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Morphofunctional Foundations of the Development of Vascular Cognitive and Emotional Disorders

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Abstract: Many studies have identified various risk factors and mechanisms that cause structural and functional changes in the brain in cerebral vascular disease (CVD). At the same time, the leading pathogenetic joint in the development of clinical signs of these syndromes remains ambiguous. Due to the frequent combination of dementia and depression in patients with cerebrovascular diseases, the term "2D syndrome" has been proposed to simplify their co-designation. It is known that the most common cause of dementia is Alzheimer's disease. "Pure" forms of Alzheimer's disease associated with increased genetically determined amyloid levels are rare, with most cases occurring sporadically, and are caused by typical changes in clearance mechanisms in the brain and amyloid beta accumulation.

Key words: emotional disorders, cognitive disorders, arterial hypertension, cerebrovascular reactivity.

Introduction. One of the approximate causes of these disorders are cardiovascular risk factors, mainly arterial hypertension (ah) [1]. The pathogenesis of the neurodegenerative process and understanding of the general risk factors and individual links of cerebrovascular disease have led to the emergence of the theory of the mixed nature of dementia, which is now widely used and supported by most researchers [2-6]. Like vascular cognitive impairment (SCN), mood disorders in cerebrovascular disease are organic in nature and are associated with vascular risk factors, leading to activation of the sympathetic-adrenal system and depression-based glucocorticoid activation [7-10]. However, a number of functional and structural changes occur in the brain under the influence of high levels of glucocorticoids in depression, such as reorganization of neurotrophic factors, in particular a decrease in BDNF levels [11-13], as well as hippocampus, olfactory and frontal cortex atrophy, tonsils [14-19], which leads to cognitive decline. Thus, the relationship between vascular, neurodegenerative processes and the development of dementia and depression has been well proven. Nevertheless, the search for the causes of the development of "2D syndrome" in cerebrovascular diseases continues. Recently, the role of the brain's own lymphatic system as a "drain" to eliminate biochemical "garbage" has helped shed light on the nature of neurodegenerative diseases [20-28].

The harmful role of B-immune cells in the development of inflammation SKN, in particular a potential therapeutic target for the Prevention of dementia, has also been confirmed [29-34]. In recent years, neuroanatomic and functional correlations of dementia and depression in cerebrovascular diseases have also been actively studied [35-39]. The localization and size of structural damage to the brain substance in cerebrovascular diseases are important in the development of SCN and depression, but the decisive role here concerns functional changes in the brain [39-41].

Thus, cerebrovascular diseases and the neurodegenerative process form a vicious circle, the main branches of which are disorders of microvascular reactivity, ischemia, inflammation, accumulation of beta-amyloid, which in turn increase the violation of microvascular reactivity [42-50].

Showed a decrease in cerebrovascular reactivity in healthy young apoe4 genotype carriers compared to non-impaired, which may reflect vascular contribution to late-life cognitive impairment in apoe4 carriers. Thus, cerebrovascular reactivity disorder (CVD) may be a major offshoot of pathogenesis and the earliest predictor of impending "2D syndrome" [51-58].

The purpose of this study was to study the state of cerebral vascular reactivity and structural changes in brain

matter in patients with moderate cognitive impairment (SOPB).

Materials and methods. The study includes 385 patients with SOPB between the ages of 57 and 79, diagnosed on neuropsychological examination and based on NIA-AA criteria (National Institute of aging and Alzheimer's Association criteria). Diagnosis of dementia was a criterion for exclusion from research. The following measures are used to assess cognitive function: mental state assessment short scale (KSHOPS), frontal dysfunction detection tests (ld), 16 – word memorization test (free and Cued Selective reminder Test-Immediate Recall, FCSRT-IR), clock drawing test, Shulte tables. Functional capabilities of patients M. Lawton and E. Brody (1969) was evaluated with an informant (a relative or a person who knows the patient well) using the daily activity scale. The emotional state of patients was studied using the Hamilton-21 scale (SHG21), Hospital Anxiety and depression scale (GSTD), taking into account the subjective and objective assessment of the patient's emotional state. The CSDD scale (Cornel scale for depression in dementia) was used to identify depression in patients with advanced dementia in the long follow-up phase and to eliminate diagnostic errors associated with the apathy present in them.

Magnetic resonance imaging (MRI) (1.5 TL) of the brain was performed in T1, t2, FLAIR, DWI modes using MRI angiography on all patients to assess structural changes in brain matter. The change in white matter was assessed using the Fazekas and arwmc (Age-Related White Matter Changes) visual scales, reflecting age-related changes. The brain substance is divided into periventricular Parts (1 cm from the lateral ventricles) and deep white matter according to the Phasecas scale. On the visual scale, ARWMC evaluated 5 brain regions in each hemisphere: frontal region to Central gyrus; parietal occipital region; temporal region; cerebellum and brainstem; basal ganglia.

The main starting points for the diagnosis of SKN in the inclusion of patients in the study were: current vascular disease (mainly arterial hypertension and atherosclerosis) lasting at least 5 years, with a well-defined clinical picture and neuroimaging data confirming several vascular brain injuries (≥3 points on the phasecas scale and ≥6 points on the scale). ARWMC) without a clear atrophy of the brain substance.

Transcranial duplex scans were performed on all patients to assess the condition of cerebrovascular reactivity. In the Vertebrobasilar Basin (VBB), the state of cerebrovascular reactivity was assessed using a functional load test (photostimulation using a stroboscopic lamp), calculating the photoreactivity index on the dynamics of blood flow rate indicators in the spinal artery, in the carotid artery system (sa) – hypercapnik. holding the breath to 30 C. Since antihypertensive drugs are able to alter cerebrovascular reactivity indicators by modulating arterial stiffness, all patients were in stable antihypertensive therapy during the entire follow-up period (20 months) at least 30 days before and after the start of the study.

The results were processed using Microsoft Excel 7,0 and statistics software with a statistical method of comparing patient groups in pairs. In a simple distribution, a comparative analysis between groups was carried out using a student criterion. The differences were considered reliable at $p<0,05$. The average and standard deviation, as well as the average error, were calculated. Without a simple distribution, the medians, 1 and 3 quartiles, were calculated. The comparison between the groups was made using the Mann– Whitney criterion.

Results and discussions. In neuroimaging testing in all patients, brain substance damage on the Phasecas scale ranged from 3 to 5 points, and from 6 to 16 points on the ARWMC scale.

Based on the results of the analysis of the neuropsychological profile of SOPB, patients are distributed as follows: 45% of them have a memory impaired multifunctional SOPB type, 32% have a memory intact multifunctional SOPB type, 16% have a monofunctional amnestic type and 7% have a monofunctional non-amnestic SOPB type. Primary attention disorders have been noted in patients with a multifunctional non-amnestic type of SOPB, as well as processes of generalization and conceptualization, difficulties in sentencing, and patients with a monofunctional non-amnestic type of sopb. At the same time, in patients M. Lawton and E. There was no significant decrease in daily activity on the Brody scale: primary activity-23.0±1.0 points (maximum-24 points); instrumental activity-19±1.0 points (maximum 21 points), which excludes the presence of dementia.

At the beginning of the observation, depression syndrome was found in 45% of those who were mainly examined with multiple and monofunctional type of amnestic SOPB.

Subclinical cases of depression have been mostly reported, rarely mild depression. Cases of severe depression have not been identified.

The correlation of the neuropsychological profile of SOPB and the localization of structural damage to the brain substance were analyzed. In Mono - and multifunctional amnestic sopb - type patients, ischemic foci are mostly localized in the parietal-occipital, temporal, frontal regions, and in non-amnestic SOPB-type patients in the basal ganglia and trunk, as opposed to mono-imultifunctional non-amnestic sopb-type patients.

However, statistical differences are only obtained in patients with a large volume of ischemic injury: ≥5 points on the fazekas scale and ≥10 points on the arwmc scale. The clinical picture of depression was reliably correlated with the localization of vascular foci in frontal areas on the phasecas scale, 4 and 5 points on the arwmc scale ≥10 points (57 and 54% of depression cases, respectively; p=0.02).

Assessment of the status of cerebrovascular reactivity in various vascular basins found a decrease in rates in all patients, with 32% of those examined showing a decrease in cerebrovascular reactivity in the carotid arteries, 24% – only in the vertebrobazi Basin, and 44% – simultaneously in the carotid arteries and in the vertebrobazi Basin. When analyzing the prevalence of different clinical variants of SOPB syndrome depending on the state of cerebrovascular reactivity in different vascular basins, multiple and monofunctional amnestic SOPB types are more common in patients with decreased cerebrovascular reactivity in carotid arteries (39% of those examined; p=0.04), mainly in carotid arteries and in the basin of vertebrobases (49%).; p=0.03). The multifunctional non-amnestic type of SOPB is often found in the sleeping arteries and in the vertebrobasilar Basin (51%; p\u003d 0.02) and monofunctional non – amnestic SOPB type-with decreased cerebrovascular reactivity in VBB (41%; p \ u003d 0.03).

The highest number of cases of depression were reported in patients with decreased cerebrovascular reactivity in the sleep arteries (54%; p=0.02), especially in the left, as well as in the basin of the sleep arteries and vertebrobases (39%; p=0.04).

After 20 months of analysis, there were 360 patients (25 people left the study), which did not affect the representation of the data. In 48% of patients, a repeated neuropsychological examination revealed mild dementia syndrome. The key to diagnosing dementia was the effect of cognitive dysfunction on the daily activities of patients.

In complex activities that are reflected in professional or daily activities that require environmental advice (M. Lawton and E. Instrumental activity on the Brody scale) the presence of regular difficulties made it possible to diagnose dementia.

At the same time, patients retained relative independence and did not require constant monitoring. In the group of patients with dementia, the average score on KSHOPS was relatively high – 24.6±2.2, on the ld scale-13.3±1.3. Moderate to severe dementia has not been identified.

Most patients with dementia (72%) were diagnosed with multifunctional amnestic SOPB at the beginning of the study. According to the neuropsychological profile, all patients with dementia are divided into mainly dysregulatory and mainly amnestic types of SOPB (67 and 33%, respectively). The correlation analysis conducted did not identify statistically significant correlations between the clinical variant of dementia and the localization of ischemic injury on the fazekas and ARWMC (p>0.05) scale.

Subsequent analysis showed that 52% of patients with dementia had an initial decrease in cerebrovascular reactivity in both vascular basins, 31% had an initial decrease in cerebrovascular reactivity in the carotid arteries, and 17% had a decrease in cerebrovascular reactivity in the vertebrobasilar Basin (p<0.05). At the same time, patients with an initial decrease in cerebrovascular reactivity in carotid arteries and both vascular basins had more pronounced memory impairment than patients with an initial decrease in cerebrovascular reactivity in the vertebrobazi Basin (total increase on the fcsrt-IR scale was 18±4.3 and 31±2.5 points respectively; p=0.02). Thus, the ratio of clinical variants of cognitive impairment and perfusion decline was maintained in a particular vascular Basin observed at the beginning of the study.

At a long stage of observation, the number of cases of depression did not increase significantly – 48% (p \ u003E compared to the initial level of 0.05), which can be explained by an increase in cognitive impairment and a decrease in the criticality of perception. However, the dynamics revealed a significant increase in the severity of depression. The highest number of cases of depression were reported in patients with decreased cerebrovascular reactivity in the carotid arteries, as at the beginning of the follow-up (52%; p=0.02).

The results of the study made it possible to determine the dependence of the clinical characteristics of SKN on the localization of vascular foci. At the same time, the most unfavorable type of multifunctional amnestic SOPB in terms of the risk of becoming dementia was associated with the preferential localization of ischemic foci in strategic zones for cognitive functions:

frontal, parietal, temporal. However, reliable differences were obtained only in patients with large amounts of ischemic injury to the brain substance, which makes it impossible to use neuroimaging data for early diagnosis of the risk of developing severe SCN. The earliest prognostic sign may be a condition of cerebrovascular reactivity in various vascular basins. This study found that the development of clinical variants of SKN depends on the condition of cerebrovascular reactivity in different vascular basins. Since cerebrovascular reactivity is a direct reflection of the state

of cerebral perfusion and functional compensatory reserve, such dependence probably indicates the malfunctioning of neurovascular units and the phenomenon of hyperemia that works in certain areas of the cognitively important brain. At the same time, a decrease in perfusion characteristics in the carotid arteries supplying the temporoparietal regions and hypothalamus is mainly accompanied by the appearance of amnestic cognitive disorders, and a decrease in simultaneous perfusion indicators in the carotid arteries and vertebrobasis Basin, reflects more significant blood supply disorders of basal nuclei and subcortical structures, and is associated with the development of cognitive disorders such thus, the disregulator type.

Terminal portions of arteries in the Vertebrobasilar Basin are known to supply blood to the posterior parts of the hypothalamus, so the cerebrovascular reactivity state in VBB may play a buffer role in developing an amnestic type of cognitive impairment. However, impaired ability to maintain active mental activity and attention associated with decreased cerebrovascular reactivity in the vertebrobasilar basin can be explained by decreased perfusion of root structures.

In this study, the highest percentage of dementia was developed in multifunctional amnestic SOPB-type patients, which confirms literature data on the highest risk of the sopb variant becoming dementia, and shows a large contribution of the neurodegenerative process to the genesis of cognitive impairment in cerebral vascular disease. This study initially included patients with a high probability of a leading cerebrovascular pathological process.

At the same time, patient monitoring and neuropsychological examination data have shown a high probability of combining SCN with neurodegeneration, reiterating the difficulty of differentiating and identifying "pure" clinical forms of the disease. Of course, the neurodegenerative process is the main reason for the development of cognitive disorders, but vascular changes contribute, firstly, to their early debut, secondly, to the formation and dominance of a dysregulatory defect at the initial stage of the disease, and thirdly, to an acceleration of the pace of cognitive decline (compared to 2-4 points "Clean"per year on SCN form-0.5-1.0 points per year and "pure" Alzheimer's disease-3 points per year, according to the literature [45]).

Therefore, for more selective and successful therapy, it is necessary to take into account the possibility of combining SCN with a neurodegenerative process and conduct a comprehensive examination, taking into account the neuropsychological profile of the cognitive impairment, the patient's emotional state, neuroimaging examination data, in addition, the assessment muhimdir.va the state of cerebrovascular reactivity.

Earlier work shows the role of decreased vasodilator Reserve and linear blood flow rate in the development of cognitive disorders, as well as the importance of cerebrovascular reactivity as an early sign of cognitive decline. However, the dependence of SKN clinical variants on cerebrovascular reactivity state in various vascular basins has not been studied. Another study noted the need to study the APO E4 genotype in all patients with cerebrovascular diseases to prevent the development of severe cognitive disorders, as well as differential transfer of different functional MRI in the absence of specific clinical-neuroimaging correlations with systemic MRI data [31, 34]. The results of this study suggest that functional changes in the cerebral perfusion Reserve as measured by the cerebrovascular reactivity condition precede morphological changes in the brain substance and may serve as an early prognostic sign of the development of cognitive disorders in cerebrovascular diseases, including neurodegeneration. The study of cerebrovascular reactivity in this case is a simpler and cheaper method that can be applied at the first stage of screening patients.

A relationship between the clinical picture of depression and preferential localization of ischemic foci in the frontal region was found in patients with high levels of ischemic injury. However, mainly in patients with cerebrovascular diseases, the development of depression is associated with a decrease in cerebrovascular reactivity in the carotid arteries, especially in the left, and a concomitant decrease in cerebrovascular reactivity in the carotid arteries and in the vertebrobasilar Basin, supporting literature on the Association of depression with damage to the left hemisphere [49], as well as depression and cognitive disorders

Conclusion. The frequency of development of depression has been found to be associated with localization of ischemic injury in the frontal lobes and decreased cerebrovascular reactivity in the left carotid Basin, as well as a concomitant decrease in cerebrovascular reactivity in the carotid Basin and vertebrobasilar basin, with impaired perfusion in the vertebrobasilar Basin playing a buffer role. The neuropsychological profile of cognitive disorders in Cerebrovascular Diseases has been found to depend on decreased cerebrovascular reactivity in a given vascular Basin, which may reflect the dominant effects of a vascular or neurodegenerative process and serve as a basis for differential therapy.

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