Effect of omeprazole on gastric bicarbonate secretion in patients with duodenal ulcer

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Background: Omeprazole, a H+K+-ATPase inhibitor, has been shown to reduce gastric bicarbonate secretion in cats. However, there has been no study on the effect of omeprazole on bicarbonate secretion in patients with duodenal ulcer (DU).

Methods: Fifteen men with duodenal ulcer (mean age 38 years, range 22-57) were included. Baseline gastric acid output, bicarbonate secretion, and parietal and nonparietal secretions were estimated before and after omeprazole therapy (20 mg/day) for four weeks. Results: Omeprazole administration did not significantly alter bicarbonate secretion (3.3 [1.2] vs. 2.4 [0.4] mmol/L, though there was significant reduction in gastric acidity (44.2 [6.6] vs. 20.7 [4.6] mmol/L, p<0.01). Conclusion: Omeprazole reduces acid secretion but does not affect gastric bicarbonate secretion in patients with DU. [Indian J Gastroenterol 1998; 17: 136-137]

Key words: Acid-peptic disease, stomach bicarbonate

The gastric mucosa is known to secrete bicarbonate, which plays an important role in protecting mucosal integrity. It neutralizes 10%-50% of the secreted acid. Ethanol, bile salts, acetazolamide, adrenergic agonists, indomethacin, aspirin and smoking have been shown to inhibit gastric bicarbonate secretion, whereas cholinergic agonists, calcium, prostaglandins E, and E, and various cytoprotective agents including sucralfate stimulate it. There is evidence that bicarbonate levels may be altered by Helicobacter pylori infection or the presence of duodenal ulcer (DU). Experiments have also provided evidence that gastric bicarbonate secretion is influenced by intraluminal acidity in the stomach.

Omeprazole is a specific inhibitor of the H+K+-ATPase enzyme located on the secretory surface of parietal cells and produces prolonged suppression of gastric acid secretion. It has been shown to reduce gastric bicarbonate secretion in cats. However, there is no study in humans on the effect of omeprazole on gastric bicarbonate secretion. We, therefore, studied the effect of omeprazole on gastric bicarbonate secretion in patients with DU.

Methods

Fifteen men with DU (mean age 38 years, range 22-57) were studied. Duodenal ulcer was diagnosed on gastroduodenoscopy. Patients taking drugs known to alter acid and bicarbonate secretion, chronic alcoholics and smokers were excluded. Informed consent was obtained from each patient. The study was approved by the Ethics Committee of our institution.

After overnight fast, a nasogastric tube was placed fluoroscopically in the gastric antrum and gastric juice was collected for an hour after discarding the overnight secretion. The patients were instructed not to swallow saliva during the study. If the gastric juice samples were stained with bile, these were discarded and the study was repeated. During the collection period, at 30 minutes, a 10 mL venous blood sample was collected in a heparinized tube for measurement of plasma osmolality. The patients were then treated with omeprazole 20 mg daily for 4 weeks; at the end of this period the above procedure was repeated.

The osmolality of gastric juice and that of plasma were measured by the freezing point depression method (Advanced Digmatic, Osmometer-model 3D 11, Advanced Instruments, Massachusetts, USA). The H+ concentration was determined by in vitro-titration with 0.1 N NaOH to pH 7.0. Gastric acid output, bicarbonate secretion and parietal and nonparietal volume secretion were calculated from the gastric juice volume and acid content and the osmolalities of plasma and gastric juice using the two-component model of gastric secretion. The two basic equations used were: 1. Measured acid output = acid secreted - acid neutralized 2. Measured osmolar output = osmoles secreted - osmoles neutralized

Under usual circumstances, acid secretion exceeds bicarbonate secretion. Thus, the amount of acid neutralized by bicarbonate equals the amount of bicarbonate secreted. The three accepted assumptions of this method are that the H+ concentration of the parietal component is 160 mmol/L, the osmolality of the parietal component is 1.05 times the plasma osmolality, and the osmolality of the nonparietal component is the same as the osmolality of plasma. The bicarbonate secretion in millimoles was calculated from the formula:

$$\text{HCO}_3^-\text{ secretion} = \frac{V_{ji} (160\Delta\text{osmol} - K\Delta H)}{320-K}$$

where $V_{ji}$ is the volume of gastric juice in liters, $\Delta\text{osmol}$ is the assumed osmolality of the parietal component minus the measured osmolality of gastric juice (in mOsmol/kg), K is the assumed osmolality of the parietal component minus the assumed osmolality of the nonparietal component, and $\Delta H$ is 160 mmol/L minus the measured hydro-
Table: Acid and bicarbonate secretion (mean [SE]) before and after omeprazole therapy

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Before</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma osmolality (mOsmol/Kg)</td>
<td>277 (6)</td>
<td>272 (5)</td>
</tr>
<tr>
<td>Gastric juice volume (mL/h)</td>
<td>58.4 (8.5)</td>
<td>39.0 (2.9)</td>
</tr>
<tr>
<td>Acidity (mmol/L)</td>
<td>44.2 (5.6)</td>
<td>20.7 (4.6)*</td>
</tr>
<tr>
<td>Gastric juice osmolality (mOsmol/Kg)</td>
<td>184 (6.6)</td>
<td>164 (5.7)</td>
</tr>
<tr>
<td>Acid output (mmol/h)</td>
<td>2.4 (0.5)</td>
<td>0.7 (0.1)*</td>
</tr>
<tr>
<td>Bicarbonate secretion (mmol/h)</td>
<td>3.3 (1.1)</td>
<td>2.4 (0.4)</td>
</tr>
<tr>
<td>Parietal volume (mL/h)</td>
<td>15.8 (12.9)</td>
<td>4.5 (3.2)*</td>
</tr>
<tr>
<td>Nonparietal volume (mL/h)</td>
<td>42.5 (7.1)</td>
<td>34.1 (3.1)</td>
</tr>
<tr>
<td>Bicarbonate concentration in nonparietal fluid (mmol/L)</td>
<td>78.0 (9.8)</td>
<td>66.0 (10.1)</td>
</tr>
</tbody>
</table>

*p<0.01

gain ion concentration of gastric juice.

Values are expressed as mean (SE) and compared using Student's t test for paired data.

Results

The gastric acidity, acid output and parietal volume secretion decreased significantly after omeprazole therapy (Table). However, nonparietal volume secretion, bicarbonate secretion and bicarbonate concentration were similar before and after omeprazole therapy.

Discussion

An alkaline microenvironment is very important for rapid restitution of the gastric mucosa. The source of this alkaline environment is bicarbonate secretion from the gastric mucosa. Gastric bicarbonate secretion is influenced by several substances and factors including intraluminal acidity. Omeprazole has been shown to reduce the gastric bicarbonate secretion by about 40% at 100 minutes after administration in cats. However, there was no decrease in bicarbonate secretion in other studies.

We conclude that omeprazole decreases acid secretion but does not influence gastric bicarbonate secretion in patients with DU.

References


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