FORENSIC ASPECTS OF ANALYTICAL COMPARISON OF TENDER INJURIES

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The occurrence of contusion foci in the area of the counterblow is explained by the theory of cavitation (Lebedev V.V., 1998). The second mechanism of development of brain contusion by the counterblow type is associated with the effect of "sliding" of the brain in the cranial cavity when foci of damage occur in the subcortical structures. Secondary brain damage develops as a result of extracranial complications that cause a disruption in the transport of nutrients and oxygen to the brain, the inability of the brain to utilize oxygen. Secondary brain damage is associated with hypoxia, ischemia, vasospasm, cerebral edema, intracranial hypertension, infection, disruption of systemic hemodynamics, cerebrospinal fluid circulation and water-electrolyte balance and increased permeability of the blood-brain barrier. Dislocation syndrome also develops due to increased intracranial pressure with displacement and deformation of various parts of the brain, then their wedging and infringement. Due to the activation of microglia, the synthesis of anti-inflammatory cytokines and leukocyte reaction increases, which in turn causes a further increase in the BBB and damage to the capillary walls. This is a prologue to the development of post-traumatic angiopathy, with the formation of clinical manifestations of remote consequences of traumatic brain injury. Eicosanoids and PAF play a significant role in this mechanism. Their effects are realized through specific receptors that are associated with phospholipase C and phospholipid metabolism processes.A feedback loop is formed with the formation of a "vicious" pathological circle: changes in phospholipid metabolism, activation of the free radical process with pathological activation of lipid peroxidation with damage to cell membranes and structural and functional disorders of the neuronal-glial-vascular complex. Primary structural changes in the brain are the trigger mechanism for a chain reaction of secondary changes at the molecular, cellular, tissue, organ and systemic levels. In young people, the frontal lobes are affected in approximately 66% of cases, the temporal lobes in 49% of cases, and up to 90% of patients have epidural hemorrhages and increased intracranial pressure. In middle-aged people, these figures are 77% and 64%. Increased intracranial pressure with the presence of subdural hematomas is found in up to 82% of affected individuals. Quite often, traumatic brain injury is accompanied by damage to the cervical spine, and post-traumatic encephalopathy with reflex-hemodynamic disorders and neuro-ophthalmological disorders develops. Atherosclerotic changes in the cerebral vessels as a result of

injuries develop mainly in the vertebrobasilar system. As a result of damage to the brain substance, serotonin or other vasoactive compounds are released into the cerebrospinal fluid, causing spasm of the cerebral vessels. The severity of traumatic brain injury and its outcomes correlate with the level of serotonin in the blood. Vestibular disorders associated with circulatory changes in the brain stem occur in individuals who have suffered traumatic brain injury.In this case, autoregulation of cerebral blood flow is disrupted, its speed decreases and local metabolism is disrupted, leading to an increase in the reaction of cerebral vessels to carbon dioxide, euphyllin, nitroglycerin, vasomotor lability, with neurotrophic changes. A special role in the development of acute vascular disorders is played by complex reflex-vascular reactions caused by the physical impact of the traumatic factor on the autonomic nervous formations embedded in the walls of blood vessels. The logical conclusion of the gradual encephalopathy of functional vascular disorders is the transition of post-traumatic to discirculatory, due to the insufficiency and depletion of the mechanisms regulating vascular tone, in particular the sympathetic-adrenal system, which is confirmed by the daily dynamics of the content of catecholamines in the urine and its changes in the insulin-adrenal test.

Autoimmune processes also play a major role in the pathogenesis of traumatic brain injury. Based on clinical data, it is not always possible to see a clear line between CCI and mild brain contusion (MCC). Neuroimmunological studies are of differential diagnostic value in this matter, in which CCI is characterized by a decrease in the nonspecific resistance of the body, a decrease in the quantitative indicators of cellular immunity, and an increase in the formation of antibodies. Mild brain contusion is characterized by suppression of the nonspecific resistance of the body, an increase in humoral immunity, and a less pronounced suppression of cellular immunity than in CCI. Damage to the soft tissues of the head, accompanying CCI, is characterized by an increase in the intensity of phagocytosis, which is associated with the cleansing of the wound surface of cicatricial adhesive processes, autoneurosensitization processes. Endocrinologists have established that injuries sustained in childhood and adolescence can cause delays in psychophysical development. According to psychiatrists, in 63-75% of cases, after a traumatic brain injury, educational, work, and social maladaptation develops. This is due to the fact that these changes are concentrated mainly in the brain stem, as well as in the cortical-subcortical formations, hypothalamus, and pituitary gland, which explains the formation of vegetative, metabolic, and neurotropic disorders in the period following a traumatic brain injury. Vegetative dysfunctions lead to changes in the cardiovascular system.In case of disorders in suprasegmental structures of the autonomic nervous system (ANS), changes in the relationships between ergotropic and trophotropic mechanisms occur, which determine deviations in the indices of autonomic tone, reactivity and activity

support. The main role in the pathogenesis of these disorders belongs to the frontal and temporal lobes of the cerebral cortex. Control and coordination of the autonomic nervous system activity is carried out through the structures of the diencephalon and the striatum. The limbic-reticular complex is involved in the regulation of sleep, wakefulness, memory, attention, in the formation of motivations and internal drives. Autonomic tone reflects the background activity of structures that ensure the regulation of body functions during adaptive activity. Autonomic reactivity forms a response in the form of autonomic changes to external disturbing factors when the body is at rest. The state of segmental and suprasegmental apparatuses to external influences can be normal, distorted, excessive and insufficient. Vegetative support of activity or vegetative components accompany all types of activity (mental, physical, etc.), which is controlled and carried out by the suprasegmental system. When it disintegrates, general somatic, mental and vegetative disorders arise.Vegetative disorders are equated with psychovegetative disorders and arise more often as a result of secondary changes in neurohumoral and vegetative regulation or in the case of pathology of internal organs and systems, including the central nervous system, as a result of a “neurotic state associated with stress and somatotropic disorders.”

The morphological substrate of the consequences of MCCI is formed as a result of primary and secondary circulatory disorders, cerebrospinal fluid dynamics and autoimmune processes due to hyperplasia of the arachnoendothelium and perivascular connective tissue. This in turn leads to obliteration and compression of blood vessels, disruption of blood flow, cerebrospinal fluid with the formation of ischemic and cerebrospinal fluid cysts of the brain. The formation of the consequences of CCCI is based on a single pathological complex consisting of a set of processes leading to a disorder of the integrative function of the brain with the formation of a new unstable nervous organization. The remote period of traumatic brain injury has several types of course: regressive, stable, remittent, progressive. D. R. Shtulman (2004) conducted a correlation of the main types of changes in the level of compensation of cerebral functions in the consequences of TBI and various syndromes of the consequences of TBI: constantly decreasing - in 38.5% of patients (hydrocephalic, psychoorganic, convulsive); stable with further decrease - in 30.4% (hypertensive, cephalgic); relatively stable - in 27.7% (cephalgic, vegetative); gradually increasing - in 3.4% of patients (asthenic). Decompensation of the post-traumatic process is associated with a breakdown of the compensatory and adaptive capabilities of the nervous system against the background of the desire of the new functional state of the nervous system to a normal level, with the resulting neurological deficit.The duration of remission of patients depends on the degree of expression of morphological post-traumatic changes and compensatory-adaptive processes. The prognosis of the disease depends on the frequency and severity of periods of decompensation of the post-traumatic state. As

the analysis of literary data showed, post-traumatic changes represent a set of compensatory-adaptive and formed pathological processes that can independently develop and at the same time determine the condition of patients. At the same time, pathological processes developing after injury have a protracted, progressive course, are transformed into various neurological symptoms and syndromes, lead to disability and early mortality. Timely detection and treatment of these pathological processes, with an assessment of the adaptive-compensatory capabilities of the central nervous system remains an urgent problem for clinical neurology and requires further study using modern research methods. Subarachnoid hemorrhages can be of both traumatic and non-traumatic origin. The first most often occur as a result of mechanical impact on the head. The causes of non-traumatic hemorrhages are quite varied: bleeding from ruptured arterial or arteriovenous aneurysms, in blood diseases. Subarachnoid hemorrhages are described in cases of general physical overload, alcohol intoxication, vitamin deficiency, etc.Despite the variety of causes, it should be considered unsuccessful to replace the term “non-traumatic” with “spontaneous”, “essential”, “idiopathic”, since the latter do not explain the essence of the hemorrhage and usually indicate only that its cause remains unknown.

The origin of the hemorrhage, as a rule, does not raise doubts for the clinician and morphologist. Along with this, forensic experts encounter serious difficulties, especially in cases where the integrity of the skull bones is intact, the consequences of the injury are limited to damage to the soft tissues of the face, and subarachnoid hemorrhages are localized mainly on the basal surface of the brain (the so-called basal subarachnoid hemorrhages). Some forensic doctors believe that the detected vascular pathology allows us to completely exclude the influence of trauma, while others consider it possible to judge the traumatic nature of such hemorrhages based only on the fact of traumatic impact on the head, which is often established only by the case materials. Naturally, the expert's opinion in such cases plays a decisive role in qualifying the actions of the suspect, accused or defendant. It is clear that two polar points of view (either trauma or pathology) cannot be correct at the same time. Who is right? Some experts see confirmation of their opinion in the court's verdict, while the court is confident in the correctness of its decision, based on the expert's conclusion on the cause of death. The correctness of the forensic medical conclusion can be verified by the results of scientific research, but even here there are diametrically opposed judgments. Various aspects of expert assessment of hemorrhages are presented in the works of many authors. These works are descriptions of individual cases from practice, or generalizations of expert opinions performed by different experts and with varying degrees of detail.

Analysis of clinical, pathomorphological and forensic literature and our own 25 years of experience in studying the problem of subarachnoid hemorrhages allows us to

propose criteria for forensic medical assessment of trauma and pathology in the genesis of hemorrhages.

If hemorrhages are an integral part of severe craniocerebral trauma and a consequence of some pronounced pathological conditions, then there is no need to discuss their etiology (origin, cause). It is obvious. On the contrary, basal subarachnoid hemorrhages that occur during domestic conflicts are assessed in the most contradictory way. In such cases, their traumatic or non-traumatic nature must be proven during a forensic medical examination. An attempt to evaluate differential diagnostic criteria for the origin of hemorrhages can be based primarily on a comparison of the morphology of obviously traumatic and obviously non-traumatic hemorrhages under the arachnoid membrane of the brain.

Traumatic hemorrhages are represented by two main morphological types: spotted and limited-diffuse. Both types can be observed both with preservation and with violation of the integrity of the soft meninges.

In craniocerebral trauma, hemorrhage into the subarachnoid space is localized mainly in the impact and counter-impact zones, which determines the asymmetry of their topography on the surface of the brain.Symmetrical arrangement of traumatic subarachnoid hemorrhages is rare. They are observed only with anteroposterior and posteroanterior central blows, as well as with special mechanisms of craniocerebral trauma - direct traumatic impact on the central sections of the skull base: for example, with a blank shot to the mouth or neck in the direction of the skull base.

Isolated traumatic subarachnoid hemorrhages in the basal cistern are also rare and occur with direct trauma to the central sections of the skull base.

Traumatic hemorrhages are typically combined with skull fractures and contusions of the cerebral cortex, as well as erosive (ulcer-like) ruptures of the soft meninges.

Non-traumatic hemorrhages may be diffuse, focal-diffuse or petechial (point) in nature, their localization is determined by the location of the bleeding source [15]. If the damaged vessel is located near the basal cistern, it is filled with blood clots, and diffuse subarachnoid hemorrhages are symmetrically located around it, the intensity of which gradually decreases towards the periphery of the base of the brain. In some cases, blood penetrates into the subarachnoid space of the convex surface of the brain. An asymmetric location of non-traumatic subarachnoid hemorrhages is also possible: for example, if the bleeding source is no closer than 2.0-2.5 cm from the basal cistern.

Non-traumatic subarachnoid hemorrhages very often fill the basal cistern. They are usually observed with ruptures of aneurysms of the vessels of the circle of Willis - an arterial ring located in the center of the base of the brain. There may be no blood in the basal cistern if, upon rupture of a pathologically altered vessel, blood breaks through either into the tissue or into the ventricles of the brain.

In the absence of a sufficient number of typical morphological signs that allow reliable differentiation of traumatic and non-traumatic subarachnoid hemorrhages, the judgment about their origin can be based on an assessment of the conditions and characteristics of the traumatic impact on the head.

Some authors point to specific features of the mechanism of head trauma, which they regard as particularly dangerous for the occurrence of subarachnoid hemorrhages, in particular basal localization.The following are considered to be particularly dangerous variants of the mechanism of head injury: 1) blows to the chin; 2) indirect trauma to the spine; 3) multiple subthreshold blows (impacts of low force, each of which individually cannot cause the formation of hemorrhages); 4) blows to reflexogenic zones.

Particular attention should be paid to the fact that there is no information in the literature on basal subarachnoid hemorrhages in traffic accidents, which are abundant in whiplash injuries.

The data presented indicate a low probability of damage to the vertebral arteries, but there is no reason to completely exclude the possibility of rupture of these vessels with direct or indirect trauma to the cervical spine.

H. Groh, giving a detailed list of whiplash injuries, includes ruptures of the vertebral arteries and damage to the cervical spinal cord. Some authors admit the possibility of trauma to the brain stem in whiplash injuries. A number of authors claim that compression of the vertebral arteries can occur with mutual displacements of the atlas (1st cervical vertebra) and the occipital bone. The judgment on the possibility of damage to the vertebral arteries with displacement in the occipitovertebral joint is permissible only if there is morphological evidence confirming the fact of such overstretching that actually took place.

Anatomical evidence of displacement is primarily a violation of the integrity of the joint capsules and ligamentous apparatus of the atlanto-occipital and atlanto-axial articulations. J.-Y. de la Caffiniere et al. who performed an experimental X-ray anatomical study, cite the following morphological criteria: 1) displacement of the atlas is impossible without damage to the transverse ligament; 2) stabilization of the I and II cervical vertebrae is ensured by the capsule of the atlanto-axial articulation; 3) in case of fractures of the odontoid process, displacement does not occur until the capsule and ligamentous apparatus are damaged.

R. Roy-Camille et al. separately studying the possibility of displacement in the block of the I-II cervical vertebrae, established that displacement between them is possible only with damage to the ligamentous apparatus of the spine at the same level. It should be borne in mind that with overstretching of the cervical spine, damage to the vertebral arteries is by no means necessary, since there is compensation due to the anatomical and topographic position of these vessels in the form of a half-loop before

entering the cranial cavity. The compensatory value of the half-loop and the elastic properties of the vessels is so great that with complete ruptures of the atlanto-occipital joint and the brain stem, the integrity of the vertebral arteries can be preserved.

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