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An objective assessment of halitosis in children with adenoid vegetation during pre- and post-operative period

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ABSTRACT

Objectives: Although most specialists in otorhinolaryngology and pediatrics find halitosis to be a common problem in children with adenoid hypertrophy, there are no objective data on this topic in the literature. Whether adenoid hypertrophy is a risk factor for halitosis or whether halitosis is a sign of adenoid hypertrophy remains unclear. Thus, the aim of this study was to investigate whether children diagnosed with adenoid hypertrophy have a higher probability of halitosis than do children in the normal population and whether adenoidectomy can decrease oral malodor.

Methods: Forty children with adenoid hypertrophy and 40 healthy subjects aged 5–15 years were included in the study. The children with adenoid hypertrophy underwent adenoidectomy operations and were followed for 3 months. We measured volatile sulfur compounds (VSCs), hydrogen sulfide (H_2S), methyl mercaptan (CH_3SH), and dimethyl sulfide $(CH_3)_2S$ using an objective method, a portable gas chromatograph (OralChroma; AbiMedical, Osaka, Japan).

Results: The mean CH_3SH and $(CH_3)_2S$ levels were significantly different ($p < 0.05$) between the adenoid hypertrophy group and the controls. The H_2S, CH_3SH, and $(CH_3)_2S$ levels in the third postoperative month were significantly lower ($p < 0.05$) than those in the preoperative period, and there was no significant difference postoperatively between the patients with adenoid hypertrophy and controls. There was a positive correlation between age and VSC levels, and CH_3SH levels were significantly higher in patients with ventilation tube insertion, rather than just adenoidectomy.

Conclusions: There was a statistically significant association between halitosis and adenoid hypertrophy, and a significant improvement in halitosis was obtained following adenoidectomy. The present study provides an association between halitosis and adenoid hypertrophy. If there is no other oral pathology causing halitosis, halitosis can be a sign of adenoid hypertrophy in children.

1. Introduction

Halitosis (oral malodor) is a prevalent oral health disorder described as unpleasant breath generated from a person's mouth of oral or non-oral origin. Halitosis has social and personal features that may result in social shame with resulting low self-respect and self-reliance in people affected by the problem [1], especially children. This phenomenon may be a consequence of a variety of factors, and respiratory, gastrointestinal, and systemic diseases may bring about oral malodor [2]. The increase in proteolytic bacteria and the resultant increase in production of volatile sulfur compounds (VSCs) has been stated as being the main cause of foul odor [3].

The nose is one extraoral cause of pediatric halitosis. Nasal breathing is the preferred type of breathing. It has been proven that a stuffed nose disrupts the upper airway physiology and causes oral breathing, resulting in halitosis [4]. The most important anatomical reason for nasal blockage in childhood is adenoid hypertrophy. The adenoids, which are a part of Waldeyer's ring, may be a source of chronic infection despite the fact that they are a defense against

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infection. Consequently, the adenoids may become hypertrophied and obstruct the nasal airway [5]. This condition causes nasal blockage, and adenoid tissue becomes a bacterial reservoir as a result of disruption of postnasal drainage [6]. Oral breathing also causes oral mucosa changes [7].

Although a number of studies [8–11] have been carried out on pediatric halitosis, all have focused on oral causes of halitosis. There are no objective data linking halitosis and adenoid hypertrophy despite the fact that most specialists in otorhinolaryngology and pediatrics have observed halitosis in children with adenoid hypertrophy.

Therefore, the goal of this study was to determine the potential effects of adenoid vegetation on oral malodor production in pediatric patients using an objective method, a portable gas chromatograph (OralChroma; AbiMedical, Osaka, Japan) [12] and to determine whether adenoidectomy can decrease the effect of oral malodor. To the best of our knowledge, this is the first study to investigate halitosis in pediatric patients with adenoid hypertrophy.

2. Materials and methods

This prospective study was conducted at the Department of Otorhinolaryngology, GOP Taksim Education and Training Hospital (Istanbul, Turkey). The study protocol was approved by the ethics committee (GOPTEAH-KAEK, approval number: 13) and was performed according to the principles outlined in the Declaration of Helsinki. The parents of the children were informed of the nature of the study, and written consent for participation of the children in the study was obtained from all parents.

Forty randomly selected children with adenoid hypertrophy were included in the study; the children underwent adenoidectomy operations and were followed for 3 months. The study also included an age- and sex-matched control group of 40 healthy children free of adenoid hypertrophy and conditions causing halitosis.

To differentiate nasal pathologies and assess estimated adenoid size, endoscopy was performed with a rigid pediatric nasal endoscope (2.7-mm diameter; Karl Storz, Tuttlingen, Germany) in all children. Participants with previously diagnosed chronic sinusitis, allergic rhinitis, nasal polyposis, or clinically significant nasal septal deviation and other causes of nasal obstruction; tonsil disorders that may cause halitosis; systemic disease (including diabetes mellitus, renal disease, gastrointestinal tract disorders, or respiratory disease); or a history of head and neck surgery were excluded from the study.

A standardized oral examination of every child was performed by the same dentist. Patients with gingival inflammation, gingivitis, advanced periodontitis, active or severe caries, substantial false dentition, distinct tongue coating, or oral thrush were excluded. None of the children in the study wore orthodontic appliances or retainers.

The hypertrophied adenoid rhinopharyngeal obstructions were graded into four classes based on the endoscopic findings [13]. Grade 1: the findings seemed to be more or less normal; <25% of the choana was obstructed with adenoid tissue. Grade 2: adenoid tissue was limited to the upper part of the nasopharynx; <50% of the choana was obstructed, and the eustachian tube ostium could be observed. Grade 3: adenoid vegetation occupied ≤75% of the nasopharynx with significant blockage of the choanal opening and partial involvement of the eustachian tube ostium. Grade 4: the adenoid tissue reached the lower border of the choana, and >75% of the choana was obstructed; the eustachian tube ostium could not be observed. Only patients with grade 3 and 4 adenoid hypertrophy according to this classification were included in the study.

2.1. Measurement of sulfur compound levels

VSCs consist of hydrogen sulfide (H_2S), methyl mercaptan (CH_3SH), and dimethyl sulfide $(CH_3)_2S$; they were measured using a portable gas chromatograph (OralChroma; AbiMedical) [14,15]. All participants and parents were informed about the procedure. To prevent dietary or cosmetic odors from influencing VSC analysis, the participants were told to stay away from foods related to oral malodor (i.e., garlic, onion, and spicy food) and not to use commercial mouthwash for 1 day prior to the assessment. Participants were told to follow their normal oral hygiene routine on the evening prior to testing. The morning of testing, participants needed to avoid food intake as well as chewing gum, mints, drops, scents, and mouth rinses. Tooth brushing with water was allowed to avoid morning “bad breath.” All measurements were recorded in the morning between 8:30 and 11:30 a.m. by the same breath specialist. Exhaled gas samples were collected with disposable syringes (1-ml plastic syringes), which were inserted into the volunteers' oral cavities. The patients were asked to close their mouths and to breathe through the nose for 30 s before the OralChroma reading was taken. A volume of 0.5 ml of mouth air was injected into the inlet on the main unit of the OralChroma. Measurements were started automatically; the process was completed after 8 min, and the concentrations of the three gases were displayed in units of either ng/10 ml or ppb (nmol/mol). All measurements were repeated three times to improve the reliability of the study, and each concentration measured by the OralChroma was determined with analysis software (OralChroma Data Manager; AbiMedical) for validation of the data [16,17].

2.2. Statistics

Mean, standard deviation, median, minimum, maximum, frequency, and ratio were used as descriptive statistics. The distributions of variables were assessed using the Kolmogorov–Smirnov test. The Mann–Whitney *U* test was used to analyze quantitative data, and the chi-square test was used to analyze qualitative data. The Wilcoxon test was used to analyze repeated measures. SPSS software (ver. 22.0) was used for the analyses.

3. Results

A total of 80 children aged 5–15 years participated in the study: 40 in the adenoid hypertrophy group [23 boys (57.5%) and 17 girls (42.5%)] and 40 without adenoid hypertrophy as the control group [26 boys (65.0%) and 14 girls (35.0%)]. The sex ($p = 0.491$) and mean age ($p = 0.630$) of the patients and controls were not significantly different. The demographic characteristics of the patients and controls are summarized in Table 1.

The median levels of H_2S, CH_3SH, and $(CH_3)_2S$ in the patients with adenoid hypertrophy and controls are presented in Table 2. The median (first quartile, third quartile) CH_3SH level was 28.0 (8.5, 77.0) ppb in the patients with adenoid hypertrophy and 4.5 (0.0, 14.5) ppb in the controls, which was a statistically significant difference ($p < 0.001$), and the median (first quartile, third quartile) $(CH_3)_2S$ level was 9.5 (2.5, 19.0) ppb in the patients with adenoid hypertrophy and 1.0 (0.0, 3.0) ppb in the controls, which was a statistically significant difference ($p < 0.001$). The H_2S levels were not significantly different between the patients and controls ($p > 0.05$).

The mean H_2S, CH_3SH, and $(CH_3)_2S$ levels in the third postoperative month were significantly lower ($p = 0.025$, $p < 0.001$, $p < 0.001$) than in the preoperative period in both patients and controls. There were no significant differences in all three VSC levels between the two groups postoperatively ($p > 0.05$) (Table 2).

Table 1
Demographic characteristics of patients with adenoid hypertrophy and controls.

		Study group (n = 40)	Control group (n = 40)	p
Age (years)		8.45 ± 2.83	8.75 ± 2.72	0.630[a]
Sex, n (%)	Female	17 (42.5)	14 (35.0)	0.491[b]
	Male	23 (57.5)	26 (65.0)	
Ventilation tube, n (%)		14 (35.0)	–	–

Age is reported as mean ± standard deviation.
[a] Student's *t*-test.
[b] Pearson's chi-square test.

Table 2
Preoperative and postoperative VCSs (ppb) of patients with adenoid hypertrophy and controls.

	Study group (n = 40)		Control group[3] (n = 40)	$p_{1\ vs\ 3}$	$p_{2\ vs\ 3}$	$p_{1\ vs\ 2}$
	Preoperative[1]	Postoperative[2]				
H_2S	7.0 (3.0, 78.5)	4.0 (2.0, 9.5)	8.0 (4.0, 10.0)	0.259[a]	0.150[a]	0.025[b] *
CH_3SH	28.0 (8.5, 77.0)	1.5 (0.0, 22.5)	4.5 (0.0, 14.5)	<0.001[a] *	0.652[a]	<0.001[b] *
$(CH_3)_2S$	9.5 (2.5, 19.0)	0.0 (0.0, 3.0)	1.0 (0.0, 3.0)	<0.001[a] *	0.222[a]	<0.001[b] *

[1]Preoperative values of study group; [2]Postoperative values of study group, [3]Control group values; p_{1vs3}: Significance level of comparison of preoperative values of study group and control group values; p_{2vs3}: Significance level of comparison of postoperative values of study group and control group values; p_{1vs2}: Significance level of comparison of study groups' preoperative and postoperative values.
*$p < 0.05$.
Values are reported as median (Q_1, Q_3), where Q_1 = first quartile and Q_3 = third quartile.
[a] Mann–Whitney *U* test.
[b] Wilcoxon signed rank test.

A striking finding was that the CH3SH levels were significantly higher in patients with ventilation tube insertion, rather than just adenoidectomy ($p = 0.009$) (Table 3). We also observed a correlation between age and VSC levels. There were significant positive correlations between patients' age and H_2S and CH_3SH levels ($r = 0.457$, $p < 0.003$ and $r = 0.472$, $p < 0.002$, respectively) (Table 4).

4. Discussion

Although children with adenoid hypertrophy are assessed for airway blockage and ear pathologies, the assessment of halitosis is essential in childhood because halitosis has the potential to cause social restriction and reduced quality of life. However, most patients with oral breathing have halitosis, and chronic halitosis can be an important clue for adenoid hypertrophy.

There are three generally accepted methods for testing and examining the degree of oral malodor in subjects with halitosis: organoleptic measurement, sulfide monitoring, and gas chromatography (GC). The subjective halitosis detection test, commonly called the organoleptic test, has limited application because it depends on the olfactory capacity of the examiner [18]. It is practical (no need for equipment) and commonly used, but in this study, we did not use this method; instead, we used an objective method that evaluates halitosis by measuring VSC levels. Sulfide monitors such as the Halimeter (Interscan Co., Chatsworth, CA) can analyze the total sulfur content of a subject's mouth air. On the other hand, one limitation of the Halimeter is its inability to distinguish among the three VSCs, which are the primary elements of human oral malodor [19]. GC is accepted as a proper diagnostic tool for halitosis. It can measure the levels of specific VSCs. Therefore, GC results are highly objective and reproducible. GC is regarded as the gold standard for measuring oral malodor because of its capability to specifically analyze VSCs [14,15]. We used a portable gas chromatograph, the OralChroma, to measure the VSCs of morning mouth air taken from each subject. A study revealed that the gas chromatograph used in this study can evaluate the concentrations of VSCs in mouth air as well as diagnose halitosis [15].

Adenoid hypertrophy can cause halitosis by several mechanisms in pediatric patients. One of the main factors seems to be oral breathing caused by airway blockage due to adenoid hypertrophy. The literature describes the prevalence of mouth breathing as ranging from 5% to 75% of children tested [20–23]. It is probable that the oral cavity is dry because the mouth remains open most of the time. This causes disruption of oral cleaning and proliferation of gram-negative organisms that cause halitosis [24]. Saliva contains significant antibacterial features, including secreted immunoglobulin A as well as specific types of glycoproteins that adhere to and eliminate bacteria; moreover, salivary peroxidase, lysozyme, and lactoferrin affect bacterial metabolism. Any condition that changes

Table 3
CH_3SH levels were significantly higher in patients with ventilation tube insertion, rather than just adenoidectomy.

Study group (n = 40)	Ventilation tube insertion		p
	No (n = 26)	Yes (n = 14)	
H_2S	6.0 (4.0, 32.0)	13.5 (0.0, 93.0)	0.457[a]
CH_3SH	11.0 (6.0, 60.0)	62.0 (28.0, 94.0)	0.009[a]*
$(CH_3)_2S$	9.0 (0.0, 15.0)	14.0 (5.0, 23.0)	0.254[a]

*$p < 0.05$.
Values are reported as median (Q_1, Q_3), where Q_1 = first quartile and Q_3 = third quartile.
[a] Mann–Whitney *U* test.

Table 4
There were statistically significant positive correlations between patients' age and H_2S and CH_3SH levels.

Study group (n = 40)	Age	
	r	p
H_2S	0.457	0.003*
CH_3SH	0.472	0.002*
$(CH_3)_2S$	0.308	0.053

r = Spearman's correlation coefficient.

these barrier systems may make the host considerably weaker and open to microorganisms [25]. Kara et al. [11] revealed that more than 70% of children analyzed had morning halitosis as a consequence of dry mouth from breathing through the mouth during sleep.

Another mechanism contributing to halitosis is adenoid tissue acting as a bacterial reservoir because of disruption of postnasal drainage. These organisms degrade the sulfur-containing amino acids cystine, cysteine, and methionine to foul-smelling VSCs [24]. It has been demonstrated that a number of bacterial pathogens are reduced and commensal microorganisms in the nasopharynx are increased after adenoidectomy [26]. Furthermore, it has been reported that adenoidectomy has a positive effect on the treatment of chronic sinusitis in children [27,28].

As a final point, reduced mucociliary clearance has been demonstrated in patients with adenoid hypertrophy [29], with improvement noted after adenoidectomy [30]. Impaired mucociliary clearance may enhance sensitivity to upper and lower respiratory tract infections due to the extended presence of microorganisms on the respiratory mucosa and because of the existence of factors that hinder the capacity to protect against microbial colonization and invasion.

There was a significant association between halitosis and adenoid hypertrophy in this study. When compared with the control group, CH_3SH and $(CH_3)_2S$ levels were significantly higher in the adenoid hypertrophy group and decreased after adenoidectomy. The removal of the adenoid pad from the nasopharynx may relieve posterior nasal obstruction, removes the bacterial reservoir, and improves mucociliary clearance, thus improving nasal, nasopharyngeal, and oral physiology.

Additionally, the current investigation produced two remarkable findings. First, we found a correlation between age and VSC levels. There were statistically significant positive correlations between patients' age and H_2S and CH_3SH levels ($r = 0.457$, $p < 0.003$ and $r = 0.472$, $p < 0.002$, respectively (Table 3)). Second, it is interesting to note that CH_3SH levels were significantly higher in patients with ventilation tube insertion, rather than just adenoidectomy. These findings may indicate a relationship between chronic otitis media with effusion and nasal and nasopharyngeal bacterial colonization in pediatric patients with adenoid hypertrophy. It has been shown that 75.4% of children affected by obstructive adenoid hypertrophy (grade 3–4) also presented with chronic rhinitis [31]. Saylam et al. [32] pointed out the role of chronic infection of the nose and nasopharynx. Otitis media with effusion has been recently proposed, and adenoid bacterial biofilms seem to play a particularly fundamental role.

One important limitation of this study was the small sample size. Although adenoid hypertrophy is a frequent childhood disease, few potential participants met all the criteria. Therefore, the adenoid hypertrophy group included only 40 patients.

5. Conclusion

In conclusion, this study aimed to describe the relationship between adenoid hypertrophy and halitosis in pediatric patients. There was a statistically significant association between halitosis and adenoid hypertrophy, and a significant improvement in halitosis was obtained following adenoidectomy. If there is no other oral pathology causing halitosis, it must be kept in mind that halitosis can be a sign of adenoid hypertrophy in children.

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Conflict of interest

None.

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