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# NEUROIMAGING ASPECTS OF VASCULAR DEMENTIA

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**Abstract:** The aim of our study was to study structure features of the brain in the vascular dementia. Neuroimaging was studied using magnetic resonance imaging. As a result of patients, it was found that differences in tomography of morphological cerebral changes in patients with various types of vascular dementia correlated with cognitive and neurological parameters of patients.

**Key words:** neuroimaging, magnetic resonance imaging, cortical vascular dementia, subcortical vascular dementia, leukoaraiosis.

**Relevance.** Recently, the problem of vascular dementia (VD) occupies one of the first places among the causes of irreversible disability and mortality of patients. Along with the growing number of cardiovascular diseases and strokes, the incidence of vascular dementia is increasing [2-5]. Dementia affects the elderly and seniors to the greatest extent. Thus, in the population of people 65-79 years old its prevalence is 10-15%, and at the age of 80 years and older it reaches 20%. Since DM develops gradually and is not as pronounced as Alzheimer's disease, most people do not suspect its existence until clinically evident signs appear. The prevalence of DM is about 1.5% in European countries and about 2.2% in Asian countries. To date, it is believed that about 15-20% of dementias in the elderly are due to DM. Establishing the cause of the disease allows determining the optimal treatment tactics, and lifestyle changes in many cases can prevent further progression of the disease, for example, moderate physical activity is an effective method of preventing further cognitive decline [4, 8-10]. According to Erkmjuntti T. (2005) significant vascular changes are found in 48% of Alzheimer's patients and in 33% of people of the same age without its signs. According to I.Skoog (2005), Alzheimer's disease was found in 77% of cases of vascular dementia and only in 17% of cases of vascular dementia isolated vascular pathology was detected [12]. It indicates that the combination of Alzheimer's disease with cerebrovascular pathology is observed in more than 80% of dementia patients [11, 14]. Currently, studies of vascular forms of cognitive disorders are underway, which has led to an active study of the diagnostic capabilities of various methods of structural and functional neuroimaging, especially in the early stages of the disease [1]. MRI method allows more accurately detecting changes in the white and subcortical gray matter of the brain. The absence of signs of cerebrovascular damage according to structural neuroimaging excludes the vascular genesis of cognitive disorders [6, 7].

The neuroimaging picture in vascular cognitive disorders is characterized by the presence of either multiple cortical or subcortical ischemic foci (multi-infarct dementia) or single post-ischemic foci, located in brain regions that are especially important for mnestic-intellectual activity, or so-called strategic zones (thalamus, basal ganglia, mediobasal sections of frontal and temporal lobes, angular gyrus) [1, 7].

However, the most frequent cause of vascular dementia is the lesion of small vessels, leading to the development of widespread leukoaraiosis, often combined with lacunar infarcts and posthemorrhagic small foci in the subcortical area. White matter changes and lacunes are considered as the main manifestations of small vessel pathology in MRI [13]. An important prognostic factor is the evaluation of the dynamics of white matter changes and lacunes as markers of small vessel disease progression. For this reason, we chose this topic as relevant in early diagnosis and prognosis of vascular dementia.

**The aim of the study:** to investigate neuroimaging features of vascular dementia of cortical and subcortical types.

**Materials and methods of the study.** We examined 80 patients with cortical dementia (50 women, 30 men) of different severity (group 1), and 30 patients with subcortical dementia (group 2) - the comparison group. The average age of group 1 patients was 71.8 +6.8 years (main group) and group 2 patients - 70+5 years.

According to the severity of dementia: 10 patients had severe dementia, 40 patients had moderate dementia, and 30 patients had mild dementia.

All patients underwent a comprehensive examination, which included the assessment of neurological status by the Brief Mental State Examination, MMSE (Mini-Mental State Examination), the tests "5 words" and "10 words", the frontal dysfunction battery, the assessment of the level of self-care was carried out with the help of the Barthel scale, which is based on the quantitative measurement of the individual's independence from external help in everyday life. Also, all patients underwent blood tests: cholesterol, triglycerides and coagulogram. CT/MRI of the brain was used as neuroimaging: CT to measure the brain ventricles and the width of the sylvian passages, oblique size of the hippocampal formation; MRI to measure the intercranial distance on coronal slices, to measure the ratio of the intercranial distance to the intercranial distance, and to assess changes in the white matter of the brain using the Fazekas scale**.**

**Results.** In patients with cortical variant of vascular dementia, neurological symptoms and disorders of higher cortical functions were mainly determined by the localization of the lesion. Aphasia was detected in 50.8% of cases, apraxia in 36.9%, agnosia in 16.9%, alexia in 6.2%, and agraphia in 30.8%. Motor disorders occurred in 89.2% of patients, coordination disorders - in 72.3%, sensory disorders - in 32.3%. Signs of pseudobulbar paresis were noted in 86.1% of cases, walking dyspraxia in 10.8%, frontal symptoms in 20%, extrapyramidal disorders in 7.7%, and neurogenic bladder in 6.2%. Arterial hypertension correlated with the severity of cognitive impairment ($p=0.23$; $p<0.05$).

In patients with subcortical variant of vascular dementia neurological symptomatology was as follows: speech disorders were detected in 22.8% of cases, apraxia in 32.6%, agnosia in 17.4%, alexia in 5.4%, agraphia in 22.8%. Speech disorders were characterized by a decrease in fluency and fluency. Ideatorial and spatial apraxia were more frequently observed. Motor disorders were detected in 69.6% of patients, coordination disorders - in 68.5%, sensitivity - in 17.4%, hemianopsia - in 4.3%. Signs of pseudobulbar paresis were detected in 85.9% of cases, walking dyspraxia in 33.7%, frontal symptoms in 21.7%, extrapyramidal disorders in 17.4%, and neurogenic bladder in 14.1%. The frequency of occurrence of the above symptoms increased as the severity of cognitive impairment increased. The total score of the "frontal test battery" had a negative correlation with the presence of frontal symptoms ($p= -0.28$; $p<0.01$), pseudobulbar syndrome ($p= -0.29$; $p<0.01$), walking dyspraxia ($p= -0.49$; $p<0.0001$), extrapyramidal syndrome (p- -0.27; $p<0.01$), neurogenic bladder ($p= -0.37$; $p<0.001$).

In group 1 of patients, focal changes in the form of cystic-gliotic changes in parietal and temporal lobes were predominant in 37.4 and 20.2% of cases, respectively. Among the patients 5 patients had foci localized in thalamus, 2 in basal ganglia, 1 in mediobasal parts of temporal lobe. The average volume of the damaged brain matter amounted to 46.5+9.4 cm3. Diffuse changes were represented by atrophic changes in brain tissue and white matter pathology by the type of periventricular leukoareosis. Subcortical leukoareosis was considered as a diffuse focal lesion.

In the cortical variant, the most pronounced atrophic changes were observed in the frontal and parietal lobes; in the subcortical variant, the parietal and temporal lobes were predominantly affected (Fig-1, Fig-2).

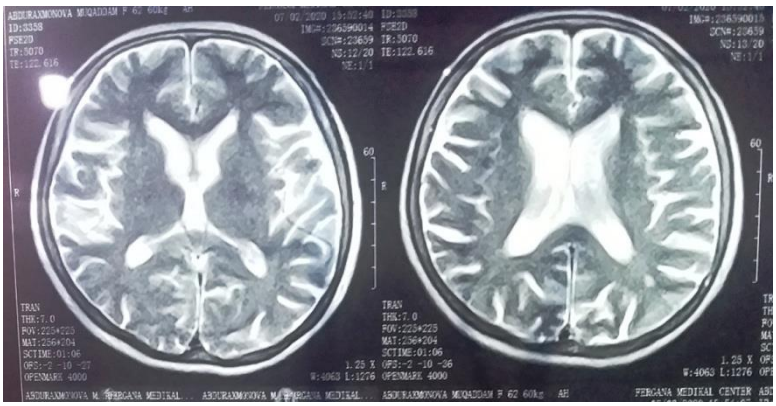

**Figure-1. MRI picture of diffuse atrophy of the cerebral cortex and cerebellum with triventricular substitutive ventriculomegaly.**

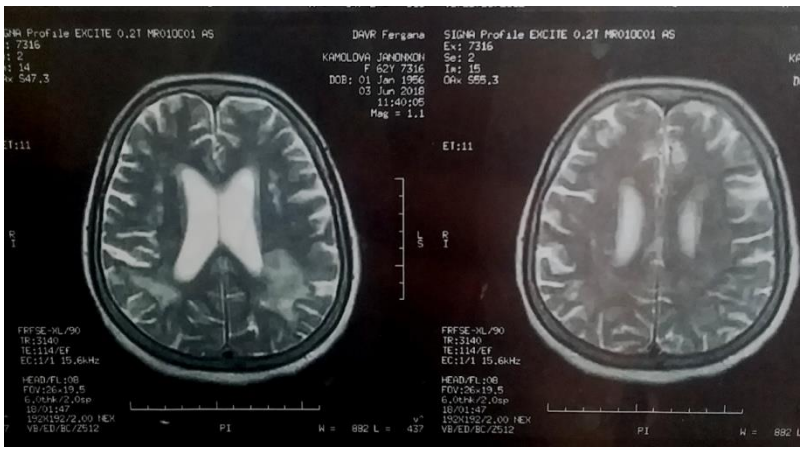

**Figure-2. MRI signs of leukoareosis of the posterior horns of the lateral ventricles in both frontal-parietal regions.**

Atrophic changes had correlation mainly with neuropsychological indicators reflecting the state of attention, memory, and general cognitive state. In patients with group 1, the index of the "attention" subscale correlated with the total index of convexital atrophy ($p= -0.31$; $p<0.05$); of the "memory" subscale - with the severity of general atrophy ($p = - 0.47$; $p<0.01$). The same correlations of general atrophy were established with the total MMSE score ($p= -0.5$; $p<0.0001$) and the "attention + memory" scale ($p= -0.3$; $p<0.05$). In subcortical variant the indices of complex scales had a close correlation with the severity of atrophic changes in temporal lobes of the brain (Scale "attention + memory" - $p= -0.37$; $p<0.01$; MMSE - $p= -0.46$; $p<0.0001$). The results of the 5-word and 10-word tests correlated primarily with atrophy of the temporal lobes ($p= -0.39$; $p<0.001$ and $p= -0.33$; $p<0.01$, respectively) and hippocampus ($p= -0.47$; $p<0.001$ and $p= -0.44$; $p<0.001$). These relationships may reflect the contribution of the concomitant neurodegenerative process. The severity and localization of atrophic changes were more significant for the progression of cognitive impairment from the stage of moderate impairment to dementia ($p<0.001$). White matter pathologic changes and lacunes had a more significant impact in the later stages.

The total score of subcortical leukoaraiosis in group 2 patients was significantly higher than in group 1 and amounted to 10.3+1.4 points in mild dementia and 17.8+6.0 points in moderate dementia. White matter changes were found predominantly in the parietal and frontal lobes, correlated with neurodynamic and regulatory disorders.

Correlation analysis also revealed the association of vascular and atrophic changes, which may reflect the interaction of vascular and neurodegenerative factors. In cortical and subcortical variants, the degree of convexital atrophy correlated steadily with the severity of periventricular and subcortical leukoareosis ($p<0.05$). For all vascular variants, especially for the subcortical variant, a direct correlation between the degree of white matter lesions and lacunes with the severity of cognitive

impairment was found. Differences between patients with moderate cognitive impairment and mild dementia were related to the severity of periventricular leukoaraiosis. As cognitive deficits progressed further, increased severity of subcortical leukoareosis was most important.

**Conclusions.** Vascular cognitive impairments are heterogeneous disorders of cognitive functions differing in etiology, pathogenesis, clinical, neuropsychological and neuroimaging manifestations. The presence of cognitive impairment syndrome of varying degrees of severity is an obligatory sign of cerebrovascular disease. An integrated approach, including along with the assessment of clinical and neuropsychological symptoms of cerebrovascular disease, the study of structural and functional changes in the brain, is the most reliable way to diagnose the nosological affiliation of cognitive disorders.

When using clinical and neuropsychological criteria to establish the vascular etiology of cognitive disorders, the identification of focal symptoms etiopathogenetically related to cognitive deficit and analysis of the profile of neuropsychological disorders are of the greatest importance. The severity of dysregulatory and neurodynamic disorders correlates with the presence of pseudobulbar syndrome, walking dyspraxia, and neurogenic disorders of pelvic organ function. The combination of periventricular and subcortical leukoaraiosis localized in the frontal and parietal lobes with lacunae in the deep parts of the gray and white matter of the brain and convexital atrophy has the greatest diagnostic significance for verification of the vascular genesis of cognitive disorders using magnetic resonance imaging. The severity of atrophic changes is associated with impaired memory, attention, global impairment of cognitive functions. Periventricular leukoaraiosis causes impairment of regulatory functions, and subcortical leukoaraiosis and lacunae cause impairment of regulatory functions and memory. The severity of periventricular leukoaraiosis is important at the initial stages of cognitive deficit development, while subcortical leukoaraiosis contributes to further progression of the disease.

Perfusion disturbance in the area of subcortical structures in subcortical dementia is an early marker of the development of vascular cognitive disorders, while their progression is caused by the appearance of cortical perfusion disturbances in the areas functionally important for the provision of regulatory activity and general cognitive state. In cortical variants of cognitive disorders, perfusion disturbances are not limited to the areas of structural brain damage and are detected both "in the neighborhood" in the homolateral hemisphere and in areas remote from the foci. In the subcortical variant, perfusion disorders predominate in the area of basal ganglia, especially in the caudate nuclei and thalamus, deep parts of the frontal lobes.

Cortical cognitive disorders, regardless of the localization of the focus of damage, are manifested by a decrease in metabolism in the parietal and temporal lobes, which reflects the presence of a concomitant neurodegenerative process.

Subcortical vascular dementia is characterized by mosaic character of metabolic disorders with predominant localization in basal ganglia, orbitofrontal cortex, anterior cingulate cortex.

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