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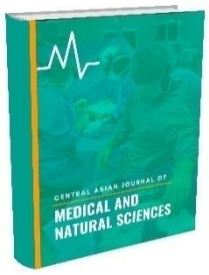

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Cognitive Disorders in Patients after Ischemic Stroke and Modern Methods of their Rehabilitation

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Abstract: Cognitive impairment is one of the most disabling complications of ischemic stroke, significantly impairing the patient's adherence to rehabilitation and long-term life-long treatment, reducing the quality of life of the patient and his relatives. The article deals with the issues of epidemiology, etiology, pathophysiology of post-stroke cognitive impairments. Improving the efficiency of rehabilitation of patients after stroke remains one of the most important strategic tasks of Russian healthcare. Cognitive impairments that develop early after a stroke negatively affect the rehabilitation process. Their timely identification and correction can improve the functional outcome. Currently, the use of many drugs with neuroprotective properties does not have a strong evidence base. Risk factors, classification, treatment and prevention of post-stroke cognitive disorders are discussed.

Keywords: ischemic stroke, rehabilitation, cognitive impairment, moderate cognitive impairment.

In recent decades, there has been a global increase in the prevalence of vascular diseases of the brain, including strokes, the consequences of which are the main cause of disability in older people [4, 6]. The proportion of patients who are incapable of independent living and self-care is approximately 30% as early as a year after a stroke [14]. In this regard, one of the main tasks of Russian health care is to slow down the growth of disability among the population, which is carried out through the active development of the rehabilitation system in our country, including neurorehabilitation.

Neurorehabilitation is one of the relatively young areas of rehabilitation. This is a progressive, dynamic and purposeful process, the task of which is to achieve the optimal level of physical, cognitive, emotional, social, communicative and functional activity by the patient with emerging disorders [3]. Restoration of impaired functions through the selection of effective means of rehabilitation and the selection of adequate drug treatment is a complex and lengthy process that requires the participation of a multidisciplinary team of specialists.

In connection with the occurrence of complex neurological deficit in most patients after a stroke, rehabilitation includes a variety of measures aimed at restoring or compensating for the impaired functions of the nervous system. The recovery processes are based on the mechanisms of

neuroplasticity - the ability of the nervous tissue to structural and functional restructuring that occurs after its damage, which contributes to the restoration of not only impaired motor functions, but also memory, learning, and the acquisition of new skills [2].

The restoration of cognitive functions is necessary at all stages of rehabilitation, since their decrease leads to insufficient involvement of the patient in the rehabilitation process, which can significantly complicate it, as well as significantly reduce the quality of life.

Cognitive abilities are important for combating the entire range of neurological disorders in a patient, since patients with a higher cognitive status at the time of the beginning of rehabilitation measures achieve better results in recovery [13]. Cognitive impairments can reduce the ability to absorb instructions, plan and perform exercises independently, and solve various problems. The level of preservation of cognitive functions in patients after stroke has an independent correlation with the degree of involvement in the rehabilitation process in the hospital [5].

Cognitive rehabilitation of stroke patients speeds up the processing of information, allows the patient to get a correct idea of the intact functions of the body and contributes to a more successful social and household adaptation, taking into account the existing deficit. Several studies have demonstrated the effectiveness of certain drugs in improving cognitive functions at different stages of recovery in patients with stroke. Thus, drugs with a neurotrophic effect can promote better recovery from stroke, although the data available to date are contradictory and require further research [1]. In a number of studies in recent years, which assessed the effectiveness of the restoration of cognitive functions in the course of rehabilitation of patients after a stroke, there was also no significant improvement in terms of impaired attention and executive functions [12].

For the formation of the correct rehabilitation strategy in the correction of cognitive deficits in patients after stroke, it is necessary, first of all, to understand the underlying causes of the development of these disorders, as well as their timely diagnosis in the early stages of their onset and assessment of their severity in order to determine the effectiveness of treatment and rehabilitation measures.

Today in Bukhara there is a three-stage system of rehabilitation of patients after a stroke:

- acute stage of stroke (stage I) - primary vascular departments;
- early recovery period (stage II) - specialized assistance in early recovery treatment units (hospital);
- late recovery period (stage III) - polyclinic and rehabilitation centers.

The staging of rehabilitation measures underlies the algorithm for identifying cognitive disorders and conducting cognitive rehabilitation in patients with acute cerebrovascular accidents (ACVI).

At the first stage, after clinical and neuroimaging examination, it is important to assess cognitive functions, which must be carried out by a neurologist or neuropsychologist (neuropsychological testing), if the general condition of the patient allows, and even if the patient outwardly gives the impression of being “intact”. Screening tests for such an assessment of cognitive functions are the Mini Mental State Examination (MMSE), the clock drawing test, the test for free and directed verbal associations, serial counting, repetition of numbers in the forward and reverse order, the test for memorization series of words or images with an assessment of both free reproduction and recognition. If violations are detected, the initiation of appropriate treatment is indicated. The wide range of drugs with neuroprotective properties used today does not have a serious evidence base for their effectiveness, however, the results of individual studies suggest an improvement in the recovery of memory functions, attention, concentration of attention while taking drugs of this group, such as cerebrolysin, citicoline, actovegin , mexidol, etc. [15].

At the II stage of rehabilitation, the assessment of cognitive functions is repeated and in the presence of impairments, specific treatment is prescribed, which may include not only drugs with

neuroprotective properties - cerebrolysin, citicoline, Mexidol, etc. [7], but also drugs with proven efficacy acting on cholinergic (cholinesterase inhibitors, such as galantamine or rivastigmine) and glutamatergic (inhibitor of NMDA-glutamate (NMDA - N-methyl-N-aspartate) receptors memantine) systems, as well as cognitive rehabilitation: cognitive training, biofeedback (BFB) by electroencephalography (6, 14], and other methods.

At stage III, it is necessary to prescribe / continue drug treatment after appropriate diagnostics of cognitive functions, as well as to carry out continued cognitive rehabilitation.

There are many scales for assessing cognitive functions, but the most common and widely used are MMSE, Montreal Cognitive Assessment (MoCA), a battery of tests for frontal dysfunction, neuropsychological research according to A.R. Luria (2010), which is standardized a method of assessing cognitive functions, including in patients with speech disorders, etc. [10, 12]. The use of these scales at each stage of rehabilitation requires the involvement of a neuropsychologist. There are also scales for a more detailed study of cognitive deficits, the use of which takes time and specialist work.

Recovery of impaired functions after a stroke is a complex problem, and knowledge of the mechanisms underlying these disorders is fundamentally important for determining the tactics and strategies of both treatment and rehabilitation.

As already noted, the restoration of functions lost due to stroke is based on the processes of neuronal plasticity, i.e. the ability of the nervous tissue to form new interneuronal connections, to perform new functions that it did not possess before the stroke, such as the performance of movements due to the involvement of new areas of the cerebral cortex [11]. These processes are induced by training certain actions to accelerate natural functional recovery due to neuronal plasticity [10]. The neuroplasticity of the neocortex can be activated by changing sensorimotor interactions and sensory impulses, which leads to the reorganization of the motor cortex in both animals and humans, this is the cornerstone of rehabilitation measures. Neuronal plasticity is tightly regulated, therefore, the timing of the start of neurorehabilitation measures is very important to achieve the maximum effect. With age, the processes of neuroplasticity slow down, therefore, the recovery processes may be less active.

Changes in the brain resulting from a stroke are extremely complex and not fully understood. Thus, it was revealed that nerve fibers undergo reorganization around the infarction area, and sprouting is observed in the terminal area of axons. In addition, the development of neurons from progenitor cells located in the subventricular zone of the lateral ventricles and subgranular layers of the dentate gyrus may also contribute to repair processes and functional recovery after stroke. In old age, the survival of neuronal progenitor cells is extremely low, so their role in the restoration of impaired functions in humans is very limited [3].

The results of studies both in animals and with the participation of humans indicate that pharmacological drugs can affect the processes of neuroplasticity [9]. Thus, the mechanisms of activation of NMDA receptors and suppression of the activity of γ-aminobutyric acid are involved in plasticity processes in the intact human cerebral cortex. Taking drugs agonists of adrenergic and dopaminergic receptors in combination with rehabilitation measures promote the activation of plasticity processes [6].

It is assumed that the local release of neurotransmitters affects the plasticity of the cerebral cortex, enhancing the excitatory glutamatergic effect in the cortex and facilitating the activation of NMDA receptors before reaching the threshold for providing synaptic changes involved in the formation of long-term potentiation. These synaptic changes are important for neuronal plasticity, and it has been suggested that drugs that increase the number of neurotransmitters may enhance it [13].

In animal studies, it has been demonstrated that the activation of muscarinic receptors is associated with the formation of memory, learning and long-term potentiation processes. In healthy volunteers, there was an increase in the formation of motor memory when taking acetylcholinesterase inhibitors [14]. It is known that acetylcholinesterase inhibitors can improve cognitive ability in patients with Alzheimer's disease, and in recent years there has been evidence of an improvement in cognitive functions while taking acetylcholinesterase inhibitors in patients after stroke [16].

A number of processes that are triggered after the development of a stroke, for example, excitotoxicity, make a significant contribution to the death of neurons in the acute period of stroke and are associated with hyperactivation of glutamate NMDA receptors. The drug memantine is a noncompetitive agonist of NMDA receptors, it is used in the treatment of Alzheimer's disease, it is assumed that the drug can prevent the excessive activation of glutamate NMDA receptors without disrupting their physiological activity [3].

Memantine is a noncompetitive low-affinity voltage-dependent antagonist of NMDA receptors and blocks cation channels at rest; however, with complete depolarization of the cell membrane, it is removed from the channel, which contributes to the normalization of synaptic transmission and restores the signal-to-noise ratio [9]. The drug is called an “open channel blocker” because a molecule can enter the channel and block the flow of ions only after the channel is open. Apparently, the binding regions of memantine and Mg2 + ions partially overlap, since an excess of Mg2 + decreases the degree of blocking of channels by memantine [7]. After memantine blocks the NMDA receptor, the ion channel is closed and the agonists are released, “blocking” memantine within the channel. At the same time, complete membrane depolarization leads to the removal of memantine from the canal, so the drug does not interfere with normal synaptic transmission.

Improvement of cognitive functions is associated with the fact that the drug eliminates synaptic “noise” caused by excessive activation of NMDA receptors, suppresses the formation of β-amyloid or reduces its toxic effect, and also changes the balance of excitation and inhibition in the brain [9].

Under conditions of stroke, memantine avoids the adverse effects caused by excessive accumulation of glutamate, in particular, cell death as a result of excitotoxicity [3].

In a number of preclinical studies in vitro and in vivo, the neuroprotective potential of memantine has been revealed [15]. It has been demonstrated that short-term administration of low-dose memantine leads to a significant decrease in the lesion volume and an improvement in the behavioral outcome of stroke [11].

A number of studies have noted the effectiveness of using memantine in combination with intensive speech rehabilitation in terms of improving speech function, lasting at least 1 year, compared with placebo [3]. Unfortunately, the amount of clinical data on the use of memantine in stroke is limited.

Table 1. Comparative characteristics of patients (abs. (%)

Specifications	1 stgroup (n = 42)	2 stgroup (n = 25)
Basin of ischemic stroke middle cerebral artery posterior cerebral artery anterior cerebral artery	27 (64,3) 12 (28,6) 3 (7,1)	18 (72,0) 6 (24,0) 1 (4,0)
Focal lateralization right hemisphere left hemisphere	26 (61,9) 16 (38,1)	17 (68,0) 8 (32,0)
Localization of brain damage cortical cortical-subcortical subcortical	13 (31,0) 20 (47,6) 9(21,4)	7 (28,0) 12 (48,0) 6 (24,0)

A pilot, double-blind, placebo-controlled study is currently underway to assess the clinical benefits of memantine in stroke (ClinicalTrials.gov identifier NCT02144584 [1]).

In the works of N.V. Vakhnina et al .; HS. Chataeva; S.E. Khatkova; S.E. Khatkova, A.N. Kuznetsov and others have shown that memantine improves the quality of life of patients with post-stroke cognitive impairment by reducing the severity of neurodynamic, regulatory and operational cognitive impairments, as well as affective disorders [1, 16].

Conclusion. The medical rehabilitation of patients with complex neurological deficits (cognitive, motor, speech) after a stroke requires the organization of a complex, highly professional and staged system for the provision of specialized (at stages I and II) and primary specialized medical and sanitary (at stage III) care with the obligatory consideration of the specifics. , clinical features and variants of the course of the disease. Particularly important are a personified, patient-oriented approach to treatment and ensuring continuity in carrying out treatment and rehabilitation measures at various stages of medical rehabilitation.

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