



## Case of Intestinal Tuberculosis Mimicking Crohn's Disease

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### Background

Although tuberculosis (TB) has traditionally been considered a third world disease, spread of the acid-fast rod was believed to be etiologically related to disease with increased prevalence of immunocompromised patients and the emergence of multidrug resistant strains [1–4]. Although pulmonary involvement is classic, primary extrapulmonary TB infection is increasing, especially among immunocompromised patients. The presentation of intestinal tuberculosis (ITB) closely mimics other more common alternative disease processes making diagnosis challenging [2,5–13]. The case presented here highlights this concept in the form of ITB mimicking Crohn's Disease (CD) and also demonstrates common impediments to diagnosis.

### Case Report

A 23-year-old female presented to the gastrointestinal clinic with multiple episodes of right-sided abdominal pain, fever, emesis, night sweats, and a 5 kg weight loss over the previous seven months. The patient denied diarrhea, melena or hematochezia. Hematologic studies revealed a leukocytosis and iron-deficiency anemia. Blood cultures were negative, including those for acid-fast bacilli. The patient reported a history of positive purified protein derivative (PPD) previously. CT scan of the abdomen revealed cholelithiasis as well as thickening and inflammation of the wall of the ascending colon to the mid transverse colon with moderate right-sided mesenteric adenopathy. Superior mesenteric venous thrombosis was also observed and subsequently treated with low-molecular weight heparin.

Esophagogastroduodenoscopy (EGD) and colonoscopy were performed to further evaluate the patient's CT findings. The EGD appeared normal while the colonoscopy revealed a large, circumferential, fungating mass near the hepatic flexure. Biopsy with histology of the mass was interpreted to be most consistent with tubulovillous adenoma with low-grade dysplasia. A second pathologic evaluation was obtained and the biopsy was thought to be most consistent with CD due to the presence of submucosal granulomas and histiocyte aggregates. Acid-fast bacilli stains of the biopsy specimens were negative, as were other immunohistochemistries.

Repeat colonoscopy with random biopsies was performed to clarify the etiology of the mass and evaluate the extent of the patient's colitis. Colonic involvement was observed to be isolated to the hepatic flexure. Biopsy specimens at this time were considered to be consistent with CD. Acid-fast bacilli staining of the biopsied specimens were again negative. Abdominal CT enterography was performed to evaluate for small bowel involvement. Significant wall thickening from the terminal ileum to the transverse colon with adjacent lymphadenopathy was found.

The patient was advised to undergo an elective right hemicolectomy with primary anastomosis. The patient declined the surgery. The patient returned with 4 kg weight loss and symptoms of imaging consistent with complete bowel obstruction. CT of the chest was unremarkable and did not show evidence of active or latent TB. The patient underwent urgent right hemicolectomy with diverting loop ileostomy (which was subsequently closed).

Histological analysis of the resected bowel segment revealed chron-

ic inflammation with abundant caseating granulomas and fibrosis. Acid-fast bacilli staining of the lymph nodes was positive, consistent with isolated tuberculous colitis. The patient was referred to infectious disease for postoperative antitubercular chemotherapy.

### Discussion

The case presented underscores the importance of considering intestinal tuberculosis despite negative diagnostic tests. Further, discriminating between CD, colon cancer, and ITB is exceedingly challenging as presentations of the three diseases display significant overlap. In this case, acid-fast bacilli staining and histological analysis of biopsy specimens failed to reveal the bacillus on two separate occasions, a chest CT scan did not display pulmonary involvement and blood cultures with acid-fast staining and culture were negative. As a result, definitive diagnosis was not obtained until surgical resection of the obstructed colon became necessary.

The pathophysiology of ITB has been attributed to swallowing infected liquid droplets with direct seeding, contiguous spread from adjacent organs, hematogenous spread from active primary or miliary TB, or ingestion of contaminated milk from cows infected with bovine tuberculosis [3,10]. Once contained within the gastrointestinal tract, the bacillus traverses the mucosa to the submucosa where infection is primarily localized. Inflammatory processes ensue with consequent edema, cellular infiltration and lymphatic hyperplasia. Over longer periods of time patients may develop submucosal noncaseating granulomas, mucosal ulceration, necrosis, fibrosis and stricture formation [14]. Grossly, ITB can present with ulceration, hypertrophy of the bowel wall, a combination of ulceration and hypertrophy, or fibrotic stricture formation [15]. Mesenteric adenopathy and increased mesenteric fat are common findings which may resemble CD. Ileocecal involvement is reported in 77% of ITB and 22–54% of CD cases [1]. The propensity of ITB to localize the ileocecal region is thought to be a result of physiologic stasis and the increased density of lymphoid tissue in the region [1,3].

ITB is most commonly misdiagnosed as Crohn's Disease or colon cancer or less likely amebiasis, sarcoidosis, *Yersinia* infection, gastrointestinal histoplasmosis and periappendicular abscess [2,3,9,10]. Differentiation of ITB and CD is especially difficult as clinical presentation, radiologic studies and histologic features are equivocal and nonspecific [10]. Furthermore, ITB must be differentiated from CD and ulcerative colitis as steroid treatment can be life saving in irritable bowel disease but fatal in cases of misdiagnosed ITB [3].

A history of younger age, aphthoid ulceration and perianal disease supports the diagnosis of CD whereas a history of immigration from endemic areas, or immunocompromise is more commonly seen in patients with ITB [1]. Abdominal pain, weight loss, fever and diarrhea are found in approximately 85%, 66%, 35–50% and 20% of patients, respectively [16]. Physical exam findings include abdominal tenderness and an abdominal mass in the right lower quadrant in 25% to 50% of ITB cases [3,16]. A majority of patients also present with partial or complete bowel obstruction, intussusception secondary to lymphoid hyperplasia, or rarely with recurrent gastrointestinal tract bleeding [1]. With the exception of an elevated erythrocyte sedimentation rate (found in 90% of cases), abnormal laboratory studies

are uncommon in ITB [17]. A positive PPD test is found in 70–86% of patients [12,17]. Importantly, immunocompromised patients are less likely to have a positive PPD test but are at an increased risk of ITB.

Although classically a pulmonary disease, recent evidence indicates that the incidence of extrapulmonary TB is increasing, especially among immunocompromised patients [1,3]. Isolated extrapulmonary TB is more common than concomitant pulmonary TB as only 20–25% of ITB cases display simultaneous active or latent pulmonary TB [2]. Disease presentation can be acute, acute on chronic, or chronic and insidious, thus, mimicking more common abdominal diseases, especially in developed nations where TB is less prevalent.

Radiographic studies are useful but nonspecific. Abdominal CT scan has a sensitivity of approximately 67% [4]. Ascitic fluid analysis from paracentesis in patients with ascites is diagnostic if acid-fast bacilli are visualized or cultured. However, many practitioners consider colonoscopy with multiple biopsies to be the most effective diagnostic tool [17]. Identification of noncaseating submucosal granulomas is more frequently identified when biopsies are obtained from ulcer margins than from nodular lesions [15,17]. Nonetheless, granulomas with or without caseation are seen in less than 50% of ITB cases and the diagnostic challenges are further complicated as CD can present with submucosal noncaseating granulomas [5,18]. Acid-fast bacilli staining of biopsy specimens has been shown to be positive in 30–60% of confirmed ITB cases [3,19]. Culture of biopsied samples is similarly insensitive and false positive results have been reported in patients with concomitant active pulmonary TB after swallowing infected particles [3]. Moreover, culture requires 3–8 weeks with a sensitivity ranging from 25–35% [1]. Nonetheless, requesting culture with sensitivities is becoming increasingly important due to the emergence of drug-resistant TB strains.

Polymerase chain reaction (PCR) has become a more rapid diagnostic test with improved sensitivity and specificity, reported to be approximately 75–80% and 85–95%, respectively [1]. Thus, PCR assay is a promising diagnostic modality for endoscopically or surgically obtained specimens where available.

Treatment for ITB, if suspected, should be empirically instituted with a full course of antitubercular agents despite negative test results. Medical management is effective and rapid resolution of symptoms within 1–2 weeks is typical [14]. Conservative management is initially preferred in patients who require surgery as there is reduced morbidity when elective surgery is performed 2–4 weeks after institution of antitubercular therapy [13]. Complications requiring surgical intervention include complete bowel obstruction, fistulization, perforation, and bleeding [12]. Colonic resection with primary anastomosis and postoperative antitubercular treatment is recommended over colectomy with colostomy [20].

### Conclusions

Signs and symptoms of ITB are usually nonspecific and diagnostic tests insensitive as highlighted in the presented case. A high index of suspicion is required for timely diagnosis. Isolated extrapulmonary TB is more frequently encountered, especially in immunocompromised patients. A positive PPD but negative chest x-ray is the most common presentation in ITB. Multiple diagnostic tests should be ordered including endoscopy with multiple biopsies for histology, staining and culture. PCR has shown improved sensitivity and specificity and should be requested where available. Negative results do not rule out ITB as false negative findings are common. A low threshold for instituting empirical anti-tubercular therapy is recommended and may lead to improved outcomes. Rapid resolution of symptoms with treatment is typical. Surgical intervention is commonly required and leads to improved outcomes in patients who received anti-tubercular therapy prior to surgical intervention.

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