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FORENSIC MEDICAL CRITERIA FOR ASSESSING CEREBROSPINAL FLUID CIRCULATION DISORDERS IN FATAL TRAUMATIC BRAIN INJURIES

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Abstract:
In almost all cases of traumatic brain injury (TBI), there is displacement and deformation of the brain, followed by its return to its original position. This is inevitably accompanied by dynamic redistribution of cerebrospinal fluid (CSF) in the subarachnoid space and ventricular system of the brain. This highlights the relevance of studying the thanatogenesis of brain injuries in TBI, particularly the CSF circulation system.
A retrospective analysis of traumatic brain injuries was conducted in this study. For the first time, morphological and morphometric indicators of CSF circulation were examined, and the main stages of thanatogenesis in fatal TBIs were determined. A systematic analysis of CSF circulation disorders in traumatic brain injury was performed, and expert criteria for assessing its severity and timing were developed.

Keywords: TBI, brain, cerebrospinal fluid circulation, thanatogenesis, morphology, morphometry, expert criteria.

Introduction

Subarachnoid hemorrhage (SAH) is one of the most severe consequences of traumatic brain injury (TBI) and rupture of cerebral aneurysms. It is characterized by the penetration of blood into the subarachnoid space, leading to a cascade of pathological reactions, including CSF circulation disorders, inflammatory responses, and ischemic processes. Understanding the pathogenesis mechanisms of these disorders is crucial for diagnosis, treatment, and prognosis. This article provides a detailed review of the primary mechanisms and consequences of CSF dynamics disturbances in SAH.

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Research Objective:

To conduct a systematic analysis of CSF circulation disorders in traumatic brain injury and develop expert criteria for assessing its severity and timing.

Materials and Methods: The material for this study consisted of 84 forensic medical examination reports on fatal traumatic brain injuries. Among the victims, 72 (85.7%) were men and 12 (14.3%) were women.

The average age of individuals who suffered fatal TBIs was 36.6±8.4 years. The circumstances of the injuries were as follows: 78.6% of cases resulted from road traffic accidents, 12.4% from falls from heights, and 9% from head trauma caused by blunt objects in domestic incidents.

In all cases of TBI, 67.8% of victims sustained their injuries while under the influence of alcohol, with intoxication levels ranging from 0.78 to 6.2‰.

Of the cases studied, 49 involved isolated TBI, while 35 cases involved TBI combined with other injuries.

The death of patients with TBI occurred within the first 24 hours (53 cases) and at later stages (31 cases).

The autopsy examination of the bodies of the injured was carried out in accordance with the procedure and sequence defined by the "Rules for Forensic Medical Examination of a Corpse" (2010).

When studying pathomorphology, we focused on the analysis of the nature of primary and secondary injuries, brain dislocation and entrapment, and postoperative complications. Secondary brain injuries were caused by disturbances in cerebral circulation, cerebrospinal fluid dynamics, brain edema-swelling, and intracranial inflammatory complications. The immediate cause of death, the presence of extracranial complications, and comorbid diseases were clarified.

Research Results and Discussion

Considering the choroid plexuses of the ventricles, the meninges, and the intermeningeal spaces as a single three-component cerebrospinal fluid (CSF) circulation system allows for a deeper understanding of the mechanisms underlying various pathologies that develop after traumatic brain injury (TBI). These conditions include leptomeningitis, choroidependymitis, pachymeningitis, hydrocephalus, arachnoid cysts, as well as subarachnoid and intraventricular hemorrhages. Despite differences in morphological presentation, they share a

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common localization and involve all three components of CSF circulation in the pathological process, albeit to varying degrees.

It is important to note that damage to CSF circulation structures always precedes secondary changes in cerebral structures (except for direct injury in TBI). In each specific case, causal relationships are determined by the characteristics of the disease, its localization, severity, and other factors.

Disruption in one component of the cerebrospinal fluid (CSF) circulation system inevitably leads to dysfunction in the other components; however, each disease exhibits its own specific characteristics. For example, in leptomeningitis, arachnoid cysts, and subarachnoid hemorrhages (SAH), the most significant factor is impaired CSF circulation in the subarachnoid space. Intraventricular hemorrhages, which occur as a complication of SAH, primarily affect CSF circulation within the brain's ventricles. In the case of hydrocephalus, the main pathogenic factor is impaired CSF outflow, leading to its excessive accumulation and increased intracranial pressure.

Disorders of the CSF circulation system are interconnected. Acute leptomeningitis can become chronic, causing fibrosis and resulting in occlusive or resorptive hydrocephalus. SAH often leads to aseptic leptomeningitis, while purulent meningitis can cause pachymeningitis, leading to secondary brain damage.

A comprehensive study of the CSF circulation structure in pathology allows for a deeper understanding of the mechanisms of these processes and their clinico-morphological manifestations in TBI. SAH is an independent form of acute cerebrovascular accident. The study of morphological changes in paracerebral barriers after aneurysm rupture and the introduction of autologous blood into the CSF has revealed three stages:

1. The spread of blood through the CSF system
2. The formation of clots
3. Their lysis

Arterial spasm in SAH occurs in two phases: a short-term phase and a prolonged phase. In favorable cases, the blood vessels restore their lumen, which can be considered the third stage of spasm. All pathological changes in SAH are interconnected and develop sequentially.

The first stage of SAH: hemorrhage and the spread of blood through the CSF system. Blood that enters the subarachnoid space spreads with the CSF flow

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through the CSF pathways, reaching their drainage openings at the tops of the gyri and penetrating the subarachnoid trabecular spaces. A significant accumulation of blood increases the volume of CSF, leading to acute CSF hypertension.

In the early stages, before clot formation, increased CSF pressure promotes enhanced CSF elimination through the pulmonary-hematological barrier, partially clearing the CSF of erythrocytes and other blood components. Erythrocytes penetrate the arachnoid membrane, intercellular spaces, the subdural space, and even the thickness of the dura mater, where they accumulate around the capillaries of its inner network.

The spread of blood through the CSF pathways, where arteries pass, leads to mechanical irritation of the vascular nerve elements and paravascular nerve plexuses, causing a short-term arterial spasm. The coagulation of blood in the CSF with the formation of clots.

The second stage of SAH: blood clot formation and disruption of CSF circulation. The formation of blood clots in the subarachnoid space partially or completely blocks the CSF pathways, leading to impaired CSF microcirculation. Depending on the location of the blockage, this may affect individual channels, one hemisphere, or both. As a result, progressive CSF hypertension develops.

Additionally, impaired CSF microcirculation in the drainage pathways hinders its outflow through the pulmonary-hematological barrier, slowing the removal of blood components. This factor, along with other mechanisms, contributes to the development of prolonged arterial spasm.

Blood clots most commonly form around paravascular structures and nerve plexuses of major arteries. Their density depends on the ratio of blood to CSF. During the coagulation process, some platelets break down, releasing serotonin—a substance with a strong vasoconstrictive effect. Even a minimal amount of serotonin in the CSF (0.05 ml) triggers an immediate spasm of arterial branches.

The fixation of blood clots near paravascular nerve trunks intensifies the irritation of nerve elements, sustaining prolonged arterial spasm. In the subarachnoid trabecular spaces, where CSF circulates more slowly, blood cells also accumulate. By days 2–3, phagocytosis becomes active: arachnoid endothelial cells and macrophages absorb clot components, facilitating CSF clearance and its normalization.

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Lysis of blood clots (the third stage of SAH). At the final stage of subarachnoid hemorrhage (SAH), the previously formed blood clots gradually undergo lysis. This process is driven by the activity of arachnoid endothelial cells lining the subarachnoid space (SAS) and is accompanied by the release of fibrin degradation products and blood elements (erythrocytes, leukocytes, and platelets) into the CSF. Fibrinolysis products can have a strong vasoconstrictive effect, contributing to the development and maintenance of prolonged arterial spasm. Studies have shown that, at the submicroscopic level, the integrity of the barriers between CSF and adjacent tissues is compromised, allowing degradation components to penetrate arterial walls, paravascular nerve trunks, and brain structures. As a result, axons and vascular smooth muscle cells are affected, leading to brain edema.

The high reactivity of arachnoid endothelial cells plays a key role in these changes. Upon contact with blood, they not only absorb its components but can also acquire macrophage-like properties. In the paravascular nerve trunks of the brain, destruction of basal membranes, loosening of collagen fibrils, and disruption of nerve fiber structure are observed, which are also associated with prolonged vascular spasm.

Similar pathological processes also occur in the major arteries of the brain. As a result of endothelial cell changes, their shape becomes rounded, intercellular spaces expand, and the internal elastic membrane undergoes folding. These phenomena are accompanied by focal rarefaction of myofilaments in smooth muscle cells, leading to pronounced and persistent vascular spasm.

Additionally, the disruption of barrier functions allows degradation products from blood clots to penetrate brain structures, exacerbating edema. It is possible that the release of serotonin stored in brain tissues plays an additional role in maintaining the spasm, as confirmed by studies conducted by several authors.

The gradual lysis of blood clots facilitates the restoration of CSF circulation, leading to the normalization of CSF pressure and the removal of degradation products beyond the SAS. As a result, the phagocytic activity of arachnoid endothelial cells decreases, indicating the recovery of the brain's barrier structures and CSF circulation.

One of the severe complications of SAH is the penetration of blood into the brain's ventricles (intraventricular hemorrhage, IVH), which significantly worsens the

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disease prognosis. Clinical data indicate that the incidence of IVH ranges from 13% to 28%, while autopsy findings report a frequency of 37% to 54%.

This leads to structural disruption of the choroid plexuses, ventricular ependyma, and arachnoid membrane, resulting in impaired CSF circulation. Postmortem analysis of patients who died after aneurysm rupture revealed changes in the cells of the choroidal epithelium and the stroma of the choroid plexuses, indicating a disruption of the blood-CSF barrier. In some cases, edema, destruction of the epithelial layer, and penetration of erythrocytes into the brain's ventricles were observed.

Damage to the ependyma lining the ventricles may be minimal, but in some cases, its destruction, rupture formation, and accumulation of blood degradation products are detected. These changes impair the function of the CSF barrier, contributing to the development of brain edema.

Conclusions

Subarachnoid hemorrhage (SAH) is one of the most severe consequences of traumatic brain injury and the rupture of cerebral aneurysms. The entry of blood into the subarachnoid space triggers a cascade of pathological reactions, including impaired CSF circulation, inflammatory processes, and ischemic changes.

Understanding the mechanisms of these changes plays a key role in diagnosis, treatment, and prognosis. This article examines the main aspects of CSF dynamics disturbances in SAH, including the mechanisms of blood clot formation and lysis, arterial spasm, brain edema, and the consequences of intracranial hemorrhage.

References

1. Akopov V.I. Expert Examination of Health Damage (Legal Issues of Forensic Medical Practice). — Moscow: Expert Bureau, 1998. — 256 p.
2. Akimov G.A., Mamrey R.K., Golovkin V.I. Autoimmune Processes in the Pathogenesis of Traumatic Brain Disease // Journal of Neurology and Psychiatry. 1978. — Vol. 78, No. 3. — P. 370-375.
3. Akimov G.A. Some Aspects of the Problem of Closed Head Injury // Military Medical Journal. 1988. — No. 11. — P. 32-34.

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4. Aleksanov N.S., Shchigolev Yu.S., Gizatullin Sh.Kh. Short-Latency Auditory Brainstem Evoked Potentials in Patients with Concussion // Issues of Neurosurgery. 1995. — No. 2. — P. 17-20.
5. Artaryan A.A., Gaevy O.V., Korolev A.G. On the Periodization of Traumatic Brain Injury in Children // Issues of Neurosurgery. 1990. — No. 6. — P. 16-18.
6. Artaryan A.A., Brodsky Yu.S., Likhterman L.B., et al. Clinical Classification of Traumatic Brain Injury in Children // Classification of Traumatic Brain Injury. Moscow, 1992. — P. 50-67.
7. Asadullaev M.M., Troshin V.M., Chirkov V.D. Fundamentals of Electroencephalography. — Tashkent: Ibn Sina Publishing and Printing, 1993. — 216 p.
8. Babichenko E.I. Recurrent Closed Head Injury // Journal of Neurology and Psychiatry. 1993. — Vol. 93, No. 2. — P. 43-47.
9. Belykh A.N. On the Relationship Between the Occurrence of Concussion and the Localization of Head Impacts // Forensic Medical Examination / Proceedings of the Interdepartmental Interregional Scientific and Practical Conference. St. Petersburg, 1997. — P. 130-132.
10. Bekhtereva N.P. The Healthy and Diseased Human Brain. — Leningrad, 1988. — 262 p.
11. Blagoveshchenskaya N.S. Otoneurological Symptoms and Syndromes. — Moscow: Medicine, 1990. — 432 p.