

**IMMUNOLOGICAL AND ENVIRONMENTAL DETERMINANTS
OF ADENOID VEGETATIONS AND TONSILLAR
HYPERTROPHY IN CHILDREN**

Abdusamatova Iroda Ilkhamovna

Senior lecturer at Tashkent State Medical University

Pardayev Chori Ikhtiyor ugli

Shaydoyeva Dildora Mizrobovna

Ismoilov Dilmurod Gulom ugli

Hamroyeva Zarina Muhiddin qizi

Students of Tashkent State Medical University

Abstract. Adenoid vegetations and tonsillar hypertrophy are among the most common pediatric ENT conditions, significantly affecting respiratory function, immunity, and quality of life. According to international data, approximately 25–45% of children experience clinically significant adenoid hypertrophy, often associated with recurrent infections and allergic conditions. This study aims to evaluate immunological and environmental determinants contributing to adenotonsillar enlargement and dysfunction. A total of 130 children (100 patients and 30 controls) were examined using clinical, laboratory, and instrumental methods. The findings indicate that immune imbalance, allergen exposure, and recurrent infections are key contributors to disease development. The study emphasizes the importance of комплекс preventive strategies and early intervention.

Keywords. Adenoid vegetations, tonsillar hypertrophy, immune response, environmental factors, pediatric ENT, inflammation

Introduction

Adenoid vegetations and palatine tonsils are integral components of the upper airway immune system, serving as the first line of defense against inhaled pathogens. However, pathological enlargement of these tissues is a common condition in pediatric populations, with prevalence rates ranging from 30% to 50% in children aged 2–12 years [14, 36, 72].

Studies suggest that repeated exposure to respiratory pathogens leads to chronic immune stimulation, resulting in lymphoid tissue hyperplasia [22, 65, 101]. Moreover, allergic sensitization and environmental pollutants such as particulate matter and tobacco smoke have been shown to exacerbate inflammatory responses in the adenotonsillar tissue [11, 49, 88].

Recent research highlights the role of cytokine imbalance, particularly elevated levels of IL-4, IL-6, and TNF- α , in promoting tissue hypertrophy and chronic

inflammation [7, 53, 109]. In addition, alterations in local microbiota and biofilm formation contribute to persistent инфекция and resistance to treatment [19, 60, 94].

Despite these findings, there remains a need for comprehensive evaluation of combined immunological and environmental influences on adenotonsillar pathology, which underlines the importance of this study.

Aim of the Study

To assess the immunological and environmental determinants of adenoid vegetations and tonsillar hypertrophy in children and to evaluate their clinical impact.

Materials and Methods

The study included 130 children aged 3–13 years, divided into a main group of 100 patients diagnosed with adenoid vegetations and tonsillar hypertrophy, and a control group of 30 healthy children.

Clinical assessment involved ENT examination using rhinoscopy, pharyngoscopy, and nasopharyngeal endoscopy to determine the degree of hypertrophy. Instrumental methods included lateral radiography and pulse oximetry for evaluation of airway patency and oxygen saturation.

Laboratory investigations included complete blood count, immunological profiling using ELISA to measure cytokines (IL-4, IL-6, TNF-α), and microbiological analysis of nasopharyngeal flora.

Environmental exposure was evaluated through structured questionnaires focusing on allergen exposure, passive smoking, frequency of respiratory infections, and living conditions. Statistical analysis was performed using SPSS software with significance set at $p < 0.05$.

Results

The results demonstrated that children with adenotonsillar hypertrophy had significantly higher exposure to environmental risk factors and showed marked immunological alterations compared to the control group.

Table 1. Environmental Risk Factors

Factors	Main Group (n=100)	Control Group (n=30)
Passive smoking	70 (70%)	9 (30%)
Allergen exposure	65 (65%)	8 (26.7%)
Urban air pollution	60 (60%)	10 (33.3%)
Frequent infections (>6/year)	78 (78%)	7 (23.3%)

Environmental factors, particularly passive smoking and frequent infections, were significantly more prevalent in the patient group, indicating their strong contribution to adenotonsillar hypertrophy.

Table 2. Cytokine Profile

Cytokine	Main Group	Control Group
IL-4 (pg/ml)	10.8 ± 2.5	5.1 ± 1.3
IL-6 (pg/ml)	13.7 ± 3.0	6.5 ± 1.6
TNF-α (pg/ml)	16.2 ± 3.4	8.0 ± 2.2

Elevated cytokine levels in the main group confirm the presence of chronic inflammatory and allergic processes contributing to tissue hypertrophy.

Table 3. Functional and Clinical Indicators

Indicator	Main Group (%)	Control Group (%)
Sleep-disordered breathing	75 (75%)	3 (10%)
Chronic nasal obstruction	85 (85%)	4 (13.3%)
Recurrent otitis media	50 (50%)	2 (6.7%)
Reduced oxygen saturation	40 (40%)	1 (3.3%)

Functional impairments, particularly respiratory disturbances and decreased oxygen saturation, were significantly more common in affected children, reflecting the clinical severity of the condition.

Discussion

The findings of this study confirm that adenotonsillar hypertrophy is a multifactorial condition influenced by both immunological and environmental determinants. The observed increase in cytokine levels aligns with previous studies demonstrating chronic inflammatory activation [53, 109].

Environmental exposures such as allergens and pollution significantly exacerbate disease progression, indicating the need for environmental control measures. Additionally, recurrent infections play a central role in maintaining chronic inflammation.

From a socio-medical perspective, untreated adenotonsillar pathology can lead to complications such as sleep apnea, cognitive impairment, and decreased academic performance. Early intervention can reduce healthcare costs and improve long-term outcomes.

Conclusion

Adenoid vegetations and tonsillar hypertrophy are strongly associated with immune dysregulation and environmental exposures. Comprehensive диагностика and preventive strategies are essential to improve clinical outcomes and reduce disease

burden in pediatric populations.

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