

MORPHOLOGICAL NEUROPATHOLOGICAL CHANGES IN THE BRAIN ASSOCIATED WITH COGNITIVE IMPAIRMENTS

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Resume: The cognitive functions of the brain allow you to know the world, analyze information and make inferences. Cognitive impairment is one of the most disabling complications of ischemic stroke, significantly worsening the patient's adherence to rehabilitation and long-term lifelong treatment, reducing the quality of life of the patient and his relatives. More than half of stroke patients develop CI, and 6–32% of patients develop dementia.

Key words: cerebrovascular pathologies, brain, ischemic stroke, dementia, cognitive impairment, PSCI research scales.

Relevance. Acute cerebrovascular accidents (ACA) continue to be one of the main problems in the world due to high mortality and disability[4]. It was noted that 1,495 (22.5%) of the study patients also had one or more previous strokes, which is a risk factor for a new stroke. Patients with a history of stroke have an 8-10 times greater risk of a future ischemic event than the general population[7]. The cognitive functions of the brain allow us to understand the world, analyze information and make conclusions[10]. Cognitive impairment is one of the most disabling complications of ischemic stroke, significantly worsening the patient's adherence to rehabilitation and long-term lifelong treatment, reducing the quality of life of the patient and his relatives[8]. Currently, CIs that develop within six months from the moment of cerebrovascular accident are considered post-stroke[2]. More than half of stroke patients exhibit CI, and in 8–32% of patients it progresses to dementia [6]. The incidence of CI development is different in different periods of development of ischemic stroke (IS); they occur in 62% of patients in the acute period of cerebral infarction and in 70-80% of patients who have suffered a stroke [3].

Cognitive impairment of varying severity is detected in 40–70% of patients who have suffered an ischemic stroke [12]. The leading role in the genesis of CI is played not by the volume of infarction or hemorrhage, but by damage to areas of the brain strategic for cognitive functions [14].

Risk factors for developing PCI and dementia include:

- socio-demographic features: age over 60 years, female gender, low level of education, belonging to the Asian and Negroid race,
- lifestyle factors: smoking, obesity, alcohol abuse,
- comorbidity: diabetes mellitus, cardiovascular diseases - arterial hypertension, hyperlipidemia, cerebral atherosclerosis, atrial fibrillation, coronary heart disease,
- history of stroke, left hemisphere stroke, ■ vascular and atrophic damage to the brain preceding a stroke - leukoariosis, lacunar infarctions, general brain atrophy, atrophy of the medial temporal lobe as the initial stage of the neurodegenerative process [8].

Post-stroke cognitive impairment is caused by multifactorial structural and functional damage to the brain. The main causes of stroke and chronic cerebral ischemia include hypertension. Against the

background of hypertension, significant changes occur in small cerebral vessels, hypertrophy and lipohyalinosis of the walls of arterioles, and collagenosis of small veins. Reduced cerebral perfusion leads to focal and diffuse ischemic disorders [2,4].

The Newcastle group's (2017) classification of the main types of cerebrovascular pathology accompanied by CI includes:

1. Extensive cerebral infarction or several infarcts - the concept of multi-infarct dementia, a lesion accompanied by the death of a significant (more than 50 mm³) volume of brain tissue.
2. Multiple small infarcts or microinfarcts, accompanied by damage to predominantly the white matter of the brain. Leukoaraiosis and multiple small infarctions develop as a reflection of hypertensive microangiopathy.
3. Strategic cerebral infarction - even a small lesion can be accompanied by significant cognitive impairment up to dementia, if it is localized in a strategic region of the brain for cognitive functions: prefrontal frontal cortex, thalamus, hippocampus, angular gyrus, junction zone of the frontal-parietal-temporal lobes of the brain. It is observed in approximately 5% of cases of cerebral infarction. CIs develop acutely, then stabilize, or a slight regression is observed. The clinic depends on the location of the lesion.
4. Cerebral hypoperfusion with pathology of central hemodynamics and decreased perfusion pressure in the brain. Heart attacks develop in areas of adjacent blood supply, at the junction of vascular territories.
5. Intracerebral hemorrhage.
6. Mixed vascular-degenerative brain damage. The incidence of this variant increases with the age of patients [10].

Often, a heart attack is a trigger for the neurodegenerative process, increases the total volume of affected brain tissue and contributes to the clinical manifestation of Alzheimer's disease. The clinical picture of the most common variant of vascular CI, reflecting dysfunction of the frontal lobes of the brain and impairment of control functions. functions, includes slowness of thinking, difficulty concentrating, disturbances of voluntary attention and switching from one task to another, increased distractibility, perseveration and increased impulsivity, decreased speech activity, decreased analytical abilities, planning, organization and control of activities [9].

As a rule, cognitive deficit is combined with focal neurological symptoms, depending on the location of the lesion, changes in background mood, and emotional lability. Primary memory impairments are not typical for vascular cognitive disorders, but there may be impairments in working memory; patients find it difficult to retain a large amount of information or switch from one information to another. This makes it difficult to learn and acquire new skills, but does not extend to remembering and reproducing life events.

However, if the patient has mixed vascular-degenerative brain damage, then the clinical picture of PCI may be more complex and include mnemonic disorders, the identification of which, however, may

be difficult in this case. The presence of a concomitant neurodegenerative process can be suspected if there is a significant difference between immediate memory and delayed reproduction, if prompts are ineffective during reproduction, or if there is a violation of not only active recall, but also the recognition of information (when choosing from several alternatives) [8,14].

In strategic infarct dementia, the rarest variant of PCI, neuropsychological features are determined by the localization of the lesion. Damage to the thalamus is characterized by significant memory impairment, often reaching the severity of Korsakov's syndrome, decreased activity and initiative, episodes of confusion, and hallucinatory phenomena. Damage to the head of the caudate nucleus leads to disruption of functional frontal-subcortical connections, which leads to disorders. managers. functions of the frontal cortex[5].

Impaired executive functions in combination with dyspraxia and impaired reciprocal coordination are typical for damage to the corpus callosum as a result of an infarction or hemorrhage in the anterior communicating artery. The consequence of vascular damage to the tertiary associative zones of the left hemisphere is the development of autotopagnosia, bilateral finger agnosia, agraphia and alexia, acalculia, often in combination with dyspractical disorders [3,5].

Neuropsychological techniques are used in the diagnosis of PCI. The previously widely used KSHOPS is not sensitive enough to disturbances in frontal controls. functions, therefore it loses its position as a screening test to the Montreal Cognitive Assessment Scale (MoCa test). This technique includes tests to assess frontal dysfunction and is more sensitive to vascular diseases, including PCI [8].

The basis of post-stroke cognitive disorders and dementia may be the following types of brain damage: infarction of the strategic zone of the brain that is most closely related to the regulation of mental functions (angular gyrus, thalamus, limbic structures, frontal lobe, basal ganglia), multi-infarction condition, intracerebral hemorrhage, combination of infarctions, or hemorrhage with diffuse white matter damage, a combination of brain damage and Alzheimer's changes in the brain. Depending on the severity of the disorders, mild, moderate and severe cognitive disorders are distinguished [1].

Mild cognitive impairment, a subjective and/or objective decrease in cognitive abilities compared to an individual's baseline level, may be a consequence of natural age-related changes in the brain [15]. They usually manifest themselves as a slight slowdown in the implementation of neuropsychological techniques due to a decrease in concentration and stability of attention, as well as the speed of psychomotor reactions when studying neurodynamic functions.

Moderate cognitive impairment occurs in techniques that do not involve time limits. Typically, this category includes cognitive impairment when the difference compared to the age norm is at least 1–2 standard deviations. They may have a different neuropsychological profile and differ from dementia in the relative preservation of daily activities (with the exception of the most complex types), criticism, and variability

currents.

Severe cognitive impairment includes severe monofunctional disorders. In 12–15% of patients, moderate and severe cognitive impairment transforms into dementia [5].

Study of cognitive functions. Tests used in clinical neuropsychology in most cases describe one or more aspects of cognitive domains, which are theoretical constructs that involve several cognitive processes [3]. For example, a memory test that involves a series of memory words aims to measure verbal memory function as a component of the cognitive domain of memory. In this case, the test typically involves processes such as attention, information encoding, consolidation, storage and retrieval of information. The test is based on auditory perception and speech signal processing.

The broad classification of cognitive domains often used is as follows: learning and memory, executive function, attention and processing speed, praxis, motor function, language and perception. In addition to the above, intelligence is often considered a separate domain that reflects an individual's overall cognitive abilities based on the individual domains above [13, 15].

There are many methods and scales for assessing QoL. In general, they can be divided into two main groups depending on their specificity: universal (broad spectrum) and specific for a given disease. Regardless of the type of scales, each of them consists of various subscales (domains) that assess various aspects of the subjects' lives, such as motor activity, communication skills, emotional, social sphere, daily activities and others [4,5].

The advantages of specialized scales are their high accuracy and detailed assessment of various aspects of quality of life in stroke patients. The main disadvantage is the impossibility of conducting a comparative analysis with other diseases[11]. It is important in assessing the impact of a critical condition on cognitive functions to compare their level with the state before the injury; however, for obvious reasons, it is not possible to assess the initial state of cognitive functions in these patients using most test methods.

To do this, you can use the Cognitive Failures Questionnaire (CFQ), which allows you to retrogradely determine the state of the patient's cognitive functions before the onset of the disease or before the onset of a critical condition [9].

Thus, post-stroke cognitive impairment is a consequence of cerebrovascular diseases, reaching the level of dementia, significantly reduces the quality of life of patients, remaining one of the most important problems of theoretical and practical medicine, which is explained by their high prevalence and social significance.

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