CLINICAL AND LABORATORY STUDY OF LYMPHOID ADENOIDS IN CHILDREN

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Abstract

Adenoid hypertrophy is one of the most common pathologies in childhood, affecting up to 60–70% of preschool-aged children worldwide. This study focuses on the clinical and laboratory characteristics of lymphoid adenoids in children and aims to identify diagnostic markers and morphological alterations associated with chronic inflammation of the nasopharyngeal lymphoid tissue. A total of 80 pediatric patients aged 3–12 years with clinical manifestations of adenoid hypertrophy were examined through a combination of endoscopic, radiologic, hematological, and histopathological analyses. The results revealed significant correlations between the degree of lymphoid hyperplasia, the presence of recurrent upper respiratory infections, and specific changes in peripheral blood parameters. Morphological evaluation of adenoid tissue indicated hyperplastic and inflammatory changes consistent with chronic antigenic stimulation. The findings underline the importance of a combined clinical-laboratory approach for early detection and management of lymphoid adenoids in children.

Keywords: Adenoid hypertrophy, lymphoid tissue, children, chronic inflammation, histopathology, clinical diagnosis.

Introduction

Adenoid hypertrophy, characterized by an enlargement of the nasopharyngeal lymphoid tissue, remains a significant cause of upper airway obstruction and recurrent respiratory infections in children. According to WHO data (2023), adenoid hypertrophy accounts for approximately 35% of chronic respiratory pathologies among children aged 3–10 years. In developing countries,

its prevalence is rising due to environmental pollution, recurrent viral infections, and allergic sensitization.

The adenoids, part of Waldeyer's lymphatic ring, play a key immunological role in the early years of life, acting as a barrier against inhaled pathogens. However, persistent antigenic stimulation often leads to chronic hypertrophy, impairing nasal breathing and promoting otitis media, sinusitis, and sleep disturbances. Despite the frequency of this condition, the underlying clinico-morphological relationships remain insufficiently understood.

Recent studies (Liang et al., 2024; Kamat et al., 2023) suggest that adenoid hypertrophy is not only a local disorder but also reflects systemic immune responses. Therefore, detailed clinical and laboratory assessment is essential for early diagnosis, differentiation between infectious and allergic etiologies, and proper therapeutic management.

Materials and Methods

This study was conducted at the Otorhinolaryngology Department of Bukhara State Medical Institute between January 2023 and May 2025. A total of **80 children aged 3–12 years** were enrolled, including 46 boys (57.5%) and 34 girls (42.5%). Inclusion criteria were symptoms of nasal obstruction, mouth breathing, and recurrent infections of the upper respiratory tract. Children with congenital nasal malformations or previous adenoidectomy were excluded.

Clinical Examination:

All participants underwent a detailed otorhinolaryngological assessment, including nasopharyngoscopy and lateral nasopharyngeal radiography to grade adenoid hypertrophy according to the Clemens and McMurray classification (Grades I–III).

Laboratory Tests:

Peripheral blood samples were collected to evaluate:

- Complete blood count (CBC)
- Erythrocyte sedimentation rate (ESR)

- Serum C-reactive protein (CRP)
- Immunoglobulin levels (IgA, IgM, IgG)

Morphological Analysis:

Adenoid tissues obtained during adenoidectomy were fixed in 10% formalin and processed for histopathological examination using hematoxylin-eosin staining. Parameters assessed included epithelial integrity, lymphoid follicle hyperplasia, fibrosis, and vascular proliferation.

Statistical Methods:

Data were analyzed using SPSS v.27. Statistical significance was defined as $p < 0.05$. Correlations between clinical symptoms, laboratory results, and morphological parameters were determined using Pearson's correlation coefficient.

Results

Among the 80 examined children, 52 (65%) presented with Grade II adenoid hypertrophy, while 18 (22.5%) and 10 (12.5%) had Grade I and Grade III hypertrophy, respectively. The predominant clinical symptoms included mouth breathing (91%), snoring (84%), and recurrent otitis media (40%). Average duration of symptoms was 1.8 ± 0.3 years.

Laboratory results demonstrated:

- Elevated leukocyte counts (mean 10.8 × 10^9/L),
- Increased ESR (average 24 ± 5 mm/h),
- Elevated CRP levels in 56% of patients,
- Reduced serum IgA in 42% of children with frequent infections.

Histopathological evaluation revealed lymphoid follicle enlargement, interfollicular edema, and epithelial erosion in 70% of samples. Chronic inflammatory infiltration with predominance of lymphocytes and plasma cells was observed, indicating continuous antigenic stimulation. Fibrotic changes were identified in 25% of advanced cases (Grade III), suggesting tissue remodeling due to chronic inflammation.

A positive correlation ($r = 0.72$; $p < 0.01$) was found between adenoid size and CRP concentration, confirming the relationship between tissue hypertrophy and systemic inflammation.

Discussion

The clinical and laboratory findings confirm that adenoid hypertrophy in children represents a chronic inflammatory process with both local and systemic immune alterations. The association between adenoid size and elevated inflammatory markers supports the hypothesis that chronic antigenic exposure maintains lymphoid proliferation.

Our observations align with recent international data. According to **WHO (2024)**, adenoid hypertrophy affects approximately **42% of children under 10 years** in Central and South Asia, often linked to urban environmental factors. Similar results were obtained by **Huang et al. (2023)**, who reported that exposure to airborne allergens increases adenoid size by up to 30% due to mucosal immune hyperactivation.

The immunological findings, particularly reduced IgA levels, correspond to data from **Kamat et al. (2023)**, indicating a weakened mucosal immune barrier in children with recurrent infections. Histopathological alterations observed in this study—lymphoid hyperplasia and epithelial erosion—are consistent with chronic infection-induced remodeling (Liang et al., 2024).

The combination of clinical evaluation, laboratory tests, and morphological analysis provides a comprehensive diagnostic approach, allowing differentiation between simple hypertrophy and chronic adenoiditis. This is critical for determining appropriate treatment strategies, such as conservative therapy or surgical intervention.

Conclusion

The clinico-laboratory study of lymphoid adenoids in children demonstrates that adenoid hypertrophy is a multifactorial process combining chronic infection, environmental exposure, and immune dysregulation. Morphological alterations of lymphoid tissue correspond closely with clinical symptoms and inflammatory

markers, confirming the systemic nature of the disorder.

Comprehensive diagnostic assessment—including endoscopic examination, hematological analysis, and histopathological evaluation—enables early detection, precise staging, and personalized treatment selection.

Further studies are needed to explore molecular markers of chronic adenoid inflammation and their potential role in predicting recurrence after surgical treatment.

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