IJCNP, Volume 2, Issue 7, 2024 ISSN: 2995-536X
https://medicaljournals.eu/index.php/IJCNP

INTERNATIONAL JOURNAL OF COGNITIVE NEUROSCIENCE AND PSYCHOLOGY

Последствия Черепно-Мозговой Травмы У Детей И Подростков

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Abstract: The consequences of traumatic brain injury (TBI) in children and adolescents represent a serious medical and social problem. Craniocerebral trauma is one of the most common types of CNS injuries and is most common in two age groups: in children under 5 years of age and in the youth group of 15-25 years of age.

Key words: craniocerebral trauma, children, central nervous system, adolescents.

Introduction. Clinical manifestations of traumatic brain injury are determined by primary and secondary CNS damage [2,6]. In the area of primary brain tissue damage, necrosis is formed with destruction and death of neurons and glia, morphologic asynapsia, violation of vascular integrity and thrombus formation. In the penumbra region around the focus of primary damage, brain cells retain viability, but suffer from lack of oxygen and nutrient delivery. Secondary damage is due to the fact that the traumatic impact triggers the processes of intracellular metabolic disorders, excitotoxicity, free radical formation, activation of lipid peroxidation, and autoimmune inflammation. The result of the cascade of violations of biochemical and immunologic homeostasis can be apoptosis - irreversible lesion of cells of the penumbra zone in the immediate vicinity of the focus of primary damage with subsequent involvement of intact brain regions in the pathologic process.

In this case, a traumatic brain injury initiates two oppositely directed processes (not only at the site of injury, but also remotely): degenerative-destructive and regenerative-reparative, which proceed with constant or variable predominance of one of them, which largely determines the presence or absence of certain clinical manifestations in the remote period [2]. Neurotrophic factors (specific intracellular neuroregulatory proteins) play an important role in the processes of neuroregeneration and neurorepair. Meanwhile, extensive CNS injuries result in the lack of neurotrophic factors or deficiency of their action, which leads to the predominance of secondary damage processes over neurorepair processes and increases the risk of unfavorable functional outcomes [7].

According to experimental and clinical studies [2, 3], pathological changes in various brain regions arising from traumatic brain injury can persist, and in some cases progress, for many months or years after trauma, leading to the formation of long-term posttraumatic disorders.

Neuroplasticity is a process of biological adaptation based on structural and functional reorganization of the CNS and aimed at restoring lost or impaired functions after brain damage. Neuroplasticity is based on modulation of neuronal functioning, restoration of synaptic transmission, activation of inter-neuronal connections, including horizontal connections at the level of the cerebral cortex, which were not involved earlier. Neuroplasticity can be realized at the molecular, synaptic, neuronal and multimodular levels (brain section or brain as a whole). To varying degrees, activation of neuroplasticity is accompanied by stimulation of expression of certain genes, biosynthesis of receptor and ion channel molecules, filamentous proteins of synaptic cytoskeleton, neurotransmitter, synaptic membrane components, intercellular adhesion molecules, formation of immature contacts, their maturation, activation, hypertrophy and reorganization of active synapses [9]. Reparative neuroplasticity provides restoration of functional brain systems after their damage and is realized by the whole spectrum of synaptic pool efficiency increase, from activation of preserved synapses to neosynaptogenesis and nerve outgrowth (synaptic sprouting phenomenon) [9].

Energetic and plastic reorganization of the brain after traumatic brain injury can last for a long time. The peculiarity of the clinical course and outcomes of pediatric traumatic brain injury is due to the fact that mechanical energy affects the brain, the growth and development of which have not yet been completed. The traumatic brain injury interferes with the normal course of neuroontogenesis, which affects the formation of the child's personality, his cognitive and emotional development, schooling, and the formation of social skills.

Previously, there was a point of view according to which the recovery of disturbed functions after traumatic brain injury in children and adolescents is more complete than in adults. It was believed that the younger the age at which the traumatic injury occurred, the greater the likelihood of complete regression of neurologic disorders. Nevertheless, the results of focused studies [10-12] are not so optimistic; moreover, the effects of traumatic brain injury may not manifest themselves immediately, but may be delayed.

It is known that functions that develop during the onset of a traumatic injury may be affected to a greater extent than those already formed before the injury. For this reason, the age of the patient in trauma is an important factor influencing its consequences. For example, a traumatic brain injury in a 3-4 year old child may result in speech development disorders, while in a teenager it may result in only mild verbal impairment. Most of the skills already formed at the time of trauma are preserved, even if there was a temporary loss of them immediately after the trauma [1, 13, 14].

According to A.I. Kemalov's study [15], in which 283 patients 4-14 years old were examined, most of them had movement coordination disorders (100%), disorders of higher mental functions (attention and memory - 75%, speech - 14%), as well as cerebrastenic phenomena (88% of patients) and chronic posttraumatic headaches (95%) within 6 months to 4 years after a moderate to severe closed traumatic injury.

Cognitive disorders in children and adolescents in the remote period of traumatic brain injury lead to difficulties in school education and social adaptation [10, 11, 16]. Neuropsychological methods in these patients have confirmed a significant decrease in the indices of auditory, visual and kinesthetic memory, sustained and distributed attention, control functions, and the speed of cognitive operations compared to healthy peers [10, 16]. Slowed speed of intellectual processes and difficulties in organizing and managing complex information are two constant manifestations of the consequences of traumatic brain injury [1, 14]. They make it very difficult for the patient to cope with tasks in the dynamic and complex environment of the educational process. Therefore, the return to learning and social life after a traumatic brain injury is associated with high fatigue, overexcitement and emotional distress.

In general, children are more likely than adults to have a favorable outcome even after severe traumatic injuries due to the high plasticity of the developing brain. At the same time, long-term consequences can be observed in children who in the acute period of traumatic injury were not found focal neurological symptoms and skull bone fractures. Therefore, even mild childhood traumatic injuries are not always without sequelae, and their consequences may appear years after the injury [1, 11].

The vulnerability of immature brain structures associated with traumatic brain injury can be expressed not only in developmental disorders of its functions, but also in paroxysmal disorders. Cognitive and behavioral disorders in children and adolescents in the remote period of traumatic brain injury are significantly increased in the presence of paroxysmal disorders: posttraumatic headache (PTHB), posttraumatic epilepsy (PTE), subclinical epileptiform activity on electroencephalogram (EEG).

PTHB is one of the most frequent complaints in patients in the remote period of traumatic brain injury. It can be both an independent symptom and a part of the structure of postcommotional syndrome. According to the results of studies [7] of the remote period of mild traumatic brain injury in 60-80% of children and adolescents, a complex of disorders, labeled as postcommotional syndrome, is formed. Along with headaches, it is characterized by dizziness, cerebrasthenic symptoms (rapid fatigability,

emotional lability, anxiety, irritability, difficulty falling asleep), mild disorders of attention and memory, slowing of psychomotor reactions and moderately pronounced disorders of movement coordination.

The pathogenetic relationship between PTHB and traumatic brain injury is reflected in the International Classification of Diseases, 10th Revision (ICD-10)1 , where this type of headaches is defined as "chronic PTHB" (G 44.3). The International Classification of Headache Disorders 3rd Revision (ICHD-3)2 categorizes PTHD as secondary headache and presents under "Headache associated with trauma or injury to the head and/or neck". It should be noted that acute headaches in traumatic brain injury, as a rule, are symptomatic and first of all require exclusion of intracranial hematoma, subarachnoid hemorrhage, brain contusion. In ICHD-3, the term "chronic PTHD" has been replaced by "persistent PTHD", which is defined as a new headache or headache that was present before the injury but became more frequent and intense after the trauma and persisted for more than 3 months. What acute and persistent PTHB have in common is that the headache appears within 7 days of injury or regaining consciousness after injury, or after discontinuation of medications that impair the ability to feel or report the presence of headache following head injury.

PTHB may have different characteristics and be similar in clinical presentation to primary headaches (tension headaches, migraine, or other type of headache), but must be clearly related to the fact of having suffered a traumatic brain injury in terms of time of onset or increasing severity.

The results of studies on the clinical features of chronic PTHS in children [8] showed that most patients had clinical features similar to those of "primary" headaches, and no headache associated with intracranial hemorrhage or the effect of brain matter compression was diagnosed. In 46.5% of children, 46.5% had migraine-like headaches, 24.8% had tension-type headaches, and 19% of patients had headaches of an unclassifiable nature. Thus, almost ½ of the patients had migraine-like type of headache, ¼ had episodic or chronic tension headaches, and the remaining patients had mixed or unclassifiable headaches; no cases of cluster headaches or associated with other neuralgias were identified [1].

The pathophysiology of PTHS is not fully elucidated, but it is assumed that even in mild traumatic brain injury diffuse axonal damage occurs, which may be accompanied by metabolic disturbances in brain tissue, changes in cerebral hemodynamics, and in combination with abnormal release of excitatory neurotransmitters and proinflammatory peptides may cause the development of headache and its transition to a chronic form [9].

The occurrence of chronic PTHB is usually independent of the severity of traumatic brain injury and changes in neurologic status. It is believed that more often chronic PTHB occurs after mild traumatic injury, can worsen several months and years after the trauma and even have a progressive course in its remote period [10]. Social and psychological factors, especially in patients with mild traumatic injury, as well as the abuse of analgesics and non-steroidal anti-inflammatory drugs are common causes of chronic PTHB.

Meanwhile, the formation of clinical manifestations of the consequences of traumatic brain injury is also explained by prolonged for many years disturbances of cerebral hemodynamics and regulation of vascular tone [11]. Another pathogenetic mechanism of the formation of the consequences of closed traumatic brain injury - liquorodynamic disorders - is directly related to the vascular factor. Changes in cerebrospinal fluid production and resorption after a closed traumatic injury are based on damage to the endothelium of choroidal ventricular plexuses, secondary disorders of the cerebral microcirculatory system, disorders of venous outflow from the cranial cavity, and fibrosis of cerebral membranes. These factors lead to the development of liquor dynamics disorders in the form of liquor hypo- or hypertension.

Regional ischemia and neurogenic inflammation play an important role in the formation of chronic PTHB and other consequences of traumatic brain injury, the development of which is possible even after mild traumatic brain injury [2]. According to the theory of excitotoxicity, these processes are

accompanied by the release of excitatory neurotransmitters (glutamate, aspartate), which leads to the development of secondary neuronal damage and contributes to the formation of neurological disorders in the remote period of traumatic injury. In the work of N.A. Bazarnaya [3], the levels of autoantibodies to glutamate receptors of the AMPA subtype (to the GluR1 subunit) and NMDA subtype (to the NR2A subunit) in the serum of 60 patients 7-16 years old with chronic PTGB after mild traumatic brain injury were studied. Group 1 included 48 children who had suffered concussion: 34 with a single traumatic brain injury and 14 with repeated traumatic brain injuries. Group 2 included 12 children after mild brain contusion. The level of autoantibodies to glutamate receptors in serum was determined 6 months and 1 year after traumatic brain injury. The increase in the concentration of autoantibodies was expressed as a percentage and was considered significant if the increase exceeded 120% of the level of healthy children of the corresponding age. The highest content of autoantibodies to NMDA receptors was found in children with mild brain contusion (165±34%) and with a single concussion (145±12.6%). In children with repeated concussions, the content of these autoantibodies did not exceed normal values (108±12.4%). An increase in the level of autoantibodies to NMDA receptors was observed during the 1st year after injury. Increased levels of autoantibodies to the AMPA receptor subtype were found in children with repeated concussion and mild cerebral contusion (150±16.8 and 167±31.3%). The results of this study indicate that children with chronic PTGB have hyperstimulation of glutamate receptors and development of autoimmune process.

The diversity of pathogenesis factors determines the existence of several clinical forms of chronic PTGB, including tension PTGB, migraine-like, hypertension, cluster, and cervicogenic pain [2]. The clinical polymorphism of cephalgias characteristic of PTGB is due to different pathogenetic mechanisms that may be combined in the same patient.

In an examination [4] of 104 adolescents 12-19 years old in the remote period of moderate and severe closed traumatic brain injury, we found that chronic PTGB were noted in 82% of patients with a high frequency: from daily to once a week. Headaches were severely tolerated by adolescents, negatively affecting their studies, performance, mood and behavior. Emotional stress (28%), weather changes (23%), mental fatigue (19%), physical exertion (12.5%), and noise (2%) were noted as triggering factors for chronic PTHS, but in 34% of patients no clear cause for headaches was identified. In the majority (51%) of the examined adolescents, headaches in their clinical picture corresponded to tension PTGB. Liquorodynamic disorders with intracranial hypertension were detected in 29% of patients. In 8% of adolescents, headaches were migraine-like in nature, with 4 patients having headache attacks debuted before the traumatic brain injury, but clearly intensified and became more frequent after it, and in 3 patients the traumatic brain injury provoked their appearance. Posttraumatic neuralgic (cervicogenic) headache was noted in 11% of patients, and in one case there was cluster PTHB.

According to A.V. Goryunova's study [2], during psychological examination of children with chronic PTGB, changes in the emotional-volitional sphere, characterological and personality traits occurred in 76% of patients, which is an important predisposing factor for the formation of pathological pain behavior and consolidation of the chronic character of headache. The majority of children showed cognitive disorders in the form of decreased efficiency, concentration of attention, reduced memory, difficulty in learning educational material, which led to school maladaptation and reduced quality of life; 80% of children with chronic PTGB often missed school classes, which had a negative impact on their social adaptation.

If there are indications of significant brain damage as a result of traumatic brain injury and frequent seizure-like PTGBs, it is necessary to exclude the epileptic genesis of paroxysmal disorders. The combination of PTGB and epilepsy, as well as epileptiform activity on EEG in patients with PTGB was first reported in 1963 by D. Cooper and D. Cavicke on the basis of two clinical studies. Cavicke on the basis of two clinical observations. R. Formisano et al. [6] found a high frequency of paroxysmal EEG abnormalities with the presence of sharp waves in 84.6% of patients with chronic PTGB, which was also associated with the presence of fractures or damage to the skull bones and dura mater either due to trauma or craniotomy.

Not only routine EEG, but also video-EEG monitoring including different functional states (especially all phases of sleep) should be used in the examination of patients with chronic PTGB. The application of multichannel EEG monitoring in combination with evoked brain potentials to study the disruption and delayed activation of neuronal networks in PTHB, especially in posttraumatic migraine, seems promising [7].

PTE refers to a serious neurological consequence of traumatic brain injury. It is characterized by several (2 or more) unprovoked epileptic seizures occurring no earlier than one week after the traumatic injury. PTE accounts for approximately 20% of symptomatic epilepsies and 5% of all epilepsy cases in both adult and pediatric populations [1]. After severe traumatic brain injury in children, the risk of epilepsy increases 7-fold [9], with cases with a Glasgow Coma Scale score of 9 or lower subsequently developing PTE in 10-15% of adult patients and 30-35% of children [30].

PTE significantly complicates the life of victims and has both medical and socio-psychological consequences, leads to the aggravation of cognitive impairment and may represent a risk factor for neuropsychiatric developmental delay in the remote period of traumatic injury.

A distinction must be made between PTE and posttraumatic seizures. Seizures that occur within 24 h after a traumatic injury are labeled as immediate. Seizures that develop during the first week after injury are considered early. According to different studies [3], early convulsive seizures occurred with an incidence ranging from 2.6 to 16.9% of cases depending on the severity of the trauma. It should be noted that early posttraumatic seizures are more often observed in children under 5 years of age.

Seizures with manifestation within 8 days or more after traumatic injury are defined as late. About 20% of patients with single late posttraumatic seizures do not have their recurrence in the future, hence PTE is not diagnosed in them. But in most cases, late posttraumatic seizures are characterized by a high recurrence of paroxysms with transition to PTE. According to a 10-year prospective follow-up [2] of a group of trauma patients, the average frequency of late seizures was 2.1%, but it was significantly higher in the subgroup of patients with severe traumatic brain injury (12%).

PTE manifests in the majority (80%) of patients as a first unprovoked seizure within a year after the trauma and in almost all patients within 2 years after the trauma. According to the study by J. Annegers et al. [2], the risk of an unprovoked epileptic seizure is 11 times higher than the population average in patients during the 1st year after trauma, 3.5 times during 4 years, 2.39 times during 5 years, and 1.56 times during 10 years after trauma. Convergence of the risk index with the population average after mild traumatic brain injury occurred within 5 years, whereas after severe traumatic brain injury it remained 4 times higher even after 10 years. In general, the risk of epileptic seizures is highest during the 1st year after trauma and gradually decreases thereafter.

Conclusions: It should also be noted that studies have shown a positive effect of levetiracetam on the background bioelectrical activity of the brain along with suppression of epileptiform discharges in patients with traumatic brain injury and other CNS lesions, indicating its neuroprotective potential.

Thus, childhood and adolescence are the periods of rapid physical and psychological growth, endocrine restructuring and, at the same time, high traumatism. The consequences of traumatic brain injury in children and adolescents can be represented by both cognitive and paroxysmal disorders. These disorders can have a long-term and significant negative impact on the success of school education and social adaptation of patients. Meanwhile, a high level of neuroplasticity in children and adolescents can determine favorable outcomes of traumatic brain injury.

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