TREATMENT OF BRAIN CONUSIONS

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Abstract: Brain damage due to traumatic brain injury is divided into primary and secondary. Primary damage is caused by the impact of traumatic force on the bones of the skull, membranes and tissue of the brain, cerebral vessels and the cerebrospinal fluid system.

Keywords: Primary damage, traumatic force, vascular damage, diffuse axonal, glial cells.

Brain damage due to traumatic brain injury is divided into primary and secondary. Primary damage is caused by the impact of traumatic force on the bones of the skull, membranes and tissue of the brain, cerebral vessels and the cerebrospinal fluid system. Primary injuries include foci of brain contusions, primary contusions of the brain stem, diffuse axonal and vascular damage to the brain. With primary damage, the structure of neurons and glial cells is disrupted, synaptic breaks are formed, vascular thrombosis occurs and the integrity of the vascular wall is disrupted. The consequence of primary injury is a decrease in the availability of adenosine triphosphate (ATP) and disruption of the permeability of the cell membrane (“membrane pump”), which leads to cell death or cytotoxic edema.

Traumatic brain injury is one of the most common types of injuries. In the overall structure of injuries, TBI accounts for about 40%. The frequency of traumatic brain injury in Russia ranges from 1.6 to 7.2 cases per 1,000 population per year, i.e. more than 600 thousand people per year. And it is the leading cause of death and disability among people under 45 years of age. The number of people with disabilities after traumatic brain injury in the United States is 3 million people, in Russia more than 2 million. Patients with concussion make up half of all patients with traumatic brain injury. The share of patients with brain contusion accounts for 30%, and 20% have traumatic intracranial hematomas.

Every year, 3.5–4 thousand patients with brain contusions of varying severity are hospitalized in neurosurgical hospitals in Moscow, and more than 2,000 patients with compression of the brain by traumatic hematomas. More than 500 victims with traumatic brain injury undergo surgical treatment. Half of the victims with traumatic brain injury experience consequences of varying severity - from functional disorders to severe neurological symptoms. In severe brain contusions, the outcome may be a vegetative state or minimal consciousness syndrome. Brain contusion is a risk factor for the development of Alzheimer's disease and parkinsonism in the long term. Traumatic brain injury. During the first year after a traumatic brain injury, the likelihood of developing an epileptic seizure is 12 times higher than in the general

population. Post-traumatic epilepsy is detected in 13% of patients who have suffered a moderate brain injury.

A perifocal zone is formed around the source of primary damage, in which cells retain their viability, but become extremely sensitive to the slightest changes in the delivery of oxygen and nutrients (penumbra zone).Secondary (ischemic) brain damage. In response to primary damage, a pathological process occurs, which is an evolutionarily developed inflammatory reaction. These changes are bidirectional, i.e. they cause both damage to cell structures and are neuroprotective in nature.

Immediately after injury, neuronal metabolism increases, which is accompanied by ATP depletion, dysfunction of the transmembrane calcium pump, increased permeability of cell membranes for calcium ions, release of calcium from intracellular stores, which causes depolarization of nerve endings and the release of "excitatory" neurotransmitters (glutamate) from them, which leads to damage to the membranes of neurons and the endothelium of brain capillaries (excitotoxicity). Glutamate, activating postsynaptic complexes, causes an influx of sodium ions into the cell, depolarization and an even greater influx of calcium ions through ion channels. The consequence of a cell overload with calcium is its damage, caused by the activation of phospholipases, proteases and nucleases, leading to disruption of the integrity of cell membranes, phosphorylation and synthesis of proteins and genome expression, and lysis of structural proteins of the cell.

Neuronal death during traumatic brain injury also occurs due to apoptosis. Apoptosis can be triggered either by the direct effect of a traumatic agent on the cell genome, or indirectly by the damaging action of inflammatory mediators. The consequence of the action of factors of secondary brain damage is the disruption of the delivery of oxygen and nutrients to brain cells and their insufficient utilization. Cells located close to the site of primary brain damage (penumbra zone) are especially affected.

Disturbances in cerebral microcirculation, oxygenation and neuronal metabolism occur, and cerebral edema and ischemia develop. Secondary ischemic brain damage occurs in 36–42.6% of patients with moderate traumatic brain injury and in 81–86.4% of patients with severe traumatic brain injury. The development of secondary brain damage significantly aggravates the severity of the condition of victims with traumatic brain injury, impairs the recovery of mental and motor activity of patients and increases the risk of developing an unfavorable outcome. In this regard, prevention and timely correction of factors of secondary brain damage are the most important task in the treatment of victims with severe traumatic brain injury.

According to the accepted classification of injury according to the clinical course and severity of damage to brain tissue, brain contusions are divided into mild,

moderate and severe bruises. There are also special forms of brain contusion: diffuse axonal injury (DAI) and traumatic subarachnoid hemorrhage [9].

The most common brain contusion is of moderate severity - 49%, mild contusion is detected in 33% and severe contusion in 18% of patients. The criteria for determining the severity of a brain contusion are the degree of disturbance of wakefulness, the severity of the patient's condition, the severity of neurological symptoms and data from instrumental diagnostic methods (CT and MRI of the brain, data from laboratory research methods).

A mild cerebral contusion is characterized by a rapid restoration of wakefulness after its initial loss, which lasts for several minutes. The clinical picture is dominated by general cerebral symptoms: headache, nausea, vomiting, dizziness, loss of attention and memory. Nystagmus (usually horizontal), anisoreflexia, and sometimes mild hemiparesis may be detected. During lumbar puncture, cerebrospinal fluid (CSF) may contain red blood cells. Mild brain contusions can be accompanied by skull fractures, but can also occur without them. In 40–50% of patients, CT scans reveal areas of low density—areas of edema-ischemia with a density of 18 to 28 units. N. Histological examination of such lesions reveals edematous brain tissue, there may be ruptures of small vessels, and pinpoint diapedetic hemorrhages. Regression of these morphological changes occurs within 2–3 weeks.

Moderate brain contusion is characterized by loss of wakefulness from several tens of minutes to 2-4 hours. The degree of wakefulness is reduced to the level of moderate or deep stupor and persists for several hours or days. General cerebral symptoms are pronounced. A characteristic feature of moderate brain contusion is the presence of traumatic subarachnoid hemorrhage. Meningeal syndrome is moderately expressed, and CSF pressure is moderately increased (except for victims who have liquorrhea). Some patients may have focal neurological symptoms: moderate hemiparesis and pathological reflexes, sensory disturbances, aphasia. Breathing disorders in the form of moderate tachypnea without rhythm disturbance and not requiring hardware correction. Craniograms reveal skull fractures in the majority of patients (62%), of which 35% have vault fractures, 50% have fractures of the base, and 15% have fractures of the vault and base of the skull.

CT scan reveals areas of brain contusion. Perifocal edema usually does not extend beyond one lobe of the brain. Typically, patients with moderate brain contusion do not require surgical treatment, with the exception of victims with depressed skull fractures.

Severe brain contusion. With this type of injury, the decrease in the degree of wakefulness to stupor or coma lasts from several hours to several days, in some

patients with a transition to apallic syndrome or akinetic mutism. Stem symptoms are characteristic, sometimes hormetonia develops - in response to painful stimulation or spontaneously, bilateral pathological foot reflexes and changes in muscle tone are determined. Violations of vital functions (respiration and circulation) in most cases require correction. Craniograms of almost all victims reveal fractures of the vault, base, or vault and base of the skull. CT scans show areas of brain contusion of varying volume, accompanied by perifocal or widespread edema of brain tissue. A pathological examination reveals foci of brain destruction over a significant extent, both in surface and in depth. The volume of detritus may be equal to or greater than the number of blood clots

A special form of brain contusion is diffuse axonal brain injury (DAI). Taking into account the pathophysiology of the development of brain damage - due to acceleration/deceleration and rotational component, displacement relative to each other of gray and white matter, which have different densities, and the resulting rupture of axonal connections, disruption of axonoplasmic flow - a clinical picture of brain stem damage develops - depression of consciousness to a deep degree. coma, disturbance of vital functions, which require mandatory drug and hardware correction. A decrease in the degree of wakefulness is a characteristic clinical sign of diffuse axonal damage, and in 25% of victims the duration of loss of consciousness exceeds 2 weeks. Mortality with diffuse axonal brain damage is very high and reaches 80–90%, and the majority of survivors develop apallic syndrome. diffuse axonal damage can occur either independently or accompanied by the formation of intracranial hematomas.

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