Primary Gastric Tuberculosis Presenting as Non-Healing Ulcer and Mimicking Crohn’s Disease

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Abstract

A 39-year-old woman was referred to our hospital for treatment of a non-healing gastric ulcer. Esophagogastroduodenoscopy (EGD) revealed an erosion in the pyloric antrum and a longitudinal ulcer on the lesser curvature of the gastric body. The histopathologic examination of biopsy specimens revealed non-caseating epithelioid granulomas. Acid-fast staining did not reveal bacilli. The differential diagnosis included gastric tuberculosis, Crohn’s disease, and sarcoidosis and empiric antituberculous therapy consisting of isoniazid, rifampicin, ethambutol, and pyrazinamide was initiated. Gastric lesions were subsequently resolved and non-caseating epithelioid granulomas were not demonstrated on the post-treatment examination. Recurrence was not observed during the follow-up period of 53 months.

Key words: gastric tuberculosis, Crohn’s disease, antituberculous therapy


Introduction

Although tuberculous involvement of the gastrointestinal tract was common in the early 1,900s, especially in patients with advanced pulmonary tuberculosis, gastrointestinal and pulmonary tuberculosis showed a decrease in the decades after the introduction of antituberculous therapy (1, 2). Gastric tuberculosis is rare and cases of isolated gastric tuberculosis without evidence of pulmonary involvement have been reported sporadically (3-12). Because histological confirmation of gastric tuberculosis is very difficult on endoscopic biopsies, full-thickness surgical biopsies are sometimes needed for adequate diagnostic histology (6, 7). Here, we report a rare case of primary gastric tuberculosis presenting as non-healing ulcer and mimicking Crohn’s disease, which was eventually diagnosed after empiric antituberculous therapy.

Case Report

A 39-year-old woman was referred to our hospital for the treatment of a non-healing gastric ulcer. A proton pump inhibitor (lansoprazole) had previously been prescribed at an outside facility for one year after successful eradication of Helicobacter pylori. However, there was no resolution of the patient’s gastric ulcer. Her past medical history was unremarkable and she denied any chills, fever, night sweats, cough, hematemesis, melena, and diarrhea, and she had no history of tuberculosis. Her physical examination was unremarkable, and no lymphadenopathy was found. The tuberculin test performed was negative with an erythema of 2 millimeters and no induration. Laboratory tests revealed the decreased hemoglobin level of 8.2 g/dL and the slightly elevated erythrocyte sedimentation test (ESR) of 20 millimeters per hour. Other blood examinations were normal including complete blood count, differential distribution of leukocytes, liver function test, and serum C-reactive protein (CRP) of 0.02 mg/dL. Serology was negative for hepatitis and HIV. Chest X-ray was normal. EGD showed erosion of the pyloric antrum and a longitudinal ulcer on the lesser curvature of the gastric body (Fig. 1). The histopathologic examination of the biopsy specimens revealed non-caseating epithelioid granulomas in the lamina propria mucosae (Fig. 2). Acid-fast staining did not reveal any acid-fast bacilli. X-ray of the stomach demonstrated localized narrowing of the pyloric antrum (Fig. 3). Chest computed tomography, small bowel se-

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Figure 1. a: Endoscopic view of a longitudinal ulcer on the lesser curvature of the gastric body. b: Endoscopic view of erosion and narrowing of the pyloric antrum.

Figure 2. Histopathological section of the biopsy specimen showing non-caseating epithelioid granulomas in the lamina propria mucosae.

Figure 3. X-ray of the stomach showing localized narrowing of the pyloric antrum.

Discussion

Within the gastrointestinal tract, the ileocecal region is the most common site for intra-abdominal tuberculosis, possibly because it contains a considerable amount of lymphoid tissue and is an area of relative fecal stasis (1). Primary and isolated gastric tuberculosis without evidence of lesions elsewhere is exceedingly rare due to the bactericidal properties of gastric acid, the scarcity of lymphoid tissue in the mucosa, and the rapid emptying of gastric contents (4, 6). Possible routes of infection include direct infection through the mucosa, hematogeneous spread, lymphatic spread, or contiguous spread through the serosa from adjacent structures (4-7). Although direct infection through the mucosa is considered to be the most likely route in primary gastric tuberculosis (7), the routes of infection to the stomach could not be identified in the present case. Although tuberculosis usually involves the antral region, involvement of the prepy-
The endoscopic appearance may be ulcerative, hypertrophic, or a combination of these findings (1, 3). In previous reports, hypertrophic lesions are the most common finding in gastric tuberculosis, sometimes resulting in gastric outlet obstruction (3, 4, 6, 7, 10). On the other hand, several cases of gastric tuberculosis presenting as non-healing ulcers have also been reported (8, 9). In the present case, both hypertrophic and erosive findings were seen in the antrum and the longitudinal ulcer, recalcitrant to proton pump inhibitor therapy, was observed on the lesser curvature of the gastric body. Thickening of the antrum and longitudinal ulcerations are considered common endoscopic features of the stomach in Crohn’s disease (13). In addition, sarcoidosis lesions may also involve the antral region resulting in gastric outlet obstruction due to narrowing of the antrum, although these changes are usually found along with lung, liver, bone-marrow, and parotid gland involvement (14). Therefore, the challenging differential diagnoses of gastric tuberculosis, Crohn’s disease, and sarcoidosis were all considered due to the similarity of endoscopic findings in this case.

The diagnosis of gastric tuberculosis can be made on histopathological examination, which shows caseating epitheloid cell granulomas or bacteriologic identification of Mycobacterium tuberculosis. In the present case, granulomas were non-caseating and staining for acid-fast bacilli was negative on biopsy specimens. Granulomas are generally present in the submucosal layer and subserosa and a nonspecific inflammatory reaction is seen in the mucosa. These biopsy findings are considered reasons for failure of endoscopic diagnosis and many patients are subsequently subjected to surgical intervention for a definitive diagnosis (5). Although non-caseating granulomas are shown on the pathological specimens in Crohn’s disease and sarcoidosis, they can also be seen in gastric tuberculosis (4, 9). Therefore, the differential diagnosis of gastric tuberculosis, Crohn’s disease, and sarcoidosis could not be refined even after pathological and endoscopic examinations. Immunosuppressive therapy which is considered to be contraindicated in tuberculosis is needed for treatment of both Crohn’s disease and sarcoidosis and it presented a management dilemma in this case. Therefore, initial empiric antituberculous therapy was attempted. Because antituberculous therapy was recommended after consultation with an infectious disease specialist, the side-effects of therapy were minimized and a safe and effective course of therapy for gastric tuberculosis could be pursued.

In conclusion, although collateral evidence was needed, diagnostic antituberculous therapy was considered effective for treating primary gastric tuberculosis presenting as a non-healing ulcer and mimicking Crohn’s disease, which could not be diagnosed by endoscopic biopsies.

The authors state that they have no Conflict of Interest (COI).

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