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CLINICAL AND IMMUNOLOGICAL FEATURES OF BRONCHIAL ASTHMA IN CHILDREN UNDER THE INFLUENCE OF ATYPICAL MICROFLORA

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Introduction

Today, bronchial asthma (BA) remains one of the most common chronic, inflammatory, and allergic diseases in pediatrics. According to the World Health Organization (WHO), approximately 10–15% of the pediatric population suffers from varying degrees of bronchial asthma. The development of the disease is significantly influenced by environmental factors, genetic predisposition, immune system imbalance, and various infectious agents.In recent years, the role of atypical respiratory pathogens such as Mycoplasma pneumoniae and Chlamydia pneumoniae has become increasingly evident, particularly in the worsening of asthma symptoms, shortening of remission periods, and persistence of inflammatory processes. These atypical infections exhibit tropism for bronchial epithelial cells and can induce prolonged, low-grade inflammation. This leads to sustained airway hyperresponsiveness, mucosal edema, and increased production of cytokines and inflammatory mediators. As a result, standard symptomatic therapy becomes less effective in such chronic inflammatory backgrounds, necessitating new approaches in treatment strategies. In this context, the role of the gut microbiota — one of the key systems involved in the regulation of immune responses — is emerging as a crucial topic of investigation. The gut microbiota is a complex biological environment composed of billions of microorganisms. It plays a vital role not only in digestion but also in maintaining immunological homeostasis, developing tolerance to allergens, and modulating inflammatory responses. Dysbiosis — characterized by a reduction in beneficial microbes and an overgrowth of conditionally pathogenic organisms — can disrupt immune responses and

exacerbate the course of allergic diseases, particularly in children. Recent scientific studies have substantiated the existence of the "gut-lung axis," a bidirectional relationship between gut microbiota and respiratory health. Disruption of the intestinal microbiota may lead to increased systemic inflammation, a dominance of Th2-type immune responses, and consequently, the development of more severe and treatment-resistant forms of bronchial asthma. Especially in cases of asthma associated with atypical respiratory infections, assessing the condition of the gut microbiota is not only essential for a deeper understanding of the disease pathogenesis but also provides a scientific and practical foundation for designing individualized and effective treatment plans.

Research Objective

The objective of this study is to identify the compositional and quantitative changes in the gut microbiota of children with bronchial asthma associated with atypical infections, and to evaluate how these changes affect immunological parameters. Based on the findings, new clinical approaches aimed at alleviating the course of asthma in children and developing immunomodulatory strategies will be proposed.

Materials and Methods

The study was conducted at the clinical base of the Tashkent Medical Academy between 2023 and 2025. A total of 60 children aged 7 to 15 years, diagnosed with bronchial asthma, were enrolled in the clinical observation. The diagnosis was established according to the Global Initiative for Asthma (GINA) guidelines, based on clinical, anamnestic, spirometric, and laboratory data. Participants were divided into two groups: Main group: 30 children with bronchial asthma in whom Mycoplasma pneumoniae or Chlamydia pneumoniae infection was confirmed by laboratory testing using PCR and serological ELISA methods. Comparison (control) group: 30 children diagnosed with bronchial asthma, but without laboratory-confirmed atypical pathogens. Stool samples were collected from each participant and analyzed using microbiological methods to assess the composition of the intestinal microbiota. The following microorganisms were identified and

quantitatively evaluated using selective differential media: Beneficial microflora: Bifidobacterium spp., Lactobacillus spp.

Conditionally pathogenic and saprophytic flora: Escherichia coli (typical and hemolytic strains), Bacteroides spp., Clostridium spp. The results were expressed in CFU (colony-forming units) per gram of stool. The degree of dysbiosis in the gut microbiota was assessed according to established normative criteria defined by specialists. In addition, peripheral blood samples were collected from all participants to determine the following immunological parameters: Total IgE level , Eosinophil count , T-lymphocyte subpopulations (CD3+, CD4+, CD8+, CD4/CD8 ratio)

Results

The conducted clinical and paraclinical studies demonstrated significant pathological changes in the composition and balance of the gut microbiota in children with bronchial asthma associated with atypical respiratory infections, particularly Mycoplasma pneumoniae and Chlamydia pneumoniae. According to the results, levels of beneficial microflora representatives—Bifidobacterium spp. and Lactobacillus spp.—were markedly decreased compared to the healthy control group, with statistically significant differences ($p<0.01$). These microorganisms play a crucial role in immune modulation, protecting the intestinal mucosa, and promoting tolerance to antigens. Their reduction creates a foundation for immunological dysfunction and activation of inflammatory processes. Conversely, the relative amounts of conditionally pathogenic and pathogenic flora—Escherichia coli and Clostridium species—were significantly increased in infected children, indicating a pronounced state of intestinal dysbiosis ($p<0.01$). Particularly, the presence of toxin-producing strains such as Clostridium difficile can be considered a factor enhancing systemic immune responses via increased inflammatory mediators. Although the quantity of Bacteroides species remained relatively stable, a decrease was observed under infectious conditions ($p<0.05$). These bacteria are key producers of short-chain fatty acids (SCFAs) that maintain a normal anaerobic environment. Their reduction may impair the metabolic support of the intestinal epithelium and enhance hyperreactivity to antigens.

Against the backdrop of microbiota imbalance, immunological hyperactivity was observed in the children. Notably, total IgE levels were significantly elevated, and peripheral blood analysis revealed an increased eosinophil count, indicating the activation of allergic inflammatory processes. Immunophenotyping showed a predominance of Th2-type immune responses, which led to increased production of key cytokines (IL-4, IL-5, IL-13) implicated in the pathogenesis of allergic diseases. Furthermore, children with severe bronchial asthma exhibited deeper microbiota dysbiosis, directly correlating with the intensity and duration of clinical symptoms. In such cases, prolonged symptoms and reduced efficacy of anti-inflammatory therapy were common, underscoring the critical role of microbiota in immune regulation.

Conclusion

The results of this study indicate that children with bronchial asthma associated with atypical respiratory infections develop a significant qualitative and quantitative imbalance in their gut microbiota. A reduction in beneficial bacteria alongside an increase in pathogenic flora amplifies immunological hyperreactivity, accompanied by elevated inflammatory markers and intensified allergic sensitization. These changes are especially pronounced in children with severe forms of bronchial asthma, significantly influencing clinical manifestations, remission duration, and treatment efficacy. The immunoregulatory mechanisms mediated via the “gut-lung axis” play a key pathogenetic role in this disease. Accordingly, systematic assessment of gut microbiota status, identification, and correction of dysbiosis through comprehensive therapeutic approaches—including probiotics, immunobiotics, prebiotics, and immunomodulators—should be considered a novel strategic direction in treating children with bronchial asthma. In the future, developing individualized therapeutic algorithms aimed at normalizing the microbiota to ensure long-term clinical remission, reduce disease relapse, and optimize immune approaches remains an urgent scientific and practical task.

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