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ETIOPATHOGENETIC MECHANISMS OF ALLERGIC STOMATITIS IN CHILDREN (LITERATURE REVIEW)

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Abstract*. Allergic stomatitis in young children is a complex inflammatory condition affecting the oral mucosa. It stems from a mix of genetic factors, immune system issues, and exposure to environmental allergens. Because their immune systems are still developing, young kids are more likely to experience hypersensitivity reactions, whether immediate or delayed. This review seeks to highlight the latest understanding of the causes behind allergic stomatitis in early childhood, focusing on the roles of immunoglobulins, cytokine imbalances, and microbial influences in both the onset and ongoing nature of the condition.*

Key words: *Allergic stomatitis, early childhood, immunopathogenesis, IgA, IgE, mucosal immunity, Th2 response, cytokines, dysbiosis.*

Introduction. Allergic stomatitis is an inflammatory condition affecting the oral mucosa, triggered by hypersensitivity reactions to various allergens, whether they come from food, microbes, or chemicals. In young children, the oral mucosa is especially susceptible to outside irritants because it's still developing and the local immune system isn't fully matured yet. Recently, there's been a noticeable rise in allergic lesions in the mouths of pediatric patients, which mirrors the increasing prevalence of atopic diseases around the globe [1]. The causes of allergic stomatitis in young children are quite intricate, involving a mix of immune responses, microbial factors, and nerve pathways. Grasping these underlying mechanisms is crucial for making accurate diagnoses, creating targeted treatments, and effectively preventing recurring inflammation in the mucosa. The main etiological triggers of allergic stomatitis in children include: food allergens such as milk proteins, egg components, citrus fruits, chocolate, and synthetic food additives; microbial allergens, including streptococcal and staphylococcal antigens that may cross-react with mucosal proteins[2]; contact allergens from oral hygiene products, medications, or dental materials; endogenous factors, such as chronic foci of infection, dysbiosis, and gastrointestinal disturbances that enhance systemic sensitization.The underdeveloped mucosal barrier and the still-maturing salivary IgA secretion in young children make it easier for allergens to pass through, which can lead to hypersensitivity reactions in the mouth [3]. The immune system's role in allergic stomatitis mainly revolves around the imbalance between local and systemic responses. Secretory immunoglobulin A (sIgA) serves as the main defense for the oral mucosa. In young children who are more prone to allergies, sIgA levels often drop, which weakens the mucosal surface's ability to fend off antigens. This local deficiency lets allergens and microbial toxins activate dendritic cells and Langerhans cells, setting off a chain reaction of inflammatory cytokines like IL-4, IL-5, and IL-13, which are typical of Th2-type immune responses [4]. On a systemic level, allergic stomatitis is linked to higher levels of total and specific IgE, increased eosinophil counts, and heightened activity of Th2 lymphocytes. These immune changes encourage mast cell degranulation and histamine release, resulting in vasodilation, swelling, and redness of the mucosa. In cases that are chronic or recurring, delayed-type hypersensitivity (Th1-mediated) reactions also play a role in tissue damage through macrophage activation and cytotoxic processes [5].

Recent pediatric case studies demonstrate that allergic and inflammatory oral le-

sions may occur even in immunocompromised children, underscoring the diagnostic complexity of mucosal immune reactions [6]. Sublingual immunotherapy has been shown to influence oral immune homeostasis and microbiota composition in children with atopic diseases, suggesting a potential therapeutic impact on allergic stomatitis [7]. The cytokine network in allergic stomatitis showcases the complex interaction between pro-inflammatory and regulatory mediators. An overproduction of IL-4, IL-5, and IL-13 fuels eosinophilic inflammation, while a drop in interferon-γ and IL-10 points to a failure in immunoregulation. This imbalance keeps mucosal inflammation going and slows down the healing of the epithelium [3]. The oral microbiota is essential for regulating our immune responses. When there's dysbiosis—meaning a drop in beneficial bacteria like Streptococcus salivarius and S. sanguis, along with a rise in harmful bacteria—it can throw our mucosal balance out of whack [2]. The byproducts of these microbes can act like boosters, making allergen presentation more effective and fueling inflammation. On top of that, environmental factors such as air pollution, preservatives in our food, and overzealous hygiene practices can further disrupt the mucosal environment, raising the chances of developing allergies. Additionally, variations in our genes related to cytokines (like IL-4 and IL-13) and IgE receptor levels might influence how susceptible we are to allergic conditions affecting our mucous membranes [1]. In young children, allergic stomatitis typically shows up as redness, swelling, itching, burning sensations, and painful sores in the mouth. This condition often appears alongside other allergic issues like skin rashes, nasal allergies, or asthma. Identifying changes in the immune system—especially a drop in sIgA and a rise in serum IgE—can help confirm the diagnosis and inform treatment options. Longitudinal studies highlight that isolated oropharyngeal allergic symptoms in children may predict a higher risk of subsequent food allergen intolerance [8]. When it comes to treatment, the focus should be on both removing allergens and correcting immune responses. Emerging evidence emphasizes the oral mucosa as a critical immunological interface capable of both initiating and modulating allergic and autoimmune responses [9]. Topical steroids and antihistamines can ease immediate discomfort, while probiotics and immune boosters work to restore balance in the mouth. To prevent future issues, it's important to manage allergic conditions early and maintain a healthy oral microbiome.

Discussion. The etiopathogenetic mechanisms of allergic stomatitis in early childhood demonstrate that this disorder is not limited to local mucosal inflammation but represents a systemic immunopathological process. Comprehensive reviews confirm that the oral cavity often reflects systemic immune dysregulation, serving as an early clinical marker for allergic and immune-mediated diseases [10] The immaturity of mucosal immunity and the predominance of Th2-mediated responses form the foundation of hypersensitivity reactions. The interconnection between the oral microbiota and mucosal immunity highlights the importance of maintaining microbial balance for preventing recurrent allergic inflammation. Current research emphasizes the need for integrated approaches combining immunological, microbiological, and genetic investigations to elucidate individual risk factors and optimize preventive care. Future studies should focus on molecular pathways regulating mucosal tolerance and the role of epithelial barrier proteins in allergen translocation.

The conclusion about allergic stomatitis in young children highlights a fascinating mix of immune, microbial, and environmental influences. The still-developing mucosal barrier and the imbalanced Th2 immune responses play key roles in how this condition develops. By grasping these underlying mechanisms, we can pave the way for earlier diagnoses, tailored immunotherapy, and ultimately better outcomes for kids dealing with allergic diseases.

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