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Cognitive Disorders in Stroke

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Abstract---*Cerebrovascular disease is one of the most important medical and social problems not only in our country, but all over the world. Because of the prevalence of the disease, the high mortality and disability rates, the inability of many survivors to continue their activities, and the loss of their place in society, the problem is further compounded. Therefore, the fate of patients with cerebrovascular disease is not limited to neurology; rather, it has become a problem for society as a whole. This disease is increasing in the developed countries of the world, including Uzbekistan. The present article discusses the cognitive disorders in stroke.*
Keywords---*atheromas, cerebrovascular disease, etiopathogenesis, hemi syndrome, hemorrhagic, ischemic, neuropsychological, pathogenesis, prevention, stroke, subcortical-forehead cognitive syndrome, treatment*

Introduction

People do not follow a healthy lifestyle, do not consult a doctor in a timely manner; most patients continue their daily activities with high blood pressure, do not take the recommended medication on time, or take it incorrectly, leading to an increase in cerebrovascular disease. In addition, the very rapid development of technology, the pollution of the external environment with various harmful substances, the proliferation of infectious diseases and poisonings also contribute to the proliferation of cerebrovascular diseases. Therefore, cerebrovascular accident is the focus of all clinicians and practicing physicians, and for many years this problem has been on the agenda of international conferences. Among cerebrovascular diseases, cerebral circulatory disorders have a special place. Stroke is a pathology that reduces the working capacity of the population, leads to long-term hospitalization, permanent disability of patients, a decrease in the quality of life in their families and significant economic costs to the state (Akhmedzhanov, 1996; Batuev & Sokolova, 2004; Batuev, 2010).

The Main Findings and Results

Cerebral stroke is a sudden disruption of blood circulation in the brain, generalized and focal neurological syndromes that persist for at least 24 hours or at this time or a disease associated with the clinical syndrome of acute cerebrovascular injury that results in the death of the patient at an earlier stage and is considered a branch of

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neurology (Beĭn & Riasik, 2001;Belova, 2000). Stroke results in destructive changes in brain tissue, resulting in persistent symptoms of organic damage. Stroke is the third leading cause of death after cardiovascular and oncological diseases, accounting for 14% (Mankovsky et al., 2000). According to studies, the prevalence of stroke is 164 to 261 per 100,000 populations Gitkina et al. (1992), the mortality rate is 35% in the acute period of the disease, and 45-50% at the end of the first year. The incidence of stroke in Uzbekistan is 0.9-1.5% per 1,000 populations, and this figure is growing from year to year (Asadullaev et al., 2002). In recent years, more than 48,000 ACSs (acute cerebral stroke) have been registered in Uzbekistan annually, of which 22,000 are hospitalized. Of these, 44.6% had mortality, 42.2% had disability, and only 10.2% had relapsed. In Tashkent, there is 1.5 per 1,000 populations. 20-25% of patients are able to continue their lives independently after stroke with various neurological defects. Most importantly, 25% of stroke patients died in the first day, 40% in 2-3 weeks, and 50% of survivors in 4-5 years. The reason for this high figure can be explained by the acute onset of the disease, in which there is a sudden onset of movement, intuition, speech, coordination and cognitive disorders, post-stroke depression and panic attacks. Therefore, early diagnosis, adequate timely treatment and prevention of CVD (cerebral vascular diseases), especially ACS, is of great importance. There are 2 different types of stroke: ischemic and hemorrhagic. Ischemic stroke is on average 5-6 times more common than hemorrhagic stroke (Evzelman, 2003; Gafurov & Alikulova, 2002).

The terms “ischemic stroke, cerebral infarction, and acute cerebral ischemia” are used synonymously in the literature. Acute ischemic stroke is characterized by a high degree of lethality. It is not only the age of the patient, the scale of the pathological lesion, the early addition of recurrent stroke, but also cardiac complications, sublingual oscillations that increase mortality. In this case, the consequences of stroke depend on various factors - the age of the patient, the presence of arterial hypertension, diabetes, heart failure, the presence of strokes, the patient's race. The main causes of circulatory failure in the brain are cerebral atherosclerosis and hypertension, rarely vascular anomalies (aneurysms, pathological bends, narrowing), rheumatism, vasculitis, heart pathology, diabetes, blood diseases. Each of the above pathological processes is important, among which cognitive disorders have a special place (Gafurov & Alikulova, 2004; Gusev & Skvortsova, 2002). Because of cognitive impairment, patients ’quality of life decreases and they experience social maladaptation.

Cognitive functions are the relatively more complex functions of the brain that are used to understand the world rationally and to interact purposefully with it. This process involves 4 main interconnected components, each of which is associated with a specific cognitive function:

1) Acceptance of information - gnosis.
2) Processing and analysis of information - the manifestation of attention, similarities and differences, formal-logical operations, the establishment of associative connections, the introduction of mental conclusions.
3) Remembering and storing information - memory.
4) Information exchange, development and implementation of action programs - speech and practice (targeted action activity).

The majority of patients with stroke show various disorders of neuropsychological functions. The concomitant onset of cognitive impairment and panic attacks, especially in strokes, is important because during this period, although the symptoms of hemisyndrome in most patients recover, the rate of cognitive impairment and panic attacks increases. As a result, most patients are unable to self-care due to cognitive impairment and panic attacks, even if the movement disorder is restored in patients (Gusev, 2003; Gusev & Skvortsova, 2003). Etiopathogenesis. The study of the causes of cerebrovascular diseases, especially cerebral strokes, reveals the presence of a number of bad factors - the risk factor. These are:

1) Heredity - hereditary predisposition. Hereditary predisposition plays a major role in the development of atherosclerosis and atherosclerosis.
2) Low mobility - various hearts - can cause vascular disease.
3) Nutrition is an alimentary factor. Eating can cause depression and atherosclerosis. Japanese scientists have concluded that excessive consumption of refined rice and table salt can lead to depression and cerebral hemorrhage. 4. Smoking. Causes atherosclerosis of the heart and cerebral vessels.
4) Psycho - emotional factor. Sudden disruption of the management of homeostasis in patients suggests that it leads to acute disturbances of cerebral circulation, especially transient disturbances. 6. Cholera. An increase in blood pressure from 200 mm Hg increases blood flow to the brain by 13 times, and from 160-200 mm Hg by 8-9 times.
5) 7. The influence of the external environment.
6) 8. Infectious diseases and poisonings.

The presence of several of the above factors in one patient accelerates the onset of cerebrovascular disease. Diseases leading to IS.

1) Atherosclerosis.

2) Cholera.
3) Concomitant atherosclerosis and coronary heart disease.
4) Symptomatic hypertension, ie an increase in blood pressure in other diseases.
5) Hypotension and symptomatic hypotension.
6) Heart disease - myocardial infarction and arrhythmias.
7) Vasculitis, endo-arthritis (rheumatic syphilitic, allergic, toxic).
8) Aneurysms.
9) Blood diseases.
10) Compression of arteries and veins (in diseases and tumors of the spine).

As a result of the influence of the above factors, IS appears. Ischemic strokes are divided into thrombotic and non-thrombotic strokes. Cerebral infarction is the result of arterial blood flow not reaching certain areas of the brain. This condition is caused by narrowing of the vascular space. Narrowing of the vascular cavity due to atherosclerotic plaque. Ischemic stroke, unlike transient ischemic attacks, is a qualitatively new condition. This involves the integration of hemodynamic and metabolic disorders, which occur at a certain stage of circulatory failure, which prepares the brain substance for necrosis (Ionova et al., 2020; Zenkov & Ronkin, 1991). Pathobiochemical cascade reactions that occur in all areas of the brain (especially in the affected areas) lead to changes in the neural pathway, astrocytosis and activation of glia, disruption (dysfunction) of the trophic supply of the brain. The onset of cascade reactions is the formation of a cerebral infarction, which can proceed through two mechanisms as necrotic cell death and apoptosis - genetically programmed cell death. The severity of an ischemic stroke is determined primarily by the depth of the decrease in blood flow to the brain, the duration of the period before perfusion, and the duration of the ischemia (Douglas et al., 1999; Norwood, 1991).

The area of decreased cerebral blood flow (maximum 10 ml / 100 g / min) becomes irreversible damage within 6-8 minutes from the moment the first clinical symptoms appear. Within a few hours, a central point infarction occurs (with a decrease in blood flow to the brain to 20–40 ml / 100g / min), but is surrounded by ischemic living tissue. It is called the ischemic hemisphere or penumbra zone, where the overall energy metabolism is still maintained and there are no structural changes. The duration of the presence of penumbra is specific to each patient and determines the boundary of the temporal period, in which there is a “therapeutic window”, within which it is possible to conduct very effective treatment procedures. The formation of the majority of cerebral infarction occurs within 3-6 hours from the onset of the first symptoms of stroke. The formation of the lesion lasts 48-56 hours, but even longer, (taking into account the preserved brain tumor). More recently, the presence of an autoimmune process in the acute phase of ischemic stroke has been shown to increase anti-DNA and OBM levels in both serum and spinal fluid. Recently, little attention has been paid to vascular spasm as a result of functional changes in the arteries in the development of cerebral ischemia. A thrombus or embolus that forms in the walls of the arteries alone cannot completely stop the flow of blood to the brain. Tickling of the vessel walls with a thrombus or embolus causes the vessel to spasm and completely closes the vascular space. Such organic angiospasms lead to ischemia of the cerebral substance due to the fact that it does not pass quickly. After vascular spasm, paralytic dilatation occurs in the vein (Gorter et al., 2004; Sukhija et al., 2004).

Through the collateral vessels, blood begins to flow into the vessel in which this paralytic dilated blood stasis occurs, and from its altered walls the erythrocytes loosen and pass into the cerebral substance. Depending on the degree of diapedesis, white or red ischemia occurs in the cerebral substance. Therefore, sometimes ischemic strokes are accompanied by hemorrhagic. An important role in the pathogenesis of ischemic stroke is played by cardiac dysfunction, narrowing of the main vessels due to stenosis, vascular insufficiency caused by a decrease in blood pressure in the cerebral arteries. Changes in cerebral vascular walls, decreased blood pressure, and increased blood clotting are vertical factors in the pathogenesis of cerebral vascular thrombosis. Cerebrovascular embolism is often associated with heart disease (Group, 1997; Sachdev, 2004; Olmez & Ozyurt, 2012). Especially rheumatic endocarditis, mitral stenosis with thrombus in the anterior ventricle of the heart is very dangerous. Emboli in the ventricles of the heart can occur in congenital malformations, myocardial infarction. Surgical procedures performed on various organs (especially the heart and lungs), fractures of the iliac bones, atheromas in the aorta and cervical arteries can also cause cerebral vascular embolism (Mehanna & Jankovic, 2013; Brandt et al., 2008).

The pathogenesis of strokes and the development of cognitive impairments and panic attacks that occur in them. Cognitive functions are complex physiological and mental processes based on complex hierarchical functional systems, including the cerebral cortex and subcortical structures. The high level of organization of mental processes is primarily associated with the work of the forehead, which carries out the analysis and synthesis of all received information and ensures the individuality of human behavior. The pathological process can lead to various cognitive disorders depending on its etiology. These include factors affecting education, the effectiveness of professional and

social activities, and the subjective or objective deterioration of higher nervous activity. Cognitive impairments resulting from strokes are very common and have significant medical and social significance. Mild cognitive impairments of vascular etiology are observed in 10% of people aged 70-90 years. Cognitive impairments in strokes are of a neuro-dynamic and regulatory nature and, according to A.R. Luria's theory, are associated with dysfunction of the frontal divisions and deep structures corresponding to the dysfunction of the I and III structural-functional blocks.

Cognitive defects are also increasing as the brain changes its qualitative and quantitative characteristics in response to vascular injury. During the late recovery period of strokes, mild neuro-dynamic disturbances in the manifestations of impaired concentration, decreased labor productivity predominate. In line with the development of cognitive disorders, along with neuro-dynamic disorders, regulatory disorders also begin to be observed (subcortical-forehead cognitive syndrome). Disturbances are observed in planning, transition to the execution of various stages of planned mental actions, and control over the achievement of planned results. Memory impairment is usually mild and secondary in nature (even with little help, neuropsychological tests are improved), which in turn makes it possible to compensate for cognitive defects. At this stage, cognitive deficits can affect patients 'quality of life. Subsequent development of a cognitive defect in CE (circulatory encephalopathy) leads to the development of its occurrence along with panic attacks. Cognitive functions include attention, memory, orientation to place and time, intelligence, perception, comprehension, comprehension, thinking, imagination, planning, etc., and the acquired acute impairment of these functions is called dementia. These functions are ensured by the balance of the cerebral cortex and subcortical structures (de Courten et al., 1992).

With the participation of mediators in the maintenance of this complex physiological process, each mediator performs a specific psychophysiological function Characteristics of cerebral neurotransmitter systems Mediator Basic structure Psychophysiological effects Symptoms of deficiency of dopamine Black substance KF activation, search for traces of memory Formation of long-term memory and attention Multiple distractions, affectivity, memory impairment Noradrenolin Blue spot Concentration Decreased mental performance Serotonin Schwann back nucleus Provide positive emotion Decreased mood Panic attacks. When talking about the dynamics of cognitive impairments, it should be noted the pathogenesis associated with relatively stable forms of stroke or recurrent episodic episodes based on cerebral circulatory failure. As a result of pathological changes in the vascular walls, autoregulation of cerebral circulation is disrupted and its dependence on the state of systemic hemodynamics increases. This is compounded by disorders of systemic and cerebral hemodynamic neurogenic rugulation. In this regard, the aging process of the nervous, respiratory and cardiovascular systems, which leads to the development and intensification of cerebral hypoxia, is also important. Cerebral hypoxia then underlies damage to the autoregulatory mechanisms of cerebral circulation. In old age, the dependence of blood circulation in the brain on the state of systemic hemodynamics is further enhanced. More than half of elderly patients with heart failure have cognitive impairments that sometimes manifest to varying degrees, leading to disability.

The severity of cognitive impairment is correlated with the degree of left ventricular failure, and its genesis is based on chronic cerebral hypo-perfusion. In conditions of left ventricular pathology, the heart contraction decreases and on this basis the rate of blood flow to the main arteries of the head decreases. It is not systemic arterial hypotension, but rather a decrease in heart rate that is a major factor determining the decrease in cerebral perfusion in the majority of patients with congestive heart failure. Frequent recurrence of systemic arterial hypotension (e.g., on the background of an arrhythmia or when an overdose of hypotensive drugs) further reduces blood flow to the brain. Panic attacks usually begin to develop in patients who experience the disease at an early age, and the number of patients increases with age. Panic attacks are observed in 1-5% of patients aged 60-65 years, while in patients aged 40-50 years the figure reaches 30%.

For the study, 86 patients aged 45 to 75 years were monitored. Patients were divided into two groups and examined. The rates of cognitive impairment and panic attacks were compared. The aim of the study was to assess the level of cognitive impairment and panic attacks in patients who had a stroke, so we took patients with stroke. All stroke diagnoses were confirmed by computed tomography (CT) and magnetic resonance imaging (MRI) examination. Ischemic stroke was observed in 70 (81.4%) patients and hemorrhagic stroke in 16 (18.6%) patients. Among the etiological factors, hypertension (84.6%), co-occurrence of atherosclerosis (69.2%) and diabetes mellitus (23%) were observed, and in 4.2% of them atrial fibrillation was observed.

Standard neuropsychological examinations were performed to assess the state of higher mental functions in all patients. Also, the speed of sensomotor reactions, attention volume and ability to concentrate, the dynamics of work ability were checked using Shulte tables. The MMSE short scale was used to assess mental status to assess time and place orientation, long and short-term memory, computation, writing, and speech status. When examined in the sphere of movement, it was found that all patients had unilateral paresis and paralysis. In rare patients, monoparesis and mild hemiparesis were observed, and in most, moderate and deep hemiparesis or hemiplegia. In the majority of

patients, a decrease in muscle tone and foot reflexes was observed on the hemiplegia side. In addition, speech disorders have been observed in most patients. This occurred when a stroke occurred in the left middle artery basin of the brain. Speech disorders took the form of motor aphasia or sensory-motor aphasia (according to the anamnesis). Decreased and lost skin reflexes were observed in all examined patients. Pathological reflexes were noted in all patients, of which Babinski and Oppenheim symptoms were more pronounced. Defensive reflexes were also observed in the paralyzed arm and leg.

The presence and obvious detection of pathological reflexes in the vast majority of patients examined suggests, on the one hand, that the central neuron of the voluntary pathway is damaged. Symptoms of oral automatism, including Marinesko-Radovich symptom and Khartoum reflex, were noted in a relatively small number of patients, and no clone of the heel and knee cap was detected. 58 A re-examination of the neurostatus of patients with stroke in the midbrain basin during late recovery revealed regression of the above symptoms in most patients: increased muscle strength, decreased spastic muscle tone, restored hemihypesthesia, called abdominal reflexes. But in the patients the symptoms of Oral automatism, including Marinesko-Radovich symptom and Khartoum reflex, were clearly evoked. This suggests that the involvement of the bilateral cortico-nuclear pathways in the pathological process is high in most patients over time after stroke. The reasons for the increase in the level of cognitive impairments and panic attacks in the post-stroke period were explained as follows. First, over time, an increase in the process of atherosclerosis in the extra and intracranial blood vessels of patients, an increase in the rate of atherosclerotic stenosis of the main arteries was observed. This, in turn, leads to a further decrease in the amount of blood flowing to the brain, an increase in the process of atrophy in the brain, and a deepening of the LCI (levels of cognitive impairment). Second, in the area where the stroke occurred, the post-stroke cyst 65 appears at the site of the subsequent cerebral infarction and there is an enlargement of the area as a result of the development of degenerative changes. Impairment of cognitive function in strokes is a very topical problem, as it is explained not only by the fact that it always occurs and deepens, but also causes disability in such patients (Sandi et al., 2017; Hepsiba & Raju, 2017).

Conclusion

In the post-stroke period, the general condition of most patients was severe, with profound impairment of the musculoskeletal system and the development of cognitive impairment and varying degrees of dementia. Decreased short- and long-term memory, concentration and concentration, and the ability to think after a stroke were observed to be relatively pronounced in patients who did not seek medical attention in a timely manner at the onset of the disease. Impairment of cognitive function in strokes is of a progressive nature. The pathophysiological basis of cognitive impairment in patients with stroke can be explained not only by the appearance of post-stroke cysts and the development of focal degenerative processes, but also by damage to the associative and projection pathways in the white matter that connect the cerebral cortex to its various centers.

In the description of patients with stroke should take into account not only their clinical and neurological indicators, but also the examination of cognitive functions affecting the patient's quality of life using neuropsychological tests. It is recommended to use neuropsychological tests such as MMSE scale, A.R. Luria 10 word memory test, clock reading and Schulte table to detect cognitive disorders. The universality of the neuropsychological test-MMSE short scale was used to assess the manifestation of cognitive impairment in stroke. Although the hemisymptomatics in a stroke are partially restored over time, the degree of cognitive impairment deepens. This in turn causes the patient to lose orientation to place and time due to the disruption of higher nervous activity. As a result, such patients are unable to self-care and need constant care. As a result, patients' quality of life decreases, their place and status in society are lost, and the result is that patients experience panic attacks and their cognitive impairments increase. Therefore, it is necessary to take into account the need for early detection of the level of cognitive impairment in patients, its correction and consideration of psychotherapeutic principles in the treatment. Otherwise it leads to severe dementia.

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