

THE IMPACT OF PARASITIC INFESTATIONS ON THE DEVELOPMENT OF ALLERGIC DISEASES AND CHANGES IN IMMUNE REACTIVITY IN CHILDREN

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ABSTRACT: Parasitic infestations remain an important pediatric health problem, particularly in regions where environmental contamination, inadequate sanitation, close contact with animals, and delayed diagnosis facilitate transmission. In children, helminths and protozoa can influence not only the gastrointestinal and nutritional status but also the functional activity of the immune system. The relationship between parasitic infection and allergy is complex: some parasites may promote type 2 immune responses characterized by eosinophilia and increased immunoglobulin E, whereas chronic helminth infections may simultaneously activate regulatory pathways that suppress excessive inflammation. These mechanisms can alter the clinical expression of bronchial asthma, allergic rhinitis, atopic dermatitis, urticaria, and other hypersensitivity disorders. This thesis summarizes current concepts regarding the immunological mechanisms linking parasitic infestations with allergic disease in children, highlights the role of Th2 lymphocytes, eosinophils, mast cells, IgE and regulatory T cells, and emphasizes the importance of accurate parasitological and immunological diagnosis. A comprehensive approach to children with recurrent or atypical allergic manifestations may improve differential diagnosis, reduce unnecessary treatment and support individualized prevention strategies.

Keywords: parasitic infestations, children, allergy, immune reactivity, helminths, IgE, eosinophilia, Th2 response, immunoregulation.

INTRODUCTION

Allergic diseases are among the most frequent chronic disorders of childhood, while parasitic infections continue to affect millions of children worldwide. Soil-transmitted helminths are especially common in areas with insufficient sanitation, and infected children may experience nutritional impairment, anemia and growth disturbances [4]. At the same time, helminths and other parasites interact intensively with the host immune system. This interaction is clinically important because many immunological features of parasitic disease overlap with mechanisms involved in allergic inflammation. Uzbek medical literature on parasitology, immunology and allergology also emphasizes the necessity of considering parasitic invasion in the differential diagnosis of eosinophilia, recurrent skin eruptions, respiratory hypersensitivity and prolonged immune reactivity in children [1-3].

The immunological relationship is not unidirectional. Acute or tissue-migrating parasites may enhance allergic-like inflammation, while long-standing helminth infections can induce regulatory mechanisms capable of attenuating allergic responses. Therefore, the clinical effect depends on parasite species, intensity and duration of infection, age at exposure, genetic susceptibility and the immunological phenotype of the child [5]. Understanding these mechanisms is essential for pediatricians, allergists and infectious disease specialists.

MAIN PART

The immune response to helminths is predominantly associated with type 2 immunity. Parasite antigens activate antigen-presenting cells and promote differentiation of CD4⁺ T lymphocytes toward the Th2 phenotype. Th2 cells produce interleukins such as IL-4, IL-5 and IL-13, which stimulate B-cell class switching toward IgE synthesis, eosinophil maturation and activation, mucus production and enhanced contractility of smooth muscle. These reactions are protective against multicellular parasites, but the same pathways participate in the pathogenesis of atopic diseases. As a result, infected children may present with elevated total IgE, peripheral eosinophilia, pruritus, urticarial eruptions, cough, wheezing or rhinitis-like symptoms that can mimic or aggravate allergy [2,3].

Eosinophils are particularly important in the interface between parasitic invasion and allergy. During tissue migration of *Ascaris*, *Toxocara* and other helminths, eosinophils accumulate in blood and tissues and release cytotoxic granule proteins, leukotrienes and cytokines. These mediators may contribute to tissue injury and respiratory symptoms. In susceptible children, cross-reactivity between parasite antigens and environmental allergens may additionally influence IgE-mediated responses. Anti-*Ascaris* IgE, for example, has been discussed as a marker associated with asthma and bronchial hyperreactivity in some populations, although the strength and direction of the association vary among studies [5].

Mast cells and basophils also participate in antiparasitic defense. IgE bound to high-affinity receptors on these cells can trigger rapid degranulation after antigen recognition, causing release of histamine and other inflammatory mediators. Clinically, this can resemble immediate hypersensitivity. However, chronic helminth infections do not simply intensify immunity. To survive for long periods, many helminths stimulate regulatory T cells, regulatory B cells and anti-inflammatory cytokines such as IL-10 and transforming growth factor-beta. These mechanisms can suppress excessive immune activation and may reduce skin-test reactivity or the clinical intensity of some allergic conditions. This dual effect explains why epidemiological studies have reported both positive and negative associations between helminth infection and allergy [5].

In pediatric practice, the most relevant manifestations include recurrent urticaria, pruritic dermatoses, prolonged eosinophilia, episodic bronchospasm, chronic cough, abdominal complaints and nonspecific constitutional symptoms. These findings should not automatically be attributed to parasites; they require differential diagnosis. A careful history should assess travel or residence in endemic areas, contact with soil and animals, pica, hygiene practices, consumption of unwashed vegetables, and family or community exposure. Laboratory evaluation may include a complete blood count with eosinophil count, stool microscopy or concentration methods for intestinal parasites, antigen tests or molecular assays when available, and serology for selected tissue helminthiases. In children with allergic symptoms, total and specific IgE tests, skin-prick testing and assessment of respiratory function may be necessary to distinguish true atopy from parasite-associated immune activation [1-3].

The interpretation of total IgE deserves particular caution. High IgE is not specific for allergy and can be observed in helminth infections. Similarly, eosinophilia may occur in allergic disease, parasitosis, drug reactions and other conditions. Therefore, diagnosis should be based on the combined clinical, epidemiological, parasitological and immunological picture. Empirical antiparasitic treatment solely because of elevated IgE or eosinophilia is not always justified; treatment should follow evidence of infection and local clinical recommendations. Public health prevention remains essential and includes

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hand hygiene, safe drinking water, washing fruits and vegetables, proper disposal of feces, deworming programs where officially indicated, and health education for families and children [1,4].

From an immunological perspective, parasite-induced modulation is also relevant for research. Modern studies show that helminths secrete molecules capable of altering dendritic-cell activity, T-cell differentiation and cytokine production. These findings have generated interest in parasite-derived immunomodulatory molecules as potential models for future anti-inflammatory therapies. Nevertheless, intentional parasitic infection is not an accepted treatment for childhood allergy, and the clinical priority remains prevention, accurate diagnosis and evidence-based management of both parasitic and allergic diseases.

CONCLUSION

Parasitic infestations can significantly modify immune reactivity in children and may either promote allergic-like inflammation or induce regulatory responses that reduce excessive immunity. The most important shared mechanisms include Th2 polarization, eosinophil activation, increased IgE synthesis, mast-cell responses and regulatory cytokine pathways. Because parasitic disease and allergy may present with similar clinical and laboratory findings, pediatric evaluation should avoid relying on a single marker such as eosinophilia or total IgE. Integration of epidemiological history, parasitological testing and allergy-focused immunological assessment provides a more reliable diagnostic strategy. Strengthening hygiene, targeted parasite control and rational differential diagnosis may reduce the burden of both parasitic infections and unnecessary allergy treatment in children.

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