ETHIOPATHOGENESIS IN CHILDREN'S EPILEPSIA: MODERN VIEWS AND THE CONCEPT OF EPILEPTIC NEURON NETWORKS

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Annotation: *Epilepsy in childhood is a complex, multifactorial neurological disease in terms of etiology and pathogenesis. In recent years, views on the mechanisms of epilepsy's origin have significantly expanded, with a transition from the classical concept of "epilepsy focus" to the concept of "epilepsy neural networks." In this literature review, the main etiological factors of childhood epilepsy, the stages of epileptogenesis, neurophysiological and neurochemical mechanisms, and the patterns of distribution of epileptic activity were analyzed. The results of the analysis show that the epileptic process is closely related to the stages of development of the central nervous system, and epilepsy, which begins at an early age, has a profound impact on the formation of neural networks.*

Keywords: *epilepsy, childhood epilepsy, etiopathogenesis, epileptogenesis, epileptic neural networks, GABA, glutamate.*

Epilepsy is one of the most complex and multifaceted diseases of the central nervous system, the development of which occurs as a result of the interaction of various etiological factors and pathogenetic mechanisms. In childhood, epilepsy has special scientific and clinical significance, since brain structures and neural networks are not yet fully developed. Therefore, epileptic activity is directly related not only to seizures, but also to the cognitive, emotional, and behavioral development of the child [4,21].

In recent decades, scientific views on the etiopathogenesis of epilepsy have fundamentally changed. If previously epilepsy was mainly explained by a local epileptic focus, now the epileptic process is considered as a complex pathological neural network, forming on the scale of the entire brain. This approach makes it possible to explain the clinical diversity of childhood epilepsy and different responses to treatment [2,19,22].

Etiological factors of childhood epilepsy

The etiology of childhood epilepsy is multifactorial, and many researchers emphasize the predominant role of genetic factors. According to the analysis of the literature, about half or more of children with epilepsy are of a genetic nature. In this case, not only traditional hereditary mechanisms, but also "de novo" mutations play an important role. The role of such mutations is increasingly being determined, especially in epileptic encephalopathies [8,15,17].

Structural causes follow genetic factors. Perinatal hypoxic-ischemic lesions, brain developmental anomalies, traumatic brain injuries, and tumor processes contribute to the formation of epileptic activity. In these cases, epilepsy often has a symptomatic course and is characterized by a predisposition to pharmacoresistant forms [1,3,14].

Metabolic, immune, and infectious factors also play an important role in the etiology of childhood epilepsy. In some metabolic disorders, epileptic seizures can be the initial and leading sign of the disease. Epilepsies associated with autoimmune processes have been identified as a separate etiological group in recent years, which expands the possibilities of targeted immunotherapy [5,6,18].

Epileptogenesis: a phased pathological process

The formation of the epileptic process occurs through a complex and prolonged process called epileptogenesis. Epileptogenesis can continue not only before the onset of seizures, but also after the formation of clinical epilepsy. During this process, structural and functional changes occur in brain tissue.

One of the main mechanisms of epileptogenesis is the disruption of the balance between the excitatory and inhibitory systems. From a neurochemical point of view, this condition is manifested by a decrease in GABA-ergic inhibition and an increase in the activity of glutamate, which is the main stimulating mediator of the central nervous system. This imbalance leads to the transition of neurons to a pathologically hyperexcitable state [9,10].

This process is especially important in childhood, as brain structures are not yet fully developed. The peculiarities of the development of the hippocampus, thalamus, and cortical structures contribute to the rapid spread and generalization of epileptic activity.

From epileptic focus to epileptic neural networks

In modern epileptology, it is becoming increasingly clear that explaining epilepsy only by one localized epileptic focus is insufficient. Recent studies show that epileptic activity is carried out through a pathological neural network, which includes several anatomically and functionally connected brain structures [7,11,13].

These epileptic neural networks can involve cortical and subcortical structures, including the hippocampus, thalamus, and brainstem. Activation of one structure leads to synchronous activation of other components in the network. It is this mechanism that explains the various clinical forms of epileptic seizures and their generalization.

In childhood epilepsy, the concept of neural networks is of particular importance, since neural connections in the developing brain have a high degree of plasticity. This leads, on the one hand, to the rapid spread of the epileptic process, and on the other hand, to the possibility of reorganizing pathological networks through proper and early treatment [12,16,22].

Antiepileptic defense mechanisms and their disorders

The central nervous system has natural defense mechanisms that limit the spread of epileptic activity. These mechanisms are realized through the inhibitory effect of neural networks located around the epileptic focus. However, insufficient functioning of this "anti-epileptic barrier" leads to the expansion of epileptic activity.

In children, these defense systems are not yet fully formed, which may explain the frequent generalization and severe clinical course of epileptic seizures. If epileptic activity

is not controlled for a long time, new pathological networks are formed, and secondary degenerative changes occur in brain tissue [20].

Clinical and prognostic significance of etiopathogenesis

A deep understanding of the etiopathogenesis of childhood epilepsy is of great importance for clinical practice. Determination of the etiological factor and mechanisms of epileptogenesis allows not only to make an accurate diagnosis, but also to choose an effective treatment strategy. In particular, early detection of genetic and structural epilepsy plays an important role in the prevention of pharmacoresistant forms [4,7].

In addition, the concept of epileptic neural networks helps to explain the long-term consequences of epilepsy in children, including cognitive and emotional disorders. The duration and prevalence of epileptic activity directly affect a child's intellectual development [3,14,15].

Final considerations

Analysis of the literature shows that the etiopathogenesis of childhood epilepsy is a complex and multi-stage process.

According to modern views, epilepsy is formed not as a separate epileptic focus, but as a result of the activity of pathological neural networks. Immaturity of the central nervous system in children leads to faster and deeper progression of these processes.

A deep understanding of the etiopathogenesis plays an important role in the early diagnosis, individualized treatment, and reduction of the consequences of childhood epilepsy.

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