RELATIONSHIP BETWEEN INFLAMMATORY CYTOKINES AND CLINICAL PHENOTYPES IN PEDIATRIC BRONCHIAL ASTHMA

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Annotation: *This research examines the characteristics of inflammatory cytokine profiles across different clinical phenotypes of bronchial asthma. The study provides scientifically grounded analysis of Th2-high and Th2-low phenotypes, eosinophilic and neutrophilic inflammation, interleukin dynamics, biomarker diagnostics, and phenotype-associated therapeutic strategies. The findings serve as a foundation for developing personalized treatment approaches.*

Keywords: *bronchial asthma, inflammatory cytokines, clinical phenotype, eosinophilic inflammation, neutrophilic inflammation, interleukin-5, interleukin-13, Th2-high phenotype, fractional exhaled nitric oxide, phenotype-associated therapy.*

Bronchial asthma represents one of the most prevalent chronic respiratory diseases among children, affecting ten to fifteen percent of children aged six to eighteen years worldwide. In the Republic of Uzbekistan, pediatric bronchial asthma prevalence comprises eight to twelve percent and has increased by twenty-five to thirty percent over the past decade. The heterogeneous nature of the disease, diverse clinical manifestations, variable treatment responses, and differing long-term prognoses necessitate classification of bronchial asthma into distinct phenotypes. Contemporary molecular biological research demonstrates that inflammatory cytokine profiles determine clinical phenotypes and possess fundamental importance in selecting

therapeutic strategies. Understanding cytokine network characteristics constitutes the foundation of personalized medicine.

Clinical phenotypes of bronchial asthma are divided into two principal groups based on inflammatory mechanisms: Th2-high and Th2-low phenotypes. The Th2-high phenotype occurs in sixty-five to seventy percent of pediatric cases and is characterized by classical allergic inflammation. In this phenotype, concentrations of interleukin-4, interleukin-5, and interleukin-13 increase significantly. Interleukin-5 levels measure three to five picograms per milliliter in healthy children but reach forty-five to sixty-five picograms per milliliter in the Th2-high phenotype. Interleukin-13 stimulates bronchial hyperreactivity, increases mucus production three to fourfold, and leads to subepithelial fibrosis development. Eosinophilic inflammation constitutes the primary characteristic of the Th2-high phenotype. Peripheral blood eosinophil counts reach three hundred to six hundred cells per microliter, representing four to six times above normal values. Sputum eosinophils exceed three percent and comprise twenty-five to thirty-five percent in severe cases. Major basic protein, eosinophil cationic protein, and eosinophil peroxidase released from eosinophil granules cause epithelial cell damage and bronchial hyperreactivity. Serum eosinophil cationic protein concentration measures thirty to fifty micrograms per liter. This indicator reflects inflammatory activity and demonstrates seventy-five to eighty percent diagnostic accuracy in assessing asthma control. The Th2-low phenotype occurs in thirty to thirty-five percent of cases and is characterized by neutrophilic or paucigranulocytic inflammation. In the neutrophilic phenotype, interferon gamma, interleukin-17, and interleukin-8 levels increase. Interleukin-17 concentration reaches eighty-five to one hundred twenty picograms per milliliter and stimulates neutrophil migration into bronchial lumen. Neutrophils comprise sixty to seventy percent of sputum cells. This phenotype demonstrates poor response to inhaled corticosteroids and presents treatment challenges. Asthma control remains inadequate in forty to

forty-five percent of cases with this phenotype, with frequent exacerbations occurring four to six times annually.

Fractional exhaled nitric oxide concentration represents a noninvasive biomarker of eosinophilic inflammation. In healthy children, fractional exhaled nitric oxide measures five to twenty parts per billion, whereas in the Th2-high phenotype it reaches thirty-five to seventy parts per billion. Values exceeding fifty parts per billion indicate high eosinophilic inflammatory activity and constitute an indication for increasing inhaled corticosteroid dosage. In the neutrophilic phenotype, fractional exhaled nitric oxide remains within normal limits or slightly elevated, measuring fifteen to twenty-five parts per billion. Periostin, a protein produced under interleukin-13 influence, participates in subepithelial fibrosis and bronchial remodeling processes. Serum periostin concentration reaches eighty to one hundred twenty nanograms per milliliter in severe asthma, representing three to four times above normal values. Elevated periostin levels assist in identifying patients who respond favorably to anti-interleukin-5 and anti-interleukin-13 therapy. Clinical trials demonstrate that biological therapy effectiveness reaches sixty to sixty-five percent in patients with periostin levels exceeding fifty nanograms per milliliter. Phenotype-associated therapy constitutes the fundamental principle of contemporary bronchial asthma management. In the Th2-high eosinophilic phenotype, inhaled corticosteroids demonstrate high effectiveness and reduce exacerbation frequency by seventy to seventy-five percent.

Inflammatory cytokine profiles in bronchial asthma determine clinical phenotypes and possess decisive importance in selecting therapeutic strategies. Distinguishing between eosinophilic and neutrophilic inflammatory mechanisms and implementing phenotype-associated treatment based on biomarkers constitutes the foundation of personalized medicine and significantly improves asthma control.

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