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 70-year-old woman with *Helicobacter pylori*-positive gastric IFP treated with eradication. Gastroduodenal endoscopy revealed a pyramidal-shaped, broad-based tumor with an ulcerated apex at the antrum. *Helicobacter pylori* was positive by both histology and tissue culture, and eradication (a proton pump inhibitor, amoxicillin, and clarithromycin) was performed. After 3 months, the tumor morphologically changed and the apex ulcer was markedly enlarged. This case suggests that *H. pylori* may play a role in the pathophysiology of IFPs.

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Key words: gastric epithelium, inflammation, cytokine

Introduction

Inflammatory fibroid polyps (IFPs) are rare benign lesions that occur in a variety of sites in the gastrointestinal tract, but are most common in the distal stomach and distal ileum (1–4). It is not clear whether IFPs are neoplasms or an inflammatory phenomenon, and the role of *Helicobacter pylori* (*H. pylori*) infection in its etiology remains unclear. In this paper, we report a case of gastric IFP which revealed a marked morphological change in response to *H. pylori* eradication.

Case Report

A 70-year-old woman presented to the Shiga University Hospital for management of a gastric tumor discovered dur-

ing an evaluation for anemia. The laboratory data indicated mild anemia with a hemoglobin level of 7.7 g/dl. Gastroduodenal endoscopy revealed a pyramidal-shaped, broad-based tumor with an ulcerated apex on the posterior wall of the antrum (Fig. 1A and B). Biopsies showed fragments of fibrous tissue with hemorrhage, edema and necrosis. The mucosa of both the antrum and the body showed active chronic inflammation, and several hyperplastic polyps (diameter 0.2–0.5 mm) were presented in the body (Fig. 1C). *H. pylori* was positive by both histology and tissue culture. The ¹³C-urea breath test was positive. Barium contrast radiographs showed a round-shaped filling defect at the antrum (Fig. 2A). Endoscopic ultrasonography (EUS) revealed a homogeneous hypoechoic lesion arising from the second echolayer (muscularis mucosae) of the gastric wall (Fig. 2B).

Since bleeding tendency of unknown etiology was observed at that time, we performed *H. pylori* eradication with triple therapy (a proton pump inhibitor, amoxicillin, and clarithromycin) under informed consent. Endoscopy indicated a similar finding at this time (Fig. 1D). After 3 months of eradication, *H. pylori* was negative by tissue culture, and the urea breath test was also negative. The 24-h gastric pH monitoring indicated that percentage of holding time under pH 3 was 39.0% before and 67.1% after eradication. Gastroduodenal endoscopy showed marked changes in the morphological features of the tumor (Fig. 3A and B). The apex ulcer was markedly enlarged. Resolution of the active chronic gastritis was observed and hyperplastic polyps had disappeared or were reduced in size (Fig. 3C). Based on an improvement in the bleeding tendency, the tumor was removed endoscopically to obtain a histological diagnosis. Endoscopic resection was performed by using polypectomy snare. Histological examinations revealed that the tumor rose from muscularis mucosae and was characterized by the infiltration of chronic inflammatory cells, increased fibroblasts and collagen fibers (Fig. 4). These findings are compatible with the histological diagnosis of inflammatory fibroid polyp. The re-

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Figure 1. Endoscopic view of the lesion on the posterior wall of the antrum (A and B) and the body (C). Seven days after, endoscopy was performed again (just before eradication, D).

section was incomplete, but 6 months after endoscopic study no sign of recurrence was seen (Fig. 3D).

Discussion

The IFP is a localized, non-neoplastic growth of the gastrointestinal wall. Stolte et al showed that gastric IFPs made up approximately 3% of one series of about 5,500 gastric polyps (4). IFPs frequently appear in the antrum, but sometimes occur in other regions (1, 4). They are characterized by non-capsulized, distinctively arranged fibrous tissue and blood vessels, with inflammatory cell infiltration dominated by eosinophils (1). Even in the light of recent studies employing on electron microscopy and immunohistochemistry, the exact origin of the main cellular component of IFP remains unclear, and many studies suggest that the majority of cells found in IFPs are derived from myofibroblasts or

fibroblasts (5–7).

The etiology and pathogenesis of IFPs remain unclear. Some investigators suggested the involvement of inflammatory mechanisms or reactive responses rather than a neoplastic growth (1). As was postulated in previous reports, the proliferation of mucosal fibroblasts or myofibroblasts in response to a variety of factors in the lumen, such as bacteria, chemicals and mechanical factors, might be involved in the pathogenesis (1). This might be responsible for its location in the stomach, because the antrum is exposed more than other sites due to various mechanical, biological and chemical factors. In this process, an ulceration or a defect in the epithelium could serve as easy access point for these factors to invade to mucosal fibroblasts and/or myofibroblasts. This concept is supported by a case described by Matsushita et al (3). They reported the accelerated growth of IFPs after incomplete resection by endoscopy, and speculated that the

Figure 2. Barium contrast radiograph (A) and endoscopic ultrasonography (B).

increased chance of the contact between luminal factors and the proliferating cells of the IFP might enhance proliferative response (3). Thus, one of the possible mechanisms responsible for the etiology of IFPs is that some luminal factors might continuously stimulate the growth of mucosal fibroblasts and/or myofibroblasts via an epithelial defect, and might induce the focal growth of these cells.

In the present case, *H. pylori* was abundant, and the gastric mucosa was severely inflamed with hyperplastic polyps. Previous studies have demonstrated that *H. pylori* infection is closely associated with chronic gastritis and hyperplastic polyps (8, 9), based on the efficacy of *H. pylori* eradication therapy in these gastric disorders. In our case, we observed the disappearance of active gastritis and the improvement of multiple hyperplastic polyps after the elimination of *H. pylori*. One interesting observation is that IFPs exhibited a marked morphological change in response to the eradication of *H. pylori*. This finding suggests that *H. pylori* infection may affect the pathophysiology of IFPs. In our case, the ulceration at the apex of the IFP was present. As mentioned above, this might serve a direct route of contact of *H. pylori* to the major proliferating cells of the tumor, and might stimulate their growth. Factors generated by *H. pylori*, such as cytotoxins, could be considered as stimulants (10). From another view point, myofibroblasts or fibroblasts are a local source of various growth factors for themselves, such as fibroblast growth factor (FGF) (11). So, direct contact of *H. pylori* to proliferating myofibroblasts might induce the secretion of growth factors that affect their growth via autocrine fashion.

Furthermore, gastric epithelial cells are the direct targets

of *H. pylori* infection (12, 13). It has been reported that *H. pylori* stimulates the secretion of various factors from gastric epithelial cells, such as inflammatory cytokines and growth factors (13). Factors derived from gastric epithelial cells in response to *H. pylori* infection might affect the growth of IFPs.

We must pay attention to the influences of improved acid secretion on the morphological changes of tumors. In the present case, the 24-h gastric pH monitoring indicated that gastric acidity was improved after *H. pylori* eradication. So, it is possible to consider that improved acidity might induce an enlargement of the apex ulcer of the tumor.

This report suggested a possibility that *H. pylori* may play a role in the pathophysiology of IFPs. Further studies using an increased number of cases are needed to define the proposed active role of *H. pylori* in the pathogenesis of IFPs.

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Figure 3. Endoscopic view of the lesion on the posterior wall of the antrum (A and B) and the body (C). After 6 months, there was no sign of recurrence (D).

Figure 4. Histologic appearance of the lesion. It shows the perivascular arrangement of the spindle-shape cells and inflammatory infiltrate composed of lymphocytes and eosinophils (HE stain, ×40).

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