Cerebral edema - possibilities of magnetic resonance imaging

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There are problems in medicine, the relevance and importance of which not only does not decrease over time, but also steadily increases. These include edema and swelling of the brain. The enduring interest in this issue of neurosurgeons, resuscitators, neurologists and neuroradiologists is dictated by the great scientific and practical significance of cerebral edema for the clinic. Before the advent of imaging techniques - CT and MRI - the presence of cerebral edema was postulated. The methods of his research were: assessment of clinical manifestations, laboratory methods (dynamics of electrolyte and protein balance, etc.), instrumental (EEG, rheoencephalography, radioisotope) and pathological studies (light, electron microscopy and histochemical analysis). Appearance in the 70s of the XX century. CT allowed us to identify areas of edema as areas of decreased density, mainly due to intercellular accumulation of fluid. Today, the main method for diagnosing and studying cerebral edema is MRI. Modern MRI methods - diffusion, diffusion tensor MRI and MR spectroscopy are actively being introduced into clinical practice, proving their promise.

Key words: edema, brain swelling, CT, MRI, diffusion MRI, diffusion tensor MRI, MR spectroscopy

This paper examines the new capabilities of MRI-DWI, DT MRI and MRS for the qualitative and quantitative assessment of cerebral edema in the light of modern ideas about the pathophysiology of the process and determining the direction of further research.

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Timely and accurate diagnosis of edema is important for choosing treatment tactics “Whoever masters the art of preventing and treating cerebral edema holds the key to the life and death of the patient,” - this is how Academician N.N. defined the significance of the problem of cerebral edema.

Before the advent of imaging techniques—computed tomography (CT) and magnetic resonance imaging (MRI)—the presence of cerebral edema was postulated. The methods of his research were: assessment of clinical manifestations, laboratory methods (dynamics of electrolyte and protein balance, etc.), instrumental (EEG, rheoencephalography, radioisotope) and pathological studies (light, electron microscopy and histochemical analysis). The latter were the most reliable. Appearance in the 70s of the XX century. CT

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Today, the main method for diagnosing and studying cerebral edema is MRI. Modern MRI methods - diffusion imaging (DWI), diffusion tensor imaging (DT MRI) and MR spectroscopy (MRS) are actively being introduced into clinical practice, showing their promise.

The purpose of this work is to consider the new capabilities of MRI - DWI, DT MRI and MRS for the qualitative and quantitative assessment of cerebral edema in the light of modern ideas about the pathophysiology of the process and to determine directions for further research.

Material and methods

Using a unified method, 85 patients were examined (men - 39, women - 46, aged from 18 to 59 years, average age -36.6 years) with various brain pathologies: intracerebral tumors (gliomas, degree of anaplasia II - IV) - 28, the meningitis spaces of the brain can vary, so different types of cerebral edema are possible. The modern classification of cerebral edema is based on the work of I.Clatzo, according to which, depending on the mechanisms of development, two main types of edema were first identified: cytotoxic and vasogenic. Some authors distinguish edema in glial cells and axons (axonal swelling, which often occurs with diffuse axonal injuries) and intermyelin edema in myelin sheaths. Swelling of myelin fibers is often observed in the acute stage of multiple sclerosis, with various types of toxic or metabolic leukoencephalopathies. Orrison divides cerebral edema into five types: vasogenic, cytotoxic, hydrostatic, hypoosmotic and interstitial. However, the views of modern authors on the nature of edema do not coincide in everything. Some provisions are controversial. Below we consider three types of edemas: cytotoxic edema with its subtypes, vasogenic and interstitial, as the most common in neurosurgical practice and the most significant for the choice of treatment tactics.

Vasogenic edema develops against the background of a local increase in cerebrovascular permeability for plasma elements, which leads to an increase in the content of extracellular fluid, with its predominant accumulation in the white matter. Vasogenic edema is most often observed around brain tumors, intracerebral hematomas, hemorrhagic strokes, abscesses, contusion lesions , hypertensive leukoencephalopathy and reversible posterior leukoencephalopathy syndrome , as a consequence of brain hyperperfusion after endarterectomy and some other interventions.

The studies were carried out on a GE tomograph Signa 1.5 T using standard pulse sequences (T 1-, T2-VI, T 2- Flair). For neoplasms, intravenous administration of a contrast agent was used (Magnevist at the rate of 0.2 ml per kg of weight). Non-enhanced areas of tissue surrounding the contrast-accumulating tumor were considered as an area of peritumoral edema.

Standard studies were supplemented by diffusion or diffusion tensor scanning - DT- MRT (TR = 8000 msec, TE = min, 6 directions of diffusion gradients, b = 1000 sec/mm2) and multivoxel spectroscopy (PRESS, TE = 144 m sec). The study results were processed on

a workstation with the construction of parametric diffusion maps based on the average (measured) diffusion coefficient (ADC) and fractional anisotropy (FA) with a quantitative assessment of changes in the edematous areas of the brain and in the conditionally healthy brain substance on the undamaged contralateral side.

Results and discussion

Cerebral edema is a term that refers to an increase in the volume of the brain due to excess fluid in the brain. Traditionally, the terms “edema” and “swelling” of the brain have been used, based on ideas about where fluid accumulation predominantly occurs - outside or inside cells. The term “swelling” was proposed by M. Reichardt in 1905. According to his observations, “the swollen brain is like a sponge and is moist, while the swollen brain is dense, sticky and relatively dry.”

The edema appears hyperintense on T2 and FLAIR tomograms, hypointense on T1 tomograms with contrast, and the diffusion map shows heterogeneous areas of increased diffusion.

In patients with tumors (meningiomas, metastases and malignant gliomas), perifocal areas of vasogenic edema of varying degrees of severity (prevalence) were diagnosed. Peritumoral vasogenic edema was determined on standard MR tomograms by a homogeneous signal change: on T2-WI (a) and FLAIR (b) - hyperintense, T1-WI (c) - hypointense. Vasogenic edema is associated with damage to the blood-brain barrier (BBB), which leads to pathological penetration of proteins, electrolytes and water molecules into the intercellular space with its subsequent expansion, occurring mainly in the white matter. On the ICD diffusion map, vasogenic edema around the tumor is characterized by an increase in the average diffusion coefficient relative to the contralateral side.

static lesion, where early and extensive vasogenic edema is observed. More specific information about the status of perifocal tumor edema to assess the integrity of white matter tracts in the immediate vicinity of the brain tumor can be provided by the use of anisotropy maps obtained from DT-MRI and the use of tractography. This task requires additional studies of white matter tracts and is considered promising for assessing glial tumor growth.

In gliomas, the peritumoral area of signal intensity changes reflect the presence of vasogenic edema and tumor infiltration, spreading along intact perivascular spaces and white matter tracts. It is difficult to differentiate areas of tumor infiltration against the background of peritumoral edema on MRI

acetylaspartate (CAL) peak, an increase and fusion of choline and creatine peaks, and an increase in myoinositol . An increase in water content in the edematous zone leads to an increase in the noise level during the study.

In brain tumors (both primary and metastatic), the development of edema is associated, first of all, with the expression of oncogenes by tumor cells - “angiogenins”, vascular endothelial growth factor. Once in the brain tissue surrounding the tumor, angiogenins

cause pathological angiogenesis and disruption of microvascular permeability. It is even believed that peritumoral cerebral edema is a sanogenetic reaction of the body aimed at reducing tumor-induced pathological angiogenesis. Apparently, these processes are more pronounced with metatask, since infiltrating tumor cells are often difficult to identify even by histological methods, the resolution of which is much higher than that of MRI. Vasogenic edema is reversible because it is associated with reactive changes in cells rather than permanent cell damage.

After surgery, there is a decrease in perifocal vasogenic edema and the appearance of an area of cytotoxic edema in the posterior temporal region are the result of surgical trauma. The area of cytotoxic edema is characterized by a decreased diffusion coefficient on the diffusion map and increased signal intensity on the diffusion-weighted image.

Vasogenic edema can be combined with cytotoxic edema. Cytotoxic edema is characterized by the penetration of excess fluid into intracellular structures. It is observed in ischemia, traumatic brain injury (TBI), metabolic disorders, demyelination, status epilepticus, in the early stages of degenerative diseases, and encephalitis. Cases of transient focal changes in the corpus callosum characteristic of cytotoxic edema have been described in patients receiving anticonvulsants. The development of cytotoxic edema has been noted in rare cases of rapidly growing malignant tumors of the central nervous system (CNS), for example, with metastases of melanoma.

The leading factor in the occurrence of cytotoxic edema is a violation of energy metabolism. Depletion of oxidative phosphorylation and dysfunction of the sodium-potassium pump leads to electrolyte disturbances, penetration of water into the cells, which leads to their swelling. Anaerobic glycolysis with the formation of intracellular lactate leads to increased cytotoxic edema. One of the most important causes of this type of edema is considered to be an increase in the content of neurotransmitters (glutamate, aspartate), cytokines and free radicals in synaptic and extracellular spaces. Cytotoxic edema leads to necrosis, cell apoptosis (delayed neuronal death), or degenerative changes combined with varying degrees of reactive gliosis. These processes also depend on the level of ATP and the presence of calcium ions, which regulate the production of proteases and lipases.

Most often, a combination of vasogenic and cytotoxic edema was observed in the early postoperative period, when, along with areas of peritumoral vasogenic edema, which is characterized by expansion of the intercellular space and increased diffusion capacity, areas with an abnormal decrease in the diffusion rate can be observed in the peritumoral zone. Causes leading to acute cell swelling include tumor devascularization , direct surgical trauma, retraction and vascular injuries.

In the group of patients with acute cerebrovascular accidents (ACVA), areas of cytotoxic edema did not have specific manifestations on standard MRI. The main method for assessing cytotoxic cerebral edema has become diffusion scanning modes, based on differences in the diffusion properties of water in tissues and diffusion maps. Only such modes made it possible to obtain contrast images of cytotoxic edema. As a result, with cytotoxic edema, a noticeable decrease in the volume of the intercellular space occurs

and, consequently, the average diffusion coefficient and ICD decrease (< 0.5 x 10-3 mm2/s), which is what we observed in cases of stroke.

Reduced diffusion in the area of cytotoxic edema in dynamics goes through a phase of increasing to the normal level (unchanged medulla), and then to the level of diffusion in vasogenic edema [2]. Against the background of post-ischemic cicatricial-atrophic changes with signs of vasogenic edema, identified areas with signs of cytotoxic edema were regarded as repeated microstrokes. An abnormal decrease in the diffusion rate can also be observed in a number of pathological intracranial processes. Signs of cytotoxic edema have been identified, for example, in carbon monoxide poisoning, status epilepticus, and brain death.

The exact mechanisms underlying this decrease in ICD are not fully understood. The most popular explanation is that extracellular water moves into the intracellular space. However, the observed decrease in ICD (by 40% or lower) cannot be a reflection of only an increase in intracellular fluid, even if we assume that all extracellular fluid will move into the cell [8]. The observed decrease in the diffusion capacity of water molecules in the intracellular space can be explained by the limited volume of diffusion movement, a decrease in metabolic processes in the cell, and an increase in the viscosity of the cytoplasm due to swelling of organelles [37].

In the group of patients with occlusive hydrocephalus, interstitial edema is observed, which is a pure model of the release of water into the interstitial spaces under the influence of mechanical pressure forces. Edema is visualized in the periventricular white matter of the lateral ventricles, and is not detected around the third and fourth ventricles. This type of edema can occur in patients with TBI and tumors when the outflow of cerebrospinal fluid is impaired. On the diffusion maps, areas of interstitial edema were characterized by a high diffusion capacity of water (B = 1.7 ± 0.09 x x 10-3 mm2/s) and noticeably reduced anisotropy, especially in the area of the posterior and anterior horns of the lateral ventricles. In the periventricular zone, an increase in anisotropy was noted compared to normal values. After installing a shunt or eliminating the cause of the occlusion, interstitial edema regresses.

An attempt was made to quantify the different types of edema by measuring diffusion and anisotropy coefficients (Fig. 9). The vertical dotted line corresponds to the lower limit of the average diffusion coefficient (B) for unchanged brain matter. The horizontal dotted line corresponds to an anisotropy level of 0.2, which is the lower limit of anisotropy in white matter. The upper left corner of the graph is occupied by values corresponding to areas of acute cytotoxic edema. (<0.6 x 10-3 mm2/s). The central part of the diagram is occupied by the values (B) of vasogenic and interstitial edema. The diffusion coefficient values for areas of vasogenic edema with tumor infiltration were grouped in the area of 1.22 ± 0.15 x 10-3 mm2/s, vasogenic and interstitial - 1.45 ± 0.15 x 10-3 mm2/s. The values measured for chronic post-ischemic changes are grouped on the right side of the diagram. In the acute stage of cytotoxic edema, anisotropy values remained unchanged compared to intact white matter; in the chronic stage they were reduced (<0.2 x 10-3 mm2/s).

We believe that the discussed quantitative neuroimaging methods will allow monitoring the effectiveness of edema treatment using new groups of antiedematous drugs, which will require additional research.

Conclusion

MRI, especially diffusion techniques, are the mainstay in visualizing and assessing cerebral edema and its extent in the brain. A comprehensive MRI examination (DT MRI, MRS, tractography) allows us to identify changes typical for various types of edema, both extracellular and intracellular fluid accumulation and provides additional information about the nature of the edema.

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