

Primary gastric cancer development after Helicobacter pylori eradication

Osamu Toyoshima¹, Yutaka Yamaji², Keisuke Hata³

¹Gastroenterology, Toyoshima Endoscopy Clinic, Tokyo, Japan; ²Health Development Center, Tokyo Pharmaceutical Industry Health Insurance Society, Tokyo, Japan; ³Department of Surgical Oncology, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan Correspondence to: Osamu Toyoshima. Gastroenterology, Toyoshima Endoscopy Clinic, Tokyo, Japan. Email: t@ichou.com. Response to: Tahara T, Horiguchi N, Nakagawa Y, et al. Gastric atrophy as an important risk factor for gastric cancer development after Helicobacter pylori eradication. Ann Laparosc Endosc Surg 2017;2:9.

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We greatly appreciate the comment (1) on our work (2). In Japan, patients infected with *Helicobacter pylori* (*H. pylori*) undergo eradication therapy funded by the health insurance system (3). Thus, the number of gastric cancers detected after *H. pylori* eradication is rising. It is essential to distinguish gastric cancers detected post-eradication from cancers accompanied by current infection, because the pathophysiology of the upper gastrointestinal tract changes dramatically on *H. pylori* eradication (4). The risk factors for, and clinical features unique to, gastric cancer detected after eradication must be defined, and appropriate treatment strategies urgently developed.

As previously noted, we explored risk factors for primary gastric cancer that developed after H. pylori eradication in a large-scale longitudinal cohort characterized by endoscopically revealed gastric atrophy at the time of eradication. We found that the principal risk factor for gastric cancer post-eradication was advanced gastric atrophy, and that the histology of such cancers was intestinal. It has been reported that the mucosa adjacent to the cancer at the time of cancer detection was more atrophic than gastric body mucosa (5). Based on a combination of this report and our report, it is assumed that atrophic mucosa that has not fully recovered after bacterial eradication is a major risk factor for cancer development. Further work is necessary. We agree with the comment made (in another report) that endoscopic detection of gastric cancer after H. pylori eradication can sometimes be very difficult; the morphology is challenging (6). We sought to conduct endoscopy as precisely as possible. We used sufficient sedation, we employed a dye, and we vigorously biopsied small and depressed lesions in seeking to partially overcome the difficulties associated with detection. However, further studies are needed to facilitate early detection of gastric cancer after bacterial eradication. Visibility improves after eradication, sometimes allowing detection of certain lesions that were difficult to see when an *H. pylori* infection was present; infection is associated with mucus on the gastric mucosa, enlarged folds, and/or mucosal edema (7). Therefore, we would recommend that surveillance endoscopy be scheduled for a short period (less than 1 year, if possible) after *H. pylori* eradication, to ensure that missed (but significant) lesions are detected.

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aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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References

- Tahara T, Horiguchi N, Nakagawa Y, et al. Gastric atrophy as an important risk factor for gastric cancer development after Helicobacter pylori eradication. Ann Laparosc Endosc Surg 2017;2:9.
- 2. Toyoshima O, Yamaji Y, Yoshida S, et al. Endoscopic gastric atrophy is strongly associated with gastric cancer

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- development after Helicobacter pylori eradication. Surg Endosc 2016. [Epub ahead of print].
- Asaka M, Kato M, Sakamoto N. Roadmap to eliminate gastric cancer with Helicobacter pylori eradication and consecutive surveillance in Japan. J Gastroenterol 2014;49:1-8.
- Graham DY. Helicobacter pylori update: gastric cancer, reliable therapy, and possible benefits. Gastroenterology 2015;148:719-31.e3.
- Tahara T, Shibata T, Horiguchi N, et al. A Possible Link between Gastric Mucosal Atrophy and Gastric Cancer after Helicobacter pylori Eradication. PLoS One 2016;11:e0163700.
- Horiguchi N, Tahara T, Kawamura T, et al. Distinct Clinic-Pathological Features of Early Differentiated-Type Gastric Cancers after Helicobacter pylori Eradication. Gastroenterol Res Pract 2016;2016:8230815.
- Mao T, Wang Y, Yin F, et al. Association of Endoscopic Features of Gastric Mucosa with Helicobacter pylori Infection in Chinese Patients. Gastroenterol Res Pract 2016;2016:6539639.