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Morphological Changes in Gastric Cancer after *Helicobacter pylori* Eradication Therapy

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Abstract

A 63-year-old woman visited her local doctor complaining of acid regurgitation. Esophagogastroduodenoscopy revealed a protruding lesion with a modulated mucosal surface in the gastric antrum, with mucosal biopsies indicating well-differentiated adenocarcinoma. Because of a positive urea breathe test, while the patient was waiting for elective surgery she underwent *Helicobacter pylori* eradication therapy. Thirty days after eradication therapy, the patient underwent laparoscopic distal gastrectomy with regional lymph node dissection. Gross examination of the resected specimen revealed a well-circumscribed, slightly depressed lesion measuring 3.0 × 2.0 cm without elevated components. Direct effects of *H. pylori* eradication therapy on the morphological appearance of gastric adenomas and carcinomas have been reported. The findings in this patient suggest that the morphology of gastric cancer may be influenced directly by *H. pylori* eradication therapy.

Keywords: Gastric cancer; *Helicobacter pylori*; Eradication therapy

Introduction

A 63-year-old Japanese woman visited her local doctor complaining of acid regurgitation. Esophagogastroduodenoscopy (EGD) revealed early gastric cancer and the patient was referred to our hospital. Physical examination and laboratory results, including serum carcinoembryonic antigen and cancer antigen 19-9, were unremarkable. EGD using indigo carmine dye in a chromoendoscopy revealed a protruding lesion with a modulated mucosal surface in the gastric antrum, with mucosal biopsies indicating well-differentiated adenocarcinoma (Figure 1). 18F-2-Deoxy-2-fluoroglucose (FDG) positron emission tomography combined with computed tomography revealed a mass lesion with intense FDG uptake in the stomach; there was no evidence of metastatic lesions in regional lymph nodes or other organs (Figure 2, arrow).

Because of a positive urea breathe test, while the patient was waiting for elective surgery she underwent *Helicobacter pylori* (*H. pylori*) eradication therapy with a 1-week triple regimen consisting of a proton pump inhibitor, clarithromycin, and amoxicillin. Thirty days after eradication therapy, the patient underwent laparoscopic distal gastrectomy with regional lymph node dissection. Gross examination of the resected specimen before formalin fixation revealed a well-circumscribed, slightly depressed lesion measuring 3.0 × 2.0 cm without elevated components (Figure 3, arrows). Microscopic examination of the tumor confirmed the diagnosis of well-differentiated adenocarcinoma confined to the gastric mucosal layer accompanied by lymphatic follicles with no lymphatic or venous invasion or lymph node metastasis (Figure 4). There were no significant differences of histological features between before and after eradication. The serum antibody of *H. pylori* was negative, and no *H. pylori* organisms were detected in the resected specimens. Follow-up after discharge was uneventful and there was no evidence of recurrence 12 months after surgery.

Discussion

H. pylori infection is known to induce a robust cellular and humoral response comprised of infiltration of neutrophils, eosinophil's, plasma cells, and lymphocytes, followed by gastric epithelial cell damage [1]. Because *H. pylori* infection is the most commonly proven risk factor for gastric cancer in humans, eradication of *H. pylori* has been shown to prevent gastric cancer in patients with this infection [1,2]. Direct effects of *H. pylori* eradication therapy on the morphological appearance of gastric adenomas and carcinomas have been reported, including typical changes to elevated lesions on endoscopy, regardless of tumor histology, of a flattening of the tumor and a switch to indistinct borders [3]. Therefore, clinicians should take this possibility of morphological change associated with *H. pylori* eradication therapy into consideration.

In the present case, the gross appearance of the gastric cancer changed dramatically from an elevated to depressed lesion over a short period of time after administration of *H. pylori* eradication therapy. Although we could not detect significant differences of histological

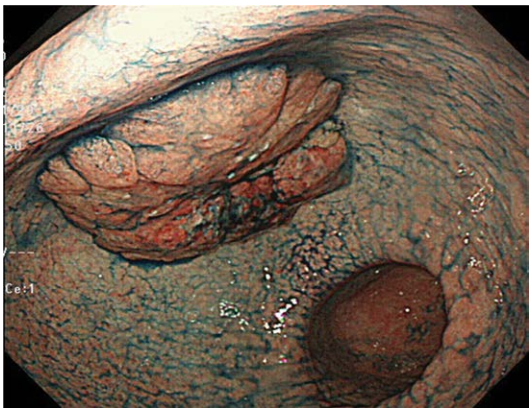

Figure 1: Esophagogastroduodenoscopy using indigo carmine dye revealed an elevated tumor in the stomach.

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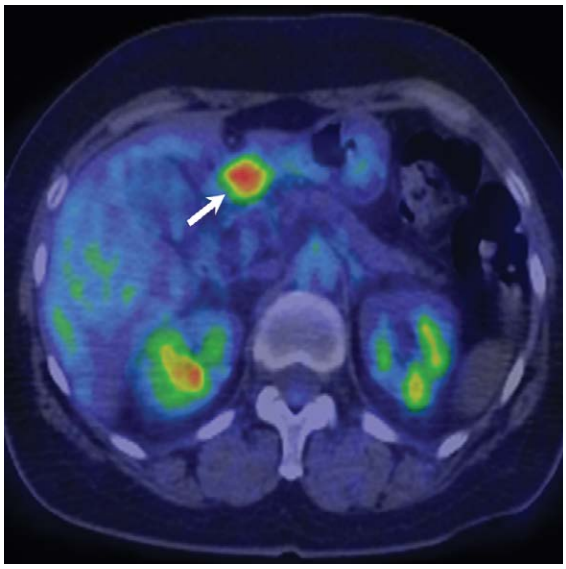

Figure 2: ^{18}F-2-Deoxy-2-fluoroglucose (FDG) positron emission tomography combined with computed tomography revealed a mass lesion with intense FDG uptakes in the stomach (*arrow*).

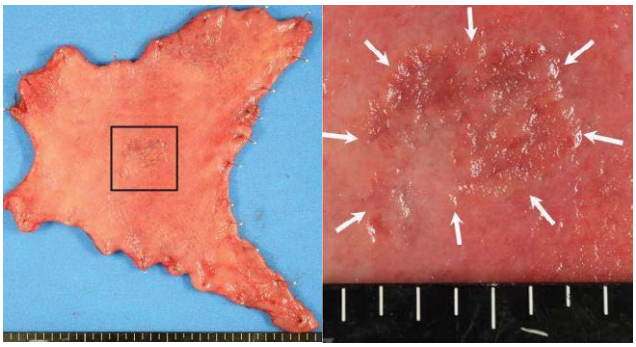

Figure 3: (a) Macroscopic appearance of the resected specimen showing gastric adenocarcinoma (*arrows*). (b) Higher-magnification image of the boxed area in (a).

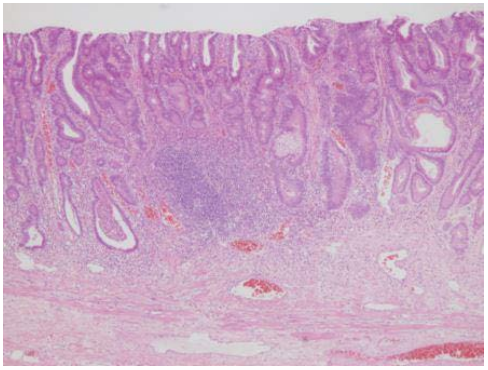

Figure 4: Hematoxylin and eosin-stained section demonstrating well-differentiated adenocarcinoma confined to the mucosa.

features caused by *H. Pylori* eradication, the morphological change might be affected by the inflammatory cell infiltration including lymphatic follicles associated with *H. Pylori* infection. The findings in this patient suggest that the morphology of gastric cancer may be influenced directly by *H. pylori* eradication therapy, possibly via inhibition of the expansive growth of the gastric tumor or reduction of *H. pylori*-induced gastritis in the background mucosa.

References

1. Kim SS, Ruiz VE, Carroll JD, Moss SF (2011) Helicobacter pylori in the pathogenesis of gastric cancer and gastric lymphoma. Cancer Lett 305: 228-238.
2. Ito M, Tanaka S, Takata S, Oka S, Imagawa S, et al. (2005) Morphological changes in human gastric tumours after eradication therapy of *Helicobacter pylori* in a short-term follow-up. Aliment Pharmacol Ther 21: 559-566.
3. Uemura N, Okamoto S, Yamamoto S, Matsumura N, Yamaguchi S, et al. (2001) *Helicobacter pylori* infection and the development of gastric cancer. N Engl J Med 345: 784-789.