PATHOGENESIS OF BRONCHIAL ASTHMA DEVELOPMENT AT THE PRESENT STAGE

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In recent years, there has been undoubted progress in both the diagnosis and treatment of bronchial asthma. In 1993, a program called The Global Initiative for Asthma (GINA) was developed, which combined the results of various diagnostic studies [13].

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A chronic inflammatory disease of the airways associated with their hyperresponsiveness leading to recurrent episodes of wheezing, dyspnea, chest tightness or coughing, especially at night or early in the morning. These symptoms are associated with widespread but variable airway obstruction, which is often reversible spontaneously or with treatment [3,4,5].

The development of bronchial asthma is based on chronic inflammation and bronchial hyperreactivity, defined as increased sensitivity of the respiratory tract to stimuli that are indifferent to healthy individuals. Specific bronchial hyperreactivity is understood as hypersensitivity of the bronchial tree to certain allergens, and nonspecific - to a variety of stimuli of non-allergenic nature. The

mechanism of airway hyperreactivity is based on the dysfunction of nervous regulation. The consequence of hyperreactivity is an increased tone of bronchial smooth muscle even outside the attack of the disease. In most patients bronchial hyperreactivity is present in the absence of symptoms of the disease and persists after prolonged anti-inflammatory therapy. Inflammation of the respiratory tract is a complex process that begins with damage to the epithelium, disturbance of microcirculation and involves all layers of the bronchi: epithelium, basal membrane, vessels and smooth muscle [11,12].

Airway inflammation in bronchial asthma is immune in nature, it is the result of release of immune mediators due to the realization of both IgE-dependent and T-lymphocyte-dependent mechanisms. In most cases, immune inflammation develops by IgE-dependent mechanism through the production of specific antibodies. Activated T-lymphocytes control the specific IgE response and exert a pro-inflammatory effect by increasing cytokine secretion. Cytokines, in turn, cause accumulation and activation of leukocytes, most notably eosinophils. Primary effector cells (mast cells, epithelial cells, macrophage cells) play an important role in forming an immediate response to antigen administration. Secondary effector cells (T-lymphocytes, fibroblasts, neutrophils, eosinophils, platelets) trigger the chronic inflammatory process, with which the long-term course of the disease is associated. Mediators of immune inflammation: histamine, leukotrienes, prostaglandins, adenosine, bradykinin, platelet and fibroblast growth factors toxically affect the surrounding tissues and lead to the development of smooth muscle spasm, increased vascular permeability of bronchial epithelium, bronchial mucosa edema, mucus hypersecretion, structural changes in the bronchi [1,2,3].

The most significant mediators in the formation of broncho obstruction caused by allergen inhalation are leukotrienes C4, D4, prostaglandins D2, F2, thromboxane A2. Leukotrienes C4, D4 are metabolites of arachidonic acid (lipooxygenase pathway of transformation), they are released within 5-10 minutes after activation of mast cells and basophils. Leukotrienes cause smooth muscle spasm, with the activity of leukotrienes being 100 times higher than that of histamine. Prostaglandins are also metabolites of arachidonic acid (cyclooxygenase pathway) and accumulate in the focus of inflammation later than leukotrienes and histamine.

Prostaglandins F2 and D2 cause contraction of bronchial musculature. Thromboxane A2 causes vasoconstriction of vessels, bronchioles and platelet aggregation. The interaction of inflammatory cells, mediators, and bronchial tissue leads to the formation of four types of obstruction. Acute bronchoconstriction is caused by smooth muscle spasm against the background of mediator release from mast cells (histamine, prostaglandins, leukotrienes) and direct action of triggers. Subacute occurs due to edema of the mucosa and bronchial wall with contraction of smooth muscle. Chronic develops due to mucus obturation of small bronchi. Irreversible is caused by remodeling of the bronchial wall (structural changes in the bronchial matrix) against the background of inadequate treatment [4,5,6].

The respiratory tract responds to inhaled antigen with two phases of reaction. Phase I is an early asthmatic reaction with rapid development of bronchospasm (due to release of mediators) 15-20 minutes after antigen exposure and resolution within one hour. This corresponds to type I allergic reactions (reactive type) and occurs in individuals with atopic bronchial asthma. Prophylactic administration of β2 -agonists blocks this bronchospasm. Phase II - secondary late asthmatic reaction occurs as a second wave of bronchoconstriction due to inflammation, bronchial edema (nonspecific hyperreactivity) within 4-8 h after antigen exposure and lasts up to 12 hours with relapses in the following days [7,8,9].

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