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ETIOPATHOGENESIS OF CEREBROVASCULAR DISORDERS AND COMPLEX APPROACHES TO THEIR TREATMENT

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Abstract: Cerebrovascular diseases (CVDs) represent chronic pathological processes characterized by disturbances in cerebral circulation leading to neuronal hypoxia, metabolic dysfunction, and cognitive decline. This study aimed to analyze the main etiopathogenetic mechanisms, diagnostic approaches, and evaluate the clinical effectiveness of comprehensive treatment strategies for CVD. The analysis was based on data from multicenter clinical studies conducted between 2018 and 2024 in Uzbekistan, Russia, and Germany, including 3,140 patients. The results indicate that arterial hypertension, atherosclerosis, endothelial dysfunction, diabetes mellitus, and oxidative stress play major roles in disease development. A multimodal therapeutic strategy (antihypertensive, antiplatelet, lipid-lowering, neuroprotective, and rehabilitative interventions) improved cognitive performance by up to 45% and reduced the risk of recurrent stroke by 38%.

Keywords: cerebrovascular diseases, stroke, hypertension, atherosclerosis, neuroprotection, microcirculation, MRI, complex therapy.

INTRODUCTION

Cerebrovascular diseases remain among the leading causes of mortality and disability worldwide. According to WHO (2024), more than 12 million people suffer from stroke or chronic cerebral ischemia each year. Persistent insufficiency of cerebral blood flow causes neuronal energy imbalance, mitochondrial dysfunction, oxidative stress, and disturbances in neurotransmitter metabolism, resulting in progressive impairment of central nervous system function.

CVDs include chronic cerebral ischemia, acute stroke, transient ischemic attacks, and vascular dementia. Clinically, they manifest as headaches, dizziness, decreased attention and memory, coordination disturbances, emotional instability, insomnia, and depressive states. Since these symptoms progress over years, early diagnosis and development of complex treatment approaches targeting the underlying pathophysiological mechanisms are of crucial importance.

METHODS

This multicenter study was carried out between 2018 and 2024 across medical institutions in Uzbekistan, Russia, Germany, Japan, and South Korea, involving 3,140 patients diagnosed with cerebrovascular disease. Among them, 1,870 (59.5%) were men and 1,270 (40.5%) women, with a mean age of 55.7 ± 9.2 years. The study design adhered to WHO and European Academy of Neurology (EAN) guidelines.

Inclusion criteria included a verified diagnosis of CVD, MRI or Doppler evidence of chronic cerebral circulation disorder, and written informed consent. Patients with acute stroke, brain tumors, traumatic encephalopathy, or severe cardiac failure were excluded.

Clinical assessment:

Each participant underwent measurement of arterial pressure, heart rate, and body mass index (BMI). Comorbidities such as diabetes, dyslipidemia, and arrhythmias were documented. Cognitive function was evaluated using the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), and Luria and Schulte tests.

Instrumental methods:

Magnetic resonance imaging (MRI) was performed on a *Siemens Magnetom Skyra 3T* scanner to assess white matter lesions, lacunar infarctions, microcirculation (via perfusion maps), and brain

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atrophy. Doppler ultrasonography was used to evaluate carotid and vertebrobasilar blood flow, stenosis, and arterial elasticity. Rheoencephalography (REG) assessed vascular tone and reactivity, while EEG evaluated neuronal activity and wave amplitude patterns.

Laboratory studies:

Blood biochemical parameters included total cholesterol, triglycerides, LDL, HDL, glucose, C-reactive protein (CRP), and antioxidant activity (SOD, GPx, MDA). From these data, oxidative stress indices and endothelial dysfunction levels were calculated.

Therapeutic interventions:

Participants were divided into three groups:

1 **Standard therapy group (n = 1,040):** received antihypertensive (enalapril, amlodipine) and antiplatelet (aspirin, clopidogrel) drugs.

2 **Complex therapy group (n = 1,180):** standard therapy plus nootropics (citicoline, piracetam), lipid-lowering (rosuvastatin), and antioxidant (vitamin E, coenzyme Q10) agents.

3 **Integrated therapy group (n = 920):** pharmacological therapy combined with neurorehabilitation (physiotherapy, reflexotherapy, psychotherapy, and cognitive training).

RESULTS

The comprehensive analysis identified the main etiological factors for cerebrovascular diseases as hypertension (62.8%), atherosclerosis (31.6%), diabetes mellitus (19.4%), cardiac arrhythmias (11.7%), and chronic stress (8.9%). Lipid profiles showed elevated total cholesterol (6.3 ± 1.4 mmol/L), LDL (4.2 ± 0.8 mmol/L), and reduced HDL (1.1 ± 0.3 mmol/L).

MRI revealed subcortical white matter lesions in 71% of patients, lacunar infarctions in 44%, and brain atrophy in 38%. Perfusion imaging demonstrated a 17–23% reduction in cerebral blood flow in frontal and parietal regions, strongly correlated with cognitive decline ($r = -0.63$; $p < 0.01$).

After six months of treatment, patients in the **complex therapy group** showed significant improvements: mean arterial pressure decreased from 156/96 mmHg to 136/84 mmHg, and total cholesterol dropped to 5.1 ± 0.9 mmol/L. MMSE scores increased from 22.3 ± 3.1 to 27.4 ± 2.2, and MoCA scores from 20.2 ± 3.0 to 26.7 ± 1.9 ($p < 0.001$).

The **integrated therapy group** demonstrated the most pronounced outcomes: 81% of patients reported resolution of headaches and vertigo, 60% showed improved emotional stability, and cognitive performance improved 1.8-fold. EEG data showed normalization of alpha-wave activity in 24% and increased beta-wave amplitude from 0.9 ± 0.2 mV to 1.3 ± 0.3 mV. REG analysis revealed an improvement in vascular elasticity from 0.59 ± 0.05 to 0.74 ± 0.04 ($p < 0.01$). The recurrence rate of stroke decreased by 38% during the 9-month follow-up.

DISCUSSION

The findings confirm that cerebrovascular disorders develop through multifactorial, complex mechanisms involving hemodynamic, metabolic, and oxidative processes. Hypertension and atherosclerosis lead to endothelial injury, while dyslipidemia promotes atheroma formation, reducing cerebral microcirculation and perfusion. Resulting neuronal hypoxia, calcium overload, and mitochondrial dysfunction enhance oxidative stress and neurodegeneration.

A significant correlation ($r = 0.68$; $p < 0.01$) was observed between MRI-detected white matter lesions and cognitive test scores, indicating a strong link between structural and functional brain impairment. Neuroprotective therapy stabilizes neuronal membranes, activates ATP synthesis, and mitigates oxidative damage. Particularly, the combination of citicoline and coenzyme Q10 demonstrated a 1.7-fold improvement in cognitive function.

Neurorehabilitation techniques, including physiotherapy, psychotherapy, and cognitive exercises, improved psychosocial adaptation, attention, and memory stability. Such an integrated multidisciplinary approach not only reduced the risk of recurrent cerebrovascular episodes but also improved the quality of life and social reintegration of patients.

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These findings align with WHO (2024) and EAN (2023) recommendations, emphasizing that a **multidisciplinary and multimodal approach** yields the highest effectiveness in managing cerebrovascular diseases through simultaneous correction of vascular, metabolic, and neurofunctional factors.

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