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NEURODEGENERATIVE CHANGES OBSERVED IN ARTERIAL HYPERTENSION AND ALZHEIMER'S DISEASE

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Abstract: The most common cause of dementia is Alzheimer's disease. But currently, the development of cognitive disorders is increasingly explained by mixed (vascular-neurodegenerative) brain damage. At a certain stage of the disease, the same amyloidogenesis, neurodegeneration and apoptosis mechanisms as Alzheimer's disease were found to be involved in the development of vascular dementia. According to pathomorphological studies, about a third of patients with dementia have signs of vascular and neurodegenerative damage, which is explained by the totality of risk factors. One of the most important risk factors for brain substance damage among middle-aged and elderly patients is arterial hypertension.

Key words: Dementia, Alzheimer's disease, vascular-neurodegenerative disorder, arterial hypertension.

Introduction. Impaired cognitive function and the development of dementia are an urgent problem not only in neurological, but also in therapeutic practice. In recent years, there has been a steady increase in the prevalence of cerebrovascular pathology, as well as neurodegenerative diseases. According to the World Health Organization (WHO), there are now about 47 million people with dementia worldwide, with numbers reaching 75 million by 2030 and nearly tripling by 2050.

According to the WHO, in 2014, arterial hypertension was more than 22% in those over the age of 18. arterial hypertension is the most important factor that determines a decrease in life expectancy and disability of the elderly population. Arterial hypertension, one of the main risk factors for cerebrovascular disease, determines a high level of vascular dementia development. However, there is a relationship between arterial hypertension and the level of cognitive decline in the absence of vascular damage to the brain, which has an acute and chronic nature. In this case, we are concerned with the neurodegenerative nature of cognitive deficits and, in most cases, Alzheimer's disease.

Some studies suggest that risk factors for cardiovascular disease such as hypertension, Type 2 diabetes, dyslipidemia, metabolic syndrome, sporadic development are associated with Alzheimer's disease [1-3]. Among these risk factors, the most important is arterial hypertension, which contributes to the cognitive decline and development of not only vascular dementia, but also Alzheimer's disease. Arterial hypertension has been found to cause dysfunction of the microvascular duct, leading to hypoperfusion of the brain, the development of micro - hemorrhage, and pathology of the cerebral arteries. Small vascular disease can lead

to lacunar infarcts and changes that are closely related to the deterioration of cognitive functions that lead to leukoareosis and vascular dementia [4]. It is known that the roots of the main pathophysiological processes lie deep at a young age, such as Alzheimer's disease, amyloidosis and neurodegeneration. In turn, the presence of cardiovascular risk factors for 10 years in middle age is a strong predictor of the development of cognitive impairment, which reaches the level of dementia [5]. Such patients are at high risk not only for the development of cardiovascular disasters, but also for the subsequent development of dementia [6]. However, unlike the mechanisms that bind hypertension and cerebrovascular diseases, the pathogenetic relationship between hypertension and Alzheimer's disease is more complex and probably involves the development of neuronal inflammation, conduction disorders of the blood-brain barrier, and cerebral hypoperfusion [7-9]. An increase in blood pressure has been found to impair the permeability of the blood-brain barrier, leading to the accumulation of neurotoxic molecules in the brain, including amyloid beta [7]. In chronic hypoperfusion of the brain, hyperphosphorylated taubelc levels in the hippocampus and cerebral cortex may increase [8] and progressive accumulation of amyloid beta (AB) [9, 10]. In addition, arterial hypertension affects the integrity of the wall of the cerebral arteries, which leads to a decrease in hypoperfusion and glucose metabolism and promotes the development of Alzheimer's disease [11].

An observation of more than 10,000 women over 65 years of age over 7 years has shown that arterial hypertension is an independent risk factor for the development of cognitive impairment [12]. Thus, an increase in blood pressure by only 10 mm HG. increases the risk of vascular cognitive impairment by 40%, especially in older patients [13, 14]. However, an increase in diastolic blood pressure is the strongest risk factor for the development of cognitive disorders, regardless of age and gender, but timely prescribing antihypertensive therapy can significantly reduce the risk of their development [15]. The Framingham study observed 1,695 elderly patients for 12-15 years with arterial hypertension at 55-88 years of age. As a result, the duration of arterial hypertension, as well as the level of systolic and diastolic blood pressure, were established a statistically significant negative inverse relationship with visual and auditory memory indicators based on the results of neuropsychological tests.

Although the exact mechanisms are not entirely clear, there is strong evidence to support the hypothesis that hypertension in old age contributes to the development of Alzheimer's disease [16]. Thus, the study [17] showed that the presence of hypertension worsens cognitive function in patients for 6 months Alzheimer's disease up to 65 years of age. The study [18] found that mental state assessment short scale (MMSE) rates in high systolic patients were significantly lower.blood pressure. The study [19] showed arterial hypertension in patients with Alzheimer's disease associated with decreased cognitive function and decreased glucose metabolism in the hippocampus.

Despite the importance of acute vascular phenomena in the genesis of cognitive disorders, the most common cause of their development is a change in subcortical white matter caused by damage to brain vessels, especially small diameter.arterial hypertension. Uncontrolled due to arterial hypertension, which often occurs with crises, changes in the vascular wall of the microcirculation channel, such as lipogialinosis, stenosis or obliteration, occur as a result of plasmoy or bleeding into the walls of blood vessels, sometimes with the accompanying trom-bose. An increase in the density of the vascular wall and, accordingly, narrowing of the vascular Lumen (arteriosclerosis) leads to a change in the physiological reactivity of the vessels.

Today, most researchers consider changes in white matter and Lacuna, which are often found in older people, to be the main MRI symptoms of minor vascular diseases. Taking into account the totality of etiology, the pathogenesis of the formation of lacunae and leukoareosis, today multi — infarcted lacunar dementia and arteriolosclerosis leukoencephalopathy (Binsvanger's disease) are combined into a single form-subcortical ischemic vascular dementia. Another sign of minor Vascular Pathology is micro-hemorrhages, the definition of which became available with the improvement of magnetic resonance

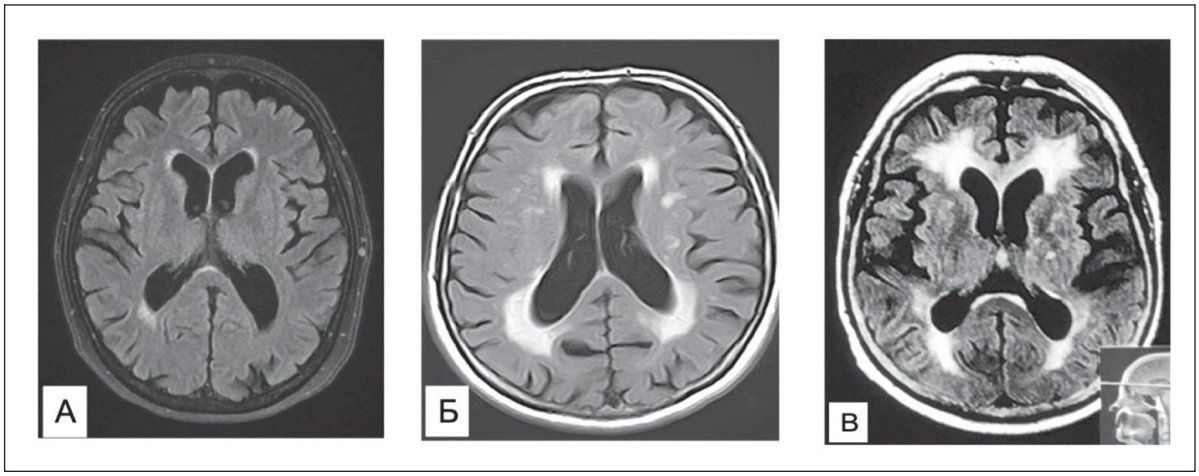
imaging (MRI). Micro hemorrhages are often localized in the gray matter of the cerebral hemispheres and subcortical ganglia and are associated with regulatory dysfunction.

When evaluating MRI, damage to white matter has a" typical "picture that manifests itself with an increase in signal intensity in T2-weighted images and is known as"leukoareosis". The periventricular zone of the brain substance is usually considered as a terminal blood supply zone, which determines its sensitivity to high blood pressure and its excessive decrease [20].

Leukoareosis often occurs in patients with vascular risk factors, including arterial hypertension or already existing cerebrovascular pathology [21]. A study using an amyloid-binding ligand on positron emission tomography results found that the severity of ligand accumulation was associated with the severity of leukoareosis in patients with amyloid angiopathy of the brain (caas), supporting the hypothesis that amyloid beta accumulation worsens or even damages the white matter of the brain, but in patients. disease Alzheimer's this connection is not confirmed [22]. The pathogenic effect of amyloid on the mechanisms of development of leukoareosis, in turn, is confirmed by the Association of Alzheimer's disease and cognitive disorders in the CAA and the severity of white matter changes with serum levels of peptide AB1–40 (the main component of amyloid vascular deposits in the CAA) [23].

To determine the severity of changes in white matter, it is recommended to use the Fazekas scale, which allows you to assess the damage to white matter (it is better to use the FLAIR mode). Assessment on a scale includes visual interpretation and scores from 0 to 3, where 0 is missing leukoareosis, 1 is a multi — point lesion, 2 is moderate Fusion leukoareosis, 3 is severe "drainage" leukoareosis (fig. 1).

Figure 1. Assessment of brain white matter damage on the phasecas scale.



А. minimal damage to white matter is "mild" leukoareosis (Fazekas = 1).

Б. intermediate Fusion leukoareosis (Fazekas = 2).

В. clear "drainage" leukoareosis (Fazekas = 3)

To date, the importance of stroke in the development of dementia is indisputable, but there are many questions regarding the patterns and conditions of the emergence of cognitive deficit. A number of researchers have found that post-stroke dementia is associated with the localization of lesion in the dominant hemisphere, with cerebrovascular pathology in the form of leukoareosis and lacunae, with the magnitude of pre-stroke focus. Today, many scientists attach fundamental importance to the localization of damage, arguing that it is associated with strategic zones, that is, zones associated with the provision of cognitive activity[27-30].

In this regard, important zones for the development of vascular cognitive disorders were identified, which are called "strategic". These small heart attacks are also areas that are particularly important for mnestico-intellectual dysfunction. According to the criteria of Ninds-AIREN (the National Institute of Neurological Disorders and Stroke, Association Internationale pour la Recherche et l'enseignement en Neurosciences), the following localization of damaged vessels and infarcts is important for the formation of strategic dementia: paramedian thalamic arteries Basin, lower medial temporal cortex, parietal and parietal occipital associative regions and angular gyrus, anterior cerebral arteries (bilateral), upper frontal and parietal watershed, because they contain a dominant hemisphere. In the pathomorphological studies conducted, the sensitivity of these criteria was 55%, and the specificity was 84%. Other recognized strategically important areas include the caudate nucleus, pale ball, and hippocampus [29].

Recent studies have shown that night blood pressure levels themselves have a higher prognostic value than daily blood pressure at risk for cardiovascular disease. The common belief of patients Alzheimer's disease in most cases does not have a hypertensive history. At the same time, among patients with Alzheimer's disease, the number of people with deviations in daily blood pressure indicators is very high.

Patients with known arterial hypertension with a violation of the circadian profile of blood pressure, there is a high risk of developing an early atherosclerotic lesion in the extracranial part of the carotid arteries, regardless of the type of change in the Daily Index. In addition, more than 50% of patients have been diagnosed with Alzheimer's disease, which has normal blood pressure indicators during the Daily measurement cycle, is characterized by insufficient pressure drops at night ("non-dipper") and 16.2% absence at all.or night fall ("reverse-dipper").

"Vascular" risk factors (arterial hypertension, hypergomosis - steinemia, diabetes mellitus, smoking, hyperlipidemia) lead to atherosclerosis of large vessels, a change in the elasticotonic properties of small arteries, which in turn disrupts the process of AB elimination, contributes to its deposition, including compensatory expansion of arterial walls and perivascular spaces, and, therefore, hypoperfusion of the brain, which enhances the development of Alzheimer's disease.

The study [31] found a significant violation of the circadian profile of hypertension blood pressure without Alzheimer's disease shown in patients. Thus, patients with Alzheimer's had higher levels of nocturnal systolic blood pressure than other groups. This was shown by [33] middle-aged people with systolic blood pressure above 160 mm HG. the risk of developing Alzheimer's disease in old age compared to people with normal blood pressure.

A sufficient drop in blood pressure at night ("non-dipper") is a strong independent risk factor for cardiovascular disease [36] and is associated with an increased risk of acute cerebrovascular events in older people [34, 38]. Because people are exposed to high levels of blood pressure at night when they are horizontally on their back, the brain is less protected from hydrostatic forces and the cerebral vessels are exposed to high pathological pulsating flow [39]. Stable growth of pulsating current then damages the vessels of the microcirculation channel, promotes the development of lacunar infarcts and damage to the white matter of the brain (leukoareosis) [40].

The purpose of the study is to study the Daily profile of blood pressure in patients with Alzheimer's disease, their cognitive functions and the level of B-amyloid protein in liquor and to establish a possible pathogenetic relationship between these indicators.

Materials and methods. In a comparative study we conducted, various parameters of daily blood pressure monitoring according to the Korotkov method with an oscillometry supplement were evaluated in 60 patients. Alzheimer's disease and 20 comparison groups (with discirculatory encephalopathy), extensive neuropsychological tests were carried out, a lumbar puncture was performed with the determination of the concentration of the amyloid protein Aß-42.cerebrospinal fluid is a solid-phase enzyme by the immunoassay

method. Non–parametric statistical methods (the Mann-Whitney criterion for two independent samples) were used to analyze the results, with correlation correlations estimated using Spearman-level correlation.

Results and their discussion.

Analysis of daily monitoring data showed generally high blood pressure among patients in the comparison group — patients with dysirculatory encephalopathy. Such results are fully consistent with the fact that arterial hypertension is the main risk factor for chronic cerebral ischemia.

The average daily systolic blood pressure in Alzheimer's patients was much lower than the comparison group (135,1 ± 16,8 and 148,5 ± 17,5 mm column of mercury). in patients in the comparison group, $p < 0,05$), at night, on the contrary, Alzheimer's disease had a high average blood pressure (141,1 ± 22,4 mmHg on average). (127,5 ± 19,6 mmHg), as opposed to patients in the comparison group (127,5 ± 19,6 mmHg). , $p < 0,05$). That is, patients with Alzheimer's disease without clinical signs and clinical diagnosis arterial hypertension, which in most cases did not receive antihypertensive therapy, was often characterized by nocturnal arterial hypertension.

However, in our opinion, the most important were the differences in diastolic blood pressure indicators at night. Thus, in patients with Alzheimer's disease, diastolic blood pressure fluctuated between 77,6 ± 17.4 mm Hg., while in cerebrovascular pathology The Mercury column is 69,3 ± 11,5 mm. ($p < 0,05$). Thus, high diastolic blood pressure rates led to a decrease in pulse blood pressure at night, which could also detect peri - vascular transport disorders.

Table 1. Changes in nighttime blood pressure in Alzheimer's disease (percentage of observations)

	There is no decrease in blood pressure	Insufficient reduction of blood pressure (< 10 %)	Adequate reduction of blood pressure (10–20 %)	Excessive decrease in blood pressure (> 20 %)
Alzheimer's disease	17,6	47,2	29,4	5,8
Dyscirculatory encephalopathy	8,3	33,4	58,3	0

In the groups of patients examined, we analyzed the decrease profile of blood pressure (table.1).

For patients with Alzheimer's at night, a sufficient decrease in blood pressure was not characteristic, despite the lesser importance of arterial hypertension during the day and during the day. In addition, 64.8% of patients with Alzheimer's disease have been characterized by insufficient blood pressure or an increase in blood pressure at night in general.

The functional relationship between the effective functioning of the perivascular-glymphatic system of the brain at night and changes in blood pressure during sleep can be important for the development of neurodegenerative disease. Therefore, for further analysis, patients were divided into 2 groups: with a sufficient night fall, with blood pressure (10-20 %) and with a sufficient decrease or increase (< 10 %). The results of protein studies were compared with Alzheimer's disease in patients with cerebrospinal fluid with different profiles for night changes in blood pressure.

In the group of patients with Alzheimer's disease with a sufficient decrease in blood pressure or an increase at night, the values of aß-42 are lower in the cerebrospinal fluid (391,8 ± 59,1 pg/ml), and at night with a sufficient decrease in blood pressure (10-20%), the concentration of pathological amyloid protein in the cerebrospinal fluid is significantly higher (801,2 ± 130,5 pg / ml, $p < 0,05$). It should be noted that the level of free AB is inversely proportional to the amyloid load of the cerebrospinal fluid, that is, the amount and density of amyloid plaque in the cerebral parenchyma. A decrease in free AB concentration may indicate

less intensive B-amyloid transport through the perivascular/glymphatic transport system. That is, a sufficient decrease in blood pressure at night is most likely due to a decrease in AB clearance of cerebrospinal fluid and, therefore, its accumulation in the brain.

A possible confirmation of such an effect is L. B. Hoffman and co-authors (2009) were obtained in a study by [40] in which they were shown in a group of deceased people.Alzheimer's disease, which has been adequately treated for hypertension, has been found to have significantly fewer amyloid plates and neurofibrillary tangles, the main neuropathological signs of Alzheimer's disease.

A correlation analysis conducted showed that in patients with Alzheimer's disease, indicators such as night diastolic blood pressure and pulse blood pressure have the most important effect on the state of cognitive functions. In particular, high diastolic rates were associated with a decrease in blood pressure free at night and associated selective recognition scale (FCSRT) indicators — a universal tool for assessing short-term oral memory (R = -0,81). Higher pulse blood pressure values were, in contrast, correlated with less time to perform the follow-up test (Part A) (r = 0,79). Since the above methods are designed to assess Mnestic and regulatory functions, it can be assumed that the negative effects of the hemodynamic factor on certain cognitive functions are not specific.

In recent years, the mechanisms of neuroprotective action of slow calcium channel blockers in relation to the central nervous system have been studied in detail. In addition to its antihypertensive and vasodilator effects, slow calcium channel blockers penetrate the blood-brain barrier and stimulate the functioning of neurotransmitters that inhibit the formation of B-amyloid, reducing apoptosis processes and intracellular calcium concentrations that stimulate the formation of free radicals. In addition, slow calcium channel blockers of the dihydropyridine series can reduce the thickness of the intima-media complex and improve the endothelial function of blood vessels due to the specific atherosclerotic effect. In this regard, the effect of slow calcium channel blockers on AB clearance through the blood-brain barrier is of particular interest.

Analysis of the results of clinical studies has shown that the most suitable therapy for arterial hypertension to prevent the development of cognitive impairment and dementia is a combination of sartan and calcium antagonist (amlodipine).

Thus, the effect of hypertension on cognitive disorders is mediated by various mechanisms. Therefore, timely and adequate prescribing of antihypertensive therapy not only prevents the development of cognitive disorders in patients or even improves cognitive function to a certain extent. The choice of antihypertensive agents requires an individual approach, taking into account the presence of the corresponding comorbid pathology, and also excludes an excessive drop in blood pressure due to the development of hypoperfusion brain complications, including increased cognitive impairment.

Conclusion. Thus, our study shows that the vast majority of patients with Alzheimer's disease, no arterial hypertension and, accordingly, do not receive, or antihypertensive therapy, the absence or adequate decrease in blood pressure at night is characterized by a decrease in pulse blood pressure, mainly due to the diastolic component. This, in turn, is accompanied by less intensive Aß clearance from brain tissue, reflecting a lower concentration of free Aß - 42.cerebrospinal fluid. Therefore, a sufficient decrease in blood pressure at night or nocturnal arterial hypertension creates the necessary conditions for low perivascular-glymphatic transport, accumulation of AB, neuronal inflammation, the onset of the tau hyperphosphorylation Cascade and the formation of neurofibrillary tangles, the destruction of the neural cytoskeleton, the development of neurodegeneration. Patients with cognitive impairment of any etiology, and the study showed that Alzheimer's disease in particular requires mandatory daily monitoring of blood pressure.

In this regard, it is of particular importance to correct such diseases of young and middle age before changes in night blood pressure and, accordingly, clinical manifestations of dementia. It is to such patients that it is indicated to prescribe antihypertensive drugs - sartans and calcium channel blockers, as well as normalize

night sleep, exclude insomnia, and in this regard, it is of particular importance to correct such diseases of young and middle age before the clinical manifestation of nocturnal blood pressure and, accordingly, dementia. It was to such patients that antihypertensive drugs - sartans and caldi.

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