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"CLINICAL AND PATHOGENETIC FEATURES OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE AND ALLERGIC BRONCHITIS"

Musojonov Bakhodir Khayotjonovich
Central Asian Medical University, Fergana, Uzbekistan
musojonovbahodir@gmail.com

Annotation. Chronic obstructive pulmonary disease (COPD) and allergic bronchitis are among the most common inflammatory diseases of the lungs, with external environmental factors (smoking, harmful industrial dust, atmospheric pollution) and immuno-allergic mechanisms playing a key role in their development. Clinically, both diseases manifest with shortness of breath, cough, and sputum production. In terms of pathogenetic mechanisms, irreversible fibrotic changes of the bronchial walls and bronchospasm are predominant in COPD, whereas in allergic bronchitis, IgE-mediated sensitization, degranulation of cells involved in the allergic reaction, and disturbance of cytokine balance play the main role.

Keywords: Chronic obstructive pulmonary disease (COPD), allergic bronchitis, clinical features, pathogenesis, bronchial obstruction, fibrotic changes, environmental factors

The conducted analyses show that in chronic obstructive pulmonary disease (COPD), a persistent and continuous inflammatory process in the airways is considered the leading pathogenetic factor [1]. This process disrupts the morphological and functional state of the bronchial walls, leading to their structural damage. As a result, the elasticity of the bronchial walls gradually decreases, which causes narrowing of the airways and the formation of bronchial obstruction. During the progression of the disease, changes at the bronchial level also involve the alveolar structures [1,2]. At this stage, the destruction of the alveolar walls intensifies, and their normal structure and functional capacity are severely impaired. Consequently, gas exchange in the lungs becomes significantly disturbed. The transfer of oxygen from the alveoli into the blood and the elimination of carbon dioxide no longer occur at a normal level. This, in turn, leads to a deepening of chronic respiratory insufficiency in the body [2]. The processes characteristic of the pathogenesis of COPD gradually take on a progressive course. The persistence of inflammatory and destructive changes ensures that the disease acquires an irreversible nature. As a result, morphological damage in lung tissue not only limits functional capacity but also contributes to the transition of the disease to more severe clinical forms [3]

In cases of allergic bronchitis, the main clinical-pathogenetic feature is reversible bronchial obstruction. The clinical aspects of this process show that patients experience symptoms such as shortness of breath, cough, and sputum production, which intensify during certain periods [4]. The exacerbation of symptoms usually occurs upon repeated exposure to external allergens. Therefore, the clinical manifestations of the disease are directly related to environmental factors, and attacks often arise under the influence of seasonal changes, industrial dust, or household allergens.

In pathogenetic mechanisms, hypersensitivity reactions of the immune system play a leading role. In particular, sensitization mediated by IgE is one of the central links of allergic bronchitis, reflecting the organism's excessive sensitivity to external allergens. During this process, degranulation of mast cells occurs, i.e., they release biologically active mediators (histamine, cytokines, leukotrienes, and others)[3,4,5]. These mediators, in turn, activate inflammatory

reactions in the bronchial mucosa. As a result, edema develops in the bronchial mucosa, hypersecretion of the mucous glands increases, and spasm of bronchial muscle fibers occurs. These three factors — edema, hypersecretion, and muscle fiber spasm — together significantly impair bronchial patency[5]. However, these changes are usually temporary in nature and may be reversed when contact with the allergen ceases or appropriate therapeutic measures are applied.

Comparative analyses show that, despite differences in the clinical manifestations of chronic obstructive pulmonary disease (COPD) and allergic bronchitis, there are several common links in their pathogenesis. First of all, dysfunction of the immune system plays a leading role in both diseases. This dysfunction not only leads to deviations in the immune response but also contributes to the prolonged persistence of the inflammatory process [5,6].

Secondly, an imbalance in the oxidation–antioxidation system is observed in both pathologies. Oxidative stress intensifies as a result of proteins, lipids, and nucleic acids being damaged by free radicals. At the same time, insufficient functioning of the antioxidant defense system further aggravates the pathological process [6].

Thirdly, a significant pathogenic link is the disruption of cytokine balance. Due to the disturbance in the ratio between pro-inflammatory and anti-inflammatory cytokines, inflammatory mediators increase excessively [7]. This leads to swelling, secretion, and muscle spasms in the bronchial mucosa, as well as to the chronic course of the process.

Conclusion: Although COPD and allergic bronchitis share common clinical symptoms, there are significant differences in their mechanisms of development. In COPD, the inflammatory process is chronic and irreversible, leading to fibrotic changes in the bronchial walls and alveolar destruction. In allergic bronchitis, however, immune-allergic mechanisms prevail, resulting in reversible bronchial obstruction. COPD is associated with external harmful factors, while allergic bronchitis is linked to hereditary and hypersensitivity factors. In both diseases, disruption of the oxidation–antioxidation system and activation of inflammatory mediators are common features. Their comparative study is of great importance for the development of early diagnostic and effective treatment strategies.

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