Intestinal Tuberculosis and Secondary Liver Abscess

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Abstract

The incidence of intestinal tuberculosis (ITB) has been increasing in the West, due to the AIDS epidemic, transglobal immigration, IV drug abuse, an aging population, and an increase in the number of immunocompromised patients. Obstruction and perforation of the intestine are the most common and serious complications of ITB. Another complication, tuberculous liver abscess (TLA), is rare and usually associated with foci of infection in the lung or gastrointestinal tract. We report a case of a 17-year-old boy with Down syndrome who presented with multiple TLAs secondary to obstructive and multiple perforated ileal tuberculosis.

Key Words: Intestinal tuberculosis, tuberculous liver abscesses, surgical treatment.

Introduction

ABDOMINAL TUBERCULOSIS (TB) is still a major problem in many regions of the world, especially in developing countries. And the incidence of ITB has been increasing in the West, due to the AIDS epidemic, transglobal immigration, IV drug abuse, an aging population, and an increase in the number of immunocompromised patients (1–3). In the abdomen, tuberculosis may affect the intestinal tract, lymph nodes, peritoneum and solid viscera (4). The lack of specific signs and symptoms of abdominal tuberculosis involving the peritoneum or intestinal tract frequently leads to missed or delayed diagnoses such as Crohn's disease, lymphoma, carcinoma, and periappendiceal abscess (5, 6). Obstruction, perforation, fistula formation, intestinal bleeding, traction diverticula and venous thrombosis are abdominal complications of ITB (7). Obstruction is the most common complication of ITB; it occurs in 12–60% of cases (7, 8). TLA is rare and usually associated with foci of infection in the lung or gastrointestinal tract (9). The bacilli reach the liver via the hepatic artery or the portal vein (10). Levine (11) classified hepatic tuberculosis as miliary tuberculosis, pulmonary tuberculosis with liver involvement, primary liver tuberculosis, tuberculoma, and tuberculous cholangitis.

We report a case of a 17-year-old boy patient with multiple TLAs secondary to obstructive and multiple perforated ileal tuberculosis.

Case Report

A 17-year-old boy with Down syndrome was admitted with a 6-week history of mild abdominal pain, fever, diarrhea, abdominal distension and 15 kg weight loss. The patient had been treated for ITB some 12 years earlier. On physical examination he was febrile (38°C) and tachycardic, and had a distended abdomen and a soft mass, about 10 cm diameter, in the middle abdomen. There were no signs of peritoneal irritation, and the liver, spleen and lymph nodes were not enlarged. Laboratory tests showed hypochromic anemia (hemoglobin 7.1 g/dL), normal white blood cells (8,000/mm^3), serum bilirubin 3.4 g/dL, alkaline phosphatase 340 IU, aspartate aminotransferase 42 U/L, alanine aminotransferase 59 U/L, high erythrocyte sedimentation rate (120 mm/h), and high

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CRP rate (15.9 mg/dL, upper limit 0.5 mg/dL). Chest x-ray showed no abnormalities, but CT scan of the abdomen showed irregular thickening of the small bowel and nonblocking mass in the abdomen (Fig. 1). The patient received metronidazole for diarrhea. After 5 days of follow-up he had acute abdominal and obstructive symptoms and his white blood count was 19,100/mm^3.

The patient had exploratory surgery, at which a mass in the middle abdomen was discovered. This mass consisted of a perforated ileum segment which was enclosed within the adjacent ileum segments. The perforated segment of the ileum was resected, and an end ileostomy was performed because of edema, dilatation and severe peritonitis. Although the patient appeared well on the first and second postoperative days, he began to have fever on postoperative day 3; on postoperative day 4 it reached 39°C. In searching for the source of the fever, blood, sputum, urine and stool cultures were taken and direct microscopies of urine, stool, and sputum were also examined. All of them were negative. We also took a chest radiogram, which was normal. Abdominal ultrasound (US) was done on postoperative day 6. It showed multiple cystic lesions, approximately 1 mm in diameter, in the liver. These were considered microabscesses. Abdominal magnetic resonance imaging (MRI) revealed multiple hypointense lesions diffusely spread in the liver (Fig. 2). Meanwhile, the pathological examination of the resected ileum revealed intestinal tuberculosis (Fig. 3).

We did a US-guided percutaneous aspiration of the cystic lesions for further diagnosis and discrimination between bacterial and tuberculous liver abscess. The aspirated material was cultured, underwent direct microscopic examination, and was sent for polymerase chain reaction (PCR) for *Mycobacterium tuberculosis* DNA. Culture and microscopical examination were negative but PCR for *M. tuberculosis* DNA was positive. The lesions were diagnosed as tuberculous microabscesses, and an anti-TB treatment for both intestinal tuberculosis and tuberculous liver abscess was ordered. The treatment consisted of four drugs (isoniaside 300 mg orally daily, pyrasinamide 2,000 mg orally daily, ethambutol 1,500 mg orally daily, rifampicin 600 mg orally daily) for a two-month period and two drugs (isoniaside, rifampicin) for the rest of the therapy period (10 months). The patient was

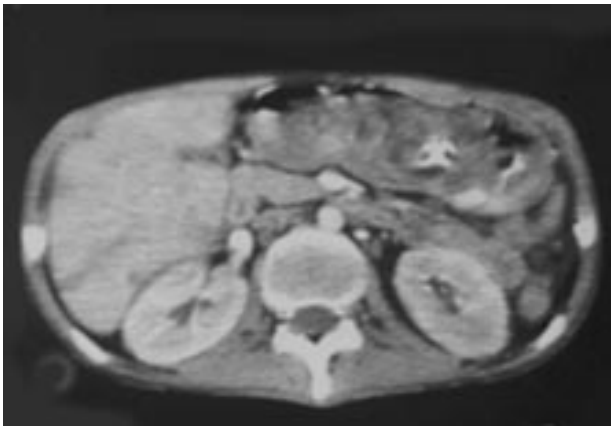

Fig 1. Contrast enhanced axial CT image through the upper abdomen shows concentric intestinal wall thickened and nodular, irregular infiltration of the omentum on the left epigastric region.

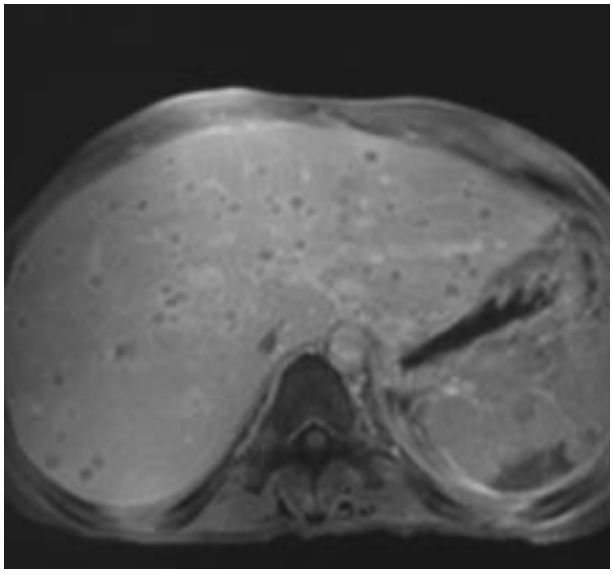

Fig 2. Post-contrast fat-saturation axial T1-weighted image through the upper abdomen shows well-defined, ring-contrast-enhanced, uniform multiple liver lesions.

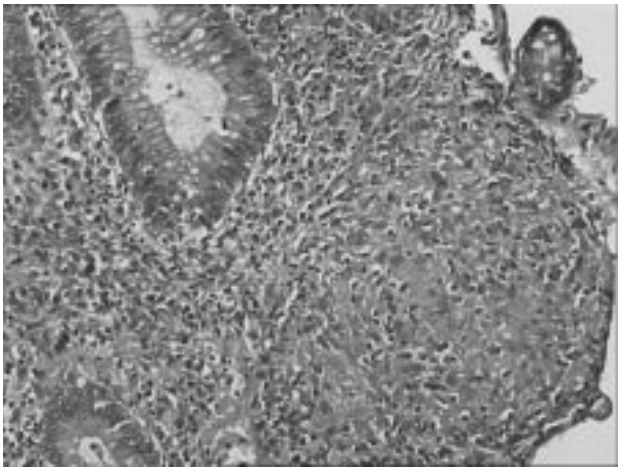

Fig 3. This section shows an intestinal mucosa and a granuloma; a round nodule with a large Langhans giant cell in the center surrounded by fibroblasts; and occasional lymphocytes.

discharged from hospital on postoperative day 15 without any other complications. The ileostomy was closed two months after the operation. An abdominal MRI was performed after the end of anti-TB therapy; there were no lesions visible on the liver (Fig. 4).

Discussion

TB can involve any part of the gastrointestinal tract, from mouth to anus, including the peritoneum and hepatopancreatobiliary system. ITB is predominantly a disease of young adults and the sex incidence is equal (12). Approximately 75% of patients with ITB have involvement of the distal small bowel and ileocecal region, possibly because of the increased physiological stasis, increased fluid and electrolyte absorption, minimal digestive activity and abundance of lymphoid tissue in this region (12–14).

It is often difficult to confirm the diagnosis of primary ITB, especially in emergency situations (15). Diagnosis of ITB is based on a comprehensive approach, including patient history as well as clinical lab tests and radiological findings (6). The most common symptoms of ITB are abdominal pain, diarrhea, weight loss, anorexia, fever, anemia, and lower gastrointestinal bleeding. Abdominal tenderness and distension and/or palpable abdominal mass are also relatively common physical findings (13–15). Many of these signs and symptoms were present in our patient.

Serum laboratory tests are usually nonspecific (15). Definitive diagnosis is essentially made by histology; Ziehl-Neelsen staining for acid-fast *bacilli* and culture can establish the diagnosis for up to 80% of the patients (16, 17). PCR of aspirated or biopsy specimens may facilitate diagnosis with higher sensitivity, and specificity and speed than routine culture, as in our case (15).

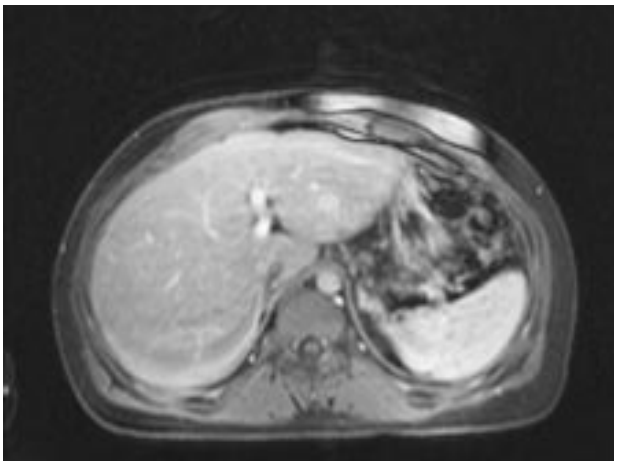

Fig 4. Post-contrast fat-saturation axial T1-weighted image through the upper abdomen appears normal.

While US and computed tomography (CT) show extramucosal changes directly, barium studies show mucosal pathology and complications (2, 18). CT also makes an important contribution in evaluating most of the complications of ITB, such as small bowel obstruction, perforation, and abscess formation (2).

The lack of specific signs and symptoms of abdominal tuberculosis involving the peritoneum or intestinal tract frequently leads to missed, delayed or erroneous diagnoses such as Crohn's disease, lymphoma, carcinoma, or periappendiceal abscess (5, 6).

Obstruction, perforation, fistula formation, intestinal bleeding, traction diverticula and venous thrombosis are reported complications of ITB (1, 7, 8, 13). Obstruction is the most common complication of ITB, occuring in 12–60 % of cases (7, 8). Though perforation is less common (1–15%) than obstruction, it is a serious complication (1, 7, 8). Multiple perforations are reported in 25–40% of cases (1, 8). Perforation can also be confined to a localized area, presumably due to the preexisting adhesive change, hypertrophied intestinal wall, and mesentery, as in our case (7, 8). The overall mortality of perforation has ranged from 25–45% (1).

Although most patients with ITB respond favorably to medical therapy, surgery should be considered for complications such as obstruction, free perforation, confined perforation with abscess, fistula and massive bleeding, or diagnostic uncertainty (1, 15, 19). The most effective surgical treatment of complicated ITB is resection, with 5 cm margins from macroscopically diseased tissue and end-to-end anastomosis. Simple closure of perforations is not recommended because of the high incidence of leak and fistula formation (1, 15). We did not perform end-to-end anastomosis because of edema, dilatation and severe peritonitis, and diagnostic uncertainty.

TLAs are rare and usually associated with foci of infection in the lung or gastrointestinal tract (9). The bacilli reach the liver via the hepatic artery or the portal vein (10). The prevalence of TLAs is 0.34% in patients with hepatic TB (20). TLAs commonly present with fever of unknown origin, anorexia, weight loss, and vague abdominal pain (10). Hepatomegaly is a common physical finding (21). Hypoproteinemia, elevated serum alkaline phosphates and mild hyperbilirubinemia are characteristic biochemical features (22).

The radiological findings of TLAs have a low specificity. Therefore, microbiological or pathological examination of specimens is needed to

make a diagnosis and to distinguish from pyogenic and amoebic abscess or neoplasm of the liver (11, 22, 23). Rahmatulla et al. reported that the diagnosis of TLAs is usually made by laparotomy, due to the nondetection of percutaneous mycobacteria in the percutaneous liver aspirate (10). Recently PCR assay was demonstrated to be useful in the diagnosis of hepatic tuberculosis liver biopsy or aspirate, as in our case. The diagnosis can be confirmed by the patient's response to anti-TB drug therapy (11, 22).

Quadruple therapy with anti-TBC drugs for 1 year is recommended. Percutaneous drainage of the abscess, combined with systemic anti-TB chemotherapy, has been used in appropriate cases as an alternative to surgery (10, 22). Mustard et al. (24) treated TLAs by percutaneous drainage combined with trans-catheter infusion of anti-TBC drugs. Surgery is best reserved for multiple large lesions and lesions refractory to medical treatment (22).

In summary, ITB should be kept in mind as a cause of intestinal obstruction and perforation, especially in endemic areas. And TLAs should be thought of in the differential diagnosis of liver abscess, especially if there is primary TB in the gastrointestinal tract.

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