Helicobacter pylori and acid secretion

H. H. GILL, K. SHANKARAN, S. HALANKAR, H. G. DESAI

ABSTRACT

Background. Helicobacter pylori has been implicated in the aetiopathogenesis of peptic ulcer but data on the effect of infection by this organism on gastric acid secretion are equivocal. We, therefore, examined the effect of the presence of Helicobacter pylori in the antrum and body of the stomach on acid secretion.

Methods. We used the augmented histamine test and intragastric titration in three groups of patients. In one group Helicobacter pylori was present in both the antrum and body of the stomach, in the second it was present in the antrum but not the body, and in the third the organism was absent.

Results. There were no significant differences in acid secretion between these three groups.

Conclusion. The presence of Helicobacter pylori in the mucosa of the gastric antrum and body has no effect on acid secretion.


INTRODUCTION

The discovery of Helicobacter pylori (H. pylori) has increased our understanding of the aetiopathogenesis and management of peptic ulcer. Acute H. pylori infection causes gastritis, reduces acid secretion and is blamed for spontaneous hypochlorhydria. Data on the effect of chronic H. pylori infection on acid secretion are conflicting because most studies have reported that the organism is present in the antrum alone. It is important to understand the relationship between acid secretion and the presence of H. pylori in the gastric antrum and body mucosa as the degree and extent of inflammation in the body of the stomach is an important determinant of acid secretion.

We therefore undertook a prospective study to evaluate this relationship.

PATIENTS AND METHODS

Five hundred and twenty-six consecutive patients (372 males, 154 females; age: 11–70 years) with dyspepsia underwent gastric endoscopy and biopsy of the antral and body mucosa to determine whether or not they harboured H. pylori. None of them had been on H2-receptor antagonists, bismuth or antimicrobial drugs in the four weeks prior to endoscopy.

The procedure was performed using an Olympus GIF P3 gastroduodenoscope and two biopsies were taken from the antrum within 5 cm of the pylorus and two from the body of the stomach in the region of the greater curvature. The endoscopic diagnoses were: duodenal ulcer (151), gastric ulcer (14), duodenitis (41), gastritis (114) and normal (206). One biopsy from each site was inoculated immediately for the biopsy urease test and the other sent in formol saline for histological examination. Paraffin-embedded sections of coded biopsy specimens were stained with haematoxylin and eosin and the Warthin–Starry stain.

Gastric secretory studies

Three hundred and forty (65%) of the patients had H. pylori and of these 111 patients consented to undergo gastric secretory studies. Acid secretion was determined by the augmented histamine test in 70 and by intragastric titration in 41. These subjects were divided into three groups depending on whether or not H. pylori was detected—Group I: when H. pylori was present in both the antrum and body; Group II: when H. pylori was present in the antrum but not in the body; Group III: when H. pylori was absent.

Augmented histamine tests were performed in 70 patients in the morning after an overnight fast. They were intubated with a 16F nasogastric tube and the resting secretion was discarded. Basal acid output was calculated for 1–60 minutes, a fixed dose of 2.4 mg of histamine acid phosphate was injected intramuscularly and then the maximal acid output was calculated for 1–60 minutes. Gastric juice was titrated with 0.1N NaOH using phenolphthalein as an indicator.

Intragastric titration was performed in 41 patients by the method described by Fordtran and Walsh. After an overnight fast, the subjects were intubated with a 16F nasogastric tube to which was attached a thin polyvinyl tube (inner diameter: 1 mm). The water recovery test was used to position the tip of the tube in the most dependent part of the stomach. The resting secretion was discarded and the gastric pH was raised to 5.5 by infusing 0.3N sodium bicarbonate through the polyvinyl tube. A standard test meal was given consisting of four slices of bread and 300 ml of milk at a pH of 5.5. During the time the patient was eating and in the subsequent two hours sodium bicarbonate was infused to maintain the gastric pH at 5.5. The net acid secretion in the first and second hours after the standard test meal was calculated from the amount of sodium bicarbonate infused.

Statistical analysis was performed by non-parametric analysis using the Mann–Whitney test.

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TABLE I. Acid secretion by the augmented histamine test and Helicobacter pylori status

<table>
<thead>
<tr>
<th></th>
<th>Group I (n=40)</th>
<th>Group II (n=11)</th>
<th>Group III (n=19)</th>
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</thead>
<tbody>
<tr>
<td>Acid output (mmol/hour)</td>
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<td></td>
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<tr>
<td>Basal</td>
<td>3.0 (1.0–9.8)</td>
<td>2.4 (0.6–5.6)</td>
<td>2.4 (0.3–6.0)</td>
</tr>
<tr>
<td>Maximal</td>
<td>8.1 (2.5–17.5)</td>
<td>9.8 (2.4–17.3)</td>
<td>6.9 (1.5–17.1)</td>
</tr>
</tbody>
</table>

Group I: H. pylori present in antrum only  Group II: H. pylori absent  Group III: H. pylori present in antrum only

TABLE II. Acid secretion by intragastric titration and Helicobacter pylori status

<table>
<thead>
<tr>
<th></th>
<th>Group I (n=13)</th>
<th>Group II (n=7)</th>
<th>Group III (n=21)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acid output (mmol/hour)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First hour</td>
<td>4.8 (0–24.9)</td>
<td>9.0 (0–11.1)</td>
<td>3.0 (0–18.3)</td>
</tr>
<tr>
<td>Second hour</td>
<td>6.7 (0–30.6)</td>
<td>9.3 (1.5–16.8)</td>
<td>6.6 (0–20.1)</td>
</tr>
</tbody>
</table>

Group I: H. pylori present in antrum and body  Group II: H. pylori present in antrum only  Group III: H. pylori absent  All values are median (range)

RESULTS
The results of acid secretion after the augmented histamine test in the various groups are shown in Table I and those after intragastric titration in Table II. There was no statistically significant difference in acid secretion between the three groups.

DISCUSSION
Data on the relationship between H. pylori infection and acid secretion is conflicting due to the different methods used to document H. pylori infection and to estimate acid secretion. Two studies reported no relationship between H. pylori and acid secretion,6,7 another two reported a higher acid output in H. pylori-positive patients with duodenal ulcer,8,9 and still another reported a lower acid secretion in H. pylori-positive subjects.8 Though attempts at a correlation between acid secretion and H. pylori have been inconclusive, post-meal gastrin levels have been reported to be higher in H. pylori-positive subjects.9,10-14 This hypergastrinaemia is due to a selective increase in the gastrin (G) 17 component.15 H. pylori infection predominantly affects the antral mucosa which is the main source of G17 whereas G34 is mainly duodenal in origin. Various explanations for the occurrence of hypergastrinaemia are (i) antral G cells responding inappropriately to a falsely high local pH resulting from the liberation of ammonia by H. pylori urease,9 (ii) stimulation of antral G cells by chronic inflammation,16 and (iii) diminished somatostatin production.17,18 Two reports have documented a fall in serum gastrin levels but no change in acid secretion up to seven months after eradication of H. pylori19,20 infection while two recent studies report a significant fall in serum gastrin and acid output after eradication of H. pylori in patients with duodenal ulcer.12,21

The relationship between the presence of H. pylori in the gastric mucosa and acid secretion is complex as several factors affect gastric acid secretion. These are:

1. The extent and duration of H. pylori infection in the body mucosa. A longer duration of infection might increase the severity of chronic body gastritis and decrease acid secretion.22
2. The degree of damage to the antral mucosa will determine the G and D cell mass16-18 and/or function23 and consequently alter serum gastrin levels.
3. The severity of chronic gastritis in the body will determine acid secretion and affect the serum gastrin levels by the acid–gastrin feedback mechanism.
4. Local inhibition of acid secretion might occur by the action of H. pylori protein independent of the presence of chronic gastritis in the body.24
5. The duration of an acute infection with H. pylori might cause hypochlorhydria which gradually increases to normal values.8,25,26

None of the previous studies have addressed these issues and it is therefore not surprising that conflicting conclusions have been reached.

We did not find any significant difference in acid secretion between the three groups of patients. This suggests that the presence of H. pylori in the antrum or body has no major effect on acid secretion, although this conflicts with reports that H. pylori protein may inhibit acid secretion.24 To counter this we would have had to study the serum gastrin levels in our patients. However, it is possible that patients with H. pylori in the body may have had higher serum gastrin levels which overcame the local acid inhibitory effect of the organism. This might increase the acid secretion and explain the similarity in acid secretion in patients with or without H. pylori in their body mucosa.

Our results indicate that the presence of H. pylori in the antrum or body of the stomach has no direct effect on acid secretion.

REFERENCES
Dystrophin assay in muscular dystrophies: An Indian experience

S. JAIN, C. SARKAR, A. K. DINDA, M. C. MAHESHWARI

ABSTRACT

Background. Dystrophin, a protein situated on the plasma membrane of skeletal muscle, is a key component in maintaining membrane integrity. In muscular dystrophies, dystrophin deficiency results in muscle weakness and degeneration.

Methods. We used dystrophin staining with monoclonal NCL-DYS (rod domain) antidystrphin antibody. We examined patients with various muscular dystrophies in a tertiary care hospital in India for dystrophin deficiency.

Results. The technique was unsuccessful in 4 cases. In the others, dystrophin staining correlated well with clinical diagnosis and detected a manifest female carrier with Duchenne's dystrophy.

Conclusion. Dystrophin staining may be useful in differentiating muscular dystrophies.

INTRODUCTION

Dystrophin is a 400 kD protein involved in membrane integrity. In muscular dystrophies, dystrophin deficiency leads to muscle weakness and degeneration. Dystrophins are present in reduced levels in Duchenne muscular dystrophy and Becker muscular dystrophy, helping in differential diagnosis.

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