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Causes of Allergic Diseases in Children

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Abstract: Currently, allergies are one of the most common pathological conditions in children. According to epidemiological studies, up to 25% of children and adolescents suffer from allergic diseases (ADs). Once an allergy occurs, it carries the risk of developing severe allergic reactions that can pose a threat to the lives of patients; allergic diseases (AD) reduce the quality of life of children and adolescents. To properly monitor their course, significant financial expenditures are required on the part of society as a whole and families with children with AD.

Keywords: allergic diseases, allergic rhinitis, atopic dermatitis, bronchial asthma.

All this puts the problem of allergic pathology at one of the first places in modern pediatrics.

The most common diseases in children are atopic diseases (allergic rhinitis - AR, atopic dermatitis - AD, bronchial asthma - BA). Over the past two decades, there has been a continuing trend towards an increase in the prevalence of AB in childhood. In their development, an important role is played by the interaction of genetic and environmental factors with the subsequent development of sensitization of the body, but in general, the immediate causes of the increase in the prevalence of AB remain insufficiently clear. Epidemiological studies conducted in many countries have shown that the high prevalence of atopic diseases is associated with a Western lifestyle, urbanization, environmental pollution with chemical compounds, and high socioeconomic status [1]. In the 2000s, there was an increase in the prevalence of food allergies (FA) in children, which is characterized by a number of studies as a new epidemic of FA, considered as an important reason for the increasing prevalence of respiratory AD in children and adolescents. The role of consumption of genetically modified foods and combination foods containing chemical ingredients as additives in the increased occurrence of PA cannot be excluded.

A significant risk factor for the occurrence of respiratory bronchitis is exposure to tobacco smoke. Passive smoking in childhood and maternal smoking during pregnancy and infancy are causally associated with asthma, the occurrence of its symptoms and the deterioration of pulmonary functions in children. Tobacco smoke can enhance the production of IgE and contribute to increased sensitization of the body.

A high risk factor for the occurrence of AB in children is the presence of atopy, characterized as an innate tendency to overproduction of general and specific IgE and the development of subsequent hypersensitivity to allergens. The risk of allergy manifestation in children and adolescents with a high atopy index is especially significant (severe hereditary burden of allergic reactions and diseases, detection of positive skin tests with exogenous allergens in a child, identification of minor clinical signs of allergy).

According to epidemiological studies, a lower prevalence of AD is observed in children living in rural areas, which is associated with more frequent contact with microbial agents that promote maturation and increased activity of the innate immune system, which can inhibit the development of AD [2].

Genetic and environmental factors are decisive in the development of allergic pathology in children. Genetic factors have a strong influence on the formation of atopic diseases. By their nature, they are multifactorial diseases, the development of which is associated with the interaction of genetic and environmental factors. Clinical observations indicate a connection between atopic diseases (BA, AR and AD) with a family predisposition to their occurrence. In recent years, research has been most intensively conducted to identify gene connections with clinical allergy variants. Allergy in general is a

polygenic disease; its development is determined by many genes encoding the synthesis of biologically active compounds involved in the pathogenesis of AD. Thus, atopic predisposition is associated with chromosome Iq21, which contains a locus of 30 genes encoding proteins involved in the construction and regulation of the function of the epithelial barrier. It was found that a mutation in the gene encoding the filaggrin protein, which plays a decisive role in the creation of the skin barrier, is the main predisposing factor for AD. Based on genomic studies in AD and PA, the possibility of identifying disease phenotypes was established.

The development of BA, AR, AD and their combined manifestations is associated with polymorphic variants of genes that play a key role in the pathogenesis of atopic diseases, determining sensitivity to pharmacological drugs of pathogenetic therapy (lipoxygenase-5 genes, glucocorticosteroid receptors, β2-adrenergic receptors, tumor necrotizing factor α, enzymes biotransformation of xenobiotics). Susceptibility to AD and other atopic diseases may be associated with variations in genes encoding components of the innate immune system. Thus, genetic variations in TLRs (Toll receptors), caused by mutations, can predispose to immune abnormalities that cause the development of AD.

A study of gene-gene interactions in atopic diseases showed that the expression of these diseases is associated with a combination of polymorphic gene variants. It has been established that an increase in the risk of developing AD may be due to the interaction of three genes: IL4, IL13 and STAT-6 [3].

In the pathogenesis of atopic diseases, gene-environmental interaction occurs, with the mutual influence of polymorphic variants of genes and environmental factors. Research into identifying complex genetic profiles associated with allergy phenotypes is promising in this direction.

The influence of the immune system on the development of AB is significant. Immunologically, the allergic response is characterized by dysfunction of allergen-specific T cells with a predominance of pathogenetic effector Th2 lymphocytes, followed by the inclusion of an IgE response and the development of allergic inflammation. The development of inflammation in the shock organ in atopic diseases depends on interactions between the innate immune system, such as dendritic cells, and the adaptive immune system, and especially T lymphocytes. This interaction is determined by the type of T-effector cells, such as Th1/Th2, Th9, Th17 and Th22, involved in the development of inflammation, while the resulting Th2 response with the release of pro-inflammatory cytokines (IL4, IL5, IL13) is the main driving force in the inflammatory immune response. answer. Th17 cells are involved in the pathogenesis of those forms of AD in which neutrophils contribute more than eosinophils to the development of inflammation [4]. At the same time, Th1 lymphocytes produce IFNγ, which has anti-inflammatory activity and the ability to inhibit the development of Th2 immune responses. The development of atopic diseases occurs due to dysregulation and imbalance of the innate and adaptive immune response, which is disrupted in patients with allergies due to gene-environment interactions. The functions of the innate immune system in a child at birth are significantly weakened. In newborns and in the postnatal period, the antigen-presenting ability of dendritic cells is reduced; at birth, the activity of NK cells, which normally have the ability to modulate the functions of dendritic cells, is weakened. In this regard, it has been hypothesized that delayed maturation of the innate immune system, including dendritic cells, is one of the factors contributing to the development of atopy. The functional maturation of dendritic cells occurs through the interaction of Toll receptors on the surface of these cells with microbial ligands. The functional competence of dendritic cells is influenced by the transplacental supply of microbial ligands in the antenatal period.

Allergen-specific Treg cells are involved in the pathogenesis of atopic diseases; their deficiency can contribute to the development of atopy and AB. Treg cells control immunopathological processes in the body and inhibit the development of allergic reactions and diseases. A high level of Treg cells protects the body against atopy and AB.

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