学 位 論 文 の 要 旨

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学 位 論 文 名	CHARACTERISTICS OF GASTRITIS IN PATIENTS WITH *HELICOBACTER PYLORI*-POSITIVE REFLUX ESOPHAGITIS
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論 文 内 容 の 要 旨

INTRODUCTION

Reflux esophagitis is caused by the reflux of acidic gastric contents into the esophagus. The prevalence of *Helicobacter pylori (H. pylori)* infection is reported to be lower in patients with, than in those without, reflux esophagitis. However, several investigators have reported that eradication therapy administered to *H. pylori*-positive patients with reflux esophagitis has improved their symptoms. In addition, the influence of *H. pylori* on gastric acid secretion differs with the status of gastritis. We therefore studied endoscopically evident gastric mucosal atrophy and the degree of histological gastritis in *H. pylori*-positive patients with reflux esophagitis, duodenal ulcer, or early gastric cancer, to clarify the characteristics of *H. pylori*-induced gastritis in patients with reflux esophagitis.

MATERIALS AND METHODS

The subjects comprised 41 *H. pylori*-positive patients with reflux esophagitis (29 men, mean age 54.1 ± 2.3 years), 41 age- and sex-matched patients with duodenal ulcer, and 41 patients with early gastric cancer. All subjects were referred to Shimane University Hospital for upper gastrointestinal endoscopy between April 1996 and March 2000, and informed written consent was obtained for upper endoscopy and biopsy procedures. Two gastric tissue samples were obtained via endoscopy from the middle antrum and middle corpus along the greater curvature. Each biopsy specimen was subjected to a rapid urease test and histological examination. Specimens for histology were subjected to hematoxylin and eosin (HE) and Warthin-Starry staining. The histological diagnosis of *H. pylori*

infection was performed on the Warthin-Starry-stained sections. All subjects were confirmed to be positive for *H. pylori* infection by the rapid urease test and/or histology.

The degree of macroscopic gastric mucosal atrophy was assessed using the classification system of Kimura and Takemoto, who categorized the grade of gastric mucosal atrophy into six groups (C1, C2, C3, O1, O2, and O3). Histological analysis was performed in accordance with the guidelines of the updated Sydney system. The grades of inflammation (mononuclear cell infiltration), activity (neutrophil infiltration), intestinal metaplasia and glandular atrophy were independently diagnosed using HE-stained specimens by three pathologists, who were unaware of the endoscopic diagnosis of the patients or the endoscopically identified atrophy. The grade of *H. pylori* density was also assessed by examination of Warthin-Starry-stained biopsy specimens, and the samples were histologically graded into one of four categories according to degree (0, absent; 1, mild; 2, moderate; 3, marked).

Statistical analysis of inter-group data was performed using the Mann-Whitney U test, when a significant difference was observed using the Kruskal-Wallis test. Chi-squared tests were applied for comparison of categorical data. Wilcoxon signed rank tests were also performed to compare the status of gastritis between the antrum and the corpus. Differences at $p<0.05$ were considered to be statistically significant.

RESULTS AND DISCUSSION

Endoscopic gastric mucosal atrophy in patients with reflux esophagitis was significantly less severe than that in patients with early gastric cancer, and was similar to that in patients with duodenal ulcer. The *H. pylori* density in the antrum of patients with reflux esophagitis tended to be higher than that of patients with early gastric cancer, and similar to that of patients with duodenal ulcer. The scores for inflammation and activity in the antrum tended to be higher than those for the corpus in patients with reflux esophagitis and duodenal ulcer. On the other hand, a corpus-predominant pattern was observed in patients with early gastric cancer, The scores for atrophy and metaplasia of the antrum were significantly lower in patients with reflux esophagitis than in patients with early gastric cancer, and tended to be lower in patients with reflux esophagitis than in patients with duodenal ulcer.

Antral-predominant gastritis, which is frequently observed in patients with duodenal ulcer, has been shown to be associated with hypergastrinemia and gastric hypersecretion. On the other hand, corpus-predominant gastritis, which is observed mainly

in patients with early gastric cancer, is associated with decreased acid secretion. Our present study demonstrated that the pattern of gastritis in patients with reflux esophagitis was antral-predominant. Therefore, gastric acid secretion in *H. pylori*-positive patients with reflux esophagitis may be increased in comparison with normal *H. pylori*-negative subjects. *H. pylori* eradication therapy is known to induce reflux esophagitis only when the therapy is administered to patients with corpus gastritis. The pattern of gastritis in patients with reflux esophagitis is quite different from that in patients who tend to develop reflux esophagitis after eradication therapy. This different pattern of gastritis may explain why eradication therapy for *H. pylori*-infected patients with reflux esophagitis often improves their esophagitis.

CONCLUSION

The degree of endoscopic gastric mucosal atrophy in *H. pylori*-positive patients with reflux esophagitis is low and the histological pattern of inflammation and activity is antral-predominant. These findings are similar to those in patients with duodenal ulcer. Therefore, gastric acid secretion in *H. pylori*-positive patients with reflux esophagitis may be augmented by *H. pylori* infection, as is the case in patients with duodenal ulcer.