**Helicobacter pylori** in gastric corpus of patients 20 years after partial gastric resection

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**Abstract**

**AIM:** To determine the long-term prevalence of *Helicobacter pylori* (*H. pylori*) gastritis in patients after partial gastric resection due to peptic ulcer, and to compare the severity of *H. pylori*-positive gastritis in the corpus mucosa between partial gastrectomy patients and matched controls.

**METHODS:** Endoscopic biopsies were obtained from 57 patients after partial gastric resection for histological examination using hematoxylin/eosin and Warthin-Starry staining. Gastritis was graded according to the updated Sydney system. Severity of corpus gastritis was compared between *H. pylori*-positive partial gastrectomy patients and *H. pylori*-positive duodenal ulcer patients matched for age and gender.

**RESULTS:** In partial gastrectomy patients, surgery was performed 20 years (median) prior to evaluation. In 25 patients (43.8%) *H. pylori* was detected histologically in the gastric remnant. Gastric atrophy was more common in *H. pylori*-positive patients compared to *H. pylori*-negative patients (*P*<0.05). The severity of corpus gastritis was significantly lower in *H. pylori*-positive partial gastrectomy patients and *H. pylori*-positive duodenal ulcer patients (*P*<0.01). There were no significant differences in the activity of gastritis, atrophy and intestinal metaplasia between the two groups.

**CONCLUSION:** The long-term prevalence of *H. pylori* gastritis in the gastric corpus of patients who underwent partial gastric resection due to peptic ulcer disease is comparable to the general population. The expression of *H. pylori* gastritis in the gastric remnant does not resemble the gastric cancer phenotype.

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scale (grade 0 = negative, grade 1 = mild, grade 2 = moderate, grade 3 = severe) in accordance with the updated Sydney system[15]. The control group consisted of H pylori-positive patients with duodenal ulcer disease who participated in previous clinical trials[7] and who were age- and gender-matched. In these patients, endoscopic biopsies have been obtained from the antrum and the corpus, and were processed as described above. All histological slides were assessed by a single pathologist.

Data analysis was performed using the statistical software package SPSS 10.0 for Windows. The Chi-square test or Fisher exact test was used when appropriate. P<0.05 was considered statistically significant.

RESULTS

A total of 57 patients with partial gastric resection were included into the study (19 males and 38 females, median age 64 years, range 26-92 years). The time period between surgery and histology assessment ranged from 8 to 47 years with a median of 20 years. None of the intraepithelial neoplasia was detected at the gastric anastomosis. In 25 patients (43.9%) H pylori was detected histologically in the gastric remnant.

Table 1 summarizes the histological features in the corpus and cardia of partial gastrectomy patients. For analysis purposes, patients with grade 0 and 1, and patients with grade 2 and 3 were combined, respectively. There was a higher proportion of patients with moderate or severe activity of gastritis in the corpus (P<0.0005) and cardia (P=0.007) among H pylori-positive patients compared to H pylori-negative partial gastrectomy patients. In addition, the proportion of patients with atrophy in the corpus mucosa was significantly higher among H pylori-positive compared to H pylori-negative patients (P = 0.047).

No significant differences were found with regard to grade of gastritis, regenerative epithelium and the presence of intestinal metaplasia between H pylori-positive and H pylori-negative partial gastrectomy patients.

The comparison of corpus gastritis between H pylori-positive partial gastrectomy patients and H pylori-positive duodenal ulcer patients is summarized in Table 2. The proportion of patients with moderate or severe grade of gastritis in the corpus (P = 0.001) and with moderate or severe regenerative epithelium (P = 0.004) was significantly lower in H pylori-positive partial gastrectomy patients than that in H pylori-positive duodenal ulcer patients, respectively. A similar trend was observed for the activity of gastritis in the corpus, however the differences were not statistically significant. The prevalence of intestinal metaplasia in the corpus mucosa was similar in both groups. The prevalence of atrophy in the corpus mucosa was higher in partial gastrectomy patients, however the difference did not reach statistical significance.

Table 2 Severity of corpus gastritis in H pylori-positive partial gastrectomy patients and H pylori-positive duodenal ulcer patients matched by age and gender

| Grade of gastritis | Partial gastrectomy (n = 25) | Duodenal ulcer (n = 25) | P |
|--------------------|-----------------------------|-------------------------|---|
| Grade 0 or 1, n (%)| 24 (96)                     | 13 (52)                 | 0.001 |
| Grade 2 or 3, n (%)| 1 (4)                       | 12 (48)                | 0.007 |
| Activity of gastritis | | | |
| Grade 0 or 1, n (%)| 16 (64)                    | 11 (44)                | 0.256 |
| Grade 2 or 3, n (%)| 9 (36)                    | 14 (56)                | 0.256 |
| Regenerative epithelium | | | |
| Grade 0 or 1, n (%)| 25 (100)                 | 17 (68)                | 0.004 |
| Grade 2 or 3, n (%)| 0 (0)                     | 8 (32)                 | 0.720 |
| Intestinal metaplasia | | | |
| Present, n (%)| 5 (20)                    | 5 (20)                 | 0.128 |
| Atrophy | | | |
| Present, n (%)| 11 (44)                  | 6 (19)                 | 0.047 |

DISCUSSION

In the present study the prevalence of H pylori gastritis in patients underwent partial gastrectomy was 43.9% which is comparable to the average population in Germany and which is within the range of other studies on the H pylori prevalence in partial gastrectomy patients[16-20]. An association between H pylori infection and an increased acute and chronic inflammatory response and a higher prevalence of chronic atrophic gastritis and intestinal metaplasia in the gastric remnant mucosa has been described[16,18] while others were non-confirmatory[20]. Our study suggests that H pylori leads to a higher proportion of atrophy in the corpus of the gastric remnant compared to H pylori-negative partial gastrectomy patients.

Several studies have shown that a severe although non-atrophic gastritis in the corpus mucosa is a particular risk factor for gastric cancer among those individuals infected with H pylori[4-7]. These findings were recently confirmed by a prospective observational study from Japan where gastric cancer developed only in those patients infected with H pylori[21] and where a

Table 1 Histology in the corpus and cardia of partial gastrectomy patients

| Grade of gastritis | Corpus | Cardia |
|-------------------|--------|--------|
| HP + n = 25       | HP - n = 32 | HP + n = 25 | HP - n = 32 | P | P |
| Grade 0 or 1, n (%)| 24 (96) | 32 (100) | 24 (96) | 32 (100) | - | - |
| Grade 2 or 3, n (%)| 1 (4)  | 0       | 1 (4)  | 0       | 0.439 | 0.439 |
| Activity of gastritis | | | |
| Grade 0 or 1, n (%)| 16 (64) | 32 (100) | 16 (64) | 32 (100) | - | - |
| Grade 2 or 3, n (%)| 9 (36)  | 0       | 9 (36)  | 0       | <0.0005 | 0.007 |
| Regenerative epithelium | | | |
| Grade 0 or 1, n (%)| 25 (100) | 32 (100) | 24 (96) | 32 (100) | - | - |
| Grade 2 or 3, n (%)| 0       | 0       | 1 (4)  | 0       | 0.439 | 0.248 |
| Intestinal metaplasia | | | |
| Present, n (%)| 5 (20)  | 7 (22)  | 4 (16) | 4 (12.5) | 0.720 |
| Atrophy | | | |
| Present, n (%)| 11 (44) | 6 (19)  | 0       | 3 (9.5)  | 0.248 |

No partial gastrectomy patient had a grade 2 or 3 regenerative type of epithelium in the corpus. Based upon this result a statistical analysis with regard to regenerative type of epithelium in the corpus was not appropriate.
corpus-dominant gastritis or pangastritis was associated with an 34-fold increased risk for gastric cancer. Based upon the increased risk for gastric cancer in the presence of severe corpus gastritis we hypothesized that patients with partial gastrectomy due to ulcer disease may develop a corpus-dominant phenotype of *H pylori* gastritis, which may contribute as a risk factor for cancer in these patients. Surprisingly, we found a significant lower grade of gastritis in the corpus of *H pylori*-positive partial gastrectomy patients compared to the control group consisting of *H pylori*-positive duodenal ulcer patients. Possible explanation for this finding might include that in some patients the infection may have disappeared spontaneously due to an altered gastric milieu, or that some of the patients may have been operated due to *H pylori*-negative ulcer caused by nonsteroidal anti-inflammatory drugs. Other patients may have received antibiotic therapy for other indications that *H pylori* infection potentially leads to coincident eradication of the bacteria. Nevertheless, we conclude that partial gastrectomy patients should be tested for *H pylori* infection and, if diagnosed positive, eradication therapy should be initiated to reduce the risk of relapse.

Other factors than *H pylori* have been implicated in the pathogenesis of the mucosal alterations in partial gastrectomy patients, including entero gastric reflux, achlorhydra and increased mucosal proliferation, effects of vagotomy and dietary factors. Bile reflux may play a promotional role by increasing permeability to initiating carcinogens. This entero gastric reflux has been reported to be more pronounced after a gastrojejunostomy than to initiating carcinogens. This enterogastric reflux has been reported to be more pronounced after a gastrojejunostomy than after a gastroduodenostomy, which may explain the higher stomach cancer risk after a Billroth II operation.

In conclusion, our study suggests that the *H pylori* prevalence in partial gastrectomy patients (former peptic ulcer patients) is comparable to the general population. *H pylori*-positive partial gastrectomy patients appear not to develop a corpus-dominant gastritis resembling the gastric cancer phenotype of *H pylori* gastritis.

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