Association of *Campylobacter pylori* with Gastritis, Duodenal Ulcer and Gastric Ulcer — A Preliminary Report of Dyspeptic Patients

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Abstract

Since studies conducted in several countries have shown an association of *Campylobacter pylori* with gastritis, gastric ulcer (GU) and duodenal ulcer (DU), we undertook a preliminary study of such association in our patients. Multiple punch biopsies (4-6) were taken from the antral mucosa in 95 patients who on endoscopy were found to have gastritis (52), DU (38) or GU (5). The biopsy specimens were examined for the detection of *C pylori* by microscopy, culture and appropriate biochemical tests.

The presence of *C pylori* was confirmed in 16 patients with gastritis (30%), seven patients with DU (18%) and in none with GU. All seven patients with DU had associated gastritis on histological examination. The overall incidence was 24%, which was significantly lower (p < 0.01) than the expected incidence of 30% in Western series. This may be because our patients had "quiescent chronic gastritis" characterized by mononuclear cell infiltration.

Key words: *Campylobacter pylori*, active chronic gastritis, chronic gastritis, duodenal ulcer, gastric ulcer.

Introduction

Although the presence of spiral organisms in the gastric mucosa was noticed over 45 years ago, Marshall and Warren were the first to culture 'Campylobacter-like' organisms (CLO) from the antral biopsy specimens of patients with gastritis (90%), gastric ulcer (87%) and duodenal ulcer (87%). The organisms were later named *Campylobacter pylori*. Since no work has been reported from India, we decided to study the presence of these organisms in our patients with gastritis, gastric ulcer (GU) and duodenal ulcer (DU).

Material and Methods

Between June 1985 and December 1986, patients with upper abdominal dyspepsia having endoscopic evidence of gastritis, DU or GU were subjected to four to six biopsies of the antral gastric mucosa. Biopsies were transferred to thymol, bacto, or brain-heart infusion broth (BHIB) for further studies. Biopsies in the first nine patients were taken both from the corpus (body) as well as the antrum and cultured separately, to see if the site of the biopsy had any relation to the positivity for *C pylori*. As none of the biopsies from the corpus grew *C pylori* even in those patients in whom the antral biopsies were positive, this practice was discontinued.

Microbiology: Direct smears of the biopsy tissue were stained with Diff-Quik. One part of the biopsy material was cultured directly on Skirrow’s medium to which amphotericin B was added. Biopsies were incubated at 37°C for 42 days under anaerobic conditions (5% H2, 10% CO2, and 85% N2). Sabouraud cultures were made 72 hours later from BHIB to Skirrow’s medium, incubated in the same manner and examined on the 3rd, 5th, 7th and 10th day.

In every positive biopsy, *C pylori* was further identified by colony morphology, motility, dark greenish ground illumination and biochemical tests such as urease test, hippurate hydrolysis, and sensitivity to antimicrobial acid.

Histopathology: Paraffin sections of the biopsy material were stained with hematoxylin and eosin and Warthin-Starry silver stain. Gastritis was graded from 0-3 as follows:

Grade 0: Inflammatory cells rarely seen; grade 1: lymphoid cells present but no other evidence of inflammation; grade 2: increased lymphoid cells with oedema and congestion of mucosa or evidence of cell damage; grade 3: polymorphonuclear (PMN) cells infiltrating mucosal glands or pits, or scattered throughout superficial epithelium, or increased PMNs in lamina propria.

Grades 0 and 1 changes were considered as normal, grade 2 as chronic gastritis (quiescent) and grade 3 as active gastritis.

Data Analysis: The percentage of patients showing the presence of *C pylori* in their biopsies was calculated along with their 95% confidence intervals (CI) and compared with the expected percentage, based on published reports, by calculating the CI for a sample of size N > 95. If the CI in the two groups did not overlap, the difference was reported as statistically significant at the probability value of 0.05 or less.
Results

C. pylori was isolated from 23 of the 95 patients studied (24%; CI 16%–34%). The Table shows the positivity rates in the three diagnostic sub-groups, along with the confidence intervals (CI), and the statistical significance of the differences. In all the three groups, the observed rates were significantly lower than the expected rates (P<0.05). In the nine patients from whom antral and corporeal biopsies were initially obtained, both biopsies were negative for C. pylori in seven patients. In the remaining two patients, the antral biopsies were positive but the corporeal biopsies were negative.

Table: Culture positivity of gastric mucosal biopsies for C. pylori

<table>
<thead>
<tr>
<th>Diagnostic group</th>
<th>Total No</th>
<th>C. pylori</th>
<th>Expected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastritis</td>
<td>32</td>
<td>16</td>
<td>19–45</td>
</tr>
<tr>
<td></td>
<td></td>
<td>90*</td>
<td>79–97</td>
</tr>
<tr>
<td>Duodenal (DU)</td>
<td>38</td>
<td>7</td>
<td>8–34</td>
</tr>
<tr>
<td></td>
<td></td>
<td>87*</td>
<td>72–96</td>
</tr>
<tr>
<td>Gastric ulcer (GU)</td>
<td>5</td>
<td>0</td>
<td>0–52</td>
</tr>
<tr>
<td></td>
<td></td>
<td>87*</td>
<td>72–96</td>
</tr>
</tbody>
</table>

* p<0.05 CI—Confidence intervals.

Ten of 16 patients with gastritis showed a histological picture of grade 2 gastritis and the remaining six showed grade 1 gastritis. Likewise, six of seven patients with DU had associated grade 2 gastritis, whereas one patient had grade 1 gastritis. All biopsies showed a preponderance of plasma cell infiltration. Polymorphonuclear infiltration, which is characteristic of active or grade 3 gastritis, was not seen in any case.

Discussion

Rathbone et al suggested that a higher positivity for C. pylori could be obtained if corpus as well as antral biopsies of the stomach were cultured. However, our data do not corroborate this suggestion.

C. pylori was present in 30% of our cases of gastritis as well as 18% of the cases of DU who had associated gastritis. This incidence is significantly lower than what is reported in other series.8–10 This difference could be explained in part by differences in the type of gastritis seen. Antral gastritis (non-immune or type B) is now recognized to be of two different histological varieties. When associated with C. pylori infection, there is marked polymorphonuclear infiltration and this is termed as 'active gastritis'. In the absence of C. pylori infection, the inflamed mucosa shows patchy atrophy and is almost always infiltrated only with mononuclear cells.2,3,4 This is termed as 'quiescent gastritis'. In our 95 cases, grade 3 or active gastritis was not seen in any case. This probably explains the low prevalence of C. pylori among our patients. Although it has been suggested that antral inflammation extending into the duodenum may be causally related to the development of DU, our preliminary observations indicate that C. pylori may not be an important factor in the aetiology of DU among Indian patients.

References


CORRECTION

In the article ‘Fatal Ischaemic Colitis in Renal Allograft Recipients’ by Rumnam et al, Indian J Gastroenterol 1988; 7: 117–8, Fig 1 should have been Fig 2 and vice versa.

NEWS AND NOTICES

XXIX Annual Conference of Indian Society of Gastroenterology in Association with Society of Gastrointestinal Endoscopy of India and Indian Association of Study of Liver, October 28—November 1, 1988, Ahmedabad.

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