Central Asian Research Journal For Interdisciplinary Studies (CARJIS). ISSN (online): 2181-2454
Volume 4 | Special Issue | July, 2025 | SJIF: 5,965 | UIF: 7,6 | ISRA: JIF 1.947 | Google Scholar |
https://carjis.afu.uz/

UDK:-616.31-002-056.3:612.017:616-053.2

ALLERGIK STOMATITLI ERTA YOSHLI BOLALARDA IMMUNOLOGIK O‘ZGARISHLAR

Shaxzodaxon Eshqulova
Toshkent davlat tibbiyot universiteti

Annotatsiya.

Erta yoshdagi bolalarda allergik stomatit — bu og‘iz shilliq qavatining yallig‘lanish kasalligi bo‘lib, u kechikkan yoki darhol rivojlanadigan gipersensitivlik reaksiyalari bilan bog‘liq. Ushbu tadqiqotda 1–4 yoshli bolalarda allergik stomatitda tizimli va lokal immun mexanizmlar o‘rtasidagi bog‘liqlik o‘rganildi.
Kalit so‘zlar: allergik stomatit, immunologik o‘zgarishlar, bolalar, sekretor immunoglobulin A, immunoglobulin E.

Аннотация.

Аллергический стоматит у детей раннего возраста представляет собой воспалительное поражение слизистой оболочки полости рта, опосредованное реакциями гиперчувствительности немедленного или замедленного типа. Целью данного исследования было изучение взаимосвязи между системными и локальными иммунными механизмами у детей 1–4 лет с аллергическим стоматитом.
Ключевые слова: аллергический стоматит, иммунологические изменения, дети, секреторный иммуноглобулин А, иммуноглобулин Е.

Abstract.

Allergic stomatitis in early childhood is an inflammatory condition of the oral mucosa mediated by immediate or delayed-type hypersensitivity. This study explored the relationship between systemic and local immune mechanisms in children aged 1–4 years with allergic stomatitis.
Keywords: allergic stomatitis, immunological changes, children, salivary immunoglobulin A, immunoglobulin E.

Introduction. Allergic diseases in young children are a major medical and social issue around the globe. In recent years, we've seen a rise in allergic conditions, which experts believe is linked to environmental influences, genetic factors, and changes in how our immune systems work [1,2]. Among these conditions, allergic stomatitis stands out because it involves both localized hypersensitivity in the mouth and a broader immune response throughout the body. The oral cavity serves as the first line of defense for our digestive and respiratory systems, constantly coming into contact with a variety of antigens—like food proteins, bacteria, and even chemicals from hygiene products. The mucosal immune system, especially secretory IgA (sIgA), is crucial for maintaining tolerance and preventing excessive inflammatory reactions.[2] When this balance is thrown off, the mucosa can become susceptible to hypersensitivity reactions, which can show up as redness, swelling, sores, and painful ulcers. Children aged 1 to 4 are particularly vulnerable since their immune systems are still maturing, and their mucosal immune responses are not fully developed. Additionally, if their parents have allergic conditions, this can heighten the risk of immune

dysregulation during this critical period. Despite how common allergic stomatitis is in pediatric care, we still don't fully understand its immunological aspects—especially how local mucosal immunity (sIgA) interacts with systemic allergic responses (like IgE and eosinophilia). This study aims to fill that knowledge gap by examining immunological markers in children with allergic stomatitis, looking closely at how mucosal and systemic immune responses relate to the severity of their symptoms.[3] Allergic diseases in early childhood have become a major medical and social concern worldwide. Over the past few decades, the incidence of allergic disorders has steadily increased, with researchers attributing this trend to environmental pollution, dietary changes, genetic predisposition, and altered immune system regulation [4]. According to epidemiological data, the global burden of pediatric allergic conditions continues to rise despite advances in prevention and treatment. This growing prevalence emphasizes the need to better understand the underlying immune mechanisms driving allergic inflammation in young children.

Among pediatric allergic conditions, allergic stomatitis occupies a special place because it involves both localized hypersensitivity reactions in the oral cavity and systemic immune activation. The oral cavity represents the first line of defense for both the digestive and respiratory tracts. It is constantly exposed to a variety of exogenous antigens—dietary proteins, microbial components, and chemical agents from oral hygiene products or medications [5] Therefore, the oral mucosal immune system plays a pivotal role in distinguishing between harmless antigens and harmful pathogens, maintaining immune tolerance while preventing overactivation. A key element of this mucosal immune defense is secretory immunoglobulin A (sIgA). This antibody is produced by plasma cells in the lamina propria and transported through epithelial cells into the saliva via the polymeric immunoglobulin receptor (pIgR). Once secreted, sIgA binds to antigens, viruses, and bacteria, blocking their attachment to the mucosal surface and preventing penetration of allergens and pathogens — a mechanism termed immune exclusion [6] In addition, sIgA modulates local inflammatory responses by interacting with dendritic cells and epithelial receptors, thus helping maintain immune homeostasis. A deficiency in sIgA can lead to increased mucosal permeability and exaggerated local inflammatory or allergic reactions. Children aged 1–4 years are particularly vulnerable to such immune dysregulation. At this stage, mucosal immune mechanisms are still developing, and the quantity and functionality of sIgA in saliva are significantly lower than in older children or adults [7]A delayed maturation of sIgA production has been associated with a higher risk of allergic manifestations, including food allergies and atopic dermatitis, in sensitized children. Genetic predisposition also contributes—children with allergic parents exhibit an increased tendency toward aberrant mucosal immune responses and elevated systemic IgE production during early immune development [8] In allergic stomatitis, the breakdown of this mucosal immune balance may manifest clinically as erythema, edema, erosions, or aphthous-like lesions on the oral mucosa, accompanied by burning, pain, and discomfort during eating. The pathophysiology of these lesions likely reflects an interplay between local immune suppression (decreased sIgA) and systemic hypersensitivity (elevated IgE and eosinophilia). However, despite its frequent occurrence in pediatric dental practice, the immunopathogenesis of allergic stomatitis remains insufficiently investigated, particularly concerning how local mucosal immune status correlates with systemic allergic parameters and clinical severity. Therefore, this study seeks to bridge this knowledge gap by examining both local (salivary sIgA) and systemic (serum IgE, blood eosinophil count) immune indicators in children aged 1–4 years with allergic stomatitis. By analyzing these parameters and their interrelationships, we aim to clarify the mechanistic link between mucosal immunity and systemic sensitization in early childhood allergy. A deeper understanding of

these processes could aid in developing targeted preventive and therapeutic strategies that strengthen mucosal defenses and reduce allergic burden in young children.

Materials and Methods . The study was conducted at the Department of Pediatric Therapeutic Dentistry, Tashkent State Medical University, from 2023 to 2025. Forty children aged **1–4 years** participated. They were divided into:

Group I (Study Group): 20 children diagnosed with allergic stomatitis;

Group II (Control Group)**:** 20 clinically healthy children of similar age and sex. We confirmed allergic stomatitis through clinical and laboratory tests, noting symptoms like redness, swelling of the mucosa, and the presence of vesicular or erosive lesions. There were no systemic diseases impacting immunity. Exclusion Criteria: 1. Participants currently on antibiotic or corticosteroid treatments. 2. Those with autoimmune, endocrine, or infectious diseases. 3. Individuals with congenital immunodeficiency. Ethical Considerations: This study followed the Declaration of Helsinki and received approval from the Ethics Committee at Tashkent State Medical University. We made sure to get written informed consent from parents or legal guardians. Clinical Assessment: Every child went through a thorough clinical examination, which included checking for mucosal changes (like redness, swelling, erosions, and ulcerations) and evaluating symptom severity (pain, burning sensations, and refusal to eat). Sample Collection: Saliva: We collected unstimulated saliva samples in the morning, one hour after eating or after oral hygiene routines. In the morning hours, specifically between 9:00 and 11:00 a.m., we collected unstimulated whole saliva samples. This was done at least an hour after the participants had eaten and completed their oral hygiene routines. The children were seated upright, and we collected about 2–3 mL of saliva through passive drooling into sterile polypropylene tubes. The samples were promptly placed on ice and then stored at –20°C until we were ready for analysis. Blood: Venous blood samples were taken to measure total IgE levels and eosinophil counts. We collected 2 mL of peripheral venous blood from the cubital vein while maintaining aseptic conditions. After collection, the samples were centrifuged to separate the serum, which we used to measure total IgE and eosinophil counts. Laboratory Methods: sIgA levels were measured using the ELISA method [4]. Total IgE was assessed through an enzyme-linked immunosorbent assay with standard diagnostic kits. Eosinophil counts were evaluated via hematological analysis ($\times 10^9$/L). Statistical Analysis: We analyzed the data using SPSS 25.0, presenting results as mean ± standard deviation (SD). We used Student's t-test to assess differences between groups, considering $p < 0.05$ as statistically significant. Correlation analysis was conducted using Pearson's coefficient.

Results. Children in the study group showed typical symptoms of allergic stomatitis: diffuse erythema, mucosal swelling, focal erosions, and small aphthous lesions on the tongue and cheeks. Some children complained of burning and discomfort while eating.

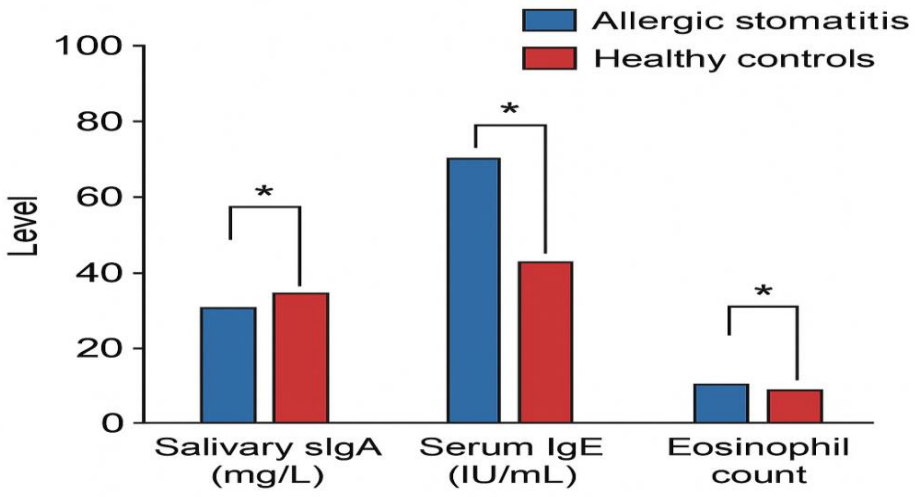
Table 1. Summarizes the primary immune parameters.

Parameter	Control group (n=20)	Allergic stomatitis (n=20)	*p*-value
Salivary sIgA (mg/L)	65 ± 10	40 ± 12	<0.05
Serum IgE (IU/mL)	45 ± 15	95 ± 22	<0.05
Eosinophil count ($\times 10^9$/L)	0.6 ± 0.2	1.5 ± 0.3	<0.05

Children suffering from allergic stomatitis showed a notable drop in sIgA levels, which points to a weakened defense in their mucosal lining. On the flip side, their serum

IgE and eosinophil counts were significantly higher, indicating a systemic allergic response. There was a strong negative relationship between salivary sIgA and serum IgE ($r = –0.68$, $p < 0.01$), hinting that lower local immunity is linked to increased systemic sensitization. Additionally, the eosinophil count had a positive correlation with IgE levels ($r = 0.59$, $p < 0.05$), reinforcing the idea of an interconnected allergic reaction. The severity of mucosal lesions was inversely related to sIgA levels. Children with the lowest sIgA (<35 mg/L) experienced severe erosions and significant pain, while those with moderate reductions (40–50 mg/L) showed milder symptoms like slight redness and swelling. As illustrated in Table 1, children suffering from allergic stomatitis showed a notable drop in salivary secretory immunoglobulin A (sIgA) when compared to healthy controls (40 ± 12 mg/L vs. 65 ± 10 mg/L, $p < 0.05$). This decrease points to a significant weakening of local mucosal immunity, which is crucial for the initial defense against allergens, pathogens, and irritants. A lower concentration of sIgA indicates that the oral mucosa is less capable of neutralizing antigens and maintaining immune balance, making it more susceptible to inflammation and damage to the epithelial layer. On the flip side, systemic immune markers displayed a contrasting trend. The average serum IgE concentration was over twice as high in the study group compared to the control group (95 ± 22 IU/mL vs. 45 ± 15 IU/mL, $p < 0.05$). Elevated IgE levels suggest a heightened systemic sensitization, aligning with allergic immune responses. Likewise, the eosinophil count was significantly higher in children with allergic stomatitis ($1.5 \pm 0.3 \times 10^9$/L) compared to controls ($0.6 \pm 0.2 \times 10^9$/L, $p < 0.05$), confirming the activation of cellular mechanisms linked to allergic inflammation. Correlation analysis uncovered significant relationships between the parameters measured. A strong negative correlation was found between salivary sIgA and serum IgE levels ($r = –0.68$, $p < 0.01$), indicating that a decrease in local mucosal immunity is closely tied to increased systemic allergic sensitization. Additionally, a positive correlation was noted between eosinophil count and serum IgE levels ($r = 0.59$, $p < 0.05$), suggesting that higher IgE levels are associated with more pronounced eosinophilic responses, which are characteristic of allergic inflammation. The clinical severity of oral mucosal lesions also correlated with immunological indices. Children with significantly reduced sIgA levels (<35 mg/L) displayed severe clinical symptoms, including multiple erosions, extensive mucosal swelling, and intense pain while eating.

Figure 1 . Results of testing.



Discussion. The findings demonstrate that allergic stomatitis in early-aged children is not merely a localized response but part of a broader immune imbalance. The suppression

of mucosal IgA production appears to remove a critical layer of protection, allowing allergen penetration and triggering systemic hypersensitivity.

Local immune deficiency:

Secretory IgA is the first immunological barrier in the oral cavity. It prevents antigen absorption and neutralizes pathogens without provoking inflammation [1]. Its decline, therefore, facilitates antigen entry, amplifying allergic reactions in susceptible individuals.

Systemic hypersensitivity:

Increased serum IgE reflects overactivation of Th2 lymphocytes and mast cell sensitization—hallmarks of atopic conditions [4]. Elevated eosinophil counts support the notion of ongoing allergic inflammation, where eosinophils release toxic proteins contributing to tissue injury and delayed healing.

Mucosal–systemic interaction:

The observed inverse correlation between sIgA and IgE levels suggests a dynamic cross-talk between local and systemic immunity. When mucosal tolerance is lost, systemic allergic responses intensify, perpetuating inflammation—a pattern consistent with the "mucosal–systemic axis" model described in pediatric allergy [2]. Clinical implications**:**

From a clinical standpoint, evaluating salivary sIgA alongside serum IgE provides a valuable diagnostic approach. Low sIgA can indicate vulnerability to mucosal inflammation, while elevated IgE predicts systemic reactivity. The combination may also help monitor treatment outcomes or disease remission. Therapeutic perspectives**:** Modern approaches to allergic stomatitis in children should extend beyond symptomatic care (antihistamines, topical corticosteroids). Restoring mucosal immunity is equally important. Probiotic therapy, vitamin D correction, and local immunomodulators (e.g., lysozyme-based agents) could support IgA synthesis and mucosal homeostasis. Comparison with existing studies:

Our data align with previous observations in pediatric allergy research. Syrlig and Sistig (2002) found reduced sIgA in oral mucosal diseases, while Assiri et al. [4] demonstrated elevated salivary IgE and eosinophil cationic protein in allergic children. Together, these studies underline the dual nature of allergic stomatitis as both a local and systemic immunopathological process.

Conclusion . Allergic stomatitis in children aged 1–4 years reflects a combined immune dysfunction—suppressed mucosal defense (low sIgA) and systemic hypersensitivity (high IgE, eosinophilia). The imbalance between these components determines disease severity and persistence. The simultaneous evaluation of salivary sIgA and serum IgE is proposed as a **non-invasive marker** for immune imbalance and disease monitoring in pediatric allergic stomatitis. Future research should investigate interventions aimed at restoring mucosal immunity and preventing allergic progression in early childhood.

References :

1. Brandtzaeg, P. (2013). Secretory IgA: Designed for antimicrobial defense. *Frontiers in Immunology, 4*(222). https://doi.org/10.3389/fimmu.2013.00222
2. Mantis, N. J., Rol, N., & Corthésy, B. (2011). Secretory IgA's complex roles in immunity and mucosal homeostasis. *Mucosal Immunology, 4*, 603–611. https://doi.org/10.1038/mi.2011.41
3. Syrlig, S., & Sistig, S. (2002). Salivary IgA and IgG subclasses in oral mucosal diseases. *Journal of Oral Pathology & Medicine, 31*(9), 499–506. https://doi.org/10.1034/j.1601-0825.2002.20844.x
4. Assiri, K. A., et al. (2023). Correlating salivary levels of IgE and human eosinophil cationic protein in allergic children. *Saudi Journal of Biological Sciences, 30*(8), 10348369. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10348369/

5. Global epidemiology of allergic diseases in children — BMC Pulmonary Medicine (2025)
6. Oral mucosal immunity and hypersensitivity mechanisms — ***Journal of Allergy and Clinical Immunology***, 2023
7. Brandtzaeg P. Role of secretory IgA in mucosal immunity — ***Frontiers in Immunology***, 2020
8. Hjelm B. et al. Maturation of salivary secretory immunity and allergy development in children — ***Pediatric Research***, 2011
9. World Allergy Organization: Allergic diseases in childhood — ***WAO Journal***, 2021