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PATHOPHYSIOLOGICAL BASES OF LYMPHOTROPIC DEEDEDEOUS THERAPY IN COMPREHENSIVE TREATMENT OF PATIENTS WITH HEMORRHAGIC STROKE

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✓ ***Resume***

The effectiveness of lymphotropic decongestant therapy in patients with hemorrhagic stroke was studied. Based on the obtained clinical and laboratory data, the effectiveness of lymphotropic decongestant therapy was revealed, which prevents the progression of cerebral edema in this category of patients.

Key words: cerebral edema, hemorrhagic stroke, lymphotropic therapy.

ПАТОФИЗИОЛОГИЧЕСКИЕ ОСНОВЫ ЛИМФОТРОПНОЙ ПРОТИВООТЕЧНОЙТЕРАПИИ В КОМПЛЕКСНОМ ЛЕЧЕНИЕ БОЛЬНЫХ СГЕМОРРАГИЧЕСКИМ ИНСУЛЬТОМ

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✓ ***Резюме***

Изучена эффективность лимфотропной противоотечной терапиицу пациентов геморрагическим инсультом. На основание полученных клинико лабораторных данных было выявлена эффективность лимфотропной противоотечной терапии, который предотвращает прогрессирования отека мозга в данной категории больных.

Ключевые слова: отек мозга, геморрагический инсульт, лимфотропная терапия.

GEMORRAGIK INSULT BILAN OG'RIGAN BEMORLARNI KOMPLEKS DAVOLASHDA LIMFOTROPTERAPIYANING PATOFIZIOLOGIK ASOSLARI

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✓ ***Rezyume***

Gemorragik insult bilan og'rigan bemorlarda shishga qarshilimfotrop terapiyaning samaradorligi o'rganildi. Olingan klinik va laboratoriya ma'lumotlariga asoslanib, ushbu toifadagi bemorlarda miya shishi rivojlanishining oldini olishda limfotrop terapiyaning samaradorligi aniqlandi.

Kalit so'zlar: miya shishi,gemorragik insult, limfotrop terapiya.

Relevance

Adverse consequences and complications of 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 damage 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].

The hypothesis put forward in 2012 noted the presence of the lymphatic system inside the skull as another component of the intracranial contents. This system is called glymphatic, and its main function is determined to be the elimination of brain waste products and toxic substances [9]. The anatomical structures of this system are the paravascular (peri-wall) Virchow-Robin spaces and the perivascular (intra) spaces through which the interstitial fluid is cleared into the cerebrospinal fluid.Aquaporin 4 channels located on astrocytic stalks play a special role in fluid filtration. There are studies indicating that glymphatic outflow is impaired in patients with severe traumatic brain injury, ischemic stroke, and 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 hemorrhagic stroke, accordingly, new methods are emerging for potentially influencing pathological processes in the brain [3,6, 7]. Currently, the molecular mechanisms of the development of cerebral edema (CE) are being actively studied and targets for targeted therapy for stroke are being sought [2,5,14,15,17].

Modern neuroimaging methods play an important role in quickly choosing the right tactics and assessing the treatment of cerebral edema in acute cerebrovascular accidents (ACVA). 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 [11,13,15].

Effective intensive care in neuroreanimation is based on dynamic monitoring of vital functions, the clinical neurological picture of stroke and a set of instrumental neuromonitoring indicators.

To date, there is no drug with a sufficient evidence base and 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 AMS in hemorrhagic stroke. New understanding of the pathophysiology of how trauma and stroke affect GS function and how this function is decompensated in brain pathologies should lead to the development of new preventive and new therapeutic targets.

Purpose of the study: to evaluate the effectiveness of decongestant lymphotropic therapy for hemorrhagic stroke.

Materials and methods

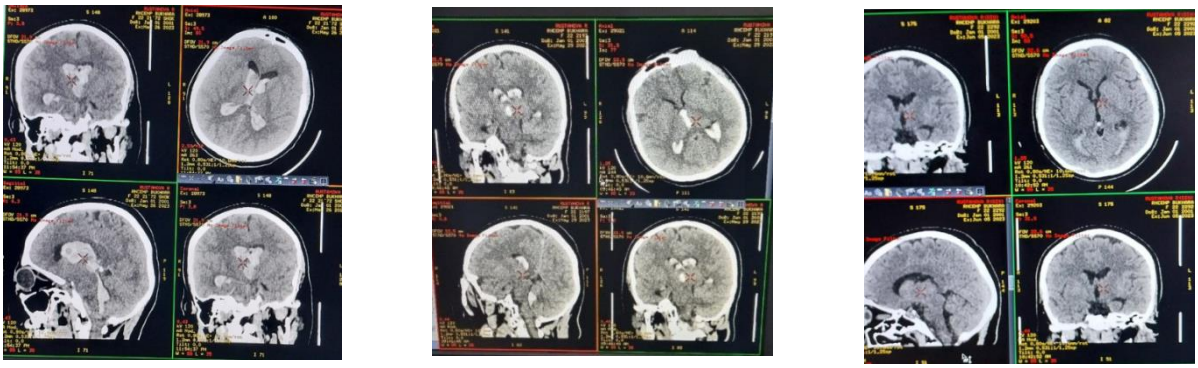
The study was carried out in the neuroreanimation department of the Bukhara branch of the Russian Research Center for Emergency Medicine. The subjects of the study were 65 patients with hemorrhagic stroke. Their age ranged from 32 to 65 years (average age was 56.3±3 years), whose clinical and laboratory data were examined. There were 38 men (58.4%), women - 27 (41.6%). Primary diagnosis was carried out on the basis of clinical and neurological data and the results of multislice computed tomography (MSCT). When assessing neurostatus using the Glasgow Coma Scale (GCS), the average score upon admission to the hospital was 9.3±2,1.According to MSCT data of patients with hemorrhagic stroke, hemispheric hematomas accounted for 51 (82.3%), brainstem 6 (9.7%), ventricular 3 (4.8%) and subarachnoid 2 (3.2%) patients.

All patients in the intensive care complex were administered lymphotropically for the purpose of decongestant therapy: 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 (comatose state and the presence of signs of dislocation of the midline structures on MSCT), the patients were transferred to mechanical ventilation. Ventilation modes and parameters were selected individually based on the severity of the somatic status and anthropometric parameters. Clinical (systemic parameters of hemodynamics and respiration, neurological status), instrumental (ECG, MSCT examination of the brain) and laboratory data (leukoformula, neutrophil-to-lymphocyte ratio index - ISNL) were analyzed. A comparison of clinical and laboratory parameters was carried out in three stages: the first stage - upon admission, the second stage: -3rd day, the third stage - 7th day of intensive therapy.

Results and discussions

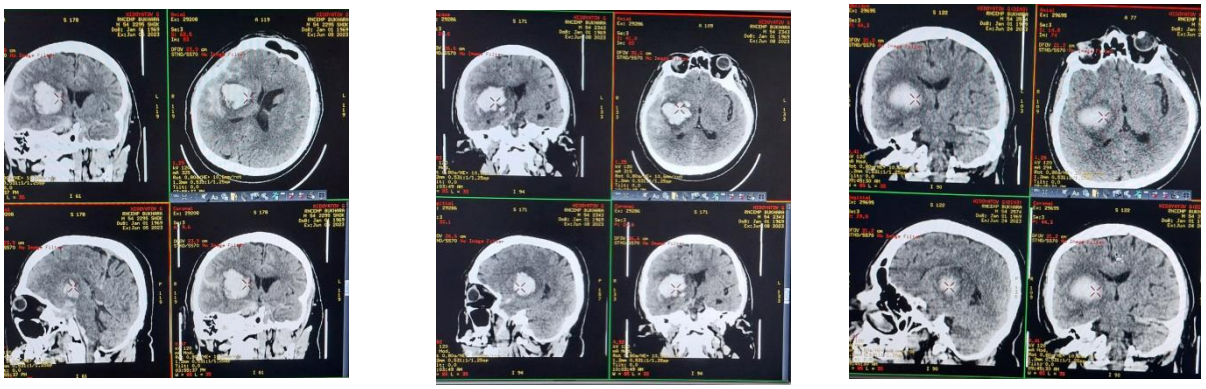
When analyzing the obtained data on the neurological status, it was revealed that upon admission in all examined patients the level of consciousness according to the GCS was from 8 to 12 points (8 points n=12; 9 points n=15,; 10 points n=13; 11 points n=8; 12 points n=14).

At the 2nd stage of the study, there was an increase in the number of patients with a more profound impairment of neurostatus (according to the GCS: 8 points n=14; 9 points n=18; 10 points n=14; 11 points n=10; 12 points n=6). This deterioration in neurological status was most likely associated with an increase in cerebral edema. These changes were confirmed by neuroimaging - MSCT, where the increase in cerebral edema was evidenced by smoothing of the furrows, narrowing of the basal cisterns and ventricles of the brain. These patients developed clinical evidence of progression of cerebral edema such as arterial hypertension, tendency to bradycardia, tachypnea and anxiety. In this regard, 14 patients were transferred to mechanical ventilation.



A) B) C)

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



A) B) C)

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

Over time, against the background of intensive therapy, 59 patients showed clinical improvement, which was confirmed by the data obtained from the neurostatus of the 3rd stage of the study (according to the GCS: 8 points n=8; 9 points n=10; 10 points n=15; 11 points n= 19,; 12 points n=10).

When conducting an MSCT study over time, regression of the previous pattern of cerebral edema was noted: smoothing of the sulci, narrowing of the basal cisterns and ventricles of the brain were absent, the SINL was below 3,1 ± 1,2.

Of all the examined patients, death was observed in 6 patients with hemorrhagic stroke, while the AISI was more than 4,2 ± 1,1.

Conclusion

Decongestant lymphotropic therapy allows impact on the pathophysiological mechanisms of cerebral edema, increases the effectiveness of intensive basic treatment and prevents the progression of cerebral edema in patients with hemorrhagic stroke. Monitoring using MSCT allows for dynamic objective monitoring of cerebral edema.

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