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PATHOMORPHOLOGY OF THE BRAIN IN EPILEPSY

Resume: There are two types of pathological changes in the brain of patients with focal epilepsy: cortical malformations caused by developmental disorders and acquired changes, the most common of which is hippocampal (or mid-temporal) sclerosis. Hippocampal sclerosis (HS) is characterized by a pronounced loss of neurons in the SA1 sector of the hippocampus, to a lesser extent — in the sa3/SA4 sectors and a relatively small loss of cells in the Sa2 zone.

Recent data show that the pattern of damage to the cells of the hippocampus HS is clearly associated with other pathologies of the neocortex (the so-called two pathologies) of the temporal lobe with a tendency to further diffuse and homogeneous damage to neurons in all branches of the hippocampus. The term "Median temporal sclerosis" is often used as a synonym, but in fact it means more extensive damage to the internal structures of the temporal lobe.

Keywords: epilepsy, brain, pathomorphology.

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ПАТОМОРМОРФОЛОГИЯ ГОЛОВНОГО МОЗГА ПРИ ЭПИЛЕПСИИ

Резюме: Существует два типа патологических изменений в головном мозге пациентов с фокальной эпилепсией: пороки развития коры, вызванные нарушениями развития, и приобретенные изменения, наиболее распространенным из которых является гиппокампальный (или срединно-

височный) склероз. Склероз гиппокампа (HS) характеризуется выраженной потерей нейронов в секторе SA1 гиппокампа, в меньшей степени — в секторах sa3/SA4 и относительно небольшой потерей клеток в зоне Sa2.

Последние данные показывают, что паттерн повреждения клеток гиппокампа HS явно связан с другими патологиями неокортекса (так называемые две патологии) височной доли с тенденцией к дальнейшему диффузному и гомогенному повреждению нейронов во всех ветвях гиппокампа. Термин "Срединный височный склероз" часто используется как синоним, но на самом деле он означает более обширное повреждение внутренних структур височной доли.

Ключевая слова: эпилепсия, головной мозг, патоморфология.

Relevance. Symptomatic focal forms of epilepsy, which are the primary manifestation of many brain tumors, as a rule, become a secondary target during surgical intervention [4]. Volumetric brain formation in itself is a complex and difficult to treat disease, and the addition of epileptic seizures significantly complicates the patient's quality of life [1]. At the same time, there are significant differences in the clinical picture, symptoms and course of the pathological process in young children compared with older children and adults [5]. In addition, the treatment of such patients is complicated by the immaturity of the physiological (including nervous) system, since the immature brain differs from the adult brain by predisposition to epileptic seizures and the body's reaction to anticonvulsants [2].Epileptogenesis in these cases may depend on a number of factors, including the histological type of tumor, its localization and changes in the peritumoral parenchyma of the brain [3]. Seizures in patients with brain tumors, as a rule, are symptomatic and depend on the localization of the tumor, they also have significant polymorphism, with the possible development of secondary generalization [1]. In turn, antiepileptic therapy in this age group is difficult due to the potential interaction of anticonvulsants with chemotherapy drugs and side effects caused by their use [3]. It should also be noted the risk of

side effects during surgery or radiation therapy. All these factors influence the nature of seizures, frequency and susceptibility to therapy [5].

There is evidence that approximately 20 to 30% of difficult-to-treat epilepsies are caused by tumors of neuroepithelial tissues, especially neuroglial tumors [4]. Diffusely growing astrocytic tumors and mixed neuronal-glial tumors are the most frequent factors leading to the formation of an epileptic complex, and serve as an additional indication for the use of surgical intervention [2]. It is worth noting that of the mixed neuronal-glial tumors, dysembrioplastic neuro-epithelial tumor (DNEO) along with ganglioastrocytoma (GG) are included in the classification of Barkovich's developmental defects as malformations arising from abnormal neoplastic neural and glial proliferation with abnormal cell types associated with abnormal functioning of the cerebral cortex [3]. It is known that only in cases with DNEO and gangliocytoma (hamartoma) of the hypothalamus, which are benign formations, in the presence of steady progression

epileptic seizures and their resistance to anticonvulsants, surgical treatment of epilepsy is indicated [4].

Usually, but not always, neurophysiological studies reveal the correspondence of changes with the localization of education on MRI and thousand tons. MPT of the brain reveals characteristic radiological signs of volumetric formation, although rarely - absolutely specific [1]. In a retrospective review of the literature, ambiguous results of surgical treatment of epilepsy were noted. In certain cases, an intraoperative intracranial EEG (ECoG), which records the bioelectric activity and determines the zones of epileptogenesis, can help during surgical operation with correction of the resected zone [2]. However, the expediency of various methods of pre-surgical and intraoperative neurophysiological examination of patients with epilepsy with brain tumors, still causes a number of discussions.

The purpose of the study. The aim was to study the pathomorphological and neuroanatomic features of epilepsy in epilepsy in children; to develop effective approaches to diagnosis, conservative and surgical treatment of these forms of epilepsy and prediction of outcomes.

Materials and methods of research. Epiprimes, especially repetitive and uncontrolled, often lead to a violation of myocardial repolarization [27], accompanied by a violation of the membrane structures of the heart and brain, dysfunction of the autonomic centers.

The consequence of this is often the development of sinus tachycardia, sinus arrhythmia, paroxysmal atrial / ventricular tachyarrhythmias, life-threatening arrhythmias, which entails a violation of general hemodynamics, a decrease in cardiac output. As a result, hypoperfusion of the brain develops, which aggravates and prolongs the pathological activity of the epileptogenic focus, and this, in turn, contributes to the formation of persistent and severe cardiac arrhythmias.

According to a number of studies, the most pronounced hypoperfusion of the brain stem, respiratory center is more often registered in patients with tonic-clonic seizures.

Results and discussions. The nature of structural and functional relationships in the epileptic focus determines the variants of clinical manifestations of epilepsy. Lesion epilepsy is characterized by a high degree of anatomical and neurophysiological correlation of structural damage and pathological epileptiform activity; sources of epileptic activity are localized in the perifocal zone of organic damage. Non-local focal symptomatic epilepsy is characterized by predominant epileptogenic damage to the hippocampal parts of the brain, which causes the predominance of temporal epilepsy in the structure of acquired epilepsy.

In alcoholic epilepsy, the dominant type of epileptic seizures are partial secondary generalized seizures with rapid clinical and encephalographic

generalization, and the source of epileptic activity is located in the mediobasal parts of the brain.

In symptomatic epilepsy of lesion and non-lesion etiology, EEG monitoring of night sleep is most informative in identifying epileptic activity, which allows identifying and localizing epileptic activity in 96.4% of patients.

In lesion epilepsy of post-traumatic, tumor and vascular etiology, the main type of seizures are partial secondary generalized seizures with slow clinical and encephalographic generalization; a frequent combination of secondary generalized seizures with simple partial and complex partial paroxysms is recorded.

Prolonged video-encephalographic studies at the stage of initial diagnosis are shown to all patients with symptomatic epilepsy.

In the absence of structural changes during initial neuroimaging in patients with focal epileptic activity, dynamic MRI is shown on the EEG in order to exclude tumor clinical and anatomical dissociation.

Conclusion. This paper presents a retrospective analysis of patients with symptomatic epilepsy in adults. Depending on the structural basis of the epileptic focus and the etiology of epileptogenic damage, groups with lesion epilepsy (patients with identified structural epileptogenic pathology of the brain) and non-lesion epilepsy (absence of clinically significant epileptogenic disorders according to neuroimaging data) were formed.

The conducted research has shown that the currently existing clinical and encephalographic classification of epilepsy requires improvement and refinement taking into account the etiopathogenetic basis of the disease.

The data obtained made it possible to form anatomical, clinical and encephalographic foundations of the taxometric division of epilepsy of various etiologies. The age-dependent onset of post-traumatic and alcoholic epilepsy developing at a young age, epilepsy due to cerebrovascular pathology - in old age is shown. Unlike idiopathic forms of epilepsy, where the age of debut is

determined by the genetic basis of the disease, in symptomatic epilepsy, the age of debut is determined by the features of social activity and age-related progression of somatic pathology.

Neurophysiological examination determined the main localization priorities of epileptic activity. In non-lesion epilepsy, the sources of epileptic activity are located mainly in the hippocampal regions, in lesion epilepsy - in the perifocal areas of the brain damaged.

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