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# Abdominal Tuberculosis

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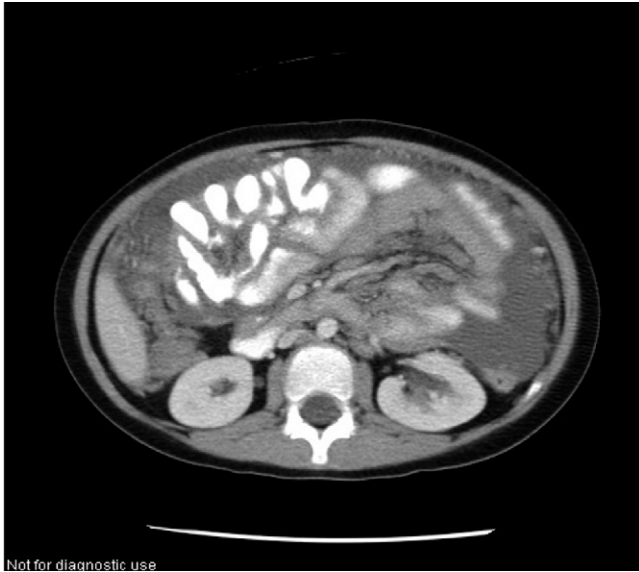
## Introduction

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.<sup>1-4</sup>

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.<sup>1-4</sup>

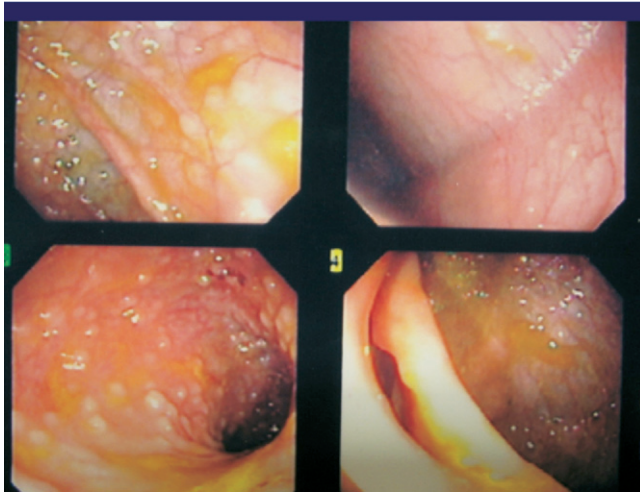
Radiographic imaging such as plain abdominal series, barium enema,

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**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 obstruction<sup>5-7</sup> (Fig 1). Patients with AIDS usually have a more severe form of involvement than those who did not have AIDS.<sup>8</sup> 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.<sup>1,8,9</sup> 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.<sup>10</sup> 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.<sup>11</sup>

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.<sup>2,12-14</sup> CT findings may include single or multiple nodules with hypodensity, miliary nodules, and calcifications. Magnetic resonance imaging is also helpful.<sup>15</sup> Biopsy of these lesions may show granulomas. PCR testing of the tissue can be helpful in making the diagnosis. Tissue culture provides bacteriological confirmation.<sup>12-14,16</sup>

Pancreatic involvement may present with findings suggestive of acute or chronic pancreatitis.<sup>17,18</sup> 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.<sup>18</sup>

## 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.<sup>19-22</sup> 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.<sup>20-22</sup>

CT features of peritoneal TB include peritoneal thickening, ascites with fine septations, and omental caking.<sup>5,6,23</sup> Ultrasonography is helpful in appreciating the loculations and stranding in ascitic fluid.<sup>5,6</sup> Analysis of ascitic fluid often shows lymphocytic predominance with a serum-to-ascites albumin gradient of  $<1.1$  g/dL.<sup>19,24</sup> The reported sensitivity of adenosine deaminase activity of tuberculous ascitic fluid varies.<sup>25,26</sup> 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.<sup>27,28</sup> 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- $\gamma$  level has been reported in TB peritonitis but not recommended for routine evaluation because of its cost.<sup>29</sup>

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.<sup>30-33</sup> PCR testing of the biopsy tissue and culture allows rapid diagnosis of tuberculous peritonitis.<sup>34</sup> 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.<sup>20,25,30</sup>

## Treatment

The recommended treatment for gastrointestinal, hepatic, and pancreatic tuberculosis is conventional antituberculous therapy for a minimum of 6 months.<sup>35</sup> (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.

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