DYSCIRCULATORY ENCEPHALOPATHY IN GENERAL MEDICAL PRACTICE: MODERN CONCEPTS OF DIAGNOSIS AND MANAGEMENT.

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Abstract.

Dyscirculatory encephalopathy (DE) is a progressive chronic cerebrovascular disorder resulting from long-term cerebral hypoperfusion and small vessel disease. It represents a major cause of cognitive decline, gait disturbances, emotional disorders, and vascular dementia in elderly patients. The present article analyzes contemporary concepts of DE pathogenesis, emphasizing the role of cerebral microangiopathy, white matter damage, and cortico-subcortical disconnection. Clinical manifestations, including executive dysfunction, vascular depression, and motor impairment, are discussed according to disease stages. Diagnostic principles are outlined with particular attention to neuropsychological screening and modern neuroimaging standards for small vessel disease. Current treatment strategies focus on vascular risk factor modification, hemodynamic stabilization, and pathogenetic therapy aimed at improving cerebral microcirculation and neuronal metabolism. Early recognition and comprehensive management in general medical practice are essential to slow progression and reduce the risk of vascular dementia.

Keywords.

Dyscirculatory encephalopathy; chronic cerebral ischemia; cerebral small vessel disease; cognitive impairment; vascular dementia; leukoaraiosis; executive dysfunction; cerebral microangiopathy; neuroimaging; vascular depression.

Introduction.

Chronic cerebrovascular disorders remain one of the leading causes of disability and cognitive decline worldwide. Dyscirculatory encephalopathy (DE) represents a progressive form of chronic cerebral ischemia characterized by diffuse and focal structural brain damage resulting from long-standing vascular insufficiency. In clinical practice, DE is frequently encountered by general practitioners, neurologists, cardiologists, and family physicians. Early recognition and comprehensive management are essential to prevent progression to dementia and severe neurological disability [1-3].

Epidemiological Significance.

Chronic cerebral ischemia and stroke contribute substantially to morbidity and mortality, particularly in elderly populations. The prevalence of chronic cerebrovascular insufficiency increases with age and is strongly associated with arterial hypertension, diabetes mellitus, and atherosclerosis. Cognitive impairment and vascular dementia often develop as long-term consequences of persistent cerebral hypoperfusion. Therefore, DE represents not only a neurological disorder but also a significant public health concern [2].

Definition and Conceptual Framework.

Dyscirculatory encephalopathy is defined as a slowly progressive syndrome caused by chronic cerebral hypoperfusion, leading to multiple diffuse and focal lesions of brain tissue. Although the term is widely used in Eastern European clinical practice, it is not formally included in ICD-10; instead, it corresponds conceptually to chronic cerebral ischemia (I67.8). The pathological process primarily affects small penetrating arteries and arterioles, resulting in white matter damage and subcortical dysfunction [5].

Etiology and Pathogenesis.

The key pathogenetic mechanism underlying DE is cerebral small vessel disease. Chronic arterial hypertension, diabetes mellitus, atherosclerosis, amyloid angiopathy, and systemic connective tissue disorders contribute to structural remodeling of small-caliber arteries. Endothelial dysfunction, oxidative stress, and impaired autoregulation lead to unstable cerebral perfusion.

The most vulnerable regions include deep white matter of the cerebral hemispheres and basal ganglia. Neuroimaging frequently reveals silent lacunar infarctions and leukoaraiosis. Damage to subcortical structures disrupts cortico-subcortical connections, producing a “disconnection syndrome” responsible for cognitive, emotional, and motor disturbances [4-6].

Clinical Manifestations.

Cognitive impairment is the earliest and most prominent manifestation of DE. Executive dysfunction predominates, including impaired planning, reduced mental flexibility, slowed information processing, and diminished attention. Memory impairment typically affects working and learning capacity, while remote autobiographical memory may remain relatively preserved in early stages.

Emotional disturbances are common and include apathy, vascular depression, irritability, and emotional lability. Organic depression associated with frontal-subcortical disconnection is frequently observed and may precede overt neurological signs [3-5].

Motor symptoms develop progressively. Gait disturbances, reduced stride length, postural instability, and slowed walking are characteristic. In advanced stages, a “lower body parkinsonism” pattern may appear, with shuffling gait and difficulty initiating movement. Pseudobulbar syndrome and pyramidal signs may also occur.

Staging.

Stage I: Predominantly subjective complaints, mild cognitive impairment, minimal focal neurological signs.

Stage II: Objective neurological syndromes, moderate cognitive impairment, pseudobulbar or extrapyramidal features.

Stage III: Severe neurological deficits, vascular dementia, marked motor dysfunction.

Diagnostic Approach.

Accurate diagnosis requires integration of clinical, neuropsychological, and neuroimaging findings. The diagnostic algorithm includes:

– Identification of vascular risk factors;

– Structured cognitive screening (e.g., Mini-Cog test);

– Comprehensive neurological examination;

– Brain MRI or CT to detect lacunar infarcts, white matter hyperintensities, and cerebral atrophy.

Modern neuroimaging standards (such as STRIVE criteria) provide structured evaluation of small vessel disease markers, including lacunes, recent small subcortical infarcts, microbleeds, perivascular spaces, and white matter hyperintensities.

Treatment Principles.

Management of DE is multidirectional and includes both etiological and pathogenetic strategies.

Etiological therapy focuses on vascular risk modification:

– Strict blood pressure control;

– Glycemic regulation in diabetes;

– Lipid profile correction;

– Management of cardiac arrhythmias and hemodynamic instability.

Pathogenetic therapy aims to improve cerebral microcirculation and neuronal metabolism. Pharmacological options may include calcium channel blockers, phosphodiesterase inhibitors, α2-adrenoreceptor antagonists, and neuroprotective agents. Treatment strategies target hypoxia reduction, membrane stabilization, oxidative stress correction, and enhancement of neuronal survival.

Hospitalization is generally indicated in cases of acute neurological deficit or severe decompensation; otherwise, most patients can be managed in outpatient settings.

Conclusion.

Dyscirculatory encephalopathy is a progressive chronic cerebrovascular disorder that significantly affects cognitive and motor functions. Early detection by general practitioners is critical to prevent transition to vascular dementia. Comprehensive management addressing vascular risk factors, cerebral hemodynamics, and neuronal protection can slow disease progression and improve quality of life. A multidisciplinary and individualized approach remains the cornerstone of effective long-term care.

Literature.

1. Skvortsov VV, Levitan BN, Golieva EA, Malyakin GI. Dyscirculatory encephalopathy in general medicine practice. Lechaschy Vrach. 2021;(5):27–31. doi:10.51793/OS.2021.49.39.006.

791ad1e5-56b0-43f9-836d-f970481…

2. Zakharov VV. Clinic, diagnosis and treatment of dyscirculatory encephalopathy. Russian Medical Journal. 2009;17(2):140.

3. Gusev EI, Bogolepova AN. Cognitive disorders in cerebrovascular diseases. Moscow: MEDpress-inform; 2013.

4. Pantoni L. Cerebral small vessel disease: from pathogenesis and clinical characteristics to therapeutic challenges. Lancet Neurol. 2010;9(7):689–701.

5. Wardlaw JM, Smith EE, Biessels GJ, et al. Neuroimaging standards for research into small vessel disease and its contribution to ageing and neurodegeneration. Lancet Neurol. 2013;12(8):822–838.

6. Yakhno NN, Zakharov VV, Lokshina AB. Syndrome of moderate cognitive impairment in dyscirculatory encephalopathy. Zh Nevrol Psikhiatr Im S S Korsakova. 2005;105(2):13–17.