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MORPHO-HISTOLOGICAL CHARACTERISTICS OF CHANGES AFTER BRAIN INJURIES (LITERATURE REVIEW)

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Abstract
The literature review showed that, brain injuries make up 30-40% of all injuries and cause disability in 25-30% of cases. More than 90% of those who died were found to be caused by ischemic damage of the brain during histological examination. The main areas of change after brain injuries are the basilar artery (a. basilaris) and the middle cerebral artery (a. cerebri media), where excessive calcium ion concentration in the smooth muscle fibers causes spastic contraction of the vessels, resulting in the death of myocyte cells, rupture of blood vessels, and bleeding into surrounding tissues, with the development of pathological changes such as necrosis and dystrophy from primary pathological changes.

Keywords Myocyte cells, rupture of blood vessels, and bleeding.

INTRODUCTION

Brain injuries make up 30-40% of all injuries and cause disability in 25-30% of cases. [15]. More than 90% of those who died were found to be caused by ischemic damage of the brain during histological examination(19). The cause of death remains unexplained in one-third of 70-72% of brain injury deaths after traumatic brain injury [20]. Clinical observations and computer tomographic examinations revealed that 3-25% of patients develop acute cerebral blood circulation disorders, that is, secondary cerebral stroke [20,25,26].

In the Russian Federation, the number of deaths due to injuries ofbrain in second place, accounting for 15.8%, and this indicator is a ratio of 6:9 among the working-age population [15].

Traumatic brain injuries are one of the leading causes of death and the leading cause of disability in people under the age of 35. In economically developed countries, the increase in motor vehicles and the automation of work processes are leading to an increase in the incidence of traumatic brain injuries. For example, in the United States, there are 180 to 250 cases per 100,000 people, and in Europe, up to 235 cases, of which 1.6 million are hospitalized, of whom 66,000 die. According to the World Health Organization, traumatic brain injuries are predominantly chronic diseases, the consequences of which are irreversible, require long-term rehabilitation, and ultimately result in disability. [18]

Complete general criteria for the correct assessment of the severity and consequences of brain damage in patients after head injuries have not been developed [15].

The most common cause of dislocation syndromes in head injuries is injuries accompanied by intracranial hemorrhage. The cause of these pathological processes in head injuries is a condition such as damage to the brain stem. As we know, we study primary and secondary forms of brain stem injuries separately. Primary changes include contusions, compression and rupture of the stem, diffuse axonal injuries, and hemorrhage into the stem. Secondary changes include damage to the structure of the stem, compression of the brain with a hematoma, swelling and congestion of the brain, pathological changes in blood circulation and cerebrospinal fluid circulation. The occurrence and development of the above changes result in clinical manifestations associated with dislocation syndrome [4]. After head injuries, intracerebral hypertension, systemic hypotension, cerebral edema, brain compression with focal hematomas, and the development of pathologies in small blood vessels (vasospasm) cause ischemic processes in the brain.

In traumatic brain injuries, the primary pathological changes in the brain are necrosis and dystrophy. Histological examination reveals rupture of axons of the corpus callosum, brain stem, cerebellar peduncle, internal capsules, rupture of blood vessels in sections, and the appearance of hemorrhagic infarction foci in numerous subcortical formations, less often in the peduncle [17].

In the late stages, post-traumatic necrosis may develop. Histological examination reveals circulatory disorders (white and red infarction foci, edematous effusions, perivascular encephalolysis), inflammatory processes (purulent and hemorrhagic effusions), and compression of adjacent tissues with the resulting scars [19].

Changes in neural tissue, such as demyelination and neuronal cell damage, are characteristic pathological conditions observed after traumatic brain injury [19].

In traumatic brain injury, signs of secondary intracranial and extracranial brain injury must develop for primary brain damage. Intracranial changes include changes such as brain hematoma compression, impaired hemo- and microcirculation, cerebral edema, intracranial infection, and hydrocephalus. Extracranial changes include signs such as hypoxemia, arterial hypotension, anemia, impaired blood-brain barrier permeability, and impaired neurohumoral control.

The system that undergoes the main pathogenetic changes in the acute period of traumatic brain injury is the dysfunction of the central nervous system [1,3,9,13]. Pathological conditions such as contusions, hematomas, dislocations, and brain contusions cause dysfunction of the central nervous system [4,16].

Clinical and pathogenetic changes in injuries are manifested depending on the location of the lesion and the nature of the central nervous system damage [5,28]. Depending on the time of the lesion, the lesion is divided into manifestations of resistance to changes, and depending on the changes in the surrounding tissue, inflammation, edema and secondary hemorrhage. Focal changes cause various pathological changes depending on their location. General cerebral symptoms develop from varying degrees of change in consciousness to a state of unconsciousness. The development of cerebral edema begins with a period of “colorful interval” and progresses to profound impairment of consciousness [19].

Depending on the mechanism of occurrence and clinical signs, it is divided into 2 types, namely epidural and subdural hematomas. As a result of

these hematomas, compression of the brain stem is observed. Compression of the brain stem manifests a set of symptoms of a diencephalic or mesencephalo-bulbar type [12] (a state of deep coma, hypo- or atonia, areflexia, divergence of the eyeballs, cardiorespiratory disorders). Epidural hematomas gradually increase in size and acquire a distinctly delimited area, in which case the dura mater of the spinal cord is in close contact with the skull [7]. In epidural hematomas, changes in the central nervous system gradually develop, and general cerebral symptoms join. After loss of consciousness, symptoms of pyramidal system insufficiency develop: anisoreflexia, weakness, or paralysis [6]. These processes occur on the opposite side of the hematoma. Dislocation and compression occur on both sides if the pyramidal system is involved. Mydriasis and pupillary light reflex are absent on the side of the hematoma. Mydriasis may be accompanied by ptosis of the upper eyelid.

Subdural hematomas are larger than epidural hematomas. The rapid appearance of the hematoma can accelerate the “color interval” period or completely eliminate it [8]. The main clinical manifestation in this process is hypertensive syndrome [10]. Hypertensive syndrome is caused by compression of the motor center of blood vessels. Bilirubin formed during hemolysis affects nerve fibers, causing clonic convulsions. After the process of cerebral edema, the meninges are affected, causing meningeal signs [11].

In the acute phase of traumatic brain injury, the diffusion of noradrenaline, serotonin, prostaglandin E into the brain and sympathetic stimulation result in spasm of arteries and arterioles, resulting in impaired cerebral blood flow [2].

Experimental studies have shown that products of erythrocyte hemolysis have a spasmogenic effect. In animal studies, the introduction of erythrocyte hemolysis products (oxyhemoglobin, methemoglobin, and mixtures thereof) into the blood of animals caused vasospasm. [21,22].

The diffusion of hemolysis products into the blood leads to the release of prostaglandins from endothelial cells. This process causes the spasmogenic effect of hemolysis [22]. Oxyhemoglobin and other products of hemolysis inhibit endothelin, which relaxes smooth muscle fibers by providing them with nitric oxide. Oxyhemoglobin enhances the release of endothelin, a protein with strong vasoconstrictive properties, from endothelial cells [24]. As a result, the distribution of hemoglobin products damages perivascular nerves and other factors that have a synergistic effect on vasoconstruction, increases the hypoxia process, and affects biologically active substances such as serotonin and potassium [22].

The calcium in the extracellular space contacts the smooth muscle layer of the blood vessel, causing the blood vessel to contract. In the study of the smooth muscle layer of the blood vessels of the brain, hemolysis products cause an increase in the amount of calcium in the extracellular space. As a result, the blood vessel muscle contracts spastically and dies. As a result of the relaxation of the smooth muscles, calcium enters the cell. The removal of calcium from the cell against the concentration gradient is carried out by the calcium-sodium antiporter due to the energy of the transmembrane sodium gradient, which is supported by the energy resources of the cell. In injuries, the release of calcium from the cell is impaired due to energy deficiency [27]. Transcranial Doppler has been studied to show that an increase in blood flow velocity of 120 cm/s is pathological, and an increase of 200 cm/s leads to a cerebral infarction. During vasospasm, the

ratio of blood flow velocity in the middle cerebral artery and internal carotid artery changes. This ratio has been found to exceed the norm. Based on this indicator, it is possible to compare the acceleration of flow due to the development of hyperperfusion with the acceleration of blood flow due to vasospasm. The following factors can also affect the blood flow velocity in blood vessels: increased intracranial pressure, age, existing stenoses in blood vessels, increased blood pressure, circulating blood volume, and hematocrit. Taking these factors into account and monitoring them in dynamics allows you to avoid mistakes [23].

CONCLUSION

In summary, the main areas of change after brain injuries are the basilar artery (a. basilaris) and the middle cerebral artery (a. cerebri media), where excessive calcium ion concentration in the smooth muscle fibers causes spastic contraction of the vessels, resulting in the death of myocyte cells, rupture of blood vessels, and bleeding into surrounding tissues, with the development of pathological changes such as necrosis and dystrophy from primary pathological changes.

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