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

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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 ≤ 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).

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

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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

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.

### 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
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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 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. 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. 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, and found in breath of patients with periodontitis. Significantly higher prevalence of intraoral halitosis also has been reported in patients with periodontitis.

![Figure 1](https://example.com/figure1.png)

Results are presented as mean $\pm$ 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.
Recent studies have evaluated the effects of nonsurgical periodontal therapy with adjunct tongue scraping and/or chlorhexidine on intraoral halitosis. 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 was defined as socially acceptable level (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, 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 malodor. The second difference is that our re-evaluation timing is only 4 weeks after the last root planning instead of 3 months because we hypothesized that nonsurgical periodontal treatment alone without tongue cleaning and/or mouth rinsing might have significant impacts on intraoral VSCs.

In conclusion, the present results showed that the risk of halitosis after nonsurgical periodontal therapy alone can be significantly reduced if VSC ≤ 75 ppb is considered normal (P = 0.035), whereas only a borderline significant change if VSC ≤ 110 ppb was considered normal (P = 0.077). 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. 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. 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 prostheses, reduced saliva flow, periodontal disease, and inflammation of peri-implant tissues are all potential causes of intraoral halitosis or oral malodor. Quirynen et al. 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 ≤ 75 ppb is considered normal (P = 0.035), whereas only a borderline significant change if VSC ≤ 110 ppb was 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.

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

| RATE:  | VSC ≤ 75 ppb | VSC > 75 ppb | Subtotal |
|--------|---------------|--------------|----------|
| Baseline | 53a (66a) | 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 ≤ 75 | 47a                          | 17                        | 64       | 0.035*  |
| Post-treatment VSC > 75 | 6                            | 10                        | 16       |         |

**VSC score ≤ 110 ppb was considered normal**

| RATE:  | VSC ≤ 110 ppb | VSC > 110 ppb | Subtotal |
|--------|---------------|--------------|----------|
| Baseline | 63a (79a) | 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 ≤ 75 | 59a                          | 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.
Conflicts of interest

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

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