



Imaging in Abdominal Tuberculosis

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Abstract

Keywords

- ▶ abdominal tuberculosis
- ▶ peritoneal tuberculosis
- ▶ intestinal tuberculosis
- ▶ hepatosplenic tuberculosis
- ▶ computed tomography
- ▶ ultrasound
- ▶ barium studies
- ▶ enterography

Diagnosis of abdominal tuberculosis is challenging due to its variable imaging appearance, which can mimic several other conditions. This review covers the imaging spectrum of tubercular involvement of abdominal lymph nodes, peritoneum, solid abdominal viscera, and the gastrointestinal tract. Tubercular lymphadenitis is the most frequent manifestation of abdominal tuberculosis and may show necrosis. Peritoneal tuberculosis is the next most commonly involved site in the abdomen and can be of wet, fibrotic, and dry forms. The ileocecal region is the most common site of involvement in gastrointestinal tuberculosis. Solid visceral involvement mostly affects the liver and spleen causing hepatosplenomegaly with a micronodular or macronodular imaging pattern. Awareness of the spectrum of imaging manifestations and a high index of suspicion is required to reach the correct diagnosis.

Introduction

Tuberculosis (TB) is a global health problem and a leading cause of death due to a single infectious agent. As per the World Health Organization's global TB report of 2023, there were 7.5 million newly diagnosed cases and an estimated 1.3 million deaths due to the disease in 2022. India has the highest burden of TB, accounting for 27% of all patients.¹ Although an infection that mostly affects the lungs, extrapulmonary disease accounts for 10 to 12% of all cases, and abdominal involvement occurs in 11 to 16% of cases of extrapulmonary TB.²

Abdominal TB has a varied clinical presentation. Paustian's criteria for the diagnosis of abdominal TB (1964) were based on histological or culture demonstration of *Mycobacterium tuberculosis* bacteria. However, due to the paucibacillary nature of abdominal TB, these criteria are difficult to fulfill. Logan's modification of Paustian's criteria is often followed since it allows the diagnosis of abdominal TB based on a response to a trial of antitubercular treatment (ATT).³ This approach has its drawbacks as well, since many other diseases may be wrongly treated with ATT, and to add to the confusion, few of the nontubercular diseases may show some response as well. Subsequently in a study, it was shown that clinical response

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Table 1 Salient imaging features of abdominal tuberculosis

Lymph nodes	<ul style="list-style-type: none"> ● Necrosis ● Periadenitis ● Conglomeration
Peritoneum	<ul style="list-style-type: none"> ● High-density ascites ● Smooth peritoneal thickening ● Smudged omentum ● Mesenteric thickening and nodules
Visceral organs	<ul style="list-style-type: none"> ● Honeycomb lesions ● T2 hypointense lesions ● Calcifications
Gastrointestinal tuberculosis	<ul style="list-style-type: none"> ● Ulcerations ● Concentric wall thickening ● Strictures ● Hypertrophic masses ● Sinus and fistula

coupled with mucosal response is diagnostic of intestinal TB in this setting and especially to differentiate from Crohn’s disease.⁴ Thereafter, it was observed that patients with intestinal TB often show a response by 8 to 12 weeks; hence, in cases of diagnostic dilemma, an assessment 2 to

3 months after the institution of ATT trial helps in diagnosis.⁵ Even after the advent of newer molecular-based laboratory tests with high diagnostic accuracy such as GeneXpert and polymerase chain reaction, imaging plays a key role in the diagnosis of abdominal TB.

The purpose of this article is to review and illustrate the imaging manifestations of tubercular involvement of various abdominal organs including tubercular lymphadenitis, peritonitis, tubercular involvement of the hepatobiliary system, pancreas, spleen, adrenal glands, and gastrointestinal tract. The salient imaging features of tubercular involvement of different abdominal organs are summarized in ►Table 1, whereas their important differential diagnoses are enlisted in ►Table 2. Although abdominal TB can involve the genitourinary system also, that is beyond the scope of this article.

Tubercular Lymphadenitis

Lymph nodal involvement is present in approximately two-thirds of patients and is the most frequent manifestation of abdominal TB.⁶ Dissemination of tubercular infection to the abdominal nodes is usually by lymphatic route from the bowel. Since TB has a predilection for the terminal ileum,

Table 2 Differential diagnoses of abdominal tuberculosis

	Imaging findings	Differential diagnosis
Tuberculous lymphadenitis	<ul style="list-style-type: none"> ● Necrotic nodes ● Conglomeration ● Periadenitis 	<ul style="list-style-type: none"> ● Nodal metastases ● Aggressive lymphoma ● Whipple’s disease
Tuberculous peritonitis	<ul style="list-style-type: none"> ● High-density ascites ● Smooth peritoneal thickening ● Smudged omentum ● Mesenteric thickening/nodules 	<ul style="list-style-type: none"> ● Peritoneal carcinomatosis ● Mesothelioma ● Bacterial peritonitis
	<ul style="list-style-type: none"> ● Encapsulating peritoneal sclerosis 	<ul style="list-style-type: none"> ● Peritoneal dialysis, surgery, β-blockers ● Internal hernia
Hepatosplenic tuberculosis	Micronodular pattern <ul style="list-style-type: none"> ● Hepatosplenomegaly ● 0.5- to 2-mm nodules 	<ul style="list-style-type: none"> ● Miliary metastases ● Sarcoidosis ● Fungal infections ● Lymphoma
	Macronodular pattern <ul style="list-style-type: none"> ● 1- to 3-cm lesions ● Honeycomb appearance ● T2 hypointensity 	<ul style="list-style-type: none"> ● Abscesses ● Metastases ● Primary malignancy ● Lymphoma
	Serohepatic tuberculosis <ul style="list-style-type: none"> ● Subcapsular hepatic lesions 	<ul style="list-style-type: none"> ● Pseudomyxoma peritonei ● Peritoneal carcinomatosis or lymphomatosis
Pancreatic tuberculosis	Focal form <ul style="list-style-type: none"> ● Solid or complex cystic lesion ● Intralesional calcifications 	<ul style="list-style-type: none"> ● Pancreatic ductal adenocarcinoma ● Pancreatic cystic neoplasms
	Diffuse form <ul style="list-style-type: none"> ● Bulky pancreas with heterogeneous enhancement 	<ul style="list-style-type: none"> ● Acute interstitial edematous pancreatitis ● Autoimmune pancreatitis ● Pancreatic lymphoma
Gallbladder and biliary tuberculosis	Gallbladder <ul style="list-style-type: none"> ● Diffuse, polypoidal, or mass-like thickening 	<ul style="list-style-type: none"> ● Cholecystitis ● Carcinoma gallbladder
	Tubercular cholangitis <ul style="list-style-type: none"> ● Multifocal intrahepatic biliary strictures ● Beaded appearance on MRCP 	<ul style="list-style-type: none"> ● Primary sclerosing cholangitis ● IgG4-related cholangiopathy ● Cholangiocarcinoma

Table 2 (Continued)

	Imaging findings	Differential diagnosis
Adrenal tuberculosis	<ul style="list-style-type: none"> ● Bilateral adrenomegaly ● Peripheral rim enhancement ● Calcifications 	<ul style="list-style-type: none"> ● Metastases ● Lymphoma ● Histoplasmosis
Gastrointestinal tuberculosis	<ul style="list-style-type: none"> ● Concentric bowel wall thickening ● Ulcerations ● Hypertrophic masses ● Strictures ● Sinus and fistula 	<ul style="list-style-type: none"> ● Inflammatory bowel disease ● Hypertrophic form mimics neoplasms
	Ileocecal tuberculosis	<ul style="list-style-type: none"> ● Crohn's disease ● Amoebiasis ● <i>Salmonella</i> enteritis ● <i>Yersinia</i> enterocolitis ● Cecal carcinoma/lymphoma
	Esophageal tuberculosis	<ul style="list-style-type: none"> ● Crohn's disease ● Carcinoma esophagus
	Gastric tuberculosis	<ul style="list-style-type: none"> ● Peptic ulcer disease ● Gastric malignancy
	Duodenal tuberculosis	<ul style="list-style-type: none"> ● Crohn's disease ● Duodenal malignancy ● Superior mesenteric artery syndrome
	Colorectal tuberculosis	<ul style="list-style-type: none"> ● Adenocarcinoma of colon ● Inflammatory bowel disease ● Amebic colitis ● Pseudomembranous colitis ● Ischemic colitis
	Anoperineal tuberculosis	<ul style="list-style-type: none"> ● Cryptoglandular fistula ● Crohn's disease ● Pelvic actinomycosis ● Lymphogranuloma venereum

Abbreviation: MRCP, magnetic resonance cholangiopancreatography.

ileocecal junction, and right colon; the mesenteric, periportal, peripancreatic, and upper paraaortic nodal stations are commonly involved, whereas retroperitoneal lymph nodes below the level of renal hilum are unusual. Other routes of spread of infection to abdominal nodes are via the hematogenous route or by contiguous spread from other affected organs.⁷

Histologically, tubercular nodes show initial lymphoid hyperplasia, followed by caseation necrosis, and eventually healing with fibrosis and calcification.⁸ Corresponding to these pathological stages, the nodes may appear homogeneous in the initial stage. Subsequently, they develop liquefactive necrosis, which appears as marked central hypoechogenicity on ultrasound (►Fig. 1), rim enhancement or heterogeneous enhancement on computed tomography (CT) or magnetic resonance imaging (MRI), and central T2 hyperintensity on MRI. Finally, the lymph nodes regress in size and may calcify.^{7,9} Tubercular lymphadenitis may also appear as an increase in the number of mesenteric nodes (>3 in a CT section), or as conglomerate nodal masses, with perinodal fat stranding and T2 hyperintensity due to peradenitis.^{7,10} The involvement of two or more lymphatic chains with poorly enhancing nodes is another pattern of tubercular lymphadenitis seen in patients with HIV/AIDS, possibly due to a weakened immune response^{11,12} (►Fig. 2).

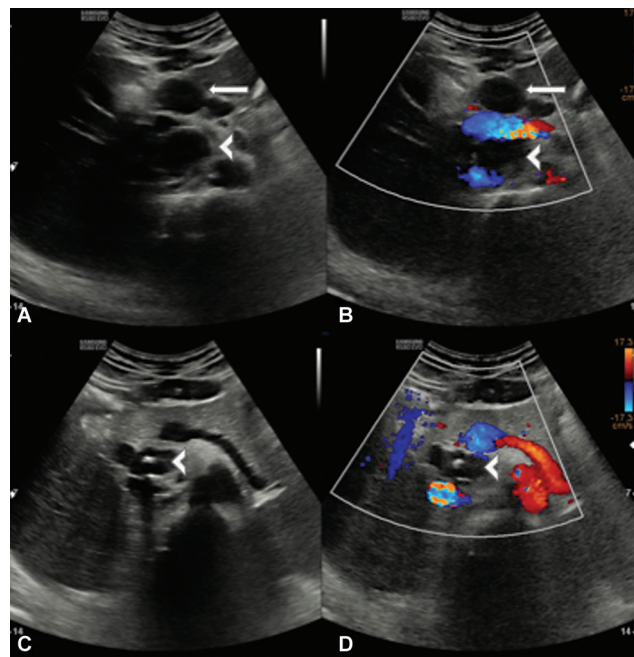


Fig. 1 Tubercular lymphadenitis. Grayscale ultrasound (A, C) and color Doppler images (B, D) show enlarged periportal (arrow) and portocaval nodes (arrowhead) with marked central hypoechogenicity suggestive of necrosis. Focal calcification is also present in the portocaval node (C, D).

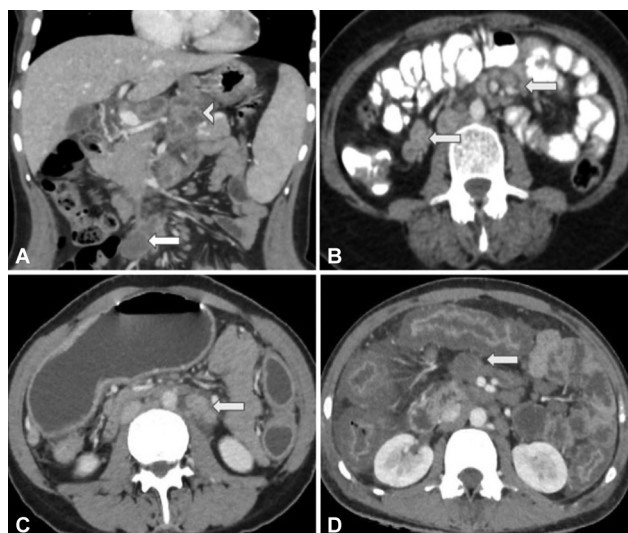


Fig. 2 Tubercular lymphadenitis. (A) Coronal contrast-enhanced computed tomography (CECT) showing rim-enhancing peripancreatic (arrowhead) and conglomerate mesenteric lymphadenopathy (arrow). Axial CECT (B–D) demonstrating heterogeneous mesenteric nodes (B), paraaortic lymphadenopathy with periadenitis (C), and nonenhancing nodes in an HIV+ patient (D).

Tubercular nodes can cause thrombosis of adjacent venous channels, especially the portal vein giving rise to cavernoma formation.⁹

Although quite characteristic, necrotic abdominal lymphadenopathy is not pathognomonic for tubercular lymphadenitis. It can also be seen in certain lymph nodal metastases, aggressive lymphoma, and inflammatory conditions such as Whipple's disease. The clinical context, characteristic distribution of nodes, and any concomitant peritoneal and intestinal changes are useful in differentiating tuberculous lymphadenitis from these conditions.¹³ Ultrasound or CT-guided sampling of the enlarged abdominal lymph nodes can also aid in confirming the diagnosis.

Peritoneal Tuberculosis

Peritoneal involvement is the second most common site in abdominal TB. It can occur by hematogenous spread or from direct spread from other involved abdominal organs such as lymph nodes, bowel, or fallopian tubes.⁹

Classically, tubercular peritonitis has been considered to be of three types: wet, fibrotic, and dry. The wet type, in which ascites is the dominant feature, accounts for most cases of tubercular peritonitis (–Fig. 3). Fibrotic or fixed type is characterized by omental and mesenteric thickening with matted bowel loops and sometimes loculated ascites (–Fig. 4). The dry or plastic type is the least common form and is characterized by caseous nodules and dense adhesions. These three types frequently occur in combination as well.¹⁴

Ascites is the most common manifestation of tubercular peritonitis present in 30 to 100% of cases. It may be free or loculated; septae within the ascitic fluid are best demonstrated on ultrasound. Due to its exudative nature, the

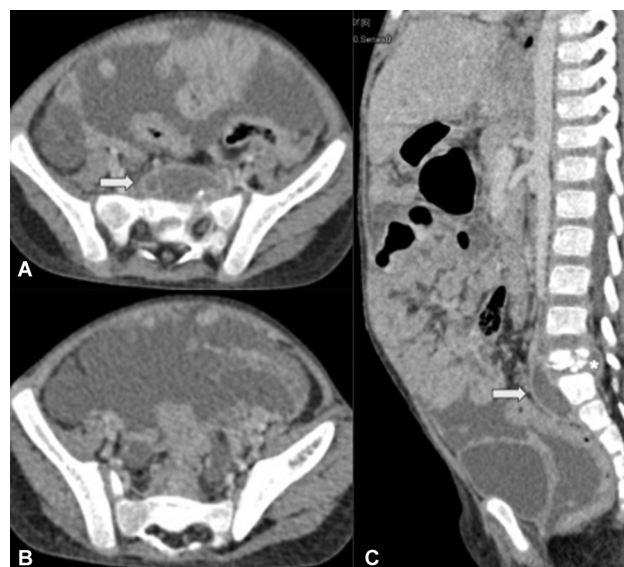


Fig. 3 Tubercular peritonitis—wet type. Axial (A, B) and sagittal (C) contrast-enhanced computed tomography images showing moderate ascites. Concomitant tuberculous spondylodiscitis at L5 to S1 level with destruction of L5 vertebral body and formation of subligamentous (arrow) and anterior epidural (asterisk) abscesses.

ascitic fluid is characteristically of high density (20–45 HU) on CT and shows delayed enhancement 15 to 20 minutes after contrast administration on MRI. Ascitic fluid examination shows low serum ascites albumin gradient (<1.1 g/dL), lymphocytosis, and elevated adenosine deaminase (>39 U/L). There may be associated peritoneal thickening (2–6 mm), which usually shows smooth enhancement, although occasionally peritoneal nodules (<5 mm) can also be seen. Omental involvement in tubercular peritonitis typically has a smudged appearance, implying infiltration with

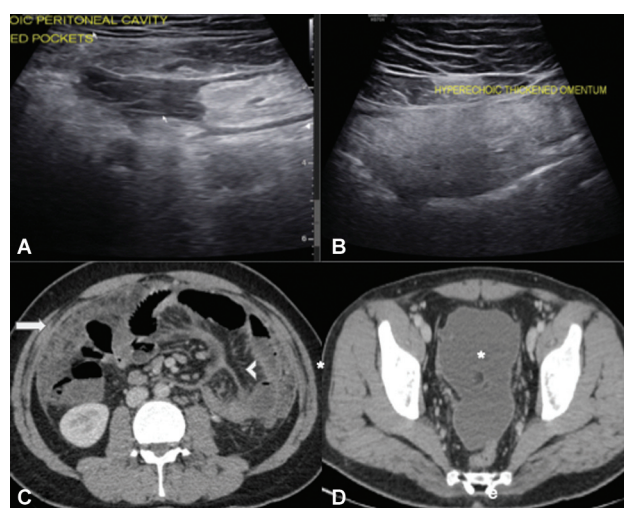


Fig. 4 Tubercular peritonitis—fibrotic type. Ultrasound abdomen showing septated ascites (A) and markedly thickened omentum (B). Axial contrast-enhanced computed tomography (CECT) abdomen (C, D) shows omental thickening with a smudged appearance (arrow), thickened mesenteric folds (arrowhead), and mild pelvic ascites (asterisk).

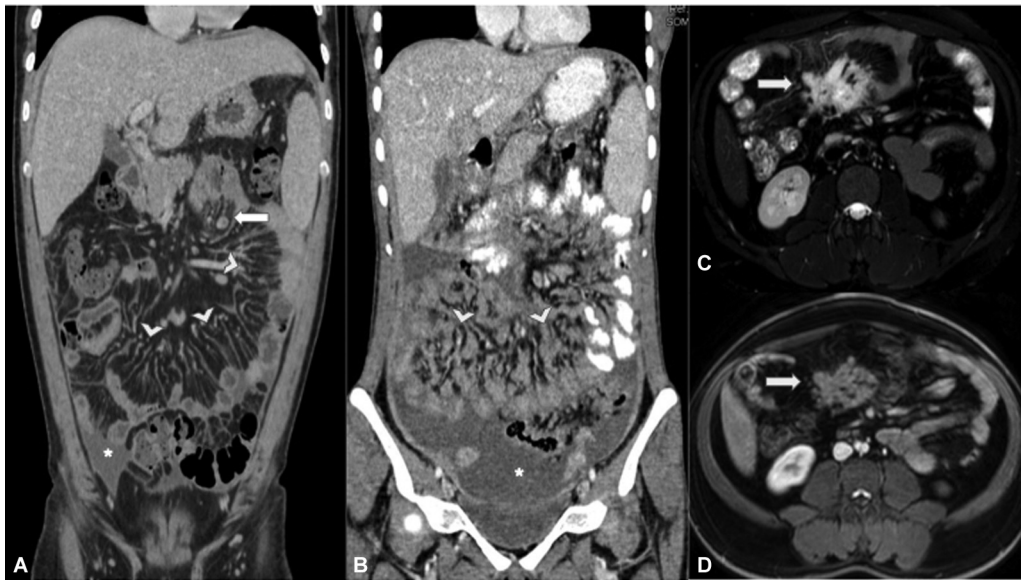


Fig. 5 Tuberculosis—mesenteric involvement. (A) Coronal contrast-enhanced computed tomography (CECT) showing thickened mesentery (arrowheads), necrotic lymphadenopathy (arrow) and ascites (asterisk). (B) Coronal CECT showing mesenteric macronodules (arrowheads) and ascites. Axial T2-weighted fat-suppressed (C), postcontrast T1-weighted images (D), showing an irregular mesenteric mass (arrow).

poorly defined lesions. Mesenteric involvement can be in the form of thickened mesenteric leaves, radiating thickened mesentery with a “stellate” configuration, mesenteric micronodules (<5 mm) or macronodules (>5 mm), and concomitant mesenteric lymphadenopathy or abscesses^{8,11} (► **Fig. 5**).

Differential diagnosis of tubercular peritonitis includes peritoneal carcinomatosis, mesothelioma, and nontubercular peritonitis. In peritoneal carcinomatosis and mesothelioma, the peritoneal and omental thickening is nodular compared with relatively smooth peritoneal thickening and smudged omentum in tubercular peritonitis. Additionally, ascitic volume is disproportionately small in mesothelioma.⁸ In a study by Ha et al, preservation of the omental line, mesenteric involvement with macronodules, necrotic or calcified lymphadenopathy, and concomitant splenic involvement also favored the diagnosis of tubercular peritonitis over carcinomatosis.^{15,16} Eventually, the diagnosis may depend upon ascitic fluid examination and/or peritoneal biopsy.

In advanced stages, the fibrotic type of tubercular peritonitis can progress to the formation of an abdominal cocoon (encapsulating peritoneal sclerosis). In this condition, there is encapsulation of small bowel loops in a thick fibrous membrane, leading to recurrent episodes of subacute intestinal obstruction. On barium meal follow-through studies, this appears as a clumping of dilated small bowel loops in a concertina-like configuration. The encapsulating membrane can also be visualized over the clumped bowel loops on high-resolution ultrasonography, CT, or MRI (► **Fig. 6**). Encapsulating peritoneal sclerosis is not always of tubercular etiology. It can also be idiopathic or secondary to long-term peritoneal dialysis, β -blockers, sarcoidosis, systemic lupus erythematosus, abdominal surgery, or malignancy. However, interloop adhesions are more common in tuberculous abdominal cocoons, which appear on CT as angulation and



Fig. 6 Sclerosing encapsulating peritonitis. Axial (A, B) and coronal (C) contrast-enhanced computed tomography images of abdomen showing clumped small bowel loops in central abdomen with overlying enhancing membrane (white arrowheads) and small pocket of loculated ascites (white asterisk).

kinking of the encapsulated bowel loops. Findings of necrotic abdominal lymphadenopathy and omental thickening with nodularity due to serosal tubercles are additional pointers on imaging indicative of a tuberculous cause. Internal hernias may also mimic an abdominal cocoon; however, the clustered bowel loops in internal hernias lack the overlying enhancing peritoneal membrane characteristic of an abdominal cocoon.¹⁷

Gastrointestinal Tuberculosis

Gastrointestinal TB usually occurs following the swallowing of infected sputum, ingestion of contaminated milk, hematogenous origin, or spread from contiguous organs. It can

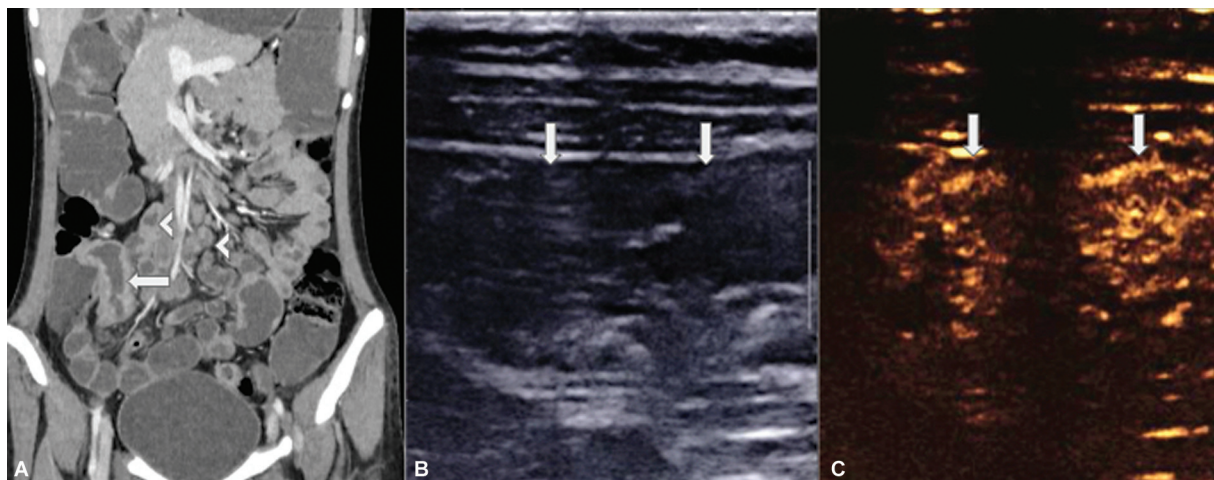


Fig. 7 Ileocecal tuberculosis. (A) Coronal contrast-enhanced computed tomography shows thickened terminal ileum and ileocecal junction (arrow) with necrotic mesenteric lymphadenopathy (arrowheads). (B) Ultrasound demonstrates concentric thickening of terminal ileum (arrows). (C) Contrast-enhanced ultrasound (CEUS) shows early full-thickness enhancement of bowel (arrows)—type 2 pattern.

involve any part of the gastrointestinal tract but has a propensity to involve the ileocecal region, which accounts for 90% of the cases.¹⁸ Clinically, patients present with abdominal pain, diarrhea, weight loss, and fever.¹⁹ Morphologically, gastrointestinal TB may be of three forms: ulcerative, hypertrophic, or ulcerohypertrophic. The ulcerative form is characterized by multiple small transverse ulcers. In the hypertrophic form, there is a pseudonodular appearance that can mimic malignancy. The ulcerohypertrophic form is a result of transverse ulceration with intervening mucosal nodularity giving rise to a “cobblestone” appearance.¹¹

Ileocecal Tuberculosis

The ileocecal region is the most frequent site of involvement (90% of cases) in gastrointestinal TB due to an abundance of lymphoid tissue and physiological intestinal stasis in this segment.²⁰ On ultrasound, concentric bowel wall thickening can be detected in the ileocecal region; when marked, it results in a pseudokidney appearance. In a retrospective study on the findings of intestinal TB on contrast-enhanced ultrasound, two patterns of enhancement were described by Yang et al: type 1 enhancement (13%) in which there was early serosal enhancement followed by gradual mucosal enhancement and type 2 enhancement (87%) in which the full thickness of the involved bowel showed early enhancement compared with normal bowel (►Fig. 7). Additionally, they categorized the enhancement of thickened bowel wall as homogeneous or heterogeneous; the heterogeneous enhancement pattern correlated with the presence of caseous necrosis in the intestinal muscle layer on histopathology.²¹

Traditionally, barium meal follow-through was performed in patients with suspected intestinal TB, which may show spasm or hypermotility of the ileocecal valve in the early stages of the disease. Subsequently, there can be nonspecific inflammatory changes in the form of mucosal fold thickening, luminal narrowing, and irregularity of the ileocecal region. Various classical signs of intestinal TB may be seen

such as Fleishner’s or inverted umbrella sign (thickening of ileocecal valve lips with wide gaping and narrowed terminal ileum), Stierlin’s sign (rapid emptying of cecum due to irritable mucosa), gooseneck deformity (pulled-up cecum with obtuse angulation of ileocecal junction and dilated terminal ileum), and string sign (fibrotic narrowing of terminal narrowing) (►Fig. 8).² However, barium studies have been largely given up as they are low-yield, high-dose studies and have been replaced by cross-sectional enterography.²²

Nowadays, CT enterography is widely employed for the evaluation of various intestinal diseases. Its main advantage over barium meal follow-through is its ability to concurrently demonstrate luminal, mural, and extraluminal features of intestinal TB.²³ Magnetic resonance (MR) enterography is a useful adjunct and its use is mostly limited to patients undergoing repeated imaging to obviate exposure to ionizing radiation. Findings of active ileocecal TB on CT or MR enterography include concentric symmetric mural thickening involving the terminal ileum, ileocecal junction, or cecum. The thickened bowel wall mostly shows homogeneous enhancement; less commonly stratified mural enhancement can be seen. Additionally, extraintestinal findings of abdominal TB such as necrotic mesenteric lymphadenitis, peritoneal, or hepatosplenic TB may be detected (►Fig. 9). In the chronic inactive stage, there is stricture formation that appears as hypoenhancing bowel wall thickening with upstream bowel dilatation; enteroliths may also be present in the dilated bowel. Commonly encountered complications of intestinal TB are obstruction and perforation; less commonly, the formation of abscesses and fistulae can occur.^{2,23,24}

Findings of active or inactive pulmonary TB are detected in up to 15% of patients with intestinal TB on chest radiographs.²⁵ A contrast-enhanced CT scan of the thorax can further aid in the diagnosis of intestinal TB; as a recent study found features of concomitant active pulmonary TB such as centrilobular/miliary nodules, consolidation, thick-walled cavitary lesions, necrotic mediastinal lymphadenopathy in 38% of patients with intestinal TB.²⁶ Therefore, a thoracic CT

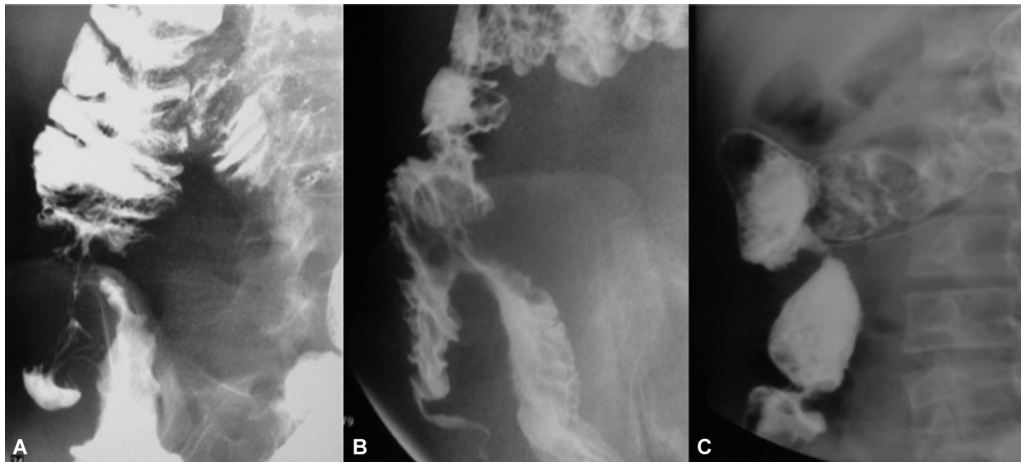


Fig. 8 Ileocecal tuberculosis—Barium meal follow-through (BMFT). (A) Stierlin's sign—narrowed ileocecal junction and contracted cecum. (B) Fleischner's sign—thickened ileocecal valve with inverted umbrella appearance. (C) Gooseneck deformity—pulled up cecum with obtuse angulation of ileocecal junction and dilated terminal ileum.



Fig. 9 Ileocecal tuberculosis—hypertrophic form. Axial (A, B) and coronal (C, D) contrast-enhanced computed tomography demonstrate markedly thickened terminal ileum (arrows). Other small bowel loops also show mild mural thickening and hyperenhancement (arrowheads) with associated necrotic mesenteric lymphadenopathy (asterisk).

should be concomitantly performed when there is high suspicion of intestinal TB.

The chief differential diagnosis of ileocecal TB is Crohn's disease; the radiological, endoscopic, and histopathological features for differentiating the two entities are summarized in ►Table 3.^{2,27,28} Other differentials of ileocecal TB include infections such as amebiasis, *Salmonella* enteritis, and *Yersinia* enterocolitis, as well as neoplasms such as lymphoma and cecal carcinoma.

Esophageal Tuberculosis

Esophageal TB accounts for approximately 2.8% of cases of gastrointestinal TB.²⁹ Primary esophageal TB is uncommon due to innate protection conferred by stratified squamous epithelial lining, esophageal peristalsis, and salivary enzymes. Esophageal TB usually occurs secondary to mediastinal, pulmonary, or spinal TB.³⁰ Patients clinically present

with dysphagia, odynophagia, retrosternal pain, hematemesis, and constitutional symptoms.

The most frequent site of involvement is the middle third of the esophagus due to its proximity to the mediastinal nodes. On barium swallow, there can be extrinsic compression over the esophagus, strictures, formation of traction diverticula, sinus tracts or fistula, or a pseudotumor appearance. CT reveals nonspecific esophageal wall thickening (►Fig. 10). However, concomitant pulmonary parenchymal abnormalities or mediastinal lymphadenopathy may aid in the diagnosis.³¹ Carcinoma of the esophagus and Crohn's disease are two close imaging differentials.

Gastric Tuberculosis

Primary gastric TB is rare accounting for only 0.4 to 2% of cases of gastrointestinal TB; this is because of the acidic environment, lack of gastric lymphoid tissue, and relatively

Table 3 Ileocecal tuberculosis versus Crohn's disease

	Ileocecal tuberculosis	Crohn's disease
Clinical features		
● Duration of symptoms	● Shorter	● Longer
● Symptoms	● Fever, abdominal distension, loss of appetite, and weight	● Diarrhea, hematochezia, pain
Radiological findings		
● Site of involvement	● Cecum > ileum	● Ileum > cecum
● Length of involvement	● Shorter	● Longer (>3 cm)
● Skip lesions	● Infrequent	● Frequent (>3 in number)
● Bowel wall enhancement	● Usually homogeneous	● Stratified enhancement
● Enteroliths	● Common	● Uncommon
● Fistulae and mesenteric abscess formation	● Uncommon	● Common
● Wall thickening	● Concentric	● Eccentric
● Comb's sign	● Absent	● Present
● Fibrofatty proliferation	● Uncommon	● Common
● Mesenteric nodes	● Necrotic, larger nodes, and at other regions as well	● Non-necrotic, mildly enlarged
● Ascites and omental thickening	● Frequently present	● Infrequent
● Solid organ involvement	● Hepatic and splenic granulomas	● Rare
● Visceral fat indices	● Lower VF/SC ratio (≤ 0.63)	● Higher VF/SC ratio (≥ 0.63)
Colonoscopic findings		
● Ulcers	● Transverse	● Longitudinal, aphthous
● Cobblestoning	● Uncommon	● Common
● Pseudopolyps	● Uncommon	● Common
● Skip lesions	● Uncommon	● Common
● Perianal lesions	● Uncommon	● Common
● Patulous ileocecal valve	● Common	● Uncommon
Histopathological findings		
● Granulomas	● Confluent, caseating	● Microgranuloma, noncaseating
● Disproportionate submucosal inflammation	● Common	● Uncommon
Laboratory investigations	Positive Mantoux test, interferon-gamma release assay (Quantiferon TB), acid-fast bacilli on Ziehl–Neelsen staining, Bactec/Lowenstein–Jensen medium culture	Positive anti- <i>Saccharomyces cerevisiae</i> antibody, anti-glycan antibody, anti-outer membrane porin C antibody

Abbreviation: VF/SC, visceral fat/subcutaneous fat.

thick gastric mucosal lining.³² Secondary gastric involvement is mostly associated with hematogenous spread from pulmonary infection or retrograde lymphatic spread from other abdominal organs. Patients may present with pain epigastrium, loss of weight, nausea, vomiting, hematemesis, or gastric outlet obstruction.³³

The gastric antrum and distal body are the most commonly involved parts. Ulcerative lesion of the lesser curvature and pylorus is the most common morphological pattern of gastric TB, which can mimic peptic ulcer disease. Less commonly, it may present as hypertrophic form (resembling gastric malignancy or pyloric stenosis) or miliary lesions.¹²

Adjacent lymphadenitis is often seen (→ **Figs. 11, 12**). Rarely, gastric TB causes sinus tracts and fistula formation.³⁴

Duodenal Tuberculosis

Duodenal TB also accounts for 2% of cases of gastrointestinal TB.³⁵ The majority of patients present with obstructive symptoms, whereas 10 to 20% of patients have dyspeptic symptoms. Duodenal involvement in TB may be intrinsic (ulcerative, hypertrophic, or ulcerohypertrophic form) or extrinsic (compression of the duodenum by periduodenal nodes).³⁶ In patients with obstructive symptoms, radiological findings include duodenal wall thickening with strictures

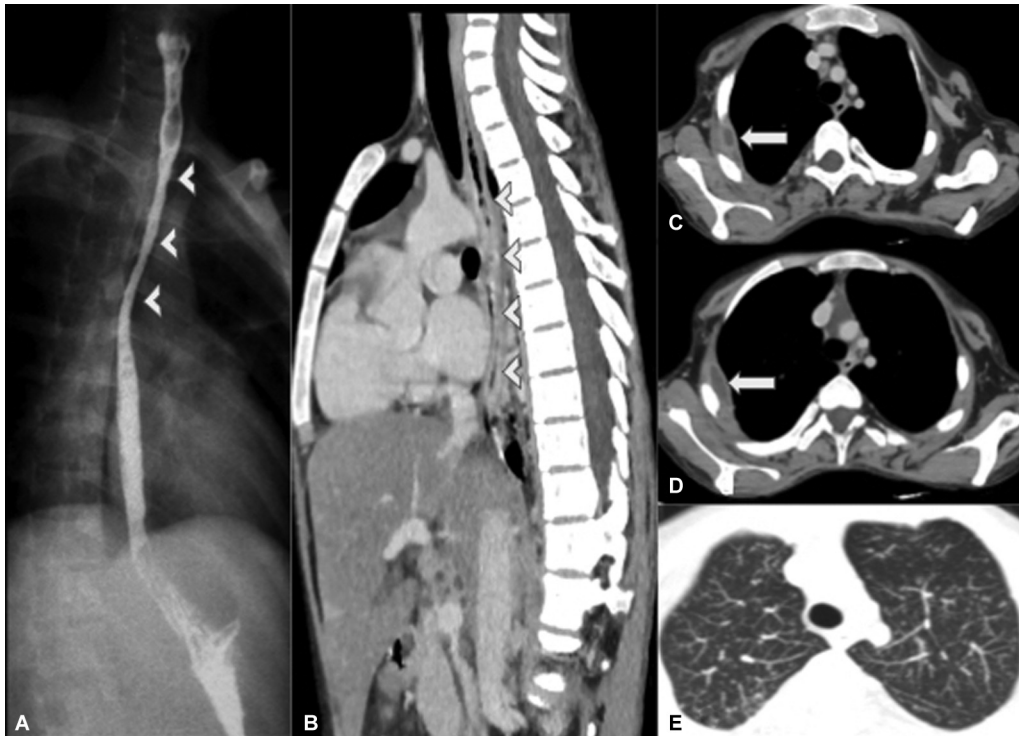


Fig. 10 Esophageal tuberculosis. Barium swallow (A) demonstrates long-segment narrowing of upper and midthoracic esophagus (arrowheads). Contrast-enhanced computed tomography thorax (B–E) shows long-segment mild thoracic esophageal thickening (arrowheads) with right loculated empyema (arrows) and centrilobular nodules in left upper lobe (E).

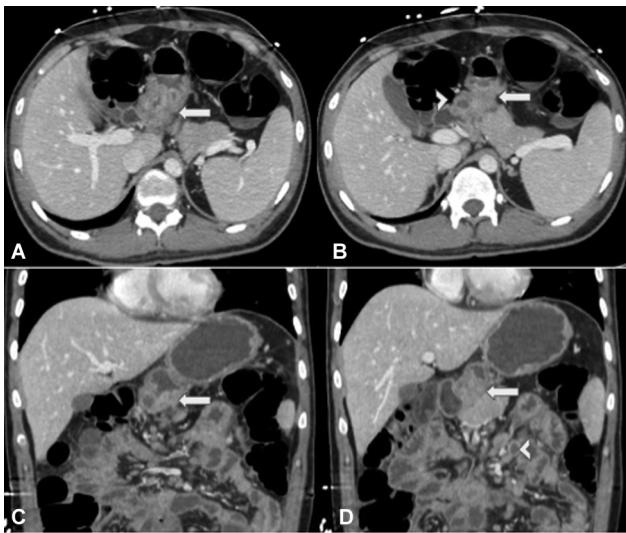


Fig. 11 Gastric tuberculosis. Axial (A, B) and coronal (C, D) images of contrast-enhanced computed tomography abdomen showing thickening of wall of stomach in antropyloric region (white arrows) with necrotic prepyloric (arrowhead in B) and mesenteric nodes (arrowhead in D).

or polypoid masses in cases of intrinsic duodenal TB, whereas widening of duodenal C-loop with luminal narrowing and periduodenal lymphadenopathy is seen in cases of extrinsic duodenal involvement. The third part of the duodenum is a common site of obstruction in duodenal TB, and its imaging appearance may resemble superior mesenteric artery

syndrome (→Fig. 13). In patients with dyspeptic symptoms, duodenal ulcers are frequently encountered³⁷ (→Fig. 14). Occasionally, the biliary system can be secondarily involved with biliary dilatation or the formation of choledochoduodenal fistula.³⁸ Upper gastrointestinal Crohn's disease and neoplasia are the important differentials of duodenal TB.

Jejunal or Ileal Tuberculosis

Isolated involvement of jejunum or ileum occurs less frequently as compared with ileocecal TB. Imaging findings are nonspecific and comprise ulcers, mucosal fold thickening and strictures on barium meal follow-through, and bowel wall thickening on CT or MRI^{8,11} (→Figs. 15, 16).

Colorectal Tuberculosis

Isolated large bowel involvement is seen in nearly 10% of cases of gastrointestinal TB.³⁹ Patients usually present with colicky abdominal pain, diarrhea, and weight loss. The ascending colon and transverse colon are the most commonly involved sites. Multifocal involvement can also be present.^{39,40} On imaging, there is spasm, increased motility, thickened mucosal folds, and aphthous ulcerations in the early stages of the disease. In advanced cases, there is transverse or circumferential ulceration, mucosal nodularity, inflammatory polyps, strictures with luminal narrowing (→Fig. 17), and hypertrophic masses mimicking adenocarcinoma of the colon.⁴¹ The other differentials of colonic TB are inflammatory bowel disease, amebic colitis, pseudomembranous colitis, and ischemic colitis.

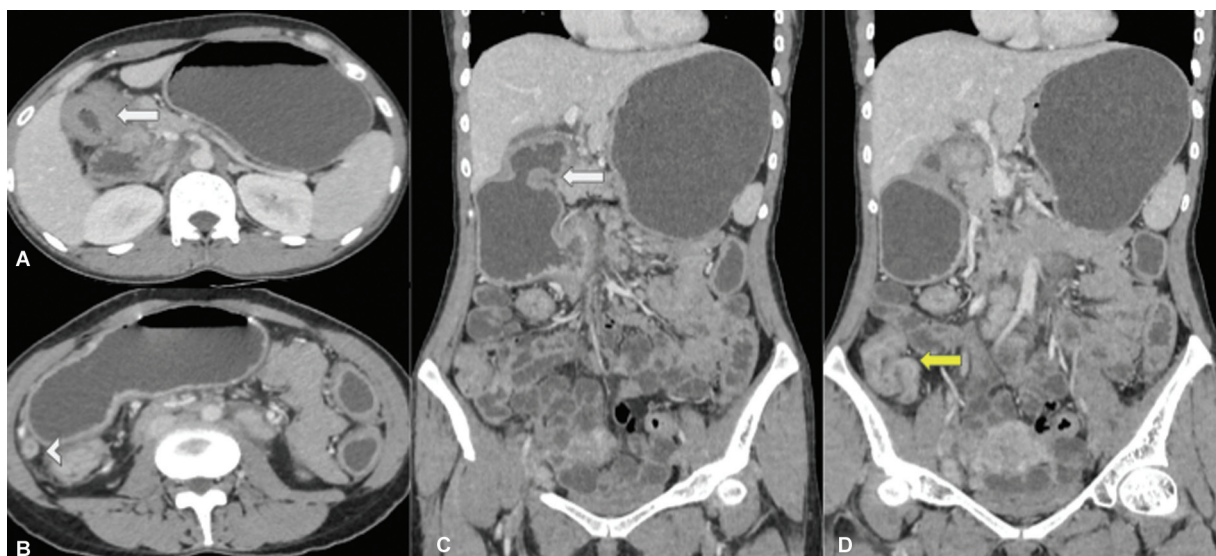


Fig. 12 Gastroduodenal tuberculosis. Axial (A, B) and coronal (C, D) contrast-enhanced computed tomography demonstrate pyloric and duodenal bulb thickening (white arrows) causing dilated stomach due to gastric outlet obstruction. Associated necrotic perigastric lymphadenopathy (arrowhead) and ileocecal thickening (yellow arrow).

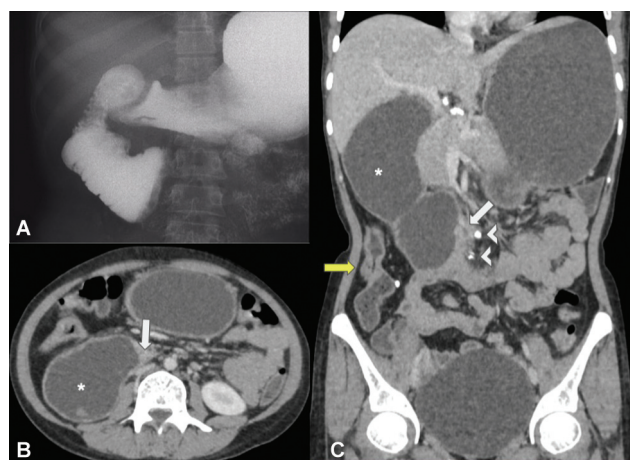


Fig. 13 Duodenal tuberculosis (obstructive form). Barium meal (A) shows vertical cutoff of duodenum. Axial (B) and coronal (C) contrast-enhanced computed tomography demonstrate duodenal third part thickening (white arrow) with proximal duodenal dilatation (asterisk), calcified nodes (arrowheads), and ileocecal thickening (yellow arrow).

Anoperineal Tuberculosis

Anoperineal TB is rare, accounting for less than 1% of cases of gastrointestinal TB.⁴² Patients present with perianal pain, discharge, hematochezia, or constipation, frequently accompanied by constitutional symptoms. Morphologically, anoperineal TB can appear as fistula-in-ano, anal ulcerations (multiple in miliary disease), short annular strictures, lupoid form (submucosal nodule with overlying ulceration), and verrucous form (wart-like).⁴³ On imaging, the most common finding is a perianal fistula (→ Fig. 18). Simple intersphincteric fistulas are the most frequently encountered type of anal TB.⁴⁴ However, the presence of nonhealing or recurrent fistula, complex fistula, suprasphincteric and transsphincteric type of fistula, underlying anorectal thickening, associated inguinal or pelvic lymphadenopathy, and occasionally evidence of osteomyelitis of pelvic bones should alert the radiologist to the possibility of underlying anal TB. The chief differentials of anoperineal TB include cryptoglandular fistula, Crohn's disease, pelvic actinomycosis, and lymphogranuloma venereum.⁴²

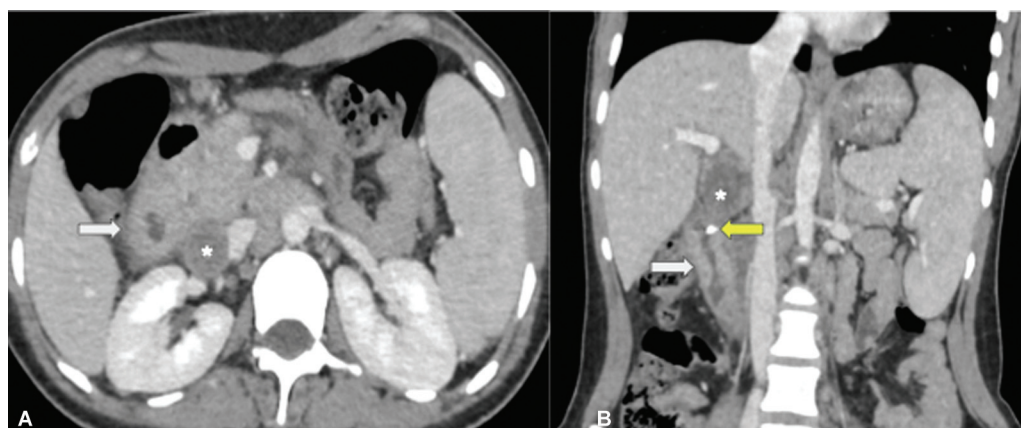


Fig. 14 Duodenal tuberculosis (dyspeptic form). Axial (A) and coronal (B) contrast-enhanced computed tomography show thickened duodenum second part (white arrow), with necrotic paracaval lymphadenopathy (asterisk). Hemostatic clip applied endoscopically for bleeding ulcer visible at proximal part of thickened duodenum (yellow arrow).

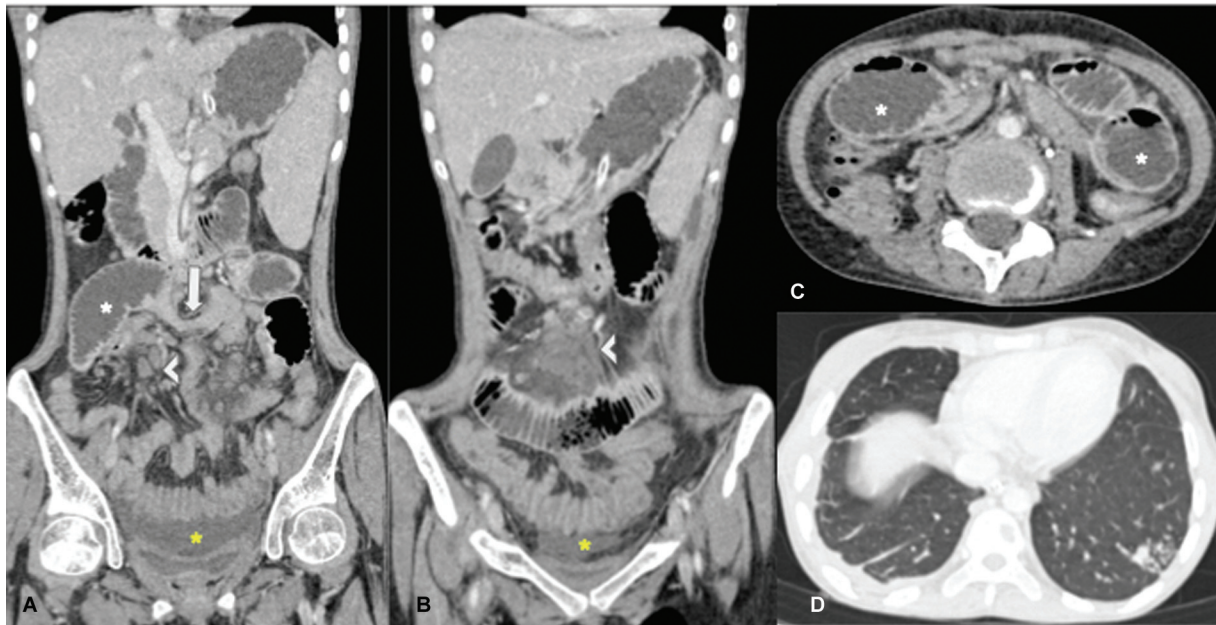


Fig. 15 Jejunal tuberculosis. Coronal (A, B) and axial (C) contrast-enhanced computed tomography shows mid-jejunal stricture (arrow) with upstream jejunal dilatation (white asterisk), necrotic mesenteric lymphadenopathy (arrowhead), and ascites (yellow asterisk). Axial computed tomography chest (D) shows centrilobular nodules in left lung base.

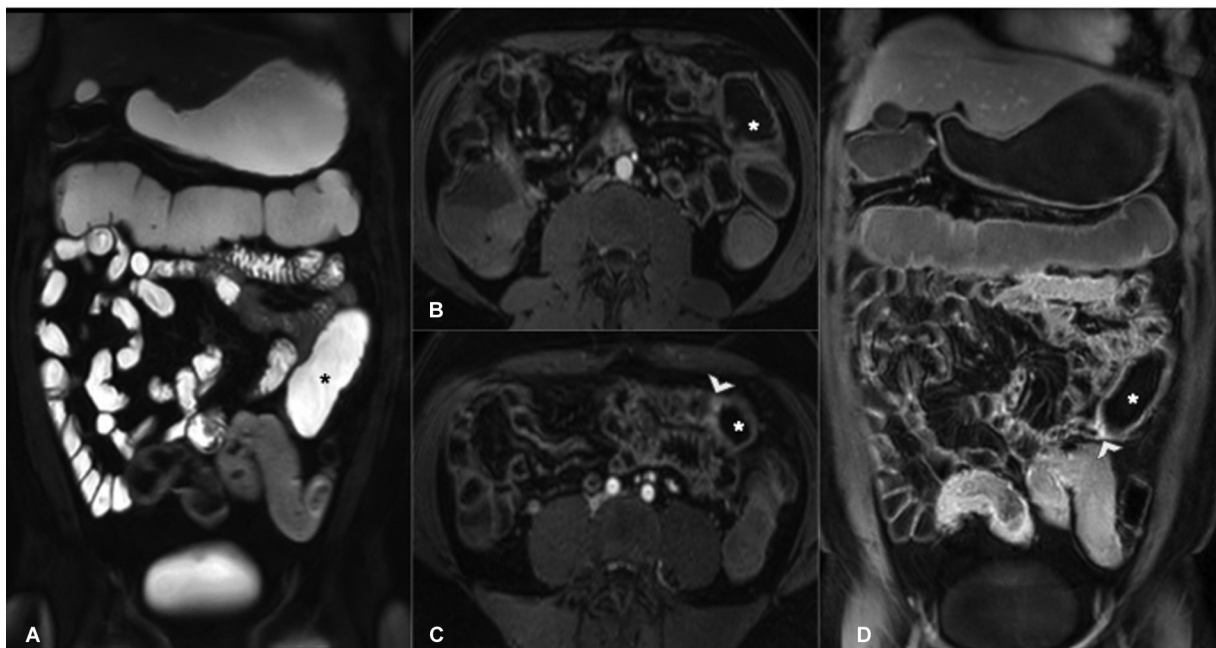


Fig. 16 Jejunal tuberculosis. Magnetic resonance enterography with coronal T2-weighted image (A), axial (B, C) and coronal (D) postcontrast T1-weighted fat-suppressed image showing jejunal dilatation (asterisk) due to short segment jejunal thickening (arrowheads in C, D).

Complications of Gastrointestinal Tuberculosis

In patients with gastrointestinal TB, serious complications such as intestinal obstruction, perforation or gastrointestinal hemorrhage, formation of abscesses and fistula, and malabsorption may ensue (►Table 4). Obstruction is the most common complication (12–60% of cases), which can occur due to hyperplastic bowel wall thickening, strictures, or adhesions.⁴⁵ On imaging, dilated bowel loops (caliber of small bowel > 3 cm and/or large bowel > 6 cm) are visualized (►Fig. 19).

Perforation occurs in 1 to 10% of cases of intestinal TB, usually seen in patients with severe disease, and is associated with a high mortality rate. In India, TB is one of the leading causes of small bowel perforation, second only to enteric fever.³² Peritoneal involvement in TB with reactive thickening and adhesion formation likely reduces the incidence of perforations in intestinal TB. Tubercular perforations are solitary in the vast majority of cases (90%), with ileum being the most common site. The perforations usually occur due to

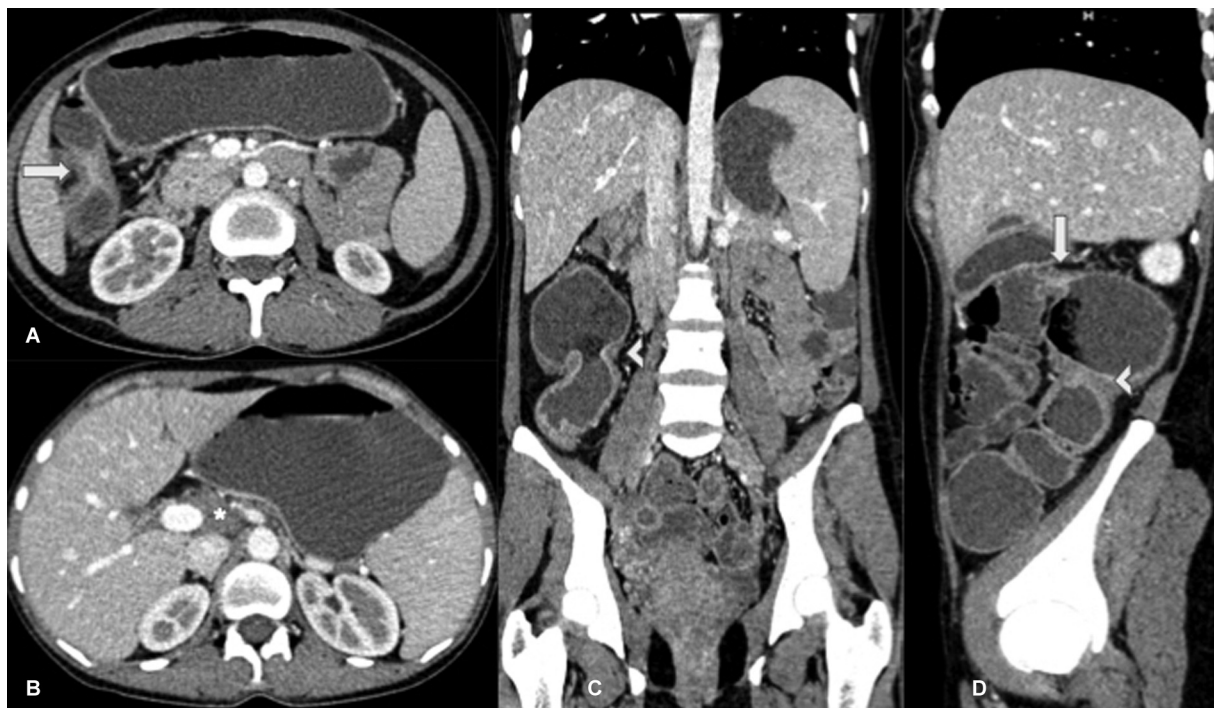


Fig. 17 Colonic tuberculosis. Axial (A, B), coronal (B), and sagittal (D) contrast-enhanced computed tomography images show thickening of hepatic flexure of colon (white arrow), with associated pulled up and thickened ileocecal junction (white arrowhead) and necrotic periportal lymphadenopathy (white asterisk).

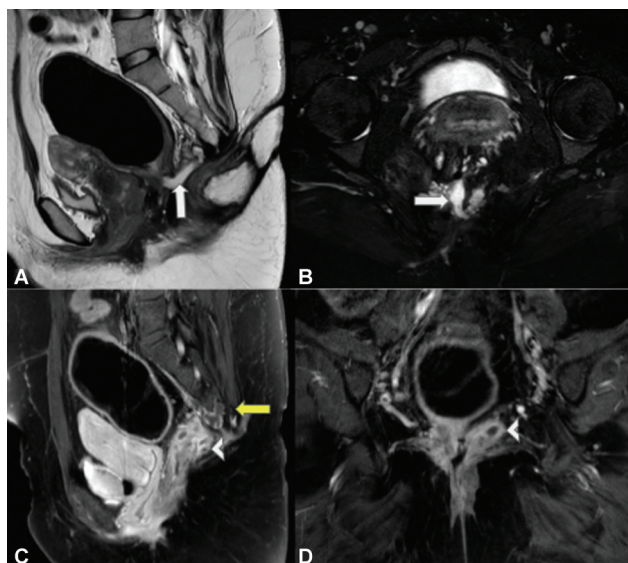


Fig. 18 Anal tuberculosis. Sagittal T2-weighted (T2W) (A) and axial T2W-fat-suppressed (B) images showing fistula extending from lower rectum to sacrum (arrows). Sagittal (C) and coronal (D) postcontrast T1-weighted-fat-suppressed images showing presacral collections (arrowheads), with extension to lower sacrum (yellow arrow).

pressure necrosis in the segment of the intestine proximal to a stricture (→ Fig. 20). Perforations may be free or localized. CT is much more sensitive than radiographs in detecting perforation and can demonstrate it in 81% of cases.⁴⁶ Ultrasound can reveal fluid collections with extraluminal air foci around the thickened diseased bowel. Rather than primary repair, these patients require resection of the affected bowel segment with primary anastomosis or exteriorization.⁴⁷

Intraperitoneal tuberculous abscess is an uncommon complication that appears on CT as a rim-enhancing multi-septated hypodense mass. These may form due to the conglomeration of necrotic nodes or due to the encapsulation of intraperitoneal caseous fluid between the bowel, solid abdominal viscera, and peritoneum. Both forms of tuberculous abscesses are associated with necrotic abdominal lymphadenopathy. However, the nodal abscesses are close to other enlarged necrotic nodes, whereas encapsulated abscesses are located at sites remote from the necrotic nodes.⁴⁸ Large abscesses may require percutaneous drainage. Although more common in Crohn's disease, patients with abdominal TB may also present occasionally with enterocutaneous (→ Fig. 21), enteroenteric, enterovesical, and perianal fistula.^{49–51}

Gastrointestinal hemorrhage is a relatively infrequent complication of intestinal TB and is rarely massive. Hemorrhage is usually due to mucosal ulceration and occasionally due to mesenteric pseudoaneurysm formation due to tuberculous vasculitis. The ileocecal region is the most common site of bleed. If the hemorrhage is not controlled medically and endoscopically, these patients require resection of the culprit segment in conjunction with ATT. Angiographic intervention may be contemplated in case of active contrast extravasation or pseudoaneurysm.⁵²

Malabsorption syndrome can occur due to stasis from intestinal strictures or due to extensive mucosal involvement. In the former case, dilated bowel loops proximal to a stricture are visualized, whereas in the latter case, there can be radiological evidence of extensive mucosal fold thickening.⁴⁵

Table 4 Complications of gastrointestinal tuberculosis

Complication	Imaging finding
Intestinal obstruction	<ul style="list-style-type: none"> ● Dilated bowel loops (caliber of small bowel > 3 cm, large bowel > 6 cm, cecum > 9 cm) ● Small bowel feces sign ● Enteroliths ● Transition zone
Intestinal perforation	<ul style="list-style-type: none"> ● Solitary (90%) ● Ileum: most common site ● Free or localized ● X-ray/CT: pneumoperitoneum ● Ultrasound/CT: fluid collections with extraluminal air foci in vicinity of thickened diseased bowel
Gastrointestinal hemorrhage	<ul style="list-style-type: none"> ● Ileocecal region: most common site ● CT: affected site shows bowel wall thickening ± active contrast extravasation ● Tuberculous vasculitis with mesenteric artery pseudoaneurysm: rare
Intraperitoneal tuberculous abscess	<ul style="list-style-type: none"> ● Rim-enhancing multiseptated hypodense mass ● Associated necrotic mesenteric lymphadenopathy and/or peritoneal tuberculosis
Sinus and fistula formation	<ul style="list-style-type: none"> ● Enterocutaneous/colocutaneous fistula: linear or complex tract from bowel to skin ● Enterointestinal fistula: clumped bowel loops with stellate configuration ● Enterovesical fistula: tract from bowel to urinary bladder
Malabsorption	<ul style="list-style-type: none"> ● Dilated intestinal loops due to stricture ● Diffuse mucosal fold thickening

Abbreviation: CT, computed tomography.

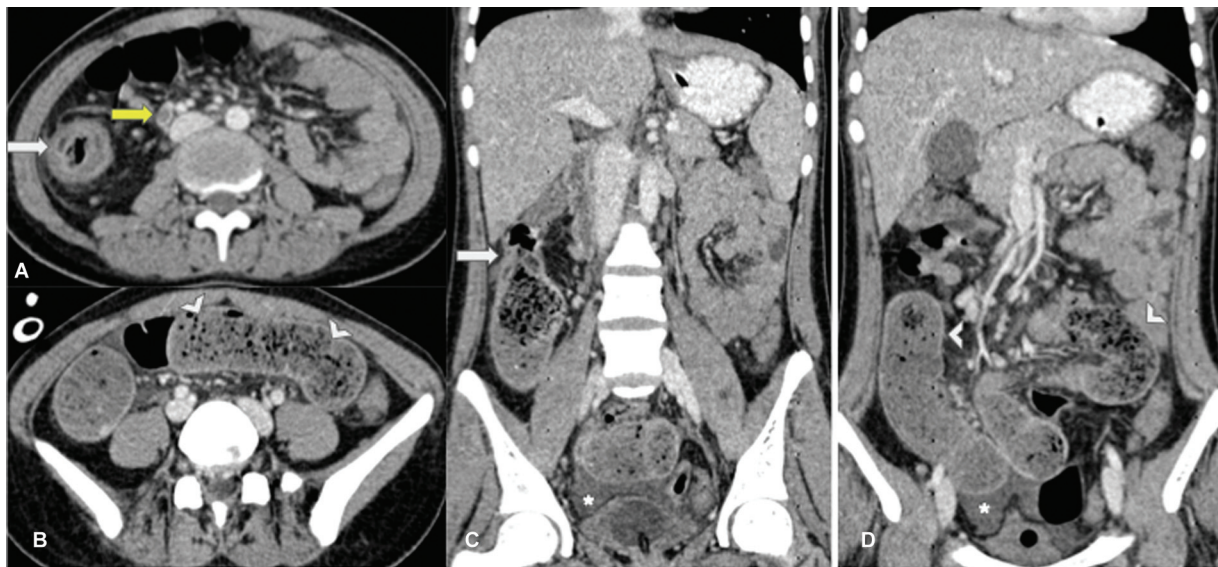


Fig. 19 Ileocecal tuberculosis causing intestinal obstruction. Axial (A, B) and coronal (C, D) contrast-enhanced computed tomography shows pulled-up thickened ileocecal junction (white arrow) causing ileal dilatation with small bowel feces sign (arrowheads). Associated necrotic mesenteric lymphadenopathy (yellow arrow) and ascites (asterisk).

Solid Organ Tuberculosis

Hepatosplenic Tuberculosis

Hepatosplenic involvement is usually secondary to pulmonary or miliary TB and has been found on postmortem evaluation in 80 to 100% cases of disseminated TB.¹⁴ Dissemination of tubercle bacilli to the liver and spleen is by hematogenous route in these cases and results in a micronodular pattern on imaging. This manifests as hepatosplenomegaly with diffuse parenchymal hyperechogenicity (“bright liver and spleen” appearance) on ultrasound. Hypoechoic miliary nodules of

0.5 to 2 mm can also be visualized on high-resolution ultrasonography. These nodules may appear as tiny hypodense lesions on CT. Alternatively, if the lesion size is less than the resolving power of CT, only hepatosplenomegaly with homogenous or heterogenous parenchymal enhancement may be apparent.¹¹ In the chronic stage, calcified nodules are seen. The differential diagnosis for this form of hepatosplenic TB includes miliary metastases, lymphoma, and other granulomatous disorders such as sarcoidosis and fungal infections.²⁰

Less commonly, in approximately 20% of cases, hepatosplenic TB occurs by dissemination through the portal vein,

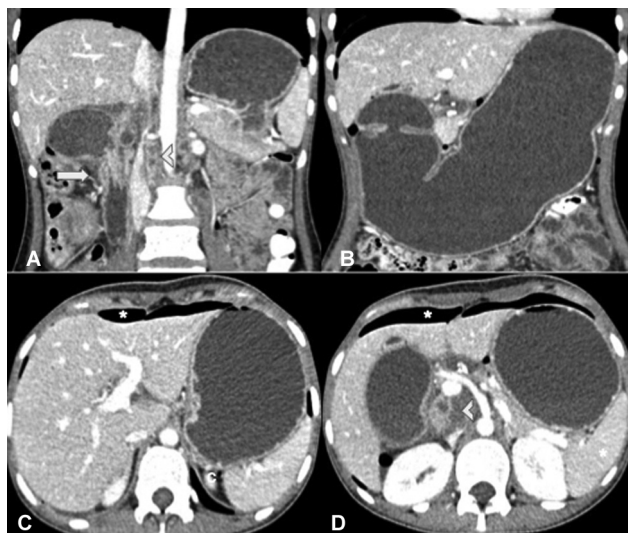


Fig. 20 Duodenal tuberculosis with perforation. Coronal (A, B) and axial (C, D) contrast-enhanced computed tomography shows circumferential thickening of duodenum second part (arrow) with necrotic upper retroperitoneal lymphadenopathy (arrowheads). Gastric outlet obstruction with overdistended stomach and perforation evident by pneumoperitoneum (asterisk).

which results in a macronodular imaging pattern due to tuberculomas or tubercular abscess formation.⁹ This is characterized by single or multiple lesions of 1- to 3-cm size, which are typically hypoechoic on ultrasound but maybe hyperechoic in earlier stages before the onset of caseous necrosis. The lesions are hypodense on CT with no or minimal peripheral enhancement. The central hypodense part is on account of the caseous necrosis, whereas the peripheral rim is due to the granulomatous inflammatory response. Conglomeration of the lesions can give a honeycomb appearance (→Fig. 22). On MRI, the lesions are hypointense on T1-weighted sequences, hyperintense with an intermediate signal intensity rim on T2W sequences and show diffusion restriction and peripheral enhancement. Like tuberculomas elsewhere, the macronodular form of hepatosplenic TB can also be hypointense on T2W sequences.⁵³ Portal vein thrombosis and portal hypertension have been infrequently reported in association with the macronodular form of hepatic TB.⁵⁴ Micronodular and macronodular lesions can also occur simultaneously in the same patient giving rise to a mixed pattern. The differential diagnosis for the macronodular form of hepatosplenic TB includes metastases, abscesses, and primary malignancies.³⁴

Serohepatic type is the rarest form of hepatic TB. This is characterized by multiple hypodense subcapsular lesions without significant enhancement on CT (→Fig. 23). The subcapsular lesions appear hypointense on T1W sequences and hyperintense on T2W sequences with subtle peripheral enhancement. This morphological pattern is known as the “frosted liver” appearance.^{55,56} Serohepatic TB should be considered in the differential diagnosis of lesions causing scalloping of liver outline, along with other conditions such as pseudomyxoma peritonei, peritoneal carcinomatosis, and lymphomatosis.⁵⁷

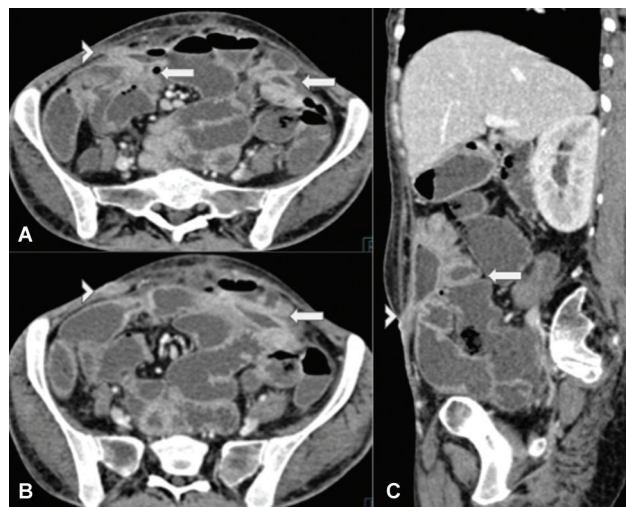


Fig. 21 Enterocutaneous fistula. Axial (A, B) and sagittal (C) contrast-enhanced computed tomography images show thickening and hyperenhancement of small bowel loops (arrows) with a fistulous tract extending from an ileal loop in right lumbar region to overlying skin (arrowheads).

Pancreatic Tuberculosis

Isolated pancreatic involvement in TB is quite rare. On imaging, it manifests as a focal pancreatic mass or less commonly as diffuse pancreatic enlargement. The focal form of pancreatic TB has a predilection for the head of the pancreas and appears as a hypoechoic solid or complex cystic mass on ultrasound.⁵⁸ On CT, it appears as a hypodense solid or solid cystic lesion, which is hypoenhancing to the normal pancreatic parenchyma⁵⁹ (→Fig. 24). On MRI, it appears as a heterogeneously enhancing mass, which is hypointense on T1W and of heterogeneous signal intensity on T2W sequences. In the diffuse form of pancreatic TB, a bulky pancreas with heterogeneous enhancement is seen on CT and MRI. Diffuse narrowing of the main pancreatic duct may also be present in magnetic resonance cholangiopancreatography (MRCP).⁶⁰

The focal form of pancreatic TB mimics pancreatic ductal adenocarcinoma or pancreatic cystic neoplasms, especially if vascular involvement and necrotic peripancreatic lymphadenopathy are present. However, unlike pancreatic ductal adenocarcinoma, upstream dilatation of the main pancreatic duct and common bile duct are uncommon in pancreatic TB. The presence of punctate or coarse calcific foci within the lesions also favors a diagnosis of TB over adenocarcinoma.⁵⁹ The differential diagnoses for the diffuse form of pancreatic TB include acute pancreatitis, autoimmune pancreatitis, and pancreatic lymphoma.⁶⁰ It is difficult to diagnose pancreatic TB in the absence of concomitant involvement of other sites such as bowel or peritoneum, and pancreatic ductal adenocarcinoma remains the first diagnosis of a focal pancreatic mass. Therefore, endoscopic ultrasound-guided fine-needle aspiration cytology is essential to clinch the diagnosis of pancreatic TB and rule out malignancy.

Tuberculosis of the Gallbladder and Biliary Tree

TB of the gallbladder is rarely encountered, as the wall of the gallbladder is relatively resistant to *M. tuberculosis*, and the

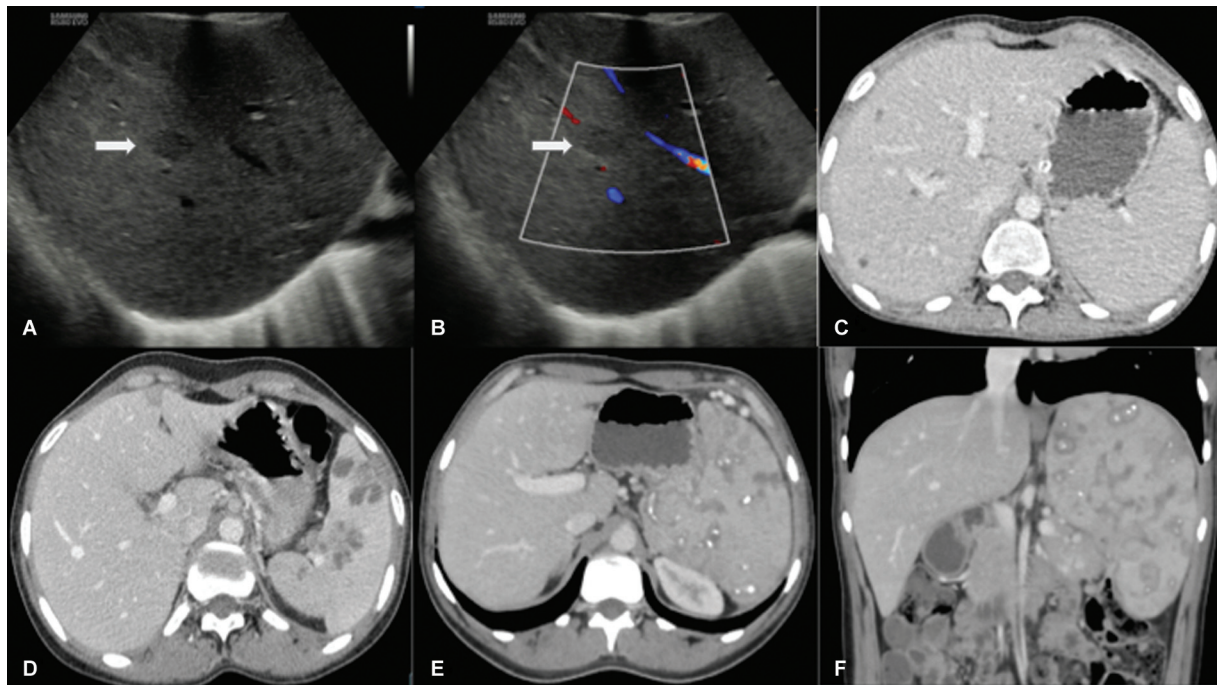


Fig. 22 Hepatosplenic tuberculosis. Ultrasound (A) and Doppler (B) show hypoechoic nonvascular liver lesion (arrow). Axial contrast-enhanced computed tomography (CECT) abdomen shows hypodense hepatic nodules (C) and splenic honeycomb lesions (D). Axial (E) and coronal (F) CECT show hypodense splenic lesions with calcification.

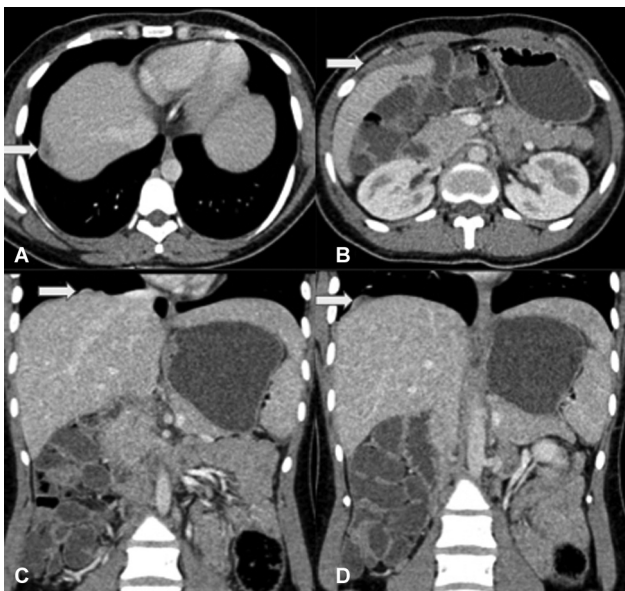


Fig. 23 Serohepatic tuberculosis. Axial (A, B) and coronal (C, D) contrast-enhanced computed tomography images of the abdomen show subcapsular hypoenhancing lesions over the liver surface (white arrows).

alkaline nature of bile also inhibits its growth.^{12,61} On CT, three patterns of involvement have been described by Xu et al. Most commonly, gallbladder TB presents as diffuse thickening of the gallbladder wall. Less frequently, it appears mass like or polypoidal.⁶² Thus, gallbladder TB mimics cholecystitis or carcinoma of the gallbladder. Diagnosis is usually made on histopathological examination of cholecystectomy specimens rather than on imaging.⁶³



Fig. 24 Pancreatic tuberculosis. Axial (A) and coronal (B) contrast-enhanced computed tomography (CECT) show hypoenhancing lesion in uncinata (asterisk), common duct narrowing (arrow) and necrotic lymphadenopathy (arrowhead). Post antitubercular treatment axial (C) and coronal (D) CECT show uncinata calcifications (arrowhead), biliary stent in situ (arrow).

Tubercular involvement of the biliary tree has an estimated annual incidence of 0.1%.⁶⁴ Biliary tree involvement is usually secondary to extrinsic compression of bile ducts by enlarged periportal nodes or hepatic granulomas. Less commonly, tubercular cholangitis occurs, which is characterized by multifocal strictures of intrahepatic bile ducts; involvement of extrahepatic bile duct is rare.⁵³ The strictures can lead to intrahepatic biliary radicle dilatation and



Fig. 25 Adrenal tuberculosis. (A) Axial contrast-enhanced computed tomography showing bulky left adrenal gland with calcific speck (arrowhead). Axial T1-weighted (T1W) (B) and T2-weighted (T2W) (C) magnetic resonance imaging showing bilateral bulky adrenal glands (arrows) with intermediate signal on T1W and hypointense signal on T2W sequence.

parenchymal atrophy mimicking cholangiocarcinoma. A beaded pattern with alternating strictures and dilatation may also be seen on MRCP; this resembles primary sclerosing cholangitis or IgG4-related cholangiopathy.²⁰

Adrenal Tuberculosis

Adrenal glands are the most commonly involved endocrine glands in extrapulmonary TB. Patients may present with adrenocortical insufficiency (Addison's disease) if more than 90% of the gland is affected.⁶⁵ The involvement is bilateral in majority of cases due to hematogenous route of spread of infection. In the initial stages of the disease, approximately half the patients may have mass-like enlargement of adrenals, whereas the remainder have enlarged adrenals with preserved triangular glandular contour.⁶⁶ Caseous necrosis accounts for the characteristic rim (\pm septal) enhancement on CT, and central isointense to hypointense signal on T2W images with peripheral enhancement on MRI (**Fig. 25**). In chronic stages, glandular atrophy with calcification ensues.⁶⁷ Differential diagnosis of adrenal TB includes other disease entities with bilateral adrenal involvement such as metastases, lymphoma and histoplasmosis.⁶⁸ T2 hypointensity and mild or peripheral contrast enhancement with preserved gland shape may be seen in both TB and histoplasmosis; round mass-like enlargement is more in favor of lymphoma. Metastases usually appear as discrete heterogeneously enhancing T2 hyperintense nodules.

Assessment of Response to Antitubercular Treatment

Standard treatment of abdominal TB requires a 6- to 12-month course of ATT. Even when the diagnosis is not reliably established, patients with a high suspicion of abdominal TB are given a therapeutic trial of ATT, following which clinical (based on symptomatology and inflammatory markers such as erythrocyte sedimentation rate, C-reactive protein) and endoscopic responses are assessed periodically. Although patients with TB and Crohn's disease may both show clinical response at 3 months, this response is not sustained in Crohn's disease. Also, mucosal healing on endoscopy would occur after therapeutic ATT trial in nearly all patients with

intestinal TB but in less than 5% of patients with Crohn's disease.^{4,69} It has been shown that patients eventually diagnosed to have Crohn's disease but given ATT trial earlier due to diagnostic dilemma had a higher chance of developing stricturing disease and higher surgical rates; hence, the clinical and mucosal response to ATT trial should be assessed at 8 to 12 weeks in such settings when there is diagnostic confusion.⁵

Imaging by CT enterography or ultrasound can also be used to assess response to ATT. In a retrospective study, Ma et al classified response to ATT on these modalities as a "good response" if the lesion area decreased by over 50% or if the lesion area decreased by less than 50% accompanied by reduced bowel wall thickening and enhancement. They found excellent agreement between ultrasound and colonoscopy (κ -value 1) and good agreement between CT enterography and colonoscopy (κ -value 0.643) in assessing response to ATT.⁷⁰

Bowel wall thickening, abdominal lymphadenopathy, and ascites usually regress with ATT. Calcification in nodes and visceral granulomas is also a sign of healing. Fibrosis can lead to contracted and pulled-up cecum (**Fig. 26**) and stricture formation. However, bowel wall thickening, homogeneous wall enhancement, and lymphadenopathy can persist post-ATT, and these do not necessarily indicate active disease.⁷¹ In fact, tubercular strictures, especially colonic ones, are particularly resilient to ATT, with response seen in only a quarter to two-thirds of patients.^{72,73} Therefore, rather than relying on imaging findings alone, the decision to continue or stop ATT should be made in conjunction with clinical and endoscopic evidence of disease activity.

More recently, perfusion CT and MRI with diffusion-weighted imaging have been applied in assessment of response to ATT. In one study, it was found that there is a reduction in intestinal mean blood flow, blood volume, and maximum peak intensity along with an increase in mean transit time and time to peak on perfusion CT following a course of ATT, likely due to reduced bowel inflammation.⁷⁴ Another study found that patients with good response to ATT showed an increase in mean apparent diffusion coefficient (ADC) values from $1.1 \pm 0.37 \times 10^{-3}$ to $2.1 \pm 0.64 \times 10^{-3}$ mm^2/s after completion of treatment.⁷⁵ Thus, CT perfusion



Fig. 26 Response to therapy. Pretreatment coronal (A) and axial (B) contrast-enhanced computed tomography (CECT) showing marked cecal thickening with mural stratification (arrows). Post antitubercular treatment axial (C) and coronal (D) CECT showing contracted cecum with reduced wall thickness and intramural fatty infiltration (arrows).

indices and ADC values could serve as objective measures of response to ATT.

Functional imaging with ^{18}F -fluorodeoxyglucose positron emission tomography/CT can be useful in monitoring the response to ATT. While anatomical imaging may reveal persistent findings of abdominal TB, the maximal standardized uptake value (SUVmax) of initially FDG-avid tubercular lesions reduces to undetectable levels on follow-up in case of good therapeutic response and may indicate treatment endpoint. On the other hand, persistent uptake suggests disease is still active and may require prolongation or modification of ATT.⁷⁶

Role of Tissue Diagnosis

Confirmation of diagnosis of abdominal TB requires histological demonstration of caseous granulomas and/or microbiological demonstration of tubercle bacilli. In case of peritoneal TB, diagnostic paracentesis is usually performed. There is low sensitivity for the detection of acid-fast bacilli (AFB) in ascitic fluid smears (3%). However, microbiological culture or GeneXpert test (sensitivity 35 and 30%, respectively) can also be performed on ascitic fluid.⁷⁷ In case of low-volume ascites, ultrasound can guide the sampling of the fluid.

For mesenteric lymph nodal or hepatosplenic TB, percutaneous ultrasound-guided sampling can be performed (sensitivity 78.6 and 87.5%, respectively).⁷⁸ CT guidance may be required for sampling of retroperitoneal nodes, whereas endoscopic-ultrasound guided sampling is preferred for locations that are difficult to access percutaneously (pancreaticoduodenal region). For gastrointestinal TB, endoscopic sampling is

done; although imaging can guide the gastroenterologist regarding the sites of the affected bowel. Here also, AFB staining has low sensitivity (<5%) compared with mycobacterial culture (40%) and GeneXpert (23%).⁷⁷

Conclusion

In conclusion, abdominal TB has a myriad of imaging manifestations, and radiologists need to be cognizant of them. Although no single radiological feature can be considered pathognomonic, the constellation of findings in different abdominal organs, as well as concomitant features of pulmonary TB or other sites of extrapulmonary TB, can help in arriving at the diagnosis. Imaging can also guide in selecting the best route for sampling the affected organ for histological or microbiological confirmation of the diagnosis. Ileocolonoscopy plays an important role in evaluating the mucosal abnormalities and in documenting healing after ATT.

Funding

None.

Conflict of Interest

None declared.

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