

## Forensic Medical Assessment of Cranio Brain Injuries

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**Annotation.** According to an analysis of the main statistical indicators, in recent years there have been serious quantitative and qualitative changes in the structure of traumatic brain injury, consisting of an increase in the number of victims with severe, life-threatening injuries. When investigating crimes against human life and health, an important place belongs to forensic medical examination, without which it is impossible to establish the mechanism and duration of injury, the nature and severity of the damage.

**Key words:** traumatic brain injury, forensic medicine, morphology, structure, expert assessment.

**Relevance.** Traumatic brain injury (TBI) is one of the main causes of mortality and disability in the population, and among young people, head injury ranks first in the structure of mortality [1, 2]. Every year, 1.5 million people worldwide die from traumatic brain injury, and 2.4 million become disabled. Traumatic brain injury, accounting for up to 30-50% of all traumatic injuries, remains an urgent problem in modern neurology, neurosurgery and forensic medicine. According to the World Health Organization (WHO), the incidence of TBI reaches 1.8-5.4 cases per 10,000 population and tends to increase by an average of 2% per year. The contingent of victims is predominantly young and middle-aged people of the most working age from 20 to 50 years [3, 4]. Most studies conducted on TBI look at the structure of the traumatic brain injury to some extent. As follows from the publications of these studies, the main part of cerebral traumatism is made up of mild closed craniocerebral injuries, which include concussion and mild brain contusion, and concussion, in turn, accounts for from 63% to 90% of their structure. .5% [7]. In developed countries such as the United States and Europe, mild traumatic brain injury, which is primarily based on concussion (MC), accounts for 60% to 90% of all victims with TBI [9, 10].

Mortality from traumatic brain injury (TBI) traditionally remains high [5, 8]. The literature describes morphological markers of TBI that vary depending on the period of its occurrence. The temporal dynamics in the area of damage to the brain and its membranes during TBI in the early stages after its occurrence, as well as the immediate causes of death at the scene of the incident, are relatively fully described. But the issues of the temporal dynamics of the immediate causes of death during TBI have been studied much less well. At the same time, it is these, often extracranial, causes that lead to death after hospitalization of patients with TBI. Our study made an attempt to decipher thanatogenesis in in-hospital TBI. We studied 70 cases of fatal TBI, distributed sequentially according to the time of death from 12 hours to 2 months. The dead were men (40 observations) and women (30 observations) aged from 14 to 72 years. Most often, TBI was associated with a motor vehicle injury (67%), less often there was a fall from a height and other types of TBI (railroad rail injury; injuries caused by hard blunt objects, etc.). Brain contusions (45 observations), isolated intrathecal hematomas (14 observations), as well as various combinations

thereof were observed. Alchohemia occurred in 25 observations, and the ethanol content in the blood varied from 2.8 to 6%; in the urine, the level of ethanol in these observations ranged from 2 to 5%. In 2 cases, death occurred due to an epileptic seizure as a result of the development of intrathecal hematomas after TBI. Death occurred in the first 12-15 hours after injury (28 observations), on the 1st day (26 observations), and at a later date (16 observations). The maximum period was 62 days. The selection criterion in this group was hospitalization of the victims, i.e. death at the scene was excluded. Methods of targeted study of brain stem sections were used [4], polarization microscopy of heart specimens, as well as standard methods of sectional and histological examination, supplemented by semi-quantitative technologies of thanatogenetic analysis [1,2]. In some observations, changes in the area of brain damage and its membranes, characteristic of the periods studied, were naturally observed. We do not describe them here, since they were generally consistent with the literature data [6, 7].

On the 1st day after the injury in the group of hospital mortality, the immediate immediate cause of death was cerebral edema, which developed as a reaction to a bruise or as a consequence of large intrathecal hemorrhages. An increase in pulmonary edema was also noted, the degree of which correlated with the duration of the agonal period. In the lungs at these times, a picture of uneven plethora, aggregation of erythrocytes in venules, stromal and focal intraalveolar edema with effusion of fibrin into the alveoli, and focal formation of hyaline membranes was observed. A focus of initial leukocyte infiltration of the bronchial walls, hypersecretion of mucus by the bronchial glands and swelling of the mucous membrane of the large bronchi were noted. At the same time, a picture of distributive leukocytosis was observed [7].

The morphological picture of the lungs was consistent with pulmonary respiratory distress syndrome. Thus, in the early stages, intracerebral mechanisms of death dominate in the form of cerebral edema, secondary hemorrhages in its brainstem, and compression of the brain substance by growing subdural hematomas. Subsequently, from 1 day to 2 weeks, the development of focal bronchopneumonia in the form of acinar, then subsegmental, segmental and, finally, confluent with frequent abscess formation is naturally observed. At the same time, natural dynamics are observed in the area of the TBI itself - initial phenomena of resorption of necrotic brain tissue, the appearance of an active vascular-mesenchymal and glial reaction to damage, resorption of hemorrhages (hemosiderosis from the 3rd day), compensatory hypertrophy of neurons surrounding the area of damage and hyperplasia of gliocytes.

At later stages the picture becomes polymorphic. There are cases of progressive abscess pneumonia leading to death through respiratory failure, as well as purulent-septic complications with the development of ascending uroinfection, bacterial endocarditis, and even a picture of pyaemia with the development of septic metastases and a primary focus in the form of purulent-hemorrhagic cystitis and pyelitis. Septic complications included abscessing pyelonephritis and purulent meningitis. The so-called “vegetative status” was also observed in the outcome of severe uncompensated TBI [8, 9]. We had two such observations - death occurred at the end of the 2nd month after the injury with symptoms of neurotrophic cachexia against the background of the so-called “respiratory brain”, diffuse cerebral gliosis, areas of partial and subtotal necrosis of brain tissue. Single observations relate to death from epistatus with the development of post-traumatic cortical gliomesodermal adhesions with the clinical picture of post-traumatic epilepsy.

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