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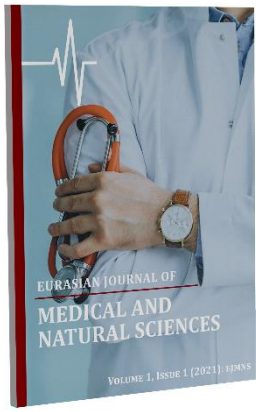

MORPHOMETRIC LIVER PARAMETERS AT DIFFERENT PERIODS OF CRANIAL BRAIN INJURY

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ABSTRACT

The article analyzes the literature data on morphological and functional changes occurring in the liver during a cranial brain injury and its relationship with periods of cranial brain injury.

The problem of severe cranial brain injury (CBI) has attracted the attention of researchers for many years. The gigantic scale of modern injuries has made it not only medical, but also acute social[1].

According to the WHO, Cranial brain injury affects mainly children, adolescents, as well as adults of working age, which leads to a further search for new solutions to reduce injuries and disabilities in the world's population[7,8,9,10,11].

Most victims with TBI have an age of 20 to 50 years, that is, the period of greatest disability, men are 2.5 times more likely to suffer than women [12].

Cranial brain injury is defined as aggression to the brain caused by external physical force, which can cause a state of reduced or altered consciousness and, therefore, affect cognitive abilities or physical functions. This directly contributes to death from external causes, the main of which are car accidents, falls, aggression and collision of pedestrians [18].

Currently, TBI is considered to be a traumatic brain injury, causing a disturbance in the level of consciousness of patients in 3 - 8 points on the Glasgow coma scale (GCS), when it is assessed at least 6 hours after the injury, under conditions of correction of arterial hypotension, hypoxia and absence any intoxication and hypothermia. In about 50% of cases, there is a combination of TBI with systemic trauma of varying severity. At present, the mortality rate in concomitant TBI reaches 68-80% [5, 6, 7].

And among surviving patients, up to 75% of the victims remain with severe neurological defects. At present, the opinion of all leading specialists in the field of neurotrauma boils down to the following basic concept: brain damage in TBI is determined not only by the primary effect at the time of injury, but also by the action of various damaging factors

during the next hours and days, the so-called factors of secondary brain damage. And if the severity of primary brain damage determines the outcome at the prehospital stage of TBI, then the development and action of secondary damaging factors determines the clinical prognosis and outcome of acute and late periods after TBI. Secondary brain damage may depend on intra- (intracranial hypertension, dislocation syndrome, cerebral vasospasm, convulsions, intracranial infection) and extracranial (arterial hypotension - blood pressure less than 90 mm Hg, hypoxemia - PaCO2 more than 45 mm Hg, severe hypocapnia - PaCO2 less than 30 mm Hg, hyperthermia, hyponatremia, anemia - Ht less than 30%, DIC, hypoglycemia) factors.

Diagnosis of traumatic brain injury

In 1977, a unified classification of closed TBI was adopted, developed at the Leningrad Neurosurgical Institute named after Professor A.L. Polenov. According to this classification, TBI is divided into:

I. Concussion

II. Brain contusion: 1 - mild, 2 - moderate, 3 - severe

III. Compression of the brain against a background of injury

IV. Compression of the brain without injury.

Concussion as a clinical form is characterized by the predominance of functional, reversible changes, which can be judged by the rapid regression of pathological phenomena (after 5-8 days). Typical signs are: short-term impairment of consciousness (20-30 minutes), headache, nausea, vomiting, oligokinesia, pallor, tachycardia or bradycardia, arterial hyper- or hypotension, sometimes bradypnea, as well as retrograde amnesia, difficulty concentrating, weakening the memorization process , horizontal nystagmus, convergence, weakness. Concussion is not accompanied by dysfunction of vital organs.

The diagnosis of a brain contusion is made immediately after the patient is admitted to the hospital. This is a more severe form of TBI, characterized by focal neurological symptoms, varying degrees of severity of cerebral, and in severe cases, stem disorders.

Compression of the brain is characterized by a life-threatening increase in cerebral (the appearance or deepening of impaired consciousness, increased headache, repeated vomiting, the appearance of psychomotor agitation, etc.), focal (the appearance or deepening of hemiparesis, unilateral mydriasis, focal epileptic seizures) and stem (the appearance or deepening bradycardia, increased blood pressure, restriction of gaze upward, tonic spontaneous nystagmus, the occurrence of bilateral pathological signs, etc.) symptoms.

One of the main clinical signs of TBI is impaired consciousness. Different authors approach the assessment of the degree of impairment of consciousness ambiguously. The following forms are most often distinguished [12,13]:

1. Stunning - manifests itself in the form of drowsiness, lethargy with slight depression of reflex activity, there is an orienting reaction, speech contact is difficult, the patient can be brought out of unconsciousness for a short time. According

to the severity of symptoms, moderate and deep stunning are distinguished.

2. Stupor - a deep clouding of consciousness, characterized by a lack of response to the environment, but with preserved reflex activity, there is a reaction to strong sound, light and pain stimuli. This reaction is manifested only by an instant awakening of attention with an inability to perceive and understand what is happening around. Distinguish between stupor with excitement and adynamia.

3. Coma - a state of sharp inhibition of higher nervous activity, manifested in a profound disturbance of consciousness and all analyzers: loss of consciousness, lack of response or sharp suppression of reflexes, visceral pathology. Voice contact is not possible. Distinguish between moderate, deep and transcendental coma.

In the process of developing a traumatic disease of the brain, in the area of its defeat, a complex set of anatomical and pathophysiological processes arises, both from the side of the brain tissue and the vascular system, which leads to impaired functions [17].

In TBI, there is not only a violation of autoregulation of the tone of the microvasculature, accompanied by a change in the density and diameter of the capillaries, but also damage to the blood-brain barrier, leading to cerebral edema [4,16].

These disorders are provoked not only by direct (primary) traumatic effects, but also by secondary factors, among which ischemic complications caused by the influence of vasoactive substances play an important role [14].

The main cause of deaths is the development of cerebral ischemia due to secondary ischemic brain damage [20].

The most significant of these are arterial hypotension and hypoxemia [19].

An acute and severe head injury often causes damage to the basal structures of the brain, involving the hypothalamic-pituitary system, and central-reflex and humoral changes occur throughout the body. The reaction of the sympathetic nervous system prevails, releasing catecholamines into the general bloodstream. As a result of these centrally conditioned reactions, microcirculation disorders occur throughout the body for the first time minutes after an injury.

In severe TBI, these disorders lead to systemic damage to all internal organs, causing multiple organ failure. In this case, changes in the liver that occur are manifested by the corresponding clinical picture [13].

The severity of the clinical course and the high mortality rate of patients with Cranial brain injury are largely due to the development of intracranial and extracranial complications, which are mainly infectious and inflammatory in nature. Clinical studies indicate that the most frequent intracranial complications in patients are arachnoiditis, meningitis, meningoencephalitis, brain abscess.

Among extracranial complications, purulent-inflammatory processes in the lungs most often develop (bronchopneumonia, tracheo-bronchitis,

lung atelectasis, etc.), then in the kidneys (cystopyelitis, renal failure) and in the liver (hepatitis, liver failure) [5].

According to various authors, TBI is accompanied by damage to the liver parenchyma and impaired function [2]. It was found that in the first hours after the injury, small focal necrosis of hepatocytes and changes in the liver microcirculation were noted. The development of circulatory and destructive necrobiotic processes in the liver manifests itself as hypoproteinemia, transient fermentemia, and an increase in the content of glucose and bilirubin in blood serum [3, 4]. However, when assessing morphofunctional changes in the liver during head injury, the degree of damage to the brain is often not taken into account, and the characteristics of changes in the microvasculature of the liver are descriptive. 3-9 days after a head injury, signs of the reverse development of circulatory and destructive necrobiotic processes appear, and by the end of the first month, the reactivity of hepatocytes increases and their saturation with glycogen is restored. These disorders of the liver parenchyma correlate with the dynamics of protein and carbohydrate metabolism in patients. The antitoxic function of the liver is also significantly reduced, which leads to endotoxemia by intermediate metabolic products. In severe TBI with a fatal outcome in the liver, pathomorphological changes progressively develop in the form of discirculatory, metabolic-dystrophic, and destructive-necrotic phenomena. In the early stages after a head injury, in the center of the lobules, discirculatory processes are observed in the form of venous plethora, diapedetic and vasculorexic hemorrhage. From 12 o'clock, dystrophic and necrotic changes in hepatocytes join them, which increase in the following periods and occupy the main area of general pathological changes by 48 and 72 hours after the injury [14,15].

In the terminal period after a severe head injury, morphological changes in the internal organs depend on the time elapsed since the injury. With rapid death, significant pathological changes are not observed with light microscopy.Over longer periods, a morphological picture develops in the form of dystrophic and necrotic changes in hepatocytes. The prevalence of acute discirculatory and hemorrhagic changes in the liver indicates rapid death in TBI, an increase in metabolic dystrophic and necrobiotic phenomena in the center of the lobules corresponds to 24 hours, the picture of "shock liver" in the form massive hemorrhages, centrolobular necrosis, and diffuse disorganization of the stroma-vascular components indicate a 3-4-day trauma. The analysis found that the most serious disorders of protein and carbohydrate metabolism are observed in patients with severe brain damage, which can persist for 10 days from the moment of injury. A violation of the pigment function of the liver occurs in every fifth patient with a head injury with a favorable outcome of the disease and in every second patient who died a day after the injury.An increase in transaminases is typical only for patients with severe TBI, whose activity level decreases with a favorable course of traumatic disease. According to the data, severe TBI is accompanied by an increase in volumetric circulation in sinusoids due to

arterialization of the hepatic blood flow. With severe damage to the brain, a "breakdown" of adaptive arterialization of sinusoidal blood flow and the development of intrahepatic portal hypertension is possible. Severe microcirculatory disorders are accompanied by activation of necrobiotic processes in the liver and serve as the general pathomorphological basis for the development of liver failure in TBI, which is consistent with published data.

Gross acute liver dysfunctions are usually associated with severe hypovolemic shock. Sectional data after prolonged unconscious conditions show that 3 weeks after the injury, almost all patients in this group have pronounced dystrophic changes in all internal organs. This is due to prolonged hypoxic, discirculatory and metabolic disorders at the tissue level. Along with other factors, the cause of the pathology is the emerging chronic decrease in the volume of circulating blood [6]. The basis for the prevention and treatment of systemic lesions of parenchymal organs is the fight against acute and chronic respiratory and circulatory disorders. Of great importance is also infusion therapy to improve the rheological properties of blood [3].

Thus, the consequences after a traumatic brain injury in the liver can be a serious complication.

However, the effect of various periods of traumatic brain injury on the morphology and function of the liver is little known, and further studies are needed to clarify the role of morphological changes in the liver caused by traumatic brain injury. This study seeks to examine whether hepatic changes in people have a traumatic brain injury. In addition, they also examined whether changes in liver morphology caused by stress stimuli, that is, the effect of various periods of traumatic brain injury, remain.

An analysis of the literature showed that in the literature available to us, data on morphological changes in various regions and tissues of the liver during head injury are not given. There are also no morphological changes occurring in the liver, taking into account periods of TBI. There is no data on the simulation of head injury in laboratory animals. All this requires a detailed study of the morphological parameters and changes in the liver during head injury.

Литература:

1. Babayan E., V. Zelman., Yu.S. Polushin, A.B. Schegolev // Anesthesiology and Intensive Care. 2005. - No. 4. - S. 4-14.
2. Badmaeva L.N. Laboratory methods for establishing the prescription of traumatic brain injury in forensic medicine // Sud.-honey. expertise. - 2003. No. 1. - S. 37-39.
3. Belyaevsky A.D., Carmen N.B. and other intensive care for traumatic brain injury. //Toolkit. Rn-D., 2000.-154s.
4. Danielyan M.A. The response of rat brain capillaries to immobilization stress // Morphology. 2007. - T. 132, No. 6. - S. 39-41.

5. Zhanaspaev A.T., Ishmukhamedov R.Sh. Optimization of the treatment of inflammatory complications of traumatic brain injury // Politrauma.- 2008.- No. 1.- P.87-91
6. Ivanov D.E., Puchinyan D.M., Ninel V.G. and other Features of changes in the activity of oxidoreductases, the content of malondialdehyde and molecules of medium blood mass of patients with traumatic brain injury of varying severity // Klin. lab. diagnostics. 2001. - N "5. - S. 40-41.
7. Krylov V.V. Diagnosis and treatment of secondary brain damage. V. Krylov, S.V. Tsarenko // Neurosurgery. 2005. - No. 1. - S. 4-8.
8. Krylov V.V. Principles of monitoring of intracranial pressure / V.V. Krylov, S.S. Petrikov, A.A. Solodov // Annals of clinical and experiment. neurology. 2014 - vol. 8, No. 1 - S.44-48.
9. Oshorov A.V. Monitoring cerebral perfusion pressure in intensive care / A.V. Oshorov, A.S. Goryachev, K.A. Parrots and others. // West. anesthesiology and resuscitation. - 2013. –T.10, No. 2. - S. 52-59.
10. Pronin I.N. Diffusion tensor magnetic resonance imaging in the assessment of the conduction pathways of the brain with diffuse axonal damage / I.N. Pronin, L.M. Fadeeva, N.E. Zakharova et al. // Vestn. X-ray and radiology. - 2009. - No. 4 - S. 12-22.248
11. Pronin I.N. Perfusion CT: Study of cerebral hemodynamics is normal / I.N. Pronin, L.M. Fadeeva, N.E. Zakharova et al. // Medical imaging. - 2007. - No. 3. - S. 8-12.
12. Senchukov C.B. Prehospital phase in the treatment of traumatic brain injury. // Medicine of critical conditions 2004. - No. 5. - P. 33-39
13. Fursov I.V., V.V. Grave, 2013. " Extracranial complications of severe traumatic brain injury "Taurida Medical and Biological Bulletin 2013, Volume 16, No. 3, Part 3 (63)
14. Tsarenko, C. B. Intensive care for diseases and brain damage / C.B. Tsarenko, V.V. Krylov // Neurological journal. 2005. - No. 2. -C. 8-13.
15. Chelnokov V.S., Ilyina E.V. Pathomorphological changes in traumatic brain injury // Sud.-honey. expertise. - 2001. - No. 1. - S. 7-9
16. Shormanoe S.V., Shormanova N.S. Morphometric characteristics of the structures of the human brain under normal conditions and in acute ethanol intoxication // Morphology. 2004.— T. 125, No. 3. - S. 56-60.
17. Armin, S.S. Traumatic subarachnoid hemorrhage: our current understanding and its evolution over the past half century / S.S. Armin, A.R.T. Colohan, J. Zhang // Neurol. Res. 2006. - Vol. 28, No. 4. - P. 445-452.
18. Leite CC, Amaro Jr E, Lucato LT. Neurorradiologia - the diagnosis of environmental diseases. Rio de Janeiro: Guanabara Coogan; 2008.S. 182-214.
19. Regional cerebrovascular and metabolic effects of hyperventilation after severe traumatic brain injury / M.N. Diringer et al. // J Neurosurg. 2002. - Vol. 96.- No. 1. P.103-108;
20. Stollings, J.L. Oxygen therapeutics: oxygen delivery without blood / J.L. Stollings, L.J. Oyen // Pharmacotherapy. 2006. - Vol. 26. - No. 10. - P. 14531464;