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BRONCHIAL ASTHMA: PATHOLOGICAL ASPECTS

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Bronchial asthma is a chronic recurrent inflammatory disease of the respiratory organs, the pathogenetic essence of which is bronchial hyperreactivity associated with immunopathological mechanisms, and the main clinical symptom of the disease is an asthma attack due to inflammatory edema of the bronchial mucosa, bronchospasm and mucus hypersecretion.

Airflow limitation in asthma is recurrent and caused by a variety of changes in the airway. These include:

Bronchoconstriction. In asthma, the dominant physiological event leading to clinical symptoms is airway narrowing and a subsequent interference with airflow. In acute exacerbations of asthma, bronchial smooth muscle contraction (bronchoconstriction) occurs quickly to narrow the airways in response to exposure to a variety of stimuli including allergens or irritants. Allergen-induced acute bronchoconstriction results from an IgE-dependent release of mediators from mast cells that includes histamine, tryptase, leukotrienes, and prostaglandins that directly contract airway smooth muscle Aspirin and other nonsteroidal anti-inflammatory drugs can also cause acute airflow obstruction in some patients, and evidence indicates that this non-IgE-dependent response also involves mediator release from airway cells. In addition, other stimuli (including exercise, cold air, and irritants) can cause acute airflow obstruction. The mechanisms regulating the airway response to these factors are less well defined, but the intensity of the response appears related to underlying airway inflammation. Stress may also play a role in precipitating asthma exacerbations. The mechanisms involved have yet to be established and may include enhanced generation of pro-inflammatory cytokines.

Airway edema. As the disease becomes more persistent and inflammation more progressive, other factors further limit airflow. These include edema, inflammation, mucus hypersecretion and the formation of inspissated mucus plugs, as well as structural changes including hypertrophy and hyperplasia of the airway smooth muscle. These latter changes may not respond to usual treatment.

Airway hyperresponsiveness. Airway hyperresponsiveness an exaggerated bronchoconstrictor response to a wide variety of stimuli is a major, but not necessarily unique, feature of asthma. The degree to which airway hyperresponsiveness can be defined by contractile responses to challenges with methacholine correlates with the clinical severity of asthma. The mechanisms influencing airway hyperresponsiveness are multiple and include inflammation, dysfunctional neuroregulation, and structural changes; inflammation appears to be a major factor in determining the degree of airway hyperresponsiveness. Treatment directed toward reducing inflammation can reduce airway hyperresponsiveness and improve asthma control.

Airway remodeling. In some persons who have asthma, airflow limitation may be only partially reversible. Permanent structural changes can occur in the airway; these are associated with a progressive loss of lung function that is not prevented by or fully reversible by current therapy. Airway remodeling involves an activation of many of the structural cells, with consequent permanent changes in the airway that increase airflow obstruction and airway responsiveness and render the patient less responsive to therapy.

Inconclusion, properly planned and implemented measures for the prevention of bronchial asthma are an effective means of preventing and treating this disease. Modern medical practice shows that the correct implementation of asthma prevention measures is often enough to cure the disease or to significantly reduce the patient's need for anti-asthma drugs.