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Research Article

FORENSIC EVALUATION OF DIFFUSE AXONAL INJURY

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ABSTRACT

Traumatic brain injury is a sudden damage to the bones of the skull and brain by various mechanical agents. Diffuse axonal injury is a type of traumatic brain injury resulting from a closed brain injury. Traumatic brain injury is the leading cause of death and disability worldwide.

KEYWORDS

Diffuse axonal injury, immunohistochemical reaction, white matter, corpus callosum.

INTRODUCTION

Diffuse axonal injury of the brain is diffuse damage to the axons and affects the white matter tracts of the brain. Its development is based on the tension and rupture of axons (long processes of nerve cells) in the white matter of the hemispheres and brain stem. This type of injury is characterized by a long multi-day coma

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from the moment of injury. In this case, the following symptoms are expressed: paresis of the reflex upward gaze, eye separation along the vertical or horizontal axis, bilateral depression or loss of pupillary reaction to light. Gross violations of the frequency and rhythm of breathing are often observed. At the same time, changes in muscle tone are extremely diverse, mainly in the form of diffuse hypotension. Pyramid-extrapyramidal paralysis of the extremities often found, asymmetric paresis is characteristic. Vegetative disorders are prominent: arterial hypertension, high fever, hyperhidrosis (sweating), hypersalivation (increased salivation). A distinctive feature of the clinical course of diffuse axonal damage is the transition from a prolonged coma to a persistent vegetative state, the onset of which is evidenced by spontaneous opening of the eyes or in response to various stimuli, but there are no signs of tracking, fixing the gaze, or following at least elementary instructions. The vegetative state lasts from several days to several months and is characterized by the appearance of a new class of neurological signs - symptoms of functional or anatomical separation of the cerebral hemispheres and the brain stem.

The largest white matter tract, the corpus callosum, is particularly vulnerable to diffuse axonal injury. The fibers of the corpus callosum have high anisotropy and unidirectional coherence. The study of its damage is important given the enormous role of this structure in providing interhemispheric transmission of auditory, visual, sensory and motor information related to a variety of cognitive processes. The most common mechanism involves acceleration and deceleration of movement, resulting in 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. 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. Comparative analyzes of data from patients in a coma with severe diffuse axonal damage, the results of the analysis show that extensive changes in the structure of the corpus callosum and corticospinal tracts occur in the first 2–17 days after severe diffuse brain damage. The most sensitive indicator of pathway damage is fractional anisotropy. Diffuse axonal injury leads to axonal degeneration, causing a more significant decrease in anisotropy from 2–3 weeks after injury. Thus, primary brain damage, and in particular diffuse axonal damage, is a trigger for degenerative changes in axons and myelin sheaths of the white matter of the brain, leading to their destruction and atrophy 2–3 months after the injury.

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

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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.

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