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MYOFUNCTIONAL DISORDERS AND DENTAL ANOMALIES AS A RISK FACTOR FOR ADENOID HYPERTROPHY (REVIEW OF LITERATURE)

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Abstract

Relevance. This review article examines the role of myofunctional disorders and dentomaxillary anomalies in the development of adenoid hypertrophy, including recurrence after adenotomy. The relevance of this problem is supported by quantitative evidence: a systematic review reported a worldwide prevalence of malocclusion of 56 %, while malocclusions were associated with poorer oral health-related quality of life (RR/PR = 1.15; 95 % CI: 1.12–1.18; 3672 participants). In pediatric samples, malocclusion was found in 49.1 % of children aged 8–10 years, and mouth breathing was associated with transverse malocclusion (PR = 6.15; 95 % CI: 2.96–12.80).

Objective: To analyze literary sources on the role of myofunctional disorders and dentomaxillary anomalies in the development of hypertrophy of the pharyngeal tonsil.

Materials and methods: We conducted an analytical review of sources from Google Scholar, Scopus, Web of Science, PubMed, and eLIBRARY scientific databases for 2019–2024. Key classical sources outside this period were also included. The final review included 92 publications that met the predefined eligibility criteria.

Results. The reviewed evidence indicates that adenoid hypertrophy, impaired nasal breathing, mouth breathing, myofunctional disorders, and dentomaxillary anomalies are closely interrelated. Hypertrophy of the pharyngeal tonsil is a common disease of the ear, nose and throat among children aged 6 years; in frequently ill children, it is reported in 70ө90 % of cases. Oral or mixed breathing associated with adenoid hypertrophy may be accompanied by articulation disorders, with speech defects reported in 81.7 % of children with breathing disorders. Although recurrent surgery is relatively uncommon, one large retrospective study reported a revision adenoidectomy rate of 0.55 %, and 21 % of revision cases were associated with tubal tonsil hyperplasia rather than true adenoid regrowth.

Conclusions: The conducted analysis of literary data provides grounds for concluding that preventing recurrence after adenotomy is clinically important and should include an interdisciplinary, comprehensive approach to postoperative management. Such an approach should combine otorhinolaryngological follow-up, early orthodontic assessment, correction of persistent mouth breathing and dentomaxillary anomalies, and myofunctional or speech therapy when indicated.

Keywords: *Myofunctional disorders, dentomaxillary anomalies, adenoid hypertrophy, relapse, risk factors, orthodontic treatment, mouth breathing, interdisciplinary management.*

Introduction

Dentomaxillary anomalies (hereinafter – DA), characterized by disturbances in the relationship between the dental arches and abnormal positioning of individual teeth, represent not only a medical but also a significant social problem. Children and adolescents with dentomaxillary anomalies often experience impaired oral health-related quality of life, including reduced self-esteem, social discomfort, and psychosocial well-being [1]. In a

systematic review and meta-analysis, 13 cross-sectional studies were included in the qualitative synthesis and four in the quantitative synthesis; malocclusions were associated with a negative effect on oral health-related quality of life (RR/PR = 1.15; 95 % CI: 1.12–1.18; 3672 participants) [1]. Moreover, dentomaxillary anomalies remain among the most common disorders encountered in pediatric dentistry and orthodontics worldwide [2-3]. A systematic review and meta-analysis that included 77 studies reported a worldwide prevalence of malocclusion of 56 % (95 % CI: 11–99), with the highest estimates in Africa (81 %) and Europe (72 %), followed by America (53 %) and Asia (48 %) [3].

Numerous factors have been implicated in the development of dentomaxillary anomalies and are generally classified into two major groups: endogenous factors, including genetic predisposition and endocrine disorders, and exogenous factors, which are further subdivided into antenatal and postnatal influences according to the timing of exposure [4]. Craniofacial growth and dentoalveolar development are highly complex biological processes regulated by genetic mechanisms and modified by environmental factors. Disruptions in these mechanisms may lead to dentofacial abnormalities [5-7]. Endocrine diseases, including endemic goiter, pseudohypoparathyroidism, hypothyroidism, and other endocrine disorders, have also been shown to adversely affect dentoalveolar development and contribute to the occurrence of malocclusion [8-9].

The etiology of dentomaxillary anomalies is multifactorial and results from complex interactions between genetic, environmental, and functional factors [3; 10].

Among postnatal factors, the type of infant feeding plays an essential role in the normal development of the dentoalveolar system [11]. Infant feeding may be classified as breastfeeding, formula feeding, or mixed feeding. The evidence on this factor remains heterogeneous: in the systematic review by Abreu et al., 817 citations were identified, but only six studies met the eligibility criteria, and the authors concluded that the available findings did not support a clear association between breastfeeding or bottle feeding and malocclusion in mixed and permanent dentitions [11]. Nevertheless, previous studies have suggested that breastfeeding is associated with more active orofacial muscle function and may contribute to favorable craniofacial growth during early childhood [12–15]. During breastfeeding, sagittal mandibular movements and coordinated activity of the perioral and masticatory muscles may stimulate mandibular development. In contrast, prolonged bottle feeding has been reported as a potential risk factor for the development of malocclusion, although the strength of this association varies across studies [14-16].

Other postnatal factors associated with dentomaxillary anomalies include previous infectious diseases, such as measles, pertussis, and diphtheria, as well as endocrine disorders, including rickets and growth hormone deficiency. Dental caries also contributes to the development of dentofacial anomalies because reduced occlusal height resulting from tooth destruction and premature tooth loss due to complicated caries may lead to tooth displacement, crowding, and various forms of malocclusion [17]. Consequently, early tooth loss often represents the first stage in a cascade of structural changes affecting the dentoalveolar system. In a representative sample of 739 children aged 8–10 years, the prevalence of malocclusion was 49.1 %, and orofacial dysfunction was identified in 33.3 % of children; in the final model, malocclusion was associated with nonnutritive sucking habits (OR = 2.26; 95 % CI: 1.25–4.08), orofacial dysfunction (OR = 1.56; 95 % CI: 1.13–2.17), and cavitated carious lesions (OR = 1.39; 95 % CI: 1.03–1.89) [17].

Myofunctional disorders associated with dentomaxillary anomalies may contribute not only to impaired craniofacial development but also to disturbances in nasal breathing and upper airway function. Mouth breathing, altered tongue posture, and dysfunctional swallowing are increasingly recognized as factors associated with pharyngeal tonsil hypertrophy and may contribute to the persistence or recurrence of adenoid disease. In a cross-sectional study of 155 children aged 6-12 years, 71 % had some type of malocclusion, including vertical malocclusion in 45.3 %, sagittal malocclusion in 52.0 %, and transverse malocclusion in 13.6 %; mouth breathing was associated with transverse malocclusion (PR = 6.15; 95 % CI: 2.96–12.80), while atypical swallowing increased the probability of malocclusion in the vertical (PR = 1.90; 95 % CI: 1.31–2.74), sagittal (PR = 1.68; 95 % CI: 1.26–2.25), and transverse planes (PR = 2.28; 95 % CI: 1.04–5.01) [18]. A study of 141 preschool children aged 4–7 years also showed that cross-bite was as-

sociated with speech disorders (OR = 3.55; 95 % CI: 1.07–11.78), and combined occlusal disorders were associated with tongue-thrust habits (OR = 3.11; 95 % CI: 0.99–9.90) [19]. Therefore, a better understanding of the relationship between dentomaxillary anomalies, myofunctional disorders, and adenoid hypertrophy is of considerable clinical importance for both orthodontists and otorhinolaryngologists.

Objective: To review the current literature on the role of myofunctional disorders and dentomaxillary anomalies in the development of pharyngeal tonsil hypertrophy.

Materials and methods

A narrative literature review was conducted using publications indexed in the electronic scientific databases PubMed, Scopus, Web of Science, Google Scholar, and eLIBRARY. The search included articles published between 2019 and 2024 in English and Russian. Key classical sources outside the range were also included.

Eligibility criteria:

Inclusion criteria

• Original clinical studies, observational studies, randomized and non-randomized clinical trials.

• Systematic reviews and meta-analyses.

• Narrative reviews and clinical practice guidelines relevant to the topic.

• Peer-reviewed publications published in English or Russian.

• Publications published between January 2019 and December 2024.

• Classical landmark publications published before 2019 that were considered essential for understanding the pathogenesis, anatomy, or historical development of the topic.

• Studies investigating one or more of the following:

1. dentomaxillary anomalies (malocclusion);
2. orofacial myofunctional disorders;
3. mouth breathing;
4. adenoid hypertrophy;
5. craniofacial growth and development;
6. orthodontic treatment;
7. interdisciplinary management of children with adenoid hypertrophy.

Exclusion criteria

• Publications unrelated to the objectives of the review.

• Duplicate publications.

• Conference abstracts without sufficient methodological information.

• Editorials, letters to the editor, expert opinions, and non-peer-reviewed publications.

• Articles without accessible full text.

• Publications in languages other than English or Russian.

• Animal studies and in vitro studies unless directly relevant to explaining pathophysiological mechanisms.

Quality assessment.

Preference was given to peer-reviewed publications, systematic reviews, meta-analyses, clinical guidelines, and well-designed clinical studies. When several publications addressed the same topic, priority was given to the most recent and methodologically robust evidence.

The literature search was performed using combinations of the following keywords and Medical Subject Headings (hereinafter – MeSH), where applicable: dentomaxillary anomalies, malocclusion, myofunctional disorders, orofacial myofunctional disorders, mouth breathing, adenoid hypertrophy, pharyngeal tonsil hypertrophy, adenoid facies, craniofacial development, orthodontics, and children.

The review included original clinical studies, systematic reviews, meta-analyses, narrative reviews, clinical guidelines, and other peer-reviewed publications addressing the relationship between dentomaxillary anomalies, myofunctional disorders, and pharyngeal tonsil hypertrophy. Studies unrelated to the topic, duplicate publications, conference abstracts lacking sufficient methodological information, and articles without accessible full texts were excluded.

Titles and abstracts were initially screened for relevance, followed by full-text assessment of potentially eligible publications. The final review included 92 publications that met the predefined eligibility criteria.

Particular attention was paid to the etiology and risk factors of dentomaxillary anomalies, the pathophysiological mechanisms underlying myofunctional disorders, the relationship between impaired nasal breathing and adenoid hypertrophy, craniofacial growth disturbances, and contemporary interdisciplinary approaches to diagnosis, prevention, and treatment.

The collected evidence was qualitatively synthesized and critically analyzed to summarize current knowledge regarding the association between dentomaxillary anomalies, myofunctional disorders, and pharyngeal tonsil hypertrophy.

Results

An important link in the formation of a pathological bite is often the incorrect attachment of the frenulum of the upper and lower lips and the tongue, as well as a violation of the physiological abrasion of the tubercles of the teeth [20]. The influence of certain bad habits and myofunctional disorders on the nature of dental development is especially noticeable [20]. All of the above contribute to the occurrence of DA.

Myofunctional disorders (hereinafter – MD) occupy a special place in the formation of bite pathology. Some scientists, such as Khamidova T. M., Abdulloev I. B., Sharipov M., Kodirova D. R., and Tursunova H. R., believe that harmful oral habits usually occur in combinations or are replaced by others, that is, some lead to subsequent ones [21]. For example, prolonged sucking of a pacifier, which leads to disturbances in the physiological movements of the perioral muscles, is later replaced by thumb sucking and nail biting [21]. The relationship between myofunctional disorders and psychological state can be caused by several factors [22].

This direction of studying the etiology of MD has become widespread in practice, and many authors argue that in order to find the most effective approach to the treatment of MD, it is necessary to use a comprehensive approach, which may include myofunctional therapy, orthodontic treatment, as well as advice on improving the quality of sleep and reducing stress levels [23-25].

Currently, the concept of «myofunctional disorders» is quite new for a wide range of specialists. According to the opinion of Kilinc D. D., Mansiz D., myofunctional disorders are orofacial muscle-mediated anomalies of the maxillofacial region (hereinafter – MFR) that can affect the structures and formation of the stomatognathic system, which, in turn, leads to changes in bite, diseases of the temporomandibular joint, and other problems of the MFR [26]. Some sources use the term orofacial myofunctional dysfunctions (hereinafter – OMD), referring to complex disorders of the oral and facial muscles that interfere with the normal growth, development, and/or functioning of orofacial structures [22; 27]. Orofacial myofunctional disorders can result from complex interactions between acquired behavioral patterns, physical and structural factors, genetic predisposition, and environmental influences.

Most authors include the so-called «bad oral habits» in the group of myofunctional disorders. Oral habits are repetitive patterns of behavior that negatively affect the dental system (sucking fingers, hair, or pencils, biting the lip, tongue, or cheek, placing the tongue between the dental arches, absence of the occlusal reflex, bruxism [18]).

Myofunctional disorders of the maxillofacial region also include infantile swallowing, oral or mixed breathing (nose and mouth), weak chewing, incorrect tongue position, and impaired sound pronunciation [8].

According to the literature, the prevalence of myofunctional disorders of the maxillofacial region ranges from 38 % to 80 % [28-30]. Although genetic predisposition contributes to the development of orofacial myofunctional disorders, environmental and acquired factors during childhood are considered the major determinants of their development [31].

It is necessary to understand the extent of myofunctional disorders' influence on the development of dental and bite anomalies. When conducting a study of the degree of interrelation of these pathologies, the authors concluded that posture, incorrect speech articulation, the habit of pressing the tongue on the teeth, mouth breathing, biting the lips, cheeks, and the position of the hand under the cheek during sleep significantly affect the development of the dental system [29; 32]. In children with mouth breathing, deformation of the facial skeleton is observed [33; 34]. There is a term «adenoid face», which implies the presence of an incompetent upper lip, a narrow upper dental arch, retropositioned lower incisors, increased height of the anterior surface of the face, a narrow or V-shaped upper jaw, an increased angle of the plane of the lower jaw, and a posterior displacement of the lower jaw [35; 36]. It was noted that patients with oral or mixed breathing have dry lips, pale skin, and a negative impact on general well-being [36].

The author Balashova M.E., as part of her own research, conducted a survey of legal representatives of children to determine the degree of their awareness of the problem of myofunctional disor-

ders, in particular, mouth breathing [37]. The opinions of parents or guardians regarding the causes of a child's mouth breathing were studied. As a result, the survey showed that parents are familiar with the problem of MD, but 100 % of respondents associated the presence of mouth breathing with pathology of the nasal cavity, none indicated malocclusion, bad myofunctional habits, etc., as the cause of mouth breathing. Almost half of the respondents reported mouth breathing in their children, indicating a high prevalence of this myofunctional problem. The survey data demonstrated the need for educational work on MD and the harms of orofacial habits in children. It is necessary to establish partnerships with parents to jointly address anomalies and correct myofunctional disorders, and to refer patients to related specialists [37].

Many orthodontists associate the presence of an open bite in the frontal area, protrusion of the upper incisors, and narrowing of the upper dental arch with the habit of sucking the tongue [38-40]. Underdevelopment of the frontal section of the lower jaw, its distal displacement is often a consequence of biting the lower lip [41; 42]. According to studies, in children with bad habits, a distal bite is observed in 47 % of cases, a mesial one in 31.7 %, and correct closure of the first permanent molars in combination with anomalies in the position of individual front teeth and their groups, in 21.4 % of cases [43].

The role of myofunctional disorders in the formation of distal occlusion

The main myofunctional disorders leading to the formation of distal occlusion in childhood are mouth breathing, sucking of the lower lip/finger, and infantile swallowing [44]. In turn, posture or bearing is a factor that has a colossal impact on the body as a whole, since a violation of the position and shape of the spine, the development of the muscular system, and the angle of the pelvis leads to various somatic diseases of various body systems, including the dental region [45; 46].

Another author defines posture as the relationship between the parts of the body (head, neck, trunk, upper and lower limbs) in a vertical position. It includes the position, shape of the body, dynamic and static balance, as well as the neuromuscular mode of operation [47]. For an orthodontist, it is important that an incorrectly positioned head, tilted back, forward, or to the side, changes the position of the tongue depending on the degree of deviation [48].

This myofunctional disorder is characterized by muscle tension in the maxillofacial region during swallowing. The tip of the tongue is pulled too far forward and down, and the lower jaw moves backward, which forces the head and neck to move forward to force food back to the back of the throat [55]. On external examination, the orthodontist will find elongation and narrowing of the maxilla and anterior head position in most patients with infantile swallowing. Other common signs include incompetent lips, tongue protrusion during swallowing, protrusion of the maxillary teeth, mouth breathing, various disorders, and neck and shoulder tension [37].

The influence of dentoalveolar anomalies on quality of life

Based on the above, changes are observed in many aspects of patients' lives with postural disorders and dental anomalies. External manifestations of these disorders significantly affect self-esteem and the psycho-emotional state of children [49]. It is described that, according to patients and orthodontists, the presence of class II and III bite anomalies according to Angle has a significantly negative impact on the quality of life, and given that the overall prevalence of class II bite anomalies, distal occlusion, is estimated at almost 20 %, this problem is relevant both for individuals and for society as a whole [50].

Thus, the ability of myofunctional disorders to cause dental anomalies is high, which, in turn, affects the overall functioning of the child's body and the quality of life. It is also important that various myofunctional disorders can be interconnected and cause both individual dental anomalies and the same anomaly [51].

Nasal breathing disorders caused by otorhinolaryngological pathology

However, some myofunctional disorders may be initially caused by other pathologies. For example, in some cases, oral or mixed breathing is secondary, a consequence of nasal breathing difficulties or inability. The causes of nasal breathing disorders may include otolaryngological diseases, among these, in childhood, a large proportion are pathologies of the lymphopharyngeal ring [52; 53]. The lymphopharyngeal ring, or Pirogov-Waldeyer ring, includes six tonsils:

two palatine, pharyngeal, lingual, and two tubal [54]. Figure 1 shows how the authors Niedzielski A, Chmielik LP, Mielnik-Niedzielska G, Kasprzyk A, and Bogusławska J depicted the anatomy of the Waldeyer ring, noting their localization and histological structure (Figure 1).

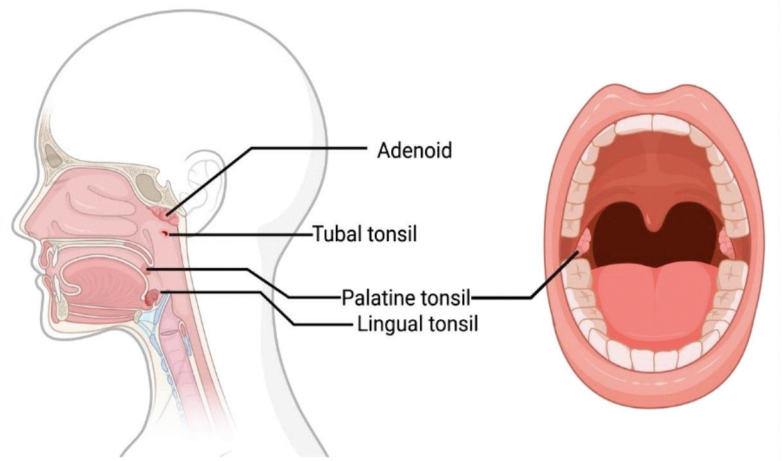


Figure 1. Anatomy of Waldeyer's ring. Adenoids, palatine, lingual, and tubal tonsils. Created with BioRender.
Source: derived from [55]

The tonsils that form the Waldeyer's pharyngeal lymphoid ring, including the pharyngeal tonsils, are already found in the embryo, but they acquire a secondary, final structure together with the lymph nodes in the postnatal period [56]. The formed pharyngeal tonsil has a quadrangular shape with rounded edges, weighing 1.5-3.3 [57].

In a normal nasopharyngeal tonsil, a surface lining is present, consisting of stratified squamous non-keratinized epithelium, followed by a basement membrane and an underlying lymphoid layer formed by both follicular and diffuse lymphoid tissue. The lymphoid follicles are arranged in a single row beneath the epithelial lining, situated between compact diffuse and parafollicular lymphoid tissue. These follicles exhibit clear polarity, with their upper poles oriented toward the covering or lacunar epithelium, while the vascular and adventitial layers are located inferiorly. The stromal framework is composed of reticular connective tissue. In certain areas, the epithelium is infiltrated by lymphocytes and granular leukocytes, particularly granulocytes, on the surface of which microorganisms are present. Exposure to microorganisms and the enzymatic activity of leukocytes lead to degeneration, death, and subsequent desquamation of some epithelial cells [57; 58].

The enlarged lymphoid tissue of the adenoids is more often subject to the inflammatory process, since normal immunological processes in the pharyngeal tonsil, associated with the function of secretory immunoglobulin A and the production of interleukins, are disrupted [59-62]. This affects the child's overall immune status [63]. Constant high antigen load due to contact with a large number of viruses, caused by social aspects, often at the age of the highest incidence of hypertrophy of adenoids (hereinafter – HA), children are in various groups (pre-school, school institutions), in combination with the immaturity of the immune system of children, leads to a violation of the regenerative processes of the mucous membrane. The damaged basement membrane and the proper layer of the mucous membrane of the tonsil stimulate fibroblasts to release transforming growth factor beta, leading to tonsil tissue hyperplasia [64]. There is evidence that hypertrophied lymphoid organs and lymphoid aggregates in the pharynx and nasopharynx of children are directly involved in barrier defense mechanisms against respiratory viral and bacterial exposure. In chronic inflammatory conditions affecting the tonsils, pronounced alterations in metabolic processes within their tissues are observed [32-35]. Scientific publications place particular emphasis on the role of local immune responses in the development of inflammation, including the evaluation of secretory immunoglobulin A levels, interleukin production, and other immune mediators [59-62].

Enlarged adenoid tissue is localized in the area of the posterior part of the nasopharyngeal

vault and disrupts normal nasal breathing. The severity of respiratory failure depends on the degree of hypertrophy of the nasopharyngeal tonsil and on congestion of the nasal mucosa. There are 4 degrees of adenoid growth: at degree I, the adenoids cover up to 1/3 of the vomer, at degree II – up to 1/2, at degree III – the hypertrophied tonsil completely covers the vomer, and at degree IV – the choanae are completely or almost completely closed [65; 66].

When adenoids grow pathologically, the passage of air through the respiratory tract becomes difficult. In the presence of such obstacles, breathing through the mouth is an adaptive mechanism [66]. Habitual mouth breathing is accompanied by hypotonia of the orbicularis oris muscle, which leads to a violation of the closure of the lips and prevents the normal development of the lower jaw [42]. To allow airflow to pass freely through the oral cavity into the nasopharynx and beyond into the respiratory tract, the child is forced to lower the tongue. Normally, the tongue should be adjacent to the hard palate. The lack of tongue support for the maxillary arch slows the growth of upper-jaw bone structures, contributing to upper-jaw narrowing [42].

According to the literature, hypertrophy of the adenoids is the most common otological, laryngological, and rhinological (hereinafter – ENT) disease among children aged 6 years, and in frequently ill children, hypertrophy of the pharyngeal tonsil is diagnosed in 70-90 % of cases [67-69]. This age is significant because it marks the beginning of the mixed dentition period, when the roots of the temporary lower central incisors undergo physiological resorption, and they then fall out, leaving a defect in the child's dental arch for a period of time [70]. When performing the function of speech and swallowing, the tongue, which occupies the position at the bottom of the oral cavity, is additionally displaced forward to fill the defect of the dental row. Thus, additional myofunctional disorders, such as tongue placement between the dental rows and infantile swallowing, arise.

Regarding the prevalence of HA by gender, the literature reports conflicting findings: the author Manuilova L.V. reports that HA occurs with equal frequency in both male and female children [71]. Abdullaeva R.R. conducted a study showing that hypertrophy of the nasopharyngeal tonsils is observed mainly in boys (60 %) [72]. Gerhardsson H, Stalfors J, Odhagen E, Sunnergren O. also indicate a higher prevalence of HA among boys [73]. Some authors associate this with the fact that allergic diseases, such as rhinitis, vernal keratoconjunctivitis, and bronchial asthma, are more common in male children than in girls [74-76].

In turn, habitual mouth breathing leads to weakness of the masticatory muscles, which affects the nature of the food predominantly consumed by children with adenoid hypertrophy, namely soft foods. This disorder is called «sluggish chewing» [77]. A bad habit, such as sluggish or lazy chewing, further contributes to a decrease in the chewing load and hypotonia of the perioral muscles, and negatively affects the formation of bone elements in the maxillofacial region [78; 79].

The oral or mixed breathing that develops with HA is associated with articulation disorders. 81.7 % of children with breathing disorders, including those due to HA, have various speech defects, such as problems with pronunciation of individual speech sounds, loss of speech fluency, and change in voice tone - rhinophonia [80].

The influence of myofunctional disorders and dentoalveolar anomalies on the character of the growth of the pharyngeal tonsil

An important aspect of the pharynx anatomy is that it is a bifurcated structure, serving as a connecting link between the nasal and oral cavities. When the entrance to the airway, such as the nasal valve, is blocked, the airflow is redirected through the oral cavity. Despite this, the nasopharynx remains under negative pressure. The pressure correlates with nasal resistance and can trigger inflammation of the nasopharyngeal mucosa, resulting in a complex inflammatory cascade [81].

Thus, there is an opinion that the presence of a myofunctional disorder, namely mouth breathing, can contribute to the occurrence of inflammation in the nasal cavity, and, subsequently, the lymphoid structure - the pharyngeal tonsil [81].

An important difference between nasal and oral breathing is that air passing through the nose is enriched with nitric oxide (hereinafter – NO) [82]. Nitric oxide is a powerful bronchodilator and vasodilator, and has antiviral and antibacterial effects [83; 84]. These data show that this protective function is not performed during oral breathing, which can contribute to frequent acute respiratory diseases of viral and bacterial origin, and that the overall allergic load on the body, and on the pharyngeal

tonsil in particular, increases.

With mouth breathing, unlike nasal breathing, there is insufficient humidification, warming, and purification of air, constant cooling of the oral cavity, pharynx, and lower respiratory tract [85]. The mass of microbes and dust particles inhaled during mouth breathing settles on the mucous membrane of the throat [86; 87]. The lymphatic pharyngeal ring is permanently exposed to viral and bacterial pathogens, which leads to repeated episodes of adenoiditis – an inflammatory process of the pharyngeal tonsil [88]. As a result of adenoiditis, the volume of lymphoid tissue increases, persistent hypertrophy of the nasopharyngeal tonsil develops, further interfering with normal breathing, and a vicious circle of interconnected links arises [89].

The author Balashova M.E. presented the influence of hypertrophy of the pharyngeal tonsil and habitual mouth breathing on the state of the upper respiratory tract and posture, and the presence of a relationship between the listed pathological conditions in the form of a drawing (Figure 2) [37].

According to the data she presented, conditions such as hypertrophy of the pharyngeal tonsil, mouth breathing, disturbance of myodynamic balance, chronic hypoxia, disturbance of the microflora, and decreased immunity are closely interconnected and interdependent [37].

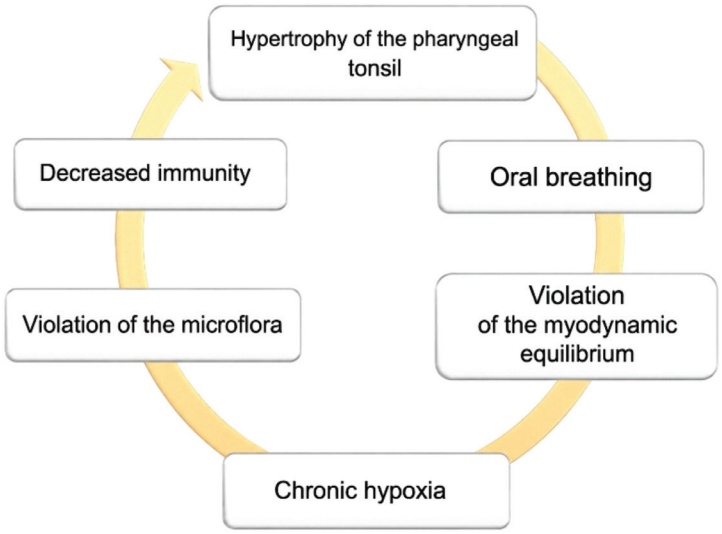


Figure 2. The role of development and interrelation of conditions associated with chronic mouth breathing.
Source: derived from [37]

Discussion

According to contemporary preventive approaches aimed at reducing the incidence of adenoiditis and preventing recurrence of adenoid hypertrophy, a complex of hygienic, therapeutic, and supportive measures is recommended [34]. These include maintaining adequate daily hydration and ensuring optimal microclimatic conditions through regular humidification and air purification in premises where the child spends prolonged periods, such as home, pre-school, and school settings. Strict adherence to hand hygiene is advised after contact with crowded environments and after coughing or sneezing. Preventive management also encompasses irrigation-elimination therapy with isotonic saline solutions twice daily, timely and rational treatment of each episode of acute respiratory viral infection, and adequate management of allergic diseases, particularly allergic rhinitis. Additional measures involve providing balanced nutrition, implementing respiratory gymnastics, administering vitamin therapy, and correcting oral breathing associated with dentofacial anomalies [90]. The clinical relevance of this preventive block is supported by data showing that chronic mouth breathing is not an isolated symptom: in a multidisciplinary study of 498 mouth-breathing children aged 9-17 years, functional mouth breathing accounted for 53.2 % of cases, allergic rhinitis for 16.9 %, adenoid hypertrophy for 14.3 %, and combined allergic rhinitis with adenoid hypertrophy for 15.7 % [80].

Recurrent adenoid hypertrophy remains a poorly understood clinical problem, with limited evidence regarding the mechanisms and predictors of recurrence [91]. In the retrospective study by Monroy et al., 13,005 adenoidectomies or adenotonsillectomies were performed over an 11-year period, and 72 children underwent revision adenoidectomy, corresponding to a revision rate of 0.55 %; the average interval between the primary and revision procedures was 4.3 years [91]. Importantly, at least 15 of 72 revision cases (21 %) were ultimately attributed to tubal tonsil hyperplasia rather than true adenoid regrowth, which further emphasizes the complexity of postoperative recurrence assessment [91].

Several studies have suggested that orofacial myofunctional disorders, particularly persistent mouth breathing, may contribute to chronic inflammation of the nasal cavity and subsequently of the pharyngeal tonsil through impaired nasal physiology, reduced filtration and humidification of inspired air, and altered upper airway function [55; 81; 85]. Persistent inflammation, in turn, may promote recurrent adenoid hypertrophy, although the exact mechanisms underlying adenoid regrowth remain incompletely understood [55; 64; 91]. Quantitative data from pediatric orthodontic samples support the clinical significance of these functional factors: in children aged 6–12 years, mouth breathing increased the probability of transverse malocclusion more than sixfold (PR = 6.15; 95 % CI: 2.96–12.80), and atypical swallowing was associated with malocclusion in all three planes of space [18].

Although conventional postoperative recommendations, including nasal irrigation, adequate treatment of upper respiratory tract infections, control of allergic diseases, and maintenance of appropriate environmental conditions, are essential for reducing infectious and inflammatory risk factors, they do not adequately address persistent functional abnormalities that may remain after adenoidectomy [34; 59; 90]. Consequently, postoperative rehabilitation should extend beyond routine otorhinolaryngological follow-up and incorporate a multidisciplinary approach involving an orthodontist and a myofunctional therapist, with the participation of a speech therapist when indicated [28; 34; 92]. This approach is also supported by data from preschool children, where cross-bite was associated with speech disorders (OR = 3.55; 95 % CI: 1.07–11.78), indicating that occlusal and functional disturbances may overlap with speech-related problems [19].

Available evidence indicates that restoration of upper airway patency does not necessarily result in spontaneous normalization of the breathing pattern. In many children, mouth or mixed breathing persists after adenoidectomy despite successful surgical removal of the mechanical obstruction [55; 81; 85]. Persistent mouth breathing should therefore be regarded not merely as a residual symptom but as a functional disorder capable of influencing subsequent craniofacial development. The multidisciplinary assessment by Alhazmi showed that 81.7 % of mouth-breathing children had orofacial myofunctional disorders combined with speech abnormalities; frontal lisp was found in 36.1 %, stuttering in 19.2 %, and two or more speech impediments occurred simultaneously in 10.6 % of children [80].

Mouth breathing has long been recognized as an important environmental factor affecting craniofacial growth and dentofacial development. If left uncorrected, it may contribute to maxillary constriction, increased lower facial height, posterior crossbite, Class II malocclusion, and the characteristic craniofacial phenotype commonly referred to as adenoid facies [33; 35; 81; 85]. These findings emphasize the importance of orthodontic assessment during postoperative follow-up in children who have undergone adenoidectomy. This is particularly important because malocclusion is common even in general pediatric samples: a systematic review and meta-analysis reported a worldwide prevalence of 56 %, and a study of 739 children aged 8–10 years reported malocclusion in 49.1 % of children [3; 17].

Furthermore, myofunctional rehabilitation should be considered an integral component of postoperative management. Growing evidence suggests that myofunctional therapy is effective in restoring physiological breathing, swallowing, and tongue posture, thereby improving orofacial muscle function and contributing to long-term functional stability [24; 26; 46]. The rationale for correcting these functions is supported by the association of orofacial dysfunction with malocclusion (OR = 1.56; 95 % CI: 1.13–2.17) and by the stronger association observed for nonnutritive sucking habits (OR = 2.26; 95 % CI: 1.25–4.08) in children aged 8–10 years [17].

Taken together, the available evidence sug-

gests that prevention of recurrent adenoid hypertrophy should not rely solely on otorhinolaryngological management. Early orthodontic assessment, timely correction of dentofacial anomalies and persistent myofunctional disorders, together with multidisciplinary collaboration involving myofunctional therapists and speech therapists when indicated, may improve functional rehabilitation and potentially reduce the risk of recurrent adenoid hypertrophy [28; 34; 37; 92]. From a public-health and clinical perspective, the need for interdisciplinary follow-up is strengthened by evidence that malocclusions negatively affect oral health-related quality of life (RR/PR = 1.15; 95 % CI: 1.12–1.18; 3672 participants) [1].

Therefore, the prevention of recurrent adenoid tissue proliferation requires the involvement of an orthodontist capable of identifying existing dentoalveolar anomalies and myofunctional disorders, including persistent mouth breathing, and implementing appropriate therapeutic measures to correct them. The reviewed numerical evidence supports this conclusion: recurrent surgery is uncommon but clinically relevant (0.55 % in one large retrospective series), while functional and occlusal abnormalities are considerably more frequent and show measurable associations with malocclusion, speech disorders, and oral health-related quality of life [1; 17-19; 80; 91].

Limitations. This review has several limitations. Only articles published in English and Russian between 2019 and 2024 were included. The heterogeneity of the reviewed studies, differences in diagnostic criteria, and variability in study designs limited direct comparison of the published evidence. Future systematic reviews and meta-analyses are required to strengthen the evidence base.

Conclusion

The relevance of the topic of our analytical review of sources is confirmed by the results of several studies published in domestic and international literature.

Hypertrophy of the adenoids is the most common ENT disease among children aged 6 years, a time when orthodontic intervention is highly effective and necessary in cases of dentoalveolar anomalies. After using the existing surgical method of treating hypertrophy of the adenoids – adenotomy, in some cases, relapses occur. The available data on the relationship between myofunctional disorders and relapses of hypertrophy of the adenoids give reason to believe that the orthodontic treatment method is mandatory after adenotomy.

The present study demonstrates the relevance of interdisciplinary collaboration between the orthodontist and other specialists, including the otorhinolaryngologist, pediatrician, osteopath, psychologist, and speech therapist-defectologist.

The implementation of a comprehensive multidisciplinary approach in the management of dentoalveolar anomalies and myofunctional disorders is essential in contemporary clinical practice. The orthodontist should maintain close collaboration with specialists from various fields, including dental and broader medical disciplines, as well as with professionals in education. In this regard, integrating a team-based, multidisciplinary approach is necessary for advancing dentistry in the Republic of Kazakhstan and globally.

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МИОФУНКЦИОНАЛДЫ БҰЗЫЛУЛАР ЖӘНЕ ТІС-ЖАҚ АУЫТҚУЛАРЫ АДЕНОИДТЫ ГИПЕРТРОФИЯНЫҢ ҚАУІП ФАКТОРЫ РЕТІНДЕ (ӘДЕБИ ШОЛУ)

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Аңдатпа

Кіріспе. Бұл шолу мақаласында аденоидты гипертрофияның, оның ішінде аденотомиядан кейінгі рецидивтердің дамуындағы миофункционалды бұзылулар мен тіс-жақ аномалияларының рөлі қарастырылады. Мәселенің өзектілігі сандық деректермен расталады: жүйелі шолу нәтижелері бойынша тіс-жақ аномалияларының әлемдік таралуы 56 %-ды құрайды, ал мұндай аномалиялар ауыз қуысы денсаулығына байланысты өмір сапасының төмендеуімен байланысты болды (RR/PR = 1.15; 95 % CI: 1.12–1.18; 3672 қатысушы). Балалар арасында жүргізілген зерттеулерде 8-10 жастағы балалардың 49,1 %-ында тіс-жақ аномалиялары анықталған, ал ауыз арқылы тыныс алу трансверсальды тістем аномалияларымен статистикалық тұрғыдан маңызды байланыста болған (PR = 6,15; 95 % СИ: 2,96–12,80).

Мақсаты. Жұтқыншақ бадамша безі гипертрофиясының дамуындағы миофункционалды бұзылулар мен тіс-жақ аномалияларының рөлі туралы әдеби дереккөздерді талдау.

Материалдар және әдістер: Google Scholar, Scopus, Web of Science, PubMed және eLIBRARY ғылыми дерекқорларындағы 2019-2024 жылдар аралығындағы жарияланымдарға аналитикалық шолу жүргізілді. Аталған мерзімнен тыс негізгі классикалық дереккөздер де енгізілді. Алдын ала белгіленген іріктеу критерийлеріне сәйкес келген 92 жарияланым қорытынды шолуға енгізілді.

Нәтижелері және талқылау. Талданған деректер аденоидты гипертрофия, мұрынмен тыныс алудың бұзылуы, ауыз арқылы тыныс алу, миофункционалды бұзылулар және тіс-жақ аномалиялары өзара тығыз байланысты екенін көрсетеді. Жұтқыншақ бадамша безінің гипертрофиясы 6 жастағы балаларда жиі кездесетін ЛОР ауруларының бірі болып табылады; жиі ауыратын балаларда ол 70-90 % жағдайда анықталады. Аденоидты гипертрофиямен байланысты ауыз арқылы немесе аралас тыныс алу артикуляциялық бұзылыстармен қатар жүруі мүмкін: тыныс алу бұзылыстары бар балалардың 81.7 %-ында сөйлеу кемшіліктері сипатталған. Қайталама операция салыстырмалы түрде сирек кездескенімен, ірі ретроспективті зерттеуде ревизиялық аденоидэктомия жиілігі 0.55 % құрады, ал ревизиялық жағдайлардың 21 %-ы аденоид тінінің шынайы қайта өсуінен емес, түтікше бадамша безінің гиперплазиясымен байланысты болды.

Қорытындылар. Әдеби деректерді талдау аденотомиядан кейінгі рецидивтердің алдын алу клиникалық тұрғыдан маңызды екенін және операциядан кейінгі кезеңді жүргізуде пәнаралық кешенді тәсілді қажет ететінін көрсетеді. Мұндай тәсіл оториноларингологиялық бақылауды, ерте ортодонтиялық бағалауды, тұрақты ауыз арқылы тыныс алуды және тіс-жақ аномалияларын түзетуді, сондай-ақ көрсетілімдер болған жағдайда миофункционалды немесе логопедиялық терапияны қамтуы тиіс.

Түйін сөздер: *миофункционалды бұзылулар, тіс-жақ аномалиялары, аденоидты гипертрофия, рецидив, қауіп факторлары, ортодонтиялық емдеу, ауыз арқылы тыныс алу, пәнаралық жүргізу.*

МИОФУНКЦИОНАЛЬНЫЕ НАРУШЕНИЯ И ЗУБОЧЕЛЮСТНЫЕ АНОМАЛИИ КАК ФАКТОР РИСКА ВОЗНИКНОВЕНИЯ ГИПЕРТРОФИИ АДЕНОИДОВ (ОБЗОР ЛИТЕРАТУРЫ)

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Аннотация

Актуальность: В данном обзорном исследовании рассматривается роль миофункциональных нарушений и зубочелюстных аномалий в развитии гипертрофии глоточной миндалины, включая ее рецидив после аденотомии. Актуальность проблемы подтверждается количественными данными: согласно систематическому обзору, распространенность зубочелюстных аномалий в мире составляет 56 %, при этом их наличие связано со снижением качества жизни, обусловленного состоянием полости рта (RR/PR = 1,15; 95 % ДИ: 1,12–1,18; 3672 участника). В исследованиях с участием детей зубочелюстные аномалии выявлены у 49,1 % детей в возрасте 8–10 лет, а ротовое дыхание статистически значимо ассоциировано с трансверсальными аномалиями прикуса (PR = 6,15; 95 % ДИ: 2,96–12,80).

Цель: анализ литературных источников о роли миофункциональных нарушений и зубочелюстных аномалий в развитии гипертрофии глоточной миндалины.

Материалы и методы: Проведен аналитический обзор публикаций, представленных в научных базах данных Google Scholar, Scopus, Web of Science, PubMed и eLIBRARY за 2019-2024 гг. Также были включены ключевые классические источники, опубликованные вне указанного периода. В итоговый обзор вошли 92 публикаций, соответствовавших предварительно заданным критериям отбора.

Результаты и обсуждение: Проанализированные данные свидетельствуют о тесной взаимосвязи гипертрофии аденоидов, нарушенного носового дыхания, ротового дыхания, миофункциональных нарушений и зубочелюстных аномалий. Гипертрофия глоточной миндалины является распространенным ЛОР-заболеванием у детей 6 лет; у часто болеющих детей она описывается в 70-90 % случаев. Ротовое или смешанное дыхание при гипертрофии аденоидов может сопровождаться артикуляционными нарушениями: дефекты речи были зарегистрированы у 81.7 % детей с нарушениями дыхания. Несмотря на то что повторные хирургические вмешательства встречаются относительно редко, в крупном ретроспективном исследовании частота ревизионной аденоидэктомии составила 0.55 %, при этом 21 % ревизионных случаев были связаны с гиперплазией трубной миндалины, а не с истинным повторным разрастанием аденоидной ткани.

Выводы: Проведенный анализ литературных данных дает основания заключить, что профилактика рецидивов после аденотомии имеет клиническое значение и должна включать междисциплинарный комплексный подход к ведению послеоперационного периода. Такой подход должен объединять оториноларингологическое наблюдение, раннюю ортодонтическую оценку, коррекцию сохраняющегося ротового дыхания и зубочелюстных аномалий, а также миофункциональную или логопедическую терапию при наличии показаний.

Ключевые слова: *миофункциональные нарушения, зубочелюстные аномалии, гипертрофия аденоидов, рецидив, факторы риска, ортодонтическое лечение, ротовое дыхание, междисциплинарное ведение.*

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