453

Primary Gastric Fundus Tuberculosis in Immunocompetent Patient: A Case Report and Literature Review

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We report on a 29-year-old Pakistani man who presented to the clinic with epigastric pain, of one-month duration. He did not report fever, cough, vomiting blood, passing black stools, loss of appetite or diarrhea. However, he had lost 7 kg since his symptoms had begun. Clinical examination was unremarkable. Laboratory results were within normal limits. An abdominal CT scan showed a mass with enhancement in the stomach. Gastric endoscopy revealed an ulcerative mass in the fundus. An endoscopic-biopsy specimen revealed caseating granulomas with acid-fast bacilli. The patient was diagnosed to have primary gastric tuberculosis, and antituberculous medications were initiated. Cultures of the gastric mass subsequently grew *Mycobacterium tuberculosis* sensitive to isoniazid and rifampcin. Follow-up after six months showed a good response to treatment; an upper gastrointestinal tract endoscopy after six months was normal.

Key-Words: Gastric tuberculosis, hematemesis, peptic ulcer, stomach perforation.

In the early 1900s, tuberculous involvement of the gastrointestinal tract due to pulmonary tuberculosis was a common occurrence, especially in patients with far-advanced lung infection [1,2]. After the introduction of successful antituberculous regimens, the percentage of pulmonary tuberculosis cases with clinically evident secondary gastrointestinal involvement decreased from 38 to less than 5% [1-4].

However, the gastrointestinal tract is the sixth-leading location of extrapulmonary tuberculosis, following nodal, genitourinary, bone and joint, miliary and meningeal locations [4]. In decreasing order, gastrointestinal localizations include: the ileocecal region, the ascending colon, the jejunum, the appendix, the duodenum, the stomach, the esophagus, the sigmoid colon, and the rectum [5].

The incidence of gastric tuberculosis varies from country to country. In the state of Qatar, the incidence of gastric tuberculosis is unknown; to our knowledge, this is the first reported case.

Case Report

A 29-year-old Pakistani man presented to the clinic with abdominal (epigastric) pain, since one month. There were no specific aggravating factors; pain was relieved at times by antispasmodics. The pain was mild, localized, associated with nausea, and sometimes with vomiting. He denied having fever, cough, blood vomiting, passing black stools, loss of appetite or diarrhea. However, he reported a 7-kg weight loss since his symptoms had begun. Clinical examination was unremarkable.

Laboratory investigations showed a white blood cell count of 6,400/µL with normal differential counts, hemoglobin 13.8

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g/dL, and platelets $289,000/\mu$ L. Blood chemistry, liver profile, lipid profile and coagulation studies were within normal limits. Abdominal sonography and chest X-rays were normal. His HIV status was negative. An abdominal CT scan showed a mass with enhancement in the stomach (Figure 1). Gastric endoscopy revealed an ulcerative mass in the fundus (Figure 2). An endoscopic biopsy specimen revealed caseating granulomas with acid-fast bacilli (Figure 3).

The patient was diagnosed to have primary gastric tuberculosis, and antituberculous therapy with isoniazid, rifampcin, ethambutol and pyrazinamide was begun. Cultures of the gastric mass subsequently grew *Mycobacterium tuberculosis* sensitive to isoniazid and rifampcin. Follow-up after six months showed good response to this treatment; the patient regained his appetite and weight and the abdominal symptoms disappeared. An upper gastrointestinal tract endoscopy after six months was normal (Figure 4).

Discussion

The gastric tract is an uncommon site of extra-pulmonary tuberculosis infection, even in parts of the world where intestinal tuberculosis is common. The incidence of gastric tuberculosis is 0.03 to 0.21 percent of all routine autopsies [6]. The rarity of gastric tuberculosis is due to gastric acid, continuous motor activity of the stomach and the scarcity of lymphatic follicles in the gastric wall [7]. Gastric tuberculosis usually develops secondary to other tuberculous lesions, most commonly pulmonary [8]; nonetheless, sporadic cases of primary gastric tuberculosis have been reported worldwide. ^[9-14] The possible routes of infection include direct infection of the mucosa, hematogenous spread, or extension from a neighboring tuberculous lesion [7].

Gastric tuberculosis is usually associated with an immunodeficient state [8]; many cases reported in developed countries are in immunodepressed patients, particularly those with HIV infection. But such relationships have not been well described in cases reported from developing countries.

The antrum and prepyloric regions are the most common sites of tuberculous lesions in the stomach [6,15]. In our patient the fundus was involved.

Figure 1. Abdominal CT with contrast shows gastric wall thickening with enhancement. (Arrow).



Figure 2. Gastric endoscopy shows ulcerative mass in the fundus.



Figure 3a. Endoscopic biopsy of fundal mass shows a giant cell granuloma near the center of the photomicrograph. (H/E stain x 200). **Figure 3b.** Same endoscopic biopsy shows at least two acid-fast bacilli within the caseous area. (Zeil Nelsen stain x 400).





Figure 4. Gastric endoscopy shows a normal fundus.



The clinical manifestations of this type of infection are nonspecific. Consequently, diagnosis is often missed. There are reports of isolated gastric tuberculosis presenting as pyrexia of unknown origin [16]. Other presentations include gastric carcinoma [17], gastric outlet obstruction [18], benign peptic ulcer [19] and, rarely, stomach perforation [20]. Our patient presented with symptoms of benign peptic ulcer.

Based on endoscopy, lesions may be described as, single or multiple ulcers and hypertrophic nodular lesions surrounding a stenotic pyloric channel [21]. In our patient, endoscopy revealed an ulcerative mass in the fundus.

The diagnosis of gastric tuberculosis can only be made by histological study of the resected stomach or of a biopsy specimen of this organ. Endoscopic brush cytology and

AA 9

biopsy is only occasionally successful in diagnosis [22]. Submucosal location of the lesion has been cited as a reason for failure of endoscopic biopsies [16,23]. Most patients are subjected to surgical intervention, and the diagnosis of gastric tuberculosis is made after surgery. In our patient, diagnosis was made based on an endoscopic biopsy.

On biopsy, granulomas are either caseous or non-caseous. Staining for acid-fast bacilli are frequently negative, and the diagnosis is either by culture or finding of confirmed tuberculosis elsewhere. In our case, endoscopic biopsy showed caseating granuloma and acid-fast bacilli, and the diagnosis was confirmed by culture.

Non-caseating granulomas are also caused by Crohn's disease, sarcoidosis and idiopathic granulomatous gastritis. Clinical and histopathological features and culture help in differentiation. Once diagnosed, complete cure can often be achieved with a course of oral anti-tuberculosis medication, with surgery being reserved for severe symptomatic or refractory lesions. Our patient responded dramatically to a six-month course of oral anti-tuberculosis medication.

In conclusion, this case highlights the diagnostic challenge of gastric tuberculosis in developing countries. A high index of suspicion is required in order to diagnose this rare condition, as it can present in patients with no particular risk factors or symptoms. Therefore, in young patients residing in endemic areas, who have short duration of symptoms, early onset of gastric outlet obstructions and who are non-responders to anti-ulcer therapy, gastric tuberculosis should always be part of the differential diagnosis.

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