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BRONCHIAL ASTHMA: MODERN CONCEPTS OF PATHOGENESIS, DIAGNOSIS AND PERSONALIZED TREATMENT (LITERATURE REVIEW)

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Abstract

Background: Bronchial asthma is a heterogeneous chronic inflammatory disease of the airways characterized by variable respiratory symptoms, bronchial hyperresponsiveness, and reversible or partially reversible airflow limitation. Despite significant advances in respiratory medicine, asthma remains an important global healthcare problem due to its high prevalence, frequent exacerbations, and substantial impact on patients' quality of life.

Objective: To review current scientific literature concerning the epidemiology, pathogenesis, clinical manifestations, diagnostic approaches, and modern treatment strategies for bronchial asthma, with particular emphasis on personalized and targeted therapy.

Materials and Methods: A narrative review of contemporary scientific publications, international clinical recommendations, and relevant literature on bronchial asthma was conducted. Particular attention was paid to publications addressing immunological mechanisms, airway remodeling, inflammatory biomarkers, pulmonary function assessment, inhaled corticosteroid therapy, and biological agents.

Results: Current evidence demonstrates that bronchial asthma is a multifactorial disease involving complex interactions between genetic predisposition, environmental exposures, airway epithelial dysfunction, immune mechanisms, and inflammatory mediators. Type 2 inflammation plays a major role in many asthma phenotypes and is associated with eosinophilia, increased immunoglobulin E, and elevated levels of interleukins IL-4, IL-5, and IL-13. Modern diagnostic approaches combine clinical assessment, spirometry, bronchodilator reversibility testing, and inflammatory biomarkers. Inhaled corticosteroid-containing regimens remain the foundation of treatment, while biological agents provide additional therapeutic opportunities for patients with severe asthma.

Conclusion: Effective asthma management requires early diagnosis, objective assessment of lung function, identification of inflammatory phenotypes, and individualized treatment. The development of precision medicine and targeted biological therapies represents an important direction for improving disease control and reducing asthma-related complications.

Keywords: bronchial asthma, chronic airway inflammation, inhaled corticosteroids, biological therapy, personalized medicine.

Bronchial asthma is a complex, chronic inflammatory disorder of the respiratory system characterized by persistent airway inflammation, increased bronchial responsiveness, and variable degrees of expiratory airflow limitation. The disease manifests through recurrent episodes of wheezing, dyspnea, chest tightness, and coughing, which may vary in frequency and severity over time[1-3]. These clinical manifestations are often associated with exposure to allergens, environmental pollutants, respiratory infections, physical activity, and other triggering factors.The development of bronchial asthma is influenced by a combination of genetic susceptibility,

immunological abnormalities, and environmental conditions. Its pathophysiological mechanisms involve interactions between airway epithelial cells, inflammatory mediators, eosinophils, mast cells, lymphocytes, and bronchial smooth muscle. Persistent inflammatory activity may contribute to structural alterations in the bronchial wall, increased mucus secretion, and progressive airway remodeling.Although substantial progress has been achieved in understanding the mechanisms of asthma and developing effective therapeutic approaches, the disease continues to represent a major public health concern worldwide[4]. Recurrent exacerbations, inadequate symptom control, impaired pulmonary function, and treatment-related complications can significantly affect patients' daily activities and overall quality of life. Therefore, early diagnosis, accurate assessment of disease severity, identification of inflammatory phenotypes, and implementation of individualized treatment strategies remain essential components of modern asthma management. The present literature review aims to provide a comprehensive analysis of contemporary scientific evidence regarding the epidemiology, etiological factors, pathophysiological mechanisms, clinical characteristics, diagnostic criteria, and therapeutic approaches to bronchial asthma. Particular attention is given to the heterogeneous nature of the disease, which is determined by the interaction of genetic predisposition, environmental influences, immunological abnormalities, and chronic inflammatory processes within the respiratory tract[5].An important aspect of this review is the investigation of the immunopathological mechanisms underlying bronchial asthma, including the involvement of T-helper 2 (Th2) lymphocytes, eosinophils, mast cells, immunoglobulin E (IgE), and pro-inflammatory cytokines such as interleukin-4 (IL-4), interleukin-5 (IL-5), and interleukin-13 (IL-13). The contribution of airway epithelial dysfunction, oxidative stress, bronchial hyperresponsiveness, and structural remodeling to disease progression is also considered.

The review further examines modern diagnostic approaches used to establish the diagnosis and evaluate disease activity, including spirometry, bronchodilator reversibility testing, peak expiratory flow monitoring, fractional exhaled nitric oxide (FeNO), blood eosinophil counts, and immunological investigations. The importance of combining clinical assessment with objective functional and laboratory parameters is emphasized to improve diagnostic accuracy and identify distinct inflammatory phenotypes.Special emphasis is placed on contemporary treatment strategies, including inhaled corticosteroids, long-acting bronchodilators, leukotriene receptor antagonists, and targeted biological agents. Particular consideration is given to monoclonal antibodies directed against IgE, IL-5, IL-5 receptor, IL-4 receptor alpha, and thymic stromal lymphopoietin (TSLP), which have expanded therapeutic possibilities for patients with severe asthma.Furthermore, the review explores the emerging role of personalized medicine in respiratory practice[7]. The identification of specific asthma phenotypes and endotypes, together with the assessment of inflammatory biomarkers, may facilitate the selection of the most appropriate therapeutic interventions for individual patients. This approach is particularly relevant for individuals with uncontrolled or severe asthma who demonstrate an inadequate response to conventional treatment.

A comprehensive narrative literature review was conducted to evaluate contemporary scientific evidence on the epidemiology, pathogenesis, clinical manifestations, diagnosis, and treatment of bronchial asthma. Relevant scientific publications, peer-reviewed journal articles, international clinical guidelines, and consensus recommendations were analyzed to identify recent advances in the understanding and management of this chronic respiratory disorder[8].Particular emphasis was placed on studies investigating the immunological and molecular mechanisms of asthma development, including type 2 inflammatory responses, eosinophilic activity, cytokine-mediated pathways, immunoglobulin E production, and airway epithelial dysfunction. In addition, publications addressing bronchial hyperresponsiveness, oxidative stress, airway remodeling, and

the relationship between persistent inflammation and progressive impairment of pulmonary function were reviewed.The literature analysis also included contemporary diagnostic approaches, such as spirometry, bronchodilator reversibility testing, peak expiratory flow monitoring, fractional exhaled nitric oxide (FeNO), peripheral blood eosinophil counts, and immunological biomarkers. Special attention was given to the clinical significance of these parameters in identifying asthma phenotypes and evaluating disease control.Therapeutic publications were examined with particular reference to inhaled corticosteroids, long-acting bronchodilators, leukotriene receptor antagonists, and targeted biological agents, including anti-IgE, anti-IL-5, anti-IL-5R, anti-IL-4Rα, and anti-TSLP therapies[9].

The reviewed evidence was comparatively analyzed and synthesized to identify major developments in personalized asthma management, existing limitations in clinical practice, and promising directions for future research. As this study represents a narrative rather than a systematic review, no formal meta-analysis or quantitative pooling of results was performed.The analysis of contemporary scientific literature indicates that bronchial asthma is a multifactorial respiratory disorder resulting from the interaction of genetic susceptibility, environmental factors, epithelial barrier abnormalities, immunological disturbances, and various inflammatory pathways. These mechanisms contribute to chronic airway inflammation, bronchial hyperresponsiveness, mucus hypersecretion, and variable airflow obstruction.One of the most extensively investigated mechanisms is type 2 (T2) inflammation, which plays a significant role in the pathogenesis of several asthma phenotypes. This inflammatory response is mediated by T-helper 2 lymphocytes, group 2 innate lymphoid cells, eosinophils, and mast cells. The production of cytokines, particularly interleukin-4 (IL-4), interleukin-5 (IL-5), and interleukin-13 (IL-13), contributes to allergic sensitization, eosinophilic inflammation, increased immunoglobulin E (IgE) synthesis, and airway hyperresponsiveness. These processes are associated with increased mucus production and structural changes in the bronchial wall. However, it is important to recognize that not all patients with asthma demonstrate a predominantly type 2 inflammatory profile.The reviewed evidence also highlights the importance of airway epithelial dysfunction and oxidative stress in maintaining chronic inflammatory activity. Damage to epithelial cells may promote the release of inflammatory mediators, including thymic stromal lymphopoietin (TSLP), IL-25, and IL-33, which activate downstream immune responses. Persistent inflammation may subsequently contribute to airway remodeling, characterized by subepithelial fibrosis, smooth muscle hypertrophy, goblet cell hyperplasia, and thickening of the bronchial wallx5ъ.Regarding diagnostic strategies, contemporary recommendations emphasize the importance of combining clinical evaluation with objective assessment of pulmonary function. Spirometry remains the principal functional investigation for identifying variable expiratory airflow limitation. Bronchodilator reversibility testing provides additional evidence supporting the diagnosisx[7]. Furthermore, fractional exhaled nitric oxide (FeNO), peripheral blood eosinophil counts, and serum IgE measurements may help characterize inflammatory phenotypes and predict treatment response in selected patients.In terms of pharmacological management, inhaled corticosteroid-containing regimens remain the cornerstone of asthma treatment. Their anti-inflammatory effects contribute to improved symptom control, reduced bronchial hyperresponsiveness, and prevention of exacerbations. Combination therapy with long-acting beta-2 agonists is recommended when additional disease control is required. Other therapeutic options, including leukotriene receptor antagonists, may be considered in selected clinical situations.

A particularly important development in modern respiratory medicine is the introduction of targeted biological therapies for patients with severe asthma. Monoclonal antibodies directed

against IgE, IL-5, IL-5 receptors, IL-4 receptor alpha, and TSLP target specific inflammatory pathways involved in asthma pathogenesis. Clinical evidence indicates that appropriately selected biological therapies can reduce exacerbation frequency, improve pulmonary function, enhance symptom control, and decrease dependence on systemic corticosteroids.

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