

Abdominal Tuberculosis: An Unusual Cause of Abdominal Pain

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Introduction

The incidence of intestinal tuberculosis (TB) in developed countries has been increasing along with the overall resurgence of TB due to the acquired immune deficiency syndrome (AIDS) epidemic, increased use of immunosuppressive drugs, and an aging population.¹ Prior to the AIDS epidemic, intestinal TB was seen most commonly in immunocompetent persons with untreated advanced pulmonary disease. Currently it is most commonly observed in association with immunosuppressive treatments. In one series, more than 40% of patients with gastrointestinal TB had AIDS. Because of the non-specific symptoms associated with intestinal TB, timely diagnosis might be missed. It is important that physicians maintain a high awareness of TB as a potential source of abdominal symptoms.

Case Summary

A 22-year-old female, a recent immigrant from India, presented with burning and worsening epigastric and left-lower-quadrant pain, for 3 days. Similar symptoms had been present for 3 years and were associated with anorexia, intermittent nausea, vomiting, and constipation. These symptoms were partially relieved by proton pump inhibitors and antacids. There was no hematemesis, melena, fever, or jaundice. The patient complained of palpitations, easy fatigability, and decreased menstrual flow for the last 2 months. She had lost 14 kg in 1 year but maintained a weight of 36 kg for 3 years. She denied cough, chills, joint pains, hemoptysis, or night sweats.

Four years prior to presentation, she had been hospitalized for pneumonia. An abdominal computed tomogram (CT), colonoscopy, and esophagogastroduodenoscopy in

India 1.5 years prior to presentation were reportedly normal. On admission she had a temperature of 38.0° C, a heart rate of 118–141 beats/min, and normal blood pressure. Physical examination showed cachexia (body mass index 14 kg/m²), pale conjunctivae, clear lungs, normal heart sounds, with no murmurs, and diffuse abdominal tenderness but no guarding or rebound. Laboratory tests showed anemia (hemoglobin 9.7 mg/dL, hematocrit 30.5 g), hypoalbuminemia (albumin 1.5 g/dL), and vitamin B12 deficiency.

Chest radiograph revealed a lingula infiltrate (Fig. 1). Chest CT showed nodular infiltrates in the lingula, associated with a calcific density in the superior segment of the left lower lobe (Fig. 2).

Abdominal CT showed dilated small-bowel loops, thickening of the cecum and terminal ileum, mesenteric and retroperitoneal lymphadenopathy, and nodular densities in the omentum (Fig. 3A). A purified-protein-derivative test was positive, with an induration of > 10 mm. Sputum acid-fast-bacilli smears and culture were negative.

An upper-gastrointestinal series was normal, but a small-bowel series revealed a 5 cm constricting lesion involving the terminal ileum, cecum, and ascending colon (Fig. 4A). Colonoscopy revealed inflammatory and ulcerative changes and a markedly thickened and hypertrophic mucosa in the region of the terminal ileum, cecum, and ascending colon. Acid-fast-bacilli smear and culture of colonoscopic biopsies were negative; there were no granulomas and no histologic evidence of inflammatory bowel disease. The CD4 count was low (313 cells/ μ L), the CD8 count was within normal limits (413 cells/ μ L), and the CD4/CD8 ratio was low (0.72 cells/ μ L, range 0.98–2.42).

The patient refused formal testing for human immunodeficiency virus. We treated her empirically for intestinal TB, with isoniazid, pyrazinamide, ethambutol, and rifampin. Peripheral parenteral nutrition was initially required, but she was able to switch to oral feedings, and she was discharged after a 1 month hospitalization. She continued treatment for a total of 9 months, and had a 14-kg weight gain and improvement of symptoms. Ten months later gastrointestinal symptoms recurred, and repeat gastrointestinal imaging (small-bowel series and abdominal CT) revealed improvement in the luminal narrowing of the

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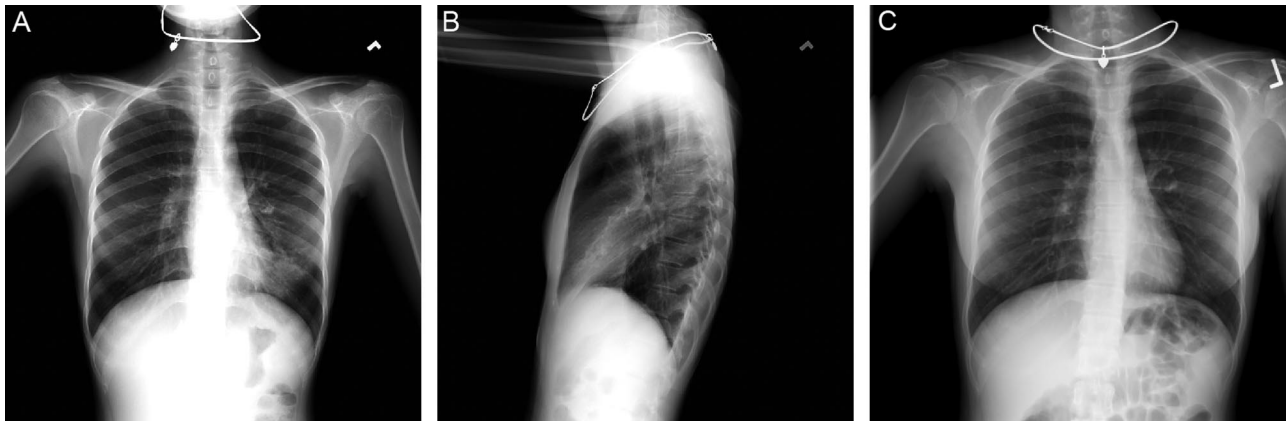


Fig. 1. A and B: Chest radiographs shows a lingula infiltrate. C: Follow-up chest radiograph shows resolution of lingula infiltrate.

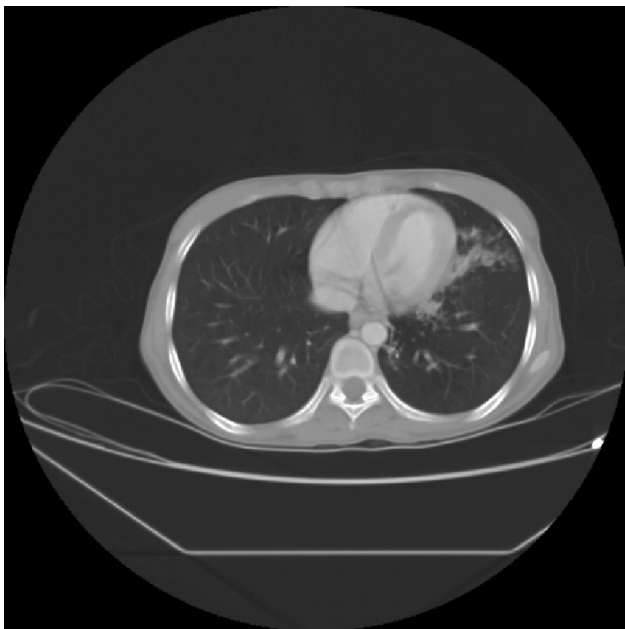


Fig. 2. Computed tomogram shows lingula nodular infiltrates and a calcific density in the superior segment of the left lower lobe.

small bowel and colon and resolution of the para-aortic and mesenteric lymphadenopathy (see Figs. 3B and 4B). The liver, spleen, pancreas, and gallbladder were normal. A follow-up chest radiograph revealed resolution of the lingula infiltrate (Fig. 1C). Her symptoms resolved promptly within 2 days with bowel rest, and were attributed to intermittent bowel obstruction. TB treatment was continued for an additional 12 months and she had complete resolution of symptoms.

Discussion

The ileocecal region is reported to be the area most commonly involved in colonic TB. This is attributed to the

tubercule bacillus's affinity for the abundant lymphoid tissue in the ileocecal region, the relative physiologic stasis and minimal digestive activity (which facilitates prolonged contact between the bacilli and the mucosa), and the high rate of absorption in this region.² Besides the ileocecal area, segmental colitis can also occur, involving the ascending and transverse colon. Colonic TB may present as an inflammatory stricture, hypertrophic lesions resembling polyps or tumors, segmental ulcers, and colitis.¹⁻³

The pathophysiology of tuberculous enteritis has been attributed to 4 mechanisms:

- Hematogenous spread from active pulmonary or miliary TB
- Swallowing of infected sputum in patients with active pulmonary TB
- Ingestion of contaminated milk or food
- Contiguous spread from adjacent organs

After the bacillus enters the gastrointestinal tract, it traverses the mucosa and lodges in the submucosa where it induces inflammatory changes that can lead to lymphangitis, endarteritis, and fibrosis, which can progress to mucosal ulceration, caseating necrosis, and narrowing of the intestinal lumen.¹

Diagnosing abdominal TB is difficult because its presenting signs, symptoms, and laboratory abnormalities are non-specific and non-diagnostic. Symptoms of abdominal TB include abdominal pain, diarrhea, fever, anorexia, weight loss, constipation, and hemorrhage. The most frequent symptom at presentation (in 90–100% patients) is abdominal colicky pain representing intermittent bowel obstruction.^{1,2} The most common laboratory abnormalities are anemia, leukocytosis, elevated sedimentation rate, and hypoalbuminemia. Routine blood tests cannot exclude intestinal TB. Our patient had both anemia and hypoalbuminemia.

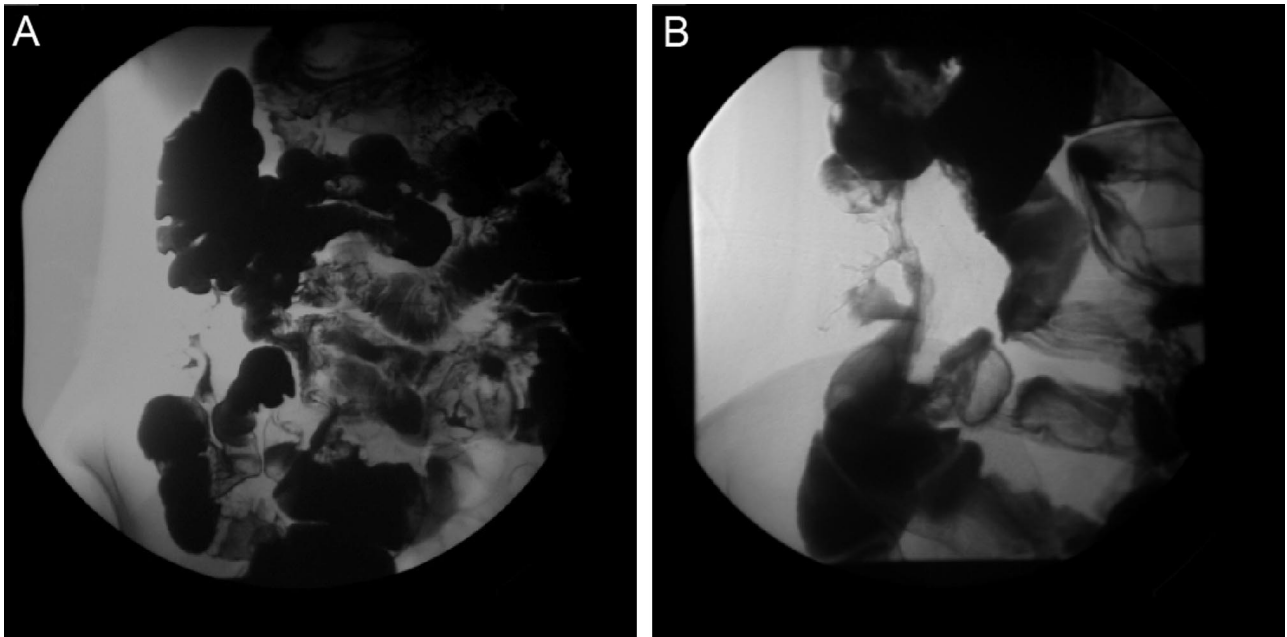


Fig. 3. A: Computed tomogram shows dilated small-bowel loops, thickening of the cecum and terminal ileum, mesenteric and retroperitoneal lymphadenopathy, and nodular densities in the omentum. B: Follow-up computed tomogram shows improvement in the degree of luminal narrowing of the small bowel and colon, and resolution of the para-aortic and mesenteric lymphadenopathy.

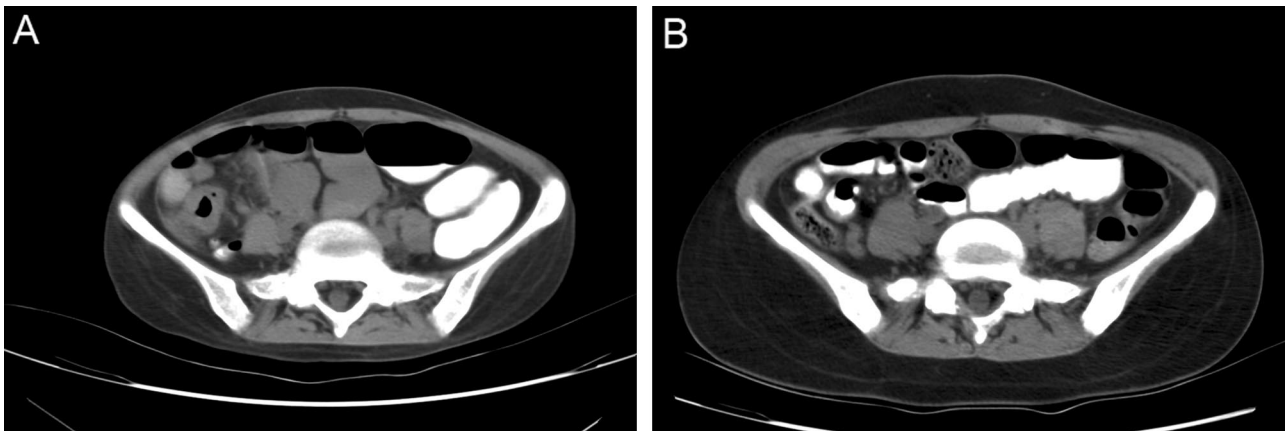


Fig. 4. A: Computed tomogram shows a 5-cm constricting lesion involving the terminal ileum, cecum and ascending colon. B: Follow-up computed tomogram shows improvement in the degree of luminal narrowing of the small bowel and colon, and resolution of the para-aortic and mesenteric lymphadenopathy.

A presumptive diagnosis of intestinal TB can be made in the setting of known active pulmonary TB or with a chest radiograph suggestive of TB together with gastrointestinal symptoms or radiographic abnormalities suggestive of intestinal TB. Chest radiographs are positive in only 20–50% of patients with intestinal TB.^{1,3,4} A barium radiograph may reveal luminal narrowing with proximal dilatation of bowel loops. CT and abdominal ultrasound findings suggestive of abdominal TB include ascites, enlarged lymph nodes, omental thickening, and bowel-wall thickening. Definitive diagnosis is based on histology,

Ziehl-Neelsen staining for acid-fast bacilli, and culture. The sensitivities of these tests differ.

Colonoscopy with biopsy is the most useful non-operative diagnostic test for intestinal TB. In a study with 209 patients, the diagnostic yield of TB on surgical specimens was 100%, whereas biopsies obtained via colonoscopy had a yield of only 83%. The combination of histology and culture can establish the diagnosis in up to 80% of patients.² Polymerase-chain-reaction testing of biopsy specimens can provide a diagnosis in 48 hours, with higher sensitivity and specificity, but in some cases of intestinal

TB the polymerase-chain-reaction test and acid-fast-bacilli culture have been negative.

A diagnosis can also be made via histologic analysis and response to TB treatment. It is important to know that the pathognomonic histologic findings of epithelioid granulomas with Langhans giant cells and central caseation necrosis may be present only in lymph nodes that are not included in biopsy specimens.²⁻⁴ In a study by Khan et al, only 68% of biopsy specimens revealed non-caseating granulomas, and 25% revealed caseating granulomas. This is not surprising, since granulomas are submucosal, and biopsy samples are at times only from the mucosal layer. Granulomas may be totally absent in patients who have received TB medication in the past. Acid-fast-bacilli culture and smear positivity are not always present. Only 35–60% of intestinal TB cases are smear-positive for acid-fast bacilli. The presence of granulomas is not always related to the yield of positive cultures.^{1,2,5}

Intestinal TB is a diagnostic challenge, especially in patients with no evidence of active TB infection. It may mimic other abdominal diseases, such as Crohn's disease. Differentiating TB from Crohn's disease is important, since treatment of these 2 disorders is very different. Both entities may present with mucosal ulcerations, nodularity, ulcers, pseudopolyps, luminal narrowing, and strictures. Characteristic endoscopic findings that distinguish the 2 diseases include multiple, large confluent granulomas, caseating necrosis, and disproportionate mucosal inflammation in intestinal TB, whereas in Crohn's disease the granulomas are small, less frequent, and surrounded by focal areas of colitis.^{5,6} Other features of Crohn's disease that are absent among patients with intestinal TB include extra-intestinal manifestations of arthralgia, ankylosing spondylitis, and peri-anal fistulas.⁶

Our patient had radiographic abnormalities in the lung and gastrointestinal tract. Although histology showed only inflammatory and ulcerative changes, without diagnostic features of TB (acid-fast bacilli and caseation), her response to TB therapy contributed to the diagnosis of TB. The recurrence of her symptoms after 9 months of treatment was probably due to intermittent obstruction from a

narrowed ileocecal lumen, visualized on colonoscopy. Her symptoms resolved completely after completing an additional 12-month course of 4-drug TB therapy.

Teaching Points

Intestinal TB should be considered in the differential diagnosis of unexplained abdominal complaints, particularly in patients originating from countries that have a high incidence of TB. A delay in diagnosis may lead to complications, such as bowel obstruction and intestinal perforation. A timely diagnosis and initiation of therapy may prevent complications and improve outcomes.

The management of abdominal TB relies on TB drugs; surgery is reserved for complications and uncertainty of diagnosis. Ideally the diagnosis should be confirmed via histology or isolation of bacteria, but typical granulomas and acid-fast-bacilli are not always detected. A high clinical suspicion for abdominal TB and empiric TB therapy may be warranted in situations where a definitive diagnosis can not be made. Clinical improvement after TB therapy indicates a correct diagnosis of abdominal TB.

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