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Comparative Characteristics Of The Morphological Parameters Of The Liver At Different Periods Of Traumatic Brain Injury

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ABSTRACT

The research paper described morphological changes in the liver in traumatic brain injury. The study was conducted on 28 deceased patients, whose death occurred from various causes of traumatic brain injury, that is, traffic accidents, falls from a height and, less often, injuries caused by blunt hard objects. Under the influence of craniocerebral trauma, the deceased developed pathological processes in the liver tissue, plethora of the sinusoid of the hepatic lobule, fatty degeneration of hepatocytes, followed by edema.

KEYWORDS

Liver, Morphology, Traumatic Brain Injury.

INTRODUCTION

The problem of severe traumatic brain injury attracted the attention of researchers for many years. The gigantic scale of modern traumatism had made it not only medical, but

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also acute social (E. Babayan et al., 2005). According to the WHO, traumatic brain injury was confirmed mainly in children, adolescents, as well as the adult population of working age, which determined the 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. Osherov et al,. 2013; I. N. Pronin et al. 2007; V. V. Krylov, S. S. Petrikov, A. A. Solodov, 2014; A. V. Osherov et al., 2013). Most of the victims with TBI were between 20 and 50 years old, that was, the period of greatest working capacity, men were affected 2.5 times more often than women (C.B.Senchukov, 2004). Traumatic brain injury (TBI) was defined as aggression to the brain caused by external physical force that would cause a state of diminished or altered consciousness and therefore affected cognitive ability or physical function.

During the progress of a traumatic brain disease, a complex of anatomical and pathophysiological processes raised in the area of its lesion, both from the brain tissue and the vascular system, which led to dysfunctions of its functions (S.S. Armin, A.R.T. Colohan, J. Zhang, 2006). With TBI, there was not only a violation of the auto regulation 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, led to cerebral edema (M.A. Danielyan, 2007; S.V.Shormanoe; N.S.Shormanova, 2004). These disorders were provoked not only by direct (primary) traumatic effects, but also by secondary factors, among which ischemic complications due to the influence of vasoactive substances played an important role (C.B. Tsarenko, V.V. Krylov, 2005). The main cause of death was 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 caused 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 occurred throughout the body. The reaction of the sympathetic nervous system predominated, releasing catecholamine into the general bloodstream. As a result of these centrally conditioned reactions, microcirculation disorders throughout the body occurred in the first minutes after the injury. In severe TBI, these disorders led to systemic damage to all internal organs, causing multiple organ failure. In this case, the resulting changes in the liver were manifested by the corresponding clinical picture (I.V. Fursov, V.V. Mogila, 2013).

THE AIM OF THE RESEARCH

The purpose of the research was to study the morphological parameters of the liver at different periods of traumatic brain injury, to develop ways of its protection. The work included data from a morphological study of 28 deceased patients, whose death occurred within two weeks after a traumatic brain injury. We studied morphological changes in the liver in deceased traumatic brain injury in combination with damage to other anatomical areas.

MATERIALS AND METHODS

A retrospective analysis of 28 forensic autopsy protocols with a diagnosis of TBI, confirmed on the basis of histological studies, was performed. Autopsy materials were used as research material. For general morphology, 2

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pieces were cut from each tissue, that is, 1.5 × 1.5 cm from the center, middle and peripheral parts of the liver, and solidified in 10% neutral formalin. After washing for 2–4 h in running water, it was dehydrated in concentrated alcohol and chloroform, then embedded in paraffin and prepared blocks. On paraffin blocks, sections of 5-8 µm were cut, stained with hematoxylin and eosin. Semi-thin 1 µm sections were obtained from Epon bricks on a Leykaultramicrotomy. Histological preparations were examined under 10, 20, 40 lenses of a light microscope and the necessary areas were photographed. During morphological examination, the lobular structure of the organ was preserved in all cases. All those who died with a mild form had hyperemia of hepatic venules, and in our observation it was found in 8 people, moderate TBI was found in 11, and severe in 9 deaths. These data indicated that there was a connection between damage to the central nervous system by functional and morphological changes in the liver.

Table 1

The severity of traumatic brain injury

Forms	Morphological changes of the liver
Mild TBI	Granular dystrophy persisted in the hepatocytes of the central hepatic lobules
Moderate TBI	Edema of the parenchyma developed and pronounced full blood vessels began to grow in the peripheral parts of the liver
Severe TBI	In severe cases, there was an increase in signs of granular dystrophy, this process covered large areas of the liver, focal necrosis of hepatic cells joined

According to ethnic groups, there were 18 (64%) men who died, and 10 (36%) women. The average age of these deceased was in our observations from 18 to 54 years, the adult population of working age.

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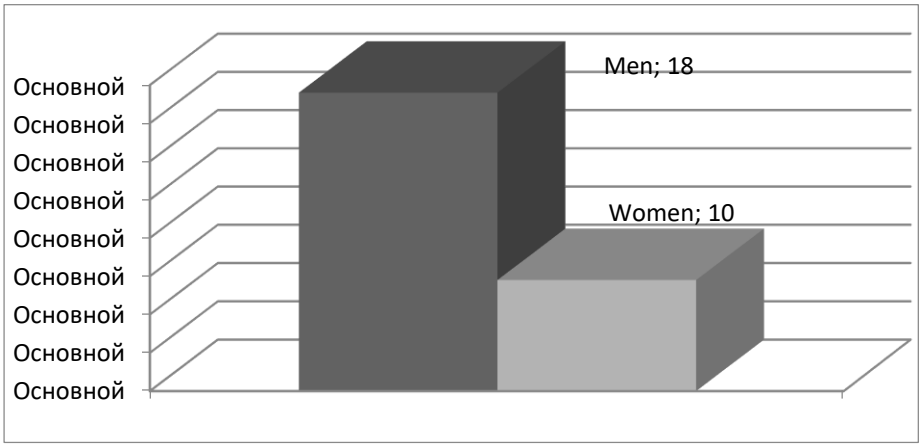

Fig. 1. Diagram of the distribution of cases by sex

CONCLUSION

As our studies showed, the following changes develop in the liver for the first time after a traumatic brain injury in the dead for the first time a day after a traumatic brain injury. Granular dystrophy was determined for histological changes in the liver; parenchymal edema persisted in the hepatocytes of the central hepatic lobules. In patients with long-term trauma, circulatory disorders already caused by trauma became more important, and in the liver tissue in severe TBI, these phenomena began to progress, focal necrosis developed in places. These conclusions might be useful when conducting examinations of victims, deaths in the hospital as a result of TBI.

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