Abdominal tuberculosis

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INTRODUCTION

Abdominal tuberculosis (TB) includes involvement of the gastrointestinal tract, peritoneum, lymph nodes, and/or solid organs [1-4]. Abdominal TB comprises around 5 percent of all cases of TB worldwide [5].

Issues related to TB involving the intestinal tract, peritoneum, and liver will be reviewed here; issues related to clinical manifestations, diagnosis, and treatment of pulmonary TB are discussed separately. (See "Clinical manifestations and complications of pulmonary tuberculosis" and "Diagnosis of pulmonary tuberculosis in adults" and "Treatment of drug-susceptible pulmonary tuberculosis in nonpregnant adults without HIV infection".)

RISK FACTORS

Risk factors for development of abdominal TB include cirrhosis, human immunodeficiency virus (HIV) infection, diabetes mellitus, underlying malignancy, malnutrition, treatment with antitumor necrosis factor agents [6], corticosteroids, and use of continuous ambulatory peritoneal dialysis [7-11]. Issues related to the epidemiology of TB are discussed further separately. (See "Epidemiology of tuberculosis".)

FORMS OF DISEASE

Abdominal TB can present with involvement of any of the following sites: peritoneum, stomach, intestinal tract, hepatobiliary tree, pancreas, perianal area, and lymph nodes. The most common forms of disease include involvement of the peritoneum, intestine, and/or lymph nodes.

TB of the abdomen may occur via reactivation of latent TB infection or by ingestion of tuberculous mycobacteria (as with ingestion of unpasteurized milk or undercooked meat). In the setting of active pulmonary TB or miliary TB, abdominal involvement may develop via hematogenous spread via contiguous spread of TB from adjacent organs (such as retrograde spread from the fallopian tubes) or via spread through lymphatic channels [2].

In general, 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, bowel obstruction, and abdominal mass [12]. Additional issues related to clinical manifestations are discussed in the following sections.

Routine laboratory tests demonstrate mild anemia and increased sedimentation rate in 50 to 80 percent of patients; the white blood count is usually normal [13]. Approximately 15 to 25 percent of patients with abdominal TB have concomitant pulmonary TB [2].

Peritoneal tuberculosis — Peritoneal TB occurs most commonly following reactivation of latent tuberculous foci in the peritoneum established via hematogenous spread from a primary lung focus [7]. In the United States in 2017, of 1887 cases of exclusively extrapulmonary TB reported to Centers for Disease Control and Prevention, 6.2 percent were peritoneal [14].

TB peritonitis can also occur via hematogenous spread in the setting of active pulmonary TB or miliary TB. Much less commonly, tuberculous mycobacteria enter the peritoneal cavity transmurally from an infected small intestine or via contiguous spread from tuberculous salpingitis [15]. As the disease progresses, the visceral and parietal peritoneum become studded with tubercles. Ascites develop secondary to exudation of proteinaceous fluid from the tubercles.

Clinical manifestations of peritoneal TB include ascites (93 percent), abdominal pain (73 percent), and fever (58 percent) [11]. Typically, symptoms have persisted for week or months before the diagnosis is established [16-18]. More than 90 percent of patients with tuberculous peritonitis have ascites at the time of presentation, with a serum-ascites albumin gradient <1.1 g/dL (in the absence of cirrhosis) [19]. In 10 percent of cases, patients present with a more advanced "dry" phase with a "doughy" abdomen, which represents a fibroadhesive form of the disease [20,21]. The absence of signs of chronic liver disease (such as palmar erythema, spider angiomata, and dilated abdominal wall veins) should increase clinical suspicion for TB peritonitis [4]. (See "Evaluation of adults with ascites", section on 'Serum-to-ascites albumin gradient'.)

In patients with renal failure on continuous ambulatory peritoneal dialysis (CAPD), clinical manifestations of tuberculous peritonitis typically develop within the first year of beginning CAPD and are usually indistinguishable from bacterial peritonitis. (See "Clinical manifestations and diagnosis of peritonitis in peritoneal dialysis".)

In one series including 60 patients, risk factors for tuberculous peritonitis included (in descending order of frequency): cirrhosis, CAPD, diabetes mellitus, underlying malignancy, use of systemic corticosteroids, and acquired immunodeficiency syndrome (AIDS) [11]. No risk factor was identified in 20 percent of cases. The mean age at presentation was 55 with an approximately equal sex distribution.

Intestinal tuberculosis — Two types of bowel lesions are seen: ulcerative and ulcerohypertrophic. The ulcerative form has been described in malnourished individuals; the ulcerohypertrophic form has been described in relatively well-nourished individuals. Ulcerative and stricturous forms are usually observed in the small intestine, while colonic and ileocecal lesions are usually ulcerohypertrophic. The most common site of involvement is ileocecal area (ranging from 25 to 90 percent across various series), followed by small intestine (6 to 67 percent), colon (2 to 32 percent), and gastroduodenal area (0.5 to 5 percent).

Signs and symptoms in a patient with intestinal TB may include:

- Clinical manifestations reflecting intestinal ulceroconstrictive disease; these include intestinal colic, abdominal distension, chronic diarrhea, nausea, vomiting, constipation, and bleeding.
- Clinical manifestations reflecting adjacent tissue involvement; these include ascites, lymph node enlargement, and tubo-ovarian symptoms.
- Clinical manifestations of chronic inflammation; these include fever, fatigue, weight loss, and night sweats.

Symptoms depend upon the site(s) of involvement and the type of lesions. Abdominal pain is the most common manifestation and can occur because of a stricture of the intestinal lumen, mesenteric inflammation, and/or peritoneal involvement. Diarrhea occurs in 11 to 37 percent of patients and may reflect small and/or large intestinal involvement. Lower gastrointestinal bleeding occurs in 5 to 15 percent of patients with intestinal TB; the bleeding is rarely massive. Constipation occurs in near half of patients with intestinal TB. Constitutional symptoms (fever, malaise, night sweats, anorexia, and weight loss) are common in patients with intestinal TB [22-25]. A palpable right lower quadrant abdominal mass is present in 25 to 50 percent of patients [26,27]. Fistula and intestinal stricture may occur. Bowel obstruction is the most common complication and may be due to progressive stricture or adhesions [28-30].

Patients with gastroduodenal involvement can present with abdominal pain, early satiety, postprandial fullness, nausea, and vomiting. Malabsorption can occur in the context of intestinal TB; etiologies may include 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 [31].

Multiple areas of the bowel can be affected. The ileocecal region is the most common site of intestinal involvement; it is affected in 75 percent of cases [1]. The affinity of *Mycobacterium tuberculosis* for this site may be due to the relative stasis and abundance of lymphoid tissue in this region. The organism penetrates the mucosa and localizes in the submucosal lymphoid tissue, where it initiates an inflammatory reaction with subsequent lymphangitis, endarteritis, granuloma formation, caseation necrosis, mucosal ulceration, and scarring. Other locations of involvement, in order of descending frequency, are the ascending colon, jejunum, appendix, duodenum, stomach, esophagus, sigmoid, colon and rectum [1].

The epidemiology of tuberculous enteritis varies widely; young adults, primarily women, are mostly affected in regions like Pakistan, Turkey, and West Africa, whereas a lower disease incidence but similar or greater numbers of male patients are reported in studies from China, Singapore, India, and the United Kingdom [32].

Hepatic tuberculosis — There are two presentations of hepatic TB: miliary hepatic disease and isolated hepatic disease. Liver involvement in miliary TB occurs in approximately 80 percent of cases of disseminated TB; in this form, TB reaches the liver via the hepatic artery. Isolated hepatic TB occurs in approximately 20 percent of cases; in this form, TB reaches the liver from the intestinal tract via the portal vein or gastrointestinal lymphatics or may represent reactivation of latent TB infection.

The most common signs and symptoms of hepatic TB include hepatomegaly (80 percent), fever (67 percent), respiratory symptoms (66 percent), abdominal pain (60 percent), and weight loss (58 percent); other signs include splenomegaly (30 percent), ascites (20 percent), and jaundice (20 percent) [33]. Local hepatic TB may present with abdominal pain, fever, and, uncommonly, jaundice, while miliary hepatic TB may present with acute respiratory symptoms (cough, sputum production).

In endemic regions, hepatic TB can present in the form of hepatic abscess, as an intrahepatic mass, or as granulomatous hepatitis (fever with hepatomegaly and mild transaminase elevation with disproportionate alkaline phosphatase elevation). TB is the most common cause of granulomatous hepatitis in endemic areas.

Presence of jaundice usually suggests biliary involvement. 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) [34,35].

Elevated liver function tests (LFTs) may reflect the site of disease; involvement of the liver parenchyma may be reflected by elevated transaminases, while involvement of the porta or biliary ducts may be reflected by elevated alkaline phosphatase and gamma-glutamyl transferase (GGT). Mild hyperbilirubinemia has been reported in miliary and local hepatic TB [33].

Pancreatic tuberculosis — Pancreatic TB is uncommon. It presents with abdominal pain, constitutional symptoms, and jaundice; it must be differentiated from pancreatic malignancy.

Other forms — Uncommon forms of abdominal TB include involvement of the stomach (rare), duodenum (1 percent of abdominal TB), pancreas (rare), and spleen (rare) [1,36].

DIAGNOSIS

Clinical approach — The diagnosis of abdominal TB should be suspected in patients with relevant clinical manifestations (fever, weight loss, abdominal pain and/or distension, ascites, hepatomegaly, diarrhea, abdominal mass, abnormal liver function tests) and relevant epidemiologic factors (history of prior TB infection or disease, known or possible TB exposure, and/or past or present residence in or travel to an area where TB is endemic).

Patients with concomitant clinical or radiographic findings suggestive of pulmonary involvement should also undergo diagnostic evaluation for pulmonary TB; this is discussed separately. (See "Clinical manifestations and complications of pulmonary tuberculosis" and "Diagnosis of pulmonary tuberculosis in adults".)

The diagnosis of abdominal TB may be definitively established by demonstration of *M. tuberculosis* in peritoneal fluid (in the setting of ascites), a biopsy specimen of an involved site (such as peritoneum, intestine, or liver), or via mycobacterial culture and/or nucleic acid amplification test (NAAT) [33,37,38]. In the United States, there is no approved NAAT for abdominal fluid or tissue; a positive validated NAAT strongly supports a diagnosis of TB. Histopathology (presence of caseating granulomas, with or without demonstration of acid-fast bacilli [AFB]) is suggestive of TB but is not pathognomonic.

Patients with suspected abdominal TB should undergo radiographic imaging; computed tomography (CT) with enterography protocol is preferred, if feasible, since it allows cross-sectional evaluation for involvement of the intestine and other organs as well as presence of ascites, peritoneal involvement, and lymphadenopathy [39-41].

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, AFB smear, mycobacterial culture, and, if available, NAAT for *M. tuberculosis*. Patients with tuberculous peritonitis (in the absence of underlying cirrhosis) typically have lymphocytic ascites with serum-ascites albumin gradient (SAAG) <1.1 g/dL. An elevated ascites ADA level (30 to 39 international units/L) is useful to support a diagnosis of TB in noncirrhotic patients but does not establish it with certainty. Patients with suspected tuberculous peritonitis and nondiagnostic ascites fluid analysis should undergo peritoneal 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. All diagnostic specimens should be sent for histopathology, mycobacterial culture, and drug susceptibility testing.

In general, a diagnostic ascites fluid analysis may be used to establish a diagnosis of abdominal TB in immunocompetent patients. However, in immunocompromised patients, the likelihood that more than one condition may be present is increased. Therefore, such patients may warrant collection of diagnostic specimens from each site of disease; the approach should be tailored to individual circumstances with assessment of risks and benefits.

It may not be possible to establish a definitive diagnosis of abdominal TB. Patients with probable abdominal TB include those with nondiagnostic mycobacterial culture and NAAT results but elevated ascitic fluid ADA and/or consistent findings in histology, with exclusion of other items in the differential diagnosis.

Patients with known or suspected TB warrant testing for HIV infection. (See "Acute and early HIV infection: Clinical manifestations and diagnosis".)

Diagnostic tools

Radiographic imaging — CT enterography/magnetic resonance enterography are the most helpful imaging modalities to evaluate for abdominal TB [42-44]:

- CT imaging in the setting of intestinal TB most 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.

- CT imaging in the setting of peritoneal TB demonstrates ascites, lymph nodes, thickening of the mesentery and omentum, and thickening of the peritoneum [45].

- CT imaging in the setting of hepatic TB depends on whether the patient has miliary hepatic TB or local hepatic TB. CT imaging in the setting of miliary disease demonstrates multiple low-density micronodules dispersed throughout the liver; it may also demonstrate hepatomegaly without intrahepatic lesions or abdominal lymphadenopathy with peripheral lymph node enhancement and/or calcifications [33,46]. CT imaging in the setting of local hepatic TB appears as a large solitary nodule or two to three low-density nodules with calcification and peripheral enhancement.

Ultrasound is useful for detecting lymphadenopathy, ascites, peritoneal thickening, omental thickening, and bowel wall thickening [1,2]. In one systematic review including 11 studies evaluating ultrasound for diagnosis of abdominal TB in patients with HIV infection, the sensitivity and specificity were 63 and 68 percent, respectively [47].

Small bowel follow-through or barium enema may demonstrate mucosal ulcerations and strictures, a deformed cecum, and/or an incompetent ileocecal valve (image 5) [2].

Plain radiography may demonstrate air fluid levels with dilated small bowel loops (suggestive of intestinal obstruction) or hepatic calcifications [33].

Chest radiography should be performed in patients with suspected abdominal TB [48]. (See "Diagnosis of pulmonary tuberculosis in adults", section on 'Radiographic imaging'.)

Ascitic fluid analysis — 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 ADA level, AFB smear, mycobacterial culture, and, if available, NAAT for *M. tuberculosis*. Patients with tuberculous peritonitis (in the absence of underlying cirrhosis) typically have lymphocytic ascites with SAAG <1.1 g/dL.

Most patients with tuberculous peritonitis have straw-colored ascites with leukocyte count of 150 to 4000 cells/mm^3, with a relative lymphocytic pleocytosis [12,26]. Patients on peritoneal dialysis with tuberculous peritonitis may have a neutrophilic response [49,50]. (See "Evaluation of adults with ascites".)

The protein content of the ascitic fluid is usually >3.0 g/dL in the setting of tuberculous peritonitis [20,51]. The cell count is 150 to 4000 cells/mcL and consists predominantly of lymphocytes [2]. Patients with tuberculous peritonitis (in the absence of underlying cirrhosis) typically have SAAG <1.1 g/dL; patients with underlying cirrhosis have SAAG ≥1.1 [19,52,53]. (See "Evaluation of adults with ascites".)

The sensitivity of AFB smear and mycobacterial culture ascites fluid is low (less than 2 percent and less than 20 percent, respectively); broth culture results may be available in 2 to 3 weeks, and solid-phase culture results require several weeks [2,26,54]. The yield of mycobacterial culture may be increased (up to 83 percent) if 1 L of ascitic fluid (concentrated by centrifugation) is cultured [55].

The utility of ascitic fluid NAAT for diagnosis of tuberculous TB peritonitis has not been well established. One review including 11 cases of abdominal TB noted a positive polymerase chain reaction (PCR) for *M. tuberculosis* of the ascitic fluid in all cases [56].

Measurement of ascites fluid ADA level is a useful tool for evaluation of patients with suspected TB peritonitis; it is most reliable in the absence of cirrhosis [57-60]. 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. One meta-analysis of 12 studies including 264 patients found that ADA levels had high sensitivity and specificity (100 and 97 percent, respectively) using cut-off values from 36 to 40 international units/L; the optimal cut-off value was 39 international units/L [59]. The sensitivity of ascites fluid ADA in patients with cirrhosis is approximately 30 percent, likely due to poor humoral and T cell–mediated response [53,61]. In such cases, it may be a helpful supportive diagnostic tool if lower thresholds are used (21 to 30 international units/L) [1,62].

Measurement of peritoneal fluid interferon-gamma concentration may be a useful tool for diagnosis of tuberculous peritonitis [58,63-65]. However, this test does not have a role in routine evaluation of suspected tuberculous peritonitis and has not been approved by the US Food and Drug Administration for this purpose.

Obtaining biopsy specimens — Tools for obtaining biopsy specimens include laparoscopy, endoscopy (upper or lower), and radiographic-guided interventions. The approach to obtaining biopsy specimens should be tailored to individual circumstances, including the dominant site of involvement and associated risks and benefits. If ascitic fluid analysis is nondiagnostic, a peritoneal biopsy should be performed via laparoscopy. In the setting of intestinal involvement, endoscopic biopsies should be obtained; in the setting of isolated hepatic involvement, a liver biopsy should be pursued. Endoscopic ultrasound can be used for fine needle aspiration biopsies from peripancreatic and celiac lymph nodes.

Peritoneal biopsy – Laparoscopy — Laparoscopy is useful for visualization of the peritoneum and peritoneal biopsy [11,20,21,26,54,56,66,67]. In one systematic review including 402 patients, the sensitivity and specificity of laparoscopic examination in making the diagnosis of peritoneal TB was 93 and 98 percent, respectively [19]. In another study including 38 patients with peritoneal TB, laparoscopic biopsy enabled a histologic diagnosis in 82 percent of cases; visual diagnosis was accurate in 95 percent of cases [21].

Laparoscopic findings in the setting of tuberculous peritonitis include [21,56]:

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

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

Blind peritoneal biopsies have a low success rate and have been associated with complications including death [68,69].

Intestinal biopsy – Endoscopy — Colonoscopy with biopsy is useful to obtain material for histology and culture [70-75]. Biopsy is also useful for investigation of the diseases that comprise the differential diagnosis of TB enteritis. (See 'Differential diagnosis' below.)

Endoscopic findings of intestinal TB may include ulcers, strictures, nodules, pseudopolyps, and/or deformed ileocecal valve (image 6 and image 7) [76].

Forms of intestinal lesions include ulcerative and hypertrophic; these forms can coexist [1,2,26,77].

- The ulcerative presentation commonly affects the ileum and jejunum; it is characterized by single or multiple transverse ulcers that form strictures during the healing process and may perforate, bleed, or form fistulas [77]. Ulcers due to TB tend to be circumferential (picture 1) and are usually surrounded by inflamed mucosa.

- The hypertrophic 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).

Deep endoscopic biopsies should be taken from ulcers and their margins. TB granulomas are often submucosal, whereas granulomas due to Crohn disease are typically mucosal (though submucosal granulomas may also be seen) [78].

Endoscopic ultrasound may be used to pursue fine-needle aspiration biopsy of the mediastinal, celiac, or peripancreatic lymph nodes [79,80].

Liver biopsy — Liver biopsy with mycobacterial culture is the most specific diagnostic test for hepatic TB [33]. Liver biopsy provides material for microbiologic studies (including culture and drug susceptibility testing as well as nucleic acid amplification testing where available) as well as histopathologic examination (including staining for AFB).

Evaluating biopsy specimens — Biopsy specimens should be sent for microbiology evaluation (including AFB smear, mycobacterial culture and/or PCR) as well as histopathology evaluation.

The sensitivity of AFB smear and mycobacterial culture for biopsy specimens is low (less than 50 percent) [33,81]. If available, PCR is more sensitive and specific for diagnosis of TB than AFB smear or mycobacterial culture, and often PCR results are available sooner [32,82]. The utility of PCR varies depending on the tissue type; sensitivity and specificity are high for peritoneal fluid and pancreatic and hepatic tissue, but intestinal tissue may be associated with false-positive PCR results. As an example, in one series including 43 liver biopsies with granulomas, PCR had sensitivity and specificity of 53 and 96 percent, respectively [81]. Thus far, no commercial NAATs (including Xpert MTB/RIF) are approved in the United States for use with nonrespiratory tract secretions.

Histologic evidence of caseating (necrotizing) granulomas had a median sensitivity of 68 percent among hepatic TB case series. Presence of caseating granulomas is suggestive of TB but is not pathognomonic; the finding may support a diagnosis of TB in the setting of other relevant clinical and epidemiologic factors [33].

Caseation granulomas and positive acid-fast stain are observed in fewer than 33 percent of cases [71,76]. Some histologic 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 [1,83].

Additional tools — A positive tuberculin skin test or interferon-gamma release assay may be observed in patients with abdominal TB but is of limited value because these assays do not differentiate between active disease and latent infection [13,26]. (See "Use of interferon-gamma release assays for diagnosis of tuberculosis infection (tuberculosis screening) in adults" and "Tuberculosis infection (latent tuberculosis) in adults: Approach to diagnosis (screening)".)

Stool PCR, generally not available in the United States, may be a useful adjunctive diagnostic tool for diagnosis of intestinal TB [84,85].

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of abdominal TB includes:

- Crohn disease – There can be marked overlap between the features of Crohn disease (CD) and intestinal TB, making the differentiation between the two conditions difficult. Distinguishing between these entities is important because use of immunosuppressive drugs for a misdiagnosis of CD may be associated with clinical deterioration in patents with TB. (See "Clinical manifestations, diagnosis, and prognosis of Crohn disease in adults".)

  A combination of clinical, radiographic, endoscopic, laboratory, and biopsy findings are used to distinguish between these diseases, as summarized in the table (table 1) [44,78,84,86-93]. Additional considerations include:

  - The presence of ascites may help to distinguish tuberculous enteritis from CD, since ascites is uncommon in CD.
  - In the setting of TB, granulomas associated with TB tend to be submucosal, large, and confluent, often with caseation necrosis [1]. In the setting of CD, in contrast, granulomas are typically mucosal, infrequent, small, nonconfluent, and noncaseating.
  - 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].
  - If there is no improvement (with respect to symptoms, endoscopic and/or histologic findings) after two months of empiric antituberculous therapy, CD (and perhaps other conditions) remain in the differential diagnosis.

- 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: Clinical manifestations" and "Spontaneous bacterial peritonitis in adults: Diagnosis".)

- Malignancy – Malignancy may be associated with ascites, peritoneal involvement, and/or liver lesions; 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 [4]. Serum CA-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 [95]. (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 – Lymphoma may present with abdominal pain and weight loss with radiographic findings demonstrating abdominal lymphadenopathy. Lymph node biopsy for culture and histopathology evaluation is the most useful tool to distinguish between lymphoma and TB. (See "Clinical presentation and initial evaluation of non-Hodgkin lymphoma".)

Presence of granuloma on histopathology may reflect other conditions besides 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 infection outside the central nervous system".)

- The differential diagnosis of noncaseating granulomas includes sarcoidosis, rheumatoid pleuritis, 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 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.

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 adenosine deaminase and/or consistent findings in histology, with nondiagnostic mycobacterial culture and nucleic acid amplification test results), an empiric trial of antituberculous therapy is reasonable.

Antituberculous therapy — In general, the approach to antituberculous therapy for abdominal TB is the same as that for pulmonary TB [96-98]. (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".)

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 [99]. Patients with tuberculous enteritis generally demonstrate clinical improvement within two to four weeks on empiric therapy [98,100]. In one study, colonoscopic follow-up after two to three months of anti-TB therapy showed complete healing of active ulcers and erosions [101].

However, healing in the setting of antituberculous therapy can also result in worsening of strictures due to scar tissue formation. Patients who develop bowel obstruction usually cannot ingest medications orally, limiting the choice of drug therapy to injectable agents, and may warrant surgical intervention. In one series including 106 patients with stricturing intestinal TB, resolution of stricture following antituberculous therapy occurred in only 25 percent of patients [102]. 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).

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 [13]. (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 [13,27,37]. 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 [30]. 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 [37]. 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 [103].

PROGNOSIS

Mortality ranges from 1.4 to 20 percent in various series [104,105]. Advanced age, delay in initiating therapy, and underlying cirrhosis have been associated with higher mortality rates [10-12,106].

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

- **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, and 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 'Forms of disease' 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 (history of prior TB infection or disease, known or possible TB exposure, and/or past or present residence in or travel to an area where TB is endemic).
    - 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; computed tomography (CT) 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. (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 (AFB) 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 (SAAG) <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 'Ascitic 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 'Obtaining biopsy specimens' 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.)

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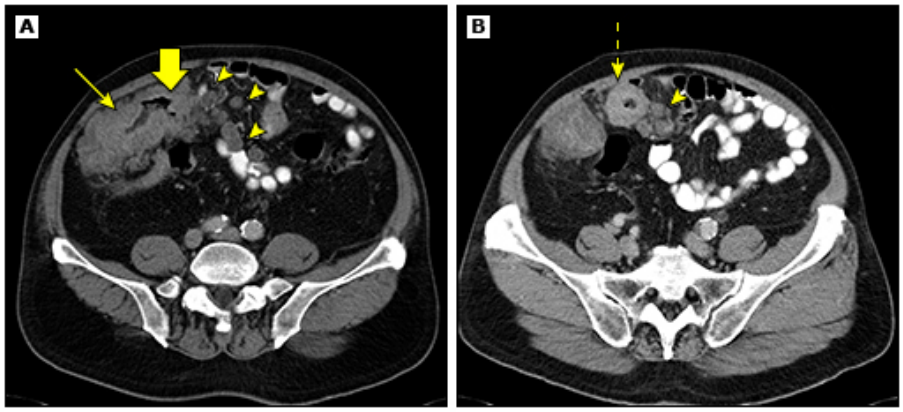
Topic 1238 Version 26.0

GRAPHICS

Tuberculous enteritis – Computed tomography

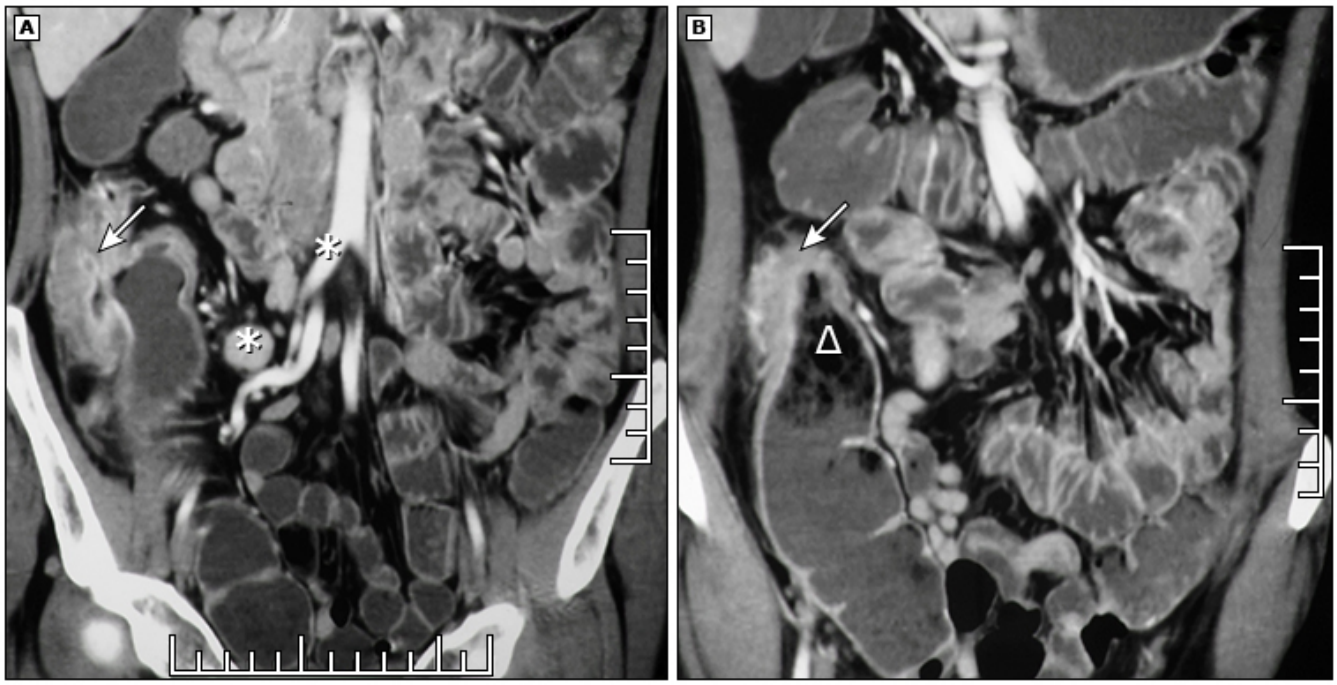


Contrast-enhanced computed tomography of histologically proven ileocecal tuberculosis showing thickened terminal ileum (arrow), ileocecal valve (thick arrow), and cecum (dashed arrow) with enlarged necrotic ileocecal lymph nodes (arrowheads).

Courtesy of Sudhakar Venkatesh, MD.

Graphic 83833 Version 3.0

Intestinal tuberculosis - CT enteroclysis I



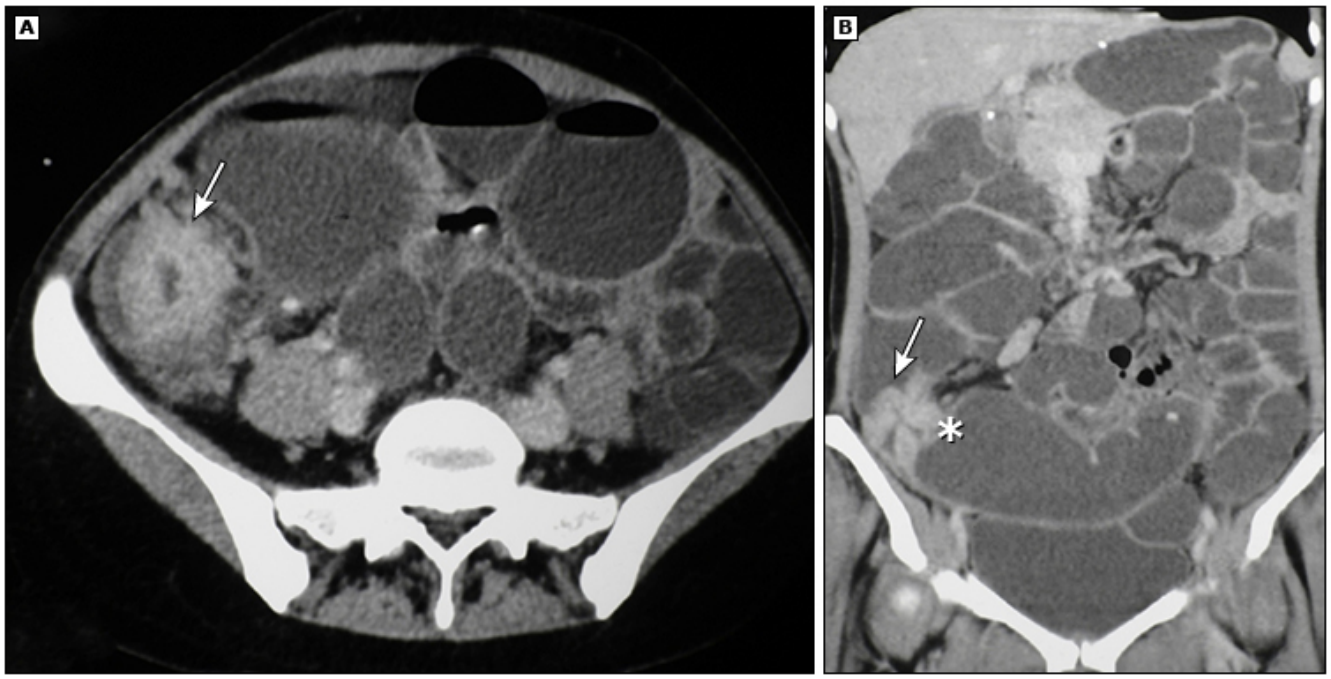
A 40-year-old male patient presented with recurrent episodes of abdominal pain for one year with an episode of partial bowel obstruction, which was treated conservatively. Coronal reformatted images from CT enteroclysis demonstrate contiguous ileocecal involvement with ileal stricture (arrows) and large pericecal and mesenteric lymph nodes (asterisk) and small bowel feces sign (delta). These features are suggestive of intestinal tuberculosis.

CT: computed tomography.

Courtesy of Vineet Ahuja, MD, DM.

Graphic 114924 Version 1.0

Intestinal tuberculosis – CT enteroclysis II



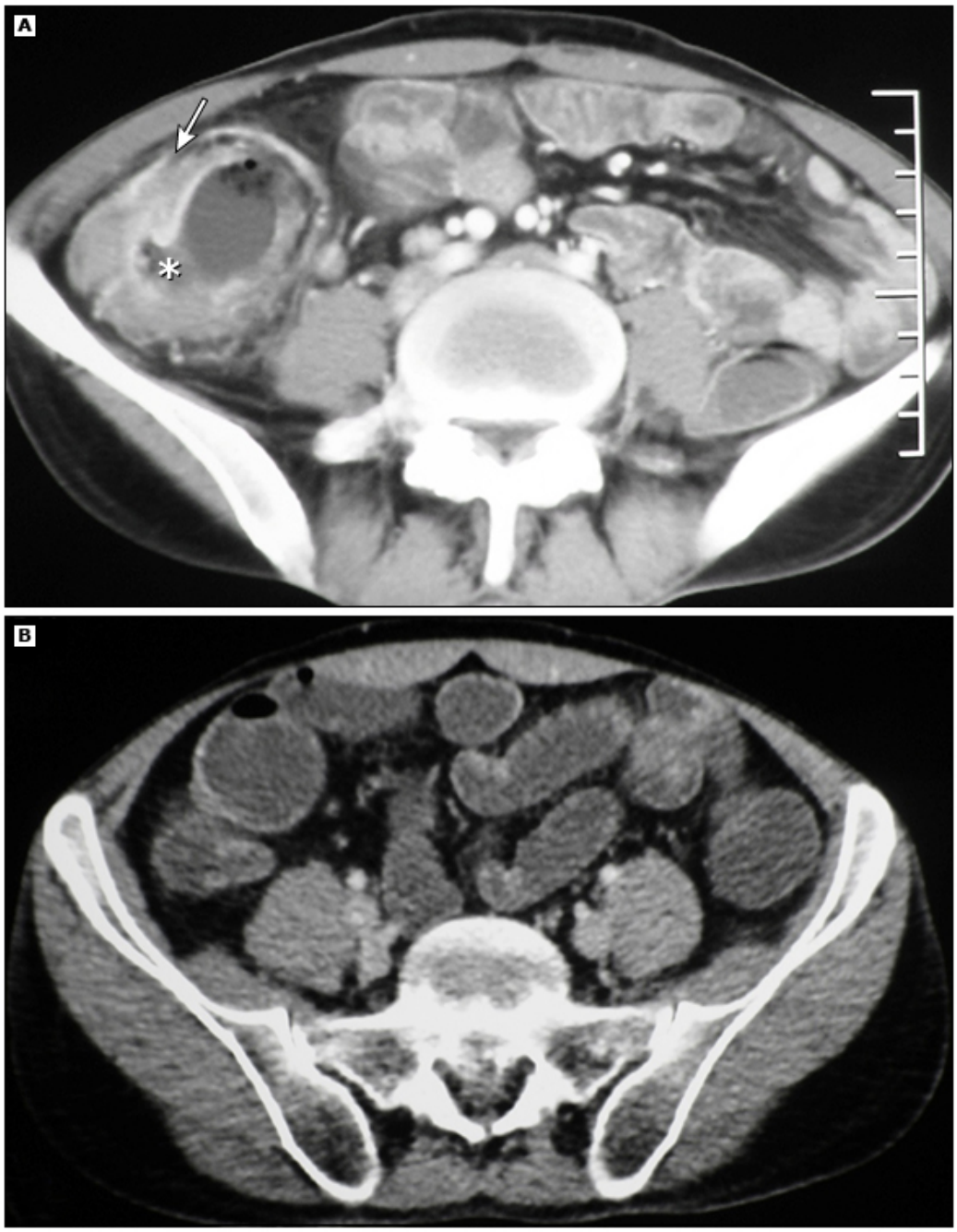
Axial (A) and coronal reformatted (B) images demonstrate short segment annular terminal ileal stricture (arrow) with contiguous ileocecal involvement (asterisk).

CT: computed tomography.

Courtesy of Vineet Ahuja, MD, DM.

Graphic 114925 Version 1.0

Intestinal tuberculosis with interval resolution



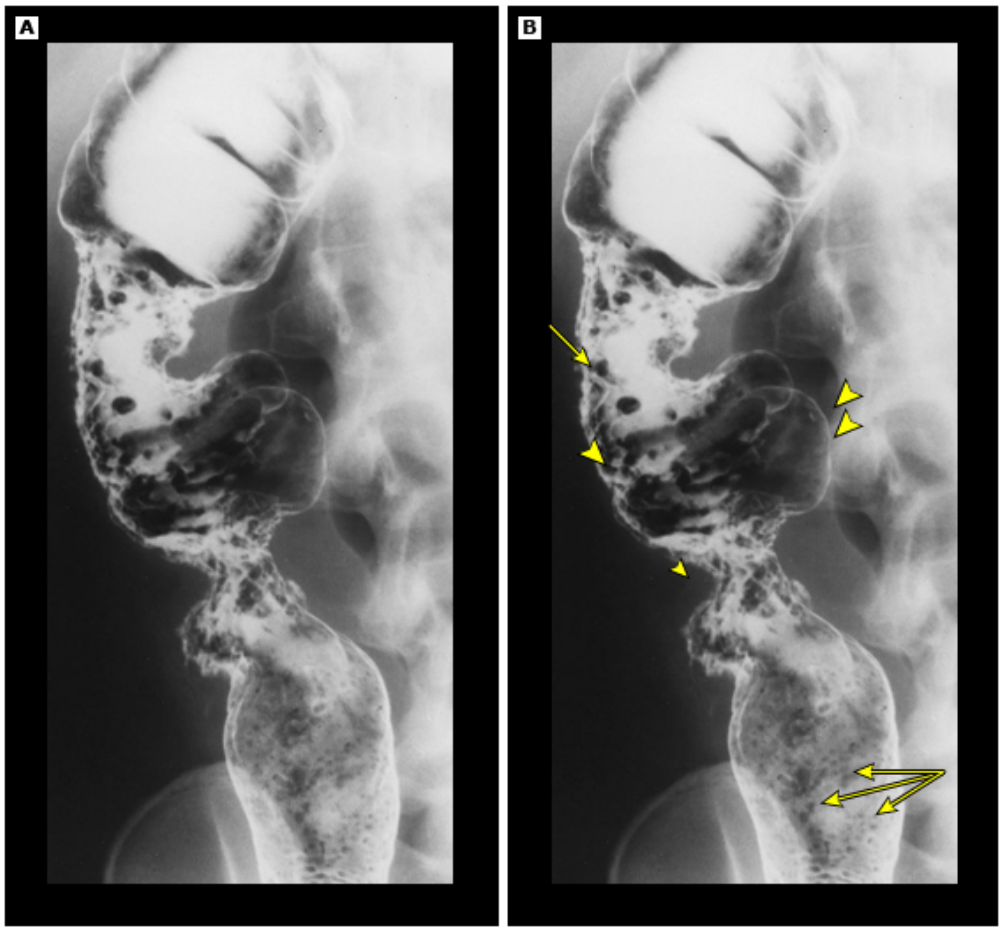
Axial images from contrast-enhanced CT demonstrating (A) ileocecal involvement (arrows) with ulceration (asterisk) and (B) complete resolution of radiographic findings after six months of antituberculous therapy.

CT: computed tomography.

Courtesy of Vineet Ahuja, MD, DM.

Graphic 114926 Version 1.0

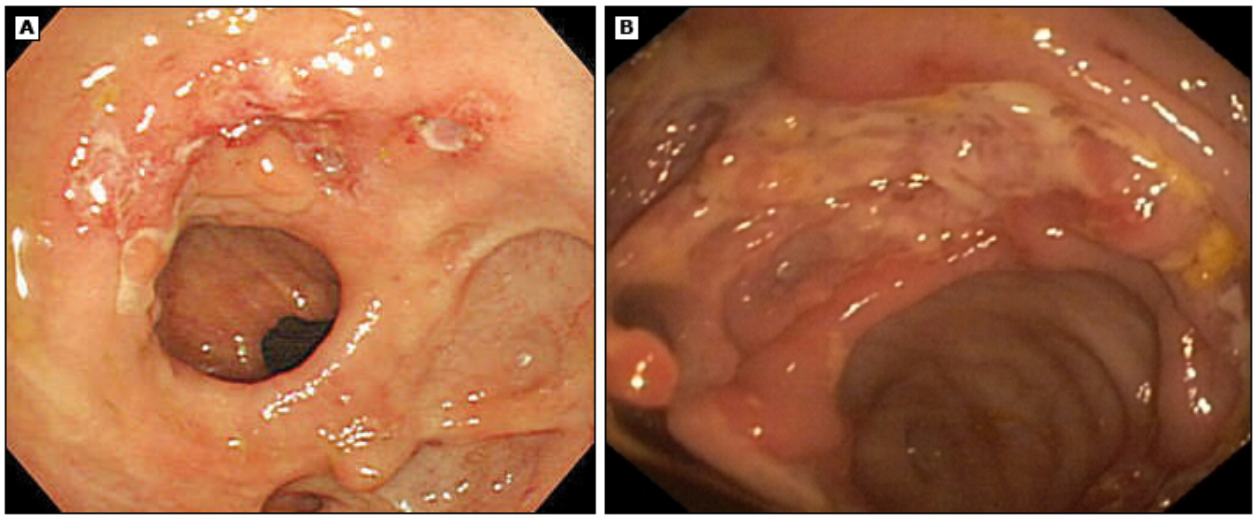
Tuberculous enteritis – Barium enema



Double contrast barium enema demonstrates ileocecal valve region. The cecum is fibrotic and contracted (arrow), the ileocecal valve is irregular, narrowed, gaping, and incompetent (small arrowhead), and the terminal ileum appears to empty directly into the ascending colon (Stierlin's sign; region of small arrowhead). Note the diffuse ulcerations in the ascending colon (large arrowheads) and the lymphoid follicles in the terminal ileum (triple arrow).

Graphic 85512 Version 3.0

Intestinal tuberculosis - Endoscopy

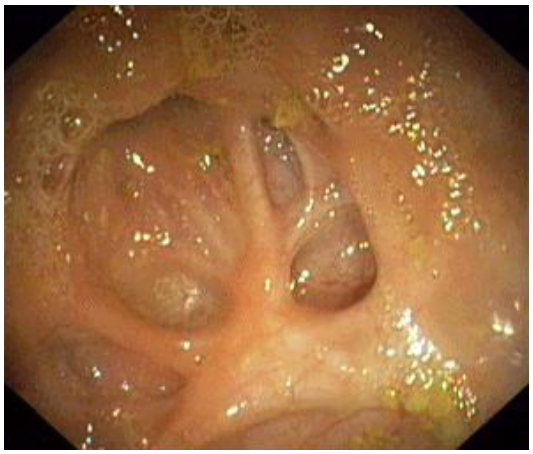


These endoscopy images of intestinal tuberculosis demonstrate a patulous ileocecal valve, scar changes, and multiple ulcers.

Courtesy of Vineet Ahuja, MD, DM.

Graphic 114942 Version 1.0

Healed intestinal tuberculosis - Endoscopy

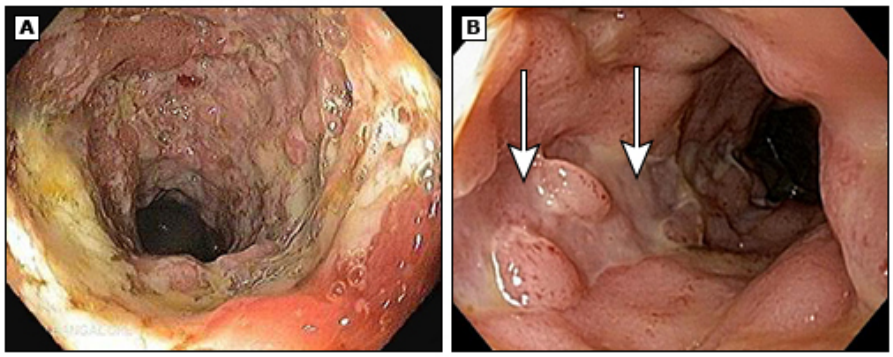


This endoscopy image of healed intestinal tuberculosis demonstrates cecal cicatrization.

Courtesy of Vineet Ahuja, MD, DM.

Graphic 114943 Version 1.0

Ascending colon tuberculous enteritis



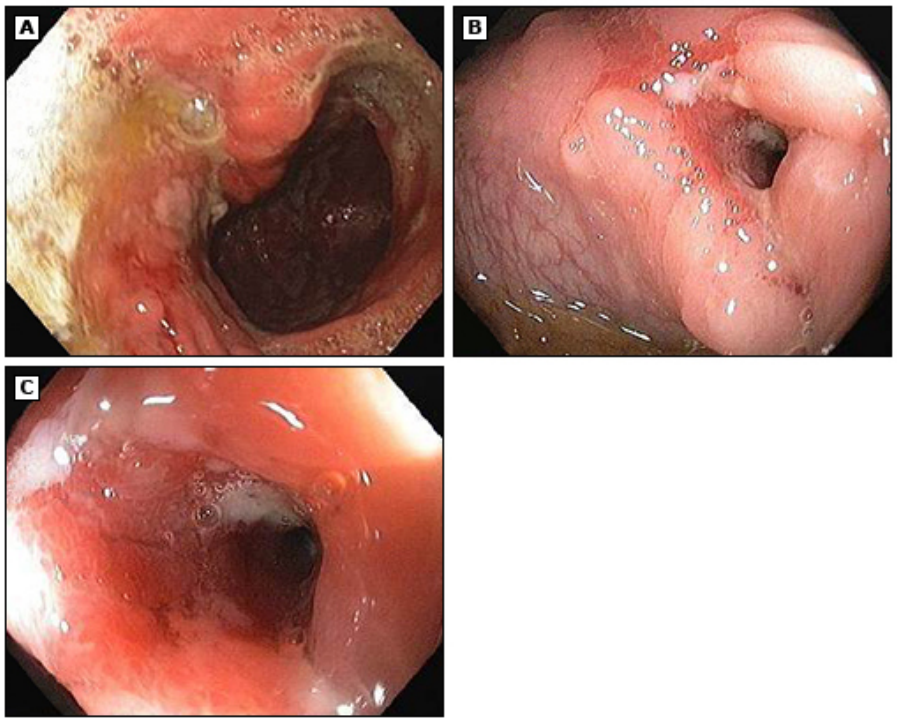
(A) Colonoscopic view of circumferential ulcers involving the cecum and ascending colon due to tuberculosis.

(B) Colonoscopic view of longitudinal ulcers (arrows) involving the ascending colon due to Crohn disease.

Courtesy of Harshad Devarbhavi, MD (image A) and Louis M. Wong Kee Song, MD (image B).

Graphic 83831 Version 3.0

Ileocecal tuberculous enteritis



Colonoscopic views of histologically confirmed ileocecal tuberculosis with ulcerations (A) and destroyed ileocecal valve (B and C).

Courtesy of Harshad Devarbhavi, MD.

Graphic 83832 Version 1.0

Features that help distinguish between Crohn disease and intestinal tuberculosis

	Crohn disease	Intestinal tuberculosis
Clinical manifestations	▪ Perianal disease	▪ High-swinging fever (>38.5°C) in absence of intraabdominal abscess ▪ Evidence of pulmonary TB on chest radiograph
Radiographic findings (CT/MRI)	▪ Symmetrical bowel wall thickening ▪ Mesenteric fibrofatty proliferation (creeping fat) ▪ Mesenteric vascular engorgement (comb sign) ▪ Small homogenous pericecal lymph nodes	▪ Asymmetrical bowel wall thickening ▪ Inflammatory mass centered around the cecum and enveloping the terminal ileum ▪ Large mesenteric nodes with necrotic centers ▪ Ascites
Endoscopic findings	▪ Longitudinal ulcers ▪ Aphthous ulcers ▪ Cobblestoned mucosa ▪ Preservation of ileocecal valve ▪ Multiple skip lesions ▪ Anorectal lesions	▪ Transverse ulcers ▪ Hypertrophic mucosa ▪ Scars/fibrous bands/inflammatory polyps ▪ Gaping/destruction of ileocecal valve ▪ Hyperemic nodules
Histopathologic findings	▪ Single granulomas ▪ Architectural distortion distant from granulomatous inflammation	▪ Caseating granulomas or positive acid-fast bacilli staining* ▪ Confluent (≥5/biopsy) and large (diameter >200 micrometers) granulomas; submucosal granulomas ▪ Ulcers lined by epithelioid histiocytes ▪ Disproportionate submucosal inflammation

TB: tuberculosis; CT: computed tomography; MRI: magnetic resonance imaging.

* Features pathognomonic for intestinal TB but present in <30 percent of cases; no single variable described above is absolutely specific for either condition otherwise.

Graphic 85948 Version 4.0

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Vineet Ahuja, MD, DM No relevant financial relationship(s) with ineligible companies to disclose. **Sanjiv Chopra, MD, MACP** No relevant financial relationship(s) with ineligible companies to disclose. **John Bernardo, MD** No relevant financial relationship(s) with ineligible companies to disclose. **Elinor L Baron, MD, DTMH** No relevant financial relationship(s) with ineligible companies to disclose.

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