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HISTOPATHOLOGICAL REMODELING OF THE NASAL MUCOSA IN PEDIATRIC ALLERGIC RHINITIS: A MORPHOLOGICAL STUDY

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Abstract. Allergic rhinitis is a common chronic inflammatory disease in children characterized by IgE-mediated hypersensitivity reactions affecting the nasal mucosa. Despite extensive clinical research, morphological alterations underlying disease progression remain insufficiently characterized. This study aimed to investigate histopathological changes in the nasal mucosa of children with allergic rhinitis and evaluate their role in inflammatory pathogenesis. A total of 30 patients aged 6–14 years with allergic rhinitis and 15 healthy controls were examined. Nasal mucosal biopsies were analyzed using standard histological techniques with hematoxylin and eosin staining. Significant epithelial disruption, goblet cell hyperplasia, glandular hypertrophy, and dense eosinophilic infiltration were observed in the study group compared to controls ($p<0.05$). These findings confirm that structural remodeling of the nasal mucosa plays a central role in disease development and chronicity.

Keywords: Allergic rhinitis, children, nasal mucosa, histopathology, eosinophils, inflammation, epithelial remodeling.

Introduction. Allergic rhinitis (AR) is one of the most prevalent chronic inflammatory diseases in childhood, affecting quality of life and contributing to significant healthcare burden worldwide [1,2]. It is characterized by IgE-mediated immune responses triggered by environmental allergens, leading to inflammation of the nasal mucosa [3]. The global prevalence of AR continues to increase, particularly among pediatric populations, making it a significant public health concern [4].

The pathogenesis of allergic rhinitis involves complex immunological mechanisms, including activation of mast cells, eosinophils, and T-helper type 2 (Th2)

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lymphocytes [5]. These cellular interactions result in the release of inflammatory mediators such as histamine, leukotrienes, and cytokines, which contribute to mucosal edema and hypersecretion [6].

Morphological changes in the nasal mucosa play a crucial role in the chronicity and severity of the disease. Previous studies have demonstrated epithelial damage, goblet cell hyperplasia, and basement membrane thickening in allergic airway diseases [7]. However, the extent of structural remodeling in pediatric patients remains insufficiently explored.

Eosinophilic infiltration is considered a hallmark of allergic inflammation and correlates with disease severity [8]. Additionally, vascular changes such as increased permeability and vasodilation contribute to nasal obstruction and rhinorrhea [9]. Understanding these histopathological alterations is essential for improving diagnostic and therapeutic strategies.

Despite advances in clinical management, limited data exist regarding the morphological basis of allergic rhinitis in children, particularly in relation to mucosal remodeling and inflammatory cell dynamics [10]. Therefore, this study aims to investigate the histological features of nasal mucosa in pediatric allergic rhinitis and clarify their pathogenetic significance.

Materials and Methods. The study included 30 children aged 6 to 14 years diagnosed with allergic rhinitis (study group) and 15 age-matched conditionally healthy children (control group). Patient selection was based on clinical symptoms including nasal obstruction, rhinorrhea, sneezing, and nasal itching, as well as allergological testing.

Diagnosis was confirmed through elevated serum total IgE levels and positive skin prick tests to common aeroallergens. Exclusion criteria included acute respiratory infections, chronic systemic diseases, and prior nasal surgery.

Nasal mucosal biopsies were obtained under local anesthesia. Tissue samples were fixed in 10% neutral formalin, processed using standard histological protocols,

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and embedded in paraffin blocks. Sections of 5–7 μm thickness were prepared using a microtome and stained with hematoxylin and eosin.

Microscopic evaluation was performed under light microscopy at ×100 and ×400 magnifications. Morphological parameters assessed included epithelial integrity, degree of desquamation, goblet cell density, mucous gland hypertrophy, stromal inflammatory infiltration, and vascular alterations.

Eosinophils, lymphocytes, and mast cells were evaluated using a semi-quantitative scoring system (mild, moderate, severe infiltration). Mucosal edema was also assessed. Statistical analysis was performed using Student's t-test, and $p<0.05$ was considered statistically significant.

Results. Histopathological examination revealed significant structural alterations in the nasal mucosa of children with allergic rhinitis compared to controls ($p<0.05$). The most prominent finding was disruption of epithelial integrity accompanied by widespread desquamation and dystrophic changes.

A marked reduction in ciliated epithelial cells was observed, indicating impaired mucociliary clearance. In contrast, goblet cell hyperplasia and mucous gland hypertrophy were significantly increased, resulting in excessive mucus production and clinically correlating with rhinorrhea.

The stromal compartment demonstrated dense inflammatory cell infiltration, predominantly eosinophils. Eosinophil counts were several-fold higher in the study group compared to controls, showing statistical significance ($p<0.05$). Increased lymphocytes and mast cells further confirmed active allergic inflammation.

Vascular changes included dilatation of blood vessels and increased permeability, leading to pronounced mucosal edema. These changes contributed directly to nasal obstruction, one of the primary clinical symptoms of allergic rhinitis.

Overall, the findings indicate that structural remodeling of the nasal mucosa is a key pathological mechanism underlying allergic rhinitis in children.

Conclusions. The present study demonstrates that allergic rhinitis in children is associated with profound histopathological remodeling of the nasal mucosa. These

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changes include epithelial damage, goblet cell hyperplasia, glandular hypertrophy, and intense eosinophilic infiltration.

Such morphological alterations lead to impaired mucociliary function, excessive mucus secretion, and persistent inflammatory activity. These processes contribute to chronic disease progression and symptom persistence.

The observed vascular and cellular changes highlight the central role of inflammatory mediators in disease pathogenesis. Therefore, assessment of nasal mucosal morphology is essential for understanding disease severity and guiding individualized therapeutic approaches.

In conclusion, nasal mucosal remodeling represents a fundamental pathogenetic component of pediatric allergic rhinitis and should be considered in both diagnostic and treatment strategies.

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