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FORENSIC EVALUATION OF DIFFUSE AXONAL INJURY

Iskandarov Alisher Iskandarovich
MD professor

Yakubov Khayot Hamidullaevich
Candidate of Medical Sciences

Ismatov Abrorkhon Askarovich
Assistant

Abstract: *Sudden damage to the bones of the skull and brain by various mechanical agents leads to craniocerebral injuries. Diffuse damage to axons occurs as a result of a closed brain injury, as a result of damage to the axons of neurons. This leads to brain injury, disability and, in the worst case, death.*

Keywords: *diffuse axonal injury, immunohistochemical reaction, white matter, corpus callosum.*

Diffuse axonal damage affects the white matter tracts of the brain. Clinically, patients may present with a spectrum of different neurological dysfunctions. This can range from mild to comatose. On examination, it measures the following three functions: eye openness, verbal response, and motor movements. According to their activity, the patient's condition is assessed. During accidents, acceleration and deceleration of movement, which leads to shear forces in the white matter tracts of the brain. This leads to microscopic and gross damage to the axons of the brain at the junction of gray and white matter. Diffuse axonal injury usually affects the white matter tracts of the corpus callosum and brainstem. Primary diffuse damage to axons leads to disconnection or disruption of the interconnection of neurons. It affects numerous functional areas of the brain. Typically, patients with diffuse axonal injury present with bilateral deficits in neurologic examination, often affecting the frontal and temporal white matter, corpus callosum, and brainstem. The Adams classification for diffuse axonal injury uses the pathophysiological lesions of the white matter tracts and the clinical presentation. Classification of diffuse axonal Adams lesions

- Grade 1: mild diffuse axonal injury with microscopic changes in the white matter of the cerebral cortex, corpus callosum and brainstem.
- Grade 2: Moderate diffuse axonal injury with coarse focal lesions of the corpus callosum.
- Grade 3: severe diffuse axonal injury with grade 2 detection and additional focal lesions in the brainstem.

The axonal parts of neurons have mechanical destruction of the cytoskeleton, which leads to proteolysis, edema, and other microscopic and molecular changes in the structure of neurons. Brain damage at different levels is reflected in the symptoms. Symptoms may include dizziness, nausea, vomiting, and fatigue. However, patients with severe diffuse axonal injury may also experience loss of consciousness and remain in a persistent vegetative state. A very small number of patients with severe diffuse axonal injury regain

consciousness within the first year after injury. Autonomic symptoms typically include tachycardia, tachypnea, sweating, loss of vascular tone, hyperthermia, and abnormal muscle tone. Often there are pyramidal-extrapyramidal paralysis of the extremities, asymmetric paresis is characteristic. Vegetative disorders appear brightly: arterial hypertension, high temperature, hyperhidrosis (sweating), hypersalivation (increased salivation). For histological comparison, a set of staining methods is proposed, which should be used taking into account the timing of the post-traumatic period. The main ones are techniques that allow detecting changes in axial cylinders - silver impregnation according to Bilshovsky or Glies, myelin sheaths - osmium impregnation according to Marks to detect early demyelination, Spielmeier stain to detect late demyelination. If necessary, examine the bodies of neurocytes - staining with hematoxylin and eosin, according to Nissl, microglia and astroglia. Careful microscopic examination using neurohistological techniques in full allows us to determine the morphological substrate of diffuse axonal damage in the form of many axonal balls. They were located everywhere in the white matter of the brain and were direct evidence of mechanical damage to the axons, because when the nerve fiber is interrupted, the axoplasm flows out of the ends of the damaged process and their club-shaped thickening occurs. After the rupture of the nerve fiber, its distal part undergoes complete degeneration. This process is referred to as the Wallerian rebirth. In the area of the axon located proximal to the site of damage, retrograde changes or the so-called retrograde degeneration are observed, which spreads in the central direction towards the cell body. Histological studies of the brain indicate that damage to axons in all cases was localized in the brainstem, corpus callosum, in the area of internal capsules, even when macroscopic changes were not found in these structures. In the first days of white matter lesions, multiple axonal balls were found. Near them, axons showed signs of initial degeneration, were uneven, swollen, and perceived color unevenly. Later, by the end of the 1st week, degenerative changes spread along the entire length of the damaged axon. Nerve fibers had a tortuous appearance, varicose thickenings, fragmented into sections of various lengths. According to their contours, a large number of small fat granules of degenerating myelin were determined. Studies of neurocytes did not reveal structural changes. During the second week after injury, in addition to the described changes, signs of secondary degeneration of the white matter (axons intact at the time of injury) along the conduction tracts of the central nervous system near the rupture site were detected. In places of primary axon breaks, a moderate macrophage reaction with the formation of granular balls was determined. When experiencing trauma, a decrease was observed, and by the end of the first month, the complete disappearance of axonal balls. The remains of damaged axons fragmented into smaller ones and gradually disappeared. Diffuse proliferation of macrophages was noted, which were loaded with granules of decaying myelin. At the same time, the secondary degeneration of nerve fibers along the conduction tracts became more pronounced. Depending on the duration of the post-traumatic period, changes in the bodies of neurocytes were determined from an “axonal reaction” (“primary irritation”) to wrinkling, ischemic changes, or severe illness. Thus, it has been proven that the fact of the existence of diffuse axonal injury as a special form of cerebral injury is beyond doubt. These data indicate the traumatic nature of damage to nerve fibers and delayed (secondary) damage to neurons. With the advent of positron emission tomography in clinical practice, intravital diagnosis of primary axonotomy became possible, not only in diffuse axonal injury, but also in other forms of TBI, the genesis of which is associated with inertial displacement of the brain in the cranial cavity. It should be noted that the information obtained fully helps to make a diagnosis in the forensic evaluation of this condition. For quantitative analysis use every fifth slice through the ganglion. X-ray computed tomography, magnetic resonance imaging of the brain with small pinpoint hemorrhages in the white matter tracts indicate diffuse damage to axons in the conditions of the corresponding clinical picture. There are currently no laboratory data available for diagnosis.

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