American Journal of Medicine and Medical Sciences 2025, 15(5): 1338-1343
DOI: 10.5923/j.ajmms.20251505.05

Features of Choice of Tactics for Treatment of Inflammatory Diseases of the Pharyngeal Tonsil

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Abstract The article presents a literature review, considers issues of modern approaches to diagnostics of chronic adenoiditis and approaches to treatment taking into account the etiology and pathophysiology of the disease. The article formulates theoretical prerequisites for changing the paradigm of treatment of chronic adenoiditis. Thus, chronic adenoiditis in children is a common pathology and requires careful diagnostics and individual choice of treatment tactics.

Keywords Chronic adenoiditis, Children, Diagnostics, Treatment

1. Introduction

Inflammatory diseases of the pharyngeal tonsil, particularly chronic adenoiditis (CA), represent a significant clinical concern in pediatric otolaryngology due to their high prevalence, recurrent nature, and potential to cause systemic complications. Chronic inflammation of the lymphoid tissue in the nasopharynx not only disrupts normal respiratory function but also predisposes children to persistent middle ear infections, hearing impairment, sleep disturbances, and alterations in craniofacial development. Despite advancements in diagnostic tools and therapeutic strategies, the management of chronic adenoiditis remains challenging, largely due to its multifactorial etiology, clinical heterogeneity, and overlapping symptomatology with other upper respiratory tract conditions such as chronic rhinosinusitis.

The differentiation between adenoiditis and adenoid hypertrophy, although often blurred in clinical practice, is essential for selecting an appropriate treatment modality. Moreover, the emergence of new concepts such as endotyping and phenotyping of chronic adenoiditis has provided a deeper understanding of the disease's underlying mechanisms, allowing for a more individualized, targeted approach to therapy. Identifying whether a case of CA is predominantly driven by infectious or allergic pathways significantly influences the choice of conservative versus surgical treatment and the selection of pharmacological agents.

This study aims to synthesize recent advances in the classification, diagnosis, and management of inflammatory diseases of the pharyngeal tonsil. Special attention is given to the role of mucolytics, corticosteroids, and biofilm-targeted therapies, as well as to the potential of non-invasive diagnostic markers in tailoring patient-specific treatment strategies. By aligning clinical tactics with pathophysiological understanding, this work contributes to the development of evidence-based, personalized care protocols for children suffering from chronic adenoiditis.

Received: Mar. 24, 2025; Accepted: Apr. 20, 2025; Published: May 8, 2025
Published online at http://journal.sapub.org/ajmms

2. Literature Review

Inflammatory pathology of the pharyngeal tonsil, particularly chronic adenoiditis (CA), has been widely studied due to its prevalence in pediatric populations and its multifactorial clinical implications. According to G.V. Volkov et al. (2020), chronic adenoiditis is not merely a consequence of hypertrophy but a distinct pathological entity characterized by persistent lymphoid inflammation, frequent infections, and mucosal hypersecretion. Differentiating CA from adenoid hypertrophy is of paramount importance, as emphasized by Belyaev et al. (2019), since therapeutic approaches diverge significantly between inflammatory and purely obstructive forms.

The role of microbial biofilms in sustaining chronic inflammation in adenoid tissues has been a focus of recent studies. Brook (2018) identified the presence of polymicrobial communities, including anaerobes, within adenoidal biofilms, which contribute to resistance to standard antibiotic therapy. Similarly, Klemens et al. (2019) demonstrated that biofilm-forming pathogens are often implicated in recurrent adenoiditis, necessitating the integration of anti-biofilm strategies in treatment.

In terms of diagnosis, endoscopic examination and radiologic assessment remain standard, but recent studies have called for the inclusion of immunohistochemical and microbiological markers to identify the underlying pathophysiological mechanism of CA (Lukashenko et al., 2021). Furthermore, the classification of adenoiditis into infectious and allergic phenotypes, as proposed by S. A. Naumov (2020), supports

the development of personalized treatment plans and helps predict therapeutic outcomes.

From a therapeutic perspective, mucolytics and topical corticosteroids have been widely endorsed in conservative management, particularly in patients exhibiting non-obstructive, inflammatory features. According to a randomized trial by Ivanov and Pustovalov (2021), a combination of anti-inflammatory and anti-edematous agents significantly improved symptom control and reduced the need for surgical intervention. On the other hand, adenoidectomy remains the treatment of choice in cases of recurrent or persistent CA unresponsive to pharmacologic therapy, especially when complicated by otitis media with effusion or obstructive sleep apnea (OSAS).

In recent years, there has been a shift toward more nuanced treatment algorithms incorporating adenoidal phenotyping, minimally invasive interventions, and biomarker-guided therapy. These developments align with global trends in evidence-based and patient-specific otolaryngological care.

3. Results

Chronic inflammation of the lymphoid structures in the nasopharynx is a frequently encountered issue in both outpatient and inpatient medical practice. It has a tendency to recur and often leads to complications affecting the middle ear. This condition is particularly prevalent in pediatric patients due to several factors, including the distinct composition of the nasopharyngeal microbiome, the virulence and invasiveness of pathogens, as well as the anatomical and physiological characteristics of the region. The proximity of the pharyngeal tonsil (PT) to the openings of the auditory tubes plays a crucial role in ensuring proper aeration of the tympanic cavity, maintaining tubotympanic pressure, and supporting the body's immunobiological defense mechanisms.

Despite the wide array of therapeutic approaches available for managing chronic inflammation of the adenoids, their effectiveness remains suboptimal. The issue of chronic adenoiditis holds significant medical and social relevance and has been a long-standing focus of attention among otolaryngologists [1,2,9,12,16,17]. Epidemiological data indicate a steady rise in incidence rates [2,3,6,11,15]. However, discrepancies persist in the literature regarding both the classification of adenoiditis and the optimal management strategies.

In international medical literature, the term «adenoiditis» is often associated with hypertrophy of the pharyngeal tonsil (HPT), a condition widely recognized across the globe. HPT significantly impacts patients quality of life, has a high comorbidity rate, and imposes substantial economic burdens [1,2,4]. In contrast, domestic literature differentiates between HPT and the term «adenoids», which refers specifically to inflammation of the pharyngeal tonsil (III) within the Pirogov-Valdeyer lymphopharyngeal ring [5].

A major point of contention among clinicians and researchers lies in the distinction between «adenoiditis» and «adenoid hypertrophy». While many practicing physicians use these terms interchangeably, scientific discourse views them as separate entities. Adenoiditis is characterized by chronic inflammation of the pharyngeal tonsil, whereas adenoid hypertrophy is considered a histological feature indicating an increase in lymphoid tissue, which may also be a physiological response [6].

Adenoiditis is further classified into acute and chronic forms, with chronic adenoiditis (CA) being a multifactorial condition. The primary clinical manifestations of CA include postnasal drip, persistent nasal congestion, and a prolonged duration of symptoms lasting over 12 weeks [7,8]. This condition is linked to immune system dysfunction or immaturity in the pharyngeal tonsil [9]. According to domestic studies, the prevalence of CA among children varies widely, affecting between 3% and 50% of the general pediatric population and up to 70% of children with recurrent illnesses [2].

The impact of CA and HPT on pediatric health is multifaceted, leading to disruptions in normal respiratory function, characteristic facial alterations, and a heightened risk of recurrent and chronic otitis media, hearing impairments, and systemic complications affecting vital organs such as the heart and kidneys. Studies have established a strong correlation between CA, HPT, and bronchopulmonary diseases in children [5,6].

Nasal breathing difficulties associated with CA and HPT contribute to mouth breathing during sleep, which in turn reduces the humidity and filtration of inhaled air. This promotes the accumulation of microbes and particulate matter on the mucosal surfaces of the larynx and trachea, increasing susceptibility to infections such as tonsillitis, pharyngitis, bronchitis, and pneumonia [2,3,10,11].

Chronic adenoiditis (CA) is a diagnosis that is most often established in the practice of an otolaryngologist and according to ICD-10 belongs to the general code J35 – «Chronic diseases of the tonsils and adenoids» [2,4]. According to ICD-10, acute and chronic adenoiditis are not distinguished as a separate nosological form. Acute adenoiditis is usually coded according to ICD-10 according to the code of acute nasopharyngitis (J02), and chronic adenoiditis - according to the code of other chronic diseases of the tonsils and adenoids (J35.8). Interest in this problem is great, which is confirmed by the tendency of recent years to write uniform consensus documents, both federal and international [7,8]. However, there is still no consensus on the duration of the inflammatory process in CA, in which these changes can be considered chronic, and there are no uniform approaches to treatment. Many authors consider inflammation of the pharyngeal tonsil in childhood as a physiological process aimed at the development of immunity [9]. At the same time, the international EPOS CA guidelines highlight cases of persistence of clinical symptoms of the disease for more than 12 weeks [7,8]. The clinical course of CA is diverse, determined by a set of clinical symptoms, duration of the course, different responses to treatment measures and concomitant diseases. Most chronic inflammatory diseases of the respiratory tract are characterized by heterogeneity of both etiological factors and pathogenesis [12]. Thus, according to most authors, in cases where

inflammation underlies the disease, it is necessary to use pathogenetic therapy, i.e. anti-inflammatory, bypassing symptomatic therapy, which will ultimately reduce the percentage of systemic complications and ensure control over the disease [13]. It is important that with the same etiological factor, the inflammatory process develops differently in different people. This is due to different endotypes, which are based on different types of inflammation: «allergic» and «infectious». Currently, the concept of «endotype» is defined as a multifactorial system of properties, including pathomorphological (inflammation and remodeling), physiological, and genetic aspects of the disease [13]. The endotype in a mono- or combined variant characterizes the pathogenetic mechanisms underlying the formation of a certain clinical phenotype [14]. The dominant endotype (subtype) of inflammation can exist in a monovariant or syncretically interact with another subtype [13]. The dominant subtype of inflammation has specific biological markers.

Pediatric chronic rhinosinusitis (CRS) is a prevalent disease with a significant impact on quality of life (QoL) [1,2]. While its diagnostic criteria are similar to adult chronic rhinosinusitis (CRS), symptoms of pediatric CRS are often overlapped with chronic adenoiditis (CA), that is at least 12 weeks of two or more symptoms of purulent rhinorrhea, nasal obstruction, facial pressure or pain, or cough [1,2]. The presence of either endoscopic or CT imaging evidence of disease confirms the presence of CRS [1,2]. There is no universally accepted diagnostic criteria for CA, and symptoms can be similar to CRS with the addition of post nasal drip, globus sensation, nasal obstruction, and headache [3].

Patient reported outcome measures (PROMs) such as the Sinus and Nasal Quality of Life Survey (SN-5) and the 22-item Sinonasal Outcome Test (SNOT-22) help to further explore the impact of disease on children suffering from CRS, considering factors that may be unaddressed based on objective findings alone [4,5,6]. These measures are often used in outcome research to measure the effect of the treatment on QoL of patients. Unlike the adult population, PROMs have been used in limited number in the pediatric CRS population. SN5 is the most widely used instrument in children of all ages [5]. SNOT-22 scores have been used randomly in certain pediatric subgroups, and mostly in the adolescent patients with CRS [6]. Another aspect specific to the pediatric population is the role of parents and/or caregivers in reporting on the child's disease-specific QoL.

Multiple studies in adult CRS patients showed discrepancy between the subjective and objective measures [7,8]. More specifically, SNOT-22 scores did not corelate with the modified Lund-Kennedy endoscopy (MLK) scores or with the CT Lund-Mackay (LM) scores [9,10]. Studies exploring the correlation between PROMs and objective findings in the pediatric patients are very limited. One study showed that SN5 scores correlate well with the CT LM scores using a cohort of 32 patients [11]. Another study in 2014 showed that CT LM scores correlate well with MLK scores in a cohort of 21 patients [12]. It is imperative to understand how both subjective and objective findings correlate in children with CRS, especially considering the direct role of parents and/or caregivers in assessing the child's QoL [12].

When identifying individual phenotypes, various authors describe the clinical and morphological characteristics of the disease, key triggering factors, the primary pathogenic mechanisms, and unique responses to treatment [12].

Endotypes are distinguished by specific immune processes occurring in a given patient, whereas inflammatory phenotypes develop under environmental influences [14]. Significant progress has been made in recent years in treating and managing bronchial asthma (BA) and rhinitis through the identification of various biological and clinical markers [13]. Similarly, patients with chronic adenoiditis (CA) form a heterogeneous group, characterized by a chronic and progressive course that significantly affects a child's quality of life and presents a high likelihood of comorbid conditions [5]. This underscores the importance of optimizing therapeutic strategies and understanding factors influencing treatment efficacy. Despite the development of numerous pathogenetic approaches for treating children with CA and hypertrophy of the pharyngeal tonsil (HPT), the prevalence of the disease continues to rise. Patients diagnosed with CA may exhibit varying responses to the same treatment regimen. Resistance to therapy may arise due to the coexistence of allergic rhinitis, high bacterial contamination, or the development of CA against a background of gastroesophageal reflux disease (GERD) [9]. Identifying distinct pathophysiological mechanisms through the classification of CA into endotypes and phenotypes could enhance treatment efficacy and improve nasal breathing outcomes.

Of particular importance in defining CA phenotypes is the «response to traditional therapy», which can facilitate the development of a stepwise, personalized treatment approach.

Endotypes such as «allergic» and «infectious» exhibit diverse clinical manifestations, influenced by symptom severity, duration, microbiological landscape, and comorbid conditions such as recurrent purulent otitis media, exudative otitis media, hearing loss, sleep apnea, purulent rhinosinusitis, and GERD. These clinical markers may serve as a foundation for identifying CA phenotypes. Literature suggests several classifications of phenotypes based on clinical presentation, pathophysiology, functional characteristics, response to therapy, and prognostic factors [13]. The heterogeneity in CA manifestations and treatment responses may be influenced by anatomical variations in the nasal cavity, nasopharynx, and facial bones; comorbid conditions such as GERD, sinusitis, recurrent otitis media, and allergic rhinitis; inadequate treatment adherence; and endogenous factors such as genetic predisposition and individual inflammatory response rates [15].

During the initial clinical assessment, which includes patient history, symptom evaluation, and endoscopic examination of the upper respiratory tract, the dominant endotype (infectious or allergic) and CA phenotype can be determined.

Diagnostic markers of the «infectious» endotype include persistent nasal congestion and prolonged rhinorrhea, particularly worsening at night. Endoscopic findings reveal hyperemia of lymphoid tissue with abundant mucopurulent

secretions in the nasal cavity or along the posterior pharyngeal wall. High bacterial contamination in nasal swabs serves as a biomarker of the infectious endotype [15].

Research indicates that irrigation therapy significantly alleviates inflammation in 77-90% of CA patients and reduces HPT from grade III to grade II in 50% of cases [16]. However, due to the immaturity of the mucociliary apparatus in children, local or systemic medications that enhance mucociliary clearance are often incorporated into initial therapy [13]. A compelling rationale for combining mucolytic drugs with irrigation therapy in the initial treatment of the infectious endotype is the role of biofilms. Biofilms are polymicrobial communities comprising bacteria, fungi, and viruses encased in a thick mucous matrix composed of sugars and proteins, adhering to various surfaces. According to leading American and European researchers, biofilms play a pivotal role in sustaining chronic inflammation in the upper respiratory tract, including the nasopharynx [17]. Clinical studies have demonstrated that mucoregulatory therapy effectively disrupts biofilms on mucosal surfaces [14].

N-acetylcysteine, a direct mucolytic agent, inhibits biofilm biomass formation and disaggregates microbial communities. Following biofilm disruption, treatment should target all components of the biopolymer matrix. Microelements in «sea water» used during irrigation prevent microbial adhesion, while the free sulfhydryl groups in mucolytics disrupt intra- and intermolecular disulfide bonds within mucopolysaccharides of viscous secretions, thereby degrading biofilms. Furthermore, mucolytics stimulate goblet cells to produce less viscous sialomucins, reducing bacterial adhesion to epithelial cells and inhibiting biofilm formation.

The hypothesis regarding the biofilm-disruptive effect of mucolytics, particularly N-acetylcysteine, has been supported by studies demonstrating a significant reduction in biofilm thickness, a decrease in viable gram-positive and gram-negative microorganisms, and a fungicidal effect against Candida species [15].

In the absence of positive dynamics, topical corticosteroids (TCS) are used to relieve the inflammatory process, since clinical experience has been accumulated in the use of mometasone furoate in the treatment of «non-allergic» inflammation of the pharyngeal tonsil in children [10].

Thus, the first step in the therapy of chronic adenoiditis (infectious endotype) can start with a combination of irrigation therapy in combination with mucolytic drugs [17]. After a follow-up examination in 3 days, the following recommendations are given: in case of significant positive dynamics, continue treatment for 4-5 days; in cases of lack of control over nasal breathing, it is recommended to intensify treatment with TCS for 14 days.

In cases of the «infectious adenoiditis with purulent rhinosinusitis» phenotype, the diagnostic markers of which will be: complaints of nasal congestion and discharge from the nose (outward or into the nasopharynx) [7,8], a history of acute respiratory viral infections and a purulent streak in the middle nasal passage during endoscopic examination, and biomarkers - high bacterial contamination in nasal smears and lack of response to initial therapy, treatment at the first stage should be intensified with topical antibiotics and systemic mucoactive drugs. In case of no response to therapy within 48 hours, it is necessary to change tactics by prescribing systemic antibiotics and irrigation therapy using ultrasound technologies.

In case of the phenotype «infectious adenoiditis with exudative otitis», the markers of which will be: complaints of hearing loss and tympanogram, type B, the initial therapy should be intensified by using decongestants against the background of the use of systemic mucolytics. If there is no effect from therapy within 48 hours, the treatment should be intensified by prescribing a systemic antibiotic according to the scheme proposed in clinical guidelines [12,17,19]. If there is no positive dynamics within 72 hours, it is necessary to determine the phenotype: «complicated infectious adenoiditis» or «HPT with exudative otitis» (recurrent purulent otitis media, hearing loss) without the effect of treatment, and adenotomy is recommended, since the pathology of the middle ear and hearing is an absolute indication for surgical treatment of the pathology of the pharyngeal tonsil.

The dominant phenotype in preschool age is CA against the background of GER; the endotype is infectious. Initial therapy is represented by irrigation therapy in combination with the use of local antibacterial drugs [9]. All children diagnosed with GER must follow a diet. To relieve GER, initial therapy should be enhanced with domperidone for 1 month. If it is necessary to enhance treatment, from the 2nd stage, irrigation therapy is carried out using ultrasound technologies. The use of low-frequency ultrasound causes a pronounced bactericidal effect, which is achieved due to the cavitation effect, damaging the cell membrane of microorganisms: swelling and subsequent destruction under the oxidative action of oxygen, which is activated by ultrasound [11,18]. Another important effect that low-frequency ultrasound has on the surface of the mucous membranes of the upper respiratory tract is the destruction of biofilm. Thus, low-frequency ultrasound and the use of local mucoactive drugs will significantly complement the sanitizing effect of irrigation therapy and add an anti-inflammatory effect, acting on all links in the formation of a biofilm. Endoscopic markers of the allergic endotype will be: pale pink color of the lymphoid tissue, pronounced edema, smoothed grooves, shiny mucous membrane, often with abundant mucous secretion. Biomarkers of the allergic endotype CA and HPT are well known: positive skin tests (scarification or prick tests) with standard allergens, high levels of total serum and specific IgE, a positive nasal provocation test (NPT) with a suspected causative allergen. Phenotyping of such pathology of the pharyngeal tonsil will depend on the form of allergic rhinitis: intermittent or persistent (seasonal or year-round); concomitant pathology (rhinosinusitis, exudative otitis media, GERD); the effectiveness of the therapy: (with the effect of basic therapy, requiring increased therapy, without effect). The tactics of treating adenoiditis or HPT with allergic rhinitis are defined by WHO in the ARIA consensus document. Elimination therapy is used to reduce the time of

contact with the allergen. To reduce the degree of hypertrophy of the pharyngeal tonsil in children, intranasal glucocorticosteroids are prescribed for at least 1 month. During an exacerbation, second-generation antihistamines and/or leukotriene receptor antagonists are prescribed [6,17]. In the case of persistent allergic rhinitis, antihistamines can be used continuously.

4. Discussion

In the case of the phenotype «adenoiditis with allergic rhinitis and purulent rhinosinusitis», characterized by endoscopic markers such as a purulent streak in the middle nasal passage and biomarkers including high bacterial contamination in nasal smears and lack of response to initial therapy, treatment should be intensified at the first stage with topical antibiotics and systemic mucoactive drugs [3]. If no response is observed within 48 hours, treatment should be adjusted by prescribing systemic antibiotics and ultrasound irrigation therapy [10,19].

For the phenotype «adenoiditis with allergic rhinitis and exudative otitis», identified by complaints of hearing loss and a type B tympanogram, initial therapy should be intensified with decongestants and systemic mucolytics. If there is no response to initial and stepwise therapy, the phenotype of «complicated infectious/allergic adenoiditis or HPT» is determined, warranting consideration of adenotomy.

Indications for adenotomy in children include severe nasal obstruction unresponsive to conservative treatment, middle ear pathology with hearing impairment despite stepwise conservative therapy, and sleep apnea syndrome. The presence of pharyngeal tonsil hypertrophy and signs of atopy in a child is not a contraindication for adenotomy. When performed under general anesthesia with proper preoperative preparation and postoperative management, adenotomy in children with allergic conditions does not exacerbate or worsen allergic diseases.

The primary goal of managing chronic adenoiditis and/or hypertrophy of the pharyngeal tonsil is to maintain nasal breathing, particularly during sleep. Effective disease control requires that therapy be tailored to the individual pathogenetic characteristics of the disease and the patient's response to treatment. Understanding the endotypes and phenotypes of CA and HPT enables a personalized and flexible treatment plan, incorporating individual genetic and clinical factors. This approach also facilitates the introduction of targeted therapies that block key pathogenic pathways dominant in a specific patient [13].

For diagnosing different CA and HPT phenotypes, common approaches include collecting anamnesis, identifying hereditary factors, evaluating living conditions and comorbidities, conducting a physical examination, and measuring allergen-specific IgE levels (a key marker of allergic and infectious endotypes). Additional diagnostic methods may include cytological analysis of nasal smears and washings, various microbiological techniques (cultural, biochemical, and molecular), endoscopic nasal examination, active anterior rhinomanometry, acoustic rhinometry, olfactory threshold assessment, mucociliary transport analysis (selectively), radiography, and computed tomography of the nasal cavity and paranasal sinuses (for complicated cases). Future diagnostic methods in pediatric practice should be simple, non-invasive, repeatable, and free from contraindications and limitations.

5. Conclusions

Chronic adenoiditis (CA) and pharyngeal tonsil hypertrophy (HPT) in children represent complex, heterogeneous conditions that require differentiated diagnostic and therapeutic approaches. The clinical variability, influenced by infectious and allergic endotypes and diverse phenotypes, underscores the necessity for individualized treatment strategies. This review highlights the paradigm shift toward personalized medicine, emphasizing the importance of phenotype-endotype classification in determining effective therapeutic tactics.

The application of irrigation therapy, mucolytics, topical corticosteroids, and, when appropriate, systemic antibiotics and surgical intervention must be guided by specific clinical markers and patient responsiveness. Furthermore, the integration of novel diagnostic tools—ranging from microbiological and immunological biomarkers to non-invasive imaging techniques—can enhance the precision of diagnosis and the efficacy of interventions.

Future directions in the management of chronic adenoiditis should focus on refining endotype-based treatment algorithms, identifying novel biomarkers, and incorporating patient-reported outcomes to optimize therapeutic success. Establishing a unified diagnostic and treatment consensus based on phenotypic variability will contribute to reducing systemic complications, preserving nasal respiration, and improving the overall quality of life in pediatric patients.

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