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EVALUATION OF THE PRESCRIPTION OF COMBINED TRAUMATIC BRAIN INJURY IN FORENSIC MEDICAL PRACTICE

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Abstract: Traumatic brain injury is one of the most common types of injuries that lead to death. Injuries to the skull and brain are observed mainly among people aged 20-50 years, i.e. the most socially active category of the population.

The study of this complex problem on forensic material is very valuable, since it preserves the morphological features inherent in normal healthy people in the event of their very rapid death from traumatic brain injury, when the body has not yet had time to develop General (reactive) morphological changes. When death occurs in the near future (hours) after the injury, you can detect signs of the onset of the disease and track their development in dynamics.

Keywords: traumatic brain injury, internal organs, blood loss, morphological changes, brain injury.

Relevance of the problem: Traumatic brain injury is one of the most common types of injuries leading to death. Damage to the skull and brain are observed mainly among people aged 20-50 years, that is, the most socially active population [1,3,5]. Traumatic brain injury, being an extreme irritant, induces a response in the body, which is a complex of various functional and morphological changes in the nervous, endocrine and other systems aimed at maintaining homeostasis [7, 10]. These changes develop in the internal organs in a certain sequence, corresponding to the phases of the general adaptation syndrome, which may be the basis for studying the limitation of the occurrence of mechanical injury [4,13]. The work of forensic doctors shows the morphological changes of some internal organs under various "stressful" influences, including with traumatic brain injury in the early and late stages [3,12]. The study of this complex problem on forensic material is very valuable, since it preserves the morphological characteristics inherent in normal healthy people in the event of a very quick death from traumatic brain injury, when the general (reactive) morphological processes in the body have not yet managed to develop changes. When death occurs in the near future (hours) after an injury, you can detect signs of the onset of the disease and track their development in dynamics [2, 8, 9, 11,14].

An experiment of this kind, injuries provides an opportunity to exclude a number of pathogenetic factors, which are not always possible to take into account in forensic practice, and to clarify the dynamics of the development of morphological changes in the area of direct mechanical impact and away from it, that is, in the internal organs, which is especially important in the early stages. For the forensic medical examination, the determination of the time elapsed from the moment of causing a traumatic brain injury to the death of the injured, that is, the limitation of the damage, is of great practical importance.

The purpose of research.To develop an algorithm for determining the prescription of traumatic brain injury in forensic practice.

Object of study: The object of the study was 50 expert cases of isolated and combined

with other injuries of a traumatic brain injury.

Research Methods. Morphological, histological and statistical research methods were used to solve the tasks.

Results of the study: Analysis of fatal traumatic brain injuries showed that isolated head injuries were mainly characterized by hemorrhages in the membranes and brain matter, contusion foci of softening. In most cases, foci of hemorrhage (subdural, epidural, subarachnoid) were localized in the parieto-occipital region, and softening was located in the midbrain, pons, the interstitial, medulla oblongata, and at the base of the brain.

Bruises of the basal surface of the frontal lobes were usually combined with foci of bruises of the pole and basal sections of the temporal lobes. They were often accompanied by subarachnoid and subdural hemorrhages.

The dependence of the location on the zone of mechanical impact was as follows: often brain damage was localized in places of shock, and later on of its impact on the inner surface of the skull, less often in places of impact, its main reason was the mixing of fragments of the bones of the skull with further damage to the brain tissue.

The zone of contusion of the brain tissue was characterized by focal crushing of the cortex with impregnation of its blood, after some time necrotic changes in this focus were revealed in the form of hemorrhagic softening.

In some cases, the lesion zone was focal hemorrhage and crush of the cortex, occupying the entire surface of the lobe of the brain or a group of its lobes; also small focal hemorrhages and crush of the cortex were located on the convex surface of the gyrus or spread to several gyruses.

In some works, it is noted that in acute blood loss there is a redistribution of cerebral blood flow with a significant deterioration in the blood supply to the diencephalon (primarily the thalamus and hypothalamus), as well as the lower sections of the trunk and cervical spinal cord. The preoptic region of the hypothalamus (paraventricular tissue around the anterior wall of the 3rd ventricle) is particularly damaged.

In addition to hemodynamic, there is cytotoxic edema of the brain. At the same time, an increase in the content of intracellular fluid is observed secondarily after cellular damage. An important role is played by osmotic, biochemical, rheological and other factors causing damage to brain cells. This edema is often called brain swelling. However, this distinction is conditional, since most often there is a combination of edema and brain swelling.

In cases of instant death after a head injury, less often at the site of the impact, more often at the site of the counterattack, barely visible foci of blue-violet color were determined, which were often combined with small point hemorrhages. Focal subarachnoid hemorrhages were also noted.

When fatal in 10-40 minutes after receiving the injury, the foci of the bruises of the tissue were dark red in color, with a more thorough examination it turned out that they are formed due to the merger of small hemorrhages. They were mainly located on ridges, and sometimes deep in the furrows. They were single (42.14%) or multiple (17.14%) and had different sizes. In some cases, signs of perifocal, and sometimes general cerebral edema have already been detected.

When death occurred 2 hours after receiving a head injury in the foci of concussion, noticeable swelling of the injured convolutions and fusion of average hemorrhages were observed. In deaths, after 3-6 hours after the injury, necrotic phenomena were observed at the top of the convolutions at the sites of foci of hemorrhage, which was especially pronounced in violation of the integrity of the pia mater and crushing of the cortex.

After 12-24 hours, in most cases, the foci of hemorrhage in the affected area were dark red in color, the surface of the bruise was isolated, slightly sagging, becoming a

granular, dull mass with a grayish tint, around which there were scattered point hemorrhages. In the section, they had the appearance of limited hemorrhages extending to all layers of the cortex to the subcortical layer, thereby resembling the shape of a wedge, the base of which was the surface of the brain.

In the interval of the first few days (1-4 days) after the injury, these changes became even more pronounced, foci of necrosis were saturated with blood, acquired a grayish-red color and, dull, spread to a white substance. The tissue around the hemorrhage is strongly edematous; when cut, it protrudes above the surface.

In some cases, the 4-8-day period was characterized by a change in the shade of hemorrhage from dark cherry to almost black, the hematoma had a layered structure - dark red on the periphery and dark brown in the center. In almost all cases, when death occurred 6-8 days after the injury, liquefaction of the central parts of the hematomas was observed, where a dark brown liquid was determined. Over the entire area of damage to the brain tissue, local edema developed perifocally, which occurred in the first minutes and hours after the damage. The phenomena of edema increased within 3-8 days, and at the same time, the permeability of the vascular wall increased.

Edema persisted for 10 days, accompanied by processes of resorption of foci of necrosis and hemorrhage.

In cases of instant death with a traumatic brain injury in the central part of the damage, the brain substance is presented in the form of a liquid gruel-like mass mixed with blood. Torn hard and soft meninges are located on the surface of the brain wound. Directly in the circumference of the wound cavity, the brain tissue is pale and dry. At the same time, one of the characteristic signs is that in the surrounding tissue of the wound cavity, blood imitation is not expressed. Microscopic examination shows that the contents of the wound cavity consists of the constituent parts of the spilled blood and tissue decays.

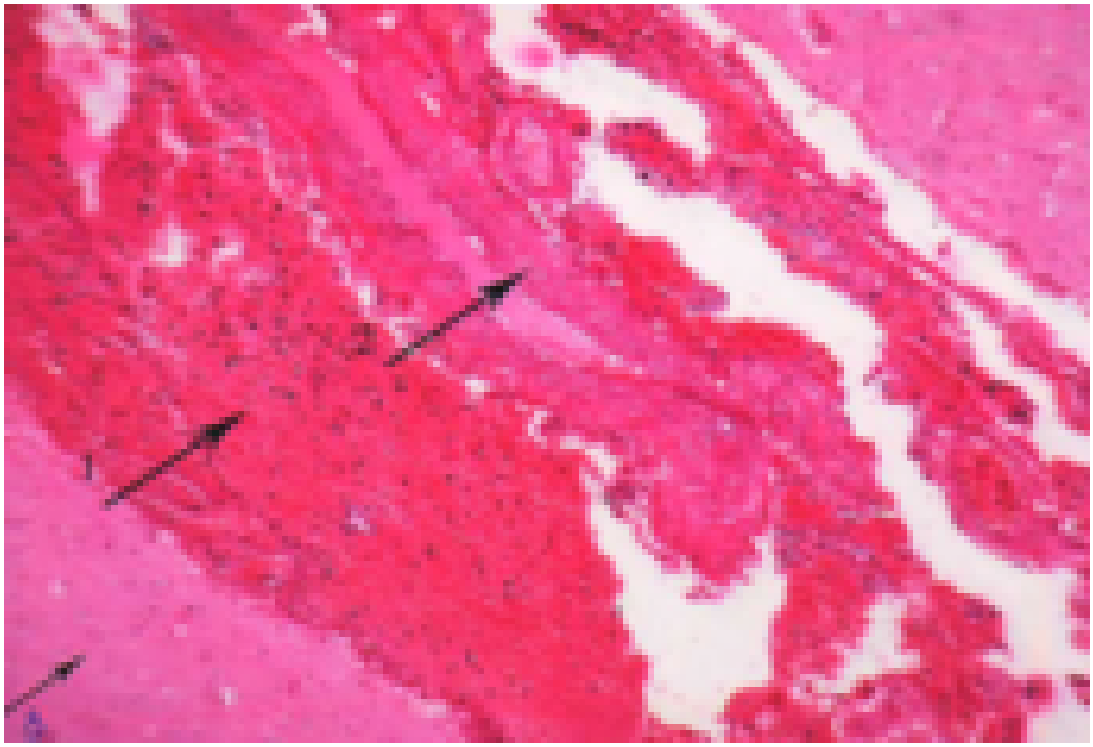

Fig. 1. Victim M. Age 22 years. Sec mat. No. 27 (protocol 635). Immediate death after craniocerebral injury. The hemorrhage center is represented by blood mass, scraps of nervous tissue. Hematoxylin and eosin stain. At century 10x20. 1. Blood masses. 2. Scraps of nerve tissue. 3. The surrounding tissue.

In the circumference of the brain wound, the arteries are slept, bloodless, veins are moderately dilated, stasis in the capillaries. From the side of neuroectodermal tissue structures, no visible changes were detected.

In case of death 2 hours after a head injury, fragments of crushing of brain tissue, liquid blood and blood clots are determined in the center of damage. In the area of contusion, the brain tissue is in a state of developing necrosis and hemorrhage. In this zone, the presence of multiple foci of hemorrhagic softening of brain tissue is noted.

In distant areas from the wound site or in the area of molecular concussion, discirculatory disorders predominate in the form of paretic expansion of small vessels with stasis and diapedetic hemorrhage. From the side of nerve cells, a small pericellular edema, wrinkling of the nuclei and focal tigrolysis are determined. In the circle of ganglion cells, almost all oligodendrogliocytes are in a state of edema. The intercellular medulla is somewhat loosened and edematous.

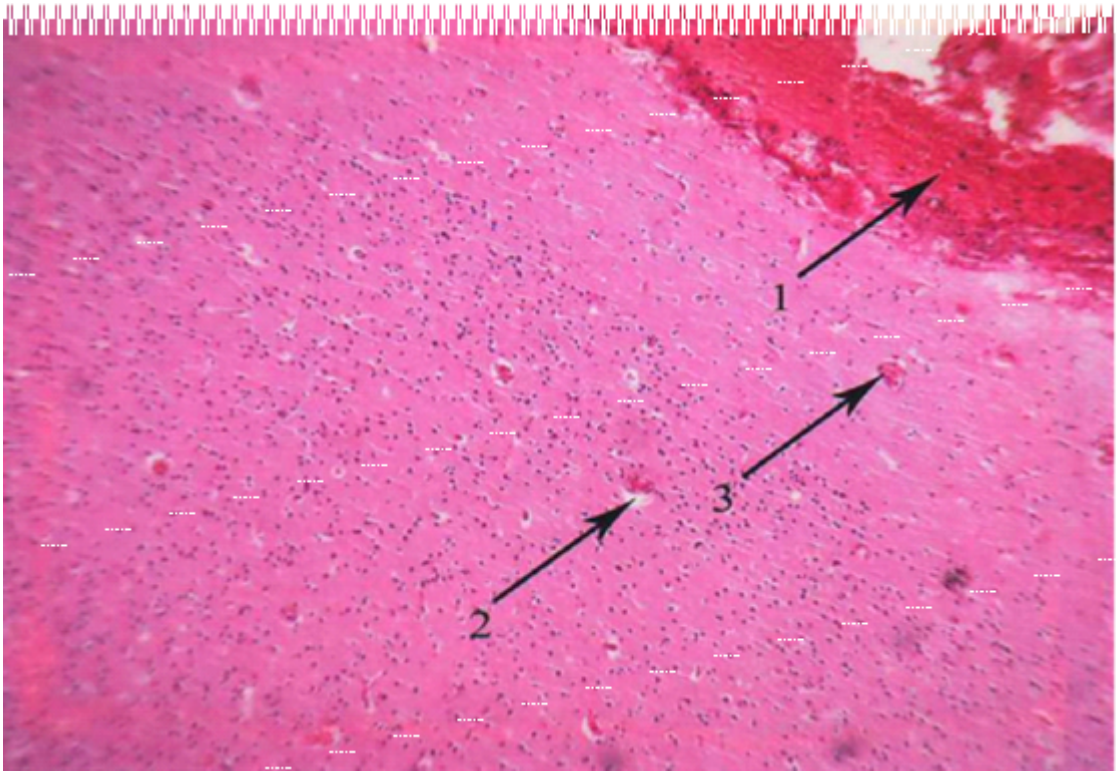

Fig. 2. Victim M. Age 36 years. Sec mat. No. 37 (protocol No. 960). Death 2 hours after Craniocerebral Injury. In the circumference of the hemorrhage focus, the arteries are spasmodic with swelling edema, veins with paretic expansion. Hematoxylin and eosin stain. At century 10x20.1. The center of hemorrhage. 2. Spasm of the artery with perivascular edema. 3. Paretic expansion of veins.

Pathomorphological changes revealed in craniocerebral injuries in various topographic areas of brain damage develop with a certain sequence depending on the period that has passed after the injury. The intensity of these changes is due to both the strength and the nature of the mechanical effect. In the early stages after Craniocerebral Injuries, if a bloody-necrotic crushed mass forms directly in the wound cavity, then discirculatory phenomena predominate in the circumference of the lesion. In the subsequent periods after Craniocerebral Injuries, secondary hemorrhagic and destructive changes develop in areas of concussion and molecular concussion due to discirculatory processes, and pronounced hyperemic and edematous phenomena develop in remote areas of the brain.

2-3 days after Craniocerebral Injuries, proliferative phenomena from both vascular and neuroectodermal tissue elements join these changes in order to develop aseptic inflammation and cleanse the focus of brain damage.

Conclusions: In general, a review of research data published in the modern scientific literature on traumatic brain injuries showed that despite the urgency of this problem and the huge number of studies in this area, there is still no sufficient unification of concepts, terms and classifications, as in clinical medicine , and in forensic practice. While the development of unified protocols for clinical trials, diagnosis, treatment and prognosis of head injuries, the standard of treatment, a unified diagnostic algorithm will undoubtedly contribute to a significant reduction in mortality from head injuries, and the development of a single set of expert criteria for assessing fatal head injuries injuries, will avoid a number of mistakes and appointments of repeated forensic medical examinations in cases of such injuries.

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