Original Article

Relationship of Halitosis with Gastric Helicobacter Pylori Infection

Farnaz Hajifattahi[1]✉, Maryam Hesari[2], Homayoun Zojaji[3], Fatemeh Sarlati[4]

[1]Assistant Professor, Department of Oral Medicine, Dental Branch Islamic Azad University, Tehran, Iran
[2]Dentist, General Practitioner, Tehran, Iran
[3]Associate Professor, Department of Gastroenterology, Shahid Beheshti University of Medical Sciences, Tehran, Iran
[4]Associate Professor, Department of Periodontics, Dental Branch Islamic Azad University, Tehran, Iran

Abstract

Objectives: Gastric infection with Helicobacter pylori (H. pylori) may be one of the main causes of halitosis. This study was performed to evaluate the relationship of H. pylori infection with halitosis.

Materials and Methods: This case control study was performed on 44 dyspeptic patients with a mean age of 34.29±13.71 years (range 17 to 76 years). The case group included 22 patients with halitosis and no signs of diabetes mellitus, renal or liver failure, upper respiratory tract infection, malignancies, deep carious teeth, severe periodontitis, coated tongue, dry mouth or poor oral hygiene. Control group included 22 patients without halitosis and the same age, sex and systemic and oral conditions as the case group. Halitosis was evaluated using organoleptic test (OLT) and H. pylori infection was evaluated by Rapid Urease Test (RUT) during endoscopy. The data were statistically analyzed using chi square, Mann Whitney and t-tests.

Results: Helicobacter pylori infection was detected in 20 (91%) out of 22 halitosis patients and seven control subjects (32%) (P<0.001).

Conclusion: Helicobacter pylori gastric infection can be a cause of bad breath. Dentists should pay more attention to this infection and refer these patients to internists to prevent further gastrointestinal (GI) complications and probable malignancies.

Key words: Halitosis; Helicobacter Pylori; Gastrointestinal Diseases; Periodontal Diseases; Dental Plaque Index

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✉ Corresponding author:
F. Hajifattahi, Department of Oral Medicine, Dental Branch Islamic Azad University, Tehran, Iran

F_hajifattahi@dentaliau.ac.ir

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INTRODUCTION

Halitosis is a common term used to describe an unpleasant or offensive odor emanating from the oral cavity, which may cause social discomfort. Halitosis may have intra- or extra-oral sources. In some cases, bad odor originates from the mouth due to inadequate plaque control, periodontal disease, excessive bacterial growth in dental caries or faulty restorations, dry mouth and hairy tongue. Halitosis may be caused by systemic disorders such as upper respiratory tract infections, liver or renal insufficiency and diabetes [1]. Recent studies suggest that H. pylori infection in patients with gastrointestinal (GI) diseases may be the principal etiology of halitosis [2-5].

Helicobacter pylori is a spiral, microaerophilic, Gram-negative bacterium, identified as the most frequent cause of gastritis; it is also believed to be responsible for peptic ulcer and gastric cancer [6]. A possible link between H. pylori and halitosis was first suggested in 1985 [2]. In a clinical study, it was established that if the main oral contributors such as dental caries and periodontal disease are ruled out in patients with halitosis, eradication of H. pylori would effectively eliminate the breath odor. Since H. pylori infection has no specific symptom, the bacterium may colonize the GI tract and cause more serious problems, and in this situation, halitosis may be the only symptom [3]. But the existing studies show contradictory results; some state that under specific circumstances, halitosis may be considered an indication for H. pylori eradication therapy [2,3]; whereas, others suggest that halitosis is related to GI diseases other than H. pylori infection such as gastro esophageal reflux (GERD)[7]. A recent study on patients with gastric pathology explained that halitosis nearly always originates from the oral cavity and not the stomach [8]. Furthermore, several studies, which linked halitosis with H. pylori infection reported elimination of halitosis following antibiotic therapy. The mechanism is questionable because even the fact that halitosis disappeared following antibiotic therapy may be the result of elimination of other bacterial species in the oral cavity [2,4]. Moreover, we know that almost half the patients with gastric H. pylori infection and chronic periodontitis harbored the bacterium in the oral cavity [9]. With regard to the existing contradictions, this case control study was performed to evaluate the relationship of gastric H. pylori infection with halitosis.

MATERIALS AND METHODS

The study design was case control. The ethical committee for clinical studies approved the study protocol. All patients gave a written informed consent. Among the outpatients referred to the GI Department of Taleghani Hospital, 44 patients were selected and enrolled in the study. They were divided into two groups in terms of presence or absence of halitosis with a mean age of 34.29±13.71 years (range 17-79 years). The case group included 22 patients who had halitosis and exhibited none of the known oral causes of malodor such as carious teeth, severe periodontitis, coated tongue, dry mouth or inadequate plaque control; besides, they had no systemic factor causing halitosis such as diabetes mellitus, upper respiratory tract infection, chronic renal or liver failure or malignancies. Smokers were excluded as well as those who were taking medications other than drugs prescribed for gastric complaints. The remaining 22 patients were assigned as controls due to absence of halitosis. The control subjects matched the case group in terms of age, sex, periodontal status, plaque control and drugs taken for gastric complaints. The exclusion criteria were similar to those for the case group. The aforementioned systemic diseases were diagnosed based on the patients' medical records and symptoms by an internist and subjects with these conditions were excluded. Each subject's oral examination was done by a dentist trained and calibrated by two experienced clinicians (periodontist and oral medicine specialist). All oral examinations were performed in the morning after at least two hours of refraining from drinking, eating or oral hygiene practice.

They were instructed not to wear perfumes and subjects who reported having spicy or odorous foods in the past 24 hours were excluded. Coated tongue was defined as the presence of tongue coating covering more than one-tenth of tongue dorsum. The following grading system was used [10]:

Grade 0 = No tongue coating

Grade 1 = Light coating of the tongue present (about 10% of the surface).

Grade 2 = Moderate coating of the tongue

present (10–50% of the surface).

Periodontal status was determined based on Periodontal Disease Index (PDI) [11] measuring clinical attachment loss at four points in the buccal and one point in the lingual aspect around Ramfjord teeth and the assessment of the inflammation of the gingiva was done and scored as follows:

Score 0: No inflammation in the gingiva

Score 1: Mild to moderate gingivitis in some areas of the gingival margin

Score 2: Mild to moderate gingivitis of the entire gingival margin surrounding the tooth

Score 3: Severe gingivitis and visible erythema, hemorrhage and ulceration

Score 4: Up to 3 mm of attachment loss

Score 5: 3-6 mm of attachment loss

Score 6: More than 6 mm of attachment loss

Plaque control was evaluated using Silness and Loe plaque index (PI) [12]. Dry mouth was diagnosed if the patient complained of it or by moving a dry swab on the buccal mucosa. Dry mucosa and swab will move together if xerostomia is present. Subjects with active dental caries, severe periodontitis (PDI grade 6), poor plaque control (PI grade 3) and dry mouth were excluded. Smokers were excluded due to the interference in halitosis diagnosis.

We used the OLT for oral malodor assessment. Halitosis was examined in the morning by the aforementioned examiner in a standard position by direct sniffing of expelled mouth air at a distance of approximately 10 cm and giving a score of 0-5 [13]:

0: Absence of odor

1: Questionable odor

2: Slight malodor

3: Moderate malodor

4: Strong malodor

5: Severe malodor

The scores of 0 and 1 were considered as negative and the subjects who were scored 2 or higher were diagnosed as positive or halitosis patients.

Gastric H. pylori infection was identified by RUT during upper GI endoscopy by a gastroenterologist who was an expert in endoscopic examinations.

The statistical comparisons between the two groups were performed by the Student's t-test for age, chi-square test for sex and medications, and Mann-Whitney test for periodontal disease and PI. The statistical analysis for H. pylori infection in case and control groups was done by chi-square test. The significance level was set at $P<0.05$.

Table 1. Age, sex, medication, Periodontal Disease Index and Plaque Index in patients with and without halitosis (Student's t-test, chi-square test and Mann –Whitney test)

	Variables	Halitosis	Control	P value
Age ± SD		32.4 ± 10.5	35.7 ± 16.7	P=0.443
Sex	Male	10 (45%)	10 (45%)	P=1
	Female	12 (55%)	12 (55%)	
Medication	Yes	15 (68%)	17 (76%)	P=0.587
	No	7 (32%)	5 (24%)	
PDI[a]	<1	6 (27%)	6 (27%)	P=0.874
	1-2	9 (41%)	10 (45%)	
	2-3	3 (14%)	3 (14%)	
	3-4	3 (14%)	3 (14%)	
	≥4	1 (4%)	0 (0%)	
PI[b]	<1	1 (4%)	1 (4%)	P=0.915
	1-2	13 (59%)	12 (55%)	
	≥2	8 (37%)	9 (41%)	

[a]: Periodontal Disease Index (Ramfjord)

[b]: Silness and Loe Plaque Index.

RESULTS

A total of 44 patients who presented to the endoscopy ward due to dyspeptic disorders were examined in two groups of case and control. The demographic and clinical parameters of both groups are shown in Table 1. There was no significant difference between the two groups regarding age, sex, periodontal status, PI, and number of people using medications for gastric complaints. Table 2 represents the distribution of patients with and without halitosis with regards to the H. pylori diagnosis. Chi-square test revealed a significant difference in this regard (P<0.001) (odds ratio=21.43, with 95% confidence interval: 3.9-118.3).

DISCUSSION

This study suggested a strong relationship between gastric H. pylori infection and halitosis. Therefore, this infection may cause bad breath irrespective of any other condition. Halitosis is a common problem and various factors can cause it. Among all, the role of GI diseases and H. pylori as the most common microorganism in the gastric mucosa has been debated for several years [2-5]. In 1998, Ierardi and colleagues reported a correlation between elimination of H. pylori infection and disappearance of halitosis when they examined halitosis as an objective finding based on the assessment of sulfide compound levels in the breath [4]. Findings of Serin et al, with the aim of investigating the frequency of halitosis before and after eradication therapy in patients with H. pylori infection suggested that although the frequency of halitosis was similar to the frequency of other symptoms of H. pylori infection, there was a higher resolution rate for malodor than for other symptoms after treatment [3].

Therefore, they suggested halitosis to be an indication for H. pylori eradication therapy. Our study had the same inclusion criteria as the study by Serin et al, [3] and confirmed their results regarding such correlation. Katsinelos and colleagues reported similar results in a survey with the aim of investigating the incidence and long-term (six to 108 months) outcome of halitosis before and after eradication therapy in patients with functional dyspepsia and H. pylori infection. They suggested that eradication of H. pylori results in sustained resolution of halitosis during long-term follow-up in the majority of cases [2].

Ierardi et al. reported similar results to ours; they showed no change in breath sulfide levels after using an antiseptic mouth rinse in H. pylori positive patients; while the infection was not eradicated in the stomach [4]. In contrast to several studies linking halitosis with H. pylori infection, there are some articles that do not confirm such correlation. Moshkowits et al. found that halitosis is a frequent symptom of GERD and it may be an extra esophageal manifestation of it [7]. They reported this in a study aiming to evaluate the relationship between upper GI conditions especially GERD and halitosis. They did not find any correlation between H. pylori infection and halitosis occurrence or severity.

Table 2. Prevalence of gastric H. pylori infection in patients with and without halitosis (Chi square test)

Group	H. Pylori	
	Negative N (%)	Positive N (%)
Halitosis	2 (9)	20 (91)
Control	15 (68)	7 (32)
P value	P<0.001	

Despite the sufficient number of patients, their study had some limitations: evaluation of halitosis was done subjectively using a questionnaire, and not with an objective method such as organoleptic method or volatile sulfur monitoring. Such approach is not valid, especially for GERD patients who experience abnormal taste and burning sensation frequently due to malodor. In addition, no dental examination was done and all patients self-reported having normal dental hygiene, which is not reliable.

Helicobacter pylori DNA was detected in the saliva of patients complaining of halitosis, using polymerase chain reaction technique in a study in Japan by Suzuki et al, with the aim of investigating the relationship between oral H. pylori and halitosis. It was shown that progression of periodontal pockets and inflammation may favor colonization by periodontopathic bacteria, and H. pylori infection may be indirectly associated with halitosis following periodontitis [14]. In the mentioned study, each subject's periodontal status was determined based on the presence of teeth with periodontal pocket depth of greater than six millimeters and tooth mobility. Whereas, in our study, these symptoms were identified as grade 6 of PDI and led to patient's exclusion, while distribution of the lower grades of PDI in case and control groups was similar. Thus, there was no link between periodontal disorders and halitosis in our case group. Moreover, there may be similarities in the mechanisms of malodor production in oral and gastric infections. In our study, the severity of malodor in each individual was determined using an organoleptic scale, which has been confirmed as a sensitive test among other methods and is regarded as the gold standard for assessing halitosis since it reflects human perception, and only a human can judge the acceptability of the mixed odors emanating from the mouth [15-17].

Since our patients exhibited none of the known systemic causes of halitosis and the oral health was assessed accurately based on the absence of periodontal disease, dental caries, dry mouth, and coated tongue, and even smokers were excluded due to the possibility of misdiagnosis of halitosis, the bad odor might be solely due to H. pylori infection in our patients and there might be a direct cause and effect relationship. A limitation of our study was that only one experienced clinician performed the OLTs. Ideally, one would use two or more odor judges, but like most similar studies, we used only one trained and calibrated examiner to avoid interobserver variations.

Training and calibration of the examiner minimized intraobserver variability. Although our findings revealed a strong positive link between H. pylori infection and halitosis, the exact pathophysiological mechanism behind this condition is controversial. For the first time, a recent survey from Korea discovered that volatile sulfur compounds (VSCs) were closely associated with changes of upper GI tract and halitosis might be the result of severely inflamed or eroded mucosa. They suggested that the mechanism by which H. pylori causes halitosis is elevated H2S levels, and bad odor might be due to the putrefactive action of H. pylori. However, there might be another possibility that H. pylori infection leads to higher chance of erosive changes in accordance with the enzyme induction prerequisite for VSCs generation [18].

CONCLUSION

In conclusion, our results strongly suggest that halitosis might be a result of H. pylori infection and it can be considered as an indicator of this infection.

Therefore, it is important for dentists as well as internists to pay more attention to consistent halitosis, in order to find H. pylori infection in the primary stages. Early diagnosis and treatment of this infection not only eliminates bad odor, but can also potentially prevent further GI disorders and malignancies.

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