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Cerebrocardial syndrome, ways of full correction.

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Abstract: The purpose of the research: to determine the clinical effectiveness of complex therapy with cholinealfoscerate drugs as a supplement to standard therapy in patients in the acute period of ischemic stroke of hemisphere localization. Materials and methods: Sixty patients with acute cerebral stroke were examined, with an average age of 56+_ to 4.38. The main methods of the study were the collection of complaints and history, analysis of medical records, assessment of somatic and neurological status, paraclinical research methods (MRI), study of cognitive functions. The examination of patients was carried out during the acute period of stroke and repeated after 3 months. The results have been processed statistically. Results.The study revealed that the use of drugs of the group cholinealphoszerate in the acute phase of stroke and in the next 2 months has a beneficial effect not only on the restoration of motor and sensitive neurological deficits, improved cardiac activity, but also reduces the incidence of early post-stroke dementia (high confidence) and reduces the severity of clinical manifestations of lung and moderate cognitive disorders. Conclusions. For the comprehensive correction of the manifestations of ACCD and the prevention of post-stroke dementia, as well as reducing the progression of cognitive disorders, stroke patients should recommend the use of drugs chodinoalfoscerat (Cerepro) (in a dose of 2000mg/day intravenous drip for 5 days, then 1000mg/day intravenous5 days, then 1 restorative tablet 2 times a day (800mg/day) - 2 months) from the first days of stroke and in the early period.

Keywords: cerebrocardial syndrome, cognitive function, chominoalphoscerate, Cerepro.

Introduction. There is a reverse effect of chronic and even more acute pathology of cerebral vessels on the occurrence and exacerbation of coronary blood flow disorders. The changes described are typical for cerebrocardial syndrome.

The concept of cerebrocardial syndrome was introduced into the medical terminology of F. Wuhrmann in the early 1950s. Initially, it was used to describe changes in myocardium (according to the Electrocardiography) during the acute period of acute cerebral circulatory disorders (ACCD).

Cerebrocardial syndrome is a variant of the violation of cervical regulation, indicating the interdependence of the brain and the system of hemodynamics[4]. 70% of patients with the localization of the ACCD hearth in the vertebral-basilar basin have clinically significant cardiac disorders[1,5,8].

There are differences in clinical manifestations of cerebrocardial syndrome not only depending on the location of the source of ischemia in the hemispheres or in the area of stem structures of the brain, but also from the lateralization of this hearth. The right- and left-sided

cerebrocardial effects are highlighted. In researches of R.D. Lane and co-study. (1992) these differences were confirmed: patients with subdominant hemisphere lesion were defined by higher numbers and variability of blood pressure (VBP) and heart rate(HR), arrhythmias were more common[6]. Under the influence of cerebral ischemic hearth may disrupt the work of centers of the autonomic nervous system. In the development of cardiac disorders it is advisable to take into account the factor of lateralization of function in the hemispheres of the brain: the right hemisphere is generally associated with sympathetic activity, while the left - with parasympathetic. Experimentally it was established that the inactivation of the left hemisphere observed an increase in the heart rate, at the same time when the right hemisphere is turned off, the rhythm is reduced. In addition, the probability of an aritmog variant of the Cerebrovascular syndrome is determined by the localization of cerebral damage, which reflects the peculiarity of cerebral regulation of the cardirvascular system. Experimentally, it was found that in the case of the defeat of the right parts of the brain more often there is supraventricular rhythm disturbances, and in lesions of the left - mainly ventricular. In clinical studies, patients with the localization of brain infarction in the right hemisphere showed a more pronounced increase in the values and variability of blood pressure than in left-sided heart attacks. They are more likely to have arrhythmias and lengthening of the interval of the QT, as well as an increase in the concentration of norepinephrine in the blood plasma. Also, the direct mechanical impact of the necrosis zone on the brain can lead to swelling of the brain, increased intracranial pressure, compression of the brain stem, impaired Likvor dynamics. As a result of these processes, there may be a violation of the regulation of cardiac activity and destabilization of systemic hemodynamics.

Due to the complex interdependence of coronary and cerebral blood supply systems, the pathology that occurs in one of them inevitably entails imbalance and possible decompensation in another system. This dictates the tactics of the earliest detection of cerebral pathology in patients of cardiac profile, as well as thorough examination and preventive cardioprotective therapy in patients with ACCD.

During the ACCD period, along with the pathophysiological interaction of the brain and myocardial at the systemic level, the main and crucial for ACCD are the processes occurring in the brain at the cytohistological level.

Considering in detail the brain stroke from the point of view of the pathophysiology of cellular and neurohumor systems, it can be described as an energy and metabolic catastrophe provoked by hypoxia.

Energy and metabolic disorders are initially divided over time. Initially, the processes of energy deficit are as active as possible, then the existing changes are supplemented by glutamate ecsytotoxicity, imbalance of intracellular calcium, lactatecidosis. There is a direct correlation between the severity of these pathological processes and the duration and depth of insufficiency of local cerebral blood flow[1, 7]. The nuclear zone of ischemia, around which a potentially recoverable zone of ischemic penumbra is formed as much and irreversibly. It is to restore the functions of neurons in the area of penumbra and directed all the efforts of neurologists in the acute period of ACCD.

During the development of ischemia, the brain tissue is locally disrupted by the utilization of glucose and oxygen by the neurons of the affected area. The processes of aerobic oxidation stop. Glucose cleavage begins to flow along the anaerobic pathway, which leads to the development of acidosis. The latter contributes to the destabilization of the enzyme systems that control the transmembrane transport of the ions of the K+, Na+, Ca2+. This contributes

to the excess accumulation of intracellular calcium with the development of cytotoxic swelling of neurons caused by excessive osmotic absorption by their water. All these processes entail a sharp reduction in the synthesis of Adenosine triphosphate and ADF molecules, the destruction of the phospholipid membranes of neurons and their organelles. The massive death of phospholipid formations of nerve cells exacerbates the massiveness of the lipid peroxidation process (POL) and contributes to the emergence of even more free radicals. Thus, there is an irreversible loss of neurons[1, 2].

Together with processes based on energy deficit and metabolic imbalance, a secondary reaction of local inflammation develops. Genes encoding apoptosis processes are activated. All this accelerates the processes of death of nerve cells and contributes to the development of secondary pathophysiological processes at systemic (including cardiac) and body levels.

Trying to resist a cascade of destructive processes, the antioxidant system depletes its capabilities, finally formalized the universal process of neuron death - oxidative stress.

All the efforts of modern neuroscience and related disciplines are aimed at eliminating the processes described above. At the same time, any of the stages of pathological process of damage and death of neurons can become a potential target for exposure.

The main efforts of neurologists should be focused on:

- timely and most active reperfusion;

- correction of the main indicators of life

- level of systolic and diastolic blood pressure, heart rate, regulation of water-electrolyte balance, respiratory rate, body temperature, diuresis, glucose, cholesterol, etc.;

- prevention and control of complications associated with underlying disease (ACCD): infectious, thromboembolic, aspirational, hypostatic, etc.;

- early rehabilitation;

- neuroprotective therapy.

Modern domestic cardioneurology gives special importance to neuroprotective therapy. Thanks to the efforts of specialists in the field of clinical and experimental pharmacology in recent years, it has been possible to obtain medicines capable of influencing each stage of the pathobiochemical ischemic cascade. The therapy, based on the principles of evidence-based medicine, is secondary neuroprotective therapy, which is currently used in practical health care to prevent the development of delayed effects of brain ischemia. This can be done by suppressing excess nitrogen oxide synthesis, inhibition of oxidative stress and cytokine imbalance, restriction and reduction of local inflammation, normalization of microcirculation, restoration of normal function of HMB (hematoencephalic barrier), inhibition of apoptosis, etc. as well as the risk of repeated ACCD. Secondary neuroprotection includes both antioxidant therapy, the use of pro-inflammatory cytokines antagonists and cell adhesive molecules, receptor regulators (gangliosids), and neurotrophic, neuroimmunomodulation drugs [7].

The leading role in the process of alteration and death of cells under the influence of ischemia belongs to oxidation stress and activation of POL (peroxide oxidation of lipids). This encourages greater use of antioxidants and their precursors in patients with ACCD and acute

coronary syndrome (ACS) or acute myocardial infarction (AMI). Also, understandable and shown the use of drugs of this group in patients with ACCD to prevent the development of cerebrocardial syndrome and in patients with ACS/AMI to prevent and advance protection of neurons from the effects of cardiocerebral syndrome.

Chronic cardiac diseases (AH (arterial hypertension), IHD (ischemic heart disease) etc.) stimulate the narrowing of the intracranial arteries, the violation of endothelial functions with increased stiffness of the vascular wall, which subsequently causes chronic cerebral blood flow failure with diffuse or focal changes of the brain substance. Basic cardiac disorders provoke the manifestation of clinical signs of chronic cerebrovascular imbalance, creating a favorable background for the development of acute vascular catastrophe.

The selection of cardioembolic and hemodynamic subtypes of cerebral disorders emphasizes the commonality and interdependence of cardiac and cardiac blood supply pools.

The purpose of the study: to determine the clinical effectiveness of complex therapy with cholinealfoscerate drugs as a supplement to standard therapy in patients in the acute period of stroke of the hemisphere localization.

Materials and methods: Sixty patients with acute cerebral stroke were examined, with an average age of 56+ _ 4.38.

The main methods of the study were: collection of complaints and history, analysis of medical records, assessment of somatic and neurological status, paraclinical research methods (MRI), study of cognitive functions. The examination of patients was carried out during the acute period of stroke and repeated after 3 months. The results are processed statistically.

Patients under observation received a standard basic treatment. Basic therapy, conducted in all patients, fully complies with national standards (the protocol of management of patients "Stroke") and standards of medical care for stroke patients. All patients were divided into two groups: the first (main) of 30 patients - the treatment included the drug group cholinealphoscerate (cereprora); the second (control) of 30 patients - took only basic therapy (cerepro was not taken). Patients of the main group were injected with cerepro2000mg/day intravenousdrip for 5 days, then 1000mg/day intravenously 5 days, then 1 tablet twice a day (800mg/day) - 2 months. Both groups had no statistically significant differences in gender, age, severity of the condition and concomitant pathology.

All patients had a concomitant cardiac pathology (confirmed by instrumental, laboratory studies and cardiologist's conclusion). All patients suffered from 3rd degree hypertension, risk 4; IBH, atherosclerotic cardiosclerosis; myocardial hypertrophy of the left ventricle. The prevailing number of strokes is atherothrombotic genesis.

Results. Cardiac symptoms of cerebrocardial syndrome. Among the patients with ACCD, the groups studied, there were various disorders of rhythm and conductivity of the heart. The presence of ischemic and hemorrhagic strokes indicated a tendency to sinus bradycardia (55.3%). Sinus tachycardia was observed in 17.5%, sinus arrhythmia in 14.4%, various kinds of extrasystole in 9.5% of cases.

In the acute period of stroke, paroxysms of atrial fibrillation, ventricular and supraventricularextrasystole were more commonly reported. The incidence of heart arrhythmias in patients with ischemic and hemorrhagic stroke reaches 25-40%, which is much higher than in patients who have not suffered a stroke. Disorders of rhythm and conductivity of the heart, associated with cerebral lesion, were found in 70-75% of patients in

the acute stage of the disease. Permanent atrial fibrillation occurs in about 25% of patients with a brain infarction.

The indicators such as the size and localization of brain damage were more and more important. The disturbance of cardiac activity is more pronounced in the localization of ischemic softening in the brain stem (57%), a large hemisphere hearth with secondary stem syndrome (43%) than in small hemisphere spots (so-called "small ischemic stroke"). as bradycardia, tachy-brady syndrome, common myocardial ischemia (up to necrosis by type of "stroke-infarction") are a bad predictive sign, indicating the involvement in the pathological process of stem structures of the brain with the suppression of the activity of respiratory and vascular centers.

Neurogenic depressions of the ST segment in the acute stage of stroke, in the studied patients, worsened in the later post-stroke period, and the appearance of significant changes in the Electrocardiography significantly worsens both the nearest (30 days) and long-term (6 months and beyond) prognosis of ischemic stroke, reliably increasing mortality rates in the control group compared to the main nuppa.

Neurological symptoms of cerebrocardial syndrome. Patients with cerebrocardial syndrome had symptoms of neurological deficiency corresponding to the lesion. In addition to the motor, sensitive focal symptoms, the study team identified and analyzed the characteristic changes in cognitive functions that were considered in the dynamics of the disease.

Thus, 13 patients (21.6%) according to clinical data and MMSE at the time of the first examination, there was no cognitive decline (average MMSE score of 28.6+/-0.84), 47 people (78.3%) and 47 people (78.3%). a mild cognitive impairment (average MMSE score of 25.38+/- 1.17) was detected. According to the results of the "frontal dysfunction battery" technique, the average score was 14.38+/-2.

MMSE score for the first (core) group was 25.86, for the second (control) - 25.96. A re-examination three months later revealed that clinically expressed dementia syndrome developed in 15 patients: in the main group - in 4 people (13.3%) 11 (36.7%) had a 95% chance of a difference of 95 percent.

Results from MMSE scores and the frontal dysfunction battery in the first group was 26.1+/-2.1 and 15.57+/-1.4 points, respectively. In the second (control) group, the average MMSE score was 24.37+/-2.5, and the average score of the frontal dysfunction battery was 12.07+/-2.3. Differences between the two groups tended to be reliable

Analysis of the obtained results. Based on the peculiarities of pathogenesis, in the therapy of patients with acute and chronic vascular diseases of the brain widely received pharmacological means that affect neurotransmitter systems. This group includes the drug Cerepro (cholynoalfoscerat). The central choline stimulant, which contains 40.5% metaboltprotectedcholine (metabolic protection contributes to the liberation of choline in the brain). When ingested, it is broken down by enzymes into choline and glycerophosphate: choline participates in the biosynthesis of acetylcholine, one of the main mediators of nervous arousal; glycerophosphate is a precursor to phospholipids (phosphathisylcholine) of the neural membrane. Provides synthesis of acetylcholine and phosphatidylcholine in neuronal membranes, improves blood flow and enhances metabolic processes in the central nervous system, activates reticular formation. Normalizing effect on reticular formation reduces the likelihood of aggravation of cerebrocardial syndrome, which plays an important role in patients of the study group.

By increasing the linear rate of blood flow on the side of traumatic brain damage, contributes to the normalization of space-time characteristics of spontaneous bioelectric brain activity; has a positive effect on the cognitive and behavioral responses of patients with vascular diseases of the brain. Dozodependently stimulates the release of acetylcholine, improves synametic transmission, receptor function. Cholinerere systems are able to protect neurons from excessive exposure to catecholamines; the metabolites of the mediator acetylcholine are part of cell membranes and provide their matrix functions; post-synaptic receptors of neurons of cholinergic systems are able to have a metabolic effect; Cholinergic factors of differentiation of neurons act as neuropoethins for nerve cells of the brain and are able to perform modulating functions.

Conclusions. The study revealed that the use of drugs of the group chotinoalfoscerate in the acute phase of stroke and in the next 2 months has a beneficial effect not only on the restoration of motor and sensitive neurological deficits, improved cardiac activity, but also reduces the incidence of early post-stroke dementia (high confidence) and reduces the severity of clinical manifestations of lung and moderate cognitive disorders. For the comprehensive correction of the manifestations of ACCD and the prevention of post-stroke dementia, as well as reducing the progression of cognitive disorders, stroke patients should recommend the use of drugs chodinoalfoscerat (Cerepro) (in a dose of 2000mg/day intravenous drip for 5 days, then 1000mg/day intravenous5 days, then 1 restorative tablet 2 times a day (800mg/day) - 2 months) from the first days of stroke and in the early period.

References:

[1] VarakinY.Y Epidemiology of Vascular Diseases of the Brain // Essays of Angioneurology / Under auth. Z.A. Suslina. - M.: Atmosphere, 2005. - p. 66-81.

[2] Z.A. Suslina, Varakin Y.Y., Vereshchagin N.V. Vascular Diseases of the Brain: Epidemiology. The basics of prevention. - M.: MEDpress-Inform, 2006. P 256

[3] VerninoS.,BrownR.D., JamesJ.S. etal. Cause- specific mortality after first cerebral infarction (a population- based study) // Stroke. – 2003. – Vol. 34. – P.1828.

[4] Bernadsky V.V. Cerebro-cardiac syndrome in the acute period of ischemic stroke./Dics. M. 2000.

[5] Martynov Y.S., Bernadsky V.V., Shuvakhina N.A., etc.Cerebro-cardiac syndrome in ischemic stroke.Journal of Neuropathology and Psychiatry.Stroke.2003; 9: 167.

[6] Martynov Y.S., Kumar Oli K, Shuvakhina N.A., et al. Cerebrocardial disorders in hemorrhagic stroke. Therapeutic Archive 2004; 76(2):44-49.

[7] Hachinski VC. The clinical problem of brain and heart./ Stroke 1993; 24: 1-2.

[8] Norris JM, Froggatt GM, Hachinski VC. Cardiac arrhythmias in acute stroke./ Stroke 1978; 9: 392-396.