

CLINICAL PICTURE AND SURGERY OF CONSEQUENCES OF CRANIOCEREBRAL INJURY

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The article analyzes aspects of diagnostics and treatment of consequences of craniocerebral injuries. The predominant mechanism of injury in PBL is a blow to the head against an object or a blow to the head. Most often, the site of application of the traumatic agent is localized in the frontal and frontofacial areas.

Key words: traumatic brain injury, neurosurgical treatment, neuroimaging

Posttraumatic basal liquorrhea

Posttraumatic basal liquorrhea (PBL) is a leak of cerebrospinal fluid (CSF) from the cranial cavity due to damage to the bones of the base and dura mater with a violation of the tightness of the subarachnoid space, as well as with injury to the walls of the ventricles or basal cisterns. PBL occurs in 2-3% of cases of TBI and is a life-threatening pathology, since it can be complicated by purulent meningitis. The predominant mechanism of injury in PBL is a blow to the head on an object or a blow to the head. Most often, the site of application of the traumatic agent is localized in the frontal and frontofacial areas. PBL most often occurs as a result of severe TBI, but is often found in moderate and even mild trauma, while TBI can be both closed and open penetrating. The favorite localization of cerebrospinal fluid fistulas (Fig. 3) is the cribriform plate, often in combination with the frontal, sphenoid sinus, and orbital roof. CSF leakage is possible selectively through the frontal and sphenoid sinuses, as well as the pyramid of the temporal bone. Most often, basal cerebrospinal fluid leak occurs within 1 month after injury, but this does not exclude its manifestation at a later date. In more than 1/2 of observations, the course of basal cerebrospinal fluid leak is complicated by the development of meningitis (single or multiple), which may be the first clinical sign of basal cerebrospinal fluid leak. The longer the basal cerebrospinal fluid leak exists, the greater the likelihood of meningitis [1, 2, 3, 4, 5, 6, 7, 8, 9, 10].



Clinical picture includes a triad of the following symptoms: 1) caused directly by liquorrhea; 2) as a consequence of meningitis associated with PBL; 3) associated with previous TBI

General cerebral symptoms.

The state of consciousness and psyche in the acute period is determined by the severity of TBI. In the intermediate and late periods of TBI, patients with PBL are usually in clear consciousness. The most frequent and constant symptom in patients with liquorrhea is headache. Its main cause is hypotension syndrome. Patients are bothered by a mild but constant squeezing headache, which is accompanied by a feeling of stuffiness in the ears, dizziness, palpitations, discomfort in the chest, abdomen, paresthesia, weakness and other vegetative-vascular and vegetative-visceral symptoms. It is characteristic that cephalgia is most clearly expressed in the vertical position of patients and significantly decreases when moving to a horizontal position. Almost 1/2 of patients with liquorrhea have mental disorders, mainly borderline syndromes - emotional-personal and asthenic. Their severity depends on the severity of the traumatic brain injury, the location of the brain damage, the age of the victim, his medical history, etc. Emotional and personal disorders manifest themselves as apathy, decreased criticism of one's own condition, limited initiative, and memory impairment, primarily for current events. Asthenic syndrome with liquorrhea is characterized by feelings of weakness, fatigue, rapid physical and mental exhaustion, impaired concentration, decreased mental activity, sometimes combined with vegetative manifestations. Patients with liquorrhea typically experience constant discomfort, decreased performance, depressed mood, isolation, fear, and those who have had purulent meningitis are afraid of its recurrence. Frequent clinical symptoms include night cough, which is a consequence of irritation of the mucous membrane of the upper respiratory tract by liquorrhea in a horizontal position. Meningeal symptoms are uncommon in the absence of severe intracranial hypertension syndrome and outside of meningitis. [11, 12, 13, 14, 15, 16] Usually, if they do manifest themselves, they are insignificant and are associated with a history of TBI. Focal neurological symptoms. Among the nesting signs in the clinical picture of post-traumatic cerebrospinal fluid leaks, the central place belongs to such craniobasal symptoms as hyposmia or anosmia. Oculomotor disorders are noted (damage to the III, IV, VI nerves), especially often with fractures of bone structures in the area of the superior orbital fissure. Damage to the optic nerve is observed in 1/3 of patients - from decreased acuity and visual field impairment to blindness. Patients with PBL often have unilateral and bilateral hearing impairment, spontaneous nystagmus, and less often -



taste impairment. Hearing loss, vestibular function, damage to the facial nerve are observed in most cases with a transverse fracture of the pyramid. Motor and sensory disorders in patients with PBL are relatively rare and, as a rule, serve as an indication of a history of TBI. Focal rhinotoneurological symptoms. Clear fluid discharge from the nasal passages is the main clinical sign of rhinoliqorrhea. Visually obvious liquorrhea is detected in 90% of cases, in most of them it is constant or intermittent. Constant fluid discharge from the nasal passages is typical of the cranionasal form of liquorrhea, and periodic, especially when changing body position, is typical of craniosinusal. Different variants of PBL have been traced by the intensity of liquor discharge: rare drops (in almost 1/2 of cases), frequent drops, profuse, portioned ("teapot symptom"), as well as moistening of the nasal passages. Rhinoendoscopically, hypertrophy of the mucous membrane is often detected on the side of liquorrhea, and synechiae and intranasal cerebral hernias are rare. Clear fluid discharge from the external auditory canal is the main clinical sign of otorrhea. Otoscopically, cicatricial deformation of the eardrum or traumatic perforation is often detected. Diagnostics Craniography. In patients with PBL, craniography in standard projections usually reveals skull fractures, primarily at the base, or combined with fractures of the vault (usually the squama of the frontal bone) [17, 18, 19, 20, 21, 22, 23, 24, 25, 26].

Fractures of the bones of the skull base are most often localized in the anterior cranial fossa, less often in the middle cranial fossa. Simultaneous damage to the anterior and middle, and sometimes posterior cranial fossae occurs. Craniography data help to clarify the biomechanics of the injury and the expected path of cerebrospinal fluid leakage. For a shock mechanism with the application of a traumatic agent in the frontal and facial areas, fractures of the frontal bone with a transition to the base - to the anterior cranial fossa are more typical. In this case, the paranasal air sinuses are often damaged, which means that conditions are created for the craniosinusal route of cerebrospinal fluid leakage. Damage to the anterior parts of the skull with an impact mechanism, for example, a fall on the back of the head, is most often localized in the area of the cribriform plate, with the cranionasal route of cerebrospinal fluid leakage predominating. CT. The method allows visualization of both the brain substance and the cerebrospinal fluid space, as well as the bones of the vault and base of the skull. In the intermediate and late periods of TBI, most patients with PBL show on CT the consequences of brain damage of varying severity, mainly in the form of atrophic or cystic-atrophic focal changes, often unilateral, with a predominance in the pole-basal parts of the frontal lobes. CT significantly complements



and surpasses the data of craniography in identifying fractures of the bones of the vault and base of the skull, pneumocephalus. However, conventional CT is often insufficient to identify and clarify the localization of small defects of the base of the skull, cerebrospinal fluid fistulas. For a more accurate determination of the nature of damage and clarification of the localization of bone defects of the bones of the base of the skull, it is advisable to use three-dimensional CT reconstruction of the skull. Accurate localization of bone pathology allows optimizing the planning of minimally traumatic surgical approaches. CT cisternography. For direct visualization of cerebrospinal fluid fistulas, CT cisternography with endolumbar administration of non-ionic radiopaque agents is used [27, 28, 29, 30, 31, 32, 33, 34]. Layered frontal reconstruction of CT sections with a step of 5 mm, as a rule, conclusively reveals penetration of contrasted cerebrospinal fluid beyond the subarachnoid space (Fig. 4). The detected "cerebrospinal fluid path" from the site of fistula formation allows for precise localization of the target of surgical intervention. CT cisternography allows for simultaneous detection of various morphological changes in the cerebrospinal fluid system (diffuse or cystic expansion of the subarachnoid space, communication of the anterior horns of the lateral ventricles of the brain with the basal cisterns in porencephaly, etc.), meningoencephalocele, pneumocephalus, as well as levels of contrasted cerebrospinal fluid in the paranasal sinuses of the skull. It should be remembered that in patients with recurrent liquorrhea and scanty nature of CSF leakage, CT cisternography data may be negative. Contraindications to CT cisternography are occlusion of cerebrospinal fluid pathways and signs of an inflammatory process. CT cisternography is a highly informative and adequate method for determining the anatomical and topographic localization of cerebrospinal fluid fistulas. The method is the "gold standard" in the diagnosis of basal cerebrospinal fluid leaks. MRI. In basal cerebrospinal fluid leaks, MRI provides polyprojection of the study and high contrast of the image of the brain matter and intracranial cerebrospinal fluid spaces, as well as the air-bearing paranasal sinuses. However, the absence of signals from bone structures in MRI limits the use of the method for diagnosing skull base injuries. Surgical treatment In the acute period of TBI, surgical intervention for basal cerebrospinal fluid leakage should be performed 2–4 weeks after the injury, if it does not stop despite conservative treatment (dehydration therapy, periodic lumbar punctures, etc.). The patterns of biological reparative processes determine the time parameters of surgery. Thus, if the fistula cerebrospinal fluid tract that exists during the 3 weeks of the acute period of TBI is epithelialized, it passes into the chronic stage of functioning. In the intermediate and late periods of TBI, the indication for surgical treatment of basal cerebrospinal fluid leakage is its constant or intermittent form, often



accompanied by recurrent meningitis [6]. The concept of reconstructive surgery for basal cerebrospinal fluid leakage is the restoration of a closed circuit of CSF circulation. Due to the low trauma of endoscopic endonasal approaches used to close cerebrospinal fluid fistulas, the latter have increasingly come to be used in basal cerebrospinal fluid rhinorrhea surgery. Less often, when endoscopic approaches are inaccessible for cerebrospinal fluid fistula plastic surgery, direct intracranial access with extra- or intradural approach to the cerebrospinal fluid fistula is used. Intracranial access is performed subfrontally with preliminary osteoplastic trepanation in the frontal region, bilateral or unilateral. Extradural approach to the cerebrospinal fluid fistula is used more often, intradural approach is less often, and sometimes mixed approach – extraintradural approach is used. Communication of cerebrospinal fluid spaces with air sinuses completely excludes the use of allo- and xenomaterials for cerebrospinal fluid fistula plastic surgery due to the high risk of developing purulent-inflammatory complications. The use of autotissues is fundamental in basal cerebrospinal fluid rhinorrhea surgery. Surgery for lumbar cerebrospinal fluid should be accompanied by correction of concomitant pathology of CSF circulation and resorption. Immediately before the main stage of the surgical intervention, an external lumbar drainage is installed. CSF drainage is continued for 5-14 days to adapt CSF dynamics to new conditions after restoration of the closed CSF circulation circuit. It is recommended to use the tunnel technique of lumbar CSF drainage (Fig. 5). If modern diagnostic methods do not allow to establish the localization of CSF fistulas, then in case of a combination of basal CSF rhinorrhea with intracranial hypertension and hydrocephalus, shunting operations are performed: lumboperitoneostomy (in the absence of occlusion of CSF pathways), ventriculoperitoneostomy (in case of blockade of CSF circulation). To prevent postoperative inflammatory complications, antibiotics are administered intravenously during induction of anesthesia (cephalosporins of the II-III generation - 1-2 g). If the duration of the surgical intervention is more than 6 hours, the administration of antibiotics is repeated. Among the early postoperative complications in the surgical treatment of cerebrospinal fluid leakage, meningitis (if defects are allowed during percutaneous installation of lumbar drainage) and cerebrospinal fluid hypotension (associated with either closed external drainage or lumboperitoneal shunting) are possible.

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