CASE REPORTS

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Lip Muscle Training Improves Halitosis and Obstructive Sleep Apnea Syndrome: A Case Report

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Halitosis is associated with mouth breathing, dry mouth, snoring, and obstructive sleep apnea syndrome (OSAS). A 40-year-old woman with moderate halitosis showed objective improvement following periodontal treatment for 1 year, but her unpleasant subjective symptoms remained. Lip muscle training using the Patakara trainer (PTR) was implemented to both increase salivary flow and treat her OSAS. After PTR training an increase in lip closure force and a decreased respiratory index (8.2 to 3.2 events/h) were observed. The patient reported resolution of mouth breathing, dry mouth, snoring, and foul odor. PTR training was associated with an improvement in halitosis and respiratory events.

Keywords: lip muscle training, halitosis, obstructive sleep apnea syndrome, dry mouth

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INTRODUCTION

Halitosis results from malodorous substances produced by anaerobic bacteria.[1] Standard treatment includes dental cleaning, tooth brushing, mechanical debridement of the tongue, and rinsing with antimicrobial agents.

Salivary flow may be central to the development of halitosis, since saliva has an antimicrobial action, and its slightly acidic pH (6.5) suppresses the growth of Gram-negative and anaerobic bacteria that produce malodorous substances.[2] Hence, dry mouth, a side effect of mouth breathing, can lead to halitosis due to reduced salivary flow.[1] Mouth breathing also increases upper airway collapsibility[3] and the occurrence[4] and severity of obstructive sleep apnea syndrome (OSAS) through a narrowed pharyngeal airway.[5]

A patient with moderate halitosis successfully treated with lip muscle training is presented.

REPORT OF CASE

A 40-year-old woman (BMI 22.1 kg/m^2) with no smoking history had a chief complaint of halitosis. She had papillary thyroid cancer and uterine fibroids (both in remission), childhood asthma and allergic rhinitis, and previously took levothyroxine sodium for hypothyroidism. She had no gastroesophageal tract problems.

Malodorous substances (H_2S, CH_3SH, and $(CH_3)_2S$) were analyzed using the Oral Chroma (Abimedical Corp., Osaka, Japan).[6] All tests were performed at least 2 hours after a meal, at the same time (10:00 am).

The respiratory disturbance index (RDI) and peripheral oxygen saturation (SpO_2) were collected for about 6 hours with the SAS-2100 (Teijin Home Healthcare Limited, Tokyo, Japan) during sleep. Data were downloaded and analyzed using QP-021W software, Ver.01-10 (Nihon Kohden, Tokyo, Japan).

Lip closure force (LCF) was measured with a lip device (BHC-V01; Patakara, Tokyo, Japan). The maximum and minimum values obtained in a 10-s period were recorded. LCFmax and LCFmin measurements were repeated three times, and mean values were calculated.

The Lip Muscle Trainer M-Patakara (Patakara Co., Ltd.) for LCF training, made from flexible, resilient plastic, and rubber, is used to increase the strength of the oral muscles. Training (5 min, 4 times/day) was performed for 2 months.[5]

In May 2012, the patient had no caries or missing teeth and no temporomandibular joint (TMJ) abnormalities. Tongue coating area was ≤ 1/3 on the dorsal tongue surface, with no soft tissue problems. She had periodontal pockets ≥ 4 mm in the molar region, and bleeding on probing around most teeth, with no tooth mobility (**Figure 1A**). H_2S was 856 ppb (recognition threshold 112 ppb), CH_3SH was 0 ppb, and $(CH_3)_2S$ was 14 ppb (recognition threshold 8 ppb, **Table 1**). The patient then received periodontal treatment for halitosis.

In July 2013, the patient showed improvement with periodontal pockets ≤ 3 mm around all teeth (**Figure 1B**). A second halitosis test showed improvement in $(CH_3)_2S$ from 14 to 5 ppb, and H_2S decreased to 206 ppb. (**Table 1**) However, halitosis remained a concern. Further questioning revealed she suffered from mouth dryness upon awakening and snoring. Her bed partner verified that she snored with her mouth open during sleep. A sleep test confirmed an RDI of 8.2 events/h and minimum SpO_2 of 91%, consistent with mild OSAS. A mandibular advancement device (MAD) was then fabricated as a routine treatment for OSAS. A Patakara trainer (PTR) was given to simultaneously treat the coexisting halitosis and OSAS. LCF measurements were taken. The patient discontinued the MAD after several days due to TMJ soreness, and lip muscle training alone was continued.

Two months after starting PTR (November 2013), both maximum and minimum LCF improved, RDI dropped to

Figure 1

A Periodontal condition at the first examination

MOB		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
BOP			• •	• •	•	• •	• •	•				• •			•••	•••
				••	• •	•••	•••	••	•••	•••	• •	•	•	•		
Depth		222	333	323	232	322	222	222	222	222	212	212	222	223	233	223
		444	434	433	313	323	323	322	222	333	322	322	333	333	233	444
	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
Depth	645	436	543	333	323	322	212	212	212	222	222	323	333	433	334	334
	322	235	434	324	223	233	223	233	233	232	233	332	223	323	333	334
BOP	•	• •	••			•••	•		•••	•••	•	•	• •	•		
		•	••	•	•	•				•	•	•		•		••
MOB	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

B Periodontal condition at 1 year after treatment

MOB		0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
BOP																
Depth		322	223	222	223	222	223	223	222	222	222	223	323	323	222	222
		323	222	232	323	222	222	222	222	222	222	323	223	323	323	223
	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
Depth	323	323	223	323	323	323	223	222	222	222	222	222	323	223	223	323
	322	333	223	222	323	223	222	222	222	222	223	323	222	323	323	223
BOP																
			•	•	•					•	•	•				
MOB	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Left panel **(A)** Periodontal condition at the first examination. Right panel **(B)** Periodontal condition at 1 year after treatment. Depth, periodontal pocket depth; BOP, bleeding on probing; MOB, tooth mobility.

Table 1—Changes of VSCs production.

	1st Test	2nd Test after periodontal treatment	3rd Test after lip muscle training
H_2S	856	206	39
CH_3SH	0	2	0
$(CH_3)_2S$	14	5	0

VSCs, volatile sulfur compounds; H_2S, hydrogen sulfide; CH_3SH, methyl mercaptan; $(CH_3)_2S$, dimethyl sulfide.

3.2 events/h, and the minimum SpO_2 increased to 97%. On the third halitosis test, H_2S was 39 ppb, below the recognition threshold (112 ppb, **Table 1**). In June 2014, the patient had continued PTR and showed favorable findings, with no snoring or malodor.

DISCUSSION

The lack of effectiveness of the periodontal approach to halitosis treatment in this case suggested other underlying contributory sources, and further questioning led to the diagnosis of OSAS. After PTR the H_2S level was below the recognition threshold, and halitosis was no longer detected. Additionally, RDI dropped and SpO_2 improved. These improvements paralleled an increase in LCF, suggesting that PTR strengthened the muscles around the lips and may have caused the decreased RDI and absence of snoring. Furthermore, following PTR training, there was a change from mouth to nose breathing with no snoring during sleep. She no longer experienced dry mouth, perhaps due to increased salivary flow associated with lip muscle training.

In conclusion, lip muscle training was associated with elimination of halitosis and improvement in the RDI and SpO_2, likely through increased LCF. A randomized, controlled trial is needed to test the efficacy of lip muscle training in patients with halitosis, OSAS, or coexisting halitosis and OSAS.

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