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The Role of Complex Perinatal Anamnesis in the Formation of Bronchial Asthma in Children (Literature Review)

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Annotation. Bronchial asthma is widespread and tends to steadily increase in incidence in children, shifting the onset to an earlier age. It was revealed that the majority of children with bronchial asthma have a history of perinatal damage to the central nervous system. However, it should be noted that the mechanisms of influence of perinatal damage to the central nervous system on the formation of bronchial asthma are numerous and have not yet been sufficiently studied. All this proves the need for preventive measures for the physiological course of pregnancy in women. Since chronic hypoxia, acute asphyxia during childbirth and, especially, the resulting premature birth contribute to the development of not only neurological pathology, but also multiple organ disorders. Moreover, in children who have suffered perinatal damage to the nervous system, somatic pathology, including bronchial asthma, is much more severe.

Key words: bronchial asthma, children, central nervous system, perinatal lesions, anamnesis

Modern ideas about the pathogenesis of asthma associate the development of the disease with congenital abnormalities of neuroimmune regulation, leading to inflammatory and allergic components with bronchial hyperreactivity [2,3]. The diathesis-stress model of AD considers the basis of the disease to be early damage to the hypothalamic-diencephalic structures, leading to disruption of autonomic-humoral regulation, especially manifested in the emotional and motivational aspects of behavior [4,6]. The psychoanalytic concept adds to the indicated pathogenesis personal problems characteristic of AD patients - alexithymia, deficiency of reflection, anticipatory failure. As a result, by adolescence and adulthood, a characteristic psychosomatic (anxious) profile of a patient with AD is formed [11,13].

Recently, special attention has been paid to the pathology of the bronchopulmonary system, including such a severe chronic disease as bronchial asthma in conjunction with perinatal damage, which contributes to the frequent development of bronchial obstruction in children of the first years of life [9,20]. The influence of perinatal neuropathology on the state of the respiratory system in children, which requires a complex of rehabilitation measures at different age periods, is beyond doubt, but the mechanisms of the formation of bronchopulmonary pathology still remain poorly understood. Domestic and foreign literature describes various forms of bronchial asthma (physical effort, aspirin, bronchial asthma in the elderly), but there is relatively little data on the characteristics of the course of this disease in premature infants. With the development of the material and technical base in medicine, it has become possible to care for children born with extremely low body weight. Such children, like premature babies in general, often require long-term artificial pulmonary ventilation (ALV), which cannot go unnoticed by the bronchopulmonary system in the future. In premature babies, whose lungs are not fully formed, the likelihood of bronchopulmonary dysplasia is high, ventilation is impaired, surfactant is restored slowly, the immune system is not mature - all this contributes to the relative ease of developing asthma [11]. In severe perinatal hypoxia, the bronchopulmonary system itself is affected quite often: circulatory

disorders lead to the development of adult-type respiratory distress syndrome and secondary surfactant deficiency. Children who have suffered respiratory pathology in the neonatal period subsequently suffer from pneumonia, bronchitis, and acute respiratory diseases 2-3 times more often, and the highest incidence was noted in children who underwent resuscitation measures [26,28]. Perinatal damage to the central nervous system increases the risk of developing bronchial asthma in children of preschool age by 3.4 times. The formation of bronchial asthma in preschool children, along with hereditary and constitutional predisposition to atopy, is significantly influenced by disturbances in the mechanisms of neuro-vegetative regulation of the cardiorespiratory system, resulting from adverse nonspecific influences in perinatal ontogenesis [14]. This is of particular importance in view of the steady growth of neurological pathology of the perinatal period. The most pressing issue in perinatal neurology is hypoxic damage to the brain of the fetus and newborn, caused by chronic intrauterine fetal hypoxia and acute asphyxia during childbirth [16,20]. The results of modern studies have proven the relationship between clinical syndromes of central nervous system damage during the neonatal period and their consequences [17,19]. It has been established that intrauterine sensitization of the fetus, which can occur during gestosis of pregnancy, acute viral or bacterial infections, exacerbation of chronic inflammatory diseases in the mother, contributes to the early onset of asthma in children [1,3]. It has been proven that sensitization of a child can occur in utero through the entry of allergens in the mother's IgG, which carry food allergens, through the fetal skin, respiratory tract and through the entry of amniotic fluid into the fetal gastrointestinal tract during swallowing movements, as well as the ability of the fetus to synthesize its own IgE already in early pregnancy. Chronic intrauterine fetal hypoxia can enhance the entry of allergens into the fetal body, as well as switch the TH1 to TH2 immune response through increased synthesis of the corresponding cytokines [5,7,15,17].

In addition, it is known that intrauterine hypoxia can influence the formation and course of asthma by increasing the hyperreactivity of the bronchial tree through disruption of the mechanisms of regulation of muscle tone and bronchial innervation. This happens because in children who have suffered chronic hypoxia in utero or acute asphyxia intrapartum, there are profound disturbances in the activity of the neuroendocrine system, caused by dysfunction of the centers of the hypothalamic level. The vulnerability of neuroendocrine centers is explained by topographical features and strong sensitivity to hypoxia of the hypothalamus and brain stem. Experimental data indicate that an imbalance in the neuroendocrine system in the neonatal period leaves long-term consequences in the form of defects in anti-stress defense and a tendency to the formation of autonomic disorders. The resulting autonomic changes are the cause of dysregulation of muscle tone and bronchial innervation [15,19,20]. Perinatal hypoxic lesions of the central nervous system lead to functional instability of the cortical-subcortical and spinal structures of the brain that regulate the respiratory complex. In addition, respiratory and cardiovascular disorders characteristic of the perinatal period, disorders of cerebral status, hemorrhagic and hematological disorders in the fetus and newborn, prematurity, morphofunctional immaturity, intrauterine growth retardation and infectious diseases specific to the perinatal period disrupt the neurogenic regulation of smooth tissue tone. bronchial muscles and damage the ciliated epithelium. The consequence of this is increased permeability of the mucous membrane to allergens, which triggers sensitization mechanisms, leads to an increase in specific and nonspecific bronchial hyperreactivity and ultimately causes the development of BA [19,20,21]. Thus, neurological and psychopathological disorders that develop as a result of perinatal damage to the central nervous system naturally close the vicious circles of the pathogenesis of AD, limiting the patient’s adaptive capabilities throughout life. In this regard, the study of clinical and dynamic features of psychoneurological disorders in children with AD is a very urgent task, as it can help improve methods of prevention and treatment of this disease.

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