# Colonic Tuberculosis and Chronic Diarrhea

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**ABSTRACT**

*We describe that often colonic tuberculosis remains unsuspected prior to surgery. We therefore draw attention to pitfalls in the diagnosis and review the literature on the diagnostic modalities available to diagnose the disease. Today, the prompt diagnosis of an unknown gastroenteritis process involves colonoscopy. Using a fiberscope, a procedure with instantaneous return can be carried out. Patients with clinical presentation suggestive of colonic tuberculosis should have had either an aggressive diagnostic work out using high-yield tests or anti tuberculosis therapy.*

***Key words**: Colonic tuberculosis, chronic diarrhea, colonoscopy.*

## INTRODUCTION

Prior to Crohn's description of regional enteritis in 1932, granulomatous ileitis was attributed to tuberculosis. Today, colonic tuberculosis is too often forgotten, especially when pulmonary disease is absent. Overemphasis on the presence of an abdominal mass or "doughy abdomen" and the insidious nature of tuberculosis peritonitis reduces suspicion.[1] The symptoms of gastrointestinal tuberculosis are nonspecific and in the absence of pulmonary tuberculosis the diagnosis may be difficult. The most common symptom associated with gastrointestinal tuberculosis is abdominal pain. Other symptoms include fever, anorexia, diarrhea, weight loss, constipation, bloating and hemorrhage.[2,3]

Chronic diarrhea is a syndrome with multi complex etiology and pathogenesis. The definition of chronic diarrhea is diarrhea more than 3 times daily, which lasts proceeded for more than 15 days. In colonic tuberculosis, diarrhea happens because of fluid, electrolyte and mucus exudation, mucosal inflammation and also malabsorption.[4]

In some cases gastrointestinal tuberculosis is diagnosed unexpectedly, when surgery is performed for another indication such as hemorrhoids, bowel perforation, appendicitis, carcinoma or colon, small bowel obstruction or Crohn's disease.[1]

## CASE REPORT

A 26-year-old woman came to the gastroenterology department at Dr. Cipto Mangunkusumo National General Hospital having suffered from continuous diarrhea for five months. She had been in her usual state of health until seven months prior to this, when she began to experience lower abdominal pain on the left and right side. After a month, she came to the hospital and was diagnosed as having appendicitis. She went for surgery and the surgeon said that from the biopsy of the appendix, there was no sign of tumor. After the surgery, she had progressive diarrhea, lethargy, fatigue, nausea, anorexia and weight loss. The symptoms developed over a 5-month period. She did not suffer from vomiting, fever, chills or night sweats. She was a kindergarten teacher. The patient did not smoke or use drugs intravenously.

On examination, the patient was thin, cachectic with a body weight of 35 kg, blood pressure of 110/70 mmHg, pulse rate of 80/min and respiratory rate of 16/min. She was afebrile and anicteric. Crackles were absent. Heart sounds were regular. The abdomen was soft, nontender and not distended. Bowel sounds were normal. Rigidity, rebound tenderness, hepatosplenomegaly and masses were absent. The remainder of the examination was unremarkable. The laboratory investigations revealed normal blood urea nitrogen, creatinine levels, platelet count, liver enzymes and bilirubin levels. Haemoglobin

value was 1.07 g/dL and erythrocyte sedimentation rate was 35 mm/hr. Total white blood cell count was 13.7 x 1,000/mm$^3$. Serum albumin was 4.0 g/dL and globulin was 5.2 g/dL. Chest X-ray film showed left upper lobe infiltrate. The gram stain acid-fast stained smear was negative. Esophagegastroduodenoscopy was normal and the endoscopic biopsy revealed mild inflammation. Colonoscopy showed mild hyperaemic mucosa on her rectum and sigmoid, granulomas in her ileocaecal valve. The histopathological findings from endoscopy and ileocaecal biopsy showed submucosal lesions with tubercle, epithelioid cell and Langhan's giant cells.

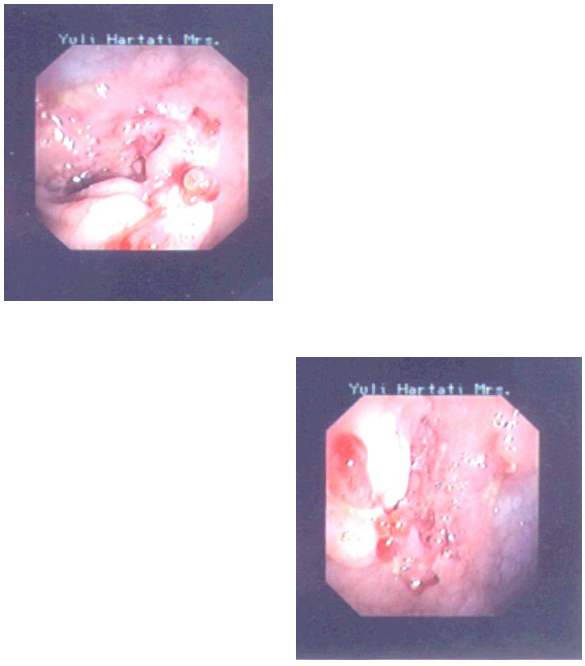


**Figure 1. Colonoscopy showed mild hyperaemic mucosa, irregular ulcers, and pseudopolyps**

The patient was treated with a full course of antitubercular therapy. She received rifampicin 1 x 450 mg, isoniazid 1 x 300 mg, pirazymanide 2 x 500 mg and ethambutol 3 x 250 mg for 9 months.

## DISCUSSION

The incidence of tuberculosis is rising. In the USA, tuberculosis occurs primarily in patients with acquired immunodeficiency syndrome, immigrants, the urban poor, native Americans on reservations, prisoners and in the elderly. Following the resurgence of tuberculosis, there have been two recently published reports of patients with abdominal tuberculosis in the United States. There are many articles warning clinicians to be aware of abdominal tuberculosis and therefore, clinicians in the developed countries are more likely to encounter patients, with colonic tuberculosis now, than in the past.[5] In the UK, pulmonary tuberculosis has declined in incidence but non-pulmonary tuberculosis continues to be common. In 1985, abdominal tuberculosis accounted for 5% of all cases of tuberculosis notified in a district in the UK.[6] In Indonesia, Simadibrata found 4 patients with colonic tuberculosis from 62 chronic diarrhea cases.[7]

The major problem in the management of colonic tuberculosis is to make a prompt diagnosis as the clinical symptoms and signs are non-specific and, more importantly, search for another foci of activity because tuberculosis in the lungs is not helpful. Only one-quarter of patients with intestinal tuberculosis have evidence of active pulmonary tuberculosis.[5]

Haematological tests reveal anaemia, leucocytosis with relative lymphocytosis and raised erythrocyte sedimentation rate (ESR). Between 50% and 80% of adults with abdominal tuberculosis have been found to be anaemic, while ESR was found to be raised in 50% to 80% of patients. Hypoalbuminaemia is frequent. The tuberculin test (Mantoux test was positive in the majority of patients but is of limited value as a diagnostic tool because it does not differentiate between the active form of the disease and previous sensititation by contact or vactination. The radiological findings of tuberculosis (active or healed) on chest X-rays support the diagnosis of abdominal tuberculosis, a normal chest X-ray does not rule it out. In patients with ulcerative intestinal and ascitis peritoneal and those with acute complication. Chest X-ray is more likely to be positive. In this case, we found the abnormality in the chest X-ray examination.

Endoscopic appearances in tuberculosis include hyperaemic nodular friable mucosa, irregular ulcers with sharply defined margins and undermined edges, pseudopolyps and cobblestoning and may mimic Crohn's disease or malignancy. The endoscopic biopsy may not reveal granulomas in all cases, as the lesions are submucosal; biopsy from the edges and the base of the ulcer, multiple biopsies at the same site and endoscopic fine needle aspiration cytology (FNAC) may increase the yield. Although acid-fast bacilli were not seen in any case, some researchers reported positive cultures in more than 40% of endoscopic specimens. Stools and gastric aspirate are rarely positive for acid-fast bacilli in patients with abdominal tuberculosis.[6,8]

Mycrobiological diagnosis of abdominal tuberculosis is difficult; the yield of organisms from abdominal lessions is low because extrapulmonary disease is paucibacillary. Acid-fast bacilli were seen on histological examination by Ziehl Nielson staining in only 6-8% of patients. The diagnosis of abdominal tuberculosis is therefore mainly histological – epithelioid cell granulomas with Langhan's giant cells, peripheral rim of lymphocytes and plasma cells and central caseation necrosis. Non-caseating granulomas, as seen in Crohn's disease, may be present in tuberculosis due to low virulence of the organism and increased host resistance. The mycrobacterial culture should be performed in all cases (although the results take 6 weeks) because it may be positive even in the absence of a characteristic histological picture.

Therapeutic trial – starting the patient in anti-tubercular therapy empirically without a definite diagnosis of tuberculosis, is advocated by many authors in such circumstances. This trial may delay the diagnosis and treatment of diseases such as malignancy, lymphoma and Crohn's disease, which can mimic tuberculosis clinically and even radiologically. Also, anti-tubercular therapy can alter the histological picture in tuberculosis so that the diagnosis cannot be confirmed or refuted at a later date and it may precipitate intestinal obstruction due to healing by fibrosis and cicatrisation or result in intestinal perforation.[6]

All patients with abdominal tuberculosis should receive a full course of anti-tubercular therapy. The conventional regimens include anti-tubercular therapy for 12 to 18 months. Short-course regimens including ethambutol, rifampicin and isoniazid for 3 months followed by rifampicin and isoniazid for 6 months or pirazymanide ethambutol, rifampicin and isoniazid for 2 months followed by rifampicin and isoniazid for 4 months, are effective for abdominal tuberculosis. It is important to administer a correct and complete course, as inadequate drugs, dose or duration is the most important cause of emergence of multi-drug-resistant tuberculosis.[8-10]

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