

Abdominal tuberculosis

Authors:

Vineet Ahuja, MD, DM
Elizabeth A Talbot, MD

Section Editors:

Sanjiv Chopra, MD, MACP, FRCP (London)
John Bernardo, MD

Deputy Editor:

Elinor L Baron, MD, DTMH

INTRODUCTION

Abdominal tuberculosis (TB) may occur with or without concomitant pulmonary TB; it may include involvement of the entire gastrointestinal tract. Abdominal TB comprises around 10 percent of all cases of extrapulmonary TB worldwide [1].

Clinical features, diagnosis, and treatment of pulmonary TB are discussed separately. (See "[Pulmonary tuberculosis disease in adults: Clinical manifestations and complications](#)" and "[Diagnosis of pulmonary tuberculosis disease in adults](#)" and "[Treatment of drug-susceptible pulmonary tuberculosis in nonpregnant adults without HIV infection](#)".)

RISK FACTORS

Risk factors for the acquisition and development of abdominal tuberculosis (TB) are similar to those for pulmonary and extrapulmonary TB. (See "[Epidemiology of tuberculosis](#)".)

In addition, unique risk factors for abdominal TB include underlying cirrhosis and use of continuous ambulatory peritoneal dialysis [2-7].

DISEASE FORMS

Abdominal tuberculosis (TB) may involve any gastrointestinal (GI) tract site from the mouth to the anus, as well as the peritoneum, abdominal and GI-associated lymph nodes, hepatobiliary tree, pancreas, and spleen. The most common sites of abdominal TB disease involve the peritoneum, intestine, and/or regional lymph nodes [1,8-12].

Abdominal TB can occur when *Mycobacterium tuberculosis* spreads from the lungs to the GI tract via hematogenous or lymphatic spread, or via contiguous spread from other infected adjacent organs (such as retrograde spread from the fallopian tubes). Abdominal TB can also occur following ingestion of *M. bovis* (a member of the *M. tuberculosis* complex), most commonly in contaminated meat or unpasteurized dairy products [13]. (See "[Mycobacterium bovis](#)".)

Peritoneal TB

— Among the forms of extrapulmonary TB reported to the Centers for Disease Control and Prevention in the United States between 2010 and 2023, peritoneal TB comprised 5.3 to 7.4 percent of cases [14,15].

Peritoneal TB develops through hematogenous or contiguous spread. Spread to the peritoneal cavity may occur transmurally from the small intestine or by ascending from tuberculous salpingitis [16].

Clinical manifestations

- **Signs and symptoms** — Typically, symptoms of peritoneal TB have been present for weeks or months before the diagnosis is established [17-19]. Clinical manifestations include ascites (93 percent), abdominal pain (73 percent), and fever (58 percent) [7].

Ascites in a patient with epidemiologic risk for TB in the absence of chronic liver disease should raise clinical suspicion for TB peritonitis [9]. Ascites develops secondary to exudation of proteinaceous fluid from the tubercles studding the visceral and parietal peritoneum. Ascitic fluid findings are described below. (See '[Ascites fluid analysis](#)' below.)

Approximately 10 percent of patients with peritoneal TB do not present with ascites; in these cases, the presentation has been characterized as a "doughy" abdomen, which represents a fibro-adhesive form of the disease [20,21].

- **Peritonitis associated with peritoneal dialysis (PD)** — In patients with kidney failure on PD, clinical manifestations of TB peritonitis typically develop within the first year of beginning PD and are usually indistinguishable from bacterial peritonitis [22]. (See "[Clinical manifestations and diagnosis of peritonitis in peritoneal dialysis](#)".)

Radiographic findings

— In the setting of peritoneal TB, radiographic imaging commonly demonstrates ascites, lymphadenopathy, thickening of the mesentery and omentum, and thickening of the peritoneum [23].

Laparoscopic findings

— Laparoscopic findings in the setting of tuberculous peritonitis include [13,21]:

- Thickened peritoneum with yellowish-white lesions, with or without adhesions
- Thickened peritoneum, with or without adhesions
- Fibro-adhesive pattern

Other findings include enlarged lymph nodes, "violin-string" fibrinous strands, and omental thickening.

Hepatic TB

Disease forms and radiographic findings

- **Hepatic TB** — There are two main presentations of hepatic TB disease:
 - **Miliary hepatic TB** — Liver involvement occurs in the majority of patients with miliary TB [24]; in this form, TB reaches the liver via the hepatic artery. Isolated hepatic TB occurs in a minority of patients with hepatic involvement; in this form, TB reaches the liver from the intestinal tract via the portal vein or GI lymphatics.

Radiographic findings include multiple low-density micronodules dispersed throughout the liver; imaging may also demonstrate hepatomegaly without intrahepatic lesions or abdominal lymphadenopathy with peripheral lymph node enhancement and/or calcifications [25,26].

- **Isolated hepatic TB** — Isolated hepatic TB can present in the form of hepatic abscess, as an intrahepatic mass, or as granulomatous hepatitis with nodules measuring 1 to 4 cm in size. TB is a common cause of granulomatous hepatitis in endemic areas.

Radiographic findings include a large solitary nodule or two to three low-density nodules with calcification and peripheral enhancement.

- **Biliary TB** — Isolated biliary TB is uncommon; manifestations include tubercular biliary stricture, obstruction of the biliary tract by enlarged lymph nodes compressing the biliary system, or, rarely, gallbladder involvement (which is usually identified as an incidental histologic finding in cholecystectomy pathology specimens) [27,28].

Clinical manifestations

— In a 2015 systematic review including 618 patients with hepatic TB, the most common signs and symptoms were hepatomegaly (median 80 percent, range 10 to 100 percent), fever (median 67 percent, range 30 to 100 percent), respiratory symptoms (median 66 percent, range 32 to 78 percent), abdominal pain (median 59.5 percent, range 40 to 83 percent), and weight loss (median 57.5 percent, range 20 to 100 percent) [25].

Liver function test results

— The pattern of abnormal liver function tests may reflect the site of disease. Elevated transaminases suggest involvement of the liver parenchyma, while elevated alkaline phosphatase and gamma-glutamyl transferase suggest involvement of the porta or biliary ducts. Mild hyperbilirubinemia has been reported in both miliary and local hepatic TB [25]. Marked hyperbilirubinemia and the presence of jaundice usually suggest biliary involvement.

Intestinal TB

— Intestinal TB can involve any area of the bowel. The most common involved site is the ileocecal area (25 to 90 percent across various series [1]). The affinity of *M. tuberculosis* for this site may reflect the relative stasis and abundance of lymphoid tissue in this region. Other locations of involvement, in order of descending frequency, are the ascending colon, jejunum, appendix, duodenum, stomach, esophagus, sigmoid, colon, and rectum [1].

Clinical manifestations

— Signs and symptoms of intestinal TB may include classic TB symptoms such as fever, fatigue, weight loss, and night sweats [29-32]. Abdominal pain is the most common manifestation of intestinal TB; it may occur because of a stricture of the intestinal lumen, mesenteric inflammation, and/or peritoneal involvement. Constipation occurs in nearly half of patients with intestinal TB, while diarrhea occurs in a minority (11 to 37 percent) [33,34]. Fistula and intestinal stricture may occur. Bowel obstruction may occur due to progressive stricture or adhesions [35-37].

Other complications of intestinal TB include perforation, intestinal fistulae, and GI bleeding [38]. Lower GI bleeding occurs in 5 to 15 percent of patients with intestinal TB; the bleeding is rarely massive. Other symptoms reflect local effects, such as when the organisms penetrate the mucosa and localize in the submucosal lymphoid tissue, which can initiate an inflammatory reaction with potential for lymphangitis, endarteritis, granuloma formation, caseation necrosis, mucosal ulceration, and scarring.

Symptoms otherwise depend on the source, site, and extent of disease. For example, patients with gastroduodenal involvement can also present with early satiety, postprandial fullness, nausea, and vomiting. Malabsorption can occur in the context of small intestinal TB due to bacterial overgrowth in a stagnant loop, bile salt deconjugation, and diminished absorptive surface due to ulceration. Rectal TB typically presents as hematochezia and constipation followed by constitutional symptoms. Anal TB may present as pilonidal sinus, anal ulceration with inguinal adenopathy, recurrent perianal growth, anal fissure, anal fistulae, or anal stricture [39]. If there is contiguous spread, there may be ascites, lymph node enlargement, and tubo-ovarian symptoms.

Radiographic findings

— Imaging commonly demonstrates concentric mural thickening in the ileocecal region, with or without proximal intestinal dilatation ([image 1](#) and [image 2](#) and [image 3](#) and [image 4](#)) [1]. Occasionally, asymmetric thickening of the medial cecal wall is seen. Lymphadenopathy with hypodense centers (representing caseous liquefaction) may be present in the adjacent mesentery.

Endoscopic findings

— Forms of intestinal lesions include ulcerative and hypertrophic; these forms can coexist [1,8,33,40].

- Ulcerative lesions have been described, especially in malnourished individuals. This presentation is characterized by single or multiple transverse ulcers that form strictures during the healing process and may perforate, bleed, or form fistulas [40]. Ulcers due to TB tend to be circumferential ([picture 1](#)) and are usually surrounded by inflamed mucosa.
- Hypertrophic lesions have been described in relatively well-nourished individuals; they are usually found in the colon and ileocecum. This presentation commonly affects the ileocecal region and causes obstruction or presents as a mass. Ileocecal valve involvement usually includes both sides of the valve, leading to incompetence. A patulous valve with surrounding heaped-up folds or a destroyed valve with a fish mouth opening may be observed ([picture 2](#)).

Upper endoscopic findings of intestinal TB may include ulcers, strictures, nodules, pseudopolyps, and/or a deformed ileocecal valve ([image 5](#) and [image 6](#)) [41].

Pancreatic TB

— Pancreatic TB is uncommon [42,43]. It often presents with abdominal pain, constitutional symptoms, and sometimes jaundice. In a systematic review of 116 reports of 166 patients, it most commonly presented as a mass lesion often confused with malignancy, with concurrent peripancreatic lymph node involvement in 47 percent of cases [43].

Laboratory findings

— Routine laboratory tests often demonstrate mild anemia and increased markers of inflammation (eg, erythrocyte sedimentation rate or C-reactive protein); the white blood count is usually normal. Liver function tests results are discussed above. (See '[Hepatic TB](#)' above.)

DIAGNOSIS

Clinical approach

- **Clinical suspicion** — The diagnosis of abdominal tuberculosis (TB) should be suspected in patients with relevant clinical manifestations and relevant epidemiologic risk for TB; the diagnosis can be challenging and often is delayed due to its nonspecific presentation.
- **Establishing a definitive diagnosis** — The diagnosis of abdominal TB may be established by positive mycobacterial culture and/or nucleic acid amplification test (NAAT) of a clinical specimen (ascites or biopsy specimen of an involved site).
- **Diagnostic evaluation**
 - **Paracentesis** — In the setting of ascites, a diagnostic ascites fluid analysis should be pursued. (See '[Ascites fluid analysis](#)' below.)
 - **Biopsy** — In the absence of ascites or if ascites analysis is nondiagnostic, biopsy should be pursued. The choice of biopsy site should be made based on the anatomic involvement demonstrated on radiographic imaging.
 - **Obtaining specimens** — The approach to obtaining biopsy specimens should be tailored to individual circumstances, including the dominant site of involvement and associated risks and benefits [20,44-47].

As an example, for patients with nondiagnostic ascites fluid, a peritoneal biopsy may be performed via laparoscopy. For patients with isolated hepatic involvement, a liver biopsy may be obtained via interventional radiology. For patients with intestinal involvement, endoscopic biopsies may be obtained; deep biopsies should be taken from ulcers and their margins. For patients with peripancreatic and celiac lymph nodes, endoscopic ultrasound can be used for fine-needle aspiration biopsies.
 - **Evaluating specimens** — Biopsy specimens should be sent for microbiologic studies (including culture and drug susceptibility testing, as well as NAAT where available) and histopathologic examination (including acid-fast bacilli [AFB] staining). Thus far, molecular tests are not US Food and Drug Administration (FDA)-approved for this context in the United States, but are available in several referral laboratories (eg, Mayo Clinic, University of Washington) and some public health laboratories.

Interpretation of biopsy results is discussed below. (See '[Biopsy interpretation](#)' below.)

- **Presumptive diagnosis** — It may not be possible to establish a definitive diagnosis of abdominal TB. Patients with probable abdominal TB include those with epidemiologic risk, positive TB infection screening, suggestive ascitic fluid analysis, and/or consistent findings in histology, with exclusion of other non-TB diagnoses. (See '[Differential diagnosis](#)' below.)
- **Evaluation for concomitant pulmonary TB** — Approximately 15 to 25 percent of patients with abdominal TB have concomitant pulmonary TB [8,10]. Therefore, patients with concomitant clinical or radiographic findings suggestive of pulmonary involvement should also undergo diagnostic evaluation for pulmonary TB; this

is discussed separately. (See "Pulmonary tuberculosis disease in adults: Clinical manifestations and complications" and "Diagnosis of pulmonary tuberculosis disease in adults".)

- **Evaluation for human immunodeficiency virus (HIV) infection** – Patients with confirmed or presumptive TB disease warrant testing for HIV infection. (See "Acute and early HIV infection: Clinical manifestations and diagnosis".)

Diagnostic tools

Radiographic imaging

— Patients with suspected abdominal TB should undergo radiographic imaging.

- **Computed tomography (CT)** – CT enterography protocol (eg, with contrast) is preferred, to facilitate cross-sectional evaluation for the involvement of the intestine and other organs, as well as the presence of ascites, peritoneal involvement, and lymphadenopathy [48-53]. In children, magnetic resonance enterography may be preferable to reduce the risk of radiation.

An alternative approach consists of abdominal CT (or magnetic resonance imaging [MRI]); if abnormalities (such as bowel wall thickening) are identified, follow-up endoscopy can be performed to facilitate biopsy of specific lesions.

Radiographic findings of peritoneal, intestinal, and hepatic TB are described above. (See 'Disease forms' above.)

- **Ultrasonography** – Ultrasound is used in many low- and middle-income settings, particularly as point-of-care ultrasound is increasingly available. Ultrasound may be useful for detecting lymphadenopathy, ascites, peritoneal thickening, omental thickening, and bowel wall thickening [1,8]. However, in one systematic review including 11 studies evaluating ultrasound for the diagnosis of abdominal TB in patients with HIV infection, the sensitivity and specificity were 63 and 68 percent, respectively [54]. The authors note these estimates are based on data that are limited, varied, and low-certainty, and these low sensitivity and specificity means ultrasound should be used in combination with other diagnostic strategies.
- **Other radiographic modalities for abdominal TB**
 - Small bowel follow-through or **barium** enema may demonstrate mucosal ulcerations and strictures, a deformed cecum, and/or an incompetent ileocecal valve (image 7) [8].
 - Plain radiography may demonstrate air fluid levels with dilated small bowel loops (suggestive of intestinal obstruction) or hepatic calcifications [25].
- **Chest imaging for all patients** – Chest imaging should be performed in all patients with suspected abdominal TB, regardless of respiratory symptoms [55]. (See "Diagnosis of pulmonary tuberculosis disease in adults", section on 'Radiographic imaging'.)

Ascites fluid analysis

— Ascitic fluid should be sent for routine tests (cell count and differential, albumin and protein concentration, Gram stain) as well as adenosine deaminase (ADA) level, AFB smear, mycobacterial culture, and, if available, NAAT for *M. tuberculosis* (MTB) [56].

- **Color** – Ascites fluid in the setting of TB peritonitis is typically straw-colored.
- **Cell count** – The typical leukocyte count is 150 to 4000 cells/mm³, with a relative lymphocytic predominance [8,33,57].

However, patients on peritoneal dialysis with tuberculous peritonitis may have a neutrophilic response [58,59]. (See "Evaluation of adults with ascites".)

- **Protein** – The protein content of ascitic fluid is usually >3 g/dL in the setting of tuberculous peritonitis [20,60].

Patients typically have serum-ascites albumin gradient (SAAG) <1.1 g/dL (in the absence of cirrhosis); patients with underlying cirrhosis have SAAG ≥1.1 g/dL [61-63]. (See "Evaluation of adults with ascites".)

- **ADA** – Measurement of ascites fluid ADA level may be a useful tool for evaluation of patients with suspected TB peritonitis; it is most reliable in the absence of cirrhosis [64]. An elevated ascites ADA level supports a diagnosis of TB in noncirrhotic patients but does not establish it with certainty.

Ascites ADA concentration >36 international units/L, with ratio of ascites to serum ADA concentration >0.98 international units/L, suggests TB, but can also be observed in the setting of malignancy [10].

One meta-analysis of 24 studies including 3044 patients found that the pooled sensitivity and specificity of ADA levels were 93 and 95 percent, respectively, with cut-off values ranging from 20 and 40 international units/L (most cut-off values were between 36 and 40 international units/L) [64]. Among patients with cirrhosis, the ADA levels in the ascitic fluid of peritoneal TB patients were significantly higher than the nonperitoneal TB patients (64 ± 21.5 international units/L versus 19 ± 11.8 international units/L, p < 0.005), such that the sensitivity and specificity were 93 percent (95% CI 87-98 percent) and 94 percent (95% CI 88-96 percent), respectively [65]. In such cases, it may be a helpful, supportive diagnostic tool if lower thresholds are used (21 to 30 international units/L) [65].

There is no role for routine use of ascites fluid interferon-gamma concentration for diagnosis of tuberculous peritonitis [66,67] and has not been approved by the FDA for this purpose.

Microbiologic evaluation

— The diagnosis of abdominal TB is definitively established by positive mycobacterial culture and/or NAAT of a fluid or biopsy specimen [25,68,69].

- **AFB smear and culture** – The sensitivity of AFB smear and mycobacterial culture of ascites fluid is extremely low (less than 2 percent and less than 20 percent, respectively) [8,33,70]. The yield of mycobacterial culture may be increased with increased volume of ascitic fluid (concentrated by centrifugation).
- **NAAT** – A positive NAAT result establishes the diagnosis of TB, given excellent specificity. However, the sensitivity of NAAT for extrapulmonary specimens is modest; a negative result does not exclude the diagnosis [71]. Thus far, molecular tests are not FDA-approved for this context in the United States, but are available in reference and public health laboratories.

In a meta-analysis evaluating the utility of ascitic fluid NAAT for diagnosis of tuberculous peritonitis, including 13 studies and 580 patients (among whom 16.2 percent had TB disease), the aggregate sensitivity of Xpert MTB/resistance to rifampicin (RIF) or Xpert Ultra was 59.1 percent, with high specificity (97.6 percent). In individual studies, Xpert MTB/RIF sensitivity ranged from 33 to 100 percent, with specificity from 90 to 100 percent [56].
- **Role of stool polymerase chain reaction (PCR)** – For patients with intestinal lesions or for patients with pulmonary TB who cough and swallow sputum containing MTB deoxyribonucleic acid (DNA), stool PCR may be helpful [72,73]. There is increasing recognition of the importance of stool-based DNA tests for pulmonary TB, especially in children [72]. (See "Tuberculosis disease (active tuberculosis) in children: Epidemiology, clinical manifestations, and diagnosis", section on 'Obtaining laboratory specimens'.)
- **Role of cell-free DNA** – Small studies suggest detection of circulating cell-free MTB DNA may be helpful for the diagnosis of abdominal TB [74]. Further study is needed.

Biopsy interpretation

— The approach to obtaining biopsy specimens is described above. (See 'Clinical approach' above.)

- **Microbiologic studies** – The sensitivity of AFB smear and mycobacterial culture for biopsy specimens is low (less than 50 percent) [25,75].

If available, PCR is more sensitive and specific for diagnosis of TB, and often PCR results are available sooner [76,77]. In a meta-analysis of intestinal biopsies evaluated with Xpert MTB/RIF (compared with a composite reference standard of clinical manifestations, histopathology, biochemical test results, smears, culture, other NAATs, and treatment response to anti-TB drugs), the pooled sensitivity and specificity were 48 and 99 percent, respectively [78].

The diagnostic utility of Xpert MTB/RIF or Xpert Ultra assays may be limited. In one study of mucosal biopsies obtained from 37 patients with intestinal TB (culture confirmed) and 62 patients with other conditions, the sensitivity, specificity, positive predictive value, and negative predictive value of Xpert MTB/RIF were 8.1, 100, 100, and 64.2 percent, respectively [69].

The utility of noncommercial PCR varies depending on the tissue type; sensitivity and specificity are high for peritoneal fluid, hepatic tissue, and pancreatic tissue.

- **Histopathology** – Presence of caseating (necrotizing) granulomas, with or without demonstration of AFB, is suggestive of TB but is not pathognomonic; this finding may support a diagnosis of TB in the setting of other relevant clinical and epidemiologic factors [25]. (See 'Differential diagnosis' below.)

Some features that help differentiate TB enteritis from other etiologies include confluent granulomas, granulomas >400 micrometers in diameter, more than five granulomas in biopsies from one segment, and granulomas located in the submucosa or granulation tissue (table 1) [1,79].

Additional tools

— A positive **tuberculin skin test** or blood-based interferon-gamma release assay may be observed in patients with abdominal TB, but is of limited value because these assays do not differentiate between TB infection (latent TB) and TB disease (active TB) [33,80]. (See "Tuberculosis infection (latent tuberculosis) in adults: Approach to diagnosis (screening)".)

DIFFERENTIAL DIAGNOSIS

— The differential diagnosis of abdominal (TB) includes:

- **Crohn disease (CD)** – Distinguishing between intestinal TB and CD is important because the use of immunosuppressive drugs for a misdiagnosis of CD may be associated with clinical deterioration in patients with TB [81,82]. A combination of clinical, radiographic, endoscopic, laboratory, and biopsy findings are used to distinguish between these diseases, as summarized in the table (table 1) [46,53,83-93]. (See "Clinical manifestations, diagnosis, and prognosis of Crohn disease in adults".)

In a meta-analysis including 38 studies (including 2117 patients with CD and 1589 patients with intestinal TB), findings favoring intestinal TB included clinical findings of fever, night sweats, lung involvement, and ascites; CT enterography demonstrating short segmental involvement; endoscopic findings of transverse ulcers, patulous ileocecal valve, and cecal involvement; pathologic findings of confluent or submucosal granulomas, lymphocyte cuffing, and ulcers lined by histiocytes, and a positive interferon-gamma release assay [93].

If definitive differentiation between intestinal TB and CD is not possible, a therapeutic trial with antituberculous therapy may be useful to help distinguish between the conditions [94]. However, patients with CD initially treated with antituberculous therapy experience a less favorable long-term disease course, characterized by a higher incidence of stricture formation and surgery. Hence, it is advisable to perform a repeat colonoscopy within 8 to 12 weeks of initiation of antituberculous therapy to evaluate for interval improvement [95,96].

- **End-stage liver disease with ascites and spontaneous bacterial peritonitis** – The presentation of spontaneous bacterial peritonitis is more acute than tuberculous peritonitis; the diagnosis may be established by examination of ascitic fluid (neutrophil count >250 or positive Gram stain or culture). (See

"Spontaneous bacterial peritonitis in adults: Pathogenesis, clinical features, and diagnosis".)

- **Malignancy** – Malignancy may be associated with ascites, peritoneal involvement, and/or liver lesions [97]; it may be distinguished from abdominal TB via abdominal imaging, evaluation of ascites fluid for cytology, and biopsy for culture and histopathology. Malignant ascites is frequently a bloody exudate [9]. Serum cancer antigen 125 concentrations can be elevated in the setting of ascites due to peritoneal TB, ovarian cancer, and other entities, so this test cannot be used to distinguish between these diseases [98]. (See "Malignancy-related ascites" and "Adnexal mass: Role of serum biomarkers in diagnosing epithelial carcinoma of the ovary, fallopian tube, or peritoneum", section on 'Cancer antigen 125'.)
- **Lymphoma** – Patients with lymphoma may present with abdominal pain, fever, night sweats, and weight loss (similar to TB), and demonstrate abdominal lymphadenopathy on radiography. Lymph node biopsy for culture and histopathology should be pursued to distinguish between lymphoma and TB. Of note, patients with lymphoma may have elevated ascites adenosine deaminase concentration [99]. (See "Clinical presentation and initial evaluation of non-Hodgkin lymphoma".)

The presence of granulomata on histopathology may reflect conditions other than TB:

- The differential diagnosis of caseating (necrotizing) granulomas includes fungal infection such as histoplasmosis and *Cryptococcus*. (See "Diagnosis and treatment of pulmonary histoplasmosis" and "*Cryptococcus neoformans*: Pulmonary and other infections outside the central nervous system".)
- The differential diagnosis of noncaseating granulomas includes CD, sarcoidosis, rheumatoid pleuritis, nontuberculous mycobacteria, and berylliosis. (See "Clinical manifestations and diagnosis of sarcoidosis" and "Overview of pleuropulmonary diseases associated with rheumatoid arthritis" and "Chronic beryllium disease (berylliosis)".)

MANAGEMENT

Clinical approach

— Patients with confirmed or presumptive abdominal tuberculosis (TB) should be treated with antituberculous therapy. In addition, surgery may be warranted for patients with complications such as perforation, abscess, fistula, bleeding, and/or high-grade obstruction.

Antituberculous therapy

— In general, the approach to antituberculous therapy for abdominal TB is the same as that for pulmonary TB [100-102]. (See "Treatment of drug-susceptible pulmonary tuberculosis in nonpregnant adults without HIV infection" and "Treatment of drug-susceptible pulmonary tuberculosis in nonpregnant adults with HIV infection: Initiation of therapy" and "Treatment of drug-resistant pulmonary tuberculosis in adults".)

For patients with intestinal involvement (who may have drug malabsorption), therapeutic drug monitoring is a useful adjunct to ensure appropriate drug levels; intravenous therapy may be warranted in some cases. In addition, drug-induced liver injury may be more likely in the setting of hepatic TB; in such cases, use of nonhepatotoxic regimens can be used, such as BPAL (bedaquiline, pretomanid, and linezolid). (See "Treatment of drug-resistant pulmonary tuberculosis in adults", section on 'BPAL: Bedaquiline, pretomanid, linezolid'.)

- **Time frame for clinical improvement**

- **Signs and symptoms** – Fever usually resolves within one week of commencing antituberculous therapy. Patients with ascites have improvement within a few weeks of initiating treatment in 90 percent of cases [103]. Patients with tuberculous enteritis generally demonstrate clinical improvement within two to four weeks on empiric therapy [102,104]. In one study, colonoscopic follow-up after two to three months of antituberculous therapy demonstrated complete healing of active ulcers and erosions [105].
- **Stricture formation** – Healing associated with antituberculous therapy can result in worsening of strictures, due to formation of scar tissue. Patients who develop bowel obstruction are usually unable to ingest medications orally; surgical intervention may be warranted in such cases. In one series including 106 patients with stricturing intestinal TB, resolution of stricture following antituberculous therapy was observed in only 25 percent of patients [106]. Stricture resolution occurred most frequently in the distal ileal and ileocecal region, followed by proximal small intestine and colon (36, 20, and 5 percent, respectively).
- **Absence of clinical improvement** – In the absence of clinical response to antituberculous therapy within four to eight weeks, repeat evaluation may be warranted for alternative diagnoses such as Crohn disease, lymphoma, or malignancy [80]. (See 'Differential diagnosis' above.)

Surgery and other interventions

— Surgery may be warranted for patients with complications such as perforation, abscess, fistula, bleeding, and/or high-grade obstruction [34,68,80]. Patients with biliary obstruction due to stricture may require biliary reconstruction.

Intestinal obstruction is the most common complication; patients with multiple and/or long strictures are less likely to respond to antituberculous therapy [37]. In addition, obstruction may be exacerbated during antituberculous therapy due to healing by cicatrization [1]. (See "Management of small bowel obstruction in adults".)

The surgical resection should be as conservative as possible; in some cases, multiple strictures of the small bowel may be amenable to strictureplasty to avoid major resection [68]. Bypass surgery for obstructing lesions should be avoided because of complications related to blind loop syndrome.

An alternative approach to management of ileal stricture is colonoscopic balloon dilatation; this technique may be useful for management of symptomatic short ileal strictures [107].

PROGNOSIS

Mortality ranges from 1.4 to 20 percent in various series [108,109]. Advanced age, delay in initiating therapy, and underlying cirrhosis have been associated with higher mortality rates [6,7,57,110].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "[Society guideline links: Diagnosis and treatment of tuberculosis](#)" and "[Society guideline links: Portal hypertension and ascites](#)".)

SUMMARY AND RECOMMENDATIONS

- **Forms of disease** – Abdominal tuberculosis (TB) can present with involvement of any of the following sites: peritoneum, stomach, intestinal tract, hepatobiliary tree, pancreas, perianal area, spleen, and gastrointestinal-associated lymph nodes. The most common forms of disease include involvement of the peritoneum, intestine, and/or lymph nodes. Clinical manifestations of abdominal TB depend on the form of disease and may include fever, weight loss, abdominal pain and/or distension, ascites, hepatomegaly, diarrhea, abdominal mass, and abnormal liver function tests. (See '[Disease forms](#)' above.)
- **Diagnosis**
 - **Clinical approach** (see '[Clinical approach](#)' above):
 - The diagnosis of abdominal TB should be suspected in patients with relevant clinical manifestations as well as relevant epidemiologic factors.
 - The diagnosis of abdominal TB may be definitively established by demonstration of *Mycobacterium tuberculosis* in peritoneal fluid (in the setting of ascites) or a biopsy specimen of an involved site (such as peritoneum, intestine, or liver) or via mycobacterial culture and/or nucleic acid amplification test (NAAT).
 - **Diagnostic tools**
 - **Radiographic imaging** – Patients with suspected abdominal TB should undergo radiographic imaging; CT or magnetic resonance with enterography protocol is preferred if feasible, since it allows evaluation for involvement of the liver and other organs as well as for presence of ascites, peritoneal involvement, and lymphadenopathy. An alternative approach consists of abdominal CT (or MRI) followed by endoscopy with biopsy. (See '[Radiographic imaging](#)' above.)
 - **Role of paracentesis** – Patients with ascites should undergo paracentesis. The fluid should be sent for routine tests (cell count and differential, albumin and protein concentration, Gram stain) as well as adenosine deaminase (ADA) level, acid-fast bacilli smear, mycobacterial culture, and NAAT for *M. tuberculosis*. Patients with tuberculous peritonitis (in the absence of underlying cirrhosis) typically have lymphocytic ascites with serum-ascites albumin gradient <1.1 g/dL. An elevated ascites ADA level (>39 international units/L) is useful to support a diagnosis of TB in noncirrhotic patients but does not establish it with certainty. Patients with nondiagnostic ascites analysis should undergo peritoneal biopsy via laparoscopy. (See '[Ascites fluid analysis](#)' above.)
 - **Role of biopsy** – In the absence of ascites, biopsy should be pursued; the choice of biopsy site should be guided by the scope of anatomic involvement demonstrated on radiographic imaging. The approach to obtaining biopsy specimens should be tailored to individual circumstances, including the dominant site of involvement and associated risks and benefits. As examples, in the setting of intestinal involvement, endoscopic biopsies should be obtained; in the setting of isolated hepatic involvement, a liver biopsy should be pursued. (See '[Biopsy interpretation](#)' above.)
- **Management**
 - **Clinical approach** – Patients with abdominal TB should be treated with antituberculous therapy. In addition, surgery may be warranted for patients with complications such as perforation, abscess, fistula, bleeding, and/or high-grade obstruction. In general, the approach to antituberculous therapy for abdominal TB is the same as that for pulmonary TB. Healing in the setting of antituberculous therapy can result in worsening of strictures due to scar tissue formation; patients who develop obstruction may warrant surgical intervention. (See '[Clinical approach](#)' above.)
 - **Empiric treatment** – It may not be possible to establish a definitive diagnosis of abdominal TB. For situations in which there is a high index of suspicion based on clinical, epidemiologic, and diagnostic findings (such as elevated ascitic fluid ADA and/or consistent findings in histology, with nondiagnostic mycobacterial culture and NAAT results), an empiric trial of antituberculous therapy is reasonable. (See '[Clinical approach](#)' above.)

REFERENCES

1. Rath P, Gambhire P. Abdominal Tuberculosis. J Assoc Physicians India 2016; 64:38.

2. Kedia S, Mouli VP, Kamat N, et al. Risk of Tuberculosis in Patients With Inflammatory Bowel Disease on Infliximab or Adalimumab Is Dependent on the Local Disease Burden of Tuberculosis: A Systematic Review and Meta-Analysis. *Am J Gastroenterol* 2020; 115:340.
3. Mehta JB, Dutt A, Harvill L, Mathews KM. Epidemiology of extrapulmonary tuberculosis. A comparative analysis with pre-AIDS era. *Chest* 1991; 99:1134.
4. Braun MM, Byers RH, Heyward WL, et al. Acquired immunodeficiency syndrome and extrapulmonary tuberculosis in the United States. *Arch Intern Med* 1990; 150:1913.
5. Rieder HL, Snider DE Jr, Cauthen GM. Extrapulmonary tuberculosis in the United States. *Am Rev Respir Dis* 1990; 141:347.
6. Aguado JM, Pons F, Casafont F, et al. Tuberculous peritonitis: a study comparing cirrhotic and noncirrhotic patients. *J Clin Gastroenterol* 1990; 12:550.
7. Chow KM, Chow VC, Hung LC, et al. Tuberculous peritonitis-associated mortality is high among patients waiting for the results of mycobacterial cultures of ascitic fluid samples. *Clin Infect Dis* 2002; 35:409.
8. Debi U, Ravisankar V, Prasad KK, et al. Abdominal tuberculosis of the gastrointestinal tract: revisited. *World J Gastroenterol* 2014; 20:14831.
9. Vaid U, Kane GC. Tuberculous Peritonitis. *Microbiol Spectr* 2017; 5.
10. Sampath A, Mani S. Diagnostic evaluation and management of abdominal tuberculosis. *Indian J Tuberc* 2025; 72 Suppl 1:S7.
11. Eraksoy H. Gastrointestinal and Abdominal Tuberculosis. *Gastroenterol Clin North Am* 2021; 50:341.
12. Jha DK, Pathiyil MM, Sharma V. Evidence-based approach to diagnosis and management of abdominal tuberculosis. *Indian J Gastroenterol* 2023; 42:17.
13. Uzunkoy A, Harma M, Harma M. Diagnosis of abdominal tuberculosis: experience from 11 cases and review of the literature. *World J Gastroenterol* 2004; 10:3647.
14. Tuberculosis in the United States, 2023 Slide Set. Centers for Disease Control and Prevention. <https://www.cdc.gov/tb-surveillance-report-2023/resources/slide-set.html> (Accessed on April 30, 2025).
15. TB by Site of Disease: 2010–2023. Centers for Disease Control and Prevention. <https://www.cdc.gov/tb-surveillance-report-2023/tables/table-20.html> (Accessed on February 06, 2026).
16. Tzelios C, Neuhausser WM, Ryley D, et al. Female Genital Tuberculosis. *Open Forum Infect Dis* 2022; 9:ofac543.
17. Tanrikulu AC, Aldemir M, Gurkan F, et al. Clinical review of tuberculous peritonitis in 39 patients in Diyarbakir, Turkey. *J Gastroenterol Hepatol* 2005; 20:906.
18. Lisehora GB, Peters CC, Lee YT, Barcia PJ. Tuberculous peritonitis—do not miss it. *Dis Colon Rectum* 1996; 39:394.
19. Gitt S, Haddad F, Levenson S. Tuberculous peritonitis: an overlooked diagnosis. *Hosp Pract (Off Ed)* 1992; 27:224.
20. Manohar A, Simjee AE, Haffjee AA, Pettengell KE. Symptoms and investigative findings in 145 patients with tuberculous peritonitis diagnosed by peritoneoscopy and biopsy over a five year period. *Gut* 1990; 31:1130.
21. Bhargava DK, Shriniwas, Chopra P, et al. Peritoneal tuberculosis: laparoscopic patterns and its diagnostic accuracy. *Am J Gastroenterol* 1992; 87:109.
22. Steven M, Reboul P, Gerbal M, Moranne O. Early diagnosis of tuberculous peritonitis after starting peritoneal dialysis. *J Nephrol* 2025; 38:1115.
23. Guirat A, Koubaa M, Mzali R, et al. Peritoneal tuberculosis. *Clin Res Hepatol Gastroenterol* 2011; 35:60.
24. Evans RP, Mourad MM, Dvorkin L, Bramhall SR. Hepatic and Intra-abdominal Tuberculosis: 2016 Update. *Curr Infect Dis Rep* 2016; 18:45.
25. Hickey AJ, Gounder L, Moosa MY, Drain PK. A systematic review of hepatic tuberculosis with considerations in human immunodeficiency virus co-infection. *BMC Infect Dis* 2015; 15:209.
26. Yu RS, Zhang SZ, Wu JJ, Li RF. Imaging diagnosis of 12 patients with hepatic tuberculosis. *World J Gastroenterol* 2004; 10:1639.
27. Amarapurkar DN, Patel ND, Amarapurkar AD. Hepatobiliary tuberculosis in western India. *Indian J Pathol Microbiol* 2008; 51:175.
28. Xu XF, Yu RS, Qiu LL, et al. Gallbladder tuberculosis: CT findings with histopathologic correlation. *Korean J Radiol* 2011; 12:196.
29. Das P, Shukla HS. Clinical diagnosis of abdominal tuberculosis. *Br J Surg* 1976; 63:941.
30. Bhansali SK. Abdominal tuberculosis. Experiences with 300 cases. *Am J Gastroenterol* 1977; 67:324.
31. Tandon RK, Sarin SK, Bose SL, et al. A clinico-radiological reappraisal of intestinal tuberculosis—changing profile? *Gastroenterol Jpn* 1986; 21:17.
32. Singh V, Jain AK, Agrawal AK, et al. Clinicopathological profile of abdominal tuberculosis. *Br J Clin Pract* 1995; 49:22.
33. Marshall JB. Tuberculosis of the gastrointestinal tract and peritoneum. *Am J Gastroenterol* 1993; 88:989.
34. Horvath KD, Whelan RL. Intestinal tuberculosis: return of an old disease. *Am J Gastroenterol* 1998; 93:692.
35. Petrosyan M, Mason RJ. Tuberculous enteritis presenting as small-bowel obstruction. *Clin Gastroenterol Hepatol* 2006; 4:xxiii.
36. García-Díaz RA, Ruiz-Gómez JL, Rodríguez-Sanjuan JC, et al. Perforation of the colon caused by intestinal tuberculosis. *Dis Colon Rectum* 2006; 49:927; author reply 927.
37. Ha HK, Ko GY, Yu ES, et al. Intestinal tuberculosis with abdominal complications: radiologic and pathologic features. *Abdom Imaging* 1999; 24:32.
38. Ju J, Liu J, Dong W, et al. Uncommon ileal perforation due to intestinal tuberculosis: A case report and literature review. *Medicine (Baltimore)* 2025; 104:e41099.
39. Mathew S. Anal tuberculosis: report of a case and review of literature. *Int J Surg* 2008; 6:e36.
40. Figueroa D, Guzman N, Isache C. Tuberculous Enteritis: A Rare Complication of Miliary Tuberculosis. *Case Rep Infect Dis* 2016; 2016:6949834.
41. Alvares JF, Devarbhavi H, Makhija P, et al. Clinical, colonoscopic, and histological profile of colonic tuberculosis in a tertiary hospital. *Endoscopy* 2005; 37:351.
42. Sharma V, Rana SS, Kumar A, Bhasin DK. Pancreatic tuberculosis. *J Gastroenterol Hepatol* 2016; 31:310.
43. Panic N, Maetzel H, Bulajic M, et al. Pancreatic tuberculosis: A systematic review of symptoms, diagnosis and treatment. *United European Gastroenterol J* 2020; 8:396.
44. Misra SP, Misra V, Dwivedi M, Gupta SC. Colonic tuberculosis: clinical features, endoscopic appearance and management. *J Gastroenterol Hepatol* 1999; 14:723.
45. Kim KM, Lee A, Choi KY, et al. Intestinal tuberculosis: clinicopathologic analysis and diagnosis by endoscopic biopsy. *Am J Gastroenterol* 1998; 93:606.
46. Prabhughate A, Rathi PM. Histopathology as a Diagnosis Tool of Abdominal Tuberculosis: A Narrative Review of Evidence. *J Assoc Physicians India* 2025; 73:e25.
47. Kocaman O, Danalioglu A, İnce AT, et al. Diagnosis of tuberculous peritonitis using endoscopic ultrasound-guided fine-needle aspiration biopsy of the peritoneum. *Turk J Gastroenterol* 2013; 24:65.
48. Demirkazik FB, Akhan O, Ozmen MN, Akata D. US and CT findings in the diagnosis of tuberculous peritonitis. *Acta Radiol* 1996; 37:517.
49. Akhan O, Pringot J. Imaging of abdominal tuberculosis. *Eur Radiol* 2002; 12:312.
50. Vázquez Muñoz E, Gómez-Cerezo J, Atienza Saura M, Vázquez Rodríguez JJ. Computed tomography findings of peritoneal tuberculosis: systematic review of seven patients diagnosed in 6 years (1996-2001). *Clin Imaging* 2004; 28:340.
51. Balthazar EJ, Gordon R, Hulnick D. Ileocecal tuberculosis: CT and radiologic evaluation. *AJR Am J Roentgenol* 1990; 154:499.

52. Suri S, Gupta S, Suri R. Computed tomography in abdominal tuberculosis. *Br J Radiol* 1999; 72:92.
53. Almadi MA, Ghosh S, Aljebreen AM. Differentiating intestinal tuberculosis from Crohn's disease: a diagnostic challenge. *Am J Gastroenterol* 2009; 104:1003.
54. Van Hoving DJ, Griesel R, Meintjes G, et al. Abdominal ultrasound for diagnosing abdominal tuberculosis or disseminated tuberculosis with abdominal involvement in HIV-positive individuals. *Cochrane Database Syst Rev* 2019; 9:CD012777.
55. Kedia S, Sharma R, Vuyyuru SK, et al. Addition of computed tomography chest increases the diagnosis rate in patients with suspected intestinal tuberculosis. *Intest Res* 2022; 20:184.
56. Kohli M, Schiller I, Dendukuri N, et al. Xpert MTB/RIF Ultra and Xpert MTB/RIF assays for extrapulmonary tuberculosis and rifampicin resistance in adults. *Cochrane Database Syst Rev* 2021; 1:CD012768.
57. al-Quorain AA, Facharzt, Satti MB, et al. Abdominal tuberculosis in Saudi Arabia: a clinicopathological study of 65 cases. *Am J Gastroenterol* 1993; 88:75.
58. Quantrill SJ, Woodhead MA, Bell CE, et al. Peritoneal tuberculosis in patients receiving continuous ambulatory peritoneal dialysis. *Nephrol Dial Transplant* 2001; 16:1024.
59. Gupta N, Prakash KC. Asymptomatic tuberculous peritonitis in a CAPD patient. *Perit Dial Int* 2001; 21:416.
60. Kim NJ, Choo EJ, Kwak YG, et al. Tuberculous peritonitis in cirrhotic patients: comparison of spontaneous bacterial peritonitis caused by *Escherichia coli* with tuberculous peritonitis. *Scand J Infect Dis* 2009; 41:852.
61. Sanai FM, Bzeizi KI. Systematic review: tuberculous peritonitis—presenting features, diagnostic strategies and treatment. *Aliment Pharmacol Ther* 2005; 22:685.
62. Poyrazoglu OK, Timurkaan M, Yalniz M, et al. Clinical review of 23 patients with tuberculous peritonitis: presenting features and diagnosis. *J Dig Dis* 2008; 9:170.
63. Hillebrand DJ, Runyon BA, Yasmineh WG, Rynders GP. Ascitic fluid adenosine deaminase insensitivity in detecting tuberculous peritonitis in the United States. *Hepatology* 1996; 24:1408.
64. Zhou R, Qiu X, Ying J, et al. Diagnostic performance of adenosine deaminase for abdominal tuberculosis: A systematic review and meta-analysis. *Front Public Health* 2022; 10:938544.
65. Dahale AS, Puri AS, Sachdeva S, et al. Reappraisal of the Role of Ascitic Fluid Adenosine Deaminase for the Diagnosis of Peritoneal Tuberculosis in Cirrhosis. *Korean J Gastroenterol* 2021; 78:168.
66. Su SB, Qin SY, Guo XY, et al. Assessment by meta-analysis of interferon-gamma for the diagnosis of tuberculous peritonitis. *World J Gastroenterol* 2013; 19:1645.
67. Sharma SK, Tahir M, Mohan A, et al. Diagnostic accuracy of ascitic fluid IFN-gamma and adenosine deaminase assays in the diagnosis of tuberculous ascites. *J Interferon Cytokine Res* 2006; 26:484.
68. Aston NO. Abdominal tuberculosis. *World J Surg* 1997; 21:492.
69. Kumar S, Bopanna S, Kedia S, et al. Evaluation of Xpert MTB/RIF assay performance in the diagnosis of abdominal tuberculosis. *Intest Res* 2017; 15:187.
70. Chow KM, Chow VC, Szeto CC. Indication for peritoneal biopsy in tuberculous peritonitis. *Am J Surg* 2003; 185:567.
71. Sharma V, Soni H, Kumar-M P, et al. Diagnostic accuracy of the Xpert MTB/RIF assay for abdominal tuberculosis: a systematic review and meta-analysis. *Expert Rev Anti Infect Ther* 2021; 19:253.
72. WHO consolidated guidelines on tuberculosis: module 3: diagnosis: rapid diagnostics for tuberculosis detection, 3rd ed. World Health Organization. <https://www.who.int/publications/i/item/9789240089488> (Accessed on February 25, 2026).
73. Gaur M, Singh A, Sharma V, et al. Diagnostic performance of non-invasive, stool-based molecular assays in patients with paucibacillary tuberculosis. *Sci Rep* 2020; 10:7102.
74. Sharma P, Anthwal D, Kumari P, et al. Utility of circulating cell-free *Mycobacterium tuberculosis* DNA for the improved diagnosis of abdominal tuberculosis. *PLoS One* 2020; 15:e0238119.
75. Diaz ML, Herrera T, Lopez-Vidal Y, et al. Polymerase chain reaction for the detection of *Mycobacterium tuberculosis* DNA in tissue and assessment of its utility in the diagnosis of hepatic granulomas. *J Lab Clin Med* 1996; 127:359.
76. Anand BS, Schneider FE, El-Zaatari FA, et al. Diagnosis of intestinal tuberculosis by polymerase chain reaction on endoscopic biopsy specimens. *Am J Gastroenterol* 1994; 89:2248.
77. Donoghue HD, Holton J. Intestinal tuberculosis. *Curr Opin Infect Dis* 2009; 22:490.
78. Ding YL, Yan RC, Chen HK, Guo XG. A meta-analysis of the accuracy of Xpert MTB/RIF in diagnosing intestinal tuberculosis. *Lab Med* 2024; 55:238.
79. Pulimood AB, Peter S, Ramakrishna B, et al. Segmental colonoscopic biopsies in the differentiation of ileocolic tuberculosis from Crohn's disease. *J Gastroenterol Hepatol* 2005; 20:688.
80. Kapoor VK. Abdominal tuberculosis. *Postgrad Med J* 1998; 74:459.
81. Kedia S, Ahuja V. Intestinal tuberculosis or Crohn's disease: Illusion or delusion or allusion. *JGH Open* 2021; 5:177.
82. Pratap Mouli V, Kedia S, Sharma V, et al. Navigating the Differential Diagnosis of Intestinal Tuberculosis and Crohn's Disease. *Gastroenterology* 2025; 169:1139.
83. Kirsch R, Pentecost M, Hall Pde M, et al. Role of colonoscopic biopsy in distinguishing between Crohn's disease and intestinal tuberculosis. *J Clin Pathol* 2006; 59:840.
84. Ramadass B, Chittaranjan S, Subramanian V, Ramakrishna BS. Fecal polymerase chain reaction for *Mycobacterium tuberculosis* IS6110 to distinguish Crohn's disease from intestinal tuberculosis. *Indian J Gastroenterol* 2010; 29:152.
85. Kedia S, Sharma R, Sreenivas V, et al. Accuracy of computed tomographic features in differentiating intestinal tuberculosis from Crohn's disease: a systematic review with meta-analysis. *Intest Res* 2017; 15:149.
86. Pulimood AB, Ramakrishna BS, Kurian G, et al. Endoscopic mucosal biopsies are useful in distinguishing granulomatous colitis due to Crohn's disease from tuberculosis. *Gut* 1999; 45:537.
87. Lee YJ, Yang SK, Byeon JS, et al. Analysis of colonoscopic findings in the differential diagnosis between intestinal tuberculosis and Crohn's disease. *Endoscopy* 2006; 38:592.
88. Makharia GK, Srivastava S, Das P, et al. Clinical, endoscopic, and histological differentiations between Crohn's disease and intestinal tuberculosis. *Am J Gastroenterol* 2010; 105:642.
89. Li X, Liu X, Zou Y, et al. Predictors of clinical and endoscopic findings in differentiating Crohn's disease from intestinal tuberculosis. *Dig Dis Sci* 2011; 56:188.

90. Pulimood AB, Amarapurkar DN, Ghoshal U, et al. Differentiation of Crohn's disease from intestinal tuberculosis in India in 2010. *World J Gastroenterol* 2011; 17:433.
91. Yu H, Liu Y, Wang Y, et al. Clinical, endoscopic and histological differentiations between Crohn's disease and intestinal tuberculosis. *Digestion* 2012; 85:202.
92. Bae JH, Park SH, Ye BD, et al. Development and Validation of a Novel Prediction Model for Differential Diagnosis Between Crohn's Disease and Intestinal Tuberculosis. *Inflamm Bowel Dis* 2017; 23:1614.
93. Limsrivilai J, Shreiner AB, Pongpaibul A, et al. Meta-Analytic Bayesian Model For Differentiating Intestinal Tuberculosis from Crohn's Disease. *Am J Gastroenterol* 2017; 112:415.
94. Pratap Mouli V, Munot K, Ananthakrishnan A, et al. Endoscopic and clinical responses to anti-tubercular therapy can differentiate intestinal tuberculosis from Crohn's disease. *Aliment Pharmacol Ther* 2017; 45:27.
95. Gupta A, Pratap Mouli V, Mohta S, et al. Antitubercular Therapy Given to Differentiate Crohn's Disease From Intestinal Tuberculosis Predisposes to Stricture Formation. *J Crohns Colitis* 2020; 14:1611.
96. Narang H, Kedia S, Ahuja V. New diagnostic strategies to distinguish Crohn's disease and gastrointestinal tuberculosis. *Curr Opin Infect Dis* 2024; 37:392.
97. Keri VC, Jorwal P, Kodan P, Biswas A. Tuberculosis masquerading as metastasis in liver: a rare and an unusual presentation. *BMJ Case Rep* 2020; 13.
98. Koc S, Beydilli G, Tulunay G, et al. Peritoneal tuberculosis mimicking advanced ovarian cancer: a retrospective review of 22 cases. *Gynecol Oncol* 2006; 103:565.
99. Buyukberber M, Sevinc A, Cagliyan CE, et al. Non-Hodgkin lymphoma with high adenosine deaminase levels mimicking peritoneal tuberculosis: an unusual presentation. *Leuk Lymphoma* 2006; 47:565.
100. Jullien S, Jain S, Ryan H, Ahuja V. Six-month therapy for abdominal tuberculosis. *Cochrane Database Syst Rev* 2016; 11:CD012163.
101. Makharia GK, Ghoshal UC, Ramakrishna BS, et al. Intermittent Directly Observed Therapy for Abdominal Tuberculosis: A Multicenter Randomized Controlled Trial Comparing 6 Months Versus 9 Months of Therapy. *Clin Infect Dis* 2015; 61:750.
102. Tony J, Sunilkumar K, Thomas V. Randomized controlled trial of DOTS versus conventional regime for treatment of ileocecal and colonic tuberculosis. *Indian J Gastroenterol* 2008; 27:19.
103. Demir K, Okten A, Kaymakoglu S, et al. Tuberculous peritonitis--reports of 26 cases, detailing diagnostic and therapeutic problems. *Eur J Gastroenterol Hepatol* 2001; 13:581.
104. Giouleme O, Paschos P, Katsaros M, et al. Intestinal tuberculosis: a diagnostic challenge--case report and review of the literature. *Eur J Gastroenterol Hepatol* 2011; 23:1074.
105. Park YS, Jun DW, Kim SH, et al. Colonoscopy evaluation after short-term anti-tuberculosis treatment in nonspecific ulcers on the ileocecal area. *World J Gastroenterol* 2008; 14:5051.
106. Aggarwal P, Kedia S, Sharma R, et al. Tubercular Intestinal Strictures Show a Poor Response to Anti-Tuberculous Therapy. *Dig Dis Sci* 2017; 62:2847.
107. Bhasin DK, Sharma BC, Dhavan S, et al. Endoscopic balloon dilation of ileal stricture due to tuberculosis. *Endoscopy* 1998; 30:S44.
108. Cho JK, Choi YM, Lee SS, et al. Clinical features and outcomes of abdominal tuberculosis in southeastern Korea: 12 years of experience. *BMC Infect Dis* 2018; 18:699.
109. Chalya PL, Mchembe MD, Mshana SE, et al. Clinicopathological profile and surgical treatment of abdominal tuberculosis: a single centre experience in northwestern Tanzania. *BMC Infect Dis* 2013; 13:270.
110. Wang HK, Hsueh PR, Hung CC, et al. Tuberculous peritonitis: analysis of 35 cases. *J Microbiol Immunol Infect* 1998; 31:113.