Abdominal Tuberculosis

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Tuberculosis remains a deadly global health problem, especially in developing nations. Extrapulmonary manifestations are prevalent in these endemic areas and on the contrary, are usually limited to the immunocompromised and immigrants in the Western world. In the United States, abdominal infection is even more exceptional, which can be problematic to clinicians in view of its non-specific presentation and investigatory findings. In this case, a 60-year-old Hispanic-American female presenting with a three-month history of abdominal pain, nausea and vomiting who was referred for an elective screening colonoscopy. Her physical and laboratory studies were unremarkable and her chest x-ray was negative. Furthermore, the patient had negative PPDs on two separate occasions. Contrast computed tomography of the abdomen showed scarring of the right lung base and sigmoid diverticulosis. The screening colonoscopy revealed two polyps and a cecal ulcer. Histological studies of biopsy samples obtained from the cecal ulcer conclusively documented the existence of gastrointestinal tuberculosis. This case illustrates the subtle and indefinable course of abdominal tuberculosis. While it can commonly mimic diseases such as inflammatory bowel disease or colon carcinoma, it can also follow a more indistinct pattern as well, particularly in the acute stages.

INTRODUCTION

According to the World Health Organization, more than 2 billion people are estimated to be infected with tuberculosis (TB) and approximately 95% of tuberculosis cases occur in developing countries. Of these cases, 1–3% are classified as extrapulmonary and among them abdominal TB, which may involve the gastrointestinal tract, peritoneum, lymph nodes or solid viscera, constitutes up to 12%. Gastrointestinal involvement is found in 66–75% of abdominal cases, with the terminal ileum and the ileocecal region being the most common sites of involvement.

Infection of the gastrointestinal system with tuberculosis is accomplished by means of ingestion, through a hematogenous route from other tuberculous foci or local spread from surrounding organs involved. It may often be confused with other intestinal inflammatory lesions and diagnosing intestinal tuberculosis is difficult not only due to variable anatomical location, but lack of efficient and sensitive tools. The most common presenting symptom of intestinal tuberculosis, seen in 90–100% of patients, is abdominal pain. Other common symptoms include weight loss (66%), fever (35–50%), and a change in bowel habit (20%). Less common but well-described symptoms include malabsorption, night sweats, malaise, anorexia, nausea, vomiting, melena, and rectal bleeding. Due to these relatively non-specific symptoms, delays in diagnosis are frequent. For our study, a definitive diagnosis of abdominal Mycobacterium tuberculosis was issued by...
histopathologic examination demonstrating caseating epithelioid cell granulomas with positive Ziehl-Neelsen staining demonstrating the presence of acid-fast bacilli (Figure 1, 2).

Ninety-nine percent of intestinal TB is a phenomenon relegated to developing nations. Unfortunately, there is an absence of current literature that elucidates the role of intestinal TB as it pertains to the developed world.

**CASE PRESENTATION**

A 60-year-old Hispanic-American woman was seen in the Emergency Room for abdominal pain, nausea and diarrhea. The pain was described as mild, intermittent, non-radiating and epigastric in origin which occurred mainly at night and was also aggravated with spicy foods. There were three episodes of non-bilious vomiting in addition to three episodes of non-bloody, non-mucoid diarrhea over the course of the day. The patient also denied any fever, weight loss or constipation. Her past medical history was unremarkable. She recently immigrated to the United States from Mexico.

Physical examination and laboratory investigations were unremarkable, with the exception of an elevated erythrocyte sedimentation rate (ESR) of 40 mm/hr. An abdominal CT with contrast was performed, which showed non-specific, pericolonic lymph nodes in the right lower quadrant (Figure 3A–B). There was no evidence of bowel wall thickening or obstruction. A presumptive diagnosis of acute gastritis was made and the patient discharged the same day on anti-acid medication and advised to follow up at the gastrointestinal clinic.

The patient remained symptom-free until her return to the GI clinic at which point a screening colonoscopy was scheduled. The colonoscopy was significant for multiple, irregular ulcers of uncertain nature located in the cecum (Figure 4A–B). The lesions were not actively bleeding; however, old blood was noted in and around the lesions. Biopsy of the ulcers showed colonic mucosa with ulceration and necrotizing granulomatous inflammation. An acid-fast stain was performed which was notable for bacilli. An inflammatory polyp, measuring 8 mm, was also located in the cecum. The ileum was unremarkable for any thickening, including in and around the region of the ileocecal valve (Figure 4C).

A review of her chart revealed a negative PPD test in 1997 and that her immunizations were up-to-date. Physical examination was unremarkable for any masses, lymphadenopathy, or hepatosplenomegaly. A fecal occult blood test was negative. Laboratory investigations were also within normal limits. Chest x-rays revealed no focal consolidations or effusions. There was no evidence of cavitary lesions or mediastinal

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**Figure 1.** Characteristic caseating granulomas with epithelioid cells.

**Figure 2.** Ziehl-Neelson stain showing an intracellular acid-fast bacillus (arrow).
lymphadenopathy. Additionally, the cardiome-diastinal silhouette was within normal limits.

Three sets of sputum samples were negative for culture and smear, and a repeat in-hospital PPD test was negative. The patient was subsequently discharged and placed on Rifampin (600 mg/daily), Ethambutol (1200 mg/daily), Pyrazinamide (1500 mg/daily), Isoniazid (300 mg/daily) and Pyridoxine (25 mg/daily). After several weeks, the abdominal pain, discomfort, nausea and diarrhea have subsided.

DISCUSSION

To this day, tuberculosis remains a global dilemma with an estimated eight million new cases each year and one-third of the world’s population at risk, according to the WHO. The Western world has encountered a resurgence since the mid-1980’s, primarily due to increased number of HIV-infected individuals and immigrants from countries where the disease is endemic, in addition to the evolution of multi-drug resistant strains of M. tuberculosis.9

The abdomen is the sixth most common extrapulmonary site of tuberculosis,10,11 in which the GI tract makes up approximately two-thirds of such cases. Any region of the gastrointestinal tract may be affected however, the site of predilection within the GI tract is the ileocecal region.12 The focal predominance of this area is believed to be a result of a few key components—an abundance of lymphoid tissue, increased physiological stasis with minimal digestion (permitting greater contact time between bacteria and intestinal lumen) and an increased rate of fluid and electrolyte absorption.5,10 The infection typically results from either reactivation and hematogenous spread from a primary lung focus, ingestion of active pulmonary secretions or contaminated food and direct spread from adjacent organs or lymph nodes9,10. While hematogenous extension from a pulmonary focus was held as the primary mode of abdominal infection, recent DNA fingerprinting studies have elucidated that roughly 40% of cases are due to reinfection.10,14 In almost all cases, the pathogen involved is M. tuberculosis; however intestinal tuberculosis may very rarely be caused by Mycobacterium bovis, secondary to ingestion of contaminated material, such as unpasteurized milk.13

Abdominal tuberculosis usually presents with vague, non-specific symptoms (Table 1), making diagnosis very difficult for clinicians. Physical exam is usually notable for abdominal tenderness, mass, “doughy abdomen,” or hepatosplenomegaly. Our patient presented with abdominal pain, nausea, vomiting and diarrhea which, in the absence of suspicion for the disease or “specific” symptoms such as ascites, fever and/or night sweats makes for a very difficult clinical diagnosis.

Laboratory values commonly exhibit anemia and an elevated ESR which are non-specific.16,17,18 PPD

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skin test is positive in the majority of patients with abdominal tuberculosis, but it lacks diagnostic significance because of the high rate of false-positives.9,19

Radiological studies act as an adjunct in the diagnosis of abdominal tuberculosis. Computed tomography has proven to be an effective tool in recognizing the pathology consistent with abdominal tuberculosis18,20 although the findings can be non-specific and may mimic many other conditions, including inflammatory bowel disease or colon cancer (Table 2).

Ileocecal tuberculosis is typically hyperplastic, exhibiting regional adenopathy as well as circumferential thickening of both the cecum and the ileum;10,21 however, in our case no bowel thickening was appreciated. There was also no evidence of lumen narrowing and associated proximal dilatation as observed in other studies. Ascites is one of the most common features seen in abdominal tuberculosis, yet it was also absent in our patient.

Chest x-rays are helpful in the work up pulmonary TB, however is often non-contributory in cases of abdominal TB. Evidence of concurrent pulmonary tuberculosis has been reported to be absent on chest x-ray in anywhere from 50–75% of cases.10,13,22 Conversely, when acute complications such as obstruction, perforation and peritonitis are present, chest x-ray is positive in 80% of patients.22

Colonoscopy has proven to be invaluable in the diagnosis of intestinal tuberculosis, due to its ability to identify the pathology both macroscopically and microscopically, via biopsy. Characteristic mucosal lesions can be classified into four types: type one, circumferential ulceration with nodules; type two, round or irregularly shaped small ulcers, arranged circumferentially, without nodules; type three, multiple erosions restricted to the large intestine; and type four, small ulcers or erosions restricted to the ileum.23 Morphology of tubercular lesions has also been described as ulcerative, hypertrophic and ulcerohypertrophic.9,13,21 Differentiation from Crohn’s disease can be difficult. Evidence of transverse ulcers, absence of crypt distortion, or extensive chronic inflammation in the lamina propria distant from ulcerated foci all suggest abdominal tuberculosis.24 Biopsy of colonic, ileal and ileocecal lesions produce the highest yield of confirmation when subjected to histopathology, acid-fast bacilli smear and culture.

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Table 1. Common Presenting Symptoms

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<thead>
<tr>
<th>Symptom</th>
<th>n</th>
<th>%</th>
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<tbody>
<tr>
<td>Abdominal pain</td>
<td>194</td>
<td>93</td>
</tr>
<tr>
<td>Fever</td>
<td>134</td>
<td>64</td>
</tr>
<tr>
<td>Night sweats</td>
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<tr>
<td>Weight loss</td>
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<td>47</td>
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<tr>
<td>Vomiting</td>
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<td>Ascites</td>
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<td>Constipation</td>
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<td>31</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>25</td>
<td>12</td>
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Table 2. Computed Tomography Features

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<tbody>
<tr>
<td>Peritoneal involvement</td>
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<td>77.5</td>
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<tr>
<td>With ascites</td>
<td>21</td>
<td>52.3</td>
</tr>
<tr>
<td>Without ascites</td>
<td>17</td>
<td>45.7</td>
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<tr>
<td>Mesenteric fat stranding</td>
<td>18</td>
<td>47.3</td>
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<tr>
<td>Omental thickening</td>
<td>9</td>
<td>23.6</td>
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<tr>
<td>Lymphadenopathy</td>
<td>23</td>
<td>46.9</td>
</tr>
<tr>
<td>GI strictures/bowel wall thickening</td>
<td>19</td>
<td>38.7</td>
</tr>
</tbody>
</table>


References
6. Davood Yadegarynia et al. Gastrointestinal Tuberculosis with Cecum Involvement in a 33-Year-Old Woman, JIMS Vol 34, No 3, September 2009

studies.25,26 Biopsy often shows caseating epitheloid granulomas among a chronic lymphocytic infiltrate, which has been used to establish diagnosis. Microbiological confirmation is regarded as definitely diagnostic, although isolation of the bacterium is very rare.17

This case represents an unusual presentation of intestinal tuberculosis, as the presenting symptoms were few and ambiguous in nature. Early diagnostic measures did not provide any clear evidence for a presumptive diagnosis of abdominal tuberculosis. It was not until colonoscopic examination that any significant pathological process was elucidated. To that end, the various combinations of signs, symptoms and radiologic findings provide little or no help in diagnosis—it is merely the suspicion of the clinician that can provide for the proper diagnostic procedures. ■