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MODERN PERSPECTIVES ON ALLERGIC DISEASES IN CHILDREN

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Abstract:

Currently, allergies are among the most common pathological conditions in children. According to epidemiological studies, up to 25% of children and adolescents suffer from allergic diseases (AD). Once developed, allergies pose a risk of severe allergic reactions that can be life-threatening. Allergic diseases (AD) also reduce the quality of life for children and adolescents. Proper control of these conditions requires significant financial expenditures from both society as a whole and families with children affected by allergies.

Keywords: Allergic diseases, children, atopic dermatitis (AD), allergic rhinitis (AR), bronchial asthma (BA), sensitization, genetic predisposition, environmental factors, immune response, inflammatory cytokines.

Introduction

All of this places allergic pathology among the top concerns in modern pediatrics. The most common allergic conditions in children are atopic diseases, including allergic rhinitis (AR), atopic dermatitis (AD), and bronchial asthma (BA). Over the past two decades, the prevalence of allergic diseases in children has continued to rise. The development of these conditions is largely influenced by the interaction between genetic and environmental factors, leading to the sensitization of the body. However, the exact reasons behind the increasing prevalence of allergic diseases remain unclear.

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 a high socioeconomic status [1]. During the 2000s, there was an increase in the prevalence of food allergies (FA) in children. Several studies describe this trend as a new epidemic

of FA, which is considered a significant factor in the rising prevalence of allergic diseases affecting the respiratory system in children and adolescents [2]. The frequent occurrence of FA may also be linked to the consumption of genetically modified foods and processed foods containing chemical additives.

One significant risk factor for the development of allergic respiratory diseases is exposure to tobacco smoke. Passive smoking during childhood, maternal smoking during pregnancy, and exposure to smoke in infancy are causally linked to bronchial asthma (BA), its symptoms, and reduced lung function in children. Tobacco smoke can enhance the production of IgE and contribute to increased sensitization of the body [3].

A strong risk factor for allergic diseases in children is atopy, which is characterized by an inherited tendency to overproduce both total and specific IgE, leading to hypersensitivity to allergens. The risk of allergy manifestation is particularly high in children and adolescents with a high atopy index—those with a strong family history of allergic reactions and diseases, positive skin tests for exogenous allergens, and minor clinical signs of allergy.

According to epidemiological studies, allergic diseases are less prevalent in children living in rural areas. This is believed to be due to their more frequent exposure to microbial agents, which contribute to the maturation and increased activity of the innate immune system, potentially slowing down the development of allergic conditions [4].

Genetic and environmental factors play a decisive role in the development of allergic pathology in children. Genetic factors have a strong influence on the formation of atopic diseases. By nature, these are multifactorial diseases, whose development is linked to the interaction between genetic and environmental factors. Clinical observations indicate a connection between atopic diseases (bronchial asthma, allergic rhinitis, and atopic dermatitis) and familial predisposition.

In recent years, research has focused intensively on identifying genetic links to clinical variants of allergies. Allergies, in general, are polygenic disorders, with their development being determined by multiple genes that encode the synthesis of biologically active compounds involved in the pathogenesis of allergic diseases. For instance, atopic predisposition is associated with chromosome Iq21, which contains a locus of 30 genes encoding proteins involved in the formation and regulation of the epithelial barrier. It has been established that a mutation in the gene encoding filaggrin, a protein crucial for maintaining the skin barrier, is a major predisposing factor for atopic dermatitis [5].

Genomic studies on bronchial asthma, atopic dermatitis, and food allergies have enabled the identification of disease phenotypes. The development of bronchial asthma, allergic rhinitis, and atopic dermatitis, as well as their combined manifestations, is associated with polymorphic variants of genes that play key roles in the pathogenesis of atopic diseases. These genes also determine sensitivity to pharmacological treatments, including lipoxygenase-5 genes, glucocorticosteroid receptors, β2-adrenergic receptors, tumor necrosis factor alpha, and xenobiotic biotransformation enzymes [6]. The predisposition to bronchial asthma and other atopic diseases may be linked to variations in genes encoding components of innate immunity. For example, genetic variations in TLRs (Toll-like receptors) due to mutations may predispose individuals to immune deviations that trigger allergic conditions [7].

Studies on gene-gene interactions in atopic diseases have shown that their expression is linked to combinations of polymorphic gene variants. It has been established that the risk of developing bronchial asthma increases with the interaction of three genes: IL4, IL13, and STAT-6 [8].The pathogenesis of atopic diseases involves gene-environment interactions, wherein polymorphic gene variants and environmental factors mutually influence each other. A promising research direction in this field is the identification of complex genetic profiles associated with allergy phenotypes.

The immune system significantly influences the development of allergic diseases. Immunologically, the allergic response is characterized by dysfunction of allergen-specific T-cells, with a predominance of pathogenic effector Th2 lymphocytes. This leads to the activation of an IgE-mediated response and the development of allergic inflammation. The inflammation that occurs in target organs in atopic diseases is dependent on interactions between the innate immune system (e.g., dendritic cells) and the adaptive immune system, particularly T-lymphocytes. This interaction is determined by the type of T-effector cells involved, such as Th1/Th2, Th9, Th17, and Th22, which contribute to inflammation. The Th2 response, which leads to the release of pro-inflammatory cytokines (IL4, IL5, IL13), is the main driving force behind the inflammatory immune response. Th17 cells are involved in the pathogenesis of bronchial asthma subtypes where neutrophils, rather than eosinophils, play a greater role in inflammation [9].

Meanwhile, Th1 lymphocytes produce IFNγ, which has anti-inflammatory properties and can suppress Th2 immune responses. The development of atopic diseases occurs due to dysregulation and imbalance between innate and adaptive immune responses, which are disrupted in allergic individuals as a result of gene-environment interactions.

At birth, the functions of the innate immune system in children are significantly weakened. In newborns and during the postnatal period, dendritic cells have reduced antigen-presenting capacity, and natural killer (NK) cells, which normally modulate dendritic cell functions, have diminished activity. This has led to the hypothesis that delayed maturation of the innate immune system, including dendritic cells, is one of the factors contributing to atopy development [10]. The functional maturation of dendritic cells occurs through the interaction of Toll-like receptors on their surface with microbial ligands. The functional competence of dendritic cells is also influenced by the transplacental transfer of microbial ligands during the antenatal period [11].

Allergen-specific regulatory T (Treg) cells are involved in the pathogenesis of atopic diseases. A deficiency in these cells may contribute to the development of atopy and allergic diseases. Treg cells play a crucial role in controlling immunopathological processes and suppressing allergic reactions and diseases. A high level of Treg cells provides protection against atopy and allergic diseases.

Atopic sensitization to environmental allergens can develop even in utero. Allergenic peptides that enter the mother's body can activate the fetal immune system, which subsequently serves as a pre-adaptation to the effects of antigenic agents in the environment. Early sensitization can significantly contribute to Th2 polarization of the immune response, which is often observed in newborns.

In children without atopy in the first years of life, Th2 polarization shifts to a predominance of the Th1 immune response. However, if a child has atopy, this transition does not occur, and Th2 lymphocyte activity remains dominant, contributing to the later manifestation of allergic symptoms. The presence of allergic diseases (AD) in the mother is the most reliable marker of allergy development in newborns, as it results from a strong genetic background and an immune environment characteristic of allergic conditions.

Apart from IgE-mediated atopic diseases, children can also develop allergic conditions caused by cytotoxic reactions (allergic reactions with blood cell damage), immune complex reactions (vasculitis, serum sickness), delayed-type hypersensitivity (contact dermatitis, severe chronic atopic dermatitis), and combined immunopathological reactions (acute toxic-allergic reactions).In addition to the strong influence of genetic factors on the development of sensitization and atopic diseases, several environmental factors modulate clinical and immunological phenotypes in early life and even prenatally. These factors include gastrointestinal and skin exposure to food allergens, respiratory exposure to household and environmental allergens, and the presence of pollutants such as tobacco smoke and other chemicals, as well as exposure to various microorganisms.

Atopic dermatitis (AD) usually manifests in early childhood and is often associated with food allergies (especially to cow's milk proteins). As children grow older, sensitization to inhaled allergens (house dust mites, Dermatophagoides pteronyssinus, Dermatophagoides farinae, pollen, fungal spores, and epidermal allergens) becomes more significant in the pathogenesis of the disease. Sensitization to these allergens can also lead to the development of urticaria and angioedema. Among young children (under two years old), sensitization to cow's milk proteins, soy, and gluten plays a crucial role in the development of AD. In most cases, AD precedes the onset of other atopic diseases.

Bronchial asthma (BA) often begins in early childhood, but its peak incidence occurs in preschool and school-age children. Sensitization to inhaled allergens (house dust mites, pollen, fungal spores, and epidermal allergens) plays a key role in its development. Mono-sensitization to food allergens is rarely the cause of BA in children.

Allergic rhinitis (AR) is relatively rare in children under two years of age. It is most commonly observed in school-age children and adolescents. Persistent AR is usually associated with sensitization to house dust mites (Dermatophagoides pteronyssinus, Dermatophagoides farinae) and mold allergens. Intermittent AR is more commonly associated with pollen allergens, pet dander, and food allergens.

The clinical manifestation of pollinosis typically occurs in preschool and school-age children. Pollinosis identified in adolescents is usually a continuation of the disease that began in childhood.

Urticaria associated with exposure to exogenous allergens is observed in half of the children diagnosed with this condition.

In children and adolescents with atopic diseases, poly-sensitization is often detected during allergy testing, especially in those with a family history of allergic reactions and diseases. In some cases, the development of AD is triggered by drug allergies.

Viral infections play a significant role in the development of allergic diseases in children. The involvement of viral infections in the pathogenesis of atopic BA is particularly well-established. The number of acute respiratory infections a child experiences in the first two years of life is linearly related to the risk of developing BA later on. Persistent BA often begins in infancy. The progression from allergic sensitization to atopic BA is more common when early atopy is accompanied by respiratory viral infections.

Viral infections enhance the effector mechanisms associated with atopy and exacerbate allergic inflammation in the infected respiratory mucosa, accelerating the development of BA. Intercurrent acute respiratory viral infections caused by RSV (respiratory syncytial virus), rhinoviruses, and parainfluenza viruses are among the most common triggers for BA exacerbations in children.

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