COMPARATIVE CHARACTERISTICS OF THE MORPHOLOGICAL PARAMETERS OF THE LIVER AT DIFFERENT PERIODS OF TRAUMATIC BRAIN INJURY

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Annotation: The morphological changes in the liver in traumatic brain injury are described. The research was carried out on 35 white outbred rats of both sexes, 3 months of age. These laboratory rats were trained for 4 days, three times a day. The training consisted of training in finding a platform located on the water's surface in the pool. Further, these outbred rats were fixed on devices prepared by hand in transport on wheels; the fixed animals were accelerated along an inclined plane in a vehicle at a speed of 6.7 km / h and hit a wooden barrier with the frontal part of the head. After receiving a craniocerebral trauma in a day, the experimental rats were immersed in the pool to find a platform with a fixation of the time indicator. Under the influence of craniocerebral trauma, the rats developed pathological processes in the liver tissue plethora of sinusoids of the hepatic lobule, fatty degeneration of hepatocytes with subsequent edema.

Keyword: Rat, liver, training, traumatic brain injury.

Introduction. The problem of severe traumatic brain injury has attracted the attention of researchers for many years. The gigantic scale of modern traumatism has made it not only medical but also acute social [E. Babayan et al. 2005]. According to the WHO, traumatic brain injury was confirmed mainly in children, adolescents, and also the adult population of working age, which leads to a further search for new solutions to reduce the level of injuries and disability of the world's population [V.V. Krylov 2014; S.V. Tsarenko 2005; A.V. Oshorov et al. 2013; I.N. Pronin et al. 2007; V.V. Krylov, S.S. Petrikov, A.A. Solodov 2014; A.V. Oshorov et al. 2013]. Most of the victims with TBI are between 20 and 50 years old, that is, the period of greatest working capacity; men are affected 2.5 times more often than women [C.B. Senchukov 2004].

Traumatic brain injury (TBI) is defined as aggression to the brain caused by external physical force that can cause a state of diminished or altered consciousness and therefore affect cognitive ability or physical function.

In the development of a traumatic brain disease, a complex of anatomical and pathophysiological processes arises in the area of its lesion, both from the brain tissue and the vascular system, which leads to dysfunctions of its functions [SSArmin, ARTColohan, J.Zhang2006]. TBI occurs not only a violation of the 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 [M.A. Danielyan 2007; S.V. Shormanoe; N.S. Shormanov 2004]. These disorders are provoked not only by direct (primary) traumatic effects but also by secondary factors, among which ischemic complications due to the influence of vasoactive substances play an important role [C.B. Tsarenko, V.V. Krylov 2005]. The main

cause of death is the development of cerebral ischemia due to secondary ischemic brain damage [J.L. Stollings, L.J. Oyen 2006; M.N. Diringeretal. 2002].

Acute and severe TBI often causes damage to the basal structures of the brain, with the involvement of the hypothalamic-pituitary system in the process, while central reflex and humoral changes occur throughout the body. The reaction of the sympathetic nervous system predominates, releasing catecholamines into the general bloodstream. As a result of these centrally conditioned reactions, microcirculation disorders throughout the body occur in the first minutes after the injury. In severe TBI, these disorders lead to systemic damage to all internal organs, causing multiple organ failures. In this case, the resulting changes in the liver are manifested by the corresponding clinical picture [IV Fursov, VV Mogila 2013].

PURPOSE OF THE STUDY

To study the morphological changes in the liver in rats in an experiment with traumatic brain injury in combination with injuries of other anatomical areas.

MATERIALS AND METHODS

The experiment was carried out on 3-month-old white outbred rats of both sexes. The quantitative ratio in sex was the same for nine outbred rats. The total number of rats was 35. The average weight value was 120-145 grams. The experiments were carried out in a circular pool with a diameter of 160 cm and a depth of 60 cm. The pool was filled with water up to 55 cm, a platform made of hardwood was placed in the pool, 10 x 10 cm in size, submerged in water by 1-2 cm, the water surface, like the platforms, was Coated with finely crushed foam to make the water surface invisible. All rats were trained 3 times a day for 4 days for several minutes. The first stage of the experiment consisted of submerging rats in water one by one in order for them to find a platform invisible from the outside. In the first days of the first stage of the experiment, the time spent on the platform was on average 150-180 seconds. On the last day, on the 4th day of the experiment, this figure was already 90-120 seconds. After receiving a craniocerebral injury by the model of a "road traffic accident", the results were as follows: on the third day after the injury, the time parameters of the rats for finding the platform were as follows: - 200-240 seconds.

Moreover, in female rats, both training and post-traumatic indicators were 8-10% better than in male rats. In terms of severity, the change in liver tissue. (Table 1).

Table No. 1. Degrees of severity of traumatic brain injury.

Forms	Morphological changes in the liver
Mild TBI	Granular dystrophy persisted in the hepatocytes of the central hepatic lobules.
TBI of moderate severity	Parenchymal edema develops, and severe vascular hyperemia began to increase in the peripheral parts of the liver.
Severe TBI	In severe form, there was an increase in signs

	of granular dystrophy; this process covered large areas of the liver, focal necrosis of hepatic cells.

Conclusion

Experimental results show that with traumatic brain injury, the following changes develop in the liver. Histological changes in the liver were determined by granular dystrophy, parenchymal edema persisted in the hepatocytes of the central hepatic lobules, and in severe TBI, these phenomena began to progress, focal necrosis developed in places.

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