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PATHOGENETIC APPROACH TO ASSESSING THE SEVERITY OF TRAUMATIC BRAIN INJURY AND ITS CRITERIA.

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Relevance of the Topic. Considering the widespread prevalence of traumatic brain injuries (TBI) and their complications, many specialists emphasize the importance of identifying their early signs at the primary healthcare level, implementing measures for their correction, and preventing the development of secondary injury manifestations. The evolving nature of various morphological signs of this disease highlights the urgency of training general practitioners with up-to-date knowledge.

Objective. To investigate the primary and secondary mechanisms of injury in a model of blunt traumatic brain injury (TBI) in rats and to confirm the induction of injury. Primary injury mechanisms occur at the moment of trauma, i.e., immediately upon impact of a blunt object on the rat's brain. These include hemorrhage (rupture of intracranial blood vessels), cerebral concussion (temporary functional disruption of internal brain structures and cells due to impact), and cerebral contusion (physical crushing of brain tissue and cell death). Secondary injury mechanisms are processes that develop minutes, hours, or days later. These include increased intracranial pressure (disruption of cerebrospinal fluid circulation), ischemia and hypotensive injury (due to decreased blood pressure or narrowing of cerebral vessels after trauma), and neuroinflammation (cell death resulting from inflammatory processes).

Materials and Methods.

Sixty adult male rats were selected for the study. The rats were healthy and had not participated in any previous experiments. They were divided into groups: a control group (no injury) and an injured group. The injury was induced using the Weight Drop model. For this, pipes of 50 cm height and various diameters (20, 25, 32 mm) were selected. Stones of corresponding weights (66, 50, 22, 10 grams) were chosen to match the pipe diameters. The experiment was observed for 7 days.The rats' heads were fixed on a soft cushion, which allowed for precise modeling of the injury induction process. After inducing the injury, the rats' reflexes were examined. These neurological assessment tests primarily focused on motor function. The following reflexes were tested: limb flexion when lifted by the tail, walking on a flat surface, startle response to sudden noise, pinna reflex, paw withdrawal reflex upon pinching, walking on a wooden beam, and corneal reflex. The initial blood sample was collected 24 hours after injury (at the peak of NSE protein). The next sample was collected on day 7 (at the end of the acute phase). This allowed for the observation of changes during the acute phase of the injury.

Conclusion. As a result of studies conducted on rats, it was initially observed that blunt traumatic brain injury (TBI) led to impaired reflexes and difficulties in maintaining balance in the rats. When assessed using the Neurological Severity Score (NSS) developed by E. Shohami and J. Chen, rats subjected to a 66g weight drop exhibited a neurological deficit ranging from 13-18 points. This scale is considered an adapted analog of the Glasgow Coma Scale (GCS) for laboratory animals. Through this scale, the presence of initial neuromotor deficits in the animals was confirmed.

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The next step involved blood analysis, where changes in the blood indicated ongoing inflammatory processes in the rats, meaning that by examining neurospecific enolase (NSE), it was possible to observe the death of brain neurons. Secondary injury, involving processes such as ischemia (impaired blood supply), cerebral edema, inflammation, and excitotoxicity, developed within several hours and days after the impact, leading to further neuronal death. NSE was released into the blood as a result of neuronal necrosis or apoptosis.

Through this work, by examining neuromotor function, we identified neurological deficits, which indicated the presence of primary injury. The increase in blood proteins (NSE) demonstrated that secondary injury processes were ongoing.