Journal of Dental Sciences (2017) 12, 156–160

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.e-jds.com

ORIGINAL ARTICLE

Comparison of oral malodors before and after nonsurgical periodontal therapy in chronic periodontitis patients

Sao-Shen Liu [a], Earl Fu [b], Hsiao-Pei Tu [a,c], Min-Wen Fu [b], Chia-Te Lin [b], E-Chin Shen [a,b*]

[a] School of Dentistry, National Defense Medical Center and Tri-Service General Hospital, Taipei, Taiwan, ROC
[b] Dental Department, Taipei Tzu Chi Hospital, Xindian, New Taipei City, Taiwan, ROC
[c] Department of Oral Hygiene, Hsin Sheng Junior College of Medical Care and Management, Longtan District, Taoyuan City, Taiwan, ROC

Received 20 October 2016; Final revision received 12 December 2016
Available online 21 March 2017

KEYWORDS
halitosis;
nonsurgical therapy;
oral malodor;
periodontitis;
volatile sulfur compounds

Abstract *Background/purpose:* Periodontal diseases have been considered as a source of oral malodor or halitosis. Improvement of oral malodor in chronic periodontitis patients has recently been observed after nonsurgical periodontal therapy in combination with tongue cleaning and/or chlorhexidine mouth rinsing. The present study, however, evaluated the impact of nonsurgical periodontal therapy alone on the oral malodor in chronic periodontitis patients by comparing the intraoral concentrations of volatile sulfur compounds (VSCs) before and after nonsurgical therapy.

Materials and methods: Using a sulfide monitor, the total VSCs in exhaled breath were measured in 80 patients with chronic periodontitis prior to and 1 month after nonsurgical periodontal therapy (re-evaluation phase). Malodor was defined as a VSC score $>$ 75 parts per billion (ppb) and $>$ 110 ppb, respectively.

Results: Significantly lower level of VSCs was recorded at periodontal re-evaluation (55 ± 9.7 ppb) than before treatment (89 ± 16.3 ppb). Before treatment, 27 (34%) patients were considered to have malodor, defined as VSCs $>$ 75 ppb. After treatment, 16 patients (20%) had VSC scores $>$ 75 ppb, including 10 of 27 patients with baseline VSC scores $>$ 75 ppb and six of 53 patients with baseline scores $\leq$ 75 ppb. The risk of malodor differed significantly before and after treatment ($P = 0.035$, McNemar's test). However, when malodor was defined as VSCs $>$ 110 ppb, the difference in risk showed only borderline significance ($P = 0.077$).

* Corresponding author. Dental Department, Taipei Tzu Chi Hospital, No. 289, Jianguo Rd., Xindian Dist., New Taipei City 231, Taiwan, ROC.
E-mail address: dentalab@tpts5.seed.net.tw (E.-C. Shen).

http://dx.doi.org/10.1016/j.jds.2016.12.004

Conclusion: On the basis of our findings, we suggest that nonsurgical periodontal therapy has a mild impact on oral malodor.

Introduction

Oral malodor, or halitosis, is a concern for many individuals, and may affect their interpersonal social communication with ensuing personal discomfort and social embarrassment.[1] Because bad breath is usually emitted from the mouth itself, the dentist or healthcare professionals are the professionals to whom individuals turn for help.[2,3] It has been shown that oral malodor may rank behind dental caries and periodontal disease as the third leading cause of patient visits to the dentist.[4]

Even though the existence of the oral malodor has been recorded in the literature, it has been a neglected problem until recently. In fact, most physicians and dental practitioners are inadequately informed about the causes and treatments for malodor. Reasons for the lack of scientific research in this area include differences in cultural and racial appreciation of odors for patients and investigators, and the absence of uniform standards in evaluation methods.[2] Moreover, there are no universally accepted standard criteria, objective or subjective, that define an oral malodor patient.

Among the various methods introduced for the measurement of oral malodor, organoleptic measurement has been suggested as a feasible chairside test for the diagnosis of intraoral halitosis in exhaled breath.[5] Moreover, organoleptic measurement is a subjective method evaluating the strength of oral malodor using a scale from 0 to 5.[6] In the present study, the portable volatile sulfur compounds (VSCs) monitor was used, based on its characteristic high sensitivity, high consistency, high accuracy, ease of use, and capacity to measure cumulative amounts of various VSCs in order to provide reliable diagnostic measurements. Recently, periodontal diseases have been considered as a major source of oral malodor,[7] and nonsurgical periodontal therapy in combination with tongue cleaning could provide improvements for the halitosis.[8–10] The present study was designed to examine whether or not the oral malodor/halitosis can be improved with nonsurgical periodontal therapy alone without tongue cleaning or mouth wash.

Materials and methods

Experimental design

A total of 80 patients (49 male and 31 female) with chronic periodontitis were included in this study. The diagnosis of chronic periodontitis was based on the American Academy of Periodontology Classification of Periodontal Diseases.[11] The mean age of the patients was 62.5 ± 10.1 years, ranging from 32 years to 78 years. The periodontal status of the patients at baseline and at the post-treatment phase, or so-called re-evaluation phase, is summarized in Table 1. Probing depth, clinical periodontal attachment level, gingival recession, and sites with plaque and bleeding upon probing were also measured at baseline and at re-evaluation. Using a sulfide monitor (Halimeter; Interscan Corporation, Chatsworth, CA, USA), the combined total sum of the VSCs in exhaled breath was measured,[12] and each patient was instructed to sit quietly without talking for 3 minutes prior to the measurement. A plastic straw was attached to the air inlet of the monitor and inserted approximately 2.5–5 cm into the oral cavity. The patients were then asked to close their mouths for 3 minutes prior to sampling to allow a full buildup of any VSC present. A series of three separate 30-second samples were collected from each patient. The peak parts per billion (ppb) values were displayed at the end of each sample period, after which an average peak ppb value for all three samples was displayed. There was a 3-minute restabilization period before each sample was taken. The VSC recorded during the first and second visit for nonsurgical periodontal therapy was used as the baseline score. All patients then received oral hygiene instructions and full mouth scaling and root planning with specific instructions not to use tongue scraping or chlorhexidine mouth rinse. When patients presented for periodontal re-evaluation in 4 weeks after the last root planning, VSCs were recorded again. A VSC score of 75 ppb was defined as the socially acceptable level as suggested in previous studies,[13,14] whereas a VSC score of ≤ 110 ppb was also considered normal according to the manufacturer's instructions (www.halimeter.com/calibration-procedure/).[15] This study received Institutional Review Board approval

Table 1 Demographics and clinical parameters of study population (n = 80) at baseline and after treatment.

	Baseline	Post-treatment	P
Age (y), mean ± SD	62.5 ± 10.1		
Sex			
Female (n, %)	31 (39)		
Male (n, %)	49 (61)		
PD (mm)	3.9 ± 0.7	3.4 ± 0.6	< 0.001*
CAL (mm)	4.7 ± 1.0	4.4 ± 0.6	< 0.001*
Rec (mm)	0.9 ± 0.5	1.0 ± 0.6	< 0.001*
Site with plaque (%)	71 ± 15	37 ± 20	< 0.001*
Site with BOP (%)	43 ± 22	26 ± 16	< 0.001*

BOP = bleeding on probing; CAL = clinical periodontal attachment level; PD = probing depth; Rec = gingival recession; SD = standard deviation.
* Significantly different measurements obtained at baseline and post-treatment.

from the Buddhist Tzu Chi General Hospital (No. 289, Jianguo Rd., Xindian Dist., New Taipei City 231, Taiwan, ROC).

Statistical analysis

Paired *t* test was used to compare the VSC scores recorded before and after treatment for periodontitis patients who received nonsurgical periodontal therapy. We measured differences in the marginal probabilities of the patients exhibiting halitosis, defined as VSC score $>$ 75 ppb or $>$ 110 ppb. Differences between baseline and the periodontal re-evaluation phase were evaluated by McNemar's test, and data are shown as the mean score and standard error of the mean. A value of $P < 0.05$ was considered statistically significant.

Results

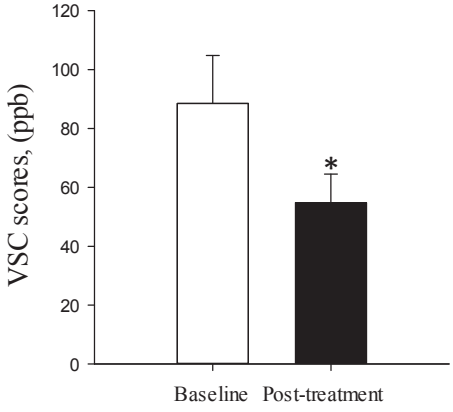
After nonsurgical periodontal therapy, the periodontal characteristics of the 80 patients improved significantly with reduced probing depths, increased clinical attachment levels, and reduced number of sites with plaque and bleeding (Table 1). In addition, the VSC score was significantly reduced at the re-evaluation phase (55 $\pm$ 9.7 ppb) if compared with the score at baseline (89 $\pm$ 16.3 ppb; $P = 0.02$; Figure 1).

At baseline, 27 out of 80 patients (34%) presented VSC scores $>$ 75 ppb (Table 2). Among these 27 patients, 17 exhibited improvements in VSC scores to $\leq$ 75 ppb at periodontal re-evaluation, whereas 10 patients remained at a level $>$ 75 ppb. By contrast, six out of 53 patients (with baseline VSC scores $\leq$ 75 ppb) had their VSC scores increased to $>$ 75 ppb. A total of 16 patients (20%) had VSC scores $>$ 75 ppb at periodontal re-evaluation. The risk of patients having oral malodor, defined as a VSC score $>$ 75 ppb, was significantly different before and after treatment ($P = 0.035$, McNemar's test). However, if halitosis was defined as a VSC score $>$ 110 ppb, the difference in the risk of patients having halitosis before and after treatment would only present a borderline difference ($P = 0.077$).

Discussion

In this study, a total of 80 patients with chronic periodontitis received nonsurgical periodontal therapy. The VSC score (89 $\pm$ 16.3 ppb) was recorded at baseline. Because there is a lack of universal agreement on what the exact VSC score should be for a patient with halitosis, the oral malodor in this study was defined as a VSC score $>$ 75 ppb according to previous studies[13,14] or $>$ 110 ppb based on the manufacturer's instructions. Before the nonsurgical periodontal therapy, 27 (34%) or 16 (20%) of 80 chronic periodontitis patients exhibited intraoral halitosis when it was defined as VSC $>$ 75 ppb or $>$ 110 ppb (Table 2). The prevalence of oral malodor in this study might be inaccurate with such a small number of sample sizes; however, halitosis is present in children, adolescents, and adults, ranging from 15% to 40% incidence.[15–19] In the general Chinese population, which is the same race as the sample group in the current study, it has been previously reported that ~28% of people have intraoral halitosis.[15]

In the present study, a lower VSC score (55 $\pm$ 9.7 ppb) was obtained at periodontal re-evaluation than the VSC score at baseline (89 $\pm$ 16.3 ppb; Figure 1). The mean change in VSC after treatment was 33.8 ppb. At the post-treatment stage, 16 (20%) or nine (11%) of 80 chronic periodontitis patients exhibited intraoral halitosis when it was defined as VSC $>$ 75 ppb or $>$ 110 ppb, respectively. Previously, elevated VSC level has been shown to be associated with the progression of periodontitis,[20] and found in breath of patients with periodontitis.[14,21] Significantly higher prevalence of intraoral halitosis also has been reported in patients with



Results are presented as mean ± SEM.

* Significantly different between baseline and post-treatment at $P < 0.05$ by paired *t* test.

ppb = parts per billion; SEM = standard error of the mean; VSC = volatile sulfur compound.

Figure 1 Comparison of VSC scores obtained from patients receiving nonsurgical periodontal therapy at baseline and post-treatment.

Table 2 Rates and distributions of patients at baseline and post-treatment ($n = 80$).

VSC score $\leq$ 75 ppb defined as socially acceptable level				
RATE:	**VSC ≤ 75 ppb**	**VSC > 75 ppb**	**Subtotal**	
Baseline	53[a] (66[b])	27 (34)	80 (100)	
Post-treatment	63 (79)	17 (21)	80 (100)	
DISTRIBUTION:	**Baseline VSC ≤ 75 ($n = 53$)**	**Baseline VSC > 75 ($n = 27$)**	**Subtotal**	***P* value**
Post-treatment VSC $\leq$ 75	47[a]	17	64	0.035*
Post-treatment VSC > 75	6	10	16	
VSC score $\leq$ 110 ppb was considered normal				
RATE:	**VSC ≤ 110 ppb**	**VSC > 110 ppb**	**Subtotal**	
Baseline	63[a] (79[b])	17 (21)	80 (100)	
Post-treatment	71 (89)	9 (11)	80 (100)	
DISTRIBUTION:	**Baseline VSC ≤ 75 ($n = 63$)**	**Baseline VSC > 75 ($n = 17$)**	**Subtotal**	***P* value**
Post-treatment VSC $\leq$ 75	59[a]	12	71	0.077
Post-treatment VSC > 75	4	5	9	

ppb = parts per billion; VSC = volatile sulfur compound.
*: Significantly different, by McNemar's test.
[a] Number of patients obtained.
[b] % of patients obtained.

periodontitis than in periodontally healthy individuals.[22] Recent studies have evaluated the effects of nonsurgical periodontal therapy with adjunct tongue scraping and/or chlorhexidine on intraoral halitosis.[9,10,23,24] However, limited information is known if nonsurgical periodontal therapy alone is effective in improving halitosis. In the present study, neither mouth rinsing nor tongue scraping were used and our results demonstrated that the prevalence of halitosis was significantly reduced, from 34% at baseline to 20% at periodontal reevaluation when VSC $\leq$ 75 ppb was considered normal (Table 2). We suggested that nonsurgical periodontal therapy alone without tongue brushing and/or mouth rinsing might have significant impacts on intraoral VSCs. Our VSC results are in agreement with a recent study that investigated the effect of periodontal treatment on halitosis,[8] with two distinct differences. One difference is that we only used nonsurgical periodontal therapy alone without tongue cleaning and/or mouth rinsing because our hypothesis was that nonsurgical periodontal treatment alone might have an impact on oral malodors. The second difference is that our re-evaluation timing is only 4 weeks after the last root planning instead of 3 months because we believe that 4 weeks is a suitable time frame since it not only is the standard re-evaluation time but it also takes ~4 weeks for soft tissue to heal.[25,26] In addition, the study showed that the subginival microbiota containing spirochetes and motile rods could be re-established after instrumentation in certain sites kept free from supragingival deposits.[25]

In fact, the detailed mechanisms or origins of intraoral halitosis are still uncertain; however, patients with halitosis, for the most part, have an oral condition[26,27] and are often associated with ear, nose, and throat, respiratory, and gastrointestinal tract infections, certain systemic diseases, metabolic disorders, and carcinomas. Halitosis has been classified into three categories: genuine halitosis, pseudohalitosis, and halitophobia. Genuine halitosis can be additionally subclassified into physiological halitosis and pathological halitosis.[28] Physiological halitosis is temporary and occurs when volatile odoriferous hematologically borne substances from foods, such as onion, garlic, and alcohol, are released into the lungs. Pathological halitosis, by contrast, stems from regional or systemic pathology such as periodontal disease, esophagitis, uremia, diabetic ketosis, respiratory and gastrointestinal conditions, hepatic and renal failure, or neoplasms.[5] In pseudohalitosis, although the patient stubbornly complains of oral malodor, it is not perceived by others. If the patient persists in believing that he or she has halitosis, even after treatment, although no social or physical evidence exists to suggest that halitosis is present, then it is classified as halitophobia.[29,30]

In ~85% of patients with persistent genuine halitosis, the odor originates from the mouth as a consequence of a complex interaction between several oral bacterial species (mainly Gram-negative anaerobic flora) with subsequent release of metabolic degradation byproducts.[31,32] The microbes ferment the peptides, mucins, and proteins found in blood, lysed neutrophils, desquamated epithelial cells, saliva, gingival crevicular fluid, and any residual food retained on the oral surface.[33] Various identified ecological niches, including the tongue coating, decayed teeth, exposed necrotic pulp, mucosal ulceration, healing site after surgical procedure, food impaction between teeth, overhang of fixed prosthesis, poor hygiene of removable prosthesis, reduced saliva flow, periodontal disease, and inflammation of peri-implant tissues are all potential causes of intraoral halitosis or oral malodor.[1,8] Quirynen et al[34] reported that halitosis cases were 43% due to tongue coating, 11% due to periodontal diseases (gingivitis and periodontitis), and 18% due to their combination.

In conclusion, the present results showed that the risk of halitosis after nonsurgical periodontal therapy alone can be significantly reduced if VSC $\leq$ 75 ppb is considered normal ($P = 0.035$), whereas only a borderline significant change if VSC $\leq$ 110 ppb is considered normal ($P = 0.077$). Consequently, we suggest that nonsurgical periodontal therapy alone has a mild impact on oral malodor. However, additional nonperiodontal etiologies still need careful investigation in the field of halitosis.

Conflicts of interest

The authors have no conflicts of interest relevant to this article.

References

1. Tangerman A. Halitosis in medicine: a review. *Int Dent J* 2002; 52(Suppl 3):201—6.
2. Bollen CM, Beikler T. Halitosis: the multidisciplinary approach. *Int J Oral Sci* 2012;4:55—63.
3. Scully C. Halitosis. *BMJ Clin Evid* 2014;2014:1305.
4. John M, Vandana KL. Detection and measurement of oral malodour in periodontitis patients. *Indian J Dent Res* 2006;17: 2—6.
5. Nachnani S, Clark GT. Halitosis: a breath of fresh air. *Clin Infect Dis* 1997;25(Suppl 2):S218—9.
6. Rosenberg M, Kulkarni GV, Bosy A, McCulloch CA. Reproducibility and sensitivity of oral malodor measurements with a portable sulphide monitor. *J Dent Res* 1991;70:1436—40.
7. De Geest S, Laleman I, Teughels W, Dekeyser C, Quirynen M. Periodontal diseases as a source of halitosis: a review of the evidence and treatment approaches for dentists and dental hygienists. *Periodontol 2000* 2016;71:213—27.
8. Iatropoulos A, Panis V, Mela E, Stefaniotis T, Madianos PN, Papaioannou W. Changes of volatile sulphur compounds during therapy of a case series of patients with chronic periodontitis and halitosis. *J Clin Periodontol* 2016;43:359—65.
9. Takeuchi H, Machigashira M, Yamashita D, et al. The association of periodontal disease with oral malodour in a Japanese population. *Oral Dis* 2010;16:702—6.
10. Tsai CC, Chou HH, Wu TL, et al. The levels of volatile sulfur compounds in mouth air from patients with chronic periodontitis. *J Periodontal Res* 2008;43:186—93.
11. Armitage GC. Development of a classification system for periodontal diseases and conditions. *Ann Periodontol* 1999;4:1—6.
12. Bolepalli AC, Munireddy C, Peruka S, Polepalle T, Choudary Alluri LS, Mishaeel S. Determining the association between oral malodor and periodontal disease: a case control study. *J Int Soc Prev Community Dent* 2015;5:413—8.
13. Miyazaki H, Sakao S, Katoh Y, Takehara T. Correlation between volatile sulphur compounds and certain oral health measurements in the general population. *J Periodontol* 1995;66: 679—84.
14. Yaegaki K, Sanada K. Biochemical and clinical factors influencing oral malodor in periodontal patients. *J Periodontol* 1992;63:783—9.
15. Liu XN, Shinada K, Chen XC, Zhang BX, Yaegaki K, Kawaguchi Y. Oral malodor-related parameters in the Chinese general population. *J Clin Periodontol* 2006;33:31—6.
16. Nalcaci R, Dulgergil T, Oba AA, Gelgor IE. Prevalence of breath malodour in 7-11-year-old children living in Middle Anatolia, Turkey. *Community Dent Health* 2008;25:173—7.
17. Villa A, Zollanvari A, Alterovitz G, Cagetti MG, Strohmenger L, Abati S. Prevalence of halitosis in children considering oral hygiene, gender and age. *Int J Dent Hyg* 2014;12:208—12.
18. Yokoyama S, Ohnuki M, Shinada K, Ueno M, Wright FA, Kawaguchi Y. Oral malodor and related factors in Japanese senior high school students. *J Sch Health* 2010;80:346—52.
19. Bornstein MM, Kislig K, Hoti BB, Seemann R, Lussi A. Prevalence of halitosis in the population of the city of Bern, Switzerland: a study comparing self-reported and clinical data. *Eur J Oral Sci* 2009;117:261—7.
20. Makino Y, Yamaga T, Yoshihara A, Nohno K, Miyazaki H. Association between volatile sulfur compounds and periodontal disease progression in elderly non-smokers. *J Periodontol* 2012;83:635—43.
21. Yaegaki K, Sanada K. Volatile sulfur compounds in mouth air from clinically healthy subjects and patients with periodontal disease. *J Periodontal Res* 1992;27:233—8.
22. Sharma NC, Galustians HJ, Qaquish J, et al. The clinical effectiveness of a dentifrice containing triclosan and a copolymer for controlling breath odor measured organoleptically twelve hours after toothbrushing. *J Clin Dent* 1999;10:131—4.
23. Quirynen M, Mongardini C, van Steenberghe D. The effect of a 1-stage full-mouth disinfection on oral malodor and microbial colonization of the tongue in periodontitis. A pilot study. *J Periodontol* 1998;69:374—82.
24. Quirynen M, Zhao H, Soers C, et al. The impact of periodontal therapy and the adjunctive effect of antiseptics on breath odor-related outcome variables: a double-blind randomized study. *J Periodontol* 2005;76:705—12.
25. Magnusson I, Lindhe J, Yoneyama T, Liljenberg B. Recolonization of a subgingival microbiota following scaling in deep pockets. *J Clin Periodontol* 1984;11:193—207.
26. Ayers KM, Colquhoun AN. Halitosis: causes, diagnosis, and treatment. *N Z Dent J* 1998;94:156—60.
27. Rosenberg M. Bad breath and periodontal disease: how related are they? *J Clin Periodontol* 2006;33:29—30.
28. Tanaka M, Anguri H, Nishida N, Ojima M, Nagata H, Shizukuishi S. Reliability of clinical parameters for predicting the outcome of oral malodor treatment. *J Dent Res* 2003;82: 518—22.
29. Toyofuku A. Psychosomatic problems in dentistry. *Biopsychosoc Med* 2016;10:14.
30. Madhushankari GS, Yamunadevi A, Selvamani M, Mohan Kumar KP, Basandi PS. Halitosis — an overview: part-I — classification, etiology, and pathophysiology of halitosis. *J Pharm Bioallied Sci* 2015;7:S339—43.
31. Ratcliff PA, Johnson PW. The relationship between oral malodor, gingivitis, and periodontitis. A review. *J Periodontol* 1999;70:485—9.
32. Scully C, Greenman J. Halitosis (breath odor). *Periodontol 2000* 2008;48:66—75.
33. Loesche WJ, Kazor C. Microbiology and treatment of halitosis. *Periodontol 2000* 2002;28:256—79.
34. Quirynen M, Dadamio J, Van den Velde S, et al. Characteristics of 2000 patients who visited a halitosis clinic. *J Clin Periodontol* 2009;36:970—5.