SEVERE BRONCHIAL ASTHMA: DEFINITION, EVALUATION OF THE INFLAMMATORY PHENOTYPE, NEW PERSPECTIVES OF BIOLOGICAL THERAPY OF PATIENTS

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Abstract

According to general estimates, from 3–5 % and up to 10 % of patients with asthma have a severe course of the disease, which often continues to remain uncontrolled, despite the maximum use of inhaled corticosteroids and additional controller drugs. Severe asthma creates a large physical, mental, emotional, social and economic burden for patients and is often associated with multimorbidity. The heterogeneity of asthma drives research aimed at defining asthma phenotypes that ultimately reflect different underlying disease mechanisms, including pathogenetic processes and response to environmental factors. The clinical phenotypes of asthma are closely related to the underlying inflammatory processes in this disease and therefore can be divided into 2 main inflammatory phenotypes determined by the dominant immunological pathways that determine the pathology of bronchial asthma: eosinophilic and non-eosinophilic.

Phenotyping patients is important for the differential diagnosis of asthma, risk assessment, treatment selection, and monitoring response to treatment. Accurate identification of the phenotype is particularly important for the selection of a biological drug for the treatment of severe asthma and for predicting the response to treatment with it. But these phenotypes are not fixed and can change over time in response to new environmental triggers (viral infections, inhaled allergens, smoking, air pollution). The main cytokines in the T2 type of inflammation in the airways, which is characteristic of severe asthma, are IL-4, IL-5 and IL- 13, IL-17, but an equally significant role in this process is also played by alarmins (inflammation mediators produced, in particular, cells of the bronchial epithelium), which include IL-33, IL-25 and thymic stromal lymphopoietin (TSLP).

Clinical features of asthma associated with TSLP include: 1) degree of severity; 2) risk and frequency of exacerbations; 3) the degree of lung function decline; 4) decrease in the patient's response to glucocorticosteroids; 5) decrease in immune response and susceptibility to acute respiratory viral infections; 6) potential remodulation of the respiratory tract; 7) bronchial hyperreactivity and mucus blockage. Therefore, the role of TSLP in the pathogenesis of severe asthma is very significant, so its therapeutic effect (anti-TSLP therapy using tezepelumab) can be considered a new promising direction of biological therapy for patients with bronchial asthma, especially in its severe form.

Key words: bronchial asthma, severe form, type 2 inflammation, biomarkers of inflammation, thymic stromal lymphopoietin, biological therapy, tezepelumab.

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