

Do We Need to Retest of *Helicobacter pylori* Infection after Gastric Cancer Surgery?

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See “Dynamic Changes in *Helicobacter pylori* Status Following Gastric Cancer Surgery” by Kichul Yoon, et al. on page 209, Vol. 11. No. 2, 2017

Helicobacter pylori is a human pathogen and can lead to chronic gastritis, peptic ulcer disease, gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma. *H. pylori* infection is associated with gastric cancer and has been categorized as a group I carcinogen in gastric cancer. In 2016, Maastricht V/Florence Consensus report emphasized *H. pylori* infection as an infectious disease and reinforced the statement that *H. pylori* eradication can cure gastritis and alter the progression to long-term complications, or recurrence of disease.^{1,2} Thus, screening and eradication for *H. pylori* is recommended in individuals at increased risk for gastric cancer.

Although *H. pylori* eradication after gastric cancer surgery is also recommended,^{1,3} it was not clear that eradication therapy could reduce the risk of metachronous gastric cancer in the remnant stomach after partial gastrectomy. A recent prospective randomized clinical trial in Korea reported that gastric cancer recurrence rate was not different according to *H. pylori* eradication treatment (5-year gastric cancer recurrence rates, 4.6% in the treatment group vs 8.5% in the placebo group; $p=0.652$).⁴ In this kind of study, it is necessary to be able to control or eliminate influences by the timing of eradication, intragastric environmental change due to gastrectomy and spontaneous regression of *H. pylori* infection in order to confirm the effectiveness of the eradication treatment.

There are several things to consider when deciding to treat. The first is the time of eradication. Postoperative eradication therapy is preferred to preoperative eradication therapy because of several reasons in clinics. Additional 1 or 2 weeks is needed for the eradication therapy before surgery. Adverse events as-

sociated with antibiotics may occur and delay the surgery. In cases with unplanned total gastrectomy, eradication therapy would not be unnecessary. The second is that even if eradication was not tried, *H. pylori* can spontaneously disappear.³ In general, *H. pylori* infection persists if not eradicated, whereas the spontaneous regression is happened in the remnant stomach. The overall spontaneous clearance rate of *H. pylori* infection after partial gastrectomy was reported 38.6% to 43%.^{4,5} Previous study reported that the spontaneous clearance rate of *H. pylori* was related to operative procedures.⁶ The Billroth-II procedure had a higher bile reflux rate and a lower *H. pylori* infection prevalence than the Billroth-I procedure. Bile reflux is a pathogenic factor in gastric remnant reflux gastritis and induces chronic histological inflammation.⁷ Third, if you decide to treat, we have to decide to re-test postoperative status of *H. pylori*. It was assumed that the change of pH and bile reflux in the remnant stomach might affect *H. pylori* colonization.^{5,8,9} Hypochlorhydric environment caused by antrectomy and increased bile reflux due to the loss of pyloric ring might inhibit the growth of *H. pylori*. And rate of *H. pylori* infection decreased over time after partial gastrectomy. A study in Taiwan reported the prevalence of *H. pylori* 1 to 15 years after surgery was 29.5%, after 16 to 30 years it was 13.6%, and after more than 30 years it was 10%.⁹ However, studies on how *H. pylori* infections change after gastrectomy are still lacking.

The article published in *Gut and Liver*, “Dynamic changes in *Helicobacter pylori* status following gastric cancer surgery” by Yoon et al.¹⁰ started from a question for clinicians about the need for retest postoperative *H. pylori* status. In this study, they

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evaluated the postoperative changes of *H. pylori* detection and analyzed the factors which affect changes of in *H. pylori* infection status after gastric cancer surgery. One hundred forty patients who underwent curative gastrectomy and had a positive *H. pylori* status without eradication therapy at the time of gastric cancer diagnosis were prospectively enrolled and annual follow-up was done to evaluate postoperative *H. pylori* status. The rate of spontaneous negative conversion at least once during follow-up was 42.9% (60/140). And of these 60 patients, eight (13.3%) showed more complex postoperative dynamic changes between negative and positive results after being confirmed as spontaneous negative conversion. The author analyzed factors associated with *H. pylori* status and demonstrated that the spontaneous negative conversion group showed a trend of more postoperative intestinal metaplasia compared to the persistent *H. pylori* group (36.7% [22/60] vs 16.3% [13/80], $p=0.001$). Unlike other previous study,¹¹ there was no significant difference between spontaneous conversion and persistently positive groups according to the surgery type which could affect the bile reflux. The peculiarities of this study were that the intestinal metaplasia of the remnant stomach had a greater effect on the presence of *H. pylori* than bile reflux which can be affected by types of surgery. The author demonstrated that these results could suggest that intestinal metaplasia could be a more important factor for the survival of *H. pylori* than bile reflux. However, this finding needs to be confirmed by further studies.

The present study showed that there was relatively high percentage of spontaneous regression and dynamic changes in status of *H. pylori* after partial gastrectomy, in case of more histologic intestinal metaplasia in the remnant stomach in postoperative period. Inaccurate diagnosis for status of *H. pylori* may lead to unnecessary eradication therapy and underestimate the protective effect of eradication therapy in patients with gastric cancer surgery. This clinical evidence could support a need to retest of postoperative *H. pylori* status before eradication therapy. However, it still cannot give answer as to how and when to test *H. pylori* after gastrectomy, more research is needed.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

REFERENCES

1. Malfertheiner P, Megraud F, O'Morain CA, et al. Management of Helicobacter pylori infection—the Maastricht V/Florence Consensus Report. *Gut* 2017;66:6-30.
2. Lee YC, Chiang TH, Liou JM, Chen HH, Wu MS, Graham DY. Mass eradication of Helicobacter pylori to prevent gastric cancer: theoretical and practical considerations. *Gut Liver* 2016;10:12-26.
3. Fock KM, Katelaris P, Sugano K, et al. Second Asia-Pacific Consensus guidelines for Helicobacter pylori infection. *J Gastroenterol Hepatol* 2009;24:1587-1600.
4. Kim YI, Cho SJ, Lee JY, et al. Effect of Helicobacter pylori eradication on long-term survival after distal gastrectomy for gastric cancer. *Cancer Res Treat* 2016;48:1020-1029.
5. Bair MJ, Wu MS, Chang WH, et al. Spontaneous clearance of Helicobacter pylori colonization in patients with partial gastrectomy: correlates with operative procedures and duration after operation. *J Formos Med Assoc* 2009;108:13-19.
6. Suh S, Nah JC, Uhm MS, et al. Changes in prevalence of Helicobacter pylori infection after subtotal gastrectomy. *Hepatogastroenterology* 2012;59:646-648.
7. Lin YS, Chen MJ, Shih SC, Bair MJ, Fang CJ, Wang HY. Management of Helicobacter pylori infection after gastric surgery. *World J Gastroenterol* 2014;20:5274-5282.
8. Kato T, Motoyama H, Akiyama N. Helicobacter pylori infection in gastric remnant cancer after gastrectomy. *Nihon Rinsho* 2003;61:30-35.
9. Abe H, Murakami K, Satoh S, et al. Influence of bile reflux and Helicobacter pylori infection on gastritis in the remnant gastric mucosa after distal gastrectomy. *J Gastroenterol* 2005;40:563-569.
10. Yoon K, Kim N, Kim J, et al. Dynamic changes in Helicobacter pylori status following gastric cancer surgery. *Gut Liver* 2017;11:209-215.
11. Cho SJ, Choi JJ, Kook MC, et al. Randomised clinical trial: the effects of Helicobacter pylori eradication on glandular atrophy and intestinal metaplasia after subtotal gastrectomy for gastric cancer. *Aliment Pharmacol Ther* 2013;38:477-489.