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FEATURES AND DYNAMICS OF STATIC-LOCOMOTOR AND COGNITIVE IMPAIRMENT AFTER POSTPONED ISCHEMIC STROKE

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Abstract:

Ischemic damage to brain cells results from a significant decrease in arterial blood flow to brain tissue or as a result of a long or shortening of the blood vessels in the brain. The result of these processes is focal or diffuse brain injury. A clear violation of microcirculation leads to several microinfarctions and occlusive lesions of large arteries - extensive cerebral infarctions.

Keywords: Vascular dementia, diagnosis, static-locomotor ischemic stroke, Diuresis.

Vascular dementia is most common in the elderly. Some authors believe that cognitive impairment in chronic heart failure is in vascular pathology (1). Even "circulatory dementia" has been identified to detect severe impairment of cognitive function associated with heart failure. An increase in systemic venous pressure against the background of a decrease in heart rate leads to a delay of cerebrospinal fluid in the cranial cavity, which leads to the development of chronic heart failure encephalopathy. Other researchers use the term "dysmetabolic encephalopathy" to describe defined neurological diseases.

Exacerbation of chronic heart failure leads to an increase in cognitive impairment (4). Although chronic heart failure therapy is adequately performed, it promotes their regression. Thus, the study of the dynamics of 6 patients with chronic heart failure IV Functional class for 50 weeks after the appointment of treatment and a reduction in the severity of symptoms of chronic heart failure observed a partial recurrence of the identified diseases. The possibility of reducing the severity of cognitive impairment, especially performance and attention, has been suggested by other authors. These facts are consistent with data from Russian neurologists who have assessed dementia as secondary and "reversible" in chronic heart failure.

The mechanism of development of cognitive impairment in patients with chronic heart failure is currently not fully understood. It is known that chronic hypoperfusion is a possible mechanism of brain injury in patients with chronic heart failure. Some authors suggest that a decrease in heart failure is the highest cause of cognitive impairment, leading to a lack of brain perfusion and oxygen (3).

Thus, a decrease in heart rate in elderly patients with stable heart disease is associated with poor performance of tests to assess performance functions, which is associated with decreased blood flow in deep regions of the brain and interactions between cortical and subcortical zones. associated with a decrease. Cognitive impairments mainly develop as a result of ischemia and cerebral hypoperfusion in the cerebral vascular system. The third group of authors defines cerebral hypoperfusion as a result of decreased cardiac output and microembolism (8). A fourth group of researchers focuses on the exacerbation of microembolism with the onset of cerebral infarction.

The contractile capacity of the myocardium is characterized by a left ventricular emptying fraction, so much attention is paid to studying its effect on the development of cognitive impairments. It has been noted that the relationship between left ventricular discharge fraction value and severity of cognitive impairment is not linear (11). in patients with chronic heart failure, a decrease in the amount of left ventricular emptying fraction is manifested by low performance during performance functions and memory testing (Hoth K.F. et al., 2008). An increase in left ventricular emptying fraction as a result of resynchronization therapy reduces cognitive deficits, especially in improving performance functions and reducing visual-field impairments (10). Other authors have not been able to determine the effect of left ventricular emptying fraction value on the development of cognitive impairment (7). An important role of myocardial contractility is that heart transplantation in patients with terminal heart failure leads to an increase in mental response and performance on the "Shortness of Mood Assessment", as well as a decrease in pathological fatigue and depression according to neuropsychological examinations. (4 months after transplantation). (6).

Several authors suggest a fact related to blood pressure. A survey of 1,583 elderly patients with chronic heart failure found that systolic blood pressure below 130 mm Hg was associated with cognitive impairment, similar to that in patients without chronic heart failure. case was not found. According to other authors, arterial hypotension is not considered a risk factor for the development of cognitive impairment in chronic heart failure (2).

Stroke is the leading cause of disability due to its effect not only on motor and sensory functions, but also on cognitive functions [10]. The combination of physical and cognitive impairments (CI) interferes with the restoration of

impaired functions and increases the risk of dependence on outside help [8]. CNs are one of the most significant factors for predicting the consequences of stroke, but for a long time they were not given due attention. Although the relationship between mental health and quality of life appears to be clear, widely used rating scales for post-stroke functional impairment, such as the Barthelindex index, do not provide a measure of cognitive deficit. One of the prerequisites for excluding cognitive function from the dimensions that consider recovery from stroke may have been the previously held belief that neurodegenerative diseases such as Alzheimer's disease (AD) are the only cause of dementia. However, further study of this problem led to an understanding of the key role of vascular disorders in cognitive dysfunction and the identification of a close relationship between vascular and degenerative changes in the brain [7]. In this regard, vascular (in particular, post-stroke) CNs have become one of the most pressing problems of modern neurosciences [2].

Stroke is a potent risk factor for the development of CI and dementia. The frequency of post-stroke CI can reach 82% [4]. Two weeks after stroke, some degree of cognitive impairment occurred in 91% of patients [5]. Data on the incidence of post-stroke dementia vary depending on the timing of the study, the location and size of the infarction, the method for determining dementia, and other methodological features. Prospective studies have shown that stroke increases the risk of developing dementia by about 10 times, and its prevalence in post-stroke patients is 20-25% [7]. In residents of Rochester, USA, the incidence of dementia increased from 7% in the first year to 48% for 25 years after stroke [1]. A recent meta-analysis concluded that 7–23% of patients develop dementia during the first year after stroke [5]. The results of prospective population studies indicated a smaller, but still significant (approximately twofold) increase in the risk of dementia after stroke [14]. Since not all CIs reach the degree of dementia and not all strokes are diagnosed, these data seem to underestimate the role of cerebrovascular pathology in the development of cognitive dysfunction [9].

Connection of scientific work with the research program of the institution. The work is carried out within the framework of the research and development of the Bukhara State Medical Institute

The degree of knowledge of the problem and the rationale for the need for scientific research.

In the modern literature, there are no unambiguous data on the effect of the severity of post-stroke dementia dramatically increases the risk of death, regardless of age, functional status and concomitant diseases [11]. These findings highlight the importance of cognitive assessment and early diagnosis of dementia in the care of stroke patients, including post-stroke rehabilitation. At the same time, the development of dementia sharply complicates the observation of the patient in scientific research, which leads to an underestimation of both the prevalence of vascular dementia and its effect on the life of patients [12].

Post-stroke CI, like any other consequences of stroke, it makes sense to consider only in surviving patients. It is logical to assume that a decrease in mortality in stroke will be accompanied by an increase in the prevalence of post-stroke cognitive dysfunction. The results of a study of 42 thousand people aged> 65 years, conducted within the framework of the National Long-Term Care Survey in the United States, showed that in 1991-2000. compared with 1984-1990. the incidence (adjusted for age) of all types of dementia increased by 53%, and post-stroke dementia by 87%. This happened against the background of a decrease in the mortality rate during this period within 1 year after stroke from 65 to 53% [13]. These data indicate an association between increased survival and prevalence of CI in patients after stroke, and also highlight the importance of prevention, diagnosis, and treatment of post-stroke cognitive dysfunction. In addition, the incidence of cerebrovascular and neurodegenerative diseases rapidly increases with age; therefore, it can be expected that the prevalence of this pathology, which is associated with CI, will increase as the population ages.

Some authors report that neuropsychological test results are related to magnetic resonance imaging of the medial surface atrophy of the left hemisphere and the extent of damage to the deep regions of the cerebral hemispheres (9). Others have been linked to memory loss in patients with chronic heart failure, impaired conditioned and unconditioned reflex functions, decreased test scores for a short scale to assess mood, and atrophy of the medial surfaces of both segments. (12). In addition to hemodynamic causes, dysmetabolic causes also play an important role in the pathogenesis of cognitive impairment, especially in the late stages, with many authors classifying encephalopathy in chronic heart failure as dysmetabolic (11). According to a survey of 151 patients with chronic heart failure after multi-stage logistic regression modeling, the likelihood of cognitive impairment is directly related to decreased levels of albumin, potassium, sodium, hemoglobin in the blood, as well as the severity of hyperglycemia detected. Normalization of glucose, potassium, hemoglobin levels is accompanied by a decrease in the level of cognitive impairment in patients with chronic heart failure. Thus, analyzing the literature, we found that not only the pathogenesis of cognitive impairments, but also the lack of data predicting their development. Although the mechanism of development of cognitive impairment in chronic heart failure is not fully understood, cerebral hypoperfusion is often considered to be their cause. It is recommended to use cerebral blood flow indices to estimate the severity of chronic heart failure as much as possible.

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