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FORENSIC ASPECTS OF CIRCULATORY DISORDERS IN BRAIN INJURIES

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Abstract: General Background: The analysis of forensic medical reports reveals persistent challenges in accurately identifying and assessing craniocerebral injuries. **Specific Background:** Repeated and additional forensic examinations often highlight shortcomings and errors attributed to a lack of understanding among experts regarding contemporary concepts related to traumatic brain injury (TBI), including its mechanisms, clinical manifestations, and differential diagnosis. **Knowledge Gap:** Despite advancements in medical knowledge, there remains a significant gap in the expertise of forensic professionals regarding the complexities of TBI, leading to inconsistencies in forensic assessments. **Aims:** This study aims to evaluate the current practices in forensic medical examinations of craniocerebral injuries and identify areas for improvement in the methodology of assessment. **Results:** The findings indicate that insufficient knowledge of TBI mechanisms and a lack of standardized approaches contribute to errors in forensic evaluations, ultimately affecting the quality of forensic medical reports. **Novelty:** This research emphasizes the need for enhanced training and a unified methodological framework for forensic experts to better understand and assess craniocerebral injuries. **Implications:** By addressing these deficiencies, the study underscores the importance of improving forensic practices, which could lead to more accurate diagnoses and assessments of TBI, thereby enhancing the reliability of forensic medical reports in legal contexts. This work advocates for the development of standardized guidelines and continuous education for forensic experts in the field.

Keywords: **:** Forensic Examination, Cerebral Blood Flow In Patients With Traumatic Brain Injury.

Introduction

Traumatic brain injury (TBI) is the most common type of mechanical damage and has a tendency to steadily increase, primarily among the most active population group — people aged 20–49 years [1]. The most common type is closed traumatic brain injury (CTBI), accounting for 60–80% of all cases of brain injury. Diagnostic errors in providing medical care are the cause of incorrect forensic classification of harm caused to human health. A major problem is the differential diagnosis of concussion and mild contusion of the brain, which explains their combination into one pathology — mild traumatic brain injury (MTBI) [1]. The greatest difficulty for a clinician and forensic expert is assessing the severity of TBI occurring against the background of an existing disease (atherosclerosis, hypertension, ischemic heart disease, etc.). These circumstances explain the need to improve diagnostics and forensic assessment of the severity of TBI.

Crystallographic studies of cerebrospinal fluid were conducted in forensic diagnostics of the severity of traumatic brain injury. Comparison of the obtained data with the results of laboratory research, as well as clinical and instrumental examination of victims, showed the high diagnostic value of the physicochemical properties of cerebrospinal fluid in brain damage. The object of attention was the area of growth of crystal colonies, the shape and size of the formed colonies, the severity of repeated crystallization, the density of the crystals in the colonies, the number of crystallization centers in healthy people and in people with traumatic brain injury and other pathological processes. In healthy individuals, the growth of crystal colonies was not observed or occurred in the form of

small, isolated colony embryos occupying no more than 10-15% of the surface of the bottom of the Petri dish. The presence of a pathological process (excluding TBI) significantly changed the properties of the cerebrospinal fluid.

In the presence of chronic hematoma, spinal cord injury, acute cerebrovascular accidents, the growth of crystals of various shapes and sizes was noted. The number of crystallization centers was different and depended on the type of pathological process and the degree of its severity.

In individuals with alcohol intoxication, the presence of pronounced secondary crystallization in cerebrospinal fluid crystallograms with the formation of a specific ring-shaped sectoral focus was established, which was determined in all cases of alcohol intoxication.

In cases of TBI of varying degrees, the crystallogram picture reliably reflected the severity of the injury and the characteristics of the post-traumatic period. Aggravation of the brain injury was accompanied by an increase in the growth area and a change in the growth form of crystal colonies, an increase in the number of crystallization centers and the density of the crystal colonies, as well as the height of the crystal colonies and their symmetrical growth. These changes were accompanied by an increase in the degree of expression of repeated crystallization, the appearance of different-sized filiform crystals.

In the crystallograms of cerebrospinal fluid in mild cerebral contusion, the growth of colonies of crystals occurred on an area of 25-50% of the surface of the bottom of the Petri dish. The bushy or sector-bushy form of colonies predominated. The number of crystallization centers reached 25-50. The density of the colonies with a moderate number of breakages increased.

Moderate brain contusion is characterized by the growth of colonies of crystals on an area of no more than 50-70% of the surface of the bottom of the Petri dish, a bushy, sectorial-bushy or sectorial form of growth of colonies of crystals, the number of crystallization centers within 25-50, high density of arrangement with multiple “breakages”, uneven distribution of crystals by thickness.

In severe brain contusion, colony growth of crystals was observed on an area of 75-100% of the Petri dish bottom surface, a sectoral or sectoral-bushy form of colony growth, and in some cases, symmetrical growth. The maximum number of crystallization centers, dense arrangement of crystals with multiple "breakages" were noted.

In severe cases of brain contusion with fractures of the vault and base of the skull, damage to the brain stem, hemorrhages under the membranes and into the substance of the brain, the appearance of symmetrical growth of colonies indicated the presence of a life-threatening condition and an unfavorable outcome of the injury.

Methods

This study employed a comprehensive observational and analytical approach to assess the interplay between traumatic brain injury (TBI) and pre-existing cardiovascular conditions, particularly in elderly patients. A cohort of individuals with diagnosed TBI was selected from clinical records, with an emphasis on those presenting with comorbid cardiovascular diseases such as hypertension and ischemic heart disease. Data collection involved detailed clinical evaluations, including neurological assessments, imaging studies, and the documentation of symptoms experienced during the acute and rehabilitation phases. Additionally, the research utilized morphological analysis of chronic subdural hematomas (CSH) through histopathological examinations to establish new diagnostic criteria. Statistical analyses were performed to identify correlations between the severity of TBI, the presence of cardiovascular conditions, and the clinical outcomes observed. The study aimed to develop a formalized chart for the comprehensive assessment

of symptoms, facilitating objective diagnosis and improving forensic evaluations. Ethical considerations were adhered to, ensuring patient confidentiality and the integrity of data throughout the research process.

Results and Discussion

Crystallographic studies of cerebrospinal fluid in cases of brain injury and other non-traumatic pathological processes have shown that cerebrospinal fluid crystallograms in the presence of TBI differ from cerebrospinal fluid crystallograms in other pathological processes.

Under the supervision of Corresponding Member of the Russian Academy of Medical Sciences, Professor Yu. I. Pigolkin, another little-studied problem in assessing the severity of TBI was studied: the influence of a disease preceding the injury on the course of TBI, the duration of rehabilitation, the establishment of a cause-and-effect relationship between them, and the occurrence of immediate and remote complications.

Any TBI causes changes in the entire body. Later, in the recovery phase, not all changes in the central nervous system may disappear, but only be temporarily compensated, which is mistakenly perceived by clinicians as recovery. When compensation is exhausted, the victim begins to experience increasing pathological phenomena that he did not have before, including in the acute post-traumatic period. For example, mental disorders, vegetative-vascular and vestibulo-vegetative reactions, changes in the immune and hormonal systems are mistakenly interpreted by forensic doctors as pathology not associated with trauma.

Patients who have suffered the same degree of TBI severity have different clinical pictures, they respond differently to conventional treatment, and the consequences of the injury manifest themselves differently in the future. This depends not only on the degree of brain damage, but also on the general premorbid state of the body. The prevalence of cardiovascular diseases (hypertension and ischemic heart disease) determines the frequent combination of these types of pathology with TBI. Therefore, it is practically important to establish the influence of cardiovascular diseases on the course of TBI in the acute and late periods, as well as the effect of brain damage on the dynamics of existing cardiovascular pathology. In addition, some diseases can develop as a result of trauma. Diagnosis of LMCT is especially difficult in older age groups with the above-mentioned diseases. Memory loss, dizziness, fatigue, headache, and neurological symptoms characteristic of cerebral vascular atherosclerosis coincide with the manifestations of MTBI.

Diseases present in elderly and senile individuals aggravate the course of the traumatic process. Even LMCT in the presence of arterial hypertension is characterized by a more severe clinical course, more pronounced neurological symptoms, and an increased frequency of secondary acute cerebrovascular accidents. Among young patients with TBI, the prevalence of hypertension is much lower, and arterial damage is characterized mainly by hyperplasia of the muscular membrane due to the proliferation of smooth muscle cells and initial signs of sclerosis.

The body's response to TBI in the elderly differs from the response of young and middle-aged victims by the prevalence of hyperergic reactions and predominantly hemodynamic disorders. At the same time, in such a multifaceted clinical picture developing with a head injury in combination with an exacerbation of hypertension, certain symptom complexes characteristic of hypertension can be distinguished: diffuse damage to the cerebral arteries in the form of elongation and tortuosity of the arteries, thickening and compaction of their walls, changes in the lumen of varying severity. Microscopic examination of the cerebral vessels reveals hypertrophy of the muscular membrane caused by the proliferation of smooth muscle cells and thickening of the basal membrane, signs of

plasmorrhagia - swelling and homogenization of the endothelium and internal basal membrane, infiltration with proteins, lipids and erythrocytes. Against the background of hypertension and ischemic heart disease, the following features of the course of TBI have been identified: more persistent and prolonged neurological symptoms, both general cerebral and focal, resistant to treatment, mnemonic and psychoemotional disorders, post-traumatic vegetative dysfunctions, severe hypertensive crises, cardialgia, and rest angina. In addition, TBI itself becomes a risk factor for hypertension and cerebral atherosclerosis, accelerating their development and aggravating the course.

Thus, the combination of TBI and cardiovascular diseases creates a qualitatively new pathological situation caused by mutual aggravating influence. At the same time, diagnosis requires a critical analysis of the entire complex of symptoms and differentiation of somatic pathology and trauma. A correct scientifically substantiated assessment of TBI, its impact on the exacerbation and course of the existing disease is of fundamental importance for objective and scientifically substantiated expert conclusions.

In order to eliminate expert errors and improve the validity of expert conclusions, a formalized chart for a comprehensive assessment of symptoms and objectification of a clinical diagnosis is proposed. The algorithm of expert actions in relation to victims with TBI includes a consultation with a neurologist with filling out a formalized chart, which allows tracking the dynamics of a complex of symptoms of the nervous system and provides significant assistance to forensic experts in determining the severity of harm to health. To prove the presence of LMCT, according to the formalized chart, at least 4-5 highly informative symptoms from the most significant diagnostic signs are required. These include: nystagmus, Gurevich-Mann symptom, tendon anisoreflexia, decreased abdominal reflexes, muscle hypotonia, static ataxia, tongue deviation, abducens nerve paresis, pallor of the skin and mucous membranes, hyperemia of the skin and mucous membranes.

In forensic practice, chronic subdural hematomas (CSH) are often encountered during autopsy studies; they are a volumetric formation limited by a capsule and located under the dura mater.

Most often, the occurrence of CSH is caused by TBI. Among other causes, the most common are cerebrovascular diseases, such as hypertension and atherosclerosis. CSH can occur as a complication of infectious diseases, blood diseases, and heart defects. The causes of subdural blood accumulation can be bleeding from vascular malformations and brain tumors. The occurrence of CSH can be facilitated by various forms of brain atrophy.

Under the supervision of Professor Yu. I. Pigolkin, new morphological criteria for forensic medical diagnostics of the causes, timing of occurrence and characteristics of chronic pulmonary hypertension have been developed.

A characteristic feature of CSH is its layered structure. Another feature of CSH is the formation of "giant" macrocapillaries. The hematoma consists of thin films, gradually and consistently superimposed on each other. A capsule is formed around the hemorrhage, consisting of external and internal walls. In the capsules of CSH, not only the cellular component is detected, but also collagen fibers. In the structure of CSH, 2 types of capsules are distinguished. The 1st type includes the presence of a distinct border between the inner surface of the capsule and the contents of CSH. The capsule is represented by granulation tissue with focal infiltration and a small group of mast cells. In the 2nd type, the CSH capsule is fused with blood clots and does not have clear boundaries with the hematoma. The outer sections are represented by connective tissue of varying degrees of maturity. Non-traumatic CSH is characterized by a distinct boundary between the inner surface of the capsule and the contents of the CSH, represented by elongated dendritic cells. Traumatic CSH is not characterized by the presence of histologically clear boundaries between the capsule and the

hematoma.

Morphological studies allow us to establish changes in the CSG depending on the time that has passed since the hemorrhage.

After 1-3 months after the subdural hemorrhage, the granulation tissue matures with a predominance of epithelioid cells and fibroblasts. The outer sections of the capsule consist of young fibrous tissue. Vascular gaps and giant capillaries are detected, as well as traces of secondary intracapsular hemorrhages of varying ages.

After 3-12 months, gradual maturation of connective tissue occurs with a decrease in cellular elements and the predominance of fibrous structures. Along with blood vessels, vascular gaps and giant macrocapillaries are found. After 1-3 years, only lymphoplasmacytic infiltrates are formed, with mature and young connective tissue. After 3 years, single lymphocytes are detected, and the capsule itself is fibrous avascular tissue.

The conducted study showed that CSH is a polyetiological space-occupying formation located under the dura mater, which, unlike acute and subacute hematomas, has a restrictive capsule. The main factors underlying the pathogenesis of CSH are mechanical trauma or pathological vascular permeability, causing changes in homeostasis and local microcirculation disorders, contributing to the formation of a capsule. Morphological signs characteristic of various variants of the course of CSH allow forensic experts to make reasonable conclusions about the nature and some features of the morphology of CSH. Analysis of a complex of signs allows us to resolve the issue of the time of occurrence of CSH within four time gradations, as well as presumably resolve issues about the genesis of CSH.

Thus, as a result of the conducted research, additional forensic criteria for diagnosing the severity of TBI have been developed, allowing for the objectification of signs of TBI in cases of concomitant diseases, including in elderly and senile individuals, and improving the diagnosis of CSH, especially with regard to its nature and time of occurrence.

Conclusion

In conclusion, this research highlights the intricate relationship between traumatic brain injury (TBI) and pre-existing cardiovascular conditions, particularly in elderly patients, demonstrating that these comorbidities significantly complicate the clinical presentation and management of TBI. The study reveals that the presence of cardiovascular diseases exacerbates neurological symptoms and leads to a higher incidence of complications, thus necessitating a comprehensive assessment that differentiates between somatic pathology and trauma. Additionally, the development of new morphological criteria for chronic subdural hematomas (CSH) enhances forensic diagnostics, allowing for more precise identification of the nature and timing of these hematomas. These findings have critical implications for clinical practice, emphasizing the need for multidisciplinary approaches in diagnosing and treating TBI, especially among vulnerable populations. Future research should focus on refining diagnostic protocols and exploring the long-term outcomes of TBI in patients with cardiovascular comorbidities, further contributing to the understanding of the pathological interplay between these conditions.

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