

CLINICAL FEATURES OF COGNITIVE DISORDERS IN HYPERTONIC DISEASE

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Urolova Dilafuz

Urolov Shokhrukh Saydullaqul o'g'li

Tashkent State Dental Institute

Abstract

The article is devoted to one of the most pressing problems of clinical medicine - impairment of cognitive functions in hypertension. The paper presents modern data on the prevalence of cognitive impairments developing against the background of hypertension, and discusses in detail their pathogenesis and clinical features. The basic methods of neuropsychological research used in the diagnosis of cognitive impairment in hypertension are presented. The results of international clinical trials on nonspecific and specific therapy of cognitive impairment in hypertension are considered in detail.

Key words

hypertension, cognitive disorders, dementia, mnestic disorders

Introduction.

Hypertension (HD) is one of the most common and socially significant diseases in clinical medicine. According to epidemiological data, the prevalence of HD in the population is approximately 40%, and among the elderly it increases to 60-70%. According to the expert committee of the All-Russian Scientific Society of Cardiology, only 30-40% of hypertensive patients are aware of their disease and no more than 10% receive adequate antihypertensive therapy. Long-term uncontrolled HD leads to secondary damage to the heart and blood vessels, and almost all vessels of the body suffer - from the aorta to the capillaries. Secondary damage to the vascular bed, in turn, causes the development of a wide range of serious complications that threaten life and health, such as myocardial infarction, acute cerebrovascular accidents, both ischemic and hemorrhagic, kidney damage, etc. [2]

The brain belongs to the so-called "target organs" in hypertension. At the same time, various mechanisms underlie the damage to the brain. First, HD is an independent, independent and strong risk factor for ischemic and hemorrhagic stroke. The risk of developing non-stroke ("silent") cerebral infarctions is even higher in hypertension. According to a number of international studies, up to 25%

of elderly people with hypertension or other vascular diseases have at least one "silent" heart attack. Secondly, HD significantly increases the risk of the formation of diffuse lesions of the white matter of the brain (leukoaraiosis). The results of the LADIS (Leukoaraiosis And Disability in the Elderly) study indicate that HD appears as the second most important predictor of the development of leukoaraiosis after the age of patients. Thus, long-term uncontrolled hypertension in most cases leads to secondary brain lesions, which are based on a combination of cerebral infarctions (both with the clinical picture of stroke and "dumb" ones) and diffuse lesions of the white matter (leukoaraiosis). [5]

The clinical picture of brain damage as a target organ in hypertension is diverse and largely depends on the localization of acute cerebrovascular accidents. However, in almost all cases, disorders of higher cerebral functions develop. Cognitive impairment is the most common manifestation of brain damage in hypertension and, therefore, can be considered as an indicator of brain damage in this disease. Cognitive disorders in hypertension are based on both lacunar infarctions of localization typical of hypertension (thalamus, subcortical basal ganglia) and diffuse changes in the white matter.

Prevalence of cognitive impairment in hypertension. Data from numerous epidemiological studies indicate that uncontrolled hypertension is a reliable, strong and independent risk factor for cognitive disorders, including severe ones (dementia). Thus, L. Skoog et al. Based on the results of a 15-year follow-up of patients over 70 years old, they concluded that initially high blood pressure (BP) (180/100 mm Hg and above) significantly correlates with the risk of dementia. The Honolulu-Asia Aging Study, which involved more than 3,700 patients from the Southeast region of Asia, showed a statistical relationship between systolic blood pressure (SBP) levels in middle age and the risk of developing cognitive disorders later. At the same time, an increase in SBP for every 10 mm Hg. Art. increased the risk of developing cognitive disorders by 7-16%. [3] The negative effect of HD on cognitive function was also demonstrated in the Rotterdam and Gothenberg studies and other works [6]

Cognitive impairment occurs in at least 73.7% of patients with hypertension. Moreover, in the absence of a history of stroke, cognitive impairment in most cases is mild (46.7%). less often moderate (26.7%). A history of stroke increases the risk of developing more pronounced cognitive impairments:

in patients with hypertension and stroke, moderate cognitive impairment prevailed over mild ones (72.7% versus 18.2%). Severe cognitive impairment (dementia) was observed relatively rarely and only in stroke patients (9% of the

surveyed patient population). [4] The presence and severity of cognitive impairments in patients with hypertension did not always correspond to the presence of complaints of a cognitive nature, which indicates the need for an objective assessment of cognitive functions in patients with hypertension. At the same time, there was a significant correlation between the severity of cognitive impairments and the level of SBP. The age of the patients was another significant predictor of the development of more pronounced cognitive impairments. Risk factors for the development of more pronounced cognitive impairments are usually advanced age, stage II and III hypertension, low educational level and the presence of concomitant dyslipidemia. In addition, a significant correlation was found between the presence of cognitive impairments and hypertensive left ventricular hypertrophy. These data indirectly confirm the assumption that left ventricular hypertrophy can be considered as an indicator of the state of cerebral vessels and, therefore, as a predictor of cerebral vascular damage in hypertension.

Mechanisms of the formation of cognitive impairment in hypertension. Cognitive disorders in hypertension are most often of a vascular nature. Arterial hypertension leads to two types of vascular changes in the brain: lacunar infarctions and diffuse changes in the white matter. Both of these morphological features can cause disorders of higher cerebral functions. Due to the peculiarities of the blood supply to the brain, the typical localization of hypertensive lacunar infarctions is subcortical gray nodes (thalamus, striated bodies, etc.). It is in these departments that cerebral infarctions associated with hypertension develop most often and earlier.

It is known that subcortical gray nodes are in close functional connection with the frontal lobes of the brain. Therefore, vascular lesion of subcortical gray nodes naturally causes secondary dysfunction of the anterior parts of the brain. Diffuse lesion of the white matter of the brain also leads to dysfunction of the frontal lobes of the brain due to a violation of their connection with other cortical and subcortical cerebral structures and functional isolation (the so-called "disconnection phenomenon").

Thus, both acute disorders and chronic insufficiency of blood supply to the brain result in dysfunction of the anterior regions of the brain. Meanwhile, it is the frontal lobes that play a key role in the cognitive process and the formation of emotions. Therefore, cognitive, emotional-behavioral, and motor symptoms of frontal dysfunction are most often observed in the clinical picture with damage to the brain as a target organ in hypertension. Numerous studies have confirmed the statistical relationship between the severity of leukoariosis, the number of lacunar

infarctions and the severity of cognitive disorders of the frontal character in patients with hypertension.

Clinical features of cognitive impairment in hypertension. Vascular cognitive disorders, usually in combination with emotional-behavioral, motor and other neurological disorders, form the core of the clinical picture of discirculatory encephalopathy. Discirculatory encephalopathy is understood as a syndrome of cerebral lesions of vascular etiology, which manifests itself in various neurological, neuropsychological and mental disorders and is formed as a result of repeated acute disorders and/or chronic insufficiency of the blood supply to the brain. Synonyms for the term "discirculatory encephalopathy" can be considered the following: "chronic cerebral ischemia", "subcortical arteriolosclerotic leukoencephalopathy" (Binswanger's disease) and similar definitions. Due to the above pathogenetic features of the formation of cognitive disorders, the leading place in their structure is occupied by a violation of executive (frontal) functions. Managing functions (syn.: Regulatory, executive functions", from the English executive functions) can be decomposed into three components:

goal-setting: the ability to arbitrarily choose and set a goal for the activity. Goal-setting is considered a function of the most anterior parts of the frontal lobe (pole) and the cingulate gyrus. If this function is insufficient, the activity of mental processes, motivation and initiative decrease. emotional indifference develops;

> stability of attention: the ability to build one's cognitive activity and behavior in accordance with the set goal, as well as the ability to inhibit less significant or unacceptable motivations in the existing situation. This component of executive functions is associated with the orbitofrontal cortex. If this factor is insufficient, the patient's behavior becomes impulsive, he is often distracted from the planned activity plan, his criticism decreases;

switchability: the ability in changed conditions to change the paradigm of activity, to move from an already achieved goal to a new one. Switchability is a function of the dorsolateral frontal cortex. If this factor is insufficient, inertia and perseveration develop.

As a rule, in the structure of cognitive impairments accompanying hypertension, one or more of the above signs of insufficiency of executive (frontal) functions are present, and it is these disorders that develop earlier than other cognitive impairments.

The memory of patients with vascular cognitive impairment against the background of hypertension suffers to a mild or moderate degree. Disturbances mostly affect short-term memory, while memory for recent and distant life events is

relatively intact. An analysis of the neuropsychological characteristics of mnestic disorders indicates that they are based on insufficient reproduction, while memorizing and storing information is relatively intact. This is indicated by the effectiveness of semantic mediation and hints when testing the mnestic function. In the sphere of gnosis and praxis, violations of spatial functions can be determined. Constructive praxis suffers to a greater extent. However, it should be noted that constructive dyspraxia is more often defined at the stage of vascular dementia and is less typical for mild to moderate cognitive impairment.

Speech in "pure" vascular cognitive disorders in the absence of a history of strokes does not suffer. The presence of a concomitant neurodegenerative process modifies the picture of cognitive disorders in hypertension. The most specific feature of concomitant Alzheimer's disease is severe memory impairment. The qualitative difference between mnestic disorders in combined vascular-degenerative brain damage from "pure" cerebrovascular pathology is the signs of primary failure to memorize new information the so-called "hippocampal type of memory impairment", the neuropsychological characteristics of which are given in Table. 1. It is important to emphasize that these features of mnestic disorders are determined already at the early stages of the neurodegenerative process. Another specific feature of concomitant Alzheimer's disease is the formation of cortical

Significant difference between direct and delayed playback

▸ Inefficiency of semantic coding of memorization and hints during reproduction Impaired recognition of information in multiple choice trials

Interlacing of foreign material during playback dysphasic disorders. This symptom, in contrast to memory impairments, is not necessary for the diagnosis and in typical cases develops somewhat later. Dysphasic disorders begin with a lack of the nominative function of speech: the patient "forgets" the names of objects, first low-frequency, and then ordinary. Over time, a complete clinical picture of acoustic-mnestic (according to another classification - transcortical sensory) aphasia may form.

Dynamic observation of patients is of great importance. "Pure" vascular cognitive disorders are characterized by a relatively slow progression or may be stationary when adequate blood pressure control is achieved. Progression is usually stepwise: pronounced deterioration (due to acute disturbance of cerebral circulation) is replaced by a period of a steady state of varying duration. With an accompanying neurodegenerative process, the progression is smoother and does not depend on the control of hypertension. [9] However, the rate of progression can vary significantly: relatively long periods of stationary status in elderly and senile

patients do not completely rule out the diagnosis of concomitant Alzheimer's disease.

Diagnosis of cognitive impairment in hypertension. Considering that HD is one of the most significant risk factors for cognitive disorders in general, when working with patients with HD, there should be special medical alertness regarding disorders of higher cerebral functions. Cognitive complaints, information from relatives about cognitive problems in daily life, or the doctor's own impression of the patient's cognitive decline are the basis for neuropsychological evaluation.

Since the most characteristic cognitive symptom in patients with hypertension is impairment of executive (frontal) functions, tests sensitive to the cognitive function of the frontal lobes should be used to detect these impairments. These tests include:

test of literal associations (in 1 minute name as many words with the letter "C" as possible, normally at least 10);

test "Symbols and numbers" (within 90 seconds put the numbers corresponding to the sample in the empty squares on the test sheet);

test of communication of numbers and letters, part B (connect numbers and letters on the test sheet in order: 1-A-2-B, etc.).

In recent years, in everyday clinical practice, the Montreal Cognitive Assessment (MoCA) scale has found wide application, which contains a simplified version of the test for the connection between numbers and letters, a literal association test, a clock drawing test, other tests for attention and executive functions, and also on the assessment of memory, orientation, nominative function of speech, etc. The technique is relatively simple, takes no more than 10-15 minutes and is highly sensitive for detecting both vascular cognitive impairments and disorders of higher cerebral functions due to neurodegenerative process.

It should be emphasized that the presence of cognitive disorders in a patient with HD does not automatically mean a causal relationship between them and the underlying disease. Therefore, severe and/or progressive impairment of cognitive functions is an indication for neuroimaging: computed tomography or magnetic resonance imaging of the brain. The main goal of neuroimaging is to exclude other serious causes of progressive cognitive disorders, such as a brain tumor, other volumetric processes, normotensive hydrocephalus, etc. In addition, one should not forget about the possibility of secondary cognitive disorders associated with systemic dysmetabolic disorders.

Conclusion. Thus, HD is a strong and independent risk factor for the onset and progression of cognitive disorders, which can be of both vascular and neurodegenerative nature. Therefore, in all patients with this disease, close attention should be paid to the state of higher mental functions. The management of patients with cognitive impairment associated with hypertension should include the achievement of adequate control over blood pressure and the conduct of specific and neuroprotective therapy.

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