Helicobacter pylori infection is associated with colon adenomatous polyps detected by high-resolution colonoscopy

Shigeto Mizuno1, Yoshinori Morita2, Toshio Inui3, Akihiro Asakawa4, Naohiko Ueno5, Takashi Ando4, Haruki Kato4, Mutsumi Uchida4, Toshikazu Yoshikawa5 and Akio Inui6*

1Department of Gastroenterology, Kinki University School of Medicine, Nara, Japan
2Division of Diabetes, Digestive and Kidney Diseases, Department of Clinical Molecular Medicine, Kobe University Graduate School of Medicine, Kobe, Japan
3Inui Clinic, Moriguchi, Japan
4Matsushita Memorial Hospital, Moriguchi, Japan
5Department of Molecular Medicine, Graduate School of Medical Science, Kyoto Prefectural University of Medicine, Kyoto, Japan
6Department of Behavioral Medicine, Kagoshima University Graduate School of Medical and Dental Sciences, Kagoshima, Japan

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Helicobacter pylori (H. pylori) infects 50% of the world population. Its prevalence varies widely in different parts of the world with average rates of 40–50% in western countries, rising to >90% in the developing world.1 Compelling evidence from epidemiological, histopathological and animal studies has linked H. pylori infection to the subsequent development of gastric cancer.2 Nevertheless, the data available currently regarding a possible link between H. pylori seropositivity and colorectal cancer risk are limited and inconclusive. Presence of H. pylori is associated with excessive and prolonged release of gastrin, which has been suggested to have a role in the development of gastric cancer and, potentially, colorectal cancer.3

We examined H. pylori infection and colonic pathology data from 334 patients who underwent colonoscopy at our clinics to determine whether this infection is associated with colon neoplasia. Patients were pretreated with polyethylene glycol-electrolyte solution and received intravenous administration of scopolamine butylbromide and diazepam just before the endoscopic examination. We used high-resolution colonoscopy of nonmagnifying or mine dye was sprayed onto the areas. Magnifying colonoscopy was performed to determine whether this infection is associated with non-neoplastic lesions,9,10 but conventional colonoscopy was carried out when patients preferred it. We observed highly significant differences in adenomatous, but not hyperplastic, polyps (Table I). Importantly, hyperplastic polyps are considered to be non-neoplastic. Most colorectal cancers arise from precursor adenomatous polyps, in concurrence with the adenoma carcinoma sequence.11

Our studies have several limitations. We could not show direct association between H. pylori and colorectal cancer. The number of such patients is small because the present study was carried out as routine colorectal cancer screening. We used IgG ELISA to detect H. pylori infection. Serological testing is non-invasive and offers better sensitivity than histology, but it might be misleading in elderly patients with mucosal atrophy who may develop seroconversion.12 Even if false negative results may occur, however, the rate of H. pylori infection might have been underestimated in patients with adenomas. Although not significant, the difference in the infection rate between males and females might also have an influence on the results.

H. pylori strains that possess cag A, vac A and babA2 genes worsen gastric mucosal inflammation significantly and may be more virulent than others.13,14 Our studies do not address whether colon adenomas are also related to such virulent strains and positive and negative results were reported on cag A seropositivity and colorectal cancer risk. Host factors such as interleukin-1β or environmental factors such as salt intake are suggested to be important for gastric carcinogenesis.15 The host response induces epithelial cell proliferation through hypergastrinemia, which is reportedly associated with mitogenic effects on colonic mucosa.

*Correspondence to: Department of Behavioral Medicine, Kagoshima University Graduate School of Medical and Dental Sciences, 8-35-1 Sakuragaoka, Kagoshima 890-8520, Japan. Fax: +81-99-275-5748, E-mail: inui@kufm.kagoshima-u.ac.jp Received 23 February 2005; Accepted after revision 8 April 2005 DOI 10.1002/ijc.21280 Published online 28 June 2005 in Wiley InterScience (www.interscience.wiley.com).
sae and with an increased risk of colonic malignancy in a subpopulation of patients. The way in which \textit{H. pylori} interact with such host and environmental factors in the lumen of the large bowel to produce neoplasia remains unknown.

Eradication therapy is not recommended for all \textit{H. pylori}-infected patients. In the U.S. and Japan, the consensus for therapy is for patients with an established peptic ulcer disease. Elimination of \textit{H. pylori} apparently increases the risk of developing gastroesophageal reflux diseases (GERD) and esophageal adenocarcinoma. Our present study of higher incidence of colon adenoma in seropositive subjects suggests that these people should also be eradicated to prevent colon cancer. More studies including prospective, long-term examination of large groups of patients are needed to evaluate exactly the clinical outcomes in the colon of \textit{H. pylori} and its eradication, as well as to examine the biological basis of \textit{H. pylori}-associated neoplasia in the gastrointestinal tract.

References


\begin{table}[ht]
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\hline
\textbf{Helicobacter positive} & \textbf{Helicobacter negative} \\
\hline
Age (years) & 58.5 ± 1.18 & 62.7 ± 1.07 \\
Gender (no.) & & \\
Male & 128 & 48 \\
Female & 84 & 47 \\
Endoscopic diagnosis (no.) & & \\
Normal & 210 & 95 \\
Hyperplastic polyp & 67 (31.9%) & 52 (54.7%) \\
Tubular adenoma & 26 (12.3%) & 18 (18.9%) \\
Villous adenoma & 2 (1%) & 0 (0%) \\
Cancer & 24 (11.4%) & 7 (7.4%) \\
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\end{tabular}
\caption{Presence or Absence of Helicobacter pylori and Colon Adenomatous Polyps}
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