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MODERN PRESENTATIONSABOUT TRAUMATIC BRAIN INJURY

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Abstract
Craniocerebral trauma – a mass pathology-is characterized by a high frequency of various consequences. Along with anatomical injuries (cranial defects, fractures, intracranial hematomas), neurotrauma triggers, among other things, two opposite processes: dystrophic-destructive and regenerative-reparative, which for months and years go in parallel with the constant or variable predominance of one of them, ultimately determining the presence or absence of one or the other consequences of brain damage.

Keywords: traumatic brain injury, consequences, complications, classification of consequences of traumatic brain injury, forensic medical examination

Introduction

Traumatic brain injury (TBI) – damage by mechanical energy to the skull and intracranial contents (brain, meninges, blood vessels, cranial nerves) – accounts for 25-30% of all injuries of the body, and among the fatal outcomes from them, its specific weight reaches 50-60%. As a cause of mortality and disability in young and younger middle-aged people, TBI is ahead of cardiovascular and oncological diseases. According to the epidemiological study of traumatic brain injuries, about 700 thousand people in our country annually receive brain damage. The socio-economic costs incurred by society due to TBI are enormous. It is no coincidence that the fight against TBI in many countries is elevated to the rank of national problems. Among the causes of traumatic brain injuries in Russia and the CIS countries, household factors dominate (49-78%); at the same time, a high proportion of intentional (criminal) injuries is noted – 28-49%. In contrast to Western countries, transport injuries (mainly road injuries) take the second place – 9.7-29.9%. Occupational injuries account for 12-15%. A complex of primary factors is involved in the biomechanics of the destructive effect of mechanical energy on brain tissues, including the leading ones: 1) a shock wave propagating from the site of application of the traumatic agent to the head through the brain and the opposite pole with rapid pressure drops in the places of impact and shockproof; 2) movement and rotation of the massive large hemispheres relative to a more fixed brain stem during an acceleration–deceleration injury. TBI, depending on its mechanism, severity and

type, leads to different degrees and prevalence of primary structural and functional damage at the molecular, subcellular, cellular, tissue and organ levels with a violation of the central regulation of all body systems, including vital ones. In response to brain damage, there are disorders of cerebral circulation, cerebrospinal fluid circulation, hypothalamic-pituitary-adrenal functions, and the permeability of the blood-brain barrier. Edema and swelling of the brain develop, which, along with other pathological reactions, causes an increase in intracranial pressure. The processes of compression and displacement of the brain are unfolding, which can lead to the insertion of stem formations in the opening of the cerebellar notch or in the occipital-cervical dural funnel. This, in turn, causes further deterioration of blood circulation.

Classification of TBI is a multidisciplinary pathology, located at the intersection of many clinical, medical and biological and social-hygienic disciplines. Hence, there is an urgent need for a unified terminology and classification of TBI. They were developed by the Institute of Neurosurgery. N. N. Burdenko, Russian Academy of Medical Sciences. The classification of TBI is based on the nature and degree of brain damage, since they usually determine the clinical course, treatment tactics, and outcomes. TBI is divided into three degrees of severity: mild, moderate, and severe. Mild TBI includes concussion and mild brain contusions; moderate-moderate brain contusions and subacute brain compression; severe-severe bruises, diffuse axonal damage and acute brain compression. According to the nature of brain damage, there are focal injuries (which occur mainly in the shock–shock biomechanics of TBI), diffuse (which occur mainly in the case of acceleration-deceleration trauma) and combined. According to the danger of infection of the intracranial contents, TBI is distinguished between closed and open. Closed TBI refers to injuries in which there are no violations of the integrity of the head coverings or there are soft tissue wounds without damage to the aponeurosis. Fractures of the bones of the cranial vault that are not accompanied by injury to the adjacent tissues and aponeurosis are included in closed TBI. Open TBI includes fractures of the bones of the cranial vault, accompanied by injury to the adjacent soft tissues, fractures of the base of the skull, accompanied by bleeding or liquorrhea (from the nose or ear), as well as wounds of the soft tissues of the head with damage to aponeurosis. If the medulla is intact, open TBI is considered non-penetrating, and if its integrity is violated – it is considered penetrating. TBI can be: 1) isolated (there are no extracranial injuries); 2) combined (at the same time there are injuries to the bones of the skeleton and/or internal organs);3) combined (different types of energy are simultaneously affected – mechanical and thermal, or radiation, or chemical, etc.). According to the peculiarities of its occurrence, TBI can be primary (when the impact of mechanical energy on the

head is not caused by any cerebral or extracerebral catastrophe immediately preceding TBI) and secondary (when the impact of mechanical energy on the head is caused by immediately preceding TBI by a cerebral catastrophe that caused a fall, for example, in an epileptic seizure or stroke, or by an extracerebral catastrophe, for example, in hypoxia due to a massive myocardial infarction). TBI can be received for the first time and repeatedly, i.e. it can be the first, or the second, or the third, etc. During TBI, there are different periods: acute, intermediate, and long-term. Their temporal and syndromological characteristics are determined primarily by the clinical form of TBI, its nature, type, age, premorbid and individual characteristics of the victim, as well as the quality of treatment.

Clinical forms There are the following main clinical forms of TBI: concussion, focal brain contusions of mild, moderate and severe degrees, diffuse axonal damage, brain compression, head compression. Concussion of the brain. It is noted in 70-80% of victims with TBI. Pathomorphologically, changes are detected only at the cellular and subcellular levels (perinuclear tyrolysis, watering, eccentric position of nuclei, elements of chromatolysis, swelling of neurofibrils, etc.). Electron microscopy reveals damage to cell membranes, mitochondria, and other organelles. There is no macrostructural pathology. Clinically, a concussion is characterized by a loss of consciousness lasting from a few seconds to several minutes. Memory loss may be detected for a narrow period of events before, during, and after trauma (retro -, con - , and anterograde amnesia). Nausea or vomiting is often observed. After regaining consciousness, complaints of headache, dizziness, weakness, tinnitus, and sleep disturbance are typical. There is pain when moving the eyes, double vision when trying to read, vestibular hyperesthesia. Vital functions without significant deviations. In the neurological status, labile, non-rough asymmetry of tendon and skin reflexes, fine-grained nystagmus, minor shell symptoms that disappear within the first 3-7 days can be detected. There are no injuries to the skull bones. The general condition of patients usually improves rapidly within the 1st, less often – the 2nd week after the injury. Computed tomography (CT) scans do not detect abnormalities in the state of brain matter and CSF spaces. Brain contusion. It is characterized by macrostructural damage to the brain substance of varying degrees (hemorrhage, destruction), as well as subarachnoid hemorrhage and fractures of the bones of the arch and base of the skull, the frequency and severity of which largely correlates with the severity of contusion. Brain contusions usually involve swelling and swelling of the brain, which can be local, lobar, hemispheric, or generalized. There are certain changes in the CSF-containing spaces (ventricular system, basal cisterns, convexital subarachnoid slits). Often, the mass effect is expressed to varying degrees. Mild brain contusion. It occurs

in 10-15% of patients with TBI. It is characterized by turning off consciousness after an injury in the range from several to tens of minutes. According to him, the complaints are similar to those of concussion. As a rule, retro-, con - and anterograde amnesia is noted. Vomiting, sometimes repeated. Vital functions are usually without significant impairments. Moderate bradycardia or tachycardia may occur, and sometimes arterial hypertension. Respiration and body temperature without significant deviations. Neurological symptoms are usually mild (clonic nystagmus, mild anisocoria, signs of pyramidal insufficiency, meningeal symptoms, etc.); regresses 2-3 weeks after the injury. Moderate brain contusion. It is noted in 8-10% of victims with TBI. It is characterized by turning off consciousness after an injury lasting up to several tens of minutes or several hours. Usually retro -, con -, anterograde amnesia is expressed for a significant period of time. Headache – often severe. Repeated vomiting may occur. There are mental disorders. Transient disorders of vital functions are possible: bradycardia or tachycardia, increased blood pressure; tachypnea without violation of the breathing rhythm and patency of the tracheobronchial tree, subfebrility. Often expressed shell symptoms. Stem signs are detected: nystagmus, dissociation of meningeal symptoms, muscle tone and tendon reflexes along the body axis, bilateral pathological reflexes, etc. Focal symptoms are clearly manifested (determined by the localization of brain contusion): pupillary and oculomotor disorders, paresis of the extremities, sensory disorders, speech disorders, etc. These nesting marks gradually (within 3-5 weeks) smooth out, but can last for a long time. Severe brain contusion. It is noted in 5-7% of victims with TBI. It is characterized by a loss of consciousness after an injury lasting from several hours to several weeks. Motor arousal is often expressed. There are severe threatening violations of vital functions: arterial hypertension, sometimes hypotension; bradycardia or tachycardia; disorders of the frequency and rhythm of breathing, which can be accompanied by violations of airway patency. Hyperthermia is expressed. Primary stem neurological symptoms often dominate (floating movements of the eyeballs, gaze paresis, tonic multiple nystagmus, swallowing disorders, bilateral mydriasis or miosis, divergence of the eyes along the vertical or horizontal axis, changing muscle tone, decerebration rigidity, suppression or irritation of tendon reflexes, reflexes from the mucous membranes and skin, bilateral pathological stop signs et al.), which in the first hours and days after the injury obscures the focal hemispheric symptoms. Paresis of the extremities (up to paralysis), subcortical disorders of muscle tone, reflexes of oral automatism, etc. can be detected. Sometimes generalized symptoms are noted or focal epileptic seizures. Focal symptoms regress slowly; gross residual phenomena are frequent, primarily from the motor and mental

spheres. Diffuse axonal brain damage (DAP). It is based on tension and rupture of axons in the semioval center, corpus callosum, subcortical and stem formations. It is characterized by a prolonged comatose state from the moment of injury (up to 2-3 weeks). At the same time, stem symptoms are expressed (paresis of the reflex look up eye difference along the vertical axis, bilateral inhibition or loss of photoreactions of the pupils, violation of the formula or absence of the oculocephalic reflex, etc.). Gross violations of the frequency and rhythm of breathing are often observed. Typical postural reactions: coma is accompanied by symmetrical or asymmetric decerebration or decortication, both spontaneous and easily provoked by pain (nociceptive) and other irritations. At the same time, changes in muscle tone are extremely variable, mainly in the form of gormetonia or diffuse hypotension. Hemi-and tetraparesis of the extremities of the pyramidal-extrapyramidal nature is often detected. Vegetative disorders hyperthermia, hyperhidrosis, hypersalivation, etc. are clearly pronounced. A characteristic feature of the clinical course of DAP is the transition from a prolonged coma to a persistent or transient vegetative state, the onset of which is indicated by opening the eyes spontaneously or in response to various irritations (there are no signs of tracking, fixing the gaze, or following at least elementary instructions). Vegetative states in DAP last from several days to several months and are characterized by the development of a new class of neurological signs – symptoms of functional and/or anatomical separation of the hemispheres and brain stem. In the absence of any manifestations of the functioning of the cerebral cortex, subcortical, oral-stem, caudal-stem and spinal mechanisms are disinhibited. Chaotic and mosaic autonomization of their activity causes the appearance of unusual, diverse and dynamic oculomotor, pupillary, oral, bulbar, pyramidal and extrapyramidal phenomena. In the clinic of persistent vegetative states due to DAP, along with the activation of spinal automatism, signs of polyneuropathy also appear. spinal and radicular origin (fibrillation of the muscles of the limbs and trunk, hypotrophy of the muscles of the hand, common neurotrophic disorders). As you get out of the vegetative state, neurological symptoms of dissociation are replaced mainly by symptoms of loss. Among them, extrapyramidal syndrome dominates with pronounced stiffness, discoordination, bradykinesia, oligophasia, hypomimia, small hyperkinesis, and ataxic gait. At the same time, mental disorders are clearly manifested. Compression of the brain. It occurs in 3-5% of patients with TBI. It is characterized by a life-threatening increase after a certain period of time after injury or immediately after it of brain-wide (the appearance or deepening of impaired consciousness, increased headache, repeated vomiting, psychomotor agitation, etc.), focal (the appearance or deepening of hemiparesis, unilateral mydriasis, focal

epileptic seizures, etc.) and stem (the appearance or deepening of bradycardia increased blood pressure, limited upward gaze, tonic spontaneous nystagmus, bilateral pathological signs, etc.) symptoms. Depending on the background (concussion, brain contusion of varying degrees) on which traumatic brain compression develops, the light gap may be expanded, erased,or absent. Among the causes of compression in the first place are intracranial hematomas (epidural, subdural, intracerebral). This is followed by depressed fractures of the skull bones, foci of brain crushing, subdural hygromes, and pneumocephaly.

Diagnosis Recognition of clinical forms of TBI is based on a correct assessment of the anamnesis and clinical signs of brain damage and all its integuments. The most informative diagnostic method is X-ray CT, which allows bloodlessly and painlessly obtaining the most complete picture of violations of anatomotopographic relationships in the cranial cavity caused by TBI. With its help, by changing the density of tissues, it is possible to visualize the location, nature and degree of brain bruises, enveloped and intracerebral hematomas and hygromes, subarachnoid and intraventricular hemorrhages, brain edema, as well as expansion or, conversely, compression of the ventricular system and cisterns of the brain base. A similar role can be played by magnetic resonance imaging.

Treatment

The scope and nature of treatment measures are determined by the severity and type of TBI, the severity of brain edema and intracranial hypertension, disorders of cerebral circulation, cerebrospinalfluid circulation, brain metabolism and its functional activity, as well as concomitant complications and vegetovisceral reactions, the age of the victim and other factors. In case of concussion, conservative treatment is performed, which includes analgesics, sedatives and sleeping pills. For mild to moderate brain injuries, moderate dehydration therapy (furasemide), desensitizing drugs (suprastin, tavegil, etc.) are also prescribed. For subarachnoid hemorrhage, hemostatic therapy is performed (calcium gluconate or chloride, dicinone, ascorutin, etc.).Lumbar puncture for therapeutic and diagnostic purposes is used when there are no signs of compression and dislocation of the brain. With open TBI and the development of infectious and inflammatory complications, antibiotics that penetrate the blood-brain barrier well (semi-synthetic analogues of penicillin, cephalosporins, aminoglycosides, etc.) are used.Torn and bruised wounds of the soft integuments of the skull require primary surgical treatment and mandatory prevention of tetanus (tetanus toxoid, tetanus serum are administered). Compression of the brain with an epidural, subdural or intracerebral hematoma, subdural hygroma, as well as

depressed fractures of the skull bones are indications for surgical intervention – bone-plastic or decompressive trepanation of the skull and removal of the substrate compressing the brain. Resuscitation measures for severe TBI (foci of crushing, diffuse axonal damage) begin at the prehospital stage and continue in a hospital setting. In order to normalize breathing, free patency of the upper respiratory tract is ensured (their release from blood, mucus, vomit, introduction of an air duct, tracheal intubation, tracheostomy), inhalation of an oxygen-air mixture is used, and if necessary, artificial ventilation (IVL). In cases of psychomotor agitation, convulsive reactions, sedatives and anticonvulsants are used (relanium, barbiturates, etc.). In case of shock, it is necessary to eliminate pain reactions, fill the deficit of circulating blood volume, etc. Therapeutic and diagnostic measures, including in patients in a coma, should be carried out in conditions of blockade of pain (nociceptive) reactions, since they cause an increase in the volume of cerebral blood flow and intracranial pressure. For the treatment of cerebral edema and intracranial hypertension, saluretics, osmotic and colloidal osmotic drugs, mechanical ventilation in hyperventilation mode, etc. are used. Saluretics (lasix at a dose of 0.5-1 mg / kg per day) are prescribed on the 1st day after injury (panangin is administered simultaneously to preventhypokalemia). With the development of a clinical picture of increasing intracranial hypertension, dislocation and compression of the brain due to edema, osmotic diuretics (mannitol, hyperhaes) are used. Use of saluretics the use of diuretics and osmotic diuretics requires careful monitoring and normalization of the water-electrolyte balance. The reduction of intracranial pressure is facilitated by mechanical ventilation in the mode of hyperventilation with an oxygen-air mixture (which also provides prevention and treatment of brain hypoxia and its consequences). To improve venous outflow from the cranial cavity and reduce intracranial pressure, it is advisable to put the patient in a position with his head raised. In severe TBI, vasoactive drugs (eufillin, cavinton, sermion, etc.), proteolysis inhibitors (kontrikal, gordox, etc.) are used according to indications. Intensive care also includes the maintenance of metabolic processes using enteral (probe) and parenteral nutrition, correction of acid-alkaline and water-electrolyte balance disorders, normalization of osmotic and colloidal pressure, homeostasis, microcirculation, thermoregulation, prevention and treatment of inflammatory and trophic complications. In order to normalize and restore the functional activity of the brain in TBI, metabolic drugs (nootropics, phenotropil, cytoflavin, etc.), drugs that improve microcirculation (tanakan, stugeron, mexidol, etc.) are prescribed. Anticholinesterase drugs (cytokoline or ceraxone, gliatilin, cereton, etc.) are used as neuroprotectors.

Measures for the care of patients with TBI include: prevention of pressure sores, hypostatic pneumonia (systematic turning of the patient, banks, massage, skin toilet, etc.), passive gymnastics to prevent the formation of contractures in the joints of paretic limbs. In patients with depression of consciousness to sopor or coma, impaired swallowing, reduced cough reflex, it is necessary to carefully monitor the free patency of the respiratory tract and use suction to release saliva or mucus from the oral cavity, and in case of tracheal inturbation or tracheostomy, sanitize the lumen of the tracheobronchial tree. Careful monitoring of physiological shipments is carried out. The necessary measures are taken to protect the cornea from drying out in comatose patients (instilling vaseline oil in the eyes, closing the eyelids with a band-aid, etc.) and the oral cavity is regularly cleaned. Patients who have received TBI are subject to rehabilitation treatment and dispensary follow-up.

Forecast

In mild TBI (concussion, mild brain contusions), the prognosis for life and recovery is usually favorable if the victim follows the recommended treatment and behavior regimen. With moderate TBI (moderate bruise), it is often possible to achieve full recovery of patients ' labor and social activity. However, a number of patients develop persistent asthenia, headaches, vegetative-vascular dystonia, static and coordination disorders, and other neurological symptoms. It should be borne in mind that with open TBI, various purulent-inflammatory complications can occur (meningitis, encephalitis, ventriculitis, brain abscesses, etc.). With severe TBI (severe bruise, diffuse axonal damage, brain compression), the mortality rate reaches 30-50%. Among the survivors, there is a significant disability, the leading causes of which are mental disorders, epileptic seizures, gross violations of motor and other brain functions. At the same time, modern possibilities of rehabilitation therapy often allow us to achieve good results even with severe brain damage.

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