Introduction

The World Health Organization (WHO) defines oral health as the absence of chronic oral pain, infections, periodontal disease, or tooth decay. 1 Mouth breathing is defined as inhalation and exhalation through the mouth instead of the nose.2

Saliva’s antimicrobial properties and mechanical cleansing effects are reduced in children with mouth breathing, so the risk of dental caries and gingival inflammation increases.3,4 Previous studies have identified adenoid hypertrophy and allergic rhinitis as the two main causes of mouth breathing in children.5 The adenoids are part of the lymphatic system and are located in the superior portion of the pharynx (behind the nose). The adenoid tissues facilitate respiratory air purification by trapping microbes. These tissues shrink after age 5, and their importance is reduced due to the development of the immune system. Adenoid hypertrophy, especially in children, occurs due to bacterial infections, leading to the narrowing or obstruction of the nasal airways.6 One important factor in determining the breathing mode is the size of the nasopharynx airway (nasal or mouth breathing). Lateral cephalometric radiographs are used to evaluate the relationship between the adenoids, the nasopharyngeal airway, and nasal resistance due to adenoid hypertrophy.7 Some previous research reports gingival inflammation in mouth-breathing children. According to the study by Sharma et al,8 gingival index (GI), plaque index (PI), and bleeding on probing (BOP) were significantly greater in mouth-breathing adolescents.
The relations between the oral health and the adenoid hypertrophy compared to healthy adolescents. Also, Tamasa et al. reported that the prevalence of dental caries was greater among children with sleep disorders and mouth breathing compared to healthy children (60% and 20%, respectively).

İnönü-Sakallı et al. compared 40 mouth-breathing with 40 nose-breathing children. They utilized flexible fiberoptic nasopharyngolaryngoscopy to determine palatal tonsil hypertrophy and concluded that adenotonsillar hypertrophy can be a risk factor for dental caries, periodontal disease, and halitosis.

Alqutami et al. investigated whether the effects of oral ventilation on the prevalence of dental caries were related but did not find a relationship. In addition, the examination of the mandibular left central incisor and the maxillary right central incisor did not reveal any association between gingivitis and mouth breathing. Ahmed reported that the severity of dental caries was higher in children with chronic tonsillitis than in healthy children. Ahmed concluded that similar microbial pathogens cause dental caries and peritonsillar infections.

He ignored the other risk factors of dental caries formation, such as tooth brushing frequency, whereas we considered this factor. Also, the sample size of our study was larger than that of his study.

In mouth-breathing children, the reduction in the cleansing effect of saliva and the openness of the mouth lead to the formation of dental caries, gingival and periodontal disease, and halitosis. Although adenotonsillar hypertrophy is one of the major causes of mouth breathing in children, few studies have been conducted to assess the severity of tooth decay and overall oral health outcomes for these children. The effect of adenoid hypertrophy on oral health status has not been sufficiently clarified. Considering the adverse effects of dental caries on the quality of life of children and their families, we investigated the relationship between dental health and adenoid hypertrophy in children aged 8–15 years in Babol city. By examining this association, it is possible to reduce the risk and severity of periodontal disease and dental caries through routine dental visits, preventive dental programs, and better oral health care in these patients.

Materials and Methods
This case-control study was conducted on 100 children (8–15 years old) in Babol city. The studied children with adenoid hypertrophy were randomly selected from the oral and maxillofacial radiology clinic in 2018 and 2019 after they were referred to the clinic by an Ear, Nose, and Throat (ENT) specialist. The ENT specialist diagnosed the children by taking a thorough history and performing a clinical examination. The most important symptoms of these patients were chronic mouth breathing, hyponasal voice quality, snoring, sleep disturbance, and halitosis.

Upper airway obstruction and adenoid enlargement were two significant features observable in the patient's lateral cephalometric radiographs. The control group included 50 healthy children.

Demographic data, including age, sex, and teeth brushing per day, were collected by face-to-face interview. After the patient's consent was acquired, their lateral cephalometric radiographs were examined. The inclusion criteria were age from 8 to 15 years, good general health, and teeth brushing at least once per day. Children with systemic or mental diseases were excluded from this study.

Our study group was divided into three age groups to reduce the error variance of the statistical results.

The lateral cephalometric radiographs of the children were collected, and the Ptm-ad variable was measured in each of them. This parameter provides more useful information on the nasopharyngeal airway compared to aggressive methods such as nasoendoscopy. Ptm is defined as the intersection between the nasal floor and the posterior contour of the maxilla, and ad refers to the intersection of the posterior nasopharyngeal wall and the Ptm-So line (the midpoint on the line joining the sella and the basion). Ptm-ad, is described as the linear distance from the Ptm point to the ad point in millimeters. This parameter was significantly shorter in children with adenoid hypertrophy due to adenoidal tissue enlargement (Figure 1).

The case group (Ptm-ad < 10 mm) and the control group (Ptm-ad > 15 mm) were clinically examined under the supervision of a pediatric dentist.

We calculated the decayed, missing, and filled teeth (DMFT and dmft for permanent and primary teeth, respectively) index according to the WHO instructions.

The gingival health of children was explored by measuring the mean periodontal pocket depth, PI, and papillary marginal attached (PMA) index for teeth in millimeters. This parameter was significantly shorter in children with adenoid hypertrophy due to adenoidal tissue enlargement (Figure 1).

<table>
<thead>
<tr>
<th>16</th>
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<th>21</th>
<th>26</th>
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<tbody>
<tr>
<td>46</td>
<td>41</td>
<td>31</td>
<td>36</td>
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</tbody>
</table>

Figure 1. The Ptm-ad parameter in the lateral cephalogram
The mean periodontal pocket depth was obtained by probing three surfaces (mesiobuccal, distobuccal, and buccal) of the teeth. A periodontal probe (Williams Periodontal Probe, Hu-Friedy, Chicago, IL, USA) was used.15

For each tooth, the PI developed by Silness and Loe was determined by inspecting the thickness of the plaque accumulated on its surfaces; then, a value in the range of 0–3 was assigned to it.16

The facial surface of the gingiva around the tooth is divided into three parts: papillary gingiva, marginal gingiva, and attached gingiva. By monitoring the signs of gingival inflammation, such as redness, swelling, bleeding, etc., a score was given to each of the three parts. Afterward, the sum of these three values was calculated as the PMA index proposed by Schour and Massler.17

Finally, the average of each of the above indices was calculated for the case and control groups to compare them.

The data were gathered and entered into SPSS 22 statistical software. The chi-square test assessed the relationship between the PI and the PMA index in children with adenoid hypertrophy. The t-test and the Mann-Whitney test were employed to investigate the relationship between adenoid hypertrophy and the prevalence of dental caries and the mean depth of periodontal pockets. The P value of 0.05 was set as the significance level in our analyses.

Results
This research was conducted on a case group of 50 children with adenoid hypertrophy (8–15 years old) and a control group of 50 children (8–15 years old). In this study, the children were divided into three groups based on age: 8–9, 10–12, and 13–15. The characteristics of these three groups are shown in Table 1.

Two-way analysis of variance (ANOVA) was utilized to investigate the effect of adenoid hypertrophy on the DMFT index. The results show a significant relationship between these two variables (F = 84.33, df = 1, P < 0.001).

Based on the independent t-test analysis for each of the study groups, a considerable discrepancy was noticed between the DMFT values of the case and healthy subjects, as shown in Figure 2A.

Using two-way ANOVA, a significant difference was found between adenoid hypertrophy and the dmft index (F = 30.99, df = 1, P < 0.001). The mean dmft index of the children with adenoid hypertrophy was significantly greater than that of the healthy children (Table 2). Also, the independent t-test examination showed a meaningful contrast between the case and control groups in each of the three age groups (8–9, 10–12, and 13–15) (Figure 2B). By assessing the mean periodontal pocket depth for the case and control groups, a significant relationship was found between this index and adenoid hypertrophy (F = 276.71, df = 1, P < 0.001) (Figure 3A).

The mean periodontal pocket depth for the case and control groups was 2.93 ± 0.35 and 1.98 ± 0.23, respectively (Table 3). Using two-way ANOVA, adenoid hypertrophy was found to influence PI significantly (F = 835.20, df = 1, P < 0.001). According to Table 3, the mean PI in children with adenoid hypertrophy was bigger than those without. Furthermore, the independent t-test examination showed a remarkable contrast between the case and control groups for the three age groups (Figure 3B). We measured the mean PMA index for the case and control groups. The obtained results were as follows (Table 3):

There was a significant relationship between adenoid hypertrophy and the PMA index (F = 575.42, df = 1, P < 0.001).

Considering the three age groups, the mean PMA index of the children with adenoid hypertrophy was significantly higher than that of the healthy children (Figure 3C).

No factually noteworthy contrast was found among the bunches regarding sexual orientation conveyance (P = 0.822) and tooth brushing frequency (P = 0.262).

Discussion
In individuals with mouth breathing, dental and gingival health are adversely affected due to decreased antimicrobial properties and the mechanical cleansing effects of their saliva.18 Also, adenoid hypertrophy is one of the main reasons for mouth breathing in children.19 According to the results for all three age groups studied in this research, in children with adenoid hypertrophy, the prevalence of tooth decay was significantly greater than that in unaffected children. Besides, a significant relationship existed between gingival inflammation and adenoid hypertrophy because the mean periodontal

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Table 1. Demographic factor distribution in the study population

<table>
<thead>
<tr>
<th>Age</th>
<th>Case group (%)</th>
<th>Control group (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Female</td>
<td>Male</td>
</tr>
<tr>
<td>8–9</td>
<td>9 (18)</td>
<td>7 (14)</td>
</tr>
<tr>
<td>10–12</td>
<td>10 (20)</td>
<td>11 (22)</td>
</tr>
<tr>
<td>13–15</td>
<td>8 (16)</td>
<td>5 (10)</td>
</tr>
<tr>
<td>Total</td>
<td>27</td>
<td>23</td>
</tr>
</tbody>
</table>
The relations between the oral health and the adenoid hypertrophy

Pocket depth, PI, and PMA index were significantly greater in the case group compared to the control group. In children, adenoid hypertrophy is more frequent than in adults.\(^1\) One of the methods of evaluating the nasopharyngeal airway and determining the breathing mode is lateral cephalometric radiography.\(^7\) According to previous research,\(^21,22\) dental caries cause pain, inconvenience, early tooth loss, and disturbances in permanent tooth eruption. Furthermore, it leads to learning disorders, inadequate nutrition, decrement in academic performance, and reduction in confidence. Considering the prevalence of adenoid hypertrophy among children, we selected the age range of 8 to 15 years in this study.

Ballikaya et al\(^{12}\) evaluated the oral health status of 150 children with adenoid hypertrophy. They assessed the GI, PI, DMFT/S, and dmft/s of these children. According to their results, 67.3% of the children had dental caries. Also, gingivitis was detected in 89.3% of the children. Motta et al\(^{23}\) examined 14 children with nasal airway obstruction and 19 healthy children. They found that, compared to healthy children, the incidence of dental caries was significantly higher in the case group.

In the present study, the mean DMFT/dmft indices were significantly higher in the case group than in the control group. Consequently, it has been shown that adenoid hypertrophy is one of the factors associated with the prevalence of oral caries in children. Also, it must be emphasized that the findings of these three studies are consistent.

Tamasa et al\(^9\) reported that the incidence of dental caries was greater among children with sleep disorders and mouth breathing than among healthy children. Also, the mean periodontal pocket depth for mouth-breathing children and the controls were 2.1 ± 0 and 0 ± 0.7, respectively. Tamasa et al focused on children with sleep disorders, whereas we concentrated on children with adenoid hypertrophy. In other words, our studies are not completely similar because of the differences in the types of diseases. Despite this difference, in both studies, the role of saliva in maintaining dental and gingival health was highlighted.

The results of our research were consistent with those of İnönü-Sakallı and colleagues’ study.\(^10\) However, our method was more suitable because it used lateral cephalometric radiography as a less aggressive approach to diagnosing adenoid hypertrophy.

The data collected about the case group in Alqutami and colleagues’ study\(^4\) were self-reported. In contrast, our approach was more precise because we used clinical examination and cephalometric radiographs to determine mouth breathing. Also, we assessed more periodontal indices to investigate gingival health.

One of the most common causes of gingivitis is dental

### Table 2. Comparison of the decayed, missing, and filled teeth (DMFT and dmft for permanent and primary teeth, respectively) index between the case and control groups

<table>
<thead>
<tr>
<th>Age</th>
<th>DMFT index (Mean ± SD)</th>
<th>Control group (Mean ± SD)</th>
<th>P value</th>
<th>Case group (Mean ± SD)</th>
<th>Control group (Mean ± SD)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>6–9</td>
<td>2.31 ± 1.195</td>
<td>1.44 ± 0.964</td>
<td>0.03</td>
<td>6.50 ± 2.875</td>
<td>2.56 ± 0.727</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>10–12</td>
<td>4.00 ± 1.378</td>
<td>2.14 ± 0.854</td>
<td>&lt; 0.001</td>
<td>3.43 ± 2.441</td>
<td>1.57 ± 1.028</td>
<td>0.03</td>
</tr>
<tr>
<td>13–15</td>
<td>6.46 ± 1.613</td>
<td>2.69 ± 0.751</td>
<td>&lt; 0.001</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>4.10 ± 2.092</td>
<td>2.06 ± 0.978</td>
<td>&lt; 0.001</td>
<td>3.52 ± 3.340</td>
<td>1.48 ± 1.249</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Figure 2. Comparison of the decayed, missing, and filled teeth (DMFT (A) and dmft (B) for permanent and primary teeth, respectively) index between the case and control groups

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The relations between the oral health and the adenoid hypertrophy

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One of the most common causes of gingivitis is dental
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Consequently, any factor that favors plaque formation and accumulation, such as dry mouth in mouth-breathing individuals, will intensify gingival inflammation. In the study by Mummolo et al., PI and bacterial count were higher in the mouth-breathing adolescents compared to healthy subjects. However, between these two groups, there were no significant differences in saliva flow rates and buffer capacities. Also, according to the study by Sharma et al., GI, PI, and BOP were significantly greater in mouth-breathing adolescents compared to healthy cases.

In our study, the mean of PI and PMA index were significantly higher in the children with adenoid hypertrophy versus the control subjects. The findings of our study are consistent with those of the studies by Mummolo et al. and Sharma et al.

Gingivitis is one of the primary and common etiological factors of periodontal diseases in children and adolescents. If the early signs of gingival inflammation are neglected, deep periodontal pocket formation, alveolar bone destruction, and progressive periodontitis will follow. Keller et al. reported that the prevalence of chronic periodontitis was significantly higher in mouth-breathing people compared to healthy individuals (33.8% and 22.6%, respectively). Furthermore, the odds ratio of chronic periodontitis in the case group was 1.75 times bigger than that of healthy individuals.

Regarding the results of this study, the periodontal indices and the mean periodontal pocket depth were higher in the children with adenoid hypertrophy, so it can

Table 3. Comparison of mean periodontal pocket depth, plaque index (PI), and papillary marginal attached (PMA) index between the case and control groups

<table>
<thead>
<tr>
<th>Age</th>
<th>Periodontal pocket depth index</th>
<th>PI index</th>
<th>PMA index</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Case group (Mean ± SD)</td>
<td>Control group (Mean ± SD)</td>
<td>P value</td>
</tr>
<tr>
<td>8–9</td>
<td>3.02 ± 0.37</td>
<td>1.80 ± 0.11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>10–12</td>
<td>2.84 ± 0.40</td>
<td>2.00 ± 0.22</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>13–15</td>
<td>2.98 ± 0.20</td>
<td>2.17 ± 0.18</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Total</td>
<td>2.93 ± 0.35</td>
<td>1.98 ± 0.23</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Figure 3. Comparison of (A) mean of periodontal pocket depth, (B) plaque index (PI), and (C) papillary marginal attached (PMA) index between the case and control groups.
be concluded that adenoid hypertrophy in children has an adverse effect on gingival health.

Dental caries and periodontal diseases are multi-factor conditions, so detecting only one factor is insufficient.\textsuperscript{28}

Ignoring the socio-economic conditions of the children’s families and the low number of samples are two restrictions of this research. It can be mentioned that we did not follow up with the studied children, so we cannot conclude the precise causal relationship between dental caries and gingival inflammation and adenoid hypertrophy, so more studies are recommended in order to find the precise mechanism of the effect of mouth breathing on oral health, especially multivariable analyses and prospective cohort studies.

**Conclusion**

Based on the results of this study, adenoid hypertrophy was found to be associated with impaired oral and dental health. To describe its mechanism of effect, it seems that the antimicrobial properties and the mechanical cleansing effects of saliva are reduced in mouth-breathing children compared to healthy children. It must be emphasized that there is an acid-base imbalance in the mouths of mouth-breathing children. Because oral and dental health influences a human’s quality of life, preventive health care and periodic dental visits are advised for these children.

**Acknowledgments**

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**Formal analysis:** Hemmat Gholinia.

**Funding acquisition:** Zahra Farahzadi.

**Investigation:** Zahra Farahzadi, Efhat Khodadadi.

**Methodology:** Zahra Farahzadi, Effat Khodadadi.

**Project administration:** Zahra Farahzadi, Effat Khodadadi, Farida Abesi.

**Resources:** Zahra Farahzadi.

**Software:** Hemmat Gholinia.

**Supervision:** Efhat Khodadadi, Farida Abesi.

**Validation:** Zahra Farahzadi.

**Visualization:** Zahra Farahzadi.

**Writing—original draft:** Zahra Farahzadi, Effat Khodadadi.

**Writing—review & editing:** Zahra Farahzadi, Effat Khodadadi.

**Competing Interests**

None.

**Ethical Approval**

This study was approved by Babol Medical University (IR. MUBABOL.HRL.REC.1398.122).

**References**


