ADENOID AND TONSILLAR DYSFUNCTION AS A FACTOR IN IMPAIRMENT OF LOCAL AND SYSTEMIC IMMUNE DEFENSE IN CHILDREN

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Annotation. Adenoid and tonsillar dysfunction represents one of the leading causes of impaired mucosal and systemic immunity in childhood. This study investigates the immunological, microbiological, and structural alterations associated with chronic adenotonsillar pathology. A comparative analysis between hypertrophy cases and healthy controls was performed using immunological assays, cytokine profiling, and bacteriological evaluation. Children with adenotonsillar dysfunction demonstrated significant deficiencies in mucosal IgA production, altered T-cell distribution, and heightened inflammatory cytokine activity. The findings confirm that early detection and management of adenotonsillar disorders may significantly reduce recurrent respiratory illnesses and improve pediatric health outcomes.

Keywords: adenotonsillar dysfunction; mucosal immunity; pediatric infections; cytokines; lymphoid tissue; immune maturation.

Introduction. Upper respiratory lymphoid tissue disorders—particularly those involving the adenoids and palatine tonsils—represent a major pediatric health challenge worldwide. Globally, recurrent adenotonsillar infections affect nearly 25–30% of children, while adenotonsillar hypertrophy is observed in up to 50% of children aged 3–10 years [1]. Environmental pollution, indoor allergens, passive smoking, and infectious burden contribute to persistently high rates in developing regions.

The adenoids and palatine tonsils serve as frontline structures within Waldeyer's ring, orchestrating early immunological responses against inhaled and ingested antigens. Their lymphoepithelial design ensures rapid antigen sampling, activation of germinal centers, and differentiation of immunocompetent cells essential for mucosal protection [2]. Tonsillar crypts contain antigen-presenting cells, T-lymphocytes, B-cell zones, and plasma cells producing immunoglobulins—especially secretory IgA, which is crucial for mucosal defense [3].

Previous research by Brandtzaeg (1996) confirmed that the adenotonsillar complex is the largest source of secretory IgA during early childhood, contributing up to 40% of mucosal antibody production [4]. Children with impaired adenotonsillar function demonstrate weakened mucosal immunity, reduced IgA synthesis, and increased susceptibility to recurrent respiratory infections [5]. Structural or inflammatory changes in tonsillar tissues disrupt the balance between innate and

adaptive responses, leading to chronic inflammation and impaired immune maturation [6].

Cytokine dysregulation is another hallmark of adenotonsillar disease. Studies reveal that hypertrophied adenoids show elevated IL-1β, IL-6, TNF-α, and reduced regulatory cytokines such as IL-10, promoting a persistent inflammatory state [7]. These findings correlate with increased local bacterial colonization, especially by S. aureus, S. pneumoniae, and H. influenzae, forming biofilms that further impair mucosal immunity [8].

Beyond localized immune disruption, adenotonsillar dysfunction contributes to systemic complications, including sleep-disordered breathing, behavioral changes, and impaired growth [9]. Despite the clinical importance of these disorders, the immunological mechanisms linking adenotonsillar dysfunction with systemic immune imbalance remain insufficiently understood.

This study aims to deepen understanding of these mechanisms through a detailed immunological comparison of children with adenotonsillar dysfunction and healthy controls.

Objective

To evaluate the effects of adenoid and tonsillar dysfunction on mucosal and systemic immunity in children through clinical, immunological, and microbiological assessments.

Materials and methods

This cross-sectional comparative study included 130 children aged 4–13 years who were examined at a pediatric immunology and otolaryngology center. They were divided into:

Main group (n = 90): Children diagnosed with chronic adenotonsillar hypertrophy (grades II–III) and recurrent adenotonsillitis (≥5 episodes/year).

Control group (n = 40): Healthy children with no history of recurrent respiratory disease.

All participants underwent detailed otolaryngological evaluation, including nasopharyngoscopy, oropharyngeal inspection, and tonsillar grading. Immunological markers were assessed via flow cytometry (CD3+, CD4+, CD8+, CD19+), serum IgA, IgM, IgG, and salivary secretory IgA levels. Cytokine concentrations (IL-6, TNF-α, IL-10) were determined using ELISA.

Microbiological cultures from nasopharyngeal and tonsillar surfaces were performed to detect pathogenic colonization and biofilm-forming organisms. Standard pediatric immunology protocols and calibrated laboratory assays ensured methodological accuracy. Statistical analysis employed Student's t-test, with $p < 0.05$ considered significant.

Results

The study evaluated clinical symptoms, immune parameters, and microbiological profiles of participants. Children with adenotonsillar dysfunction showed significantly higher rates of nasal obstruction, sleep-disordered breathing, snoring, and recurrent infections compared with healthy children.

Table 1.

Cellular Immune Indicators in the Study Population

Indicator	Control Group (n=40)	Main Group (n=90)	p-value
CD3+ T-cells (%)	64.1 ± 3.9	52.8 ± 4.0	<0.01
CD4+ T-cells (%)	39.4 ± 3.2	29.9 ± 2.7	<0.01
CD8+ T-cells (%)	21.8 ± 1.9	27.5 ± 1.8	<0.05
CD4/CD8 ratio	1.81 ± 0.05	1.08 ± 0.04	<0.01

Children with adenotonsillar dysfunction displayed significantly reduced CD3+ and CD4+ percentages, indicating impaired cellular immunity. The elevated CD8+ level and reduced CD4/CD8 ratio reflect chronic inflammatory stress.

Table 2.

Humoral Immunity and Cytokine Activity

Parameter	Control Group	Main Group	p-value
Serum IgA (g/L)	1.41 ± 0.14	0.87 ± 0.13	<0.01
Secretory IgA (mg/L)	176 ± 20	101 ± 17	<0.01
IL-6 (pg/mL)	4.1 ± 0.7	12.6 ± 1.5	<0.01
TNF-α (pg/mL)	6.8 ± 0.9	17.4 ± 2.1	<0.01
IL-10 (pg/mL)	3.9 ± 0.5	2.0 ± 0.4	<0.05

Humoral immunity in the main group was significantly impaired, demonstrated by reduced serum and secretory IgA levels. Increased IL-6 and TNF-α indicate heightened inflammatory activity, while decreased IL-10 suggests insufficient immune regulation.

Discussion. The present study confirms that adenoid and tonsillar dysfunction significantly alters both mucosal and systemic immunity in children. Reduced CD4+ T-cell levels align with previous reports by Ohlms et al. showing weakened cellular immunity in tonsillar disorders [5]. The sharp decline in secretory IgA supports the findings of Brandtzaeg, who demonstrated the vital contribution of tonsillar tissue to mucosal antibody production [4].

Increased pro-inflammatory cytokines (IL-6, TNF-α) suggest persistent lymphoid

inflammation, consistent with earlier observations by Vargas et al. regarding cytokine imbalance in hypertrophied adenoids [7]. These inflammatory patterns contribute to chronic antigenic stimulation, tissue remodeling, and susceptibility to recurrent infection.

Microbiological results (not shown in tables) revealed higher colonization by S. aureus and H. influenzae in the main group—findings that support the role of bacterial biofilms in sustaining chronic tonsillar inflammation [8].

Socioeconomic and public health implications

Adenotonsillar dysfunction contributes to:

increased antibiotic use,

higher rates of school absenteeism,

long-term respiratory morbidity,

impaired sleep and cognitive development,

substantial healthcare costs for families and health systems.

By identifying immunological impairments early, clinicians may improve management strategies, reduce unnecessary antimicrobial exposure, and prevent complications such as obstructive sleep apnea.

Conclusion. Adenoid and tonsillar dysfunction in children leads to marked disturbances in both mucosal and systemic immunity, including reduced IgA synthesis, altered T-cell regulation, and increased inflammatory cytokine activity. These changes predispose children to recurrent infections, sleep-related disorders, and chronic inflammation. Early diagnosis and integrated immunological evaluation are essential to improving long-term outcomes and reducing the socioeconomic burden associated with pediatric adenotonsillar diseases.

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