Case Report

Gastrointestinal Tuberculosis with Cecum Involvement in a 33-Year-Old Woman

Davood Yadegarynia[1], Farhad Abbasi[1],
Maryam Keshtkar-Jahromi[1],
Sharareh Gholamin[1]

Abstract

Abdominal tuberculosis is one of the most prevalent forms of extra-pulmonary tuberculosis. Various regions of gastrointestinal tract including cecum, terminal ileum, peritoneum, lymphatic system, and solid viscera can be affected by tuberculosis. Here we report a 33-year-old woman presented with fever, chills, and a history of abdominal discomfort. Lymphadenopathy was detected on physical examination. Contrast computed tomography of chest and abdomen showed patchy densities and thickening of the ileocecal wall respectively. Histological studies of the biopsy samples documented the existence of tuberculosis.
Iran J Med Sci 2009; 34(3): 213-216.

Keywords ● Tuberculosis ● extra-pulmonary ● abdominal

Introduction

Tuberculosis (TB) has plagued the human beings since ancient times. Despite advances in medicine, TB is still a major problem in developing and some developed countries. Difficulty in controlling the disease is attributed to factors such as transglobal migration, increasing population age, socioeconomic deprivations, and the acquired immunodeficiency syndrome (AIDS).

As long as pulmonary TB is a problem, extra-pulmonary TB is also a health problem.[1] In order of frequency, the extrapulmonary sites most commonly involved by TB are the lymph nodes, pleura, genitourinary tract, bones and joints, meninges, peritoneum, and pericardium. However, virtually all organ systems may be affected.[2] Extra-pulmonary TB can be classified based on the pathogenesis into three groups. The first group comprises superficial mucosal foci resulting from the spread of infectious pulmonary secretions via the respiratory and gastrointestinal tracts. Such lesions were once almost inevitable complications of extensive cavitary pulmonary disease but are now rare.

The second group comprises foci established by contiguous spread, such as from a subpleural focus into the pleural space. The third group comprises foci established by lymphohematogenous dissemination, either at the time of primary infection or, less commonly, from established chronic pulmonary or extrapulmonary foci.[3] Failure of pulmonary tubercular disease control resulted in increasing number of extra-pulmonary tuberculosis, such as abdominal tuberculosis.[1]

Abdominal TB can affect the gastrointestinal tract, the peritoneum, lymph nodes of the small bowel mesentery, or the solid viscera including the liver, spleen, and pancreas. The gastrointestinal tract is involved in 66-75% of patients with

[1]Infectious Diseases and Tropical Medicine Research Center, Shaheed Beheshti University of Medical Sciences, Tehran, Iran.

Correspondence:
Davood Yadegarynia MD.
Infectious Diseases and Tropical Medicine Research Center, Shaheed Beheshti University of Medical Sciences, Tehran, Iran.
Tel: +98 21 22439963
Fax: +98 21 22439964
Email: yadegarinia@yahoo.com

abdominal TB. The terminal ileum and the ileo-cecal region are the most common sites, followed by the jejunum and colon.[4]

Case Presentation

A 33-year-old woman presented with a history of 4-month intermittent fever and chills. There was a history of abdominal discomfort and pain of 3 weeks duration since a month ago. She also complained of weight loss during the last month. She had not been exposed to any ill people.

Physical examination of head and neck, heart, lungs, extremities, and nervous system were normal. Apart from axillary lymphadenopathy, nothing was remarkable on physical examination. No hepatosplenomegaly was found.

In initial evaluation, erythrocyte sedimentation rate of 72 mm/hr and tuberculin skin test with induration exceeding 20 mm were detected. The chest radiograph was normal but spiral computed tomography (CT) of thorax with and without contrast showed bilateral peripheral patchy densities of upper lobes with focal pleural thickening of the right side (figure 1).
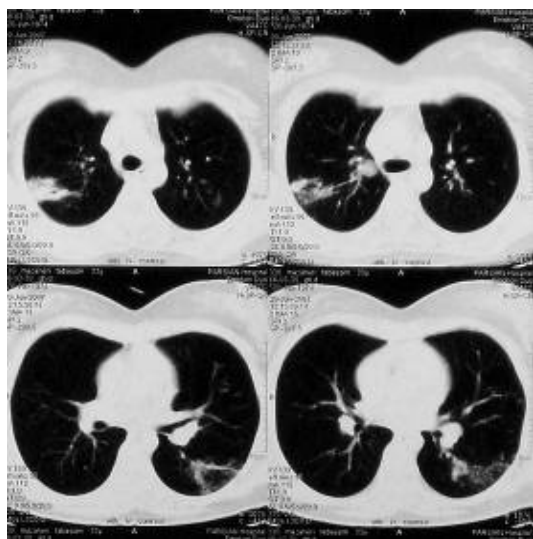

Figure 1: Bilateral peripheral patchy densities of upper lobes

Bronchoalveolar lavage was done for detection of malignancy. Also the specimen taken on the lavage was sent for detection of mycobacterium tuberculosis through acid fast bacilli staining and PCR technique. The report was negative in both studies. Abdominal CT revealed marked thickening of cecal wall and terminal ileum with irregular lumen surface. Other parts of colon, liver, spleen, kidneys and other organs were normal (figure 2).
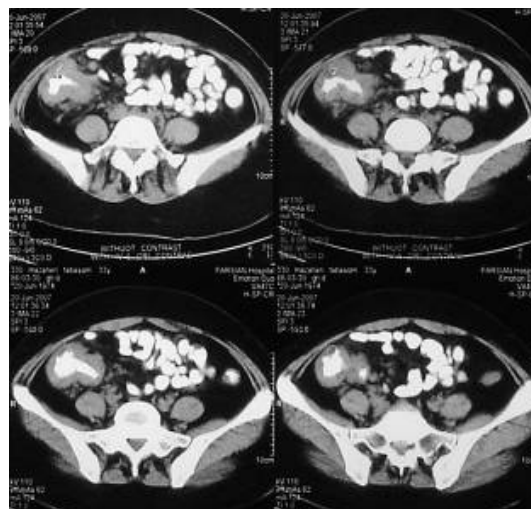Since inflammatory process was detected in abdominal evaluation and the patient had a

Figure 2: Marked thickening of cecal wall with irregular lumen surface

history of abdominal discomfort, lymphoma with abdominal source was suspected. Colonoscopic biopsy of cecum demonstrated eight soft pale tan tissue fragments measuring 1.2 × 0.2 × 0.2 cm in gross view whereas microscopic view of the sections revealed ulcerated colonic mucosa with polymorphous leukocytic infiltration and epithelioid granulomas, consistent with tuberculosis. Acid fast staining and PCR for mycobacterium tuberculosis were positive on the biopsy specimen. There was an ulcerovegetan mass in cecum extended to terminal ileum (figure 3).
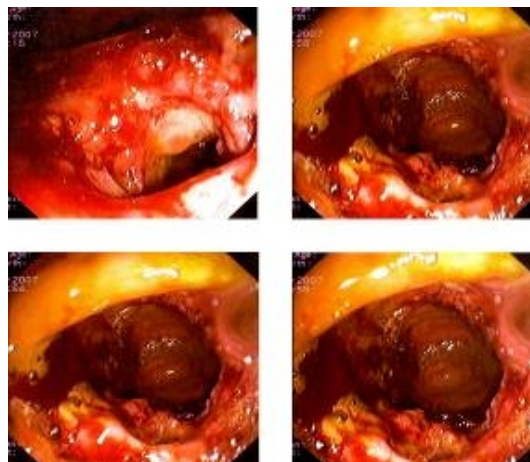

Figure 3: Ulcerovegetan mass is seen in cecum extended to terminal ileum

Axillary lymph node biopsy showed chronic necrotizing granulomatous lymphadenitis, consistent with TB and large areas of granular eosinophilic necrosis with surrounding epithelioid histiocytes and giant cells. Anti-TB drugs isoniazid (300 mg daily), rifampin (600 mg daily),

ethambutol (800 mg daily), and pyrazinamide (1200 mg daily) were administered. After several weeks, abdominal pain and discomfort gradually decreased and finally disappeared. After 6 months, the patient's general condition was good and there was no abdominal pain.

Discussion

Gastrointestinal TB, once considered common, and then a relatively rare disease is now re-emerging in association with AIDS and multi-drug resistant Mycobacterium tuberculosis. Intestinal involvement with TB may be either primary from ingesting of the organism, or secondary usually from a pulmonary source.[5]

Gastrointestinal TB can affect any part of the tract, from the mouth to the anus. The most common site is the ileocecal area. This is probably caused by several factors; I. A massive amount of lymphoid tissue, II. Physiologic stasis causing increased contact time between the bacteria and the intestinal lumen, III. Increased rate of fluid and electrolyte absorption, and IV. Minimal digestive activity, permitting greater contact time.[1]

Other commonly involved sites are the colon and the jejunum. Uncommon involvements of the esophagus, duodenum and small bowel in isolation have also been reported.[6]

Clinical manifestations of gastrointestinal TB are non-specific. The most common complaint is abdominal pain, occurring in approximately 80% of cases.[1] Patients with primary TB may present with abdominal pain, fever, and a tender, fixed palpable mass in the ileocecal area. Weight loss is more common in secondary intestinal tuberculosis. Only one third of the patients with gastrointestinal TB may present with diarrhea.[5] The exact mechanism for diarrhea is unknown but it may be caused by generalized inflammatory response of the intestine and the subsequent effect of the cytokines, leukotrienes, and prostaglandins on fluid and electrolyte transport.[1] Hemorrhage and the presence of gross blood in the stool are distinctly uncommon.[5]

Diagnosis of intestinal TB may be difficult radiologically and even histologically.[5] Radiological and histological manifestations of intestinal TB may resemble other diseases such as Crohn's, lymphoma, or malignancies.[1] It must also be distinguished from regional enteritis, sarcoidosis, actinomycosis, amebiasis, carcinoma, and periappendiceal abscess.[5] In imaging studies, chest radiology is usually normal, but evidence of pulmonary TB in chest radiography or high resolution CT supports the diagnosis. Radiographs of the abdomen are useful in patients with intestinal obstruction and perforation.[4] Lymphadenopathy is a common manifestation of abdominal TB. Mesenteric, omental, and peripancreatic lymph nodes are most commonly involved. Contrast enhanced CT shows peripherally enhancing lymph nodes with low density centers explained by a peripheral inflammatory reaction and central caseous necrosis. This appearance is highly suggestive but not pathognomonic of abdominal TB.[7] In histological study, epithelioid cell granulomata that resembles Crohn's disease makes the diagnosis difficult. However, the epithelioid cell granulomata with the peripheral rim and plasma cells, giant cells and central caseating necrosis, fibrosis, and calcification in healing lesions can be used as histological criteria for making differentiation.[4,8]

The first line of treatment for abdominal TB is medical treatment.[9] More recent reports show that 6 months of treatment is adequate for abdominal tuberculosis. Diagnosis in the early phase results in a good response to medical treatment. Patients usually show signs of improvements as early as 2 weeks after starting the treatment. Even patients with signs of incomplete gut obstruction have shown improvement and cessation of symptoms with medical treatment alone.[1] Surgery for abdominal TB is reserved for patients who develop complications, such as obstruction, perforation, and stricture formation.[1,4] Stomach and duodenum are involved in just 0.3-2.3% of patients with TB of the gut.[10] Abdominal TB is a rare manifestation of extrapulmonary TB. In a study by Chen and co-workers 21 patients with abdominal TB were identified during a 20-year period. Tuberculous peritonitis was noted in 11 patients. The remaining patients were diagnosed as having TB of gastrointestinal tract (n = 6), urinary tract (n = 2), and pelvis (n = 2).[11]

In another study by Akinkuolie and colleagues, the clinical records of 47 patients who diagnosed as having abdominal TB between January 1986 and December 2005 in Nigeria, were reviewed. Common presenting symptoms and signs were abdominal pain 76.6%, ascites 59.6%, weight loss 53.2%, and fever 29.8%. Average duration of symptoms before presentation was 3 months. Thirteen percent of the patients had earlier been treated for pulmonary tuberculosis in the hospital. Mantoux test was positive in 33% and ascitic fluid evaluation was diagnostic for TB in 29%. Chest radiography showed abnormal findings in 25% of the patients and laboratory evaluation of sputum samples showed acid fast bacilli in 14.3%.[12]

PCR might be a rapid alternative for identification of mycobacterium tuberculosis in culture

and allow for earlier setup of susceptibility testing.[13] Overall sensitivity and specificity of PCR are 100% and 99.7%, respectively. In the study by Smith and co workers, the sensitivity and specificity of PCR were 93% and 100%, respectively.[14] In another study by Webster and colleagues the Roche Amplicor Mycobacterium tuberculosis PCR test (RMtb-PCR) was compared with mycobacterial culture, with the BACTEC 460 (Becton Dickinson, USA) system and inoculation on Lowenstein-Jensen media. The results were interpreted with an adjusted "gold standard" incorporating clinical diagnosis. The sensitivity, specificity, and positive and negative predictive values of RMtb-PCR compared with the adjusted gold standard for clinical specimens were 79%, 99%, 93%, and 98%, respectively. This study demonstrates the value of a commercial nucleic acid amplification kit for rapid diagnosis of Mycobacterium tuberculosis, particularly in smear-positive specimens or BACTEC culture-positive specimens.[15]

Conflict of Interest: None declared

References

1   Faylona JMV, Chung SCS. Abdominal tuberculosis revisited. *Ann Coll Surg* 1999; 3: 65-70.
2   Raviglione MC, O'Brien RJ. Tuberculosis. In: Kasper DL, Braunwald E, Fauci AS et al, editors. Harrison's principles of internal medicine. 16th edition. McGraw-Hill, 2005. p. 957.
3   Mandlle, Bennett, & Dolin. Principles and practice of infectious diseases, 6th ed. 2005 Chuerchill Livingstone. An Imprint of Elsevier. Extrapulmonary Tuberculosis. MD Consult www.mdconsult.com
4   Kapoor VK. Abdominal tuberculosis. *Medicine* 2007; 35: 257-60.
5   Mandlle, Bennett, & Dolin. Principles and practice of infectious diseases, 6th ed. 2005 Chuerchill Livingstone. An Imprint of Elsevier. Chronic inflammatory processes. MD Consult www.mdconsult.com
6   Ong WC, Cheemalakonda R. Colonic tuberculosis mimicking a diminutive sessile polyp. *Dig Endosc* 2005; 17: 257-8.
7   Yilmaz T, Sever A, Gür S. CT findings of abdominal tuberculosis in 12 patients. *Comput Med Imaging Graph* 2002; 26: 321-5.
8   Pulimood AB, Peter S, Ramakrishna B. Segmental colonoscopic biopsies in the differentiation of ileocolic tuberculosis from Crohn's disease. *J Gastroenterol Hepatol* 2005; 20: 688-96.
9   Mert A; Bilir M; Tabak F. Miliary tuberculosis. Clinical manifestations, diagnosis and outcome in 38 adults. *Respirology* 2001; 6: 217-24.
10 Padussis J, Loffredo B, McAneny D. Minimally invasive management of obstructive gastroduodenal tuberculosis. *Am Surg* 2005; 71: 698-700.
11 Chen HL, Wu MS, Chang WH, et al. Abdominal tuberculosis in southeastern Taiwan: 20 years of experience. *J Formos Med Assoc* 2009; 108: 195-201.
12 Akinkuolie AA, Adisa AO, Agbakwuru EA, et al. Abdominal tuberculosis in a Nigerian teaching hospital. *Afr J Med Med Sci* 2008; 37: 225-9.
13 Forbes BA, Hicks KE. Ability of PCR assay to identify Mycobacterium tuberculosis in BACTEC 12B vials. *J Clin Microbiol* 1994; 32: 1725-8.
14 Smith MB, Bergmann JS, Woods GL. Detection of Mycobacterium tuberculosis in BACTEC 12B broth cultures by the Roche Amplicor PCR assay. *J Clin Microbiol* 1997; 35: 900-2.
15 Wobeser WL, Krajden M, Conly J, et al. Evaluation of Roche Amplicor PCR assay for Mycobacterium tuberculosis. *J Clin Microbiol* 1996; 34: 134-9.