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Clinical Features of Traumatic Brain Injury in Various Kinds of Brain Damage

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ABSTRACT Various circumstances of the injury lead to various types of brain damage. The main types of destructive effects are countrecoup effect and acceleration/deceleration. The high intensity injuring force creates conditions for occurrence of combinations of different types of damage leading to aggravation of pathological processes caused by trauma, complication of clinical picture, difficulties of diagnosis and treatment, prolongation of hospital stay, and requires an additional methods of research and treating the injured. Finding the genesis of symptoms observed upon neurologic examination, and especially the differential diagnosis between primary and secondary lesions of the brain stem are nessesary to choose the emergency care for victims with severe traumatic brain injury, as well as to forecast the outcomes of treatment. The dynamics of neurological symptoms (level of wakefulness, pupil size, eyeball mobility, muscle tone and limb movement disorders, pathological plantar reflexes) have significant differences in patients with various types of brain damage, which makes a regular assessment of neurological status extremely important in these patients.

Keywords: traumatic brain injury, types of brain damage, neurological status, forecasting the outcomes of treatment

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DAI — diffuse axonal injury
DC — decompressive craniotomy
DS — dislocation syndrome
EDH — epidural hematomas
ICH — intracranial hypertension
IH — intracerebral hematoma
ICP — intracranial pressure
OR — oculocephalic reflex
SDH — subdural hematomas
TBI — traumatic brain injury

INTRODUCTION

Treatment of traumatic brain injury (TBI) is one of the most important health problems in any country in the world, as it is the main cause of death and disability in the working people [1, 2]. The number of patients with severe TBI, brain contusion, and intracranial hematomas of traumatic origin is increasing annually, reaching 40–50% of cases from all victims with a head injury [3]. The fatal outcome occurs in 60–80% of patients with severe head injury [4, 5].

PATHOPHYSIOLOGY OF TBI

Different types of brain damage occur depending on the mechanism of injury. There are two main types of destructive effects leading to brain injury: contrecoup effect and acceleration/deceleration [1, 3]. In the contrecoup type of impact, the damage is caused by direct contact of the traumatic agent with the victim's head and the resulting acceleration leading to a contrecoup injury due to the collision of the cerebral hemispheres with the bones of the skull from the opposite side. At the site of application of traumatic force, linear or depressed fractures of the skull bones occur, foci of bruises and damage to the vessels of the dura mater with the formation of epidural hematomas (EDH) are formed [6–10]. Acceleration/deceleration causes the movement and rotation of the cerebral hemispheres relative to a more fixed trunk, and there may be no direct contact of the traumatic force with the victim's head. Under these conditions, there are both diffuse lesions (concussions, diffuse axonal lesions), and focal brain contusions and subdural hematomas (SDH). In real conditions, both types of dynamic effects of mechanical energy are most often encountered simultaneously and, depending on the prevalence of the shock or impulse mechanism, one or another anatomical form of damage or their combination occurs [1, 5, 11].

Subdural hematomas are formed as a result of injuring blood vessels supplying the cortex and lining of the brain, and veins flowing into the sinuses. They make up the majority of intracranial hematomas which is about 50%. Postoperative mortality in patients operated on for acute SDH is 40-60%. In patients operated on with a decrease in the degree of wakefulness to coma, this indicator increases to 57–82% [12–16]. EDH is most often formed due to damage to the main trunk or branches of the middle membranous artery at the fracture site or during bone deflection. Postoperative mortality among patients operated on for traumatic EDH is 7–30%. Risk factors include an increase in EDH volume of more than 50 cm^3, displacement of median structures by more than 10 mm, compression of basal cisterns and the traumatic subarachnoid hemorrhage [5, 14, 17, 18].

Patients with brain injuries account for 15% of the total number of victims with head injury [1, 10, 11]. Over time, evolutionary changes in the foci of contusion occur either in the direction of reverse development, or in the direction of increase. Clinically, the process of organizing contusuon is manifested by coagulation of neurological symptoms [3, 10, 19]. The progression of foci of contusion, according to different authors,

is observed in 30-51% of victims. It leads to an increase in intracranial hypertension, dislocation of the brain stem, which is the main cause of deaths in 62% of victims [21–23].

With diffuse brain lesions at the morphological level, ruptures of axons and small vessels with the formation of hemorrhages in the white matter, corpus callosum and brain stem are revealed. The key pathogenetic factor here is the development of angular acceleration of 0.75–1 rad/s^2, while there may be no contact of the head with an object at all. Axon rupture leads to the development of coma from the moment of trauma, as well as stem symptoms, severe muscle tone disorders, symptoms of oral automatism and autonomic disorders [4, 9].

Different types of brain damage are not a simple sum, but mutually aggravate each other's course [1]. For example, the risk of herniation of the hippocampal uncus increases sharply with a combination of crush foci in the temporal lobe with intracranial hematomas, which increases postoperative mortality [10, 15]. Many authors have confirmed that contusion in combination with hematomas tend to increase more often. The presence of foci of contusion, in turn, aggravates the course of small volume hematomas, which in themselves do not create a mass effect and do not require surgical treatment [4, 23]. On the other hand, many researchers argue that surgery itself to remove hematomas is one of the triggers for the progression of foci of brain contusion due to a sharp increase in blood flow in the penumbra and parapenumbra zones [11, 22].

It seems extremely important to determine the genesis of the symptoms observed during a neurological examination of patients with a head injury and differential diagnosis between primary lesions of the brain stem and dislocation syndrome (DS), which is necessary to decide on the feasibility of surgical intervention or conservative therapy and to predict treatment outcomes.

The most important thing in the TBI clinic belongs to DS. Anatomical and morphological phases of the dislocation process (protrusion, herniation and strangulation) were identified by S.M. Blinkov and N.A. Smirnov (1967). Other researchers distinguish from 4 to 7 stages of DS [1, 2, 9, 24]. The classification of *F. Plum, J.B. Posner developed* in 1980 is most common. In this publication the authors suggested a stepwise description of the DS in supratentorial brain compression based on an assessment of level of consciousness, stem reflexes, motor system disorders and respiratory function. However, this work was carried out with patients with acute cerebrovascular accidents and with oncological diseases of the brain, in which the increase in intracranial hypertension (ICH) and stem dislocation occur much more slowly than with TBI [24].

CLINICAL PICTURE OF HEAD INJURY

Among the neurological symptoms characteristic of TBI, the state of consciousness is the most important. There are qualitative (productive) and quantitative (deficient) forms of impaired consciousness [1, 4, 24, 25]. Deficit forms of consciousness depression are of great importance in neurotraumatology, because they are often the result of increased intracranial pressure and dislocation of the brain stem in TBI, which requires immediate decompressive craniotomy (DC). When describing deficient forms of impaired consciousness, the term “wakefulness suppression” is often used recently, which makes it possible to distinguish between the different nature of these symptoms more clearly. In our country, the classification of A.N. Konovalov et al. (1982) is mostly used to describe the degree of consciousness depression according to which clear consciousness, obtundation, sopor, moderate, deep and atonic coma are distinguished (Table 1).

Table 1

The ratio of the states of consciousness and the Glasgow Coma Scale

State of consciousness	Description	Glasgow Coma Scale (score)
Clear	Arbitrary eye opening, targeted reactions, performance of instructions, correct orientation, free speech	15
Moderate obtundation	Drowsiness, slowness when performing instructions, elements of disorientation, difficult speech	13-14
Deep obtundation	Severe drowsiness, execution of only elementary instructions, disorientation, minimal verbal contact	10-12
Sopor	Eye opening to a pain stimulus, location of pain, inability to perform instructions, no verbal contact	8-9
Moderate coma	Unarousable unresponsiveness, uncoordinated motor response to a pain stimulus, possible spontaneous motor activity, impaired sphincters regulation	6-7
Deep coma	Unarousable unresponsiveness, no motor activity	4-5
Terminal (atonic) coma	Unarousable unresponsiveness, deep muscle atony, areflexia	3

According to the literature, in patients with a decrease in the level of wakefulness to a deep coma upon admission to a hospital in 87.5% of cases, the ICP level was more than 30 mmHg , whereas if the level of wakefulness was reduced due to the initial contusion of the brain stem, there could be no ICH. Therefore, to select the optimal treatment tactic, it is necessary to differentiate the causes of the decrease in the level of wakefulness using neuroimaging methods. Lowering the level of wakefulness in victims with various types of brain damage has fundamental differences. Primary contusions are characterized by a decrease in consciousness to a coma from the moment of injury, while in the event of a hematoma, consciousness depression occurs gradually, after a bright interval, as the volume of the hematoma increases due to dislocation of the brain stem [4, 26].

The anatomical proximity of structures responsible for the level of wakefulness and formations that provide innervation of the eye determines the close relationship between pupillary symptoms, mobility of the eyeballs and changes in the level of wakefulness [4, 8]. Disorders of the innervation of the pupils upon admission to the hospital are observed in 85% of patients with severe head injury. In 86.7% of patients who did not have pupil changes upon admission, these symptoms occur during the first 3 days of observation, and much more often (in 87% of cases) in unoperated patients. These symptoms are of great importance in predicting the outcome of treatment of patients with severe head injury. There is a direct dependance between the volume of various intracranial hematomas and the incidence of pupil innervation disorders. Anisocoria, a symptom of the temporal lobe herniation into the opening of the cerebellum, is of major diagnostic value in the dynamic observation of victims in the acute period of TBI. Anisocoria develops at intracerebral hematomas (IH) of more than 49 cm^3, multiple hematomas larger than 73 cm^3 and acute SDH, subacute SDH and EDH of average volume over 100 cm^3 [26]. A statistically significant correlation between anisocoria and ICP level in the early postoperative period was also revealed. When the ICP level was less than 20 mmHg, mydriasis of one pupil was noted in 12.5% of the victims, in ICP from 21 to 30 mmHg it was observed in 25%, in ICP from 31 to 40 mmHg — in 33.3%, more than 41 mm Hg — in 83.3% of patients [11].

The restriction of mobility of the eyeballs is detected in 36.64% of patients [7]. Fully preserved passive movements of eyeballs when checking the oculocephalic reflex (OCR) or conducting a vestibuloocular test in a patient in a coma indicates disintegration of the cortex and brain stem in the absence of gross damage to the midbrain and bridge. Moreover, an increase in the mobility of the eyeballs correlates with a deepening of the coma [4, 24]. According to some authors, the restriction of OCR in the vertical plane is one of the main signs of a dislocation lesion of the midbrain and is not typical for primary trauma of the brain stem [1, 24]. However, it is believed that in patients with diffuse axonal injury (DAI), the horizontal component of the OCR is much better than the vertical, which is a manifestation of stem reflexes dissosiation in the rostrocaudal direction [27]. OCR is of great prognostic value in patients with head injury: patients are discharged from the hospital with preserved eyeball

mobility in 84.62% of cases, and in its absence, 61% of patients die [7]. However, despite the high diagnostic and prognostic significance of this symptom, the characteristics of management of patients with severe head injury do not allow verification of the oculocephalic and vestibuloocular reflexes. First, the cervical spine trauma which often accompanies traumatic brain injury is a contraindication to head rotations when checking OCR; second, the damage to the tympanic membrane excludes the possibility of infusion of cold water into the ear for conducting the vestibuloocular test [8, 24]. This is probably why there is little information in the literature about the use of this important neurological symptom in patients with severe head injury.

The diagnostic and prognostic significance of changes in muscle tone, and especially posotonic reactions, is not in doubt among researchers, which is covered in a large number of literature [1, 4, 7, 12, 27]. Being a manifestation of functional or anatomical dissociation of the cortex and brain stem, posotonic reactions are always combined with depressed level of wakefulness, which is already a poor prognostic sign itself [4, 7, 27]. Various disorders of muscle tone occur in 32.7% of patients with severe head injury, with 42.9% of patients experiencing decerebral rigidity, 34.7% with hypotension with hyporeflexia, 16.3% with atony with areflexia, and 6.1 % - decortication rigidity. A fatal outcome occurs in 78.6% of patients with impaired tone [7]. In patients with brain injuries in the presence of muscle tone disorders, mortality reaches 67.4%. The same symptoms as signs of brain stem compression are risk factors for the progression of contusion foci [11]. There is a correlation between the type of traumatic hematomas and the incidence of various disorders of muscle tone: most often these symptoms are found in subacute SDH (77%), victims with acute EDH (64%) are on the second place, and third are victims with acute SDH and ICH (28–29%). We also revealed the dependence of impaired muscle tone on the lateral dislocation: if the tone is not impaired, the median values axial dislocation is 5 mm, 11 mm in decorticate posturing, 12 mm in decerebrate posturing , and 13 mm in diffuse muscular hypotonia [4]. In 80% of patients with acute SDH, ICH, brain injuries and their combinations, with normal muscle tone, an increase in ICP level to 20 mmHg is detected. If muscle tone is increased, ICH is observed in 75%, in 85.7% with posotonic reactions and in 100% of patients with diffuse muscle hypotension. At the same time, ICP for muscle hypotension in 75% of victims reaches 30 mmHg and more. These results make it possible to attribute violations of muscle tone and postural reactions to reliable signs of an increase in ICP in patients with severe head injury [26]. However, the prognostic significance of changes in muscle tone in various types of traumatic brain injuries remains unexplored at the moment.

Disorders of movements in the limbs play an important role in topical diagnosis and in the dynamic monitoring of patients with severe head injury. By the presence of pyramidal mono- or hemiparesis, one can judge the localization of cortical lesions of the brain. Contralateral hemiparesis is a manifestation of cerebral hemisphere injury, and with the development of dislocation syndrome, paralysis of the ipsilateral limbs occurs. Pyramid triple and tetrapareses occur for lesions in the sagittal and parasagittal region, with bilateral injuries. The pronounced extrapyramidal component during tetraparesis in the form of various disorders of muscle tone is characteristic of DAI [4, 7, 27]. It should be noted that the diagnostic significance of assessing the state of motor function in patients with various anatomical forms of head injury during clinical observation and in terms of prognosis in literature is insufficiently covered.

Pathological foot signs are markers of damage to the central motor path of various origins. In clinical practice, the Babinsky symptom is most often assessed. The presence of a unilateral Babinsky symptom may indicate both damage to the supranuclear part of the pyramidal pathway on the contralateral side due to contusion or hematoma, and compression of the cerebral peduncle on the ipsilateral side due to the dislocation process (an early stage of the oculomotor nerve according to Plum and Pozner) [4, 7, 24]. Bilateral foot reflexes in TBI patients are a formidable sign indicating damage to the stem structures of both primary and secondary (dislocation) genesis. A bilateral symptom of Babinsky is detected during the progression of the dislocation process to the late stage of the oculomotor nerve and at deeper stages [4, 24, 27]. However, the significance of this symptom in the diagnosis and prognosis of victims of head injury in literary sources is practically not covered.

The severity of the symptoms described above, the dynamics of their development and combination with other symptoms largely depend on the anatomical form of brain damage. In particular, in patients with acute SDH and EDH, a violation of the level of wakefulness almost linearly depends on the volume of the hematoma, which is associated with the staging of DS [3, 4, 28].

Epidural hematomas in 70–85% of cases are manifested by a rapid increase in DS. A “lucid space” occurs in 45–50% of the injured, and 22–56% of patients are admitted to neurosurgical hospitals with a decrease in the level of wakefulness to coma [3, 4, 10]. Focal symptoms prevail over common cerebral signs and contralateral pyramidal syndrome is the leading one. Typically, deep hemiparesis is observed with a hematoma volume is more than 80 cm^3. Anisocoria is less common than hemiparesis (in 25–60% of cases), and in 85–90% of patients with altered pupils, mydriasis is observed on the side, which is ipsilateral hematoma [1, 3, 4]. In contrast, a predominance of cerebral symptoms over focal is typical for victims with SDH. From 37 to 80% of victims with SDH are admitted to the hospital with a decrease in the level of wakefulness to coma. The decrease in the level of wakefulness in these cases occurs more smoothly than with EDH, and a lucid space is found in 12–38% of patients. The leading focal symptom in 30-50% of patients is homolateral mydriasis with inhibition of photoreaction and limitation of the mobility of the eyeball, which indicates the radicular origin of the lesion. The neurological picture in acute SDH is characterized by extreme variability due to the fact that in 95% of cases they are combined with brain injuries [3, 4, 21].

Most researchers note a direct dependence of neurological symptoms and treatment outcomes on the volume of hematomas, but there are works that refute this opinion. In a study by Dutch scientists, *W.A. van den Brink et al.* (1999), which had been in the Rotterdam hospital for 9 years, the dependence of treatment results on the presence of subarachnoid hemorrhages, and not on the volume of hematomas, was revealed. However, the materials and methods of this work indicate that the study included patients with a combination of hematomas and small focal brain injuries, which could not but affect the patients' condition, treatment outcomes and study results [29].

The clinical manifestations of brain contusions depend both on the location of the lesion, and its volume and tendency to evolution. Primary centers of crushing in the convexital parts of the cerebral hemispheres in 20% of cases are formed as a result of the depression of the bones of the cranial vault [1, 4, 11, 19]. In prognostic terms, cortical and subcortical contusion are the most favorable. Contusion of the frontal lobes account for 40–50% of brain contusion. In 40% of patients with injuries this location developed disturbances of consciousness of the disintegration type contralateral mono- and hemiparesis, but more often, especially when large amounts of damage, signs of dislocation of the brain stem prevail [4, 25]. Injuries to the temporal lobes account for 35–45% of all brain injuries. Temporal location of contusion is extremely dangerous, since herniation of the temporal lobe can occur even at a normal ICP level [1, 3, 30]. Contusion of the parietal lobes are much less common than contusion of the frontal and temporal location. Central paresis of the contralateral limbs with a decrease in muscle tone of varying severity are common. The comparative remoteness of the parietal lobe from the stem structures ensures a slower development of the stem syndrome even with extensive lesions [1, 4, 8]. Contusion of the occipital lobes is relatively rare, since the shock-absorbing effect of the cerebellum affects. Clinically, cerebral symptoms, visual impairment, and ataxia predominate [3, 4, 12].

Lesions of the median structures of the brain can occur both by the mechanism of crushing or the formation of an ICH due to contusion, as a result of DAI, and without anatomical destruction due to impaired functioning of neuromediator systems. The clinical picture of DAI is primarily characterized by decreased level of wakefulness to coma from the moment of injury, often turning into a transient or persistent vegetative state, disturbances of OCR both in the vertical and horizontal planes, tetrasyndromes of pyramidal and extrapyramidal type, various changes in muscle tone [4, 7, 27].

The combination of brain contusion with EDH and SDH noticeably transforms the clinical picture of isolated hematomas. Due to primary contusion, the lucid space eliminates, and the initial decrease in wakefulness reaches a coma. Hemiparesis significantly deepens even to the degree of plegia and becomes ipsilateral or bilateral [4]. Mydriasis is more common on the side of the contralateral hematoma, due to contusion formed

by the counter-shock mechanism. They also diagnose diffuse disorders of muscle tone (including post- tonic reactions) and bilateral pathological reflexes [1, 4, 7].

With a combination of hematomas and contusion of the brain, cerebral symptoms often prevail over local ones, which mask the clinical signs characteristic of isolated forms of brain damage, which unifies the clinical picture. The prevalence of disturbances in vital functions and depression of wakefulness in the late stages of DS are clinically indistinguishable from primary contusion of the brain stem and extensive contusion of the cerebral hemispheres. These features determine the complexity of early diagnosis, assessing the dynamics of the condition of the victim, as well as the choice of therapy and predicting the outcome of severe head injury [1, 4, 28].

CONCLUSION

Despite the apparent clarity of the clinical symptoms of a traumatic brain injury, further research and systematization of the neurological manifestations of various types of traumatic brain injuries and their combinations will improve the quality of diagnosis, optimize the treatment process and improve treatment outcomes for patients with severe traumatic brain injury.

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