Gastric Inflammatory Fibroid Polyp Treated with *Helicobacter pylori* Eradication Therapy

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Abstract

Gastric inflammatory fibroid polyps (IFPs) are rare benign lesions that occur in the distal stomach. We describe a 56-year-old woman with *Helicobacter pylori* (*H. pylori*)-positive gastric IFP treated with eradication. Endoscopic examination revealed a submucosal tumor, 35 mm in diameter, with an ulcerated apex at the antrum. *H. pylori* were positive by both histology and tissue culture, and eradication (a proton pump inhibitor, amoxicillin, and clarithromycin) was performed. After 6 months, the tumor morphologically changed and decreased in size. This case suggests that *H. pylori* may play a role in the pathogenesis of gastric IFPs.

Key words: gastric submucosal tumor, inflammation, endoscopic biopsy

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Introduction

Inflammatory fibroid polyp (IFP) is a relatively rare disorder, which is thought to be clinically and histologically benign, and was first described as “polypoid fibroma” in 1920 by Konjetzny (1). It can appear in many different locations throughout the gastrointestinal tract. The most common site is the gastric antrum (about 70% of cases), followed by the small bowel (about 20% of cases) (2). Gastric IFP often appears as an incidental finding on examination of the upper digestive tract, and this tumor might sometimes be accompanied by adenocarcinoma of the stomach (3-5). Recently, several reports have suggested the relationship between gastric IFP and *Helicobacter pylori* (*H. pylori*) (6, 7). Herein, we describe a case of gastric IFP that morphologically changed and decreased in size after *H. pylori* eradication therapy.

Case Report

A 56-year-old woman visited our hospital for a follow-up study of gastric submucosal tumor (SMT). She had been diagnosed with gastric SMT in the antrum 10 years previously, but we could not confirm the histological diagnosis of SMT because the biopsy specimen obtained repeatedly from the lesion revealed normal gastric mucosa. No specific family history was identified. Her body temperature was 36.4°C,

blood pressure was 126/78 mmHg, and radial pulse rate was 66 beats/min and regular. She had neither anemia nor jaundice. A neurological examination revealed no abnormal findings and there was no lymphadenopathy. No specific family history was identified. Routine hematological examination and biochemical tests were within normal limits. Serum anti-*H. pylori* immunoglobulin G (IgG) antibody was positive. Endoscopic examination of the upper digestive tract revealed an SMT with an ulcerated apex, about 35 mm in diameter, in the pyloric gland area, in the anterior wall of the antrum (Fig. 1). She had undergone endoscopic examination the year before, and the SMT was about 25 mm in size at that time (Fig. 2). A biopsy specimen obtained from the ulcer bottom of the lesion showed the proliferation of fibroblasts (Fig. 3A) and the infiltration of inflammatory cells such as plasma cells and eosinophils (Fig. 3B). These findings were compatible with the histological diagnosis of IFP. There were some red patches with erosions in the antrum; however, there was no diffuse red area in the fundic region. The culture of gastric mucosa propagated microaerophilic bacteria, *H. pylori*. Her gastric IFP had increased in size over the previous year, but she was not willing to have the tumor resected. Thus, she underwent *H. pylori* eradication with triple therapy (a proton pump inhibitor, amoxicillin, and clarithromycin) under informed consent. Eradication therapy included the oral administration of a proton-pump inhibitor (lansoprazole 30 mg) associated with amoxicillin

Figure 1. Endoscopic examination revealed a submucosal tumor (SMT) with an ulcerated apex, about 35 mm in diameter, in the pyloric gland area, in the anterior wall of the antrum.

Figure 2. Endoscopic examination one year previously revealed a submucosal tumor (SMT) of about 25 mm in diameter, in the pyloric gland area, in the antrum.

(750 mg) and clarithromycin (400 mg) twice daily for one week. After 6 months, *H. pylori* were negative by tissue culture, and the urea breath test was also negative. Endoscopic examination 6 months after *H. pylori* eradication therapy showed marked changes in the morphological features and size of the tumor. The cure of *H. pylori* infection made this tumor thin and center of IFP became depressed (Fig. 4).

Discussion

IFP is a rare mucosal or submucosal lesion of the gastrointestinal tract that follows a benign course. Most of the fi-

Figure 3. Microscopic findings of the biopsy specimen obtained from the tumor ulcer. (A) Proliferation of fibroblasts was seen and the fibroblasts presented whorl patterns around the small vessels (HE stain, ×20). (B) Inflammatory cells such as plasma cells and eosinophils were also seen (HE stain, ×50).

Figure 4. Endoscopic examination 6 months after *H. pylori* eradication therapy revealed marked changes in the morphological features and size of the tumor. The tumor became thin and center of inflammatory fibroid polyp (IFP) developed a depressed shape.

broids reported are located in the mucosa and submucosa, although Ishikura et al reported six lesions (8) and we also reported one lesion limited to the mucosa (5). Endoscopic findings of IFPs are smooth sessile or pedunculated polyps. The final diagnosis of IFP depends on the pathological findings; however, the histological findings of the biopsy specimen are often difficult to diagnose (5, 9). In the present case, the diagnosis of gastric IFP was made for the histological findings of the biopsy specimen obtained from the ulcer bottom of the SMT.

The pathogenesis of IFP remains unknown; however, some authors have proposed that IFP is caused by an allergic reaction to inflammatory stimulus such as bacterial, chemical traumatic, etc, or is a reactive lesion of fibroblastic or myofibroblastic nature (10). Gastric IFP frequently appears in the antrum, and the incidence of gastric IFP was reported to be 3.1% in one series of 5515 gastric polyps by Stolte et al (11). Recently, Nishiyama et al (6) reported a case of IFP that morphologically changed after *H. pylori* eradication therapy. They claimed that factors derived from gastric epithelial cells in response to *H. pylori* infection, such as inflammatory cytokines and growth factors, might affect the growth of IFPs. Their theory is not proven yet, but there is another report describing the relation between IFP and *H. pylori* infection (7). It is interesting that their report suggested a relationship between gastric IFP and *H. pylori* infection.

As gastric IFP is a benign tumor, it is seldom resected by endoscopic resection or surgery; however, IFPs are sometimes treated with endoscopic mucosal resection (EMR) or endoscopic submucosal dissection (ESD) if the tumor is accompanied by malignant lesion (5) or is increasing in size (12). We reported two cases of gastric IFPs resected by ESD, one case accompanied by gastric carcinoma and an-

other case accompanied by gastritis cystica polyposa (GCP) (9). In this case, the tumor had shown a tendency to enlarge in a year, but she was not willing to have the tumor resected. The cure of *H. pylori* infection decreased the size of gastric IFP. Recently, the relationship between *H. pylori* and several gastric diseases such as gastric ulcer (13) or gastric mucosa-associated lymphoid tissue lymphoma (14) has been discussed and clarified. Further studies on the relationship between IFP and *H. pylori* are certainly required. If we could confirm the theory that the cure of *H. pylori* infection decreases the size of gastric IFP, endoscopic resection of IFP might be unnecessary because IFP is a submucosal lesion of the gastrointestinal tract that follows a benign course (5, 9).

We should be aware of complications of adenocarcinoma or adenoma with gastric IFP. Mori et al described that 8% of their 50 patients with gastric IFP were accompanied by an adenocarcinoma or adenoma in the same area (3). Kolodziejczyk et al (15) reported 3 cases with gastric IFP in which a carcinoma was overlying or immediately adjacent to the IFP. Thus, if a patient has a gastric IFP, we must take care to search for small gastric neoplasms during endoscopic examinations. Endoscopic resection of IFP is unnecessary unless IFP increases in size or associates with gastric neoplasm, but periodic follow-up endoscopy and careful observation is necessary in treating patients with gastric IFP. In addition, if the patient has a gastric IFP, we must investigate *H. pylori* infection taking into consideration the relation between gastric IFP and *H. pylori* infection.

In conclusion, we reported a case of gastric IFP that morphologically changed and decreased in size after the cure of *H. pylori* infection. Based on the findings in the present case, we suggest that *H. pylori* infection may be related to gastric IFP in etiology.

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