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DIAGNOSIS AND TREATMENT OF TRAUMATIC BRAIN INJURY

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The article analyzes aspects of diagnostics and treatment of craniocerebral injuries. Posttraumatic pathology of the brain and skull can manifest itself already in the acute period, but mainly in the intermediate and late periods of traumatic disease. The consequences of craniocerebral injury (CCI) are widespread and have a clear tendency to continuously accumulate.

Key words: traumatic brain injury, neurosurgical treatment, neuroimaging

Post-traumatic pathology of the brain and skull can manifest itself already in the acute period, but mainly in the intermediate and late periods of traumatic disease. The consequences of traumatic brain injury (TBI) are widespread and have a clear tendency to continuously accumulate. They are a "hard-to-dissolve sediment" that accumulates over many years and, in essence, determine the health of the population, the humanitarian, social and economic significance of the problem under analysis. Most chronic patients with neurological, psychiatric and somatic profiles have a traumatic anamnesis, suffering from one or another consequence of TBI. Suffice it to say that the number of disabled people due to TBI in Russia exceeds 2 million, in the USA - 5.5 million. The first studies conducted by the N.N. N.N. Burdenko's probing epidemiological studies revealed that the frequency of only surgically significant consequences of TBI is 15–18 new cases per 100 thousand of the population, or about 25 thousand victims in Russia annually require neurosurgical care for cranial post-traumatic pathology [1, 2, 3, 4].

Clinical classification of TBI consequences

The classification of the consequences of TBI developed at the N.N. Burdenko Neurosurgery Research Institute is based on the following principles:

• pathogenesis of consequences;

• morphological substrate;

• clinical manifestations.

As is known, the intracranial space is occupied by brain matter (≈85%), cerebrospinal fluid (≈10%) and blood (≈5%), which, like the hard and soft tissues of the head, are affected by mechanical energy. Accordingly, we consider 3 groups of morphological consequences of TBI:

1) fabric:

• brain (atrophy, scars, adhesions, etc.);

• cranial (defects, osteolysis, osteosclerosis, etc.);

2) liquorodynamics (dyscirculation, disresorption, liquorrhea, liquoroma, etc.);

3) vascular (dyscirculation, ischemia, thrombosis, etc.).

They correlate with 3 identified groups of clinical forms of TBI consequences: 1) predominantly tissue; 2) predominantly cerebrospinal fluid; 3) predominantly vascular [5, 6, 7, 8, 9, 10]. We identified the following clinical forms of tissue consequences of TBI:

• post-traumatic brain atrophy: – local, – diffuse;

• post-traumatic arachnoiditis;

• post-traumatic pachymeningitis;

• meningeal-cerebral scars: – without foreign bodies, – with foreign bodies;

• cranial nerve damage;

• skull defects;

• post-traumatic skull deformation;

• combined. Clinical forms of cerebrospinal fluid dynamic consequences of TBI:

• hydrocephalus: – active, – passive;

• porencephaly;

• meningoencephalocele;

• chronic hygromas;

• cerebrospinal fluid cysts; liquorrhea: – without pneumocephalus, – with pneumocephalus;

• combined. Clinical forms of vascular consequences of TBI:

• ischemic lesions; • chronic hematomas;

• aneurysms: – true, – false;

• arteriosinus anastomosis: – carotid-cavernous anastomosis, – other arteriosinus anastomoses;

• sinus thrombosis; • combined.

Of course, in reality, tissue, cerebrospinal fluid and vascular consequences of TBI are often combined, but identifying their main component is always important for treatment tactics, as well as social protection of patients. All consequences of TBI, in addition, should be divided into traumatic and iatrogenic. This is practically important; for example, most skull bone defects are iatrogenic. It is clear that each clinical form of TBI consequences is characterized by its own symptoms and development dynamics. But it is advisable to highlight the leading post-traumatic syndromes common to all consequences of TBI: 1. Neurological deficit. 2. Mental dysfunction. 3. Autonomic dysregulation. 4. Epileptic. Based on the study of patho- and sanogenesis, clinical analysis using a set of neuroimaging methods, we have developed and introduced into practice conceptual approaches to the treatment of the main surgically significant consequences of TBI [11, 12, 13, 14, 15, 16]. Chronic subdural hematomas Post-traumatic chronic subdural hematoma (CSH) is an encapsulated voluminous hemorrhage located under the dura mater (DMA) and causing local and general compression of the brain. CSH differs from acute and subacute traumatic hematomas by a delimiting capsule that determines all the features of their patho- and sanogenesis, clinical course and treatment tactics. The capsule of the CSH is usually distinguishable and begins to function 2 weeks after the subdural hemorrhage. This period is accepted by most authors to differentiate chronic hematomas from acute and subacute ones. At the same time, the development and organization of the CSH capsule is a process that continues for months and years. The capsule of chronic hematomas consists of connective tissue fibers and newly formed thin-walled vessels. The contents of the CSH cavity are usually altered blood - a dark brown or brownish-green

liquid with small fibrin clots. By closing the contents of the CSH, the capsule forms a relatively autonomous formation that coexists with other components of the intracranial space. The dynamic equilibrium of intracranial volumes is easily disturbed under the influence of a variety of circumstances and factors. The volume of the CSH usually increases due to repeated micro- or macrohemorrhages from defective vessels of the capsule, which is facilitated by the accumulation of fibrin degradation products in the hematoma cavity. The convexital-parasagittal arrangement with the spread to 2-3 lobes or the entire hemisphere is typical for CSH. About 1/10 of CSH has a bilateral arrangement. The volume of CSH varies from 50 to 250 ml, most often it is 100-150 ml. Most often, CSH is caused by TBI. According to our data, it was the cause of CSH formation in 80% of patients. Then follow vascular catastrophes: ruptures of arterial aneurysm, bleeding from arteriovenous malformation or hemorrhagic strokes. Other causes of CSH formation can be infectious diseases, hemorrhagic diathesis, hemophilia, toxic lesions, bleeding from meningiomas, angiomas and metastases, craniocerebral disproportions, etc. It should be noted that sometimes CSH are iatrogenic, especially in connection with the spread of bypass operations. CSH accounts for 1–7% of all space-occupying lesions of the brain: among surgically significant cerebral hemorrhages, their share increases to 12–25.5% [17, 18, 19, 20, 21, 22, 23, 24, 25, 26]. The main reasons for the increase in CSH in recent years are the prevalence of TBI and cerebrovascular diseases. An important role is played by the aging of the population, which, due to age-related brain atrophy, changes in the vascular system, and rheological properties of the blood, creates additional prerequisites for the formation of CSH. Craniocerebral anomalies in children are of similar importance. A certain role in the increase in CSH is played by alcoholism and adverse effects on the brain and other organs. Previously, CSH was diagnosed exclusively in elderly and senile individuals. Currently, CSH has become significantly “rejuvenated” and is often found in young and middle-aged individuals, as well as in children. The incidence of CSH in different countries varies from 2 to 13 cases per 100,000 population per year, significantly increasing in elderly and senile individuals. Operations for CSH are becoming more and more common in neurosurgery. Clinical picture The "light" period in CSH can last for weeks, months, and even years. The clinical manifestation is polymorphic. Both gradual development of compression syndrome and sudden sharp deterioration of the patient's condition to stupor and coma are observed spontaneously or under the influence of various additional factors (mild repeated head injury, overheating in the sun, alcohol consumption, colds, etc.). The clinical picture may resemble various diseases of the central nervous system: benign and malignant brain tumors, stroke, spontaneous subarachnoid

hemorrhage, cerebral atherosclerosis, encephalitis, epilepsy, etc. During the period of the developed clinical picture of CSH, changes in consciousness in the form of stunning or amentive confusion with memory impairment and orientation are common. The headache has a meningeal tint. Dullness of percussion sound over the hematoma area may be noted. Among the focal signs of CSH, the leading one is pyramidal hemisyndrome. Speech disorders, hemihypalgesia, hemianopsia, etc. are also encountered. Extrapyramidal symptoms (hypomimia, bradykinesia, general stiffness, increased muscle tone of the plastic type, subcortical tremor, etc.), which go beyond age-related changes, have a large specific weight in the clinical picture of CSH. Often, proboscis and grasping reflexes are detected, as well as static and gait disorders. In the phases of clinical decompensation, secondary dislocation midbrain symptoms appear (upward gaze paresis, bilateral pathological reflexes, anisocoria, spontaneous nystagmus, etc.). The following main patterns were established in the age-specific analysis of the clinical picture of chronic cerebral hypertension: • the usual predominance of general cerebral symptoms over focal ones in all age groups; • a change in the structure of general cerebral symptoms with increasing age of patients: from hypertensive in childhood, young and middle age to hypotensive in the elderly and senile, which is associated with the predominance of cerebral edema and intracranial hypertension in younger age groups, and of cerebral collapse and intracranial hypotension in older age groups; • an increase (with age) in the role of vascular factors in the genesis of symptoms in chronic pulmonary hypertension, which, together with involutional changes, determines a higher frequency of mental disorders in elderly and senile individuals compared to younger age groups; • a predominance of motor disorders among focal symptoms. The degree of their severity and stability usually worsens with the age of the patient.

Treatment

Previously, the only radical method of treating patients with CSH was considered to be osteoplastic craniotomy with total removal of the hematoma together with the capsule. Now, minimally invasive surgery is usually used. Evacuation of the hematoma contents through a burr hole (imposed in the projection of the greatest thickness of the CSH) with washing of its cavity with isotonic sodium chloride solution and subsequent installation of a closed external drainage system for 1-3 days is effective. Drainage is performed unforced, which reduces the risk of formation of postoperative intracerebral and meningeal hematomas, creates better opportunities for gradual straightening of the long-compressed brain, allows simultaneous drainage of bilateral hematomas and allows manipulations to

be performed under local anesthesia. Strict adherence to the surgical technique prevents air penetration into the hematoma cavity and the occurrence of tension pneumocephalus. Evacuation of the contents of the hematoma and lavage of its cavity are the most important techniques that break the self-sustaining cycle of hemorrhage from newly formed vessels of the capsule. In case of multi-chambered hematoma and hematoma containing dense blood clots, endoscopic removal is successfully performed. In case of planar hematoma in the phases of clinical compensation and subcompensation, conservative management of patients under CT and MRI control is acceptable. Dynamic CT and MRI indicate complete disappearance of hematoma within 1–3 months (see Fig. 1, 2) when using gentle treatment methods. Usually, the rate of clinical improvement significantly outpaces the process of resorption of hematoma. In this regard, the presence of residual fluid in the hematoma cavity after lavage and closed external drainage of its contents in the absence of clinical deterioration cannot in itself serve as an indication for reoperation and patient detention in hospital. Bone-plastic trepanation is indicated in the presence of extensive dense clots in the hematoma cavity and in rare cases of its calcification, as well as in the case of relapse of CSH after unsuccessful double drainage. Possible complications of surgical treatment of CSH are relapses of subdural hematoma, postoperative epidural hematomas, intracerebral hemorrhages, tension pneumocephalus, cerebral edema, cerebral collapse, purulent-inflammatory processes. When using gentle techniques that allow regulating the rate of internal decompression of the brain, complications are much less common and proceed more easily. The prognosis for life and restoration of impaired functions when choosing an adequate method of treatment of CSH is usually favorable even in elderly people. According to our data (427 observations of CSG), good results were achieved in 91.8% of patients, in 3% (although CSG was completely resorbed) the condition remained severe due to other consequences of TBI, repeated operations were required in 4.1% of patients, and the fatal outcome was 1.1%.

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