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**NEUROIMAGING MONITORING FOR EVALUATING THE
EFFECTIVENESS OF LYMPHOTROPIC
THERAPY IN STROKE**

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Abstract

The objects of the study were 65 patients with stroke, whose age ranged from 32 to 65 years. The study included patients with hemorrhagic (n=32) and ischemic (n=33) forms of stroke. The most important criterion for evaluating the effectiveness of the ongoing lymphotropic decongestant in combination with standard therapy was the timing of recovery of the level of consciousness, which was recorded according to GCS and according to MSCT data, which allows dynamic and objective control of cerebral edema.

Key words: Glasgow scale, cerebral edema, lymphotropic therapy.

Relevance

Modern methods of neuroimaging play an important role in the rapid selection of the correct tactics and evaluation of the treatment of cerebral edema in acute cerebrovascular accidents (ACV). The enduring interest in this issue of neurosurgeons, resuscitators, neurologists and neuroradiologists is dictated by the

great scientific and practical importance of cerebral edema for the clinic [11,13,15]. Effective intensive care in neurocritical care is based on dynamic monitoring of vital functions, the clinical neurological picture of stroke, and a set of instrumental indicators of neuromonitoring.

Adverse consequences and complications in stroke are associated with secondary damage and the development of neuroinflammation [4,10,11] and cerebral edema. The development of cerebral edema plays a central role in the development of secondary injury after stroke and is closely associated with neurological outcomes and remains the most significant predictor of outcome in acute cerebrovascular accidents [1,8,12,19].

In the hypothesis put forward in 2012, it was noted that there is a lymphatic system inside the skull, as another component of the intracranial content. This system is called glymphatic, and its main function is determined, which consists in the elimination of brain waste products and toxic substances [9]. The anatomical structures of this system are the paravascular (parietal) Virchow–Robin spaces and the perivascular (inside) spaces through which the interstitial fluid is cleared into the cerebrospinal fluid. A special role in fluid filtration is played by aquaporin 4 channels located on astrocytic legs. There are works indicating that glymphatic outflow is disturbed in patients with severe traumatic brain injury, ischemic stroke, subarachnoid hemorrhage [3,5,6,7,16,18].

The discovery of the glymphatic system (GS) has expanded our understanding of the physiology of the brain in normal and pathological conditions, which opens up new prospects for further research on the treatment of cerebral edema in STBI, and, accordingly, new methods of potential impact on pathological processes in the brain are emerging [3,6,7]. At present, the molecular mechanisms of the development of

cerebral edema (CSE) are being actively studied and the search for targets for targeted therapy in strokes is underway [2,5,14,15,17].

To date, there is no drug with a sufficient evidence base and a convincing effect in the treatment of post-traumatic cerebral edema. Therefore, it is necessary to search for new methods for effective direct influence on the pathogenetic links of BT.

The purpose of our study

To determine the significance of neuroimaging monitoring for assessing the

Materials and methods of research

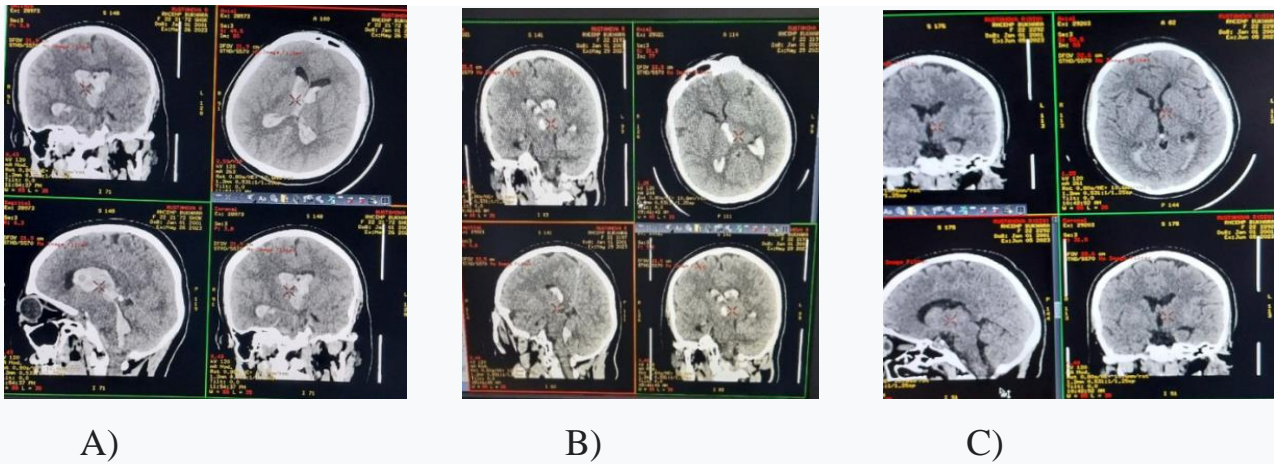
The study was carried out in the Department of Neuro-Resuscitation of the Bukhara branch of the RNCEM. The objects of the study were 65 patients with acute disorders of cerebral circulation, whose age ranged from 32 to 65 years (mean age was 56.3 ± 3 years), in whom clinical and laboratory data were studied. There were 38 men (58.4%), women - 27 (41.6%). The study included patients with hemorrhagic (n=32) and ischemic (n=33) forms of stroke. Primary diagnosis was carried out on the basis of clinical and neurological data and the results of multispiral computed tomography (MSCT). the average score upon admission to the hospital was 9.3 ± 2.1 . According to MSCT data of patients with hemorrhagic stroke, hemispheric hematomas were 51 (82.3%), stem 6 (9.7%), ventricular 3 (4.8%) and subarachnoid 2 (3.2%). Ischemic foci were diagnosed in the basin: middle cerebral artery 48 (77.4%), anterior cerebral artery 3 (4.8%), posterior cerebral artery 4 (6.5%), and vertebrobasilar basin 7 (11.3%).

For the purpose of decongestant therapy, all patients were administered lymphotropically: lidocaine 2% -1 ml, dexamethasone 4 mg, 10% glucose solution 3 ml. in one syringe submastoidally for 5 days, along with conservative treatment, including: antibacterial, decongestant, membrane stabilizing, hemorheological, cerebroprotective and symptomatic therapy. If necessary (coma and if there were signs of dislocation of the median structures on MSCT), the patients were transferred

to mechanical ventilation. 14 patients, which accounted for 22.6% (with hemorrhagic (n=9 (64.2%)) and ischemic (n=5 (35.8%)) patients, were on prolonged mechanical ventilation. Modes and parameters of ventilation were selected individually according to the severity of the somatic status and anthropometric parameters. Clinical (systemic indicators of hemodynamics and respiration, neurological status), instrumental (ECG, chest X-ray, MSCT examination of the brain) and laboratory data (leukoformula, index of the ratio of neutrophils to lymphocytes - SNL) were analyzed. Comparison of clinical and laboratory parameters was carried out in three stages: the first stage - on admission, the second stage - the 3rd day, the third stage - the 7th day of intensive therapy.

Results and their discussion

When analyzing the obtained data on the neurological status, it was found that upon admission in all examined patients, the level of consciousness according to the GCS was from 8 to 12 points (8 points n=12, 9 points n=15, 10 points n=13, 11 points n=8, 12 points n=14). At the 2nd stage of the study, there was an increase in the number of patients with a more profound impairment of neurostatus (GCS: 8 points n=14, 9 points n=18, 10 points n=14, 11 points n=10, 12 points n=6). This worsening of the neurological status was most likely associated with an increase in cerebral edema. These changes were confirmed by the method of neuroimaging - MSCT, where the increase in cerebral edema was evidenced by the smoothness of the furrows, narrowing of the basal cisterns and ventricles of the brain. These patients had clinical evidence of progression of cerebral edema such as arterial hypertension, susceptibility to bradycardia, tachypnea and restlessness. In this regard, 14 patients were transferred to mechanical ventilation.

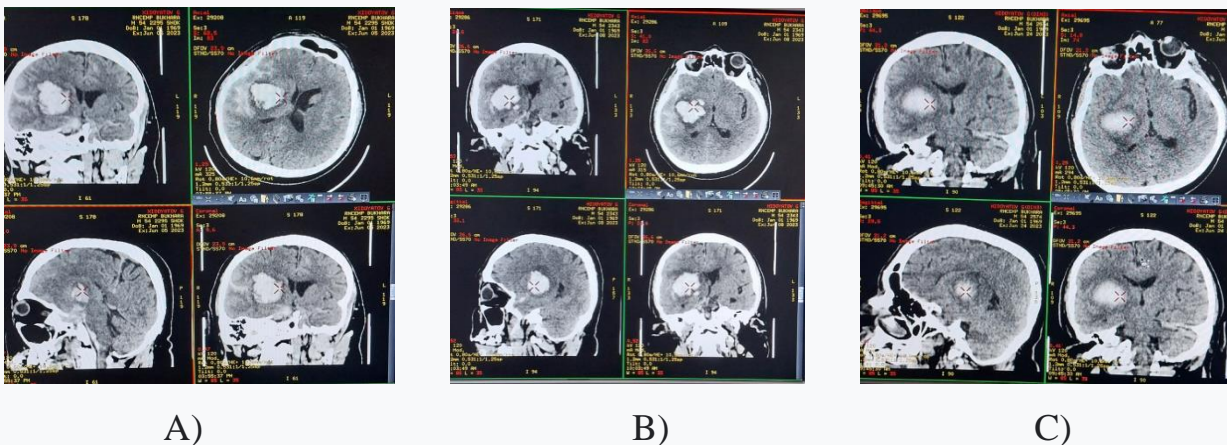


A)

B)

C)

Fig.1. MSCT of patient R. with hemorrhagic stroke who received lymphotropic therapy (A - on admission, B - on the 5th day, C - on the 10th day).



A)

B)

C)

Fig.2. MSCT of patient G. with hemorrhagic stroke who did not receive lymphotropic therapy (A - on admission, B - on the 5th day, C - on the 10th day).

When conducting MSCT studies in patients who received lymphotropic therapy during treatment, there was a regression of the previous picture of cerebral edema: smoothness of the furrows, the absence of narrowing of the basal cisterns and ventricles of the brain, rapid resorption of the hematoma as early as on the 10th day (Fig. 1). In patients without lymphotropic therapy, the resorption of intracerebral hematoma occurred slowly and at a later date (Fig. 2)

In the dynamics against the background of intensive therapy, 56 patients showed clinical improvement, which was confirmed by the obtained data on the

neurostatus of the 3rd stage of the study (according to GCS: 8 points n=8, 9 points n=10, 10 points n=15, 11 points n= 19, 12 points n=10).

Of all the examined patients, a lethal outcome was observed in 6 (2 patients with ischemic and 4 patients with hemorrhagic stroke).

Conclusion

Lymphotropic decongestant therapy increases the effectiveness of basic treatment, prevents the progression of cerebral edema in patients with stroke. Monitoring using MSCT allows dynamic objective control of cerebral edema.

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