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

Mehmet Emre Dinc [a, *], Aytug Altundag [b], Denizhan Dizdar [c], Mehmet Ozgur Avincsal [a], Ethem Sahin [d], Seckin Ulusoy [a], Ceki Paltura [a]

[a] Istanbul Gaziosmanpasa Taksim Research and Training Hospital, Department of Otolaryngology, Head and Neck Surgery, Istanbul, Turkey
[b] Istanbul Surgery Hospital, Department of Otolaryngology, Head and Neck Surgery, Istanbul, Turkey
[c] Istanbul Kemerburgaz University, Faculty of Medicine, Department of Otolaryngology, Head and Neck Surgery, Istanbul, Turkey
[d] Bayındır Hospital Icerenkoy, Department of Otolaryngology, Head and Neck Surgery, Istanbul, Turkey

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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

* Corresponding author. Istanbul Gaziosmanpasa Taksim Research and Training Hospital, Department of Otolaryngology, Head and Neck Surgery, Istanbul, Turkey.
E-mail address: dremredinc@hotmail.com (M.E. Dinc).

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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).