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A Challenging Case of the Forgotten Abdominal Tuberculosis in the Developed World

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Abstract: Differentiating abdominal tuberculosis (TB) from Crohn's disease (CD) despite the rarity of the condition remains vital to avoid catastrophic consequences of disseminated miliary TB as a result of mistakenly starting an immunosuppressive medication. We highlight a challenging pediatric abdominal TB case of a 5-year-old male that presented with failure to thrive, ascites, and diarrhea. Our case aims to shed light on a forgotten disease in our developed world by highlighting subtle clinical, endoscopic, and histologic features. Findings of caseating necrosis on biopsy, positive smear for acid-fast bacillus (AFB), AFB culture, and necrotic lymph node on imaging are diagnostic of TB but are rarely present. Clinicians should be vigilant in screening pediatric patients with elusive symptoms, history, and exam. TB should be suspected, and one should not shy away from empirical antituberculous treatment as it could be the only way of establishing the diagnosis.

Keywords: Intestine, Pediatrics, Crohn's disease, Tuberculosis

INTRODUCTION

The incidence of Tuberculosis (TB) worldwide is slowly declining; however, inflammatory bowel disease (IBD) incidence is rising in developing countries (1). Abdominal TB is a communicable disease caused by *Mycobacterium tuberculosis*. Abdominal TB represents a small fraction of TB cases. Nevertheless, it is significant to recognize as its clinical, endoscopic, and histologic features overlap with IBD, specifically Crohn's disease (CD) (2–4). Both pediatric abdominal TB and CD can present with growth failure and diarrhea, whereas ascites is a feature more commonly seen in abdominal TB (3,4). While necrotizing caseating granulomas and the finding of acid-fast bacilli (AFB) in intestinal biopsies are pathognomonic features of abdominal TB, they are not always present and have low sensitivity (2,3). This overlap causes a diagnostic and therapeutic dilemma. We present a challenging case of abdominal TB in a pediatric patient in the United States. Given the rarity and elusive nature of abdominal TB, our case presentation will shed light on a forgotten disease in our developed country. We aim to review the clinical presentation and challenges faced in establishing a diagnosis and starting treatment. Parents provided informed consent for publication of the case details.

CASE

A 5-year African American male presented to the emergency department with low-grade fever, worsening abdominal distention and watery diarrhea for 1 week. He had been fatigued with intermittent shortness of breath. He had no abdominal pain, vomiting, joint pain, oral ulcers. Upon examination, his abdomen appeared distended, with shifting dullness and no appreciated tenderness. Malnutrition was noted with upper extremity muscle wasting.

On admission, his blood work was notable for microcytic hypochromic anemia (hemoglobin of 9 mg/dL [11.5–13.5 mg/dL]), thrombocytosis (512 [100–400 × 10^9L]), mildly low albumin (3.2 gm/dL [3.4–4.7 gm/dL]), elevated inflammatory markers (ESR 79 MM/HR [0–13 MM/HR], CRP 5 mg/dL [<0.5 mg/dL]). Chest radiograph showed small bilateral pleural effusions.

Abdominal ultrasound and CT showed large volume ascites, and small bowel wall thickening suggestive of enteritis, cecum, and terminal ileum were normal although on imaging (Fig. 1). Endoscopy showed congested mucosa at the cecum and ulceration at the ileocecal valve (Fig. 2), rest of the colon, terminal ileum, and upper endoscopy were unremarkable. Histology showed acute active colitis with moderate neutrophilic infiltration of the surface epithelium and crypts, no granulomas were identified. Immunostaining for cytomegalovirus (CMV) was negative along with AFB and Grocott methenamine silver (GMS) for acid fast and fungal organism consecutively. Paracentesis showed exudative fluid, straw colored, with the following analysis: total nucleated cells count of 1,243 (0–10 × 10 E6/L) with a lymphocytic predominance, elevated protein, mildly low glucose. Serum ascites albumin gradient (SAAG) was <1.1. Cytology of the ascitic fluid was negative for malignancy, and Gram stain, AFB, fungal stains, bacterial and fungal cultures of the same were negative. Fecal calprotectin was elevated at 923 (0–120 μg/g). Collaboratively these findings pointed more toward a chronic infectious process rather than IBD.

Sulfasalazine was started for nonspecific colitis for 1 week then discontinued as further investigations revealed a positive Mantoux tuberculin skin test (TST) with raised, erythematous, indurated area of 3 cm along with interferon-gamma release assays (IGRAs). Upon further questioning, it was revealed that the patient participated in a cruise trip to an area in South America, where TB is endemic 6 months before presentation and that the mother works in a nursing facility.

While the patient's ascites slowly resolved, his weight continued to decline, along with persistent anemia and elevated ESR up to 127 (0–13 MM/HR); CXR, and sputum culture for AFB were negative.

Further collaboration between the health department, infectious disease, and the pulmonology service concluded that symptoms, labs, and histologic findings suggested abdominal TB despite

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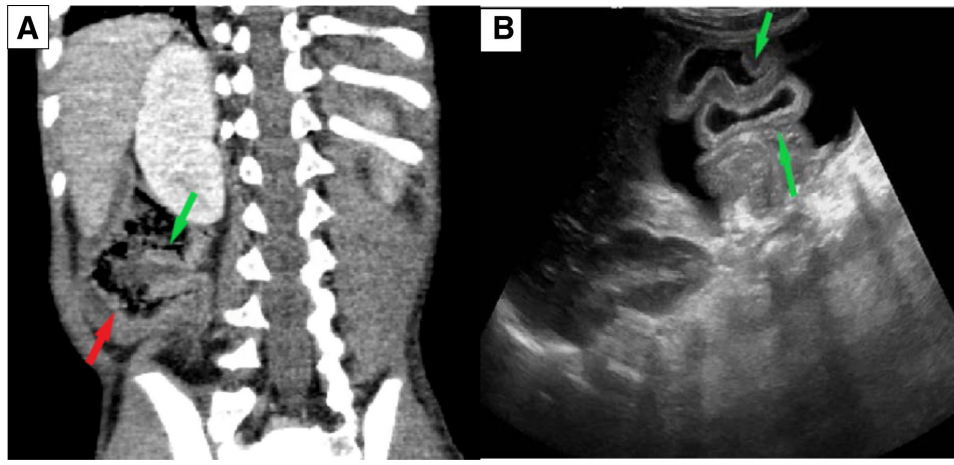

FIGURE 1. A, CT abdomen showing normal Ileum (green arrow) and cecum (red arrow). B, Ultrasound abdomen small bowel wall thickening (green arrow).

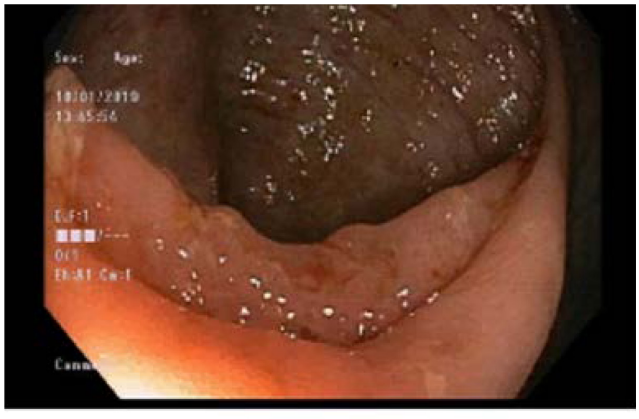

FIGURE 2. Ileocecal valve ulcer.

culture-negative ascitic fluid. The patient was started on quadruple therapy (isoniazid, rifampin, pyrazinamide, ethambutol) for 2 months, then switched to dual therapy (Isoniazid, Rifampin) for 6 months. Monitored labs showed no signs of toxicity, resolved anemia, normalized inflammatory markers, and improved nutritional status. Full resolution of symptoms and labs after completion of antituberculous therapy was observed.

DISCUSSION

Abdominal TB may affect the gastrointestinal tract, peritoneum, lymph nodes, liver, and spleen. The peritoneum and abdominal lymph nodes are most commonly affected. Abdominal TB is chronic in nature; its presentation takes weeks to months to evolve. Symptoms of abdominal TB are usually vague, the most common being low-grade fever, fatigue, abdominal distention, abdominal pain, and weight loss. It mimics other diseases such as CD, thus, a delay in diagnosis often occurs (5). In a developed country with low incidence of tuberculosis, differentiating abdominal TB from CD is crucial, as starting immunosuppressive medication carries the risk of developing disseminated miliary TB, a fatal complication. As in our case, history of exposure is not always available at presentation (5,6). While ascites is regarded as one of the key differentiating factors between abdominal TB and CD, it is not sufficient to make the diagnosis. Inflammatory markers and fecal calprotectin are elevated in both diseases (7). TST combined with T cell–based IGRAs can increase the sensitivity of detecting TB when miliary TB is suspected and in immunosuppressed patients (8).

Transverse ulceration of the ileocecal valve and stricturing of the small bowel are endoscopic features found more common in intestinal TB (ITB) (3). The histologic finding of necrotizing caseating granuloma is confirmatory of abdominal TB, along with positive AFB smear or culture. Nonetheless, these have low sensitivity (2). Paracentesis showing exudative fluid with lymphocytic predominance and SAAG <1.1 is typical for abdominal TB. Adenosine deaminase level can be helpful and specific for diagnosis (9); it could not be obtained in our patient.

Given our high suspicion for abdominal TB, in spite of negative biopsies and cultures, we proceeded with a therapeutic antituberculous trial after careful literature review and a discussion between multiple services and the family. This is a common practice in developing countries with a high incidence of TB. Antituberculous therapy as (Isoniazid, Rifampin Pyrazinamide, Ethambutol) has several side effects and requires close monitoring. Typical treatment duration is 2 months for the quadruple therapy and 6–9 months with dual therapy (Isoniazid, Rifampin). Medication choice and duration can vary based on known resistance of the bacteria in different countries (6). Response to therapy and a repeat endoscopy is the preferred approach to demonstrate macroscopic and microscopic healing, which is considered confirmatory of the diagnosis of Abdominal TB in challenging cases (3,4). Our patient had an excellent response to therapy with complete resolution of clinical and biochemical abnormalities. Unfortunately, we are unable to provide endoscopy results as of yet, due to scheduling difficulties. We urge clinicians to be vigilant in screening pediatric patients with elusive symptoms, especially in nonclassical IBD cases. The decision to treat despite lack of microbiological or histopathological evidence should be taken in collaboration with appropriate subspecialties and local health authorities. As missing the diagnosis of TB and starting immunosuppressive medications could have devastating consequences.

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