FORENSIC ASPECTS OF DIAGNOSING DIFFUSE AXONAL BRAIN DAMAGE IN TRAUMATIC BRAIN INJURIES

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https://doi.org/10.5281/zenodo.11410732

Abstract*. Diffuse axonal brain injury is a variant of severe traumatic brain injury, the main substrate of which is diffuse ruptures or tears of axons. Clinically, from the first minutes of injury, a coma with symptoms of brain stem damage is observed, which can turn into a vegetative state. Diagnosis is carried out based on traumatic history, clinical features and tomographic data. Treatment of coma consists of mechanical ventilation and intensive therapy; after recovery from coma, vascular, nootropic, metabolic drugs, physical therapy, psychostimulation, and speech therapy correction are used.*

Keywords*: TBI (traumatic brain injury), cranial injury, shock, DAP (Diffuse axonal damage).*

Relevance. Unfortunately, to date there is no clear definition of the concept of “traumatic brain injury” that would reveal its content, scope and establish its meaning. Oddly enough, in many domestic textbooks, manuals and monographs on neurosurgery and forensic medicine, the definition of cranial injury is either absent altogether, or it is stated that “the meaning of the term TBI itself There's probably no need to explain..."Foreign authors more often use the terms Traumatic Brain Injury, sometimes head injury or cranial trauma (cranial injury). Traumatic brain injury (TBI) is a combination of injuries to the soft coverings of the head, skull, membranes and brain tissue. A logical analysis of these definitions shows their incommensurability, their scope is either wider, narrower than the scope of the concept being defined, or generally not clearly defined. From them, it is not entirely clear the presence of what kind of head injuries gives grounds to consider them a traumatic brain injury, and what head injuries should be included in this diagnosis.

The purpose of this study is to develop clinical and morphological criteria for expert assessment of DAP in traumatic brain injuries In this regard, a number of clinicians, especially in pediatric practice, consider it possible to establish a diagnosis of TBI in the presence of even small superficial injuries on the head, while others, on the contrary, classify only damage to the skull and intracranial contents as TBI, ignoring damage to the skin. A similar situation can be observed in forensic medical practice - some experts collectively estimate all injuries on the head as a single complex, while some evaluate each injury on the head in isolation from the others.

Materials and methods of research. Traumatic brain injury (TBI) is an optional set of injuries intracranial formations (brain substance, meninges, vessels, cranial nerves) and fractures of the bones of the skull (vault or base), as well as associated damage to the soft tissues of the head and facial bones, which occurs both due to trauma to the head itself and other parts of the body (by an indirect mechanism). Damage to the soft tissues of the head and bones of the face due to repeated impacts on the head should be included in the complex of traumatic brain injury in those cases when they are associated with skull fractures or intracerebral injuries by a single mechanism, i.e. they are localized in areas of application of traumatic forces that led to the formation of the

main elements of TBI, or located along the course of their action vector. If superficial injuries to the face differ in location from injuries to the skull and intracerebral structures or the vector the action of the force that led to their formation does not coincide with the force vector, formed the components of TBI.

Classification is a necessary basis for both scientific generalization and quantitative study of any phenomenon. In relation to the cranial trauma, it is necessary to unify the expert assessment of head injuries, ensuring uniformity of diagnosis formulations, the use of modern terminology by experts, for an adequate comparison of clinical and forensic diagnoses. There are numerous classifications of traumatic brain injury, developed in different countries and by different authors, which, for all their value, are often built on opposing principles, are either overly broad, or fragmentary, sometimes based on the use of individual, although extremely important signs, sometimes contradictory in the interpretation of the same defeats.

Based on biomechanics, TBI is classified into:

 shock-anti-shock (shock wave propagating from the place application of a traumatic agent to the head through the brain to the opposite pole with rapid pressure changes at the points of impact and counter-impact);

 acceleration-deceleration (movement and rotation of massive cerebral hemispheres relative to a more fixed brain stem);

 combined (when both mechanisms act simultaneously)

By type of damage there are:

 focal, caused predominantly by impact-shock trauma (characterized by local macrostructural damage to the brain

substances of varying degrees, including areas of destruction with the formation of detritus,

hemorrhagic impregnation of brain tissue, pinpoint, small and large focal hemorrhages - at the site of impact, counter-impact, along the shock wave),

 diffuse, predominantly trauma-induced acceleration and deceleration (characterized by transient asynapsia, tension and widespread primary and secondary axonal breaks in the centrum semiovale, subcortical formations, corpus callosum, brain stem, as well as point

and small focal hemorrhages in the same structures);

 combined, when there are simultaneously focal and diffuse brain damage. According to the genesis of brain damage, TBI is differentiated:

 primary lesions - focal contusions and crushes of the brain, diffuse axonal damage, primary intracranial hematomas, ruptures trunk, multiple intracerebral hemorrhages;

 secondary lesions:

a) due to secondary intracranial factors - delayed hematomas

(epidural, subdural, intracerebral), hemo- and liquor circulation disorders as a result of subarachnoid or intraventricular hemorrhage,an increase in brain volume or swelling due to edema, hyperemia or venous congestion, intracranial infection, etc.;

b) due to secondary extracranial factors: arterial hypotension, hypoxemia, hypercapnia, anemia and others.

In this regard, it can be considered an evolutionarily predetermined and genetically fixed process in response to damage to the brain, its integument and intracranial vessels. That is, if you follow the logic of the definition given by the authors of this classification, brain compression is

not a separate form of damage, but a consequence one or another injury - brain contusion, intracranial hematomas, depressed fractures, etc.

Researches and discussion. DAP is supported by the mechanism of the resulting TBI with angular acceleration of the head, the onset of coma immediately after the injury and the characteristic features of its clinic. A distinctive feature is also the absence of congestive changes in the optic discs during ophthalmoscopy of patients with DAP, even in the case of prolonged coma. However, it is quite difficult for a neurologist and traumatologist to diagnose DAP clinically. A reliable diagnosis of "Diffuse axonal damage" can be made by the presence of pathognomonic macroscopic signs on an MRI or CT scan of the brain. CT and MRI of the brain in the acute period reveals cerebral edema with a decrease in the ventricles and subarachnoid spaces; fluid accumulation is often visualized above the frontal lobes. More difficult is the diagnosis of mild and moderate degrees of DAP, in which macroscopic signs are usually absent, cerebral edema and hemorrhages are mild, and in some cases the tomographic picture does not differ significantly from the norm. In such a situation, they rely on typical tomographic dynamics - regression of hemorrhages and edema with a tendency to dilate the ventricles, subsequent ventriculomegaly and an increase in diffuse atrophy of cerebral structures.

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