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Lymphotropic Deunedeous Therapy In Complex Treatment Of Patients With Hemorrhagic Stroke

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Summary. The effectiveness of lymphotropic decongestant therapy in patients with hemorrhagic stroke was studied. Based on the obtained clinical and laboratory data, comparatively the main and control groups, 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.

Relevance. Stroke is a worldwide public health problem and one of the leading causes of acquired disability worldwide. Every year, 15 million people suffer from a stroke. Of these, 5 million die and 5 million remain without permanent disability, placing a heavy burden on individuals, families and society. Mortality varies significantly depending on the degree of socioeconomic development: about 85% occurs in underdeveloped or developing countries and one third of cases reach the economically active part of the population [12] .

Despite the fact that hemorrhagic stroke (HI) is almost five times less common than IS, it is characterized by a higher level of disability (about 75% of the number of cases) and mortality (from 40 to 50%). HI typically affects a large percentage of the working age population. About 20% of patients who have suffered a stroke require constant care due to severe motor and cognitive impairment and social maladjustment. After 50-55 years, the incidence of stroke increases by 1.8-2 times in each subsequent decade and reaches a peak by 70 years. In connection with the above, this disease is of a high social nature, which puts it among the priority problems in economically developed countries with a high standard of living [1].

Secondary injuries are a cascade of biochemical inflammatory and immunological stress reactions that develop in response to primary injury. Due to the increase in secondary damage reactions, irreversible ischemic damage and death of cells located in the immediate vicinity of the primary damage site ultimately occur, and with further development, undamaged brain cells are involved in the pathological process [10, 14,15,16,18] .

Modern directions in the search for means of pharmacological correction of cerebral edema (CED). Despite the fairly extensive arsenal of drugs available for the pharmacotherapy of AMS, the search for new effective compounds that can inhibit the development of this formidable pathological condition continues. To this end, various biochemical factors involved in the development of AGM are being studied, water channels and ion transporters, cellular enzymes and other proteins are being studied as potential targets for the effects of drugs [2] .

One factor that can increase swelling is intraventricular hemorrhage (IVH) because there is an imbalance in the absorption of cerebrospinal fluid. This often leads to acute hydrocephalus, which in turn increases edema by increasing ICP, reduces cerebral perfusion and causes ventricular wall distension, white matter damage and inflammation [21].

Hemorrhagic stroke includes intracerebral hemorrhage (ICH), in which bleeding

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occurs within the brain parenchyma, and subarachnoid hemorrhage (SAH), in which bleeding occurs in the subarachnoid space. Non-traumatic ICH is caused by high blood pressure in approximately 50% of cases and is associated with the time of year, occurring most often in winter and more often in men than women. The size of the IUD is usually determined at the onset of bleeding, and in very rare cases bleeding continues [5].

Neurological symptoms usually appear within 48 hours, and the severity of hemorrhagic stroke depends on whether the hematoma has enlarged and the size of peripheral cerebral edema [8,21].

Perihematomal edema (PHE) occurs when there is an increase in water content in the brain tissue adjacent to the intraparenchymal hematoma. The development of PHO is considered a quantitative marker of AGM and is associated with thrombin activation, inflammatory immune response, blood-brain barrier (BBB) dysfunction, and hemoglobin cytotoxicity after ICH. PGO also causes significant mass effect, and rapid growth of PGO can lead to severe intracranial hypertension [25] .

Brain swelling causes a mass effect that puts pressure on the surrounding tissue. This increase in pressure is amplified by the rigid lining of the skull, which sets an upper limit to the volume to which the brain can expand. When the brain swells, it exerts a mechanical force on the inside of the skull, thereby increasing intracranial tissue pressure. When tissue pressure exceeds capillary pressure, the capillary lumens are compressed, initiating a feedforward process in which ischemia of the surrounding lining causes further edema formation and further swelling of the next lining [19] .

In addition to the initial mass of the hematoma, ICH causes disruption of the blood-brain barrier and swelling of parenchymal cells, which leads to cerebral edema And intracranial hypertension affecting the patient's prognosis. Reducing cerebral swelling is an important part of post-ICH care. However, there are limited effective methods treatment to reduce perihematomal cerebral edema and intracranial pressure with ICH [20] .

ICH causes initial (primary) brain damage due to the mass effect caused by the hematoma and physical destruction. Secondary damage occurs as a result of this primary damage, as well as the effect of thrombus-forming factors on the surrounding brain tissue [4] .

Cerebral edema increases progressively in the first 24 hours and increases rapidly 3 days after onset, reaches an initial peak on days 4-5 and slowly increases until 9-14 days, and then decreases. Perihematomal Edema (EED) develops in response to clot retraction and changes in hydrostatic pressure, mass effect, thrombin formation, red blood cell lysis, hemoglobin toxicity, complement activation, plasma protein leakage, and disruption of the blood-brain barrier (BBB). All inflammation, thrombin activation and erythrocyte lysis contribute to the destruction of the BBB, leading to the formation of edema [6,22,26].

Since blood is extremely toxic to brain tissue, hemorrhage itself is a form of focal damage to the central nervous system, which causes the formation of cerebral edema in the tissue lining immediately surrounding the hemorrhage, i.e. in the perihematomal space, a phenomenon called perihematomal edema. Perihematomal edema occurs in three stages: ionic edema, vasogenic edema and delayed vasogenic edema [7, 24] .

The discovery of the glymphatic system opens a new direction in the understanding of brain diseases, which shifts the focus from changes in specific brain structure to the general circulation of fluid in the brain [11,17].

More and more studies highlight the importance of lymphatic function in various neurological diseases. As a widespread clearance system in the brain, the glymphatic system may influence many neurological disorders, providing a new direction for future research into

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disease mechanisms and treatments [9,23] .

Assessing glymphatic function after stroke or traumatic brain injury may indicate that this system maintains brain homeostasis, is involved in immune surveillance, and represents a new therapeutic target in the treatment of stroke [3,27,28] .

Understanding the mechanisms of the glymphatic system in cerebral edema after stroke may provide new targets and treatment options, thereby promoting restoration of neurological function and improving patient prognosis. The development of therapeutic strategies aimed at maintaining the integrity of the brain's lymphatic drainage system may provide an innovative approach in the treatment of stroke.

The purpose of the study is to evaluate the effectiveness of lymphotropic decongestant therapy for cerebral edema in patients with hemorrhagic stroke .

Materials and methods of research : The study included data from patients who were treated in the neurointensive care unit of the Bukhara branch of the Russian Research Center for Emergency Medicine. Materials from 37 patients with HI were studied, at an age which ranged from 37 to 68 years (average age was 58.2±2 years). All patients underwent standard diagnostic methods (assessment of neurological status during a joint examination by a neurologist and neurosurgeon, multislice computed tomography (MSCT), as well as laboratory tests (leukoformula, neutrophil-to-lymphocyte ratio index - ISNL)). Neurostatus was assessed using the Glasgow Coma Scale (GCS), with the average score upon admission to hospital being 9.3±2.1. On MSCT in patients with hemorrhagic stroke, hemispheric hematomas accounted for 29 (78.3%), brainstem 4 (10.8%), ventricular 2 (5.4%) and subarachnoid 2 (5.4%).

Patients with HI were divided into two groups. The first group is the main one, whose patients received lymphotropic decongestant therapy. For the purpose of anti-edematous therapy, submastoid on one side a solution of lidocaine a 2% - 1 ml, dexamethasone a 4 mg, 10% glucose solution 3 ml was introduced. in one syringe (the method was approved at a meeting of the Ethics Committee of the Ministry of Health of the Republic of Uzbekistan, protocol No. 7 of November 9, 2023). Submastoid lymphotropic injections were performed in within 5 days, along with conservative treatment, including: antibacterial, decongestant, membrane stabilizing, hemorheological, cerebroprotective and symptomatic therapy.

The second group was the control group, which received standard therapy. A comparison of clinical and laboratory parameters was carried out in three stages: the first stage - upon admission, the second stage: day 5, the third stage - 10 days of intensive therapy.

Results and their discussions : When analyzing the results of patients in the main group with HI (n=15), the following data were revealed - at the first stage of the study, the level of impairment of consciousness averaged 9.1 points on the Glasgow scale. In blood tests, the number of segmented neutrophils was 56%, and lymphocytes - 16%, while the ISNL indicator was 3.5. At the second stage of the study, the assessment of the level of consciousness was 10.2 points on the GCS. Segmented neutrophils accounted for 60%, lymphocytes 22%, the ISNL index was 2.7. At the third stage of the study, a noticeable increase in all these indicators was observed. There was clearing of consciousness to stupor, the average score of which was 13.1 points. And the indicators of the general blood test were: segmented neutrophils 62%, lymphocytes 25%, and ISNL 2.5 (graph 1).

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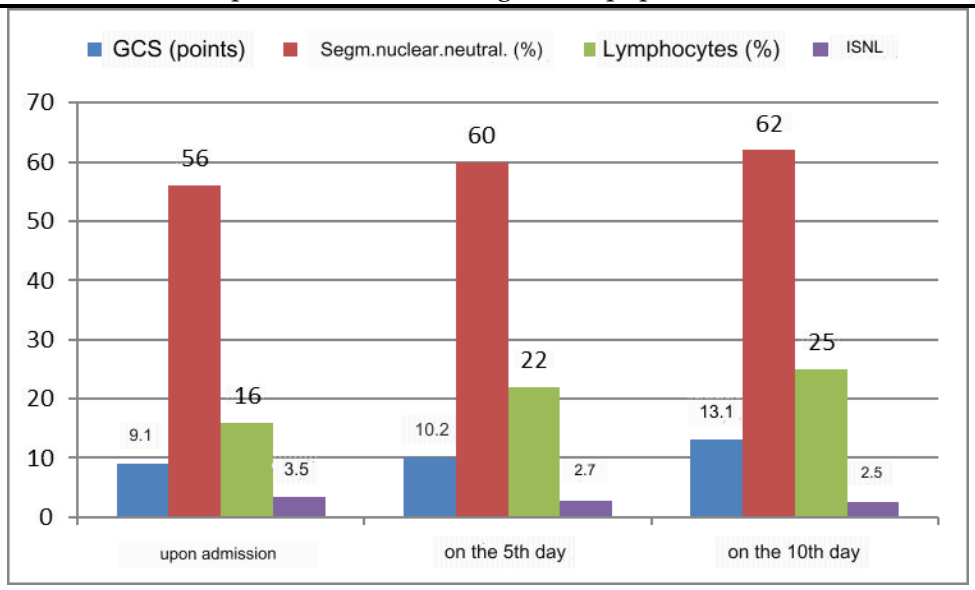
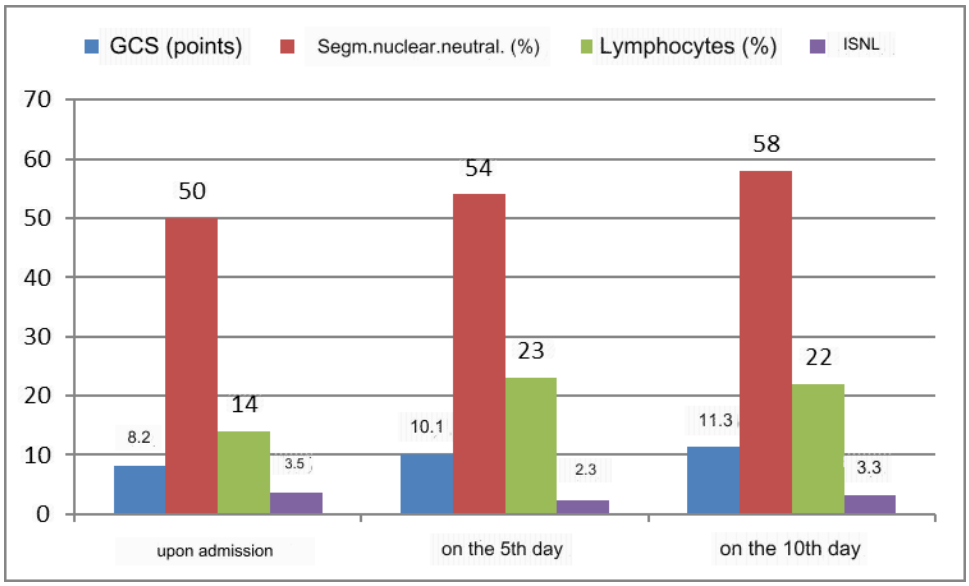


Graph 1. Dynamics of indicators of the main group of patients with HI who underwent lymphotropic decongestant therapy

Processing of the results obtained from the control group of patients with HI (n=22) showed the following data: at the first stage of the study, the level of impairment of consciousness averaged 8.2 points on the Glasgow scale. In laboratory blood tests, the number of segmented neutrophils was 50%, and lymphocytes - 14%, while the ISNL indicator was 3.6. At the second stage of the study, the assessment of the level of consciousness was 10.1 points on the GCS. Segmented neutrophils accounted for 54%, and lymphocytes 23%, respectively, the ISNL indicator was 2.3. At the third stage of the study, the level of consciousness according to the GCS was 11.3 points. The general blood test indicators were: segmented neutrophils 58%, lymphocytes 22%, and ISNL 3.3 (graph 2).



Graph 2. Dynamics of indicators in the control group of patients with HI.

Dynamic neuroimaging control (MSCT of the brain) made it possible to obtain information about the state of the hemorrhage, the bruise zone, and the perifocal zone. Analysis of the obtained data from the MSCT study revealed that all patients in both groups on the primary MSCT had parenchymal as well as hemorrhages and cerebral ventricles. In addition,

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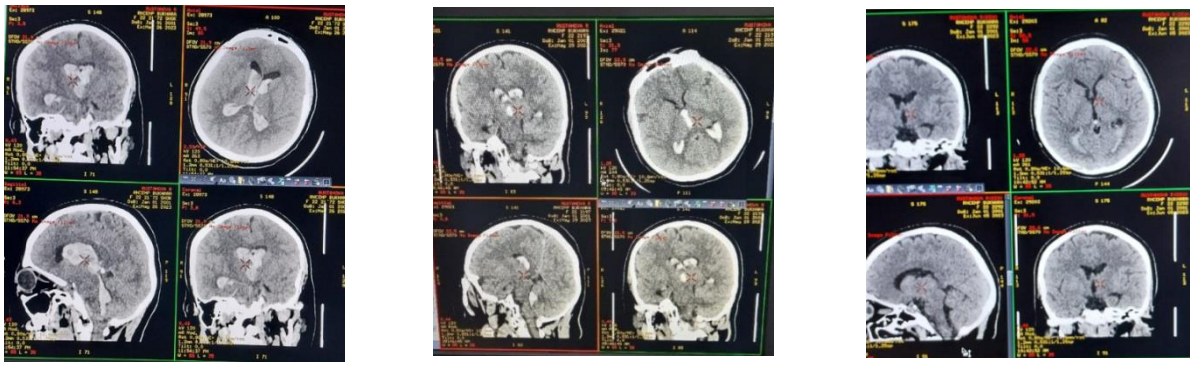
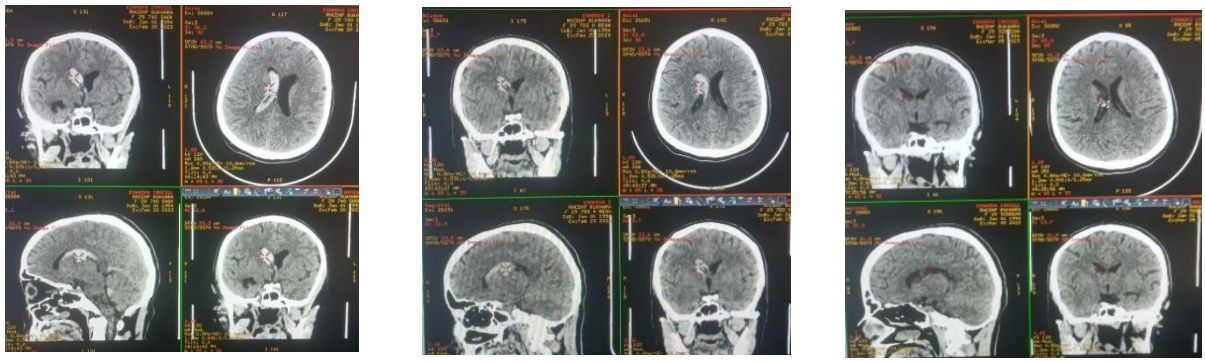
congestion of the sulci of the cerebral cortex and narrowing of the basal cistern were signs of intracranial hypertension with symptoms of impaired consciousness. In patients of the main group, against the background of lymphotropic decongestant therapy on the second and third MSCT, regression of cerebral edema was evidenced by the appearance of signs of improvement in the architectonics of the cerebral cortex, restoration of the normal sizes of the basal cisterns and ventricles of the brain (Figure 1). All these manifestations corresponded to positive neurological changes .



A) B) C)

Figure 1. MSCT of the patient. with hemorrhagic stroke of the main group (A – upon admission, B – on the 5th day, C – on the 10th day).

And in patients in the control group, the above changes on MSCT of the brain appeared noticeably slowly compared to the main group (Figure 2).



A) B) C)

Figure 2. MSCT scan of a patient with hemorrhagic stroke in the control group (A – upon admission, B – on the 5th day, C – on the 10th day).

Dynamic changes in the neurological status and computed tomography in both groups corresponded to changes in laboratory parameters that reflected changes in the leukocyte formula (graph 1, 2). Rapid positive dynamics were observed in the main group, which received lymphotropic decongestant therapy.

Conclusion. Lymphotropic decongestant therapy increases effectiveness in a complex of intensive basic treatment , prevents progression of cerebral edema in patients with HI . Neuromonitoring using MSCT and comparison of clinical data allows dynamic objective monitoring of cerebral edema.

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