Alterations in duodenal disaccharidases in chronic smokers

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Objective: To assess the effect of smoking on activity of intestinal disaccharidases. Methods: The study was conducted on patients with non-ulcer dyspepsia who were smokers (n=20) or non-smokers (n=20). Smokers were classified according to smoking index into mild, moderate and heavy smokers. Biopsy specimens were taken from the second part of the duodenum at endoscopy and examined histologically, and for disaccharidase (lactase, sucrase, maltase and trehalase) activities. Results: Mean duration of symptoms was more in smokers than in non-smokers. None of the smokers had endoscopic evidence of duodenal inflammation. Lactase and trehalase levels were significantly decreased in smokers. There was no difference in enzyme levels between mild smokers and non-smokers. Decreased lactase, maltase and trehalase activities were observed in moderate smokers compared to mild smokers. Duration of symptoms had no relation to enzyme activities. Conclusions: Intestinal disaccharidase levels are diminished by smoking. [Indian J Gastroenterol 2001;20:62-63]

Keywords: Non-ulcer dyspepsia, smoking

Tobacco smoke is a complex mixture of some 4000 constituents, a majority of them toxic.1 Risks due to cigarette smoking vary with differences in smoking habits and with presence of other risk factors like age.2 Smoking has been implicated in several disorders of the gastrointestinal tract.3,4 Smokers have a significantly higher risk of developing cancer of the mouth, larynx, pharynx and esophagus.5

A relationship between smoking and intestinal disaccharidase activity has not been reported in literature. Recently, cigarette smoking has been associated with dyspepsia.6

We undertook this study to find out any relationship between smoking and disaccharidase levels.

Methods

Forty men with ulcer-like non-ulcer dyspepsia were enrolled in the study. Non-ulcer dyspepsia was defined as a constellation of symptoms that suggested a diagnosis of peptic ulcer, in the absence of a demonstrable ulcer at endoscopy or barium studies and the absence of any other demonstrable organic disorder or evidence of irritable bowel syndrome.7 Twenty of these patients were smokers and were classified according to smoking index (SI) into mild (SI <100), moderate (SI 100-300) and heavy (SI >300) smokers.8

Patients who had taken anti-ulcer therapy or antibiotics in the previous 2 weeks, chronic alcoholics, those with ascariasis or giardiasis, and those with any malignancy or duodenal ulcer at endoscopy were excluded. The study was approved by the Ethics Committee of the Institution.

All patients were subjected to a detailed history, with emphasis on the type and duration of smoking and history of milk intolerance. After an overnight fast, a forward-viewing flexible endoscopy was done after intravenous diazepam as premedication. Two pairs of biopsy specimens were taken from the second part of the duodenum. One pair was preserved in normal saline for histological examination (hematoxylin-eosin stain) and the other was wrapped in parafilm and stored at -70°C for enzyme estimation. The activities of lactase, sucrase and maltase were measured using the method of Dahlquist.9 Proteins were estimated by the method of Lowry et al.10

Statistical analysis

Comparison between the two groups was done using Student's t test for unpaired data. Coefficient of correlation was drawn between age, duration of symptoms, smoking index and enzyme levels, and between milk intolerance and lactase levels. A p value less than 0.05 was considered significant.

Results

The age of the smokers ranged from 22 to 42 years (mean [SD] 30.9 [2]) and that of non-smokers from 20 to 42 years (30.2 [1.5]). All smokers were either mild (n=6) or moderate (n=14) smokers; there was no heavy smoker. All smokers had pain in the abdomen, 16 had postprandial fullness, five had heartburn, and two had milk intolerance. Corresponding figures among non-smokers were 20, 11, four and one, respectively. Seven smokers and three non-smokers had antral gastritis; the rest had normal endoscopy. Histologically, no smoker and seven non-smokers had duodenal inflammation (p<0.05; χ² test).

Duodenal lactase and trehalase levels were significantly reduced (p<0.05) in smokers as compared to non-smokers. Sucrase and maltase were comparable in the
Table: Mean (SD) levels of duodenal lactase, sucrase, maltase and trehalase (U/g protein) in smokers and non-smokers

<table>
<thead>
<tr>
<th>Groups</th>
<th>Lactase</th>
<th>Sucrase</th>
<th>Maltase</th>
<th>Trehalase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smokers (n=20)</td>
<td>7.4 (1.1)*</td>
<td>55.4 (6.9)</td>
<td>120.2 (12.4)</td>
<td>9.2 (1.0)*</td>
</tr>
<tr>
<td>Mild smokers</td>
<td>9.4 (2.4)*</td>
<td>61.4 (13.3)</td>
<td>161.4 (24.7)*</td>
<td>9.2 (1.3)</td>
</tr>
<tr>
<td>Moderate smokers</td>
<td>6.5 (1.2)*</td>
<td>52.9 (8.3)</td>
<td>102.5 (11.3)*</td>
<td>9.2 (1.4)*</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>12.4 (1.2)</td>
<td>48.9 (4.6)</td>
<td>136.9 (10.5)</td>
<td>14.4 (1.8)</td>
</tr>
</tbody>
</table>

*p<0.05 as compared to *non-smokers and *moderate smokers

two groups (Table). There was no significant difference in enzyme activities between mild smokers and non-smokers. Moderate smokers had significantly lower (p<0.05) lactase, maltase and trehalase activities as compared to non-smokers. Lactase and maltase activities were significantly (p<0.05) lower in moderate smokers as compared to mild smokers, while sucrase and trehalase levels were comparable in the two groups.

No correlation of enzyme activities with age or duration of symptoms was observed in either group.

Discussion

In our study, smokers had significantly lower levels of duodenal lactase and trehalase as compared to those in non-smokers. These results are in agreement with those reported by Vetrivik et al.11 in patients with duodenal ulcer. Lactase and maltase levels were lower in our study in moderate as compared to mild smokers.

Cigarette smoke contains various stimuli in gas and particulate phase,1 some of which might exert a direct effect on intestinal disaccharidases. Smoking has also been shown to decrease mucosal blood flow,12 which in turn may effect expression or turnover of enzymes. A cumulative effect of smoking might be the determining factor of the enzyme levels in smokers.

Some studies have shown a decrease in enzyme activities with increase in age,13 while others have found no such effect.14 The present study found no change in enzyme activities with increase in age or duration of symptoms in either group.

To conclude, our study suggests that smoking decreases the levels of duodenal lactase and trehalase.

References


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