J Tokyo Wom Med Univ
71 (11・12) 787～797 (2001)

Association Between Halitosis and Gastric *Helicobacter pylori* Infection

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(Received Aug. 15, 2001)

The aim of the present study was to assess whether oral odour and tongue coating odour in the gastric *Helicobacter pylori* (*H. pylori*) positive patients are more severe organoleptically than those of the negative patients. Moreover, this is the first study of halitosis on *H. pylori* in which qualitative-quantitative measurements were made by gas chromatography (GC). In addition, the backgrounds (age, gender, oral periodontal parameters and tongue coating) were compared between the 31 *H. pylori* positive and 49 negative patients. The *H. pylori* positive group was significantly older than the *H. pylori* negative group. There were no significant differences in gender, periodontal parameters or visual tongue coating assessment between the two groups. The oral odour assessed with patient holding breath by organoleptic measurement, the gastric *H. pylori* positive patients was more severe than that of the negative patients (Odds ratio: 2.76, 95% CI: 1.78 to 3.74). There were no significant differences in oral odour assessed with patient exhaling breath and tongue coating odour. The levels of H_2S and $(CH_3)_2S$ but not CH_3SH, in oral air measured by GC were significantly higher in the *H. pylori* positive patients than in the negative patients ($p<0.05$). Further research confirming the relation between gastric *H. pylori* infection and halitosis is needed.

Introduction

Halitosis (oral malodour, bad breath) is mainly caused by periodontal disease and tongue coating. Volatile sulphur compounds (VSCs: hydrogen sulphide, methyl mercaptan and dimethyl sulphide) are claimed to be the principal gases responsible for halitosis[1)~3)]. In old medical accounts, gastrointestinal diseases are mentioned as causes of halitosis[4)5)]. By contrast, there are also warnings against being misled, since no gases originating in the gastrointestinal tract are released through the mouth[6)]. Despite that, an association between gastric *Helicobacter pylori* (*H. pylori*) infection and halitosis has been hypothesized[7)8)]. Since there are evidence to suggest that infection with *H. pylori* is the risk factor for duodenal and gastric ulceration[9)], the hypothesis indicates that gastrointestinal diseases relate to halitosis.

The aim of the present study was to assess whether oral odour and tongue coating odour of gastric *H. pylori* positive patients is more severe than that of patients who are negative. This study is also significant among studies[7)10)11)] conducted on halitosis in persons infected with gastric *H. py-*

lori previously reported because it is the first time that qualitative-quantitative measurements made by gas chromatography have ever been reported.

In addition, parameters such as the background of patients' oral status relating to oral odour were compared between the two groups.

Materials and Methods

Subjects

Between January 1999 and December 2000 a total of 8,475 patients were examined in the outpatient clinic of the Department of Oral and Maxillofacial Surgery, Tokyo Women's Medical University Hospital. A simple history was taken by interview in the 183 patients who presented with halitosis as their chief complaint at the time of the initial examination, and they were placed under the care of a dentist who specializes in halitosis from the second visit. In the Breath Odour Clinic of the Department of Oral and Maxillofacial Surgery, the patients were randomly assigned to 4 dentists, with each patient always being treated by the same dentist. The patients were allotted to 4 dentists in proportion to the amount of time they were on duty in the outpatient clinic. The 102 patients for whom one of these dentists was responsible were included in this study. Ten of the total of 102 patients were immediately referred to the Department of Psychiatry for the treatment of halitophobia[12] and they were excluded from the study. Informed consent was obtained from each patient prior to the examination. None of the 92 remaining patients had liver disease, kidney disease, or diabetes mellitus or had taken antibiotics within the preceding 3-week period.

Almost all data collection was performed during ordinary treatment in the Outpatient Clinic of Breath Odour, and it was included within the scope of care provided by the individual patient's health insurance. However, it was explained that because they were outside the range of health insurance coverage, the patients would be extra-charged for the oral odour measurements by gas chromatography and a urea breath test for gastric *H. pylori* infection. It was explained to the 92 remaining patients that, "It has never been determined whether the presence or absence of *H. pylori* infection is associated with halitosis, and while some investigators think that halitosis is cured by bacterial eradication therapy, there is still no consensus". When informed of this, 4 patients refused the urea breath test for gastric *H. pylori* infection and were excluded as subjects. Starting on the day before the test, foods with strong odours, such as garlic, as well as alcoholic beverages, smoking, and the use of cosmetics and perfumes with strong odours was prohibited, but the subjects were instructed to carry out oral cleaning at bedtime as usual. On the day of the test, consumption of food and drink, teeth brushing, tongue cleaning, and oral rinse were prohibited, and the subjects were strictly cautioned not to place anything in their mouth after arising.

However, without prior notice 4 subjects cancelled their appointment on the day of the test, 6 subjects smoked on the day of the test, and 2 subjects had eaten, and they were all excluded as subjects.

As a result, 80 persons participated in this prospective study. The 80 subjects comprised 78 Japanese, 1 Chinese, and 1 Korean.

Methods

To minimize the effects of the other tests, all measurements were performed in the order as shown in Table 1.

Examination for halitosis

The two methods of analyzing oral odour-human organoleptic measurement and gas chromatography (GC) were performed.

1) Human organoleptic measurement

With the nose of an examiner approximately 5

cm from the mouth of the patient opened mouth and simply sniff the patient's breath. Odour was assessed with the patient holding his/her breath (A, Table 1) and it was assessed again with the patient exhaling alveolar air (B, Table 1). Organoleptic scoring scale was rated on a scale of 0 to 5 (0, absence of odour; 1, questionable odour; 2, slight malodour; 3, moderate malodour; 4, strong malodour; 5, severe malodour)[12].

2) GC measurement of VSCs[13] (C, Table 1)

A gas chromatograph equipped with a flame photometric detector (GC-17A, Shimadzu, Japan) was used to make the gas-chromatography measurements, and three VSCs (hydrogen sulphide: H_2S, methyl mercaptan: CH_3SH and dimethyl sulphide : $(CH_3)_2S$) were quantitatively determined by a computed soft ware (805 Data Station Version 1.01 d, MILLIPORE, Japan). The patients were instructed to breathe quietly through their nose, and they were asked to hold the tip of a teflon tube between their lips, close them tightly, and advance the tube approximately 3 cm inside. Three minutes after inserting the tube, an auto injection system was used to draw 10 ml of the gas in the oral cavity into a column (Teflon column 4.0 m × φ3.0 mm packed with 1, 2, 3-tris (2-cyanoethoxy) propane (TCEP) 25% Shimalite 80/100 mesh, AW-DMCS-ST). Standard gas for the quantitative determinations was generated with a Permeater PD-1B gas generator (Gastec, Japan).

Examination of tongue coating

1) Visual examination of tongue coating (D, Table 1)

Visual examination of tongue coating was performed by the method as described below. The "extent covered by tongue coating" was defined as the extent of the dorsal surface of the tongue anterior to the vallate papillae. The total area and thickness of the tongue coating were determined. The area was recorded on a scale 0 to 3 (0, no tongue coating; 1, tongue coating covering less than 1/3 of tongue dorsum; 2, tongue coating covering 1/3～2/3 of tongue dorsum; 3, tongue coating covering more than 2/3 of tongue dorsum). The thickness was recorded on a scale of 0 to 2 (0, no tongue coating; 1, thin tongue coating-tongue papillae visible; 2, thick tongue coating-tongue papillae invisible). The tongue coating was scored by multiplying the area score by the thickness score[14].

Table 1 Order of measurements[a]

A	Human organoleptic measurement with patient holding breath (Score 0, 1, 2, 3, 4 or 5)
B	Human organoleptic measurement with patient exhaling alveolar air (Score 0, 1, 2, 3, 4 or 5)
C	Gas-chromatography (GC) measurement of volatile sulphur compounds (VSCs)
	Hydrogen sulphide : H_2S
	Methyl mercaptan : CH_3SH
	Dimethyl sulphide : $(CH_3)_2S$
D	Visual examination of tongue coating (Score 0, 1, 2, 3, 4 or 6)
E	Tongue coating weight (mg)
F	Human organoleptic measurement of tongue coating odour (Score 0, 1, 2, 3, 4 or 5)
G	Plaque index (Score 0 ～ 3)
H	Gingival index (Score 0 ～ 3)
I	Number of pockets 4 mm or deeper
J	Number of pockets bleeding on probing
K	Diagnosis of gastric *H. pylori* infection (^{13}C-labeled urea breath test)

[a]Measurements were performed in order from A to K.

2) Measurement of tongue coating weight (E, Table 1)

Since January 2000, tests have been performed to weigh the tongue coating.

An examiner pulls on the tip of the tongue, and after absorbing the moisture with paper wipers, the examiner collects the tongue coating on its dorsal surface anterior to the vallate papillae by scraping with a plastic spoon until all possible coating materials have been collected. The material is then weighed. The total weight (mg) of the tongue coating is determined on an electronic balance.

3) Human organoleptic measurement of tongue coating odour (F, Table 1)

All collected coating was simply sniffed with the nose of an examiner approximately 5 cm from tongue coating. Organoleptic scoring scale of tongue coating odour was rated on a scale of 0 to 5 according to oral odour measurement[12].

Assessment of periodontal parameters

The following clinical parameters were examined and recorded by one dentist.

The plaque index (Silness and Löe, 1964)[15] (G, Table 1) and the gingival index (Löe and Silness, 1963)[16] (H, Table 1) were assessed by examining the 6 teeth advocated by Ramfjord[17]. The mean values for the 6 teeth were recorded as the indices for that patient, and they were subsequently analyzed. Periodontal pockets at six sites (mesial, central and distal; buccally as well as orally) of all remaining erupted teeth were examined by Single end color coded periodontal probe (PCP 11: Hu-Friedy, U.S.A). The probing depth recorded to the nearest 1.0 mm at each site. The total number of pockets 4 mm or deeper was calculated (I, Table 1). The bleeding tendency up to 20s after pocket probing was examined. The total number of pockets bleeding on probing (J, Table 1) was also calculated.

Diagnosis of gastric *H. pylori* infection (K, Table 1)

The urea breath test[18] was performed to diagnose *H. pylori* infection. 100 mg ^{13}C-labeled urea was dissolved in 100 ml sterile water, and exhaled air was collected before drinking. Immediately after drinking, the patient rinse mouth and pharynx with a total of 150 ml of tap water. After assuming the left lateral position for 5 min, the patient sat up, and 20 min after drinking, exhaled air was collected again. The change in ^{13}C in the exhaled air was measured with a mass spectrometer (VG Isochrom-μG and VG Isochrom-MS, VG Organic, UK), and values of 2.5‰ or more were considered positive for gastric *H. pylori* infection.

Statistical methods

All analyses were performed with SAS for Windows version 6.12 (SAS Institute Inc. USA). Because of time restrictions in some patients, not all of the tests could be performed on the same day that oral odour was measured, such data were handled as missing data. Two-tailed t-tests for unpaired data were used for the comparison of age, plaque index, gingival index, the number of pockets 4 mm or deeper, the number of pockets bleeding on probing and tongue coating weight between the *H. pylori* positive and negative patients. Gender distributions were compared between the *H. pylori* positive and negative patients by chi-square test.

Based on the score for visual examination of the tongue coating, the patients were divided into two groups. Scores of 3, 4 or 6 were considered to be high, scores of 0, 1 or 2 low. Subsequently, a chi-square test was used to compare tongue coating scores on visual examination between the *H. pylori* positive and negative patients.

Based on the organoleptic score for oral odour, the patients were divided into two groups. Scores of 2, 3, 4 or 5 were taken to indicate oral odour positivity and scores of 0 or 1 oral odour negativ-

ity. Subsequently, chi-square tests were used to compare organoleptic oral odour scores assessed while the patients held their breath and while they exhaled, between the *H. pylori* positive and negative patients. Odds ratios and 95% confidence intervals (CI) were calculated.

Based on the tongue coating odour score obtained by organoleptic measurement, the patients were divided into two groups. Scores of 3, 4 or 5 were considered to be high, scores of 0, 1 or 2 low. Subsequently, a chi-square test was employed to compare tongue coating odour scores between the *H. pylori* positive and negative patients. Odds ratio and 95% CI was computed.

The common logarithms of the levels (ppm) of VSCs (H_2S, CH_3SH and $(CH_3)_2S$) measured by GC between the *H. pylori* positive and negative patients were compared by two-tailed t-tests for unpaired data.

All p-values quoted are two sided and values less than 0.05 were taken as indicating significance.

Results

Based on urea breath test results, the patients were divided into a group of 31 who were positive for gastric *H. pylori* infection and a group of 49 who were negative. The mean ages of the gastric *H. pylori* positive and negative patients were 44.7 (ranged 21～77) years and 33.8 (ranged 18～67) years, respectively. The difference in age between the two groups was significant (t-test, $p<0.01$; Table 2). Gender (males/females) ratios of the *H. pylori* positive and negative patients were 14/17 and 20/29, respectively. Male/female ratios did not differ significantly between the two groups (Table 2). Mean values and standard deviations for plaque index, gingival index, the number of pockets 4 mm or deeper, the number of pockets bleeding on probing and tongue coating weight (mg) were compared between the two groups (Table 2). The numbers of patients scored as high or low on tongue coating visual examination are shown in Table 3. There were no significant differences in plaque index, gingival index, the number of pockets 4 mm or deeper, the number of pockets bleeding on probing, tongue coating weight (mg) (Table 2) or tongue coating score on visual examination (Table 3).

Depend on the numbers of subjects organoleptically assessed as positive or negative in the measurement of oral odour (Table 4) and tongue coating odour (Table 5), odds ratios (95% CIs and

Table 2 Characteristics of patients with gastric *Helicobacter pylori* (*H. pylori*) infection, distinguished by ^{13}C-urea breath test

Variables means ± SDs	*H. pylori* infection		Significance
	Positive n=31	Negative n=49	
Age, mean (range) years	44.7 (21 ～ 77)	33.8 (18 ～ 67)	$p<0.01$[a]
Gender, Male/Female	14/17	20/29	NS
Plaque index	1.74 ± 0.53	1.72 ± 0.47	NS
Gingival index	1.00 ± 0.56	1.16 ± 0.47	NS
Number of pockets 4 mm or deeper	33.23 ± 21.04	39.35 ± 24.11	NS
Number of pockets bleeding on probing	45.50 ± 26.21	59.41 ± 28.72	NS
Tongue coating weight, mg	17.50 ± 16.20 (n=23[b])	15.0 ± 16.20 (n=24[b])	NS

[a] Mean values were compared by two-tailed t-test for unpaired data.

[b] Since January 2000 tests have been performed to measure the tongue coating weight. Eight of 31 *H. pylori* positive patients and 25 of 49 *H. pylori* negative patients did not undergo tongue coating assessment. The data were handled as missing data.

Table 3 Comparison of tongue coating scores on visual examination between *H. pylori* positive and negative patients

Score of tongue coating on visual examination[a]	Number of subjects (%) *H. pylori* Positive	Negative
High (3, 4 or 6)	15 (48.4%)	20 (40.8%)
Low (0, 1 or 2)	16 (51.6%)	29 (59.2%)
Total	31 (100.0%)	49 (100.0%)
	NS	

[a] Tongue coating was scored by multiplying the area score (0, 1, 2 or 3) by the thickness score (0, 1 or 2).

Table 4 Comparison of oral odour by organoleptic measurement between *H. pylori* positive and negative patients

Organoleptic score of oral odour assessed with patient	Number of subjects (%) *H. pylori* Positive	Negative
Holding breath		
Oral odour positive (score 2, 3, 4 or 5)	23 (74.2%)	25 (51.0%)
Oral odour negative (score 0 or 1)	8 (25.8%)	24 (49.0%)
Total	31 (100.0%)	49 (100.0%)
Exhaling breath		
Oral odour positive (score 2, 3, 4 or 5)	22 (71.0%)	30 (61.2%)
Oral odour negative (score 0 or 1)	9 (29.0%)	19 (38.8%)
Total	31 (100.0%)	49 (100.0%)

Table 5 Comparison of tongue coating odour scores obtained by organoleptic measurement between *H. pylori* positive and negative patients

Tongue coating odour score	Number of subjects (%) *H. pylori* Positive	Negative
High (3, 4 or 5)	7 (25.0%)	15 (33.3%)
Low (0, 1 or 2)	21 (75.0%)	30 (66.7%)
Total	28[a] (100.0%)	45[a] (100.0%)

[a] Three of 31 *H. pylori* positive patients and four of 49 *H. pylori* negative patients did not undergo tongue coating odour assessment. The data were handled as missing data.

chi-sguare) of oral malodour with the patients holding breath, oral malodour with the patients exhaled breath and tongue coating malodour were 2.76 (95% CI: 1.78 to 3.74, χ^2= 4.248, $p<0.05$), 1.55 (95% CI: 0.58 to 2.52, χ^2= 0.792) and 1.50 (95 % CI: 0.44 to 2.56, χ^2= 0.569), respectively (Table 6).

Mean common logarithms of H_2S, CH_3SH and $(CH_3)_2S$ levels in the *H. pylori* positive patients were −0.59, −0.90 and −1.36, in the *H. pylori* negative patients −0.84, −1.16 and −1.57, respectively (Table 7). While there were no significant difference between the groups positive and negative for gastric *H. pylori* infection in CH_3SH measured by gas chromatography, significant differences were found in H_2S and $(CH_3)_2S$ ($p<0.05$) (Table 7).

Discussion

Recently, halitosis research has received considerable attention from the general population,

Table 6 Association between oral malodour/tongue coating malodour and gastric *H. pylori* infection in organoleptic measurement, as assessed by chi-square test

	Odds ratio	95% CI[a]	chi-square
Oral malodour assessed with patient holding breath	2.76	1.78 to 3.74	4.248[b]
Oral malodour assessed with patient exhaling breath	1.55	0.58 to 2.52	0.792
Tongue coating malodour	1.50	0.44 to 2.56	0.569

[a] CI: confidence interval, [b] $p<0.05$.

Table 7 Comparison of volatile sulphur compounds (VSCs) levels assessed with gas chromatography between *H. pylori* positive and negative patients

	VSCs levels[a] *H. pylori*		
	Positive (n=28[b])	Negative (n=41[b])	p-value[c]
Hydrogen sulphide : H_2S	− 0.59 ± 0.45	− 0.84 ± 0.47	0.033
Methyl mercaptan : CH_3SH	− 0.90 ± 0.52	− 1.16 ± 0.60	0.059
Dimethyl sulphide : $(CH_3)_2S$	− 1.36 ± 0.39	− 1.57 ± 0.36	0.023

[a] Values are means ± SDs of common logarithms of measured values (ppm).
[b] Three of 31 *H. pylori* positive patients and eight of 49 *H. pylori* negative patients were not assessed by gas chromatography. The data were handled as missing data.
[c] Mean values of common logarithms of measured values (ppm) were compared by two-tailed t-test for unpaired data.

clinicians and researchers. In the literatures, the cause of halitosis has focused on the oral cavity especially tongue coating and periodontal diseases[2)3)12)~14)19)~23)]. On the contrary, in a university student questionnaire survey, as an example, there were far more respondents who thought that halitosis was caused by bad odors emerging from the gastrointestinal tract than from gases emerging from the lungs and bronchi[24)]. Moreover, quite a few of the patients examined in our department had already visited a gastroenterologist thinking that a gastrointestinal disease was the cause of their halitosis. There were even patients who had been advised to undergo gastrointestinal examinations, as part of a halitosis assessment series, by physicians or dentists. While gas analysis data have demonstrated that malodorous gases travel through the bloodstreams of hepatic and renal disease patients and are excreted via their lungs[25)26)], studies examining associations between gastrointestinal diseases and halitosis that have included analyses of malodourous gases have been rare. Marshall and co-workers first reported "putrid" breath in persons infected with *H. pylori*[27)] and the hypothesis that gastric *H. pylori* infection and halitosis are linked has been put forward[7)8)]. The levels of volatile sulphides measured with a portable monitor have even been presented[11)]. A portable monitor used to obtain the data in the literature did not measure individual gases.

In the present study, oral odour and tongue coating odour were organoleptically compared between the gastric *H. pylori* positive and negative patients, and GC measurements of three VSCs (H_2S, CH_3SH and $(CH_3)_2S$) in oral air were made. Moreover, parameters as the backgrounds of patients relating to oral odour were compared

between the two groups.

Since no significant differences were observed between the two groups in dental plaque, gingivitis, periodontal disease, or tongue coating despite the age difference, the two groups could be compared by hypothesizing that the only difference between them was in the presence or absence of gastric *H. pylori* infection.

The reason for the significant difference of age in the comparison between those who were positive and negative for *H. pylori* infection in this study is that the infection rate increased with age[28], and this is apparently a natural result.

The *H. pylori* infection rate is said to be higher in the populations of developing countries and in populations exposed to unsanitary environments[29]. The oral health status of such populations is presumed to be poor, and it is possible that their oral odour is worse. However, while the group of patients who were *H. pylori* positive was demonstrated to have more severe oral malodour in the group of patients in the present study, the results did not show that their index used as an indicator of oral health status was worse.

The results showed a significant difference in oral odour when the patients did not exhale, but no significant difference in oral odour when the patients exhaled. This may be attributable to the fact that the air was diluted by the exhaled air from the lungs and bronchi.

In addition, significant differences among the three sulphur compounds measured by GC were in H_2S and $(CH_3)_2S$. However, it is suggested that $(CH_3)_2S$ originates from the digestive tract or respiratory tract[30][31], and few past studies have focused on this gas. The possibility of other sites besides the oral cavity, such as the pharynx, larynx, bronchi, lungs, gastrointestinal tract, etc., being the source should be examined. If it is possible for *H. pylori* to cause halitosis without being present in the oral cavity, it is also possible that the cause lies in the very small amount of gas that leaks out of the upper gastrointestinal tract. CH_3SH level is strongly associated with periodontitis and oral bacteria[2][31]. This may be the reason for the absence of any significant difference in CH_3SH between the two periodontally equal groups in this study.

While there has been previous report regarding the elimination of halitosis after bacterial eradication with metronidazole and colloidal bismuth subcitrate in the patients with *H. pylori* infection[7], the data do not include objective gas measurement. If the patients stopped complaining, since there is very little correlation between the severity of the halitosis that patients complain of and the actual odour[32], objective-quantitative measurements are preferable. In addition, since metronidazole, which is used for eradication of *H. pylori*[33], also show promise of being effective against the causative agent of periodontal pathogens[34], the possibility that they exerted a temporary inhibitory effect and there by a decreased the odour was cannot be ruled out. Moreover, the use of these drugs cannot be recommended only because of a desire to suppress halitosis. Nevertheless, while in our breath odour clinic on the course giving an instruction of oral cleaning, a dentist noted an improvement in halitosis, and the patients wanted bacterial eradication therapy. Although they were referred to a gastroenterologist for this, as a treatment of the gastrointestinal tract, it was not encouraged as a procedure for treating halitosis.

While the report by Kojima[35] contains a statement that the tongue coating is a common finding among patients with gastrointestinal diseases, it was impossible to extract data from the present study showing coated tongue to be common in patients with gastric *H. pylori* infection. This also seems to be related to the fact that not all lesions are positive for *H. pylori* infection[9].

One of the shortcomings of the present study is that the subjects were limited to persons who came to the clinic complaining of halitosis and the gastric *H. pylori* positive and negative patients do not constitute a matched pair.

While the occurrence of *H. pylori* infection via the oral route is an essentially established theory, *H. pylori* is present in the oral cavity in the coccoid form, and since it is claimed to be viable but not culturable (VNC), and isolation culture is also difficult. Culture supernatants of oral microorganisms inhibited growth of the *H. pylori* strain and caused formation of the coccoid form[36]. Further research will clarify the factors confounding gastric *H. pylori* infection and halitosis.

Conclusions

In the organoleptic assessment, however, halitosis (oral malodour) with patients holding their breath was more severe in the gastric *H. pylori* positive patients than in the negative patients, oral odour with patients exhaling alveolar air and tongue coating odour did not significantly differ between the groups.

The levels of H_2S and $(CH_3)_2S$ but not CH_3SH, in oral air measured by GC were significantly higher in the *H. pylori* positive patients than in the negative patients.

Further research which confirming the relation between gastric *H. pylori* infection and halitosis is needed.

Acknowlegement

The authors would like to express their thanks to Dr. Shimizu S of the Department of Hygiene and Public Health for assistance with the statistical analysis.

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口臭と胃内ヘリコバクタピロリ感染との関連

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口臭の原因は，ほとんどが口腔内の舌苔や歯周病に起因し，その主な原因ガスは揮発性硫黄化合物（VSCs）だと言われている．胃内ヘリコバクタピロリ感染も口臭の一因であるとする報告がなされたが，VSCsを定性・定量的に測定した研究はない．この研究の目的は，胃内ヘリコバクタピロリ感染の陽性者群と陰性者群の口臭と舌苔臭を官能試験およびガスクロマトグラフィによるVSCsの測定を用いて比較することである．また，両群の背景因子として，年齢，性別，口臭に関連すると考えられる口腔内パラメーター（歯垢指数，歯肉炎指数，4mm以上の歯周ポケット数，出血歯周ポケット数，視診による舌苔スコア，舌苔重量）を検討した．80名の患者のうち，胃内ヘリコバクタピロリ感染の陽性者は31名（平均44.7歳）で，陰性者は49名（平均33.8歳）であった．年齢は陽性者の方が有意に高かったが，性別比や口腔内パラメーターなどの背景因子には差を認めなかった．口臭の官能試験では，息をとめた状態の口臭に有意差を認め（オッズ比：2.76，95% 信頼区間：1.78～3.74），胃内ヘリコバクタピロリ感染の陽性者の方が陰性者に比較して口臭は臭（くさ）いことが確認された．しかし，息を吐いた状態の口臭および舌苔臭には両群間に差を認めなかった．ガスクロマトグラフィによるVSCsの測定では，メチルメルカプタンに差を認めなかったが，硫化水素とジメチルサルファイドに有意差（$p<0.05$）を認めた．今後，胃内ヘリコバクタピロリ感染と口臭との関連について，さらに検討が必要である．