypodense to background liver parenchyma. Differential diagnosis includes fungal infections, sarcoidosis, and rarely, leukemia and lymphoma and other granulomatous infections such as brucellosis [23, 24]. Clinical, biochemical correlation as well as histopathologic examination may be necessary to reach a diagnosis in certain cases but PCR is more sensitive. Finding AFB or culture of *M. tuberculosis* is the most specific for diagnosing hepatic TB. In cases with high index of suspicion, especially in endemic settings, empirical anti-tubercular therapy (ATT) is usually started [25].

The macronodular, or pseudotumoral, form may present either as a localized focal tuberculoma or multiple

large lesions. This is primarily acquired via the portal vein from the bacilli in the gastrointestinal tract. Compared to micronodular hepatic TB, macronodular TB usually lacks associated pulmonary infection and is characterized by > 2 mm nodules located near the portal triads [26]. The pathological feature of hepatic TB is granulomatous inflammation that occurs as a result of cell-mediated immunological response, consisting of aggregates of macrophages that may coalesce to form Langerhans giant cells associated with surrounding lymphocytes [27]. These undergo progressive central caseous necrosis with peripheral granulation tissue that eventually undergo fibrosis and calcification. Many of these granulomas will coalesce forming a large tuberculoma; liquefaction necrosis within a tuberculoma can result in a tubercular abscess [11]. Imaging appearances vary based on the stage of the disease, from solid homogeneously enhancing nodules in the granulomatous stage, rim-enhancing nodules when they develop caseous necrosis (Fig. 1), to calcified nodules in the chronic stage. On magnetic resonance imaging (MRI), these lesions appear hypointense on T1-weighted

sequences and hyperintense on T2-weighted sequences relative to the background parenchyma. On post gadolinium images, enhancement pattern of the lesions varies from rim-enhancement to heterogeneous patterns of enhancement [28, 29]. Macronodular lesions can show heterogeneous enhancement with central necrosis that can have an appearance like a necrotic hepatic malignancy or metastases (Fig. 2).

Imaging features that favor a diagnosis of TB include the presence of “target sign,” used to describe the rim enhancement of tuberculomas with caseous necrosis, and “cluster sign,” which represents the coalescence of smaller tuberculomas to form an abscess [20, 28]. The target sign is suggestive of, but not specific for, TB as this sign can also be seen in liver metastasis (Fig. 2a) and other liver abscesses. The cluster sign is commonly seen with a liver abscess and rarely with a liver tumor. These signs in disseminated TB can suggest hepatic involvement, otherwise biopsy may be needed.

Hepatic tuberculomas tend to calcify when a normal immune response is mounted, and the presence of calcified liver and/or splenic lesions, particularly in endemic areas,

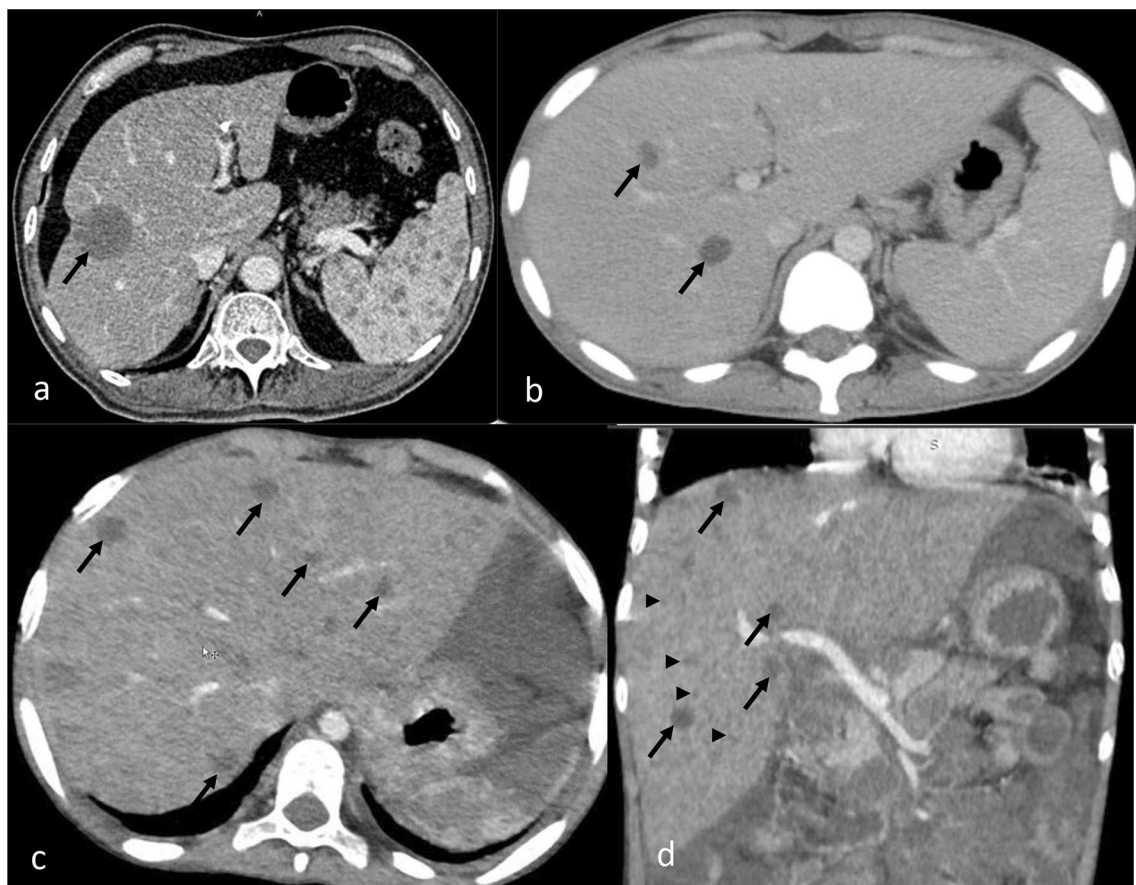


Fig. 1 CECT axial images of abdomen show different pattern of liver involvement in various cases of TB (arrow) as (a) large focal low attenuation lesion, (b) few hypodense nodules and, (c, d) hepatomeg-

aly with multiple hypodense nodules, including 0.5–2 mm nodules that can be seen with the micronodular type (arrowhead)

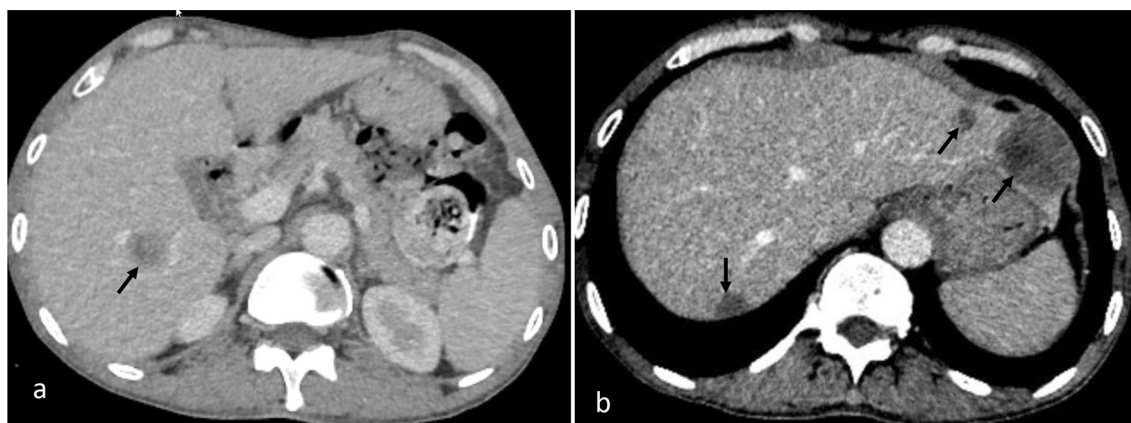


Fig. 2 CECT abdomen axial image (a) shows a solitary hypoenhancing focal lesion (arrow) in liver with “target” appearance of peripheral enhancement in a known case of rectal cancer suggesting liver

metastasis. CECT axial abdomen image (b) shows similar multiple hypoenhancing lesions (arrows) in a patient with colorectal carcinoma, suggesting liver metastases

strongly favors TB. In non-endemic areas, calcified granulomas in the liver and spleen can suggest fungal infection.

Splenic tuberculosis

Like liver, splenic involvement in TB is usually a part of disseminated disease. Isolated splenic TB has been rarely reported [19]. Manifestations include splenomegaly as well as micronodular and/or macronodular nodules [30]. Micronodular lesions may or may not be visualized on ultrasound or CT except as splenomegaly. Macronodular disease can appear as solitary or multifocal hypoechoic nodules on ultrasound, and most commonly hypodense lesions with peripheral enhancement on CT (Fig. 3). Differential considerations include leukemia or lymphoma, metastases (Fig. 4), sarcoidosis, hemangioma, cyst, and fungal infection [31]. Fungal abscesses, most commonly *Candida*, are a greater consideration in immunosuppressed populations. Among malignancies, the most common to the spleen are melanoma, lymphoma, and breast cancer.

Splenic involvement of these is almost invariably commonly part of disseminated stage IV cancer. In this setting, TB is difficult to diagnose and often a high suspicion of TB is needed [32, 33] with confirmatory biopsy.

Gallbladder tuberculosis

Tuberculosis of the gallbladder (GB) is rare and formulating a prospective diagnosis is not easy. Cholelithiasis or obstructed cystic duct are considered risk factors for GB TB. It may present as a nodule, mass or circumferential thickening of the GB, with homogeneous or heterogeneous enhancement. The differential diagnoses include carcinoma, chronic cholecystitis, and xanthogranulomatous cholecystitis. The most important feature that may help to prospectively diagnose TB is the presence of calcific foci within the GB wall lesion, as well as associated necrotic rim-enhancing mesenteric nodes and concomitant liver involvement [34].

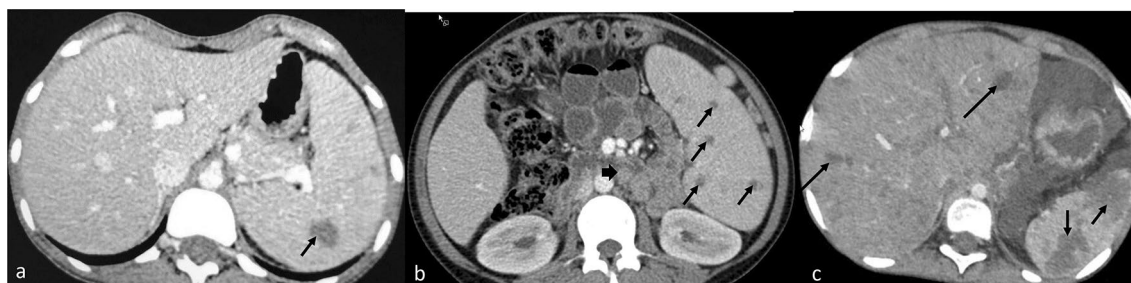


Fig. 3 CECT axial images of abdomen show splenic involvement in various cases of TB as (a) large focal lesion with rim enhancement (arrow), splenomegaly with multiple small focal lesions (arrow) (b),

and large splenic abscesses (c). Note retroperitoneal adenopathy (thick arrow) in (b) and liver lesions (long arrow) in (c)

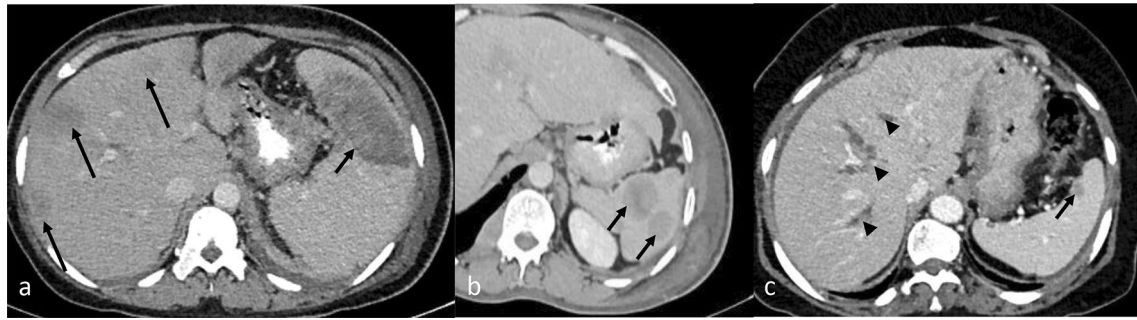


Fig. 4 CECT abdomen images showing various forms of splenic metastases which can mimic TB involvement: (a) large low attenuation splenic metastasis (arrow) and liver metastases (long arrow) from

breast cancer, (b) enhancing lesions in the spleen due to non-Hodgkin's lymphoma, (c) solitary low attenuation splenic metastasis from breast cancer. Intrahepatic ductal dilatation is also seen (arrowheads)

Biliary tuberculosis

Tuberculosis involving the biliary ducts is a rare cause of biliary stricture and is very difficult to diagnose preoperatively in isolation. Biliary TB most commonly occurs due to spread from the portal tracts to biliary radicles; rarely it may spread from periportal tubercular lymph nodes or ascending infection via the Ampulla of Vater [14, 35]. The biliary system can also be involved via direct spread from the hepatic granulomas or by the haematogenous route. Both large and small caliber bile ducts can be affected, however involvement of the extrahepatic bile ducts is rare. This form of TB can present with obstructive jaundice, morphological features similar to cholangiocarcinoma, or inflammatory biliary stricture (Fig. 5). In the presence of other manifestations of abdominal TB, endoscopic or ultrasound-guided fine needle aspiration biopsy or brush cytology can yield the diagnosis pre-operatively, avoiding unnecessary complex surgery. Biliary involvement is often best evaluated by magnetic resonance cholangiopancreatography (MRCP). Wall thickening and dilatation may be seen when large ducts are involved. Multiple strictures at various segments with intervening sections of dilatation can mimic primary sclerosing cholangitis [36]. Ancillary findings of hepatic granulomas, periportal lymph nodes or calcification along the bile ducts favor biliary tuberculosis [24, 37]. Segmental parenchymal atrophy of the involved segments may be seen [38]. Other differential diagnoses include inflammatory/sclerosing cholangiopathies, IgG4-related strictures, AIDS cholangiopathy, recurrent pyogenic cholangitis and malignant strictures secondary to cholangiocarcinoma or carcinoma of the gallbladder.

Pancreatic tuberculosis

Few cases of isolated pancreatic involvement of TB have been reported. It has been postulated that pancreatic enzymes confer a protective effect from tubercular bacilli

[39]. Pancreatic TB is most frequently noted in immunocompromised patients, with the haematogenous route being the most common mode of spread. The pancreatic body is most commonly involved, followed by head and tail [39]. There can be diffuse involvement of the pancreas or there may be multifocal or solitary focal lesions that mimic pancreatic cancer (Fig. 5d and e). The most common morphology is that of a hypodense, peripherally enhancing necrotic mass that can be difficult to distinguish from adenocarcinoma. Biomarkers such as CA-19-9 may aid distinction. Associated prominent peripancreatic necrotic lymphadenopathy is common with TB, in up to 75% of cases [40], but uncommon in pancreatic adenocarcinoma. The main pancreatic duct may be normal or mildly dilated with TB, but is usually dilated with pancreatic carcinoma. Although vascular invasion is not expected in TB, it may cause vasculitis and mimic vascular invasion, as can be seen with adenocarcinoma [41]. The presence of calcifications in both the pancreatic mass and adjacent lymph nodes suggests the diagnosis of TB rather than malignancy. Calcification is rare in pancreatic cancer, although calcification may be seen in the presence of chronic pancreatitis [42].

Kidney ureter bladder (KUB) tuberculosis

The kidneys are affected in 4–17% of patients with TB, with genitourinary TB commonly affecting patients 15–40 years old. Often there is a long latent period between the primary infection and dissemination to the kidneys [43]. Transmission is via the haematogenous route, with bacilli lodging in the glomeruli and peritubular capillaries. Caseous necrosis and subsequent calcifications occur resulting in calcified granulomas. If the disease progresses, the tubercular granulomas coalesce and subsequent capillary rupture results in spread to collecting tubules, renal pelvis, ureter, and urinary bladder. Clinical manifestations are non-specific, often leading to a delay in diagnosis. Increased urinary frequency,

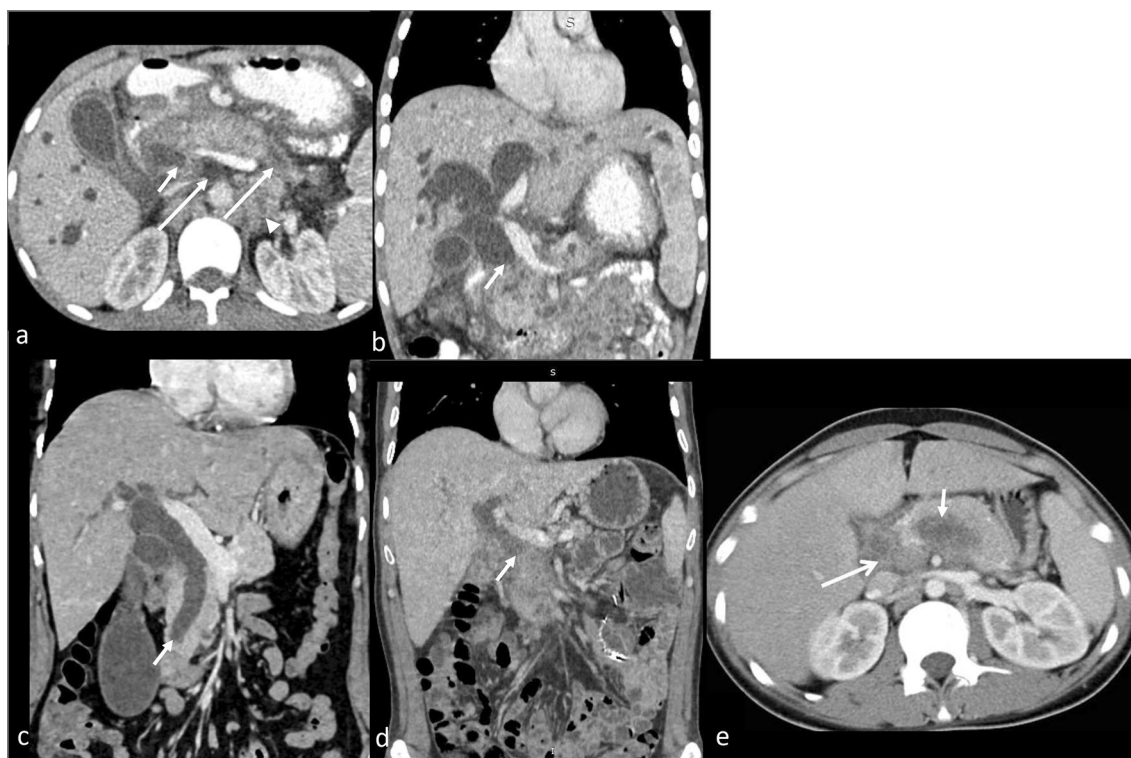


Fig. 5 CECT abdomen images (**a** axial, **b** cor) in a case of TB reveal dilated bilateral intrahepatic biliary radicles and CBD with its abrupt cut off (arrow). Pancreas is bulky with hypodense area in tail and uncinate process (long arrow). Necrotic retroperitoneal nodes (arrowhead) are noted. Similar appearance can be seen with (**c**, cor) distal cholangiocarcinoma (arrow) and (**d**, cor) pancreatic adenocar-

cinoma showing abrupt cut off of the dilated common bile duct. Axial CT (**e**) image showing a hypodense lesion (short arrow) in pancreatic body with necrotic peri pancreatic node (long arrow) highly suspicious for pancreatic malignancy. On biopsy, it was found to be pancreatic tuberculosis

dysuria and flank pain are the most common presenting symptoms and tend to occur once the lower urinary tract is involved. On routine laboratory investigations, sterile pyuria is commonly found. Twenty-four hour urine AFB analysis is positive in ~80–90% of cases. At least 6–8 weeks are required for urine culture to become positive. The false-negative rate is approximately 10–20% [44]. Radiologic manifestations in the kidneys depend on the stage of the disease and may appear as focal pyelonephritis, focal tubercular abscess, or parenchymal scarring.

Intravenous pyelography may detect the early findings of mucosal irregularity and edema. Abdominal radiographs may show calcifications along the genitourinary tract or lymph nodes in late stages. Renal calcifications may be granular or amorphous in cases of focal involvement. Ring-like calcifications may be seen with papillary necrosis. In the end-stage, there may be homogeneous or nodular often dense calcifications of the entire kidney, called putty kidney (Fig. 6). Ultrasound and CT scan may demonstrate parenchymal lesions in the stage of evolving tuberculomas in the parenchyma and CT may show inflammation within the collecting system in the form of

irregular asymmetrical urothelial enhancement. In the end-stage, due to the presence of fibrosis, there may be strictures within the infundibulum, resulting in focal caliectasis or partial opacification of the calyx called a “phantom calyx.” Renal pelvis involvement may appear as a smooth, angulated kink, also referred to as “Kerr’s kink,” with diffuse pelvic narrowing or asymmetrical hydronephrosis. The differential diagnosis of parenchymal renal TB includes other pyogenic or fungal renal infections. Chronic infection can mimic xanthogranulomatous pyelonephritis. Solitary renal tuberculoma, although rare, can be difficult to distinguish from renal cell carcinoma, metastases and lymphoma. Metastasis to the kidneys tends to occur in the setting of disseminated stage IV cancer, and solitary renal lymphoma is rare.

In the early stages, ureteral involvement may manifest as irregular urothelial enhancement with hydronephrosis (Fig. 6). Later, multi-level ureteral strictures can form resulting in a beaded appearance due to alternate segments of dilatation and constriction. In advanced cases, extensive fibrosis can result in short and straightened ureter, referred to as a “pipe stem ureter” and parenchymal calcifications associated

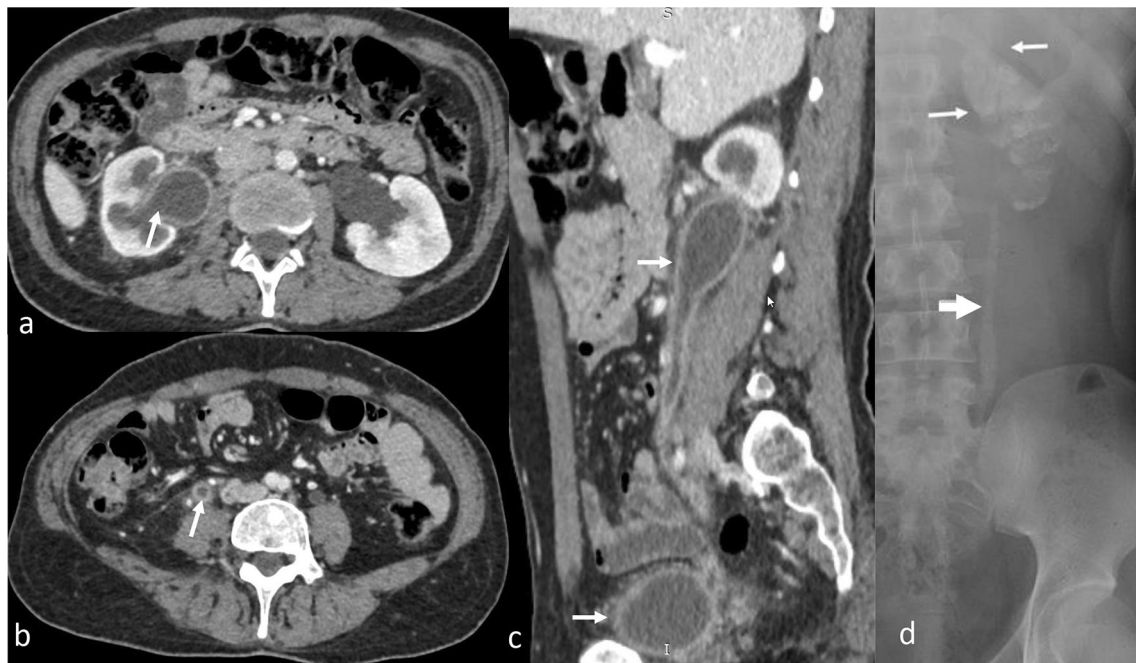


Fig. 6 Genitourinary TB (arrow), CECT axial (a, b), and sagittal (c) images depict dilatation, irregular thickening and enhancement of (a) right pelvicalyceal system, (b, c) ureter, and (c) urinary bladder. These findings can be confused with urothelial carcinoma. Plain

radiograph (d) shows lobar-pattern of calcification of the entire left kidney signifying putty kidney (white arrows) and calcification of the left upper ureter (block arrow) in an advanced case of TB

with putty kidney [45]. Findings may mimic urothelial carcinoma (Fig. 7).

Urinary bladder tuberculosis is nearly always secondary to renal TB and is seen in about 33% of cases [46]. In the early stage, there may be no imaging findings. Later, asymmetrical wall thickening may mimic transitional cell carcinoma on imaging (Fig. 8). Transurethral biopsy may help in distinguishing from malignancy. In the chronic form, a small capacity, thick walled bladder known as a “thimble bladder” may develop and vesicoureteral reflux may be seen. Thimble bladder usually presents with voiding symptoms such as dysuria, frequency and incontinence [47].

Adrenal tuberculosis

Although TB involving the adrenal glands can present as unilateral or bilateral adrenal mass(es), approximately 90% are bilateral, thought to be due to haematogenous mode of spread [48]. The morphology depends on the stage of involvement. In the early stage of caseous granulomatous necrosis, adrenal glands increase in size, with or without preservation of the contour, and may include peripheral rim-enhancement and central hypodensity corresponding to caseous necrosis (Fig. 9). In the late fibrotic stage, calcification is found within the masses, followed by atrophy at the end stages. Differential diagnoses include metastases, which

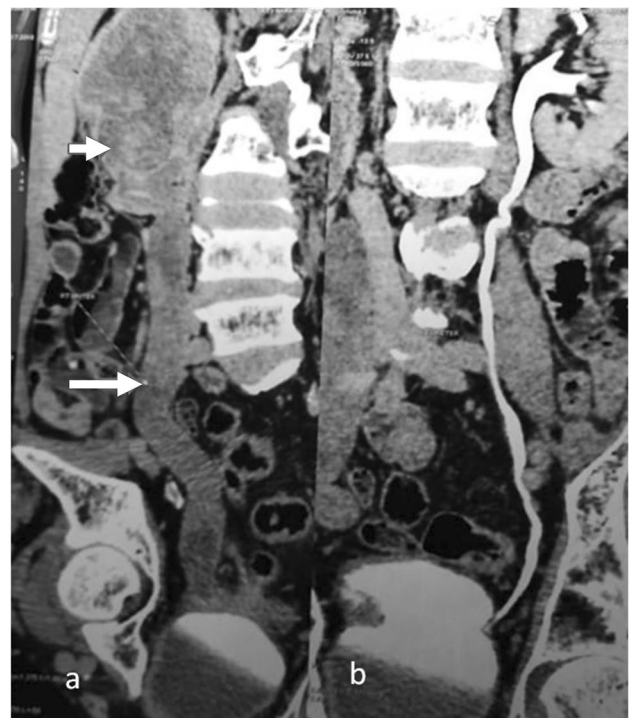


Fig. 7 CECT coronal images of the abdomen in a patient with urothelial carcinoma shows (a) a mass in right pelvis (arrow) and ureter with no contrast excretion (long arrow) whereas (b) normal contrast excretion is seen in the pelvicalyceal system and ureter on the opposite side

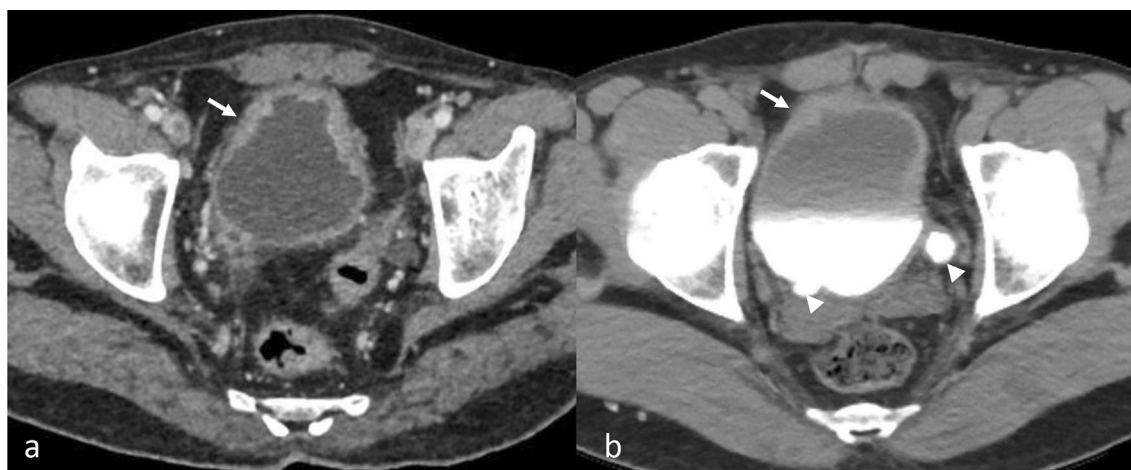


Fig. 8 CECT pelvis axial images (a) show irregular wall thickening of the urinary bladder (arrow) in a patient suspected for urothelial carcinoma who underwent cystoscopy and biopsy. Biopsy showed caseating granuloma suggestive of genitourinary TB. Small blad-

der capacity is due to thimble bladder. CT urography (b) showing irregular wall thickening with proximal dilatation of bilateral ureters (arrowheads) in a case of urothelial carcinoma of the urinary bladder

are more commonly seen in the setting of known malignancy and more often unilateral; lymphoma, which rarely involves the adrenal glands; adrenal haemorrhage, which tends to evolve, may calcify or resolve, and does not enhance with intravenous contrast; as well as fungal infection (most commonly *Histoplasma*), which is rare. Other differential considerations of primary adrenal carcinoma and pheochromocytoma are most commonly unilateral. Features favoring adrenal tuberculosis from an adrenal neoplasm include bilateral involvement, calcification, peripheral enhancement, and central hypodensity [48], although central hypodensity also may be seen with adrenal carcinoma or metastases. Blood biochemical markers (metanephrins) are commonly elevated with pheochromocytoma. The radiological features of adrenal TB and histoplasmosis are fairly similar but, as stated, adrenal fungal involvement is rare in TB. Taking into

consideration the prevalence of TB in the area may further aid in distinguishing between TB and histoplasmosis. Tuberculosis remains the most common cause of Addison's disease or primary adrenal insufficiency in endemic countries [49, 50].

Visceral TB summary

Visceral TB (Table 1) can mimic many diseases and may not be considered in areas where it is thought to be of low prevalence. In areas of high prevalence, it may be mistaken for other diseases such as cancer, thus both a high index of suspicion and knowledge of local demographics are important. Imaging features that favor TB in abdominal viscera are systemic TB, associated caseating lymphadenopathy



Fig. 9 CECT abdomen axial images show (a) enlarged, hypoenhancing and nodular right adrenal gland in a patient with adrenal TB involvement, (b) coronal image shows enlarged right adrenal gland (arrow) with dilated and thickened pelvicalyceal system (thick arrow) of the right kidney consistent with renal and adrenal TB as

well as left hydronephrosis (arrowhead). These appearances can mimic malignancy. CECT coronal image (c) showing bilateral low attenuation adrenal metastasis (arrow) from melanoma with associated liver (long arrow) and splenic (arrowhead) metastases

Table 1 Summary of visceral TB

Type	Clinical features	Imaging findings	Differential diagnoses
Hepatic tuberculosis	Hepatomegaly, abdominal pain, jaundice, fever, and weight loss	Focal/ diffuse, miliary/macronodular Serohepatic variant (rare) Late: calcifications, architectural distortion	Primary malignancy or metastatic disease, fungal infections, sarcoidosis, and rarely, leukemia and lymphoma and other granulomatous infections such as brucellosis
Splenic tuberculosis	Splenomegaly Abdominal pain	Micronodular and/or macronodular nodules, sometimes splenomegaly Late: calcifications, architectural distortion	Metastatic disease, leukemia or lymphoma, sarcoidosis, hemangioma, cyst, and fungal infection
Gallbladder tuberculosis	Right upper quadrant pain, mass, anaemia	Nodule, mass or circumferential thickening of the GB Late: calcifications, architectural distortion	Carcinoma, chronic cholecystitis, and xanthogranulomatous cholecystitis
Biliary tuberculosis	Abdominal pain, hepatomegaly, jaundice, fever, and chills	Multifocal biliary strictures with/without intervening sections of dilatation Extrahepatic biliary duct involvement is rare Late: calcifications	Cholangiocarcinoma, primary sclerosing cholangitis, inflammatory/sclerosing cholangiopathies, IgG4-related strictures, AIDS cholangiopathy, recurrent pyogenic cholangitis
Pancreatic tuberculosis	Epigastric pain, nausea, vomiting, mass, diarrhea, jaundice	Diffuse involvement of the pancreas or multifocal or solitary focal lesions, peripancreatic necrosis Late: calcifications, architectural distortion	Pancreatic carcinoma, pseudocyst, chronic pancreatitis
Kidney ureter bladder (KUB) tuberculosis	Increased urinary frequency, dysuria and flank pain	Parenchymal tuberculoma in kidney, papillary necrosis, renal calcifications, phantom calyx, Kerr's kink, irregular asymmetrical urothelial enhancement Pipe stem ureter Asymmetrical wall thickening of UB, thimble bladder Additional late: calcifications and further architectural distortion	Xanthogranulomatous pyelonephritis, renal cell carcinoma, metastases, lymphoma, urothelial carcinoma
Adrenal TB	Symptoms of adrenal insufficiency- fatigue and abdominal pain	Unilateral or bilateral adrenal masses, early stage-adrenal glands increase in size, with or without preservation of the contour, peripheral rim-enhancement and central hypodensity corresponding to caseous necrosis. In the late fibrotic stage, calcification within the masses, followed by atrophy at the end stages	Metastasis, lymphoma, fungal infection, primary adrenal carcinoma and pheochromocytoma

Caseating or calcified lymphadenopathy may be seen. Visceral TB is often in the setting of systemic disease

(i.e. non-enhancing, low attenuation center) in early TB or calcified lymph nodes in later disease, micronodules or macronodules in early disease, and architectural distortion in later disease. Tumor markers may be elevated in correlated cancers but not with TB. Imaging can guide biopsy. PCR, rather than simply AFB staining, may be needed for confirmation. Imaging may assess response to anti-TB therapy. CT and MRI can be used both for the diagnosis as well as follow up of these patients after antitubercular therapy.

Conclusion

In conclusion, abdominal visceral tuberculosis is common, especially in countries where tuberculosis is endemic. Diagnosis is challenging as clinical presentations are often non-specific and imaging appearances may mimic other diseases, especially cancer. Tissue sampling may be necessary for definitive diagnosis. Radiology can aid detecting TB, providing a differential diagnosis, assessing extent of spread, guiding biopsy, and evaluating response.

Author contributions CJD: contributed to the concept of the manuscript, wrote the manuscript, contributed images and captions, and provided final approval of the manuscript. NR, ZV, AA, SHC: contributed to writing of the manuscript and provided final approval of the manuscript. VK: contributed to the concept of the manuscript, helped write the manuscript, contributed images and captions, oversaw the process, and provided final approval of the manuscript.

Data availability Not applicable.

Declarations

Conflict of interest The authors have no competing interests to declare that are relevant to the content of this article.

Ethics approval Not applicable.

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