

REVIEW

The effects of adenoid hypertrophy and oral breathing on maxillofacial development: a review of the literature

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Abstract

According to modern epidemiological surveys, the prevalence of adenoid hypertrophy in children and adolescents ranges from 42% to 70%. Adenoid hypertrophy can lead to airway obstruction; thus forces a child to breathe through their mouth, thus affecting the normal development of the dental and maxillofacial area, and can lead to malocclusion. Long-term mouth breathing can cause sagittal, vertical and lateral changes in the maxillofacial area. In this article, we review the current research status relating to the association between adenoid hypertrophy, oral breathing and maxillofacial growth and development in children and adolescents. We also discuss the personalized formulation of treatment plans.

Keywords

Adenoid hypertrophy; Mouth breathing; Maxillofacial developmental deformities

1. Introduction

The adenoids, also known as pharyngeal tonsils, are the peripheral immune organs of the human body and are located at the junction of the nasopharyngeal parietal and posterior walls. The adenoids represent the first defense portal of the respiratory tract and the earliest site of exposure to various antigens by inhalation or ingestion. This can result in adenoid hypertrophy (AH) due to repeated stimulation by various antigens and their own inflammation [1]. AH is a common respiratory disease in childhood; according to modern epidemiological surveys, the prevalence of adenoid hypertrophy in children and adolescents ranges from 42% to 70% [2]. This can lead to several diseases, including sinusitis [3], mouth breathing [4], snoring [4], secretory otitis media [5] and craniofacial abnormalities [6], and can even exert serious impact on sleep structure, quality-of-life and the patient's families [7].

2. Adenoid hypertrophy

2.1 Etiology

Adenoids are present at birth, reach their maximum size in early childhood, and usually shrink slowly around the age of 10 years [1]. However, in cases where adenoid hypertrophy has caused airway obstruction, the symptoms of dental and facial malocclusion are irreversible [8].

The etiology of adenoid hypertrophy has yet to be fully elucidated [9], although several factors are known to be related to upper respiratory tract infections, including abnormal immune regulation, pharyngeal reflux, allergic reactions, passive smoking, food intolerance, micronutrient abnormalities and the irrational application of antibiotics; all of these factors

could lead to adenoid hypertrophy.

2.2 Diagnostic methods for the degree of adenoid hypertrophy

Several methods can be used to evaluate the degree of adenoid hypertrophy, including ultrasound examination, lateral nasopharyngeal films, lateral cranial localization films, Computed Tomography (CT), Magnetic Resonance Imaging (MRI), nasal acoustic reflex, electro nasopharyngoscopy, polysomnography (PSG) and nasal endoscopy.

Of these techniques, nasal endoscopy is a commonly used diagnostic test in the (Otolaryngology) ENT department. Cephalometric lateral radiographs are commonly used in orthodontics to investigate adenoid hypertrophy. Some scholars [10, 11] have suggested that cephalometric lateral radiographs are better than nasopharyngeal lateral radiographs to evaluate the relationship between the degree of adenoid hypertrophy and developmental deformities in the jaw, and that these methods can be used to evaluate the results of adenoid surgery and to detect jaw development after surgery. In a previous study, Zou [12], proposed that an A/N ratio ≤ 0.60 is normal, while an A/N ratio of 0.61–0.70 indicates moderate hypertrophy, and an A/N ratio ≥ 0.71 indicates pathological hypertrophy. The A/N ratio refers to the ratio of the adenoid thickness to the width of the nasopharyngeal airway; in other words, a line is generated along the outside of the sloping skull in a lateral cephalometric film, and the distance between the most protruding point of the adenoid and the most protruding point of the adenoid is recorded as the adenoid thickness (A). The reverse extension of the line connecting the most protruding point of the pendulous foot and the adenoids intersects the anterior edge of the nasopharyngeal

airway, and the distance between the intersection point and the pendulous foot of the pendulous line is recorded as the nasopharyngeal airway width (N) [13].

2.3 Correlation between adenoid hypertrophy and malocclusion

2.3.1 The mechanism that leads to malocclusion has yet to be identified

In 1872, Tomes [14] proposed that adenoid hypertrophy causes malocclusion in the development of the dentition and the face. Harvold [15] found that blocking the nostrils of monkeys and the absence of normal nasal breathing resulted in open-mouth breathing and jaw recession. Furthermore, some of the monkeys exhibited an open-mouth but extended their jaws forward in order to be able to breathe. One of the mechanisms considered in the current study is long-term nasal congestion; this forces a patient to breathe with an open mouth, thus leading to upper lip insufficiency owing to the physiological feedback effect of the joint muscles. This causes the patient to open his/her lips and teeth. Long-term mouth breathing causes the patient's jaw to rotate backwards and downwards, thus changing the morphological position of the jaw and increasing the anterior height. Open mouth breathing causes the position of the tongue to fall, and the upper jaw does not have the counterbalancing effect of the tongue muscles, thus leading to an increase in the force of the buccal muscles. This leads to an increase in the force of the buccal muscles, which compresses the maxillary dental arch, which adopts a narrow or "V" shape, thus resulting in an "adenoid face" [16, 17]. In another study, Kim found that the presence of adenoid tonsillar hypertrophy was significantly associated with the prevalence of dental and maxillofacial deformities [18].

Some children or adolescents presenting to the clinic with dental and maxillofacial malformations are also found to have adenoid hypertrophy but not associated with mouth breathing. Some scholars [19, 20] suggest that this may be due to blockage of the upper airway due to the hypertrophy of the adenoids, thus leading to poor breathing; furthermore, physiological feedback through the facial neuromuscular leads to changes in the muscles and soft tissues of the jaws, thus disrupting the muscular balance around the teeth and jaws and causing the appearance of odontofacial deformities. However, due to the short duration of this disease, adenoid hypertrophy does not cause open-mouth breathing [21]. It has also been suggested that facial developmental abnormalities in children may be the cause of airway obstruction; this means that airway obstruction may be due to dental and maxillofacial developmental abnormalities [19].

2.3.2 The longer the course of adenoid hypertrophy, the more severe the effect on dental and maxillofacial malformation

Yang Rong *et al.* [22] reported that the longer the course of adenoid hypertrophy, the more severe the effect on the development of the dentition and the maxillofacial surface of the child. Xue Xiaochen [23] also concluded that the effect of pathological hypertrophy of the adenoids and/or tonsils on jaw development was already present during the mammary

dentition, and the longer the course of the disease, the more severe the effect. In another study, Jieying Dai [24] studied the effect of adenoidal hypertrophy on children with An's class II malocclusion and showed that the trend of mandibular recession became more pronounced with age and the duration of disease, thus, the effect on An's class II malocclusion became more severe.

2.3.3 The more severe the degree of adenoid hypertrophy, the more pronounced the dental and maxillofacial malocclusion

Yingjiao *et al.* [25] compared lateral cephalometric radiographs from children and adolescents with moderate adenoidal hypertrophy and physiological hypertrophy and concluded that the more severe the adenoidal hypertrophy, the more pronounced the craniofacial deformity. Yaqiu *et al.* [26] investigated the correlation between different degrees of adenoid hypertrophy and dental and maxillofacial deformities and showed that patients with different degrees of adenoid hypertrophy had statistically significant differences in the labial inclination of the upper and lower incisors; the more hypertrophic the adenoids were, the greater the labial inclination of their incisors.

2.3.4 Various types of malocclusion caused by adenoid hypertrophy

Osiatuma [27, 28] found a significantly higher incidence of An's Class II, Division I classification in patients with adenoid hypertrophy than in normal children. In another study, Grippaudo *et al.* [29] found that patients with adenoid hypertrophy had a prolonged open mouth, thus resulting in overgrowth of the mandible and an imbalance in muscle function due to the low tongue position, thus resulting in inadequate lateral and sagittal development of the maxilla and eventually producing a Class III malocclusion. Many scholars also believe that adenoid hypertrophy is not directly related to odontofacial malocclusion. Feres *et al.* [30] found no differences in all cephalometric variables when compared between obstructive and non-obstructive patients by comparing the lateral cephalometric films of 100 boys and girls aged between 4 and 14 years. These authors concluded that specific craniofacial patterns may not be associated with adenoid hypertrophy.

3. Mouth breathing

Mouth breathing is one of the most common deleterious oral habits in children and a symptom of sleep disordered breathing (SDB). The prevalence of this condition ranges from 11 to 56% in children [31–34].

The normal breathing pattern in children and adolescents involves the majority of the airflow passing through the nasal cavity; if the airflow passes through the oral cavity and exceeds 25%–30% [35], then a child would be considered to be mouth breathing; this is an abnormal state. If all of the airflow passes through the oral cavity, then this is a condition known as severe mouth breathing.

3.1 Etiology and classification of mouth breathing

Anatomical mouth breathing refers to mouth breathing caused by anatomical factors and is characterized by abnormal dental and jaw development, a short upper lip and severe protrusion of the upper front teeth.

Obstructive mouth breathing is caused by nasopharyngeal diseases such as hypertrophy of the adenoids and tonsils that can cause narrowing of the upper airway. Obstructive mouth breathing is more common in children and is one of the symptoms of obstructive sleep apnea hypoventilation syndrome in children [36].

Habitual mouth breathing occurs in patients who form habits by breathing through their mouths for a long time. The formation of habits takes some time, so it often occurs in adolescents and is rare in young children [37].

3.2 Diagnostic methods for mouth breathing

History testing refers to the collection of valid information by the doctor who can ask the child and parents about the medical history during the child's visit. It is also an important basis for confirming the diagnosis, such as whether the child snores during sleep, whether the child has frequent colds, and whether there are symptoms of nasal congestion and a runny nose. It is also carefully observed whether the child opens his/her mouth to breathe in a relaxed state and whether the child has an adenoidal hypertrophy face, including open lips and leaky teeth. Sano [38] designed a questionnaire that helps to make an earlier and more accurate diagnosis of habitual mouth breathing.

Clinical tests [39] involve the cotton wool test, the closed-lip test, the mirror test and the water-containing test.

In the cotton wool test, some cotton wool is placed outside the child's mouth and lips when the child is asleep; if the child opens his/her mouth to breathe, then the cotton wool will flutter. For the closed lip test, it is important to observe the child when he/she is asleep; of the child's upper and lower open lips are closed and if the child wakes up, then the child is considered to be mouth breathing. For the mirror test, we place the mirror below the nostrils and in front of the lips of the patient; if the child is mouth breathing, then water mist will appear on the mirror in front of the lips. For the water test, the child is asked to sit still and hold about 15 mL of water for 3 min. If the child cannot hold the water for this time period, then the child is considered to have a mouth breathing habit.

In a recent study, Xing *et al.* [40] determined the tidal volume and transoral airflow of patients by improving the use of pulmonary function instruments which can qualitatively and quantitatively determine the presence of mouth breathing and the severity of mouth breathing in patients during the daytime. This is a simple method with strong clinical operability that is suitable for the quantitative examination of transoral airflow.

There are also simultaneous oronasal airflow measurement systems [41] such as polysomnography and portable polysomnography methods that can be used to diagnose the presence of mouth breathing in children.

3.3 The effect of mouth breathing on craniofacial growth

Prolonged mouth breathing can cause changes in the three-dimensional direction of dental and maxillofacial growth and development.

3.3.1 Lateral alterations

Habumugisha *et al.* [42] studied cone-beam CT images from 70 children aged 10 to 12 years in the sagittal position. These scans showed that the intermolar width, maxillary width of the molars, intercuspal width, maxillary width of cuspids and palatal area, were significantly greater in nasal-breathing patients than in mouth-breathing patients, thus indicating that the maxillary arch morphology of mouth-breathing children was narrower and longer than that of children in a nasal-breathing group. Kai Yang further suggested [43] that this is a result of an imbalance of lateral muscle forces inside and outside the upper dental arch and the relative relaxation of labial muscle tone in mouth breathers. The relatively narrow and long posterior mandibular dental arch in mouth breathing children may represent a compensation for the morphology of the maxillary dental arch.

3.3.2 Sagittal alteration

The traditional view is that mouth-breathing children will form a bony Class II malocclusion with maxillary protrusion and mandibular recession. However, the results of a study by Yang Kai [44] showed that the sagittal facial shape of mouth breathing patients can exhibit various types of I, II and III malocclusion; in addition, there were no significant differences between different groups of patients. A study by Zerui Shan and Hao Yu also demonstrated that mouth breathing was distributed across all three types of facial bone patterns [45, 46].

3.3.3 Vertical orientation alterations

A previous systematic review and meta-analysis [47] assessed the effect of mouth breathing on facial bone development and malocclusion in children and found that angle of inclination of palatal plane (SN-PP) cant of occlusion plane (SN-OP), basal plane angle (PP-MP) and sella nasion plane–Mandibular plane (SNGoGn), SN-PP, SN-OP, PP-MP and SNGoGn were all higher in patients with mouth breathing, thus indicating that the mandible and maxilla rotated backwards and downwards and that the occlusal plane was steep.

4. Correlation of adenoid hypertrophy and mouth breathing with maxillofacial deformities

Qiehui *et al.* [48] reported a correlation between mouth breathing and adenoid hypertrophy and tonsillar hypertrophy in children. Yaqiu *et al.* [26] studied the differences between simple adenoid hypertrophy and adenoid hypertrophy with mouth breathing and showed that their two groups had statistically significant (the facial plane) Npog-SN and (the former and the mandibular plane) GoGn-SN angles, thus indicating that mouth breathing affects the development of the dental and maxillofacial surfaces. By comparing lateral cranial localiza-

tion films of 11–14-year-old adolescents with different degrees of adenoidal hypertrophy with different breathing patterns, Yaling [49] found that adenoidal hypertrophy can lead to increased vertical facial distance, chin recession and increased steepness of the mandibular body. Adenoid hypertrophy with mouth breathing additionally leads to the occurrence of labial tilt and opening of the lower central incisors. In another study, Chen Jinzhao *et al.* [50] studied the craniofacial development of children with adenoid hypertrophy ($A/N > 0.6$) and mouth breathing at different stages of bone age and found that children with adenoid hypertrophy ($A/N > 0.6$) and mouth breathing were more likely to exhibit craniofacial developmental malformations. Furthermore, these authors believed that the early detection of mouth breathing behavior and the symptomatic treatment of patients with mouth breathing can prevent increased deformity following growth and development. However, further studies of moderate adenoid hypertrophy and mouth breathing are still required.

5. Treatment

5.1 Treatment of adenoid hypertrophy

5.1.1 Adenoidectomy treatment

The most effective treatment for adenoid hypertrophy is adenoidectomy. The removal of enlarged adenoids improves the symptoms of upper airway obstruction and is associated with high levels of safety [51]. Some scholars [52] have reported an increase in the width of the maxillary arch and a reduction in the frequency of lateral crossbite after surgery. The existing literature suggests that the treatment of mast adenoids and/or tonsils affects dental and maxillofacial deformities. This may indicate a relationship between nasopharyngeal obstruction (*i.e.*, upper airway obstruction) and dental and maxillofacial growth patterns.

5.1.2 Conservative treatment

Patients who do not have an indication for surgery can be treated conservatively by hormone inhalation [53], Chinese medicine [54] or a combination of Chinese and Western medicine [55].

5.2 The treatment of mouth breathing

According to its etiology, mouth breathing can be divided into obstructive mouth breathing, habitual mouth breathing and anatomical mouth breathing. When a sub-type of mouth breathing is detected and diagnosed by a doctor, appropriate treatment measures should be taken as soon as possible to avoid the development of more serious health conditions [56].

5.2.1 Family therapy

Early habitual mouth breathing can be treated by masks, lip patches and vestibular shields. These treatments are simple and can be undertaken by the patient and parents alone at home after being diagnosed by a doctor. The mask method [57] involves preparation of half of the daily-use mask, covering only the lips and exposing the nostrils. Lip patches are used to seal the child's mouth and lips, thus allowing the child to breathe through the nose. Vestibular shields are placed in the

vestibule of the mouth with the anterior portion in contact with the upper anterior teeth and the posterior portion positioned 2 to 3 mm away from the posterior teeth to induce the incisors to press in and the distance from the posterior teeth to the dental arch is expanded.

5.2.2 Treatment in the clinic

Some cases of mouth breathing can require conventional orthodontic treatment with myofunctional training to address dental and bony protrusions.

Most maxillofacial anomalies created by mouth breathing require orthodontic treatment; common methods include lower jaw anterior functional orthodontic appliances, maxillary rapid arch expansion, and maxillary anterior traction. Lower jaw anteriorly shifted functional appliances include the Twin-Block [57] and Herbst appliances [58]. The rapid maxillary arch expansion method can solve narrowing of the upper dental arch. Rapid palatal expansion (RPE), when performed by orthodontists, improves obstructive sleep apnea in children by reducing nasal airway resistance, increasing nasal volume, raising tongue posture and enlarging the pharyngeal airway [59]. Previous research indicates that RPE significantly reduces the size of both the adenoid and palatine tonsils and is associated with long-term benefit. RPE treatment can be considered as a valid and effective treatment option for the pediatric obstructive sleep apnea syndrome (OSAS) population with a narrow high arch palate and adenotonsillar hypertrophy.

Patients with small mandibular class II malocclusion should be treated during late teething by muscle agonist strategies and functional regulators. The aim of such treatment is to change the position of the lower jaw by occlusal reconstruction, thus producing muscle contraction force for the teeth, jaws, joints and other soft and hard tissues to promote growth and development [60].

Anterior jaw breathing caused by adenoid tonsillar hypertrophy can affect functional anterior jaw position and functional anterior dentition. These patients should receive orthodontic treatment as early as possible. The best age for orthodontic treatment is 3 to 5 years. The orthodontic method can be performed with a jaw joint pad orthodontic device, mandibular joint crown type bevel guide, or 2×4 orthodontic treatment. This form of correction can be completed within six months.

5.2.3 Surgical treatment

For obstructive mouth breathing, the first step is to eliminate the etiology of mouth breathing; in other words, to remove the hypertrophic adenoids. However, some patients retain the mouth breathing pattern after removal. Subsequently, multidisciplinary cooperation is required to incorporate orthodontic sequential treatment to treat their malocclusion and reduce the impact on dental and maxillofacial development.

The treatment of adenoids and/or tonsillar hypertrophy is usually a sequential process. The excision of adenoids and/or the tonsils may only be the beginning of treatment. Such patients should have their adenoids and the tonsils resected as soon as possible according to surgical indications to block a series of secondary malformations. Furthermore, the follow-up and treatment of patients needs to be multidisciplinary. Attention should not be focused solely on the upper respiratory

tract; it is important to consider the prevention and treatment of mouth breathing and the dental deformity it induces. Previous researchers [61] found that adenoid hypertrophy may represent a risk factor for dental caries, periodontal disease and bad breath. If needed, orthodontic treatment should be commenced as soon as possible. In such cases, the otolaryngologist, pediatrician and orthodontist should work as a team to treat children with adenoid tonsillar hypertrophy.

6. Conclusion

Simple adenoid hypertrophy and prolonged mouth breathing in children and adolescents can lead to oral and maxillofacial developmental deformities and exert negative impact on the psychological and physiological health of patients. Furthermore, when these conditions coexist, they can aggravate the development of malocclusion in children and cause irreversible effects on the child's jaw and face as well as the whole body. Currently, an increasing number of children with adenoid hypertrophy and mouth breathing symptoms are being seen in dentistry, although studies have shown that the recognition rate of these children by orthodontists is low [62], thus resulting in delays and even missed treatment opportunities for these children. If doctors can identify the symptoms of adenoid hypertrophy and mouth breathing in time, correctly diagnose them and commence sequential treatment as early as possible, the occurrence of malocclusion can be reduced or even avoided. Therefore, we should be fully aware of the dangers of adenoid hypertrophy, especially in children. Doctors should make an early diagnosis and deal with this condition by involving multi-disciplinary teams and the parents in order to avoid serious consequences in later life.

AVAILABILITY OF DATA AND MATERIALS

Not applicable.

AUTHOR CONTRIBUTIONS

LLX and WHW—designed the research study. YNM—wrote the manuscript. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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