Eradication rate and histological changes after *Helicobacter pylori* eradication treatment in gastric cancer patients following subtotal gastrectomy

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**Abstract**

**AIM:** To investigate the eradication rate and histological changes after *Helicobacter pylori* (*H. pylori*) eradication treatment following subtotal gastrectomy for gastric cancer.

**METHODS:** A total of 610 patients with *H. pylori* infection who had undergone surgery for either early or advanced gastric adenocarcinoma between May 2004 and December 2010 were retrospectively studied. A total of 584 patients with proven *H. pylori* infection after surgery for gastric cancer were enrolled in this study. Patients received a seven day standard triple regimen as first-line therapy and a 10 d bismuth-containing quadruple regimen as second-line therapy in cases of eradication failure. The patients underwent an esophagogastroduodenoscopy (EGD) between six and 12 mo after surgery, followed by annual EGDs. A further EGD was conducted 12 mo after confirming the result of the eradication and the histological changes. A gastric biopsy specimen for histological examination and *Campylobacter*-like organism testing was obtained from the lesser and greater curvature of the corpus of the remnant stomach. Histological changes in the gastric mucosa were assessed using the updated Sydney system before eradication therapy and at follow-up after 12 mo.

**RESULTS:** Eradication rates with the first-line and second-line therapies were 78.4% (458/584) and 90% (36/40), respectively, by intention-to-treat analysis and 85.3% (458/530) and 92.3% (36/39), respectively, by per-protocol analysis. The univariate and multivariate analyses revealed that Billroth II surgery was an independent factor predictive of eradication success in the eradication success group (OR = 1.53, 95%CI: 1.41-1.65, *P* = 0.021). The atrophy and intestinal...
INTRODUCTION

Helicobacter pylori (H. pylori) infection is a leading cause of gastric cancer[1,2] and H. pylori eradication therapy is thought to convey beneficial effects in preventing metachronous cancer after endoscopic resection of early gastric cancer[3]. As H. pylori infection remains a risk factor for malignancy after subtotal gastrectomy, several guidelines recommend H. pylori eradication therapy in patients who have undergone surgery for gastric cancer[4,5].

Several guidelines recommend H. pylori eradication in patients following surgery for gastric cancer[4,5] but its beneficial effects have not been established. In general, glandular atrophy from H. pylori infection is reversible after eradication but intestinal metaplasia (IM) cannot be improved in patients who have not undergone surgery[6,7]. Onoda et al[8] reported significant changes in glandular atrophy after eradication in the remnant stomach. However, Matsukura et al[9] determined no significant improvements in glandular atrophy or IM. Cho et al[10] reported that H. pylori eradication following subtotal gastrectomy significantly reduced both glandular atrophy and IM scores, 36 mo after eradication.

CONCLUSION: Patients with H. pylori following subtotal gastrectomy had a similar eradication rate to patients with an intact stomach. H. pylori eradication is recommended after subtotal gastrectomy.

Key words: Helicobacter pylori; Eradication; Atrophy; Intestinal metaplasia; Esophagogastroduodenoscopy; Gastrectomy

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Core tip: This is the first study to investigate the eradication rate and histological changes after Helicobacter pylori (H. pylori) eradication treatment in patients following subtotal gastrectomy for gastric cancer. The patients with H. pylori infection who had undergone a subtotal gastrectomy for gastric cancer had a similar eradication rate to patients with an intact stomach. H. pylori eradication in gastric cancer patients following subtotal gastrectomy resulted in histological improvement, especially in the Billroth II group.

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MATERIALS AND METHODS

Study population

A total of 610 patients with H. pylori infection who had undergone surgery for either early or advanced gastric adenocarcinoma at Seoul National University Bundang Hospital between May 2004 and December 2010 were retrospectively studied. The patients underwent either open or laparoscopically-assisted distal gastrectomy and either Billroth I (gastrojejunostomy) or II (gastrojejunalostomy) surgery was used for reconstruction. The exclusion criteria were as follows: (1) age below 18 years; (2) previous H. pylori eradication before diagnosis of malignancy; (3) previous gastric surgery or endoscopic resection for gastric cancer; (4) severe concurrent disease (hepatic, renal, respiratory or cardiovascular); (5) pregnancy; (6) palliative therapy; and (7) any condition probably associated with poor compliance (e.g., alcoholism or drug addiction).

All patients gave written informed consent and the study was performed according to the directions of the Declaration of Helsinki. The study protocol was approved by the Ethics Committee at Seoul National University Bundang Hospital (Institutional Review Board number: B-1306/206-109).

H. pylori infection and histological evaluation

The patients underwent an esophagogastroduodenoscopy (EGD) between six and 12 mo after surgery, followed by annual EGDs. Of the 610 patients who were offered eradication therapy, all consented to the treatment. A further EGD was conducted 12 mo after confirming the result of the eradication and the histological changes. H. pylori infection and eradication failure were defined on the basis of at least one of the following three tests: first, a positive 13C-urea breath test (13C-UBT); second, histological evidence of H. pylori by modified Giemsa staining.
in the lesser and greater curvature of the corpus of the remnant stomach; and third, a positive rapid urease test (CLOtest; Delta West, Bentley, Australia) in gastric mucosa biopsy samples from the lesser and greater curvatures of the corpus of the remnant stomach. An endoscopic specialist performed the biopsies and described the endoscopic findings (Dong Ho, Lee). A gastric biopsy specimen for histological examination and Campylobacter-like organism testing was obtained from the lesser and greater curvature of the corpus of the remnant stomach and immediately fixed in formalin. The tumor location was determined with reference to the Japanese Classification of Gastric Cancer[11]. The degree of atrophy and IM, polymorphonuclear neutrophil activity and mononuclear cell count were graded according to the updated Sydney system (0 = none, 1 = mild, 2 = moderate and 3 = marked)[12].

**H. pylori eradication regimen**

All the patients received seven day standard triple therapy [amoxicillin 1000 mg twice a day (b.i.d.), clarithromycin 500 mg b.i.d. and esomeprazole 20 mg b.i.d.] as first-line therapy. Patients who failed to respond to first-line therapy received a 10 d bismuth-containing quadruple regimen [tripotassium dicitratobismuthate 300 mg four times a day (q.i.d.), tetracycline 500 mg q.i.d., metronidazole 500 mg three times a day and esomeprazole 20 mg b.i.d.] as second-line therapy, with a subsequent 13C-UBT for evaluation of eradication. Patients who took < 85% of the prescribed medication were considered to have low compliance.

**Statistical analysis**

*H. pylori* eradication rates were determined using intention-to-treat (ITT) and per-protocol (PP) analyses. ITT analysis compared treatment groups, including all the patients as originally allocated. PP analysis compared treatment groups, including only those patients who completed the treatment as originally allocated. The mean ± SD was calculated for quantitative variables. The Student's *t* test was used to evaluate continuous variables and the χ² test and Fisher's exact test were used to assess non-continuous variables. Additionally, univariate and multivariate analyses were conducted to assess the effects of factors on the eradication rate. Updated Sydney system scores were compared using the Wilcoxon signed-rank test and Mann-Whitney *U* test for unpaired data. All of the statistical analyses were performed using Predictive Analytics Software (PASW) version 20.0 for Windows (SPSS Inc., IBM, Chicago, IL, United States). A *P* value of less than 0.05 was defined as carrying statistical significance. The statistical methods of this study were reviewed by Medical Research Collaborating Center at Seoul National University Bundang Hospital.

**RESULTS**

**Characteristics of patients**

A schematic diagram of the study is provided in Figure 1. Of the 610 patients, 26 were excluded from the study because of previous *H. pylori* eradication before diagnosis of malignancy (nine patients), endoscopic submucosal dissection before surgery (nine patients), palliative surgery (four patients), liver cirrhosis (three patients) and chronic renal failure (one patient). A total of 584 patients underwent first-line eradication treatment. Forty-four were lost to follow-up and 10 had low treatment compliance. Eradication was surveyed using a 13C-UBT in all the patients after treatment but histological changes were surveyed in only 326 patients after 12 mo. The remaining 204 patients were not examined for histological changes after eradication because they were lost to follow-up. Finally, 326 patients had histological changes of their gastric mucosa analyzed after eradication of *H. pylori*. Of these, 290 patients in whom *H. pylori* was successfully eradicated with first-line therapy were assigned to the eradication success group and 36 patients in whom first-line therapy failed were assigned to the eradication failure group. In patients in the eradication failure group, second-line therapy was used to eradicate *H. pylori* infection. However, this was not successful in most patients because of either poor compliance or adverse events. The mean ages of the eradication success and failure groups were 56.7 ± 10.4 and 56.9 ± 10.7 years (*P* = 0.124), respectively. The enrolled patients’ baseline demographic and clinical characteristics are provided in Table 1. The eradication rate of patients who underwent Billroth II surgery (96.7%, 89/92) was significantly higher than that of patients who underwent Billroth I surgery in the eradication success group (85.8%, 201/234, *P* = 0.012). There were no statistically significant differences in gender distribution, smoking status, alcohol use, underlying disease, early gastric cancer/advanced gastric cancer or tumor location between the two groups (*P* > 0.05).

**H. pylori eradication rates of first- and second-line therapy**

Table 2 shows the rates of eradication of *H. pylori* infection according to the ITT and PP analyses. The eradication rates of first-line therapy by ITT and PP analyses were 78.4% (95%CI: 74.9-81.6%) and 85.3% (95%CI: 82.1-88.1%), respectively. Forty of the 72 patients who failed first-line therapy underwent second-line treatment and their eradication rates by ITT and PP analyses were 90% (95%CI: 77.0-96.0%) and 92.3% (95%CI: 79.7-97.4%), respectively.

**Clinical factors influencing *H. pylori* eradication**

To evaluate the clinical factors influencing the efficacy of *H. pylori* eradication, univariate analyses were performed.
performed, which are listed in Table 1. The eradication rate of patients who underwent Billroth II surgery was significantly higher than that of patients who underwent Billroth I surgery \( (P = 0.012) \). The other factors did not affect the eradication response. The multivariate analysis revealed that Billroth II surgery \( (OR = 1.53, 95\% CI: 1.41-1.65, P = 0.021) \) was an independent factor predictive of eradication success in the eradication success group (Table 3).

**Sequential histological changes after eradication**

Table 4 shows the histological changes of the gastric mucosa according to eradication. Histological changes were compared before and after eradication and scored according to the updated Sydney system. The atrophy scores were significantly lower than the baseline after eradication in the eradication success group \( (0.25 \pm 0.04 \text{ vs } 0.46 \pm 0.04, P < 0.001) \). The atrophy scores were not significantly different compared to the baseline after eradication in the eradication failure group \( (0.51 \pm 0.12 \text{ vs } 0.56 \pm 0.14, P = 0.226) \). The IM scores after eradication were significantly lower in the eradication success group than in the eradication failure group \( (0.27 \pm 0.04 \text{ vs } 0.51 \pm 0.12, P = 0.015) \). Activity and chronic inflammation scores decreased in all the groups. In metachronous cancer patients \( (n = 7) \), the atrophy and IM scores were lower than the baseline after eradication but the differences were not statistically significant \( (P > 0.05; \text{Table 5}) \).

**Sequential histological changes according to reconstructive surgery method**

Table 6 shows the histological changes of the gastric mucosa according to reconstructive surgery method after successful eradication. Thirty-six eradication failure patients were excluded to analyze the histological changes of the gastric mucosa after successful eradication between the Billroth I and Billroth II groups. The atrophy scores were significantly lower than the baseline after successful eradication in the Billroth II group \( (0.13 \pm 0.09 \text{ vs } 0.53 \pm 0.19, P < 0.001) \). The atrophy scores were not significantly different compared to the baseline after successful eradication in the Billroth I group \( (0.31 \pm 0.12 \text{ vs } 0.33 \pm 0.10, P = 0.831) \). The atrophy scores after successful eradication were significantly lower in the Billroth II group than in the Billroth I group \( (0.13 \pm 0.09 \text{ vs } 0.31 \pm 0.10, P = 0.021) \).
Atrophy or IM scores after eradication in the remnant stomach, whereas another study showed significant improvements in glandular atrophy compared with normal mucosa. Second, it is unknown whether the eradication rate differs between patients who have undergone and those who have not undergone gastric surgery and whether the eradication rate in patients after surgery decreases as in the current study, because histological improvement was significantly higher in the eradicated group.

In this study, we evaluated histological changes, particularly glandular atrophy and IM, and the eradication rate of *H. pylori* infection in patients who underwent a subtotal gastrectomy for gastric cancer. Our data indicated that only the eradication success group had significantly improved glandular atrophy and IM scores 12 mo after treatment, suggesting the importance of *H. pylori* eradication rate. The eradication rate in patients who underwent Billroth II surgery was significantly higher than that of patients who underwent Billroth I surgery. The multivariate analysis revealed that Billroth II surgery was an independent factor predictive of eradication success in the eradication success group. The atrophy and IM scores were significantly lower than the baseline after eradication in the eradication success group. The atrophy and IM scores after eradication were significantly lower than the baseline after successful eradication in the Billroth II group. These results suggest that *H. pylori* eradication would result in histological improvement in patients who underwent surgery for gastric cancer.
Hwang JJ et al. Gastric cancer and *H. pylori* infection

### Table 4 Histological changes of gastric mucosa according to eradication

|                      | Baseline | 12 mo | *P* value |                      | Baseline | 12 mo | *P* value |                      |
|----------------------|----------|-------|-----------|----------------------|----------|-------|-----------|----------------------|
| Atrophy              | 0.46 ± 0.04 | 0.25 ± 0.04 | < 0.001   |                      | 0.50 ± 0.11 | 0.47 ± 0.12 | 0.698 | 0.023 |
| IM                   | 0.41 ± 0.05 | 0.27 ± 0.04 | < 0.001   |                      | 0.56 ± 0.14 | 0.51 ± 0.12 | 0.226 | 0.015 |
| Neutrophil count     | 2.12 ± 0.04 | 0.40 ± 0.04 | < 0.001   |                      | 2.08 ± 0.12 | 1.11 ± 0.18 | < 0.001 | < 0.001 |
| Mononuclear cells    | 2.30 ± 0.03 | 1.45 ± 0.03 | < 0.001   |                      | 2.08 ± 0.08 | 1.64 ± 0.11 | 0.004 | 0.085 |

0 = none, 1 = mild, 2 = moderate, and 3 = marked. IM: Intestinal metaplasia.

### Table 5 Comparison of histological changes of gastric mucosa in metachronous cancer patients (*n* = 7)

|                      | Baseline | 12 mo | *P* value |
|----------------------|----------|-------|-----------|
| Atrophy              | 1.29 ± 0.42 | 0.71 ± 0.36 | 0.436 |
| Intestinal metaplasia| 1.34 ± 0.41 | 1.00 ± 0.38 | 0.457 |
| Neutrophil count     | 2.00 ± 0.22 | 0.86 ± 0.34 | 0.071 |
| Mononuclear cells    | 2.00 ± 0.31 | 1.85 ± 0.20 | 0.289 |

0 = none, 1 = mild, 2 = moderate, and 3 = marked.

especially Billroth II surgery.

At least 50% of the Korean population has a *H. pylori* infection and its eradication rate with standard triple therapy is reported to be between 72.5% and 83.7%[21]. A 14 d bismuth-containing quadruple therapy as the second-line treatment resulted in an eradication rate of 82.6%–93.6% in Korea[22]. After surgery, the efficacy of eradication therapy varies from 70% to 90%[9,23]. In our results, the eradication rates of the seven day standard triple therapy as first-line therapy and the 10 d bismuth-containing quadruple therapy as second-line therapy were 78.4% and 90%, respectively, using ITT analysis and 85.3% and 92.3%, respectively, using PP analysis. Our data suggest a slightly higher result than previous studies. As histological improvement was seen only in the *H. pylori* negative group, the eradication rate is important[8,10]. In our results, there were also significant histological improvements in the eradication success group. Therefore, *H. pylori* eradication following subtotal gastrectomy might lead to histological improvement.

Billroth II surgery has been reported to result in a higher reflux rate than Billroth I surgery[24] and bile reflux plays a role in the eradication of *H. pylori* after subtotal gastrectomy. Rino et al[25] reported that the overall rate of *H. pylori* infection was 37.1%; 39.6% in Billroth I reconstruction, 0% in Billroth II reconstruction and 55.6% in pylorus-preserving gastrectomy. We hypothesized that the reconstructive surgery method would influence eradication and histological changes.

In our study, atrophy and IM scores were significantly lower than the baseline after successful eradication in the Billroth II group. The atrophy and IM scores after successful eradication were also significantly lower in the Billroth II group than in the Billroth I group, suggesting that the reconstructive surgery method influences eradication and histological changes. Previous studies have shown that the *H. pylori* infection rate was significantly lower in Billroth II patients than Billroth I due to the role of bile reflux which interferes with *H. pylori* colonization. If *H. pylori* is still left after Billroth II surgery, the gastric carcinogenesis process is promoted because of the synergistic effect of bile reflux and *H. pylori* infection[24-26]. Therefore, *H. pylori* eradication should be strongly recommended following subtotal gastrectomy, especially in the Billroth II group.

Metachronous cancer developed in seven patients in this study and all of these were patients in which *H. pylori* eradication had been successful. The atrophy and IM scores were lower than the baseline after eradication but the differences were not statistically significant. However, the atrophy and IM scores in this group were higher than the mean for all the patients and increased after eradication. These results indirectly indicate that *H. pylori* eradication alone does not ensure prevention of metachronous cancer after surgery.

This study had several limitations. First, due to its retrospective nature at a single center, only two thirds of the patient population underwent histological examination after eradication and only 40 of the 72 patients who failed first-line treatment went on to second-line eradication. Second, the follow-up time to evaluate the changes in atrophy and IM was relatively short.

Although our study was limited by its retrospective nature, we enrolled a large number of patients and evaluated the eradication rate of second-line treatment and histological changes according to eradication and reconstructive surgery method. The patients with *H. pylori* infection who underwent subtotal gastrectomy for gastric cancer had a similar eradication rate when compared with the patients with an intact stomach. The eradication success group showed histological improvement in glandular atrophy and IM. After successful eradication, the Billroth II group showed a significant decrease in atrophy and IM scores over the Billroth I group. Therefore, *H. pylori* eradication is needed for these patients and more active treatment is required in the Billroth II group. Our study may support the recommendation that *H. pylori* should be treated even after gastrectomy for gastric cancer, especially after Billroth II reconstruction.
eradication does not ensure prevention of metachronous cancer after successful eradication, the Billroth II group showed a significant decrease in atrophy and IM scores compared with the Billroth I group. So, they should make H. pylori eradication an important factor in the treatment of patients following Billroth II reconstruction.

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