Halitosis and gastroesophageal reflux disease: a possible association

M Moshkowitz, N Horowitz, M Leshno, Z Halpern

OBJECTIVE: Previous reports have suggested that gastrointestinal (GI) diseases may cause halitosis. The aim of this study was to evaluate the relationship between upper GI conditions, especially gastroesophageal reflux disease (GERD), and halitosis.

PATIENTS AND METHODS: One hundred and thirty two consecutive patients complaining of upper GI symptoms were included in the study. All the patients completed a validated questionnaire that was designed to characterize and measure the severity of their symptoms. The questionnaire also contained questions about awareness and severity of oral bad breath. Following the filling of the questionnaire, the patients were physically examined and subjected to an upper GI endoscopy.

RESULTS: The final diagnosis among the 132 patients (M/F = 70/62, mean age 45.2 years, range 20–87 years) was GERD in 72 patients (55%), Functional dyspepsia in 52 (39%), Peptic ulcer in seven patients (5%) and gastric cancer in one patient (1%). Halitosis was significantly associated with the occurrence and severity of heartburn (P = 0.027), regurgitation (P = 0.002) sour taste (P < 0.001), belching (P = 0.001) and burburigmus (P = 0.006). Halitosis was not associated with upper abdominal pain, bloating, early satiety and chest pain. In relation to the final diagnosis, halitosis was significantly associated only with GERD (P = 0.002) but not with functional dyspepsia (P = 0.855) and peptic ulcer disease (0.765). No correlation was found between Helicobacter pylori infection status and halitosis occurrence and severity (analysis of variance F = 0.001, P = 0.977).

CONCLUSIONS: Halitosis is a frequent symptom of GERD and may be considered as an extra-esophageal manifestation of GERD. On the other hand, we did not find an association between functional dyspepsia, peptic ulcer disease and H. pylori infection with halitosis occurrence or severity.
cases of extraoral halitosis resulted from bacterial growth in the posterior-dorsal part of the tongue, it is reasonable to assume that halitosis might be also a result of GERD.

The aim of this study was to evaluate the rate of halitosis in patients with dyspepsia, and to compare patients with GERD with those with other types of dyspepsia.

Subjects and methods

Subject selection
A total of 139 consecutive subjects presented to the Gastroenterological Department in the Tel Aviv Sourasky Medical Center from January 2002 through May 2003 with uninvestigated upper abdominal symptoms were prospectively investigated. Inclusion criteria were (1) presence of dyspeptic symptoms such as upper abdominal pain or discomfort, bloating, nausea, vomiting, heartburn, or early satiety; (2) persistence of symptoms for at least 3 months in the last year, and (3) no previous abdominal surgery.

All participants completed a detailed GI symptom questionnaire, which was followed by an interview and physical examination by a gastroenterologist. All subjects were referred to gastroscopy, and 132 out of 139 patients consented to undergo upper GI endoscopy.

Written informed consent was obtained from all the patients and the study was approved by the local ethics committee.

Symptom questionnaire
To classify dyspepsia and to discriminate GERD from other types of dyspepsia a diagnostic symptom questionnaire was developed. The questionnaire was designed to measure both the presence and severity of reflux and other types of dyspepsia using a 5-point Likert-type scale. The evaluated symptoms included the following items: epigastric pain/discomfort, retrosternal pain, heartburn, regurgitation, nausea, vomiting, belching, bloating, early satiety, burburigmus, hiccups, upper abdominal distension, halitosis, sour taste, and stress. The questionnaire also measured the influence of different factors on the patient’s symptoms; these factors included: eating, drinking milk, hunger, avoiding certain foods, use of antacids, bending or lying down, and heavy meals. Presence of alarm symptoms (such as GI bleeding, weight loss, vomiting, dysphagia, anemia, GI polyps or tumors and past abdominal surgery) was also addressed.

Subject evaluation
Upper GI endoscopy was performed using Pentax video endoscope. Endoscopists were aware that all patients were being investigated for this study but were blinded to the patients’ questionnaire answers. The macroscopic appearance of the esophageal mucosa was recorded according to Los Angeles classification (Lundell et al., 1999). Subjects with any grade of esophagitis as seen in endoscopy were diagnosed as having ‘reflux disease’ (GERD) vs ‘non-reflux disease.’ Finding of ‘esophagitis’ was made by the physician who performed the gastros-
Table 2 Severity of symptoms in patient with or without halitosis

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Patients with moderate/severe halitosis</th>
<th>Patients with none/mild halitosis</th>
<th>Independent-sample t-test</th>
<th>Sig. (two-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heartburn</td>
<td>3.80 (±1.47) 2.79 (±1.53)</td>
<td>2.41 (±1.42)</td>
<td>-2.9**</td>
<td>0.004</td>
</tr>
<tr>
<td>Regurgitation</td>
<td>3.53 (±1.18) 2.41 (±1.42)</td>
<td>2.41 (±1.42)</td>
<td>-2.9**</td>
<td>0.004</td>
</tr>
<tr>
<td>Sour taste</td>
<td>3.66 (±1.44) 1.98 (±1.25)</td>
<td>1.98 (±1.25)</td>
<td>-4.8**</td>
<td>0.000</td>
</tr>
<tr>
<td>Dysphagia</td>
<td>2.40 (±1.29) 1.79 (±1.20)</td>
<td>1.79 (±1.20)</td>
<td>-1.79*</td>
<td>0.075</td>
</tr>
<tr>
<td>Chest pain</td>
<td>2.42 (±1.34) 1.92 (±1.23)</td>
<td>1.92 (±1.23)</td>
<td>-1.41</td>
<td>0.159</td>
</tr>
<tr>
<td>Belching</td>
<td>2.66 (±1.58) 2.25 (±1.42)</td>
<td>2.25 (±1.42)</td>
<td>-1.04</td>
<td>0.299</td>
</tr>
<tr>
<td>Slow digestion</td>
<td>3.20 (±1.42) 2.61 (±1.44)</td>
<td>2.61 (±1.44)</td>
<td>-1.46</td>
<td>0.146</td>
</tr>
<tr>
<td>Burburigmus</td>
<td>3.06 (±1.22) 2.33 (±1.32)</td>
<td>2.33 (±1.32)</td>
<td>-2.03**</td>
<td>0.044</td>
</tr>
</tbody>
</table>

*P ≤ 0.075, **P < 0.05.

between the severity of halitosis and dyspeptic symptoms. Patients with GERD-typical symptoms have also more severe halitosis than patients with non-GERD.

Table 3 represents the distribution of final diagnoses among patients with and without halitosis. Only diagnosis of GERD was significantly different between patients with and without halitosis.

Figure 1 shows the occurrence and severity of halitosis in various diagnostic groups. Halitosis was significantly more prevalent and more severe among patients with GERD than in patients with other causes of dyspepsia. No correlation was found between the severity of halitosis and dyspeptic symptoms. Patients with GERD-typical symptoms have also more severe halitosis than patients with non-GERD.

![Figure 1](image-url)

Figure 1 Severity of halitosis in reflux vs non-reflux patients

Table 3 Severity of halitosis (1–5) among patients with reflux, peptic ulcer, and functional (non-ulcer) dyspepsia

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>n</th>
<th>Mean rank</th>
</tr>
</thead>
<tbody>
<tr>
<td>GERD</td>
<td>72</td>
<td>74.52*</td>
</tr>
<tr>
<td>Non-ulcer dyspepsia</td>
<td>52</td>
<td>55.01</td>
</tr>
<tr>
<td>Peptic ulcer disease</td>
<td>7</td>
<td>58.1</td>
</tr>
</tbody>
</table>

GERD, gastroesophageal reflux disease.

*Kruskal–Wallis test, P < 0.01

Discussion

The results of the present study indicate a strong association between the occurrence and severity of halitosis and GERD, and the absence of such association with other causes of dyspepsia such as peptic ulcer and H. pylori infection. This is the first study to establish a possible association between GERD and halitosis.

There is a common popular belief that once a periodontal disorder has been ruled out, halitosis might be the result of GI disorders and some patients are referred often for GI consultation. However, a direct cause-and-effect relationship has not been established.

It is well recognized that more than 90% of cases of halitosis originate from the oral cavity and it is attributed to VSC produced by oro-pharyngeal bacteria (Tonzetich, 1977). The implicated bacteria are located in stagnant areas in the oral cavity, such as the dorsal surface of the tongue, periodontal pockets, and interproximal areas. Several factors that support the growth of these bacteria and predispose a person to halitosis are well known and include: accumulation of food within pockets around the teeth, along the bumps at the back of the tongue, or in small pockets in the tonsils; sloughed cells from the mouth; diminished saliva flow and mucus in the throat or sinuses. (Kleinberg and Westbay, 1992).

The findings of the present study suggest that GERD might be another possible predisposing factor that serves as a breeding ground for bacteria in the oropharynx.

Extraesophageal manifestations of GERD are frequent, and consist broadly of pulmonary disease, noncardiac chest pain, and ear-nose-throat (ENT) disease (Koufman, 1991). It has been shown that patients often do not have classic symptoms of heartburn or regurgitation (Fennerty, 1999). In a recent study that investigated the prevalence and severity of esophagitis in 405 patients with suspected GERD-related chronic ENT symptoms, almost 40% of patients had halitosis and its occurrence was similar to that of cough, throat ache, and globus sensation (Poelmans et al, 2004). Interestingly, in a study that examined oral signs and symptoms in patients with inflammatory bowel diseases (IBD), Katz et al (2003) found high rates of GERD and halitosis in this patient group. They did not explain these findings; however, consistent with the results of our study, the high halitosis rate they found is probably related to GERD rather than IBD which is located in the small or large bowels.

There are several possible mechanisms by which GERD might cause halitosis. The first probable mechanism is of direct damage to the oropharyngeal mucosa by the gastroesophageal refluxate, which spills across the upper esophageal sphincter and into the oral cavity. This mechanism is similar to the process suspected to operate in the ear, nose, and throat and asthma presentations of GERD (Irwin et al, 1993).
Oral Diseases

The first mechanism is a direct injury of the oropharyngeal mucosa by the gastric refluxate that may cause inflammation. Mamede et al (2004) showed that the prevalence of severe hypertrophy of lymphoid follicles at the base of the tongue markedly increased from 1.6% in healthy population to 7.5% among patients presenting with GERD symptoms such as heartburn, regurgitation, retrosternal burning feeling, and dysphagia. Another possible indirect mechanism is that halitosis is a result of dental erosions, which is a common complication of GERD (Schroeder et al, 1995; Gregory-Head et al, 2000). Although professional dental examination was not a part of our study, all patients reported a normal and routine dental hygiene, without difference between the patient groups.

In contrast to several studies which linked halitosis with H. pylori infection (Tiomny et al, 1992; Ierardi et al, 1998; Serin et al, 2003; Adler et al, 2005), our study does not confirm a relation between gastric H. pylori infection and halitosis. Serin et al (2003) found a higher rate of infection among H. pylori-positive patients with non-ulcer dyspepsia (NUD) than in those who were H. pylori negative. They also found that H. pylori eradication led to dramatic improvement in the halitosis complaints. Helicobacter pylori infection in NUD patients is usually limited to the antral mucosa causing mild gastritis and the mechanism by which such condition might cause halitosis is not clear. Even the fact that halitosis disappeared following triple antibiotic therapy might be a result of a temporary eradication of other bacterial species in the oropharynx rather than H. pylori eradication. In another study using PCR techniques, H. pylori was detected in the saliva, supragingival, and subgingival plaques of periodontitis patients (Gebara et al, 2004). Hoshi et al (2002) found that levels of hydrogen sulfide and dimethyl sulfide in mouth air were significantly higher in H. pylori-positive patients than in H. pylori-negative patients; however, odor strength in exhaled breath did not differ significantly between the two groups. Several authors described an association between glossitis, H. pylori, and halitosis. Adler et al (2005) investigated 46 patients with lingual dorsum hyperplasia and halitosis, and found 40 out of 46 (87%) to be H. pylori positive in the dorsum of the tongue and 93% of them with H. pylori in the stomach also. Although the study population included patients with dyspepsia and burning sensation, they did not refer at all to the possibility of GERD as the cause of glossitis and halitosis, and H. pylori colonization of the dorsal of the tongue as a marker of reflux. Gall-Troselj et al (2001) described an association between H. pylori colonization in the oral mucosa and atrophic glossitis and burning mouth syndrome. They concluded that mucosal changes in these conditions might make the oral environment more acceptable for H. pylori colonization compared with normal mucosa. In a recent study by Lee et al (2006), H. pylori was shown to produce hydrogen sulfide and methyl mercaptan. This suggests that this microorganism can contribute to the development of halitosis.

A possible explanation for the contradictory findings in relation to H. pylori infection and halitosis between the present study and the above-mentioned studies might be the fact that we investigated only gastric H. pylori infection, and not oro-pharyngeal H. pylori infection.

An important limitation of our study should be noted. The evaluation of halitosis was made subjectively using a questionnaire and not with more objective methods such as organoleptic method or volatile sulfide monitoring by a halimeter. Obviously, these objective methods could add to the power of the study; however, a statistically significant correlation was found recently between clinical organoleptic diagnosis of halitosis, SC level by halimeter and subjective patients’ opinion evaluated with a questionnaire (Iwanicka-Grzegorek et al, 2005).

In conclusion, our results indicate that halitosis might be a result of GERD and that it should be considered as an extraesophageal manifestation of this disease. Thus, it is important for the dentist as well as for the primary care physician to be familiar with and inquire about typical and atypical reflux symptoms. Early diagnosis and suppression of refluxed acid through lifestyle changes and medications could potentially prevent further unnecessary investigations of this disease, and a communication between gastroenterologists and dentists is imperative for the success of the overall treatment of the patients.

References


