ISSN: 2582-4686 SJIF 2021-3.261,SJIF 2022-2.889,
2023-5.384 ResearchBib IF: 8.848 / 2023

THE MULTIDISCIPLINARY JOURNAL OF SCIENCE AND TECHNOLOGY

VOLUME-3, ISSUE-5

# COGNITIVE IMPAIRMENTS IN CEREBROVASCULAR DISEASE

**Rasulova Raykhon Pardaevna**
Termez Branch of Tashkent Medical Academy
**Safarova Rokhatoy Shafaatovna**
Termez Branch of Tashkent Medical Academy
**Juraev Ilkhomjon Khujakulovich**
Termez Branch of Tashkent Medical Academy

**Annotation**: Cerebrovascular diseases belong to a group of the major causes of cognitive impairments, in the elderly in particular. The paper presents current ideas on the etiology and pathogenesis of vascular cognitive impairments (VCI). The etiological factors of VCI may be divided into genetic, sociodemographic, and common risk factors for vascular and other diseases. The pathogenesis of VCI is multifactorial; cognitive function decrement results from brain damage due to cerebral circulatory disorders. Damage to the deep white matter portions and basal ganglions plays a leading role in the development of cognitive deficit in cerebral circulatory insufficiency, disrupting the connections between the frontal lobes and subcortical structures (a dissociation phenomenon). Regulatory functions are impaired; instability of volitional attention develops; the speed of thinking processes and the performance of professional and everyday skills are suffered, mnestic functions being impaired to a lesser extent. Impairments in other higher cortical functions, such as speech, gnosis, praxis, thinking, generally occur in the later stages of cognitive deficit. The comprehensive approach to examining patients with cognitive dysfunctions, which encompasses physical examination with a mandatory evaluation of neurological symptoms, neuropsychological testing, laboratory studies, instrumental diagnostic methods, and structural and functional neuroimaging techniques, are most justified now. VCI therapy is a challenging task requiring the specific features of different types of cognitive deficit to be analyzed, by providing a rationale for the choice of medications. Therapeutic effectiveness may be enhanced by rational combined multimodal therapy, by keeping in mind a variety of factors for the pathogenesis of VCI.

**Key words:** vascular cognitive impairments; vascular dementia; diagnostic criteria; treatment.

Cerebrovascular diseases (CVD), along with Alzheimer's disease, are the main causes of cognitive impairment (CI), especially in the elderly. Considering the importance of not only the medical, but also the socio-economic component of the problem of CI, interest in it from doctors of various specialties is constantly growing.

The prevalence of dementia in older people in Europe averages 6.4%, with vascular dementia accounting for 1.6%; the incidence of the latter increases with age, amounting to 0.3% in people aged 65–69 years and 5.2% in people over 90 years of age [1]. The incidence of vascular dementia varies significantly - from 1.5 to 3.3 cases per 1000 elderly people, with men getting sick much more often than women [2].

Vascular CIs (VCs) represent a large group of conditions characterized by different clinical and morphological features and different pathophysiology.

Etiological factors for the development of SCI can be divided into genetic (presence of the epsilon-4 allele of the APOE gene, cerebral autosomal dominant arteriopathy with subcortical

ISSN: 2582-4686 SJIF 2021-3.261,SJIF 2022-2.889,
2023-5.384 ResearchBib IF: 8.848 / 2023



infarctions and leukoencephalopathy - CADASIL), socio-demographic (age over 60 years, Mongoloid or Negroid race, male gender, low educational level level), general risk factors (RF) for the development of vascular diseases (arterial hypertension - hypertension, coronary heart disease, orthostatic hypotension, diabetes mellitus, hypercholesterolemia, hyperhomocysteinemia, obesity), others (smoking, alcoholism, sleep apnea) [3, 4].

The pathogenesis of SCI is multifactorial, and cognitive decline develops as a result of destructive brain damage caused by cerebrovascular accidents. The morphological basis of SCI is most often infarctions (more precisely, post-infarction cysts) or diffuse ischemic damage to the subcortical white matter (subcortical leukoencephalopathy) and strategically important areas (thalamus, frontobasal, limbic regions) [5].

There is experimental and clinical evidence of cholinergic deficits in vascular dementia, regardless of the presence or absence of signs of a neurodegenerative process. This position is confirmed by a decrease in the level of acetylcholine in the cerebrospinal fluid and a decrease in the activity of choline acetyltransferase in the brain. The cholinergic basal nucleus of Meynert is supplied by penetrating arterioles and is therefore very sensitive to the effects of hypertension and cerebral ischemia. Of great interest are also the results indicating the importance of glutamatergic disorders in the development of cognitive deficits. During ischemia of brain tissue, the functioning of cellular transport systems is disrupted. In addition, due to sustained pathological depolarization of the cell membrane, an increased release of glutamate from synaptic vesicles occurs, leading to hyperactivation of glutamate NMDA receptors and an excessive influx of Ca++ into the cell. Thus, glutamate, which is an essential component of the neurotransmitter systems of the brain, in the development of a number of pathological conditions, such as ischemia, can have a damaging effect on the nerve cell, acting as a neurotoxin [6].

In addition to cholinergic and glutamatergic transmission, other neurotransmitter terminals of cells projecting to the neocortical cortex are also affected: serotonergic terminals of the median raphe and noradrenergic terminals of the nucleus coeruleus, which may be associated with the development of affective and behavioral disorders in patients.

Damage to interneuronal transmission is closely related to various biochemical and metabolic pathogenetic reactions: disorders of glucose metabolism, decreased overall energy metabolism, oxidative stress and a number of other mechanisms. Glucose metabolism affects many cellular processes occurring in the brain, and primarily energy metabolism, which is determined by its participation in the synthesis of adenosine triphosphate. The free radical oxidation process that develops against the background of ischemia, which is closely associated with inflammatory reactions and endothelial dysfunction, is also of great importance. Activation of lipid peroxidation processes contributes to the accumulation of free radical molecules in the body, which react with polyunsaturated fatty acids and other chemical compounds of cellular structures, causing irreversible changes both at the cellular level and in the body as a whole.

CIs arising as a result of a cerebrovascular process are quite heterogeneous, which is explained, on the one hand, by different localizations of brain lesions, and on the other, by differences in the characteristics of the formation of dementia. In general, vascular dementia is characterized by a “mosaic pattern” of cognitive deficits, an inconsistent pattern of cognitive impairment, and fluctuations in symptoms.

The leading role in the formation of cognitive deficit in cerebrovascular insufficiency is played by damage to the deep parts of the white matter of the brain and the basal ganglia, which

ISSN: 2582-4686 SJIF 2021-3.261,SJIF 2022-2.889,
2023-5.384 ResearchBib IF: 8.848 / 2023



leads to disruption of connections between the frontal lobes of the brain and subcortical structures (disconnection phenomenon) [7]. Regulatory functions are disrupted (planning, the sequence of performing certain actions, monitoring the results of actions, the ability to generalize the received material), instability of voluntary attention develops, the speed of thought processes, the performance of professional and everyday skills suffer, while mnestic functions are damaged to a lesser extent . A reliable sign that allows us to distinguish the secondary nature of memory impairments in vascular lesions from primary mnestic disorders of neurodegenerative origin is the distinct effect of prompts [4].

Violations of other higher cortical functions - speech, gnosis, praxis, thinking - arise, as a rule, at later stages of development of cognitive deficit and can be either caused by the development of a lesion in certain areas of the brain, or develop as a result of a violation of interneuronal relationships when deep parts of the brain. Patients with vascular dementia demonstrate greater deficits in speech fluency than patients with Alzheimer's disease. Although motor aspects of speech may be affected in patients with vascular dementia, primary speech function tends to be preserved. Agnosia is characterized by the inability to recognize sensory signals as a holistic image while maintaining the perception of its individual features; they are often modality-specific in nature, which is due to the localization of the lesion. Apraxia is manifested by a violation of purposeful motor activity, which may be based on various pathogenetic mechanisms. As a result of the loss of certain skills, disturbances in professional activity and daily activities, including dressing, are observed. Perseverations (stereotypical repetitions of the same movements) and violation of the sequence of actions are characteristic.

The uniqueness of the clinical picture of vascular dementia also appears in combination with other psychopathological and neurological disorders. The most significant correlations of vascular cognitive deficit are observed with the presence of pseudobulbar syndrome, gait dyspraxia, pyramidal symptoms, more pronounced in the legs, and dysfunction of the pelvic organs.

Currently, the most justified is a comprehensive approach to the examination of patients with impaired cognitive functions of varying severity, including a general examination with a mandatory assessment of neurological symptoms, neuropsychological testing, laboratory tests, instrumental diagnostic methods, structural and functional neuroimaging methods.

The collection of complaints and medical history must necessarily include a conversation with the patient himself and with his relatives or persons capable of providing the necessary information. Of great importance is the collection of information about problems associated with the performance of everyday and professional skills, the presence of behavioral and affective disorders. During an objective examination, it is necessary to pay attention to identifying symptoms indicating the presence of somatic, infectious pathology to exclude the secondary nature of dementia, as well as to identify possible risk factors for the development of dementia. A neurological examination can reveal symptoms indicating focal brain damage.

To verify and determine the severity of CI, it is mandatory to conduct a neuropsychological study. The scope of neuropsychological testing and the choice of methods are determined by the severity of cognitive disorders, the nature of the existing impairments, and the goals of the researcher. The most informative methods for identifying SCI are the frontal dysfunction battery, the clock drawing test, the Montreal Comprehensive Test (MoCA-test), the tracking test, and the verbal association test.

ISSN: 2582-4686 SJIF 2021-3.261,SJIF 2022-2.889,
2023-5.384 ResearchBib IF: 8.848 / 2023

THE MULTIDISCIPLINARY JOURNAL OF SCIENCE AND TECHNOLOGY

VOLUME-3, ISSUE-5

Laboratory diagnostics are carried out to identify current somatic diseases, other risk factors and include a general blood and urine test, a biochemical blood test (sugar, liver enzymes, urea, creatinine, electrolytes, thyroid hormones, homocysteine, lipid profile, folic acid, vitamin B12) , serological reactions to syphilis and AIDS, determination of the APOE gene isoform, proinflammatory markers

Among the instrumental diagnostic methods, it is advisable to use vascular research methods, such as Doppler ultrasound and duplex scanning of extra- and intracranial arteries of the brain, which allow one to assess the speed of cerebral blood flow, cerebral vasomotor reactivity, and identify signs of an occlusive or stenotic process. Instrumental diagnostics can also be used to assess the severity of changes in other organs and systems, which may, in particular, reflect systemic damage in the pathology of small vessels (retinopathy, nephropathy).

It is mandatory to use structural neuroimaging methods in the diagnosis of vascular dementia, not only to confirm the vascular nature of brain damage, but also to identify possible curable diseases, such as tumor, normal pressure hydrocephalus and chronic subdural hematoma.

The neuroimaging picture in SCI of post-stroke origin is characterized by the presence of either multiple cortical or subcortical ischemic foci, or single post-ischemic foci located in areas of the brain that are especially significant for mnestic-intellectual activity - the so-called strategic zones (thalamus, basal ganglia, mediobasal regions of the frontal and temporal lobes , angular gyrus) [7, 8]. However, the most common cause of vascular dementia is damage to small vessels, leading to the development of widespread leukoaraiosis, often combined with lacunar infarctions and post-hemorrhagic small lesions in the subcortical region. Almost always, in cases of vascular dementia, structural neuroimaging reveals signs of cerebral atrophy in the form of enlargement of the cerebral ventricles and subarachnoid spaces, which can cause certain difficulties in differential diagnosis with degenerative type dementia [9].

Functional neuroimaging methods (single photon emission computed tomography - SPECT, positron emission tomography - PET) have additional diagnostic capabilities, allowing to detect a decrease in perfusion and metabolism in the projection of the deep parts of the brain (thalamus, subcortical ganglia, caudate nucleus), even in the absence of pronounced structural changes [10].

**Literature:**

1. Lobo A, Launer L, Fratiglioni L, et al. Prevalence of dementia and major subtypes in Europe: a collaborative study of populationbased cohorts. Neurologic diseases in the elderly research group. Neurology. 2000;54(11 Suppl 5):S4–9.

2. Ruitenberger A, Ott A, van Swieten JC, et al. Incidence of dementia: does gender make a difference? Neurobiol Aging. 2001;22(4):575–80.

3. Roman G. Vascular dementia prevention: a risk factor analysis. Cerebrovas Dis. 2005;20(Suppl 2):91–100.

4. Одинак ММ, Емелин АЮ, Лобзин ВЮ. Нарушение когнитивных функций при цереброваскулярной патологии. Санкт-Петербург: ВМедА; 2006. 158 с. [Odinak MM, Emelin AYu, Lobzin VYu. Narushenie kognitivnykh funktsii pri tserebrovaskulyarnoi patologii [Violation of cognitive functions at cerebrovascular pathology]. St-Petersburg: VMedA; 2006. 158 p.]

5. Jellinger K. Morphologic diagnosis of «vascular dementia» – a critical update. J Neurol Sci. 2008;270(1–2):1–12.

ISSN: 2582-4686 SJIF 2021-3.261,SJIF 2022-2.889,
2023-5.384 ResearchBib IF: 8.848 / 2023



VOLUME-3, ISSUE-5

6. Parsons C, Danysz W, Hesselink M, et al. Modulation of NMDA receptors by glycine – introduction to some basic aspects and recent developments. Amino Acids. 1998;14(1–3):207–16.

7. Яхно НН, Захаров ВВ. Когнитивные и эмоционально-аффективные нарушения при дисциркуляторной энцефалопатии. Русский медицинский журнал. 2002;10(12/13):531–51. [Yakhno NN, Zakharov VV. Cognitive and emotional and affective violations at discirculatory encephalopathy. Rusckii meditsinskii zhurnal. 2002;10(12/13):531–51. (In Russ.)]

8. Roman GC, Sachdev P, Royall DR, et al. Vascular cognitive disorder: a new diagnostic category updating vascular cognitive impairment and vascular dementia. J Neurol Sci. 2004;226(1–2):81–7.

9. Pantoni L, Poggesi A, Inzitari D. Cognitive decline and dementia related to cerebrovascular diseases: some evidence and concepts. Cerebrovasc Dis. 2009;27(S1):191–6.

10. Hachinski VC, Lassen NA, Marshall J. Multi-infarct dementia. A case of mental deterioration in the elderly. Lancet. 1974;2(7874):207–10.