RISK FACTORS AND PATHOPHYSIOLOGICAL CHANGES IN THE ADENOTONSILLAR SYSTEM IN CHILDREN

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Abstract. The adenotonsillar system plays a crucial role in the immune defense of the upper respiratory tract, especially in early childhood. However, pathological hypertrophy and chronic inflammation of adenoids and palatine tonsils remain among the most prevalent ENT disorders worldwide. According to global epidemiological data, up to 30–50% of children aged 3–10 years suffer from varying degrees of adenotonsillar pathology, often associated with recurrent infections and environmental exposures. This study aims to analyze risk factors and morphological-functional changes in the adenotonsillar system and assess their clinical implications. A comparative clinical study involving 120 children (90 patients and 30 controls) was conducted using комплекс clinical, laboratory, and instrumental methods. The findings demonstrate a significant association between environmental, immunological, and infectious factors with adenotonsillar hypertrophy and dysfunction. The results highlight the importance of early diagnosis and targeted preventive strategies.

Keywords. Adenotonsillar system, adenoid hypertrophy, tonsillitis, risk factors, children, immunity, ENT pathology

Introduction. Adenotonsillar tissue represents a key component of the mucosa-associated lymphoid tissue (MALT) system, contributing significantly to local immune responses against inhaled and ingested pathogens. Epidemiological studies indicate that adenotonsillar hypertrophy affects approximately 35–40% of preschool-aged children globally, often leading to airway obstruction, sleep-disordered breathing, and recurrent infections [12, 45, 78].

Recent investigations have shown that chronic inflammation of the adenoids and tonsils is closely associated with microbial colonization, including *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Staphylococcus aureus*, which disrupt normal immune responses [23, 67, 102]. Furthermore, environmental factors such as air pollution, passive smoking, and allergen exposure significantly increase the risk of adenotonsillar pathology [15, 39, 84].

According to several authors, immunological imbalance—particularly alterations in cytokine profiles (IL-6, TNF-α, IL-10)—plays a central role in the progression of chronic adenotonsillar inflammation [9, 51, 110]. Additionally, genetic predisposition and early-life infections have been identified as contributing factors influencing the severity and recurrence of the disease [18, 63, 95].

Despite extensive research, there remains a need for integrated evaluation of risk factors and their combined effect on adenotonsillar structural and functional changes, which justifies the relevance of this study.

Aim of the Study

To investigate the risk factors and pathophysiological changes in the adenotonsillar system in children and to evaluate their clinical and socio-medical significance.

Materials and Methods

This study was conducted as a prospective comparative clinical investigation involving 120 children aged 3 to 12 years. The main group consisted of 90 patients diagnosed with adenotonsillar pathology (including adenoid hypertrophy and chronic tonsillitis), while the control group included 30 clinically healthy children without ENT disorders.

Clinical examination included detailed анамнез collection, ENT examination using anterior rhinoscopy, oropharyngoscopy, and эндоскопия of the nasopharynx. Instrumental methods such as lateral nasopharyngeal radiography and pulse oximetry were used to assess airway obstruction and oxygen saturation levels.

Laboratory methods included complete blood count, immunological assays (measurement of cytokines such as IL-6, TNF-α, IL-10 using ELISA), and microbiological analysis of nasopharyngeal swabs.

Additionally, environmental and lifestyle factors were assessed using structured questionnaires, including exposure to passive smoking, frequency of respiratory infections, and socio-economic conditions. Statistical analysis was performed using descriptive and inferential methods, including Student’s t-test and chi-square analysis, with significance set at $p<0.05$.

Results

The study demonstrated that adenotonsillar pathology is significantly associated with multiple interacting risk factors and leads to pronounced functional and immunological changes. Children in the main group showed higher rates of recurrent respiratory infections, immune dysregulation, and environmental exposure compared to controls.

Table 1. Distribution of Major Risk Factors

Risk Factors	Main Group (n=90)	Control Group (n=30)
Passive smoking exposure	62 (68.9%)	8 (26.7%)
Frequent ARVI (>5/year)	70 (77.8%)	6 (20.0%)
Allergic diseases	48 (53.3%)	5 (16.7%)
Poor socio-economic status	40 (44.4%)	7 (23.3%)

The data indicate that environmental and infectious factors significantly contribute to adenotonsillar pathology. Passive smoking and recurrent viral infections were the most prominent risk factors, suggesting their critical role in chronic inflammation development.

Table 2. Immunological Parameters

Parameter	Main Group	Control Group
IL-6 (pg/ml)	12.5 ± 2.3	6.2 ± 1.4
TNF-α (pg/ml)	15.1 ± 3.1	7.8 ± 2.0
IL-10 (pg/ml)	4.3 ± 1.2	8.5 ± 2.1

A significant increase in pro-inflammatory cytokines (IL-6, TNF-α) and a decrease in anti-inflammatory IL-10 were observed in the main group, indicating an imbalance in immune regulation and chronic inflammatory activity.

Table 3. Clinical Manifestations

Symptoms	Main Group (%)	Control Group (%)
Nasal obstruction	80 (88.9%)	3 (10.0%)
Snoring	65 (72.2%)	2 (6.7%)
Mouth breathing	72 (80.0%)	4 (13.3%)
Recurrent tonsillitis	60 (66.7%)	1 (3.3%)

Clinical symptoms such as nasal obstruction, snoring, and mouth breathing were significantly more frequent in patients, reflecting functional impairment of the upper airway due to adenotonsillar hypertrophy.

Discussion

The results of this study confirm that adenotonsillar pathology is a multifactorial condition influenced by environmental, infectious, and immunological factors. The observed increase in pro-inflammatory cytokines supports the hypothesis of chronic immune activation, consistent with previous studies [51, 110].

Environmental exposures, particularly passive smoking, were strongly associated with disease prevalence, highlighting the importance of preventive public health strategies. Furthermore, recurrent infections contribute to a vicious cycle of inflammation and tissue hypertrophy.

From a socio-economic perspective, early diagnosis and management of adenotonsillar pathology can reduce healthcare costs associated with repeated

infections, hospital visits, and surgical interventions. Improved screening and preventive measures may enhance children's quality of life and reduce the burden on healthcare systems.

Conclusion

Adenotonsillar pathology in children is strongly associated with environmental, infectious, and immunological risk factors, leading to significant structural and functional changes. Early identification of these factors and implementation of preventive and therapeutic strategies are essential to reduce disease burden and improve clinical outcomes.

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