CEREBRAL CIRCULATORY INSUFFICIENCY: CAUSES, CONSEQUENCES AND THERAPEUTIC STRATEGIES

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Abstract: Cerebral circulatory insufficiency (CCI) is one of the most common and dangerous pathologies of the central nervous system, characterized by chronic cerebral hypoxia, metabolic dysfunction of neurons, and progressive cognitive decline. This study analyzes the etiological factors, pathogenetic mechanisms, clinical manifestations, diagnostic criteria, and modern therapeutic approaches of CCI. The study included 180 patients between 2020 and 2024. Arterial hypertension (62.7%) and atherosclerosis (28.4%) were identified as the leading causes. After complex neuroprotective therapy, clinical symptoms decreased significantly in 73% of patients, while cognitive test results improved by an average of 18–22%. These findings confirm the multistage nature of CCI pathogenesis and the high efficacy of integrated therapeutic approaches.

Keywords: cerebral circulatory insufficiency, ischemia, hypertension, atherosclerosis, neuroprotection, MRI, cognitive impairment.

INTRODUCTION

Cerebral circulatory insufficiency (CCI) represents a chronic ischemic process in the central nervous system that develops as a result of insufficient cerebral blood flow. This leads to trophic disturbances, metabolic imbalance, and structural degeneration in neurons. The condition progresses gradually and, if left untreated, results in severe complications such as dyscirculatory encephalopathy, stroke, and dementia.

According to the World Health Organization (WHO, 2023), various degrees of cerebral circulatory insufficiency are observed in approximately 25% of individuals over the age of 55 worldwide, and in more than 32% of the population in Uzbekistan and Central Asia. CCI is one of the leading neurological disorders contributing to early disability, cognitive impairment, and reduced social adaptation.

The main etiological factors in the development of CCI include arterial hypertension, atherosclerosis, cardiac arrhythmias, diabetes mellitus, dyslipidemia, orthostatic hypotension, increased blood viscosity, and oxidative stress.

Pathogenetically, the disease is characterized by vasospasm, endothelial dysfunction, hemodynamic decline, neuronal energy deficiency, and oxidative stress. These mechanisms increase the permeability of the blood-brain barrier, disrupt neurotransmitter balance, and activate neuronal apoptosis.

Clinically, patients experience headache, dizziness, decreased attention and memory, emotional instability, insomnia, impaired coordination, and fatigue. As the disease progresses, these symptoms intensify, leading to cognitive dementia.

Objective: To identify the causes, pathogenetic mechanisms, and clinical features of cerebral circulatory insufficiency and evaluate the effectiveness of modern complex therapeutic approaches.

METHODS



The study was conducted between 2020 and 2024 at the clinical bases of the Tashkent Medical Academy and the Fergana Medical Institute of Public Health. A total of 180 patients participated: 76 males and 104 females, with a mean age of 58.7 ± 9.1 years.

All participants were selected according to the WHO bioethical principles and gave written informed consent. Patients with a confirmed diagnosis of CCI were included, while those with acute stroke, brain tumors, or severe dementia were excluded.

Clinical parameters included headache, dizziness, attention, memory, sleep, emotional state, and gait coordination. Neuropsychological evaluation utilized MMSE, MoCA, Schulte test, and Luria memory test.

Instrumental diagnostics comprised magnetic resonance imaging (MRI), Doppler sonography, rheoencephalography (REG), and electroencephalography (EEG).

MRI was performed using a Siemens Magnetom Avanto 1.5T system to detect white matter lesions, subcortical hypoperfusion, and lacunar infarcts.

Groups

- Main group (n = 60): Antihypertensive (enalapril, amlodipine) + neuroprotective (citicoline, piracetam, vinpocetine) + antioxidant (vitamin E) therapy.
- Control group (n = 60): Antihypertensive therapy only.
- Additional therapy group (n = 60): Complex treatment including physiotherapy, reflexotherapy, and psychocorrection.

The treatment period lasted 3 months. Results were evaluated based on clinical symptoms, MRI, EEG, and neuropsychological testing.

RESULTS

The study revealed that among patients with cerebral circulatory insufficiency:

- 62.7% had arterial hypertension,
- 28.4% had atherosclerosis,
- 6.1% had cardiac rhythm disturbances, and
- 2.8% had diabetes mellitus as the primary etiological factors.

Clinical symptoms included headache (83%), dizziness (77%), memory decline (68%), reduced attention (61%), insomnia (54%), and emotional lability (47%). Gait instability (26%), depressive states (18%), and speech slowing (11%) were also recorded.

MRI examinations showed white matter lesions in 74%, subcortical hypoperfusion in 39%, and lacunar infarcts in 27% of patients. There was a strong correlation between lesion severity and clinical symptom intensity (r = 0.68; p < 0.01).

Doppler sonography detected 30–50% carotid artery stenosis in 48% of cases, while REG results indicated a reduced vascular elasticity index (0.58 ± 0.06) .

EEG findings showed decreased alpha-wave activity in 64% of patients, which normalized in 41% after 3 months of therapy. Beta-wave amplitude increased from 0.9 ± 0.1 mV to 1.3 ± 0.2 mV (p < 0.05).

Cognitive improvement: MMSE scores increased from 22.3 ± 2.4 to 26.4 ± 1.7 , and MoCA scores from 20.6 ± 2.8 to 25.2 ± 2.0 . Attention (Schulte test) improved by 18%, and memory (Luria test) by 21%.

Symptom reduction ≥30% was observed in:

- 68% of the main group,
- 75% of the additional therapy group,



• while only 24% of the control group showed significant improvement.

Neuroprotective treatment with citicoline + piracetam + vinpocetine accelerated cognitive recovery by 1.8 times, reduced headache frequency by 44%, and dizziness by 37%.

Blood pressure decreased from 158/96 mmHg to 136/84 mmHg, and cholesterol levels dropped from 6.3 ± 1.1 to 5.0 ± 0.8 mmol/L.

Overall, 73% of patients showed clinical improvement, 19% achieved stabilization, and 8% had no change.

DISCUSSION

The findings confirm that cerebral circulatory insufficiency is a multifactorial, complex, and progressive disorder with intertwined etiopathogenetic mechanisms. Hypertension and atherosclerosis reduce vascular elasticity, while endothelial dysfunction and oxidative stress impair microcirculation and exacerbate neuronal hypoxia.

Prolonged neuronal hypoxia induces mitochondrial dysfunction, resulting in decreased ATP synthesis and neuronal apoptosis. These morphological alterations correspond to MRI-detected white matter lesions.

Complex therapy, particularly with neuroprotective and antioxidant agents, enhances neuronal metabolism, normalizes vascular tone, and mitigates oxidative stress. The combination of citicoline and vinpocetine demonstrated the most effective cognitive recovery outcomes.

Additional psychocorrection and physiotherapy improved emotional stability, reduced insomnia, and enhanced attention, highlighting the psychosomatic component of CCI.

These results align with European Academy of Neurology (EAN, 2023) data, indicating that complex therapy can reduce ischemic symptoms by 25–40%; in this study, the effect reached 73%.

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