
Abdominal Tuberculosis

Angeline A. Lazarus, MD, FACP, FCCP and
Bennett Thilagar, MD

Introduction

Tuberculosis (TB) can involve the entire gastrointestinal tract (GI) including the peritoneum and the pancreatobiliary system. The incidence and severity depends on the prevalence of TB and infection with human immunodeficiency syndrome (HIV). Abdominal TB is seen more commonly between 25 and 45 years of age. The modes of infection of the GI include hematogenous spread from a primary lung focus that reactivates later or miliary tuberculosis, spread via lymphatics from infected nodes, ingestion of bacilli either from the sputum or from infected sources such as milk products, or by direct spread from adjacent organs. Involvement of the abdominal lymph nodes and the peritoneum may occur without other organ involvement. The most common site for abdominal TB is the ileocecal area. Infection often results in granuloma formation, caseation, mucosal ulceration, fibrosis, and scarring.¹⁻⁴

The clinical presentation of abdominal TB may be acute or chronic. Patients often have fever (40–70%), weight loss (40–90%), abdominal pain (80–95%), abdominal distension, diarrhea (11–20%), and constipation. Fatigue, malaise, and anorexia are also seen. Dysphagia and odonophagia are seen in esophageal TB. Gastric TB may mimic peptic ulcer disease or gastric carcinoma. Duodenal TB may present with dyspepsia or duodenal obstruction. Abdominal pain, nausea and vomiting, and symptoms of malabsorption may be seen in ileocecal TB. Colonic tuberculosis may be focal or multifocal with pain as the predominant symptom. Other symptoms such as fever, anorexia, weight loss, and change in bowel habits are often reported. Rectal and anal involvement by TB presents with hematochezia as the predominant symptom with constipation in approximately one-third of patients. Multiple fistulae may be the presenting feature in anal TB.¹⁻⁴

Radiographic imaging such as plain abdominal series, barium enema,

The views expressed in this article are those of the authors and do not reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States Government.
Dis Mon 2007;53:32-38
0011-5029/2007 \$32.00 + 0
doi:10.1016/j.disamonth.2006.10.004



FIG 1. Computed tomography of abdomen shows peritoneal and bowel wall thickening in tuberculous enteritis.

upper GI series with small intestinal follow-through, chest radiograph, computed tomography (CT), and/or ultrasonography (US) of the abdomen are often utilized. In the diagnostic evaluation of abdominal TB, CT of the abdomen is helpful in visualizing thickened peritoneum, ascites, mesenteric disease, lymph node enlargement, caseation within lymph nodes, bowel wall thickening, omental thickening, and bowel obstruction⁵⁻⁷ (Fig 1). Patients with AIDS usually have a more severe form of involvement than those who did not have AIDS.⁸ Other GI diagnostic studies include upper endoscopy and colonoscopy. Endoscopy reveals intestinal lesions that may appear as ulcers (60%), ulcerohypertrophic (30%), or hypertrophic (10%) (Fig 2). Other notable changes include fibrous bands, fistulae, pseudopolyps, and ileocecal valve deformities. A deformed, patulous cecal valve with heaped up mucosal folds is suggestive of tuberculous etiology.^{1,8,9} Alvares et al. reported colonoscopic findings of ulcers (70%), nodules (56%), a deformed ileocecal valve (40%), strictures (23%), polypoid lesions (14%), and fibrous bands (7%). The most common sites were the cecum and ascending colon. In nearly half of these patients, more than one site was involved.¹⁰ Histopathology revealed granulomas with caseation in two-thirds of granulomas. Even in the absence of granulomas, biopsies should be sent for culture to increase diagnostic yield. Repeated biopsies may be needed for confirmatory

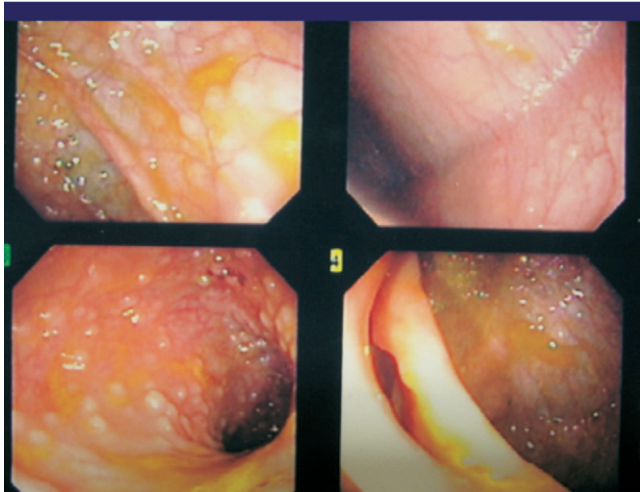


FIG 2. A 10-year-old boy with bovine intestinal tuberculosis after eating unpasteurized cheese from Mexico. (Color version of figure is available online.)

diagnosis. Polymerase chain reaction (PCR) testing of biopsy tissue has shown higher sensitivity and specificity.¹¹

Hepatic tuberculosis is often seen in miliary TB and presents with hepatomegaly and hepatic failure. Tuberculoma and tuberculous liver abscesses are uncommon manifestations of hepatic TB. When they appear as discrete nodules, diagnosis can be difficult. The most common symptoms are right upper quadrant pain, fever, anorexia, and weight loss. Elevation of transaminases may be present in two-thirds of the cases. Anemia and elevated erythrocyte sedimentation rate (ESR) are often seen. Hepatic TB abscesses may represent decreased host immunity to tubercle bacilli, resulting in caseous necrosis. In miliary TB, the mode of spread to the liver is via the hepatic artery with tubercles seen near the hepatic veins. In localized forms of hepatic TB, the mode of spread appears to be via the portal vein.^{2,12-14} CT findings may include single or multiple nodules with hypodensity, miliary nodules, and calcifications. Magnetic resonance imaging is also helpful.¹⁵ Biopsy of these lesions may show granulomas. PCR testing of the tissue can be helpful in making the diagnosis. Tissue culture provides bacteriological confirmation.^{12-14,16}

Pancreatic involvement may present with findings suggestive of acute or chronic pancreatitis.^{17,18} The most common symptoms include abdominal pain (75%), anorexia and weight loss (69%), malaise and weakness (64%), fever and night sweats (50%), back pain (38%), and jaundice (31%). US imaging showed enlargement of the head of the pancreas in 12

of 16 patients. CT showed pancreatic mass with hypodensity in all patients and peripancreatic nodules in 38% of cases. Diagnosis was confirmed in all cases either by the presence of granulomas or by PCR positivity of tissue. Rarely, pancreatic TB can occur without other organ involvement. Response to chemotherapy and resolution of abnormality are usually seen.¹⁸

Tuberculous Peritonitis

Tuberculous infection of the peritoneum is rare in developed countries but not infrequent in countries with a high prevalence of TB. It is commonly seen in individuals less than 40 years of age. Tuberculous peritonitis often exhibits female predominance. Individuals with HIV infection, cirrhosis, diabetes, malignancy, and those receiving continuous ambulatory peritoneal dialysis are at high risk for tuberculous peritonitis.¹⁹⁻²² Pathogenesis usually involves peritoneal infection via hematogenous spread or direct extension from an intestinal site or pelvic organ. Both visceral and parietal peritoneal layers are affected with the formation of multiple tuberculous nodules and ascites. The clinical presentation is that of a slowly progressive abdominal swelling from ascites and abdominal pain. Constitutional symptoms of fever and night sweats may be present. Small-bowel obstruction can occur due to adhesions. Diffuse abdominal tenderness, doughy abdomen, hepatomegaly, and ascites may be noted on physical examination. Tuberculin skin tests are positive in two-thirds of cases. Diagnosis is often delayed due to nonspecific symptoms and physical findings.²⁰⁻²²

CT features of peritoneal TB include peritoneal thickening, ascites with fine septations, and omental caking.^{5,6,23} Ultrasonography is helpful in appreciating the loculations and stranding in ascitic fluid.^{5,6} Analysis of ascitic fluid often shows lymphocytic predominance with a serum-to-ascites albumin gradient of <1.1 g/dL.^{19,24} The reported sensitivity of adenosine deaminase activity of tuberculous ascitic fluid varies.^{25,26} In noncirrhotic patients, adenosine deaminase activity (ADA) of >33 U/L in ascitic fluid is shown to have a sensitivity of 97% and specificity of 100% in TB peritonitis.^{27,28} The yield of *Mycobacterium tuberculosis* on smear and culture of peritoneal fluid is low and larger amounts of fluid on centrifugation are required to increase the yield. In HIV patients with tuberculous peritonitis, ADA levels may be low. A high interferon- γ level has been reported in TB peritonitis but not recommended for routine evaluation because of its cost.²⁹

The smear and culture of ascitic fluid have low diagnostic yield. A peritoneal biopsy should be done via laparoscopy or laparotomy to

minimize any possible diagnostic delay. Thickened peritoneum, studding of the peritoneum with multiple tubercles, and adhesions are often seen on laparoscopy or laparotomy. Biopsy of these tubercles shows granulomatous changes.³⁰⁻³³ PCR testing of the biopsy tissue and culture allows rapid diagnosis of tuberculous peritonitis.³⁴ Microbiological confirmation and/or histological appearance of granulomas, with or without caseation, establishes the diagnosis. Individuals with underlying liver disease, HIV, malignancy, or other risk factors usually have higher mortality.^{20,25,30}

Treatment

The recommended treatment for gastrointestinal, hepatic, and pancreatic tuberculosis is conventional antituberculous therapy for a minimum of 6 months.³⁵ (The reader is referred to the article on management of tuberculosis for details.) Addition of corticosteroids is controversial. Complications of abdominal TB depend on the site of involvement. They include ulcer, perforation, adhesion, obstruction, bleeding, fistulae formation, and stenosis. Patients may require surgical therapy, based on clinical presentations, to relieve obstruction or repair perforations/strictures.

In summary, the signs and symptoms of abdominal TB are nonspecific. Delays in diagnosis often result in an increase in complications and mortality. In the evaluation of abdominal tuberculosis, CT and US are helpful. Endoscopic and laparoscopic visualization along with biopsy can increase diagnostic yield. Prompt diagnosis and treatment can minimize morbidity and mortality.

REFERENCES

1. Marshall JB. Tuberculosis of the gastrointestinal tract and peritoneum. *Am J Gastroenterol* 1993;88:989-99.
2. Bernhard JS, Bhatia G, Knauer CM. Gastrointestinal tuberculosis: an eighteen-patient experience and review. *J Clin Gastroenterol* 2000;30:397-402.
3. Sharma MP, Bhatia V. Abdominal tuberculosis. *Indian J Med Res* 2004; 120:305-15.
4. Jakubowski A, Elwood RK, Enarson DA. Clinical features of abdominal tuberculosis. *J Infect Dis* 1988;158:687-92.
5. Suri S, Gupta S, Suri R. Computed tomography in abdominal tuberculosis. *Br J Radiol* 1999;72:92-8.
6. Akhan O, Pringot J. Imaging of abdominal tuberculosis. *Eur Radiol* 2002;12: 312-23.
7. Malik A, Saxena NC. Ultrasound in abdominal tuberculosis. *Abdom Imaging* 2003;28:574-9.
8. Balthazar EJ, Gordon R, Hulnick D. Ileocecal tuberculosis: CT and radiographic evaluation. *AJR Am J Roentgenol* 1990;154:499-503.

9. Uzunkoy A, Harma M, Harma M. Diagnosis of abdominal tuberculosis: experience from 11 cases and review of the literature. *World J Gastroenterol* 2004;10:3647-9.
10. Alvares JF, Devarbhavi H, Makhija P, et al. Clinical, colonoscopic, and histologic profile of colonic tuberculosis in a tertiary hospital. *Endoscopy* 2005;37:351-6.
11. Akgun Y. Intestinal and peritoneal tuberculosis: changing trends over 10 years and a review of 80 patients. *Can J Surg* 2005;48:131-7.
12. Oliva A, Durate B, Jonasson O, et al. The nodular form of local hepatic tuberculosis. A review. *J Clin Gastroenterol* 1990;12:166-73.
13. Essop AR, Posen JA, Hodkinson JH, et al. Tuberculosis hepatitis: a clinical review of 96 cases. *Q J Med* 1984;212:465-77.
14. Huang HT, Wang CC, Chen WJ, et al. The nodular form of hepatic tuberculosis: a review with five additional new cases. *J Clin Pathol* 2003;56:835-9.
15. Yu RS, Zhang SZ, Wu JJ, et al. Imaging diagnosis of 12 patients with hepatic tuberculosis. *World J Gastroenterol* 2004;10:1639-42.
16. 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;127:359-63.
17. Franco-Paredes C, Leonard M, Jurado R, et al. Tuberculosis of the pancreas: report of and review of the literature. *Am J Med Sci* 2002;323:54-8.
18. Xia F, Poon RTP, Wang SG, et al. Tuberculosis of pancreas and peripancreatic lymph nodes in immunocompetent patients: experience from China. *World J Gastroenterol* 2003;9:1361-4.
19. Talwani R, Horvath JA. Tuberculous peritonitis in patients undergoing continuous ambulatory peritoneal dialysis: case report and review. *Clin Infect Dis* 2000;31:70-5.
20. Sanai FM, Bzeizi KI. Systematic review: tuberculous peritonitis-presenting features, diagnostic strategies and treatment. *Aliment Pharmacol Ther* 2005;22:685-700.
21. Wang HK, Hsueh PR, Hung CC, et al. Tuberculous peritonitis: analysis of 35 cases. *J Microbiol Immunol Infect* 1998;31:113-8.
22. Bastani B, Shariatzadeh MR, Dehdashti F. Tuberculous peritonitis—report of 30 cases and review of the literature. *Q J Med* 1985;56:549-57.
23. Vasquez Munoz E, Gomez-Cerezo J, Atienza Saura M, et al. Computed tomography findings of peritoneal tuberculosis: systematic review of seven patients diagnosed in 6 years (1996–2001). *Clin Imaging* 2004;28:340.
24. Shakil AO, Korula J, Kanel GC, et al. Diagnostic features of tuberculous peritonitis in the absence and presence of chronic liver disease: a case control study. *Am J Med* 1996;100:179-85.
25. Hillebrand DJ, Runyon BA, Yasminah WG, et al. Ascitic fluid adenosine deaminase insensitivity in detecting tuberculosis peritonitis in the United States. *Hepatology* 1996;24:1408-12.
26. Aguado JM, Pons F, Casafont F, et al. Tuberculous peritonitis: a study comparing cirrhotic and noncirrhotic patients. *J Clin Gastroenterol* 1990;12:550-4.
27. Dwivedi M, Misra V, Kumar R. Value of adenosine deaminase in tuberculous ascites. *Am J Gastroenterol* 1990;85:1123-5.
28. Voigt MD, Kalvaria I, Trey C, et al. Diagnostic value of ascites adenosine deaminase in tuberculous peritonitis. *Lancet* 1989;1:751-4.
29. 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;26:484-8.
30. Demir K, Okten A, Kaymakoglu S, et al. Tuberculous peritonitis—report of 26 cases, detailing diagnostic and therapeutic problems. *Eur J Gastroenterol Hepatol* 2001;13:581-5.
 31. 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;35:409-13.
 32. al-Quorain AA, Facharzt, Satti MB, et al. Abdominal tuberculosis in Saudi Arabia: a clinicopathological study of 65 cases. *Am J Gastroenterol* 1993;88:75-9.
 33. Bhargava DK, Shriniwas, Chopra P, et al. Peritoneal tuberculosis: laparoscopic patterns and its diagnostic accuracy. *Am J Gastroenterol* 1992;87:109-12.
 34. Lye WC. Rapid diagnosis of mycobacterium tuberculous peritonitis in two continuous ambulatory peritoneal dialysis patients, using DNA amplification by polymerase chain reaction. *Adv Perit Dial* 2002;18:154-57.
 35. Blumberg HM, Leonard MK Jr, Jasmer RM. Update on the treatment of tuberculosis and latent tuberculosis infection. *JAMA* 2005;293:2776-84.