Extrapulmonary tuberculosis, part 3: Abdominal involvement

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Abstract: In addition to causing pulmonary disease, infection with Mycobacterium tuberculosis can result in a wide range of extrapulmonary manifestations, including abdominal involvement. Patients with acute tuberculous peritonitis typically present with fever, weight loss, night sweats, and abdominal pain and swelling. Intestinal tuberculosis is characterized by weight loss, anorexia, and abdominal pain (usually in the right lower quadrant). A palpable abdominal mass may be present. Patients with primary hepatic tuberculosis may have a hard, nodular liver or recurrent jaundice. The workup may involve tuberculin skin testing, imaging studies, fine-needle aspiration, colonoscopy, and peritoneal biopsy. Percutaneous liver biopsy and laparoscopy are the main methods of diagnosing primary hepatic tuberculosis. Treatment includes antituberculosis drug therapy and, in some cases, surgery. (J Respir Dis. 2005;26(11):485-488)

Abdominal tuberculosis is one of the most common forms of extrapulmonary tuberculosis. It includes tuberculous infection of the GI tract; peritoneum; omentum; mesentery and its nodes; and other solid intra-abdominal organs, such as the liver, spleen, and pancreas.

This infection is typically caused by Mycobacterium tuberculosis. Abdominal infection caused by Mycobacterium bovis is rarely seen in the present era because of stringent pasteurization of milk and control of tuberculosis on dairy farms.

In the August and September 2005 issues of The Journal of Respiratory Diseases, we reviewed pleural and lymph node involvement and CNS involvement, respectively. In this article, we will focus on the presentation of abdominal manifestations of tuberculosis.

PERITONITIS
Tuberculous peritonitis constitutes 4% to 10% of all cases of extrapulmonary tuberculosis.1 The reactivation of long-standing latent tuberculous infection of the peritoneum or hematogenous spread of bacilli from an active pulmonary lesion results in peritonitis. The contiguous spread of infection from an intestinal lesion or fallopian tube is relatively infrequent.

Acute tuberculous peritonitis has an onset resembling that of acute abdomen. Laparotomy may reveal straw-colored fluid, with tubercles scattered over the peritoneum and greater omentum.1 Chronic peritonitis occurs in patients in the third to fifth decade of life, with a slight preponderance in women. In addition to constitutional features (fever, weight loss, and night sweats), abdominal pain and swelling are the most common presenting symptoms.

There are 3 forms of chronic tuberculous peritonitis: ascitic, encysted (loculated), and fibrous. The ascitic form often has an insidious onset, with constitutional features and abdominal distention. Abdominal pain is usually absent. The rolled-up greater omentum infiltrated with tubercles may be felt as a transverse solid mass in the patient's abdomen.

The clinical presentation of the encysted form of chronic peritonitis resembles that of the ascitic form. Patients often present with localized abdominal swelling.

In the fibrous form, widespread adhesions may cause the intestine (especially in the ileal region) to be matted together and distended. The disease frequently presents as acute or subacute intestinal obstruction, especially in developing countries such as India.

The matted intestine may also act as a “blind loop” leading to the development of steatorrhea, malabsorption syndrome, and abdominal pain. On physical examination, the adherent loops of intestine and the thickened mesentery may be felt as a lump in the abdomen.1

GI TUBERCULOSIS
Although GI tuberculosis is rare in industrialized countries, it is common in developing countries. In India, tuberculosis has been reported to be the cause of intestinal obstruction in 3% to 20% of patients. About 5% to 7% of all GI perforations (excluding appendiceal perforations) are caused by tuberculosis.2

Pathogenesis and pathology
M tuberculosis often reaches the abdomen as a result of a person's swallowing sputum containing the bacilli or by hematogenous dissemination from active pulmonary tuberculosis or miliary tuberculosis or by local spread from infected adjacent viscera. Any region of the GI tract, from oral cavity to anus, can be affected.
The striking predilection for the ileocecal region is the result of the abundance of lymphoid tissue (Peyer patches) there. In the ileocecal region, there generally is increased physiologic stasis, increased rate of fluid and electrolyte absorption, and minimal digestive activity. The greater contact time between the organism and the mucosal surface renders the area more vulnerable to the development of intestinal tuberculosis.

GI tuberculosis can manifest as ulcerative, hypertrophic, ulcerohypertrophic, or diffuse colitis. The ulcerative form usually occurs in malnourished persons. Tuberculous ulcers may be solitary or multiple and usually lie transverse to the long axis of the gut ("girdle ulcers"). The healing and fibrosis result in stricture formation ("napkin ring strictures"), which leads to obstructive symptoms. "Skip lesions" of normal mucosa are also observed in the diseased segment. Adhesions between the bowel loops prevent free perforation but promote formation of intestinal fistulae.

Unlike the ulcerative form, hypertrophic intestinal tuberculosis commonly occurs in relatively well-nourished patients. The cecum is the most commonly affected site. The hypertrophic form is caused by infection with a less virulent organism in a host with good resistance and wound-healing capacity.

Caseation is often seen in the mesenteric lymph nodes but is occasionally absent in tuberculous granulomas. These noncaseating granulomas may be difficult to distinguish from the findings in Crohn disease. The important findings supporting the diagnosis are confluence of the granulomas, relative absence of fissures, and submucosal edema.

Diffuse colitis is a rare form of intestinal tuberculosis that cannot be easily distinguished from ulcerative colitis on the basis of mucosal appearance on endoscopy. Clinical presentation

Intestinal tuberculosis is a chronic illness with protean manifestations, including weight loss and anorexia. Patients are most often aged 20 to 40 years, and the disease more commonly affects women.

Abdominal pain is the most common symptom and is present in almost all patients. The pain is most commonly located in the right lower quadrant of the abdomen, although a significant proportion of patients may complain of diffuse, central, epigastric, or left lower quadrant pain. The pain is spasmodic, particularly in patients with intestinal obstruction.

Diarrhea and/or constipation occurs in fewer than 20% of patients. Fever has been reported in 40% to 70%. Menstrual abnormalities have been described in nearly one third of female patients. Tuberculous duodenitis may produce distress that mimics duodenal ulcer disease. Tuberculous appendicitis mimics acute appendicitis. Incidental tuberculous involvement of the vermiform appendix is fairly common in patients who have active ileocecal tuberculosis.

The abdominal examination reveals tenderness, most frequently in the right iliac fossa. A palpable abdominal mass may be present. This mass results from hyperplastic cecal tuberculosis, lymph node enlargement, and/or rolled-up omentum. The classic "doughy abdomen" has been described in fewer than 10% of patients.

SPLENIC TUBERCULOSIS

This condition presents as hypersplenism or splenic abscess. Fever, left upper quadrant abdominal pain, weight loss, and diarrhea are common features. Multiple tuberculous abscesses have been described in patients with HIV infection.

The preoperative diagnosis of splenic tuberculosis is difficult. The diagnosis is often established by histopathologic examination of the excised specimen. Hepatobiliary tuberculosis can occur in several ways. Hepatic involvement is very common in congenital tuberculosis. Congenital tuberculosis should be considered in the differential diagnosis for an infant who has hepatomegaly, jaundice, and failure to thrive and whose mother has active tuberculosis. Primary hepatic tuberculosis is the involvement of the hepatobiliary tract by tuberculosis without apparent disease elsewhere. It is more common in males, and most patients are in the first 3 decades of life. Patients are symptomatic for about 1 to 2 years before the diagnosis is established.

Primary hepatic tuberculosis is the involvement of the hepatobiliary tract by tuberculosis without apparent disease elsewhere. It is more common in males, and most patients are in the first 3 decades of life. Patients are symptomatic for about 1 to 2 years before the diagnosis is established. The 2 major forms of clinical presentation are fever and weight loss with a hard, nodular liver mimicking malignancy, and recurrent jaundice mimicking extrahepatic obstruction.

ABDOMINAL TUBERCULOSIS AND HIV

Abdominal tuberculosis in patients with HIV infection is almost invariably a manifestation of disseminated disease, and it causes significant mortality. Fever, weight loss, and intra-abdominal lymphadenopathy are more common in patients who have HIV infection, whereas ascites and omental thickening are more frequent in HIV-negative patients. Diagnostic tests

In patients with abdominal tuberculosis, laboratory studies generally show nonspecific findings, such
as anemia, leukopenia with relative lymphocytosis, and elevated erythrocyte sedimentation rate. The serum transaminase levels are usually normal, although the serum alkaline phosphatase level may be elevated. A positive tuberculin test result in an appropriate clinical setting supports the diagnosis.

**Imaging studies**

A plain radiograph of the abdomen may show calcified lymph nodes or calcified granulomas in the spleen, liver, or pancreas. Other radiographic features include dilated loops with fluid levels and dilatation of the terminal ileum. The pneumoperitoneum may be evident in patients who have intestinal perforation.

Barium contrast study is valuable in the evaluation of intestinal tuberculosis. Enteroclysis followed by barium enema is the best protocol. Ultrasonography often reveals a mass consisting of matted small-bowel loops with thickened walls, diseased omentum, mesentery, loculated ascites, and lymphadenopathy. Fine septae may be seen in the ascitic fluid.

These strands usually arise from the serosa of the small bowel and are caused by the high fibrin content of the exudative ascitic fluid. They are considered to be diagnostic of abdominal tuberculosis.

Loculated ascites represent concealed peritoneal inflammation. An abdominal CT scan is better than an ultrasonogram for detecting high-density ascites. In addition, mesenteric infiltration, omental masses, peritoneal enhancement or thickening, or disorganized masses of soft tissue densities may be seen. Retroperitoneal, peripancreatic, porta hepatitis, and mesenteric/ omental lymph node enlargement may also be evident. An example of CT findings in a patient with tuberculous peritonitis is shown in the Figure.

Ultrasonographic or CT-guided fine-needle aspiration of palpable masses with culture and cytology has a high diagnostic yield.

**Ascitic fluid examination**

The ascitic fluid is exudative (protein level greater than 2.5 g/dL), and the serum ascitic fluid albumin gradient is less than 1.1 g/dL in more than 90% of patients. Ascitic fluid white blood cell count is usually 150 to 4000/µL and consists primarily of lymphocytes. However, a neutrophilic response may also be observed. The ascitic fluid culture for *M tuberculosis* is positive in fewer than 20% of patients. Adenosine deaminase (ADA) activity in ascitic fluid is elevated in peritoneal tuberculosis. The specificity and sensitivity of ADA activity are 95% and 98%, respectively, when the cutoff value is 32 IU/L. In patients who have both HIV infection and tuberculous peritonitis, ADA levels may be lower. False-positive ADA results have been reported in patients with malignant ascites. The level of interferon gamma in ascitic fluid is significantly higher in persons with tuberculous peritonitis than it is in those with malignancy or cirrhosis.

**Endoscopy**

Colonoscopy with biopsy is a direct method for establishing the diagnosis of tuberculous colitis. On colonoscopy, the ileocecal valve may be edematous or deformed, and nodules, ulcers, pseudopolypoid folds, or strictures may be seen. Ulceration is the most common finding and is observed in the ileocecal region. However, endoscopic findings are not pathognomonic of tuberculosis.

**Laparoscopy and biopsy**

Direct inspection, with biopsy of the peritoneum, is probably the most effective method of diagnosing tuberculous peritonitis. However, it is an invasive procedure with potential morbidity and mortality.

Percutaneous liver biopsy and laparoscopy are the main methods for confirming the diagnosis of hepatobiliary tuberculosis.**TREATMENT**

The management of abdominal tuberculosis includes medical treatment and conservative or radical surgery when appropriate. Conventionally, patients have been treated with antituberculosis drug regimens of 6 to 12 months' duration, depending on the clinical response (Table). Most patients with hepatobiliary tuberculosis respond to antituberculosis treatment. Those with obstructive jaundice usually require biliary decompression.

Stricturoplasty is recommended for obstructive lesions, since bypass surgery (enteroenterostomy, ileotransverse colostomy) may result in blind loop syndrome. Acute intestinal obstruction, perforation, and peritonitis are treated conservatively, although surgery may have to be performed when conservative treatment fails. The surgical risk is very high in these patients.

**References: REFERENCES**


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