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Features of the Clinical Course of Post-Traumatic Epilepsy, Psychiatric and Neurosurgical Approaches (Literature Review)

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Abstract: People with head injuries are at least three times more at risk for epilepsy than the population. Traumatic brain injury refers to the main risk factors for epilepsy, along with a severe family history for bacterial meningitis, heroin use, or epilepsy. Although head trauma accounts for only 5% of all cases of epilepsy [1], post-traumatic epilepsy (PTE) is a very large problem. Most importantly, traumatic brain injury refers to etiological factors that can prevent epilepsy.

Key words: post-traumatic epilepsy, traumatic brain injury, head trauma.

Introduction. The main offshoot of the term" post-traumatic attacks " is that trauma is not only the cause of the previous, but also the cause of the attacks. Post-traumatic attacks start early within 1 Week of injury and more than 7 days after injury. However, in some studies, all attacks that occur before the acute symptoms of injury disappear are classified as premature [1-3].

The group of early seizures also includes a separate subgroup – "immediate" or "acute" seizures in the first minutes after injury. Early attacks are acute symptomatic; late attacks are long symptomatic. They can be one and several. According to the ILAE classification, only repeated late seizures correspond to the definition of "posttraumatic epilepsy". Nevertheless, in practice, the terms "PTSD" and "PTSD" are often used in turn [4-6].

Epidemiology of post-traumatic epilepsy. Attacks develop on average in the first 3 years, often in the first year after injury (57.7%). The total frequency of post-traumatic seizures is about 6.5 %, early seizures are about 2%, and late seizures are about 4.5% [7].

Risk factors. Factors associated with high risk for penetrating injuries include post-traumatic epilepsy, inhibition of consciousness up to 3-8 points on the Glasgow Coma Scale; severity of injury; metal fragments in the skull cavity; intracranial hematoma and parenchymal bleeding; permanent neurological deficits and decreased brain tissue volume through computed tomography [8, 9]. In turn, depressed skull fractures and intracranial hematomas (both subdural and intracerebral) are risk factors for early and late attacks for non-penetrating injuries. This increases the risk of seizures and repeated concussions several times [10]. Attacks in children after intentional injuries are more common than accidental injuries, while intentional injuries turned out to be more "malignant" according to other parameters [11].

Despite the lack of objective information, it cannot be completely ruled out that genetic predisposition increases the risk of seizures in patients with mild head injuries, but these patients are a relatively small group among those suffering from post-traumatic epilepsy. Obviously, the use of seat belts and helmets also leads

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to a decrease in the severity of brain damage and the frequency of late seizures as a result of cycling, Moto and car accidents [12-15].

Pathogenesis posttraumatic epilepsy. Trauma involves a number of structural, biochemical and physiological changes. Mechanical action leads to rupture of nerve fibers and blood vessels and diffuse axonal damage, which is histologically manifested by gliosis, the formation of microglial scars, back strain of axons and Valler degeneration. Focal injury is a combination of bleeding, swelling, and necrosis; in addition, traumatic brain injury can be isolated large bleeding. Neurodegeneration of the hippocampus and germination of bryophyte fibers play an important role in the development of epilepsy in surviving patients [16-18].

Parenchymal bleeding, followed by hemosiderin accumulation, is one of the main risk factors for epilepsy, and experimental studies show that the inclusion of iron or its chloride in the neocortex leads to regional epileptiform activity in EEG and seizures [19].

The epileptogenic effect of iron is associated with the appearance of free radicals and can prevent seizures by administering antioxidants. However, the exact mechanism of this process remains unknown. In patients with reduced hemoglobin binding protein, resorption of intraserebral hematomas slows down, which can increase the risk of posttraumatic seizures [20-23].

Attacks that occur immediately or shortly after brain damage in rats are accompanied by increased levels of glutamate and aspartate [24]. Excitatory amino acids are not only highly epileptogenic, but also cytotoxic in relation to neurons.

NMDA receptor antagonists can block their negative effects. The release of excitatory amino acids can also cause a dramatic increase in extracellular potassium concentration observed in experimental models of brain damage [25]. The increase in extracellular potassium, in turn, leads to excessive excitability of neurons and facilitates the development of attacks. But it is unlikely that the "universal" mechanism of PTSD development will ever be found [26].

Hidden or" dumb", the period from the moment of injury to the onset of attacks W. R. It has been known since the days of Gowers. Injury can probably trigger a dynamic epileptogenic process that increases the excitability of nerve cells, shapes or destroys important connections between neurons, which can cause such a restructuring of the brain, in which a network of epileptogenic neurons can form sufficiently.clinically manifested by attacks [27].

In addition, in some cases, head injuries can reduce the "limit of attacks" relatively quickly. In these cases, the time interval between trauma and first attack does not have to be a progressive process, but rather reflects the rate at which pathological arousal spreads before engaging the critical mass of neurons. With traumatic brain injury (unlike a brain tumor), the timing of the onset of a potential epileptogenic process is clearly known, which makes it possible to start using neuroprotectors in a timely manner [28-30].

In experiments in rats, according to some data, it was possible to establish an important period for therapeutic intervention [31]. Nevertheless, experimental data on the use of NMDA receptor antagonists as neuroprotectors in rats has not yet been confirmed in human studies.

Post-traumatic epilepsy clinic. Attacks caused by brain damage, are not semiologically different for other reasons [32]. A head injury may result in simple and complex partial attacks of all types, as well as general tonic-clonic seizures.

In a number of patients with partial and general seizures, it is impossible to accurately calculate the number of seizures of each type. The study found that 52% of posttraumatic epilepsy patients developed general seizures following moderate to severe traumatic brain injury, 33% developed partial seizures, and 15% developed secondary generalizing partial seizures [33-35].

In another study, posttraumatic epilepsy among patients with partial seizures, 57% temporal epilepsy, 35% frontal, and 3% parietal and occipital [16]. 44% of patients with Temporal epilepsy had medial temporal sclerosis and 26% had temporal neocortex injury [36].

In a long-term EEG follow-up (for a week) in patients after moderate to severe traumatic brain injury

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in 11 out of 94 cases, electroencephalographic seizures were reported that were not clinically manifested [37]. These attacks are generally not recognized; they are not associated with increased intracranial pressure or worsening cerebral blood flow, and do not predict a negative prognosis.

Diagnostics. EEG does not play an important role in the examination of patients with early seizures. It is mainly used for differential diagnosis of behavioral disorders in patients with altered and impaired consciousness [38-40].

The initial diagnosis of head injury is based on the nature of the injury and the results of clinical and neurological examination. A patient with moderate to severe traumatic brain injury is shown an emergency CT scan [41].

Intracerebral bleeding, subdural hematoma and hydrocephalus may not appear immediately, but a repeated examination after a certain period of time after the injury is indicated for patients who, contrary to expectations, do not improve or worsen against the background of adequate treatment. The debut of attacks is also a guideline for repeated tomography [42].

The need for Neuroimaging after mild traumatic brain injury is a matter of debate, but it is clearly indicated in patients with seizures or other unexpected state changes [43-46]. Predicts the presence of intracranial abnormalities in patients with mild seizures brain damage. The need for MRI in the acute period of injury may be due to the presence of additional risk factors for epilepsy. The patient with the first late seizure should be examined as if he had a seizure without the first cause, others should also be taken into account.traumatic brain injury, causes of seizures [47-50].

Differential diagnosis posttraumatic epilepsy. Fluctuations in the level of consciousness and behavioral disorders can lead to diagnostic difficulties, especially in patients with head injuries in the first 1-2 weeks after injury. In fact, moderate to severe traumatic brain injury means that there are other neurological disorders that can be the cause of encephalopathy and/or attacks. Errors and warnings that are not accompanied by other symptoms, the appearance of strong emotions without changing consciousness, or episodes of aggression should not be considered as manifestations of attacks [51-56]. Persistent cognitive, emotional, and behavioral disorders are probably not associated with pathological electrical brain activity. However, it should be borne in mind that it can be very strange to monitor some patients with frontal attacks, and they have been misdiagnosed that the seizures are not epileptic. It is not uncommon for patients with cognitive disorders associated with encephalopathy to find it difficult to detect typical complex partial seizures. To develop a treatment strategy, it is necessary to determine which of the patient-specific clinical manifestations is an attack and what is not. In some cases, only EEG video monitoring can answer this question. Despite not having a serious impact on traumatic brain injury to the development of non-epileptic seizures, this is one of the risk factors. However, most patients with pseudoprevitis have traumatic brain injury, and have had various mental illnesses before the injury, while some have faked head injuries to address pathologies or social problems [57-60].

Treatment post-traumatic epilepsy. Patients with moderate to severe head injuries are particularly susceptible to the physiological effects of attacks such as metabolic acidosis, acute increase in intracranial pressure, as well as respiratory diseases, including pulmonary edema. Convulsions can cause additional damage in patients with severe combined injuries. Postictal (post-attack) confusion makes it difficult to adequately assess the neurological condition. In this regard, many patients use antiepileptic drugs for brain damage, especially in its acute stage. Valproates, carbamazepine, and phenytoin do not affect the mechanism of development of posttraumatic epilepsy, being antiepileptogenic drugs rather than antiepileptogenic drugs [61-65].

These findings are also valid for other epileptogenic brain injuries: prophylactic appointment antiepileptic drugs do not reduce the risk of First seizures in patients with brain tumors [66].

According to leading experts, severe patients with brain damage should receive phenytoin during the first week after injury, which reduces the risk of attacks and their complications during the period of acute injury. Where phenytoin and phosphenitoin are not noted for Parenteral use, an alternative to these drugs are

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injectable valproates and levetiracetam, as well as oral antiepileptic drugs. It should be borne in mind that injectable diazepam (relanium, Valium, seduxen), which is used in our country to prevent attacks in patients with frequent injuries, is a brain injury that retains therapeutic concentration in the brain substance within minutes of ingestion, making it meaningless to use this drug to prevent premature attacks.

Conclusions. The decision to start treatment in patients who experience the only post-traumatic attack antiepileptic drugs depends on risk factors for repeated attacks. Long-term use of antiepileptic drugs is usually impractical in a patient with early seizures. On the contrary, the first late attack is repeated in most cases, and such patients are prescribed drug therapy. There is no consensus on the duration of use of antiepileptic drugs in patients with PTSD. In the case of isolated early seizures, treatment should be discontinued after a few weeks or months. On the contrary, in patients with late attacks, it should last at least 2 years.

In late attacks, all the main antiepileptic drugs are effective, the choice of which is mainly determined by the potential for side effects, among which special attention should be paid to sedation and behavioral disorders. The epileptic condition caused by head trauma is treated as it is for other reasons.

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