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PREDICTION OF THE DEVELOPMENT OF BRONCHIAL ASTHMA IN CHILDREN WHO HAD ACUTE OBSTRUCTIVE BRONCHITIS AT AN EARLY AGE

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Abstract:

The article presents the results of treatment of acute obstructive bronchitis (AOB) in young children. It was found that hypoxic-ischemic lesions of the central nervous system increase the incidence of AOB by 2.3 times. A method for predicting the risk of developing bronchial asthma in children suffering from AOB was developed.

Keywords: acute obstructive bronchitis, bronchial asthma, prognosis. pharmacotherapy, inhaled glucocorticosteroids.

Introduction

Acute obstructive bronchitis in young children remains particularly relevant in modern pediatrics, despite scientific and practical achievements in the field of etiopathogenesis of this disease and the introduction of modern treatment methods into medical practice [1, 2, 3]. The prevalence of obstructive bronchitis, according to a number of authors, ranges from 15% to 50% [1, 2]. In infants, the incidence of acute obstructive bronchitis reaches 90% [5, 6]. Most often, AOB manifests itself in children of the first years of life, which is due to the multifunctional features of the respiratory organs at this age: narrow airways, insufficient elasticity of the bronchial tree cartilages, insufficient development of the smooth muscles of the bronchi, which is one of the confirmations of the theory of relative immaturity of all organs and systems in young children [7, 4]. A special place in the formation of increased bronchoreactivity in infants and the development of acute obstructive pulmonary disease is occupied by neuroreflex mechanisms, the basis of which are dysfunctions of

the autonomic nervous system, which causes bronchial spasms, vasodilation, disturbance of the peristaltic rhythm, which is accompanied by the development of pastosity, edema, hyperproduction of thick secretion, gastroesophageal reflux, intestinal dyskinesia and is a provoking factor for the development of acute obstructive pulmonary disease. [3, 5]. The development of acute obstructive pulmonary disease in young children is facilitated by hypoxic-ischemic disorders of the central nervous system, as a consequence of perinatal hypoxia. These disorders are manifested by a syndrome of movement disorders, a syndrome of vegetative-visceral dysfunctions, hydrocephalic syndrome, convulsive syndrome, and a delay in the rate of psychomotor development. Analyzing the incidence of acute obstructive bronchitis in children in the first half of the year, it was found that in the group with CNS disorders there were 26.4% of them, and in the group without CNS disorders - 11.3%, which was a difference of 2.3 times. At the age of 9 to 12 months, against the background of CNS disorders, acute obstructive bronchitis was observed in 14.1% of patients versus 6.6% in children without CNS disorders, which is 2.1 times more often. Thus, our studies have shown that children of the first year of life who have the consequences of hypoxic-ischemic damage to the central nervous system are more likely to suffer from acute obstructive bronchitis (almost 2 times more). Recurrent course of acute obstructive bronchitis in young children, their anatomical and physiological characteristics, ever-increasing allergization of the modern world, the consequences of perinatal hypoxia are the pathophysiological complex that contributes to the further development of bronchial asthma in children.

Bronchial asthma (BA) in children is one of the most common allergic diseases. In recent years, there has been a worldwide trend towards an increase in the incidence of asthma in children, including young children, and a more severe course of asthma. In this regard, the main attention of specialists is focused on new methods of diagnosing this pathology and developing an optimal treatment regimen for the disease. The review presents the main approaches to the treatment of asthma, taking into account the patient's age and the degree of disease control. Optimal combinations of drugs for basic therapy and the most promising areas of asthma treatment are considered. Constant attention to the problem of asthma is also due to the fact that with insufficiently effective treatment, frequent exacerbations of the disease, the quality of life of patients decreases, limiting their vital functions. Severe forms of asthma are accompanied by dysfunction of not only the respiratory organs, but also other body systems.

Disability develops in 7% of officially registered children with bronchial asthma. Currently, this problem has acquired not only medical, but also socio-economic significance. Based on the pathogenesis of bronchial asthma, modern therapy is aimed at eliminating allergic inflammation of the bronchial mucosa, reducing bronchial hyperreactivity, restoring bronchial patency and preventing structural reorganization of the bronchial wall. Untimely diagnosis and inadequate therapy are the main causes of severe disease and mortality in patients with bronchial asthma. The choice of treatment is determined by the severity of the course, the degree of control and the period of bronchial asthma. However, in any case, an individual approach is necessary in choosing the means and methods of treatment. The main defining definition of bronchial asthma is chronic inflammation. The definitions that exist to date are descriptive: for example, specific cell types (mast cells, eosinophils, etc.), the time of symptom onset (especially at night or early in the morning), reversibility (partial) or triggers (viral infection, physical activity and exposure to allergens). However, the role and relative contribution of each of these additional elements can be debated: they are not absolute (exclusive) features of asthma. The causal relationships between chronic inflammation, airway hyperreactivity and asthma symptoms remain to be elucidated. Current asthma management guidelines do not differentiate between adult and pediatric asthma. Asthma is therefore a chronic inflammatory disorder associated with transient and variable airway obstruction and airway hyperreactivity, characterized by recurrent episodes of wheezing, cough, shortness of breath and chest tightness. Chronic inflammation, airway hyperreactivity and the underlying structural changes (remodeling)are associated with an increase in the number of mast

cells, eosinophils, lymphocytes, macrophages, dendritic and other cell types. Inflammatory and structural (epithelial and smooth muscle) cells jointly produce cytokines, chemokines and cysteine leukotrienes - various mediators that enhance the inflammatory response and contribute to the narrowing and hyperreactivity of the airways [11]. Airway hyperreactivity is associated with excessive contraction of smooth muscles in response to non-specific irritants and viral infections, and in the case of patients suffering from atopy - in response to exposure to specific allergens. Neural mechanisms initiated by inflammation are highly likely to contribute to the development of airway hyperreactivity [12]. Airway obstruction is initiated by a combination of edema, infiltration, increased mucus secretion, smooth muscle contraction and epithelial desquamation. These changes are largely reversible; However, as the disease progresses, airway narrowing may become progressive and permanent. Structural changes associated with airway remodeling include smooth muscle hyperplasia, hyperemia with increased vascularity of the subepithelial tissue, thickening of the basement membrane and subepithelial deposition of various structural proteins, as well as loss of normal airway compliance [13]. Remodeling, initially described in detail in adult asthma, is also present in children, at least in severe persistent forms [14, 15]. At present, despite the advances in understanding the mechanisms of asthma pathogenesis, unfortunately, allergists do not have successful prevention programs in their arsenal. Conflicting results of the few interventional observations do not allow the formulation of clear recommendations. Many guidelines mention infections, microbial exposure, stress, airborne pollutants, allergens and tobacco smoke as possible triggers. High levels of specific antibodies (sIgE), particularly in early life, are a strong risk factor for asthma, especially in developed countries. Potential preventive measures such as a hypoallergenic diet during pregnancy or vitamin D supplementation [16] require confirmation; the use of drugs that could mobilize immune mechanisms for primary prevention of asthma (oral bacterial immunomodulators) is also being actively investigated [17]. At present, the only proven modifiable environmental factor that can be confidently recommended for primary prevention of asthma is limiting exposure to tobacco smoke during pregnancy and in the neonate (level of evidence B).Chemokines and cysteine leukotrienes are various mediators that enhance the inflammatory response and contribute to airway narrowing and hyperreactivity [11]. Airway hyperreactivity is associated with excessive smooth muscle contraction in response to nonspecific irritants and viral infections, and in the case of atopic patients, in response to specific allergens. Neural mechanisms initiated by inflammation are also likely to contribute to the development of airway hyperreactivity [12]. Airway obstruction is initiated by a combination of edema, infiltration, increased mucus secretion, smooth muscle contraction, and epithelial shedding. These changes are largely reversible; however, as the disease progresses, airway narrowing may become progressive and permanent. Structural changes associated with airway remodeling include smooth muscle hyperplasia, hyperemia with increased vascularity of the subepithelial tissue, thickening of the basement membrane and subepithelial deposition of various structural proteins, as well as loss of normal airway compliance [13]. Remodeling, initially described in detail in adult asthma, is also present in children, at least in severe persistent forms [14, 15]. At present, despite the advances in understanding the mechanisms of asthma pathogenesis, unfortunately, allergists do not have successful prevention programs in their arsenal. Conflicting results of the few interventional observations do not allow the formulation of clear recommendations. Many guidelines mention infections, microbial exposure, stress, airborne pollutants, allergens and tobacco smoke as possible triggers. High levels of specific antibodies (sIgE), particularly in early life, are a serious risk factor for the development of asthma, especially in developed countries. Potential preventive measures such as a hypoallergenic diet during pregnancy or vitamin D supplementation [16] require confirmation; the use of drugs that could mobilize immune mechanisms for primary prevention of asthma (oral bacterial immunomodulators) is also being actively investigated [17]. At present, the only proven modifiable environmental factor that can be confidently recommended for primary prevention of asthma is limiting exposure to tobacco smoke during pregnancy and in the neonate (level of evidence B).Chemokines and cysteine leukotrienes are various mediators that enhance the

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Remodeling, initially described in detail in adult asthma, is also present in children, at least in severe persistent asthma [14, 15]. The key point in the diagnosis of bronchial asthma in children is a history of recurrent episodes of wheezing (usually more than three). The goal of asthma treatment is to achieve stable remission and high quality of life in all patients, regardless of the severity of the disease. The onset of symptoms and the development of asthma exacerbations are provoked by various specific and non-specific irritants [18]. Naturally, limiting their impact on the patient's body can have an effect on reducing the activity of the disease. All elimination measures should be personalized and are cost-effective and effective only in the case of a thorough preliminary allergological examination, including anamnesis to assess the clinical significance, skin testing and/or determination of the sIgE titer (evidence level B). Indoor allergens (dust mites, pets, cockroaches and mold fungi) are considered the main triggers and are the target of specific interventions (evidence level B–D depending on the allergen and procedure). Complete elimination of allergens is usually impossible, and some measures entail significant costs and inconvenience and often have only limited effectiveness. Outdoor allergens are even more difficult to manage, and the only recommended approach may be staying indoors for certain periods of time (for pollen sensitization). Stopping smoking and limiting exposure to tobacco smoke in children with asthma has a positive effect (evidence level C).

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