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НЕВРОЛОГИЧЕСКИЕ ПРОЯВЛЕНИЯ ПОСТИНСУЛЬТНОЙ ЭПИЛЕПСИИ ПРИ ГЕМОРРАГИЧЕСКОМ ИНСУЛЬТЕ

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АННОТАЦИЯ

Инсульт представляет собой острое заболевание, которое сопровождается гибелью клеток головного мозга вследствие острого нарушения мозгового кровообращения. Он проявляется общемозговыми и локальными симптомами. Развитие инсульта возможно по двум сценариям: либо после 24 часов от начала заболевания его признаки сохраняются, либо наступает летальный исход. Изучение нарушений мозгового кровообращения (НМК) по геморрагическому типу который представляет актуальную задачу из-за тенденции к увеличению частоты патологии, а также высокого уровня связанных с НМК летальности и инвалидизации.

Ключевые слова: неврологические проявления, постинсультная эпилепсия, геморрагический инсульт

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NEUROLOGICAL MANIFESTATIONS OF POST-STROKE EPILEPSY IN HEMORRHAGIC STROKE

АННОТАЦИЯ

Stroke is an acute disease that is accompanied by death of brain cells due to an acute disorder of the cerebral circulation. It is manifested by general cerebral and local symptoms. The development of stroke is possible in two scenarios: either after 24 hours from the beginning of the disease, its symptoms persist, or it is fatal. The study of cerebral haemorrhagic circulatory disorders (CABD) is of current interest because of the tendency for the pathology to increase in frequency as well as a high level of mortality and disability associated with CABD.

Keywords: neurological manifestations, post-stroke epilepsy, hemorrhagic stroke.

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GEMORRAGIK INSULTDAN SO'NG EPILEPSIYANING NEVROLOGIK KO'RINISHLARI (ADABIYOT SHARHI)

ANNOTATSIYA

Insult- o'tkir miya qon aylanishining buzilishi tufayli miya hujayralarining o'limi bilan kechadigan o'tkir kasallik bolib miya va mahalliy alomatlar bilan namoyon bo'ladi. Insultning rivojlanishi ikki turda kechadi umumiy va mahalliy belgilar bilan kechadi. Ushbu kasallik boshlanganidan 24 soat o'tgach, uning belgilari saqlanib qoladi yoki o'limga olib keladi. Gemorragik tipdagi serebrovaskulyarinsultni o'rganish bu patologiya chastotasining ko'payishi tendentsiyasi, shuningdekmiya faoliyatining buzilishi bilan kechadi va bu o'lim va nogironlikga olib keladi. Shu tufayli insult dunyoda dolzarb muammolardan biri hisoblanadi.

Kalit so'zlar: nevrologik namoyishlar, insultdan keyingi epilepsiya, gemorragik insult.

Introduction: Stroke is the second most common cause of death and the leading cause of disability worldwide. Although recent advances in acute stroke therapy have improved life expectancy, there has been a consistent increase in the prevalence of stroke-related epilepsy [1, 2]. Principles of management of patients with post-stroke seizures have not evolved as rapidly as other aspects of post-stroke care. To date, there

are insufficient clinical practice guidelines addressing most of the fundamental issues of post-stroke seizure management [3, 4]. The management of patients with poststroke epilepsy is of great clinical importance because patients with seizures after stroke have a higher mortality and disability rate than those without seizures [5]. In addition, epilepsy worsens long-term functional outcomes in those who have had a stroke [6]. Seizures impair patients' quality of life, may delay recovery of stroke-damaged functions, and aggravate cognitive impairment. The social consequences of poststroke epilepsy play an important role. Therefore, prevention and treatment of epileptic seizures are important in patients with stroke [7]. Frequency of epilepsy in stroke. Stroke is the cause of about 10% of all epilepsies and 55% of newly diagnosed seizures in the elderly [2, 8]. Statistically, 1 in 10 adult patients with first-onset epilepsy is associated with a stroke, and this etiology occurs in 1 in 4 patients aged 65 years or older with epilepsy [9]. Incidence rates are higher in patients with intracranial, i.e., intracerebral or subarachnoid hemorrhage. Hemorrhage increases the risk of seizures up to 10-16% [10, 11]. The risk of subsequent epilepsy is highest in patients with distant and after-effects of ischemic stroke [12]. Classification of seizures in stroke and their frequency Seizures develop in different stages of stroke. Depending on the time of occurrence, they can be divided into the following types: precursory, early, and late seizures. Unfortunately, there is currently no consensus among neurologists about the timing of these seizures. The most common definitions are that seizures occurring within the first 24-48 h, the first week, the first 2 weeks, and 1 month of stroke onset are called early seizures. In addition, many neurologists use a classification that distinguishes: pre-stroke precursors, early seizures that begin within the first 7 days after an acute stroke, and late seizures that appear 7 days after an acute stroke [1, 5]. According to the new ILAE revision, if a patient has at least one seizure and there is a possibility of further ones, the patient is diagnosed with epilepsy [1, 6]. The incidence of epileptic seizures in stroke patients varies widely, according to different authors, from 2-33% to 50-78% for early seizures. The frequency of late poststroke epileptic seizures, according to the literature, also varies in different studies from 3-4.5% to 67% [1, 8]. It has been found that most early seizures occur within the first 24 hours of the onset of stroke [9]. According to some authors' studies, the average interval between the onset of a stroke and the first epileptic seizure is 18 months (the interval is 0-7 years). According to other data, most epileptic seizures appear in the 2nd-10th month of ischemic stroke. Late poststroke epileptic seizures more often occur between 6 months and 2 years after the stroke [2]. The incidence of poststroke epilepsy ranges from 2.3% to 43%, according to different authors. Analyzing the age and sex peculiarities, it should be noted that most seizures in men occur at the age of 50-69 years, and in women - at the age of 60-79 years [1]. Seizures occur more frequently in relatively young and middle-aged patients. For example, in people younger than 40 years of age, 30% of cases occur, and in those under 50 years of age, 23.1% of men and 20% of women (22.6% of cases total). Patients in older age groups (over 60 years) tend to have fewer attacks after ischemic stroke. At the same time, patients aged 40 to 60 years revealed the development of more severe seizures compared to the group of patients aged 60-80 years [2]. Pathogenesis of epilepsy in stroke Epileptic seizures, divided according to the time of their occurrence, have different pathogenetic mechanisms. The precursor seizures may occur before the appearance of clinical symptoms of stroke and are often the only clinical symptom of a transient cerebral circulation disorder or so-called "silent" stroke, the diagnosis of which can be made retrospectively according to computed tomography or magnetic resonance imaging [2,3]. Early seizures are believed to be a consequence of local cellular biochemical dysfunction. Disruption of the blood-brain barrier caused by acute ischemia leads to dysfunction of ion channels, disruption of neurotransmitter homeostasis [2,4]. In areas of hypoxic-ischemic damage, the extracellular concentration of glutamate increases, which may lead to secondary neuronal damage and discharge of epileptiform activity. It has also been suggested that the seizures observed in severe stroke are the result of depolarization of the peri-infarct area [2,5]. The latter has been further confirmed in an animal model of mechanical occlusion of the middle cerebral artery, which demonstrated altered membrane properties and increased excitability of neuronal populations in the neocortex and hippocampus. In rodent models, stroke causes changes in ion channels that lead to an increase in extracellular potassium and intracellular calcium and sodium [2, 6]. These local ionic shifts can decrease the threshold of neurotransmitter capture during depolarization. In addition to focal ischemia, global hypoperfusion can induce seizure activity, especially when highly epileptogenic regions such as the hippocampus are involved. In addition, extravasated thrombin may also contribute to epileptic seizures by causing prolonged reactivity to afferent stimulation. Finally, hemosiderin deposition after intracerebral hemorrhage or hemorrhagic transformation can lead to increased neuronal excitability [2,7]. Late seizures occur when epileptogenesis is observed and the brain becomes predisposed to seizures after stroke in men and women if the lesion is localized in the frontal lobe of the brain. seizure susceptibility due to gliosis, deafferentation, selective neuronal loss, chronic inflammation, angiogenesis, neurodegeneration, collateral synaptic sprouting and altered synaptic plasticity [3, 8]. Seizures lead to the destruction of the blood-brain barrier and contribute to local inflammation, which is involved in the formation of the focus for the development of late seizures [2, 9]. In addition, vasogenic cerebral edema, collapse of cellular ion gradients, and mitochondrial dysfunction may contribute to secondary irreversible brain damage (gliosis) and lower seizure threshold. Persistent seizure activity in cerebral ischemia significantly increases infarct size and impairs functional recovery [3]. The latter suggests that stroke-related seizures and stroke share common pathogenic mechanisms and influence each other. The relationship between stroke and epileptic seizures or epilepsy is bidirectional [4]. Interestingly, middle-aged and elderly patients with newly diagnosed epilepsy have a 2-3-fold increased risk of subsequent stroke within the next two years [3, 11]. The hypothesis behind this is that epilepsy in these patients may be caused by microangiopathic changes predisposing to later cerebrovascular changes, causing seizures to be an early biomarker of subsequent stroke [3,12].

Risk Factors: One important issue is the risk factors leading to the development of post-stroke seizures and, in particular, to the further formation of post-stroke epilepsy. Much attention is currently being paid to finding risk factors for the development of vascular epilepsy. Most studies in hemorrhagic stroke have revealed a higher incidence of epileptic seizures and their early occurrence compared to ischemic stroke. Hemorrhagic transformation is a risk factor for early seizures and an independent predictor of epileptic status in the acute period of ischemic stroke [3,13]. Some studies have found a higher incidence of epileptic seizures after cardioembolic stroke compared with other subtypes of ischemic stroke. At the same time, there are a large number of studies in which the relationship between epileptic seizures and the cardioembolic stroke subtype has not been confirmed [3,4]. The role of stroke severity in the development of epilepsy has also been discussed. There have been population-based and prospective multicenter studies demonstrating that severe stroke is an important predictor of both the occurrence of early seizures and poststroke epilepsy [3,5]. A population-based study described by M. Lofthouse, showed that severe stroke is an important predictor of post-stroke epilepsy. A similar result was obtained in a study by L. Kammersgaard, T. Olsen [6]. The results of a prospective Copenhagen study suggest that severe stroke may be an independent predictor of early seizures, and the association of stroke severity with the development of late epileptic seizures was also found. Currently, there is an ambiguous attitude of different researchers toward the volume of ischemic focus as a risk factor for the development of epileptic seizures. There are supporters of the point of view that a large volume of a stroke focus is a predictor of seizure development. There are researches in which it is shown, that the size of a focus more than 1 /2 hemispheres of a brain can be the independent predictor of late post-stroke seizures. In a study by A. Alberti, M. Paciaroni, V. Caso in a single-factor analysis, large focal volume was a significant predictor of early seizures, but was insignificant in a multivariate analysis [3,12]. Many risk factors for post-stroke seizures and epilepsy remain poorly understood and the data are mixed. Among new factors, the role of involvement of the parieto- temporal cortex, supratentorial, and superior temporal gyrus in poststroke epileptogenesis has been confirmed [8]. It was also revealed that the spread of the ischemic focus to the large

hemisphere cortex can be a predictor of both early and late epileptic seizures [3,5]. A retrospective population study has shown that ethnicity and localization of the stroke focus are not risk factors for early seizures [4]. The authors also showed in a retrospective study, taking into account multivariate analysis, that the functional state of patients before stroke, assessed more than 1 point on the modified Rankin scale, is a predictor of early seizures. According to various authors, qualitative disturbances of consciousness in the acute period of stroke are predictors of early epileptic seizures in patients with hemorrhagic and ischemic stroke [13]. About 30% of all epilepsy syndromes are thought to have a genetic origin and are caused by more than 500 loci present in humans and mice [4]. Researchers studied that the ALDH2 (aldehyde dehydrogenase) and rs671 polymorphism was associated with seizures and increased plasma concentrations of the ALDH2 substrate, 4-hydrosinonenal (4-HNE). In addition, the CD40-1C/T polymorphism was associated with susceptibility to seizures by increasing the plasma concentration of sCD40L, which is involved in the inflammatory response. Transcrip profiling has shown that a functional link exists between many of the genes that modulate ischemic stroke outcomes [5]. There have been no other studies proving that this polymorphism may increase the association of seizures with certain comorbidities. There are studies citing "young age" as a predictor of post-stroke epilepsy. There is evidence that post-stroke epilepsy is most common in the elderly, particularly in people 85 years and older [4,10]. It is also worth noting that children are no less likely to develop seizures. Cohort studies have noted that seizures in children occur in 58% of cases. Seizures were also found to be 18 times more common in children than in adults within 24 h of stroke onset [46]. The findings suggest that factors such as arterial hypertension, diabetes mellitus, smoking, hyperlipidemia (cholesterol >200 mg/dl or TGC.

Seizure syndrome can be provoked by the following factors:

- emotional overstrain;
- physical or psychological fatigue;
- side effects of certain medications.

The main causes of cramps are considered to be the occurrence of cysts, adhesions and other neoplasms in areas of dead tissues.

- The main signs of a convulsion syndrome
- During convulsions, there is a contraction or twitching of certain muscle groups. The duration of the convulsive syndrome varies from a few seconds to ten minutes. A patient after a stroke most often suffers from short-term cramps throughout the body. They may occur in the legs, arms, neck, or face. After a seizure, the patient becomes unconscious or falls asleep as a result of the brain going into shock. The manifestation of the seizures may vary. Some patients have a slight tremor, while others suffer from severe seizures.

Seizures during and after a stroke may manifest as:

- Short-term contractions of the facial muscles (this may skew one half of the face, resulting in a mask-like face);
- numbness of the limb muscles, complete loss of control over them, the feeling of a wooden arm or leg;
- contraction of facial muscles against the background of limb numbness.
- Consequences of convulsions after a stroke

Seizures are an extremely dangerous consequence of a stroke, and without adequate therapy can cause serious complications. Deterioration is possible with seizures with loss of consciousness during the period of acute impairment of cerebral circulation. With seizure syndrome, the following consequences may occur:

- The development of a recurrent stroke;
- prolonged or short-term loss of consciousness (with more intense and pronounced seizures, the patient may fall into a coma);
- disability - permanent seizures that are difficult to treat are evidence of serious disorders in the brain, which leads to disability without the possibility of restoring the lost functions;
- lethal outcome - if seizures occur frequently and treatment fails to yield positive results, it can be assumed that there is an acute circulatory disorder in the brainstem, which causes respiratory and cardiac arrest.
- The patient who has regular attacks of stroke faces problems of a psychosomatic nature. Loss of motor function, inability to perform tasks that previously seemed simple, self-care, combined with a seizure syndrome make the patient feel inferior.
- Treatment of seizures after a stroke Immediately it is necessary to call an ambulance. Before the arrival of doctors, first aid is provided by people who are close to the patient at the time of the attack. It is necessary to provide the patient with access to oxygen. If it happened indoors, you should open the windows, in another case, you need to take off the patient's tight clothing. Under the head of the patient should be placed a pillow or a roller. If there are dentures in the mouth they should be removed, clean the oral cavity from food debris. If the patient began to breathe hoarsely, he should be put on his side and check that nothing impedes breathing. If pain in the muscles develops it is necessary to make a massage. It is advisable to lubricate the skin with olive oil before the massage. Treatment of seizures is carried out in the intensive care unit. Treatment of seizures begins with intravenous administration of anticonvulsants. If ineffective, switch to a combination of anticonvulsants - parenterally and through the probe:

- diazepam 0.15 to 0.4 mg/kg intravenously at a rate of 2-2.5 mg/min, repeating 0.1-0.2 mg per 1 kg body weight per hour if necessary;
- valproic acid intravenously 20-25 mg per 1 kg of body weight for the first 5-10 minutes, followed by a continuous infusion of the drug at a rate of 1-2 mg per 1 kg per hour, or bolus infusion four times a day at a daily dose of 25-30 mg per 1 kg of weight per day;
- in refractory status epilepticus and if diazepam is ineffective, sodium thiopental is used under control of external respiratory function.

Finlepsin or carbamazepine are used to relieve the patient's condition. Finlepsin has the following side effects:

- Reduces intellectual activity when the drug is used for an extended period of time;
- causes impotence, apathy;
- provokes the development of osteoporosis or increased bone fragility (this problem is corrected with calcium preparations);
- Increases the risk of bleeding in combination with anticoagulants.

Finlepsin treatment is administered to persons not older than 65 years. A maximum of 400 mg of the drug can be taken per day.

Conclusion: Thus, stroke currently remains one of the most important medical and social problems. This is due to their high share in the structure of morbidity and mortality of the population. In addition, cerebrovascular diseases often lead to the development of structural epilepsy. Given that most cases of poststroke epilepsy occur at a young age, and the fact that there are conflicting data on risk factors for poststroke epilepsy, it is necessary to conduct research to better understand the pathophysiological mechanisms of poststroke epilepsy, which will ensure timely prevention and diagnosis of poststroke epilepsy and improve patients' quality of life. At Yusupov Hospital, neurologists use the latest medications to relieve seizures, which are effective and have a minimal range of side effects. The resumption of symptoms is not indicative of an incorrect tactic of treatment, therapy, the development of repeated acute impairment of cerebral circulation, a large area of necrosis or the formation of a cyst in place of dead tissues. Patients with seizures after a stroke are treated by doctors at a neurology clinic or intensive care unit. Professors, doctors of the highest category are individualized in their choice of therapy regimen for stroke seizures. Severe cases of brain infarction or hemorrhage are discussed at the meeting of the Expert Council and a collegial decision is made on further tactics for treating patients with a convulsive syndrome. Seizures in men and women can occur after ischemic or hemorrhagic stroke. They are the result of nerve cell death in the brain. When foci of necrosis begin to form, the body tries to stop the lesion and restore normal blood flow to the brain. This task is accomplished by activating neurons that are located around the area of the infarction or brain hemorrhage. Cavities with fluid are formed in place of the dead tissue. This formation does not cause discomfort. Sometimes it irritates the neurons, which is accompanied by convulsions after the stroke.

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