

MAIN PATHOGENETIC MECHANISMS IN THE DEVELOPMENT OF Cerebrovascular Insufficiency and Discirculatory Encephalopathy

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Abstract: This article examines the main etiopathogenetic factors in the development of discirculatory encephalopathy (DE), their interrelations, and the clinical and neuropsychological characteristics observed in affected patients. The study involved 150 patients diagnosed with DE. Arterial hypertension, atherosclerosis, dyslipidemia, endothelial dysfunction, and oxidative stress were identified as the key pathogenetic mechanisms. Neuropsychological tests, MRI, EEG, REG, and Doppler ultrasound findings were analyzed to reveal the complexity of DE pathogenesis. The results confirmed the effectiveness of personalized, comprehensive therapeutic strategies in improving cognitive and vascular function in DE patients.

Keywords: discirculatory encephalopathy, cerebrovascular insufficiency, cognitive impairment, neuroprotection, atherosclerosis, oxidative stress.

INTRODUCTION

Discirculatory encephalopathy (DE) is a progressive neurodegenerative disease caused by chronic insufficiency of the cerebral vascular system, manifested through cognitive, emotional, and motor dysfunctions. According to the World Health Organization (WHO), DE accounts for 20–25% of dementia cases among people over 65 years old worldwide. Approximately 70% of DE cases are associated with arterial hypertension, atherosclerosis, and dyslipidemia.

In Uzbekistan, the incidence of DE has been steadily increasing due to an aging population and the high prevalence of cardiovascular diseases. DE requires early diagnosis and comprehensive therapy to prevent irreversible brain damage. The purpose of this study was to identify the key pathogenetic factors involved in DE, analyze their relationship with clinical manifestations, and evaluate the effectiveness of complex treatment approaches.

METHODS

The study included 150 patients aged 45–75 years diagnosed with DE. Clinical, laboratory, neuropsychological, and instrumental evaluations were conducted. Clinical assessments included measurements of blood pressure, heart rate, neurological symptoms, headache, dizziness, sleep disturbances, attention, and memory decline.

Neuropsychological evaluations involved the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), and Schulte test. Instrumental investigations included magnetic resonance imaging (MRI), electroencephalography (EEG), rheoencephalography (REG), and Doppler ultrasonography. Laboratory tests measured lipid profile, glucose, C-reactive protein, and oxidative stress markers (MDA, SOD).

Patients underwent a 3-month course of complex therapy, including antihypertensive (enalapril, amlodipine), antiatherosclerotic (atorvastatin), nootropic (piracetam, citicoline), angioprotective (vinpocetine), and antioxidant (vitamin E, mexidol) drugs. Physiotherapy and reflexotherapy were used as adjunct treatments. Statistical analyses were performed using SPSS 25.0 software, with $p < 0.05$ considered significant.

RESULTS

Among 150 patients, arterial hypertension was found in 59.3%, atherosclerosis in 33.4%, and vertebrobasilar insufficiency in 7.3% of cases. Diabetes mellitus, cardiac arrhythmias, and

metabolic syndrome were identified as aggravating factors in DE pathogenesis. Clinically, headache was observed in 84% of patients, dizziness in 76%, memory decline in 71%, decreased attention in 65%, emotional lability in 48%, fatigue in 37%, and sleep disturbances in 29%.

According to neuropsychological tests, 82% of patients demonstrated mild to moderate cognitive impairment. The mean MMSE score improved from 22.8 ± 2.3 to 26.1 ± 1.9 , and MoCA from 20.7 ± 2.5 to 25.3 ± 2.1 ($p < 0.05$), indicating cognitive improvement. Schulte test results showed a 15–18% increase in attention stability.

MRI revealed subcortical hypoperfusion in 78% of patients, diffuse white matter lesions in 46%, and lacunar infarcts in 32%. REG demonstrated a decrease in vascular wall elasticity index to 0.62 ± 0.04 , while Doppler ultrasound revealed carotid artery stenosis of 30–50% in 40% of patients. EEG findings showed normalization of alpha wave activity in 22% and an increase in beta-wave amplitude from 0.8 ± 0.1 mV to 1.1 ± 0.2 mV.

After complex therapy, 71% of patients showed a reduction of symptoms by more than 30%, 19% achieved clinical stabilization, and 10% had no significant improvement. Mean blood pressure decreased from 156/94 mmHg to 134/86 mmHg, and the lipid profile improved (cholesterol decreased from 6.2 ± 1.3 to 5.1 ± 0.9 mmol/L). Neuroprotective treatment contributed to a significant improvement in cognitive performance.

DISCUSSION

The study confirmed that DE pathogenesis is a multifactorial, multistage process. Arterial hypertension, atherosclerosis, dyslipidemia, endothelial dysfunction, and oxidative stress were identified as the major pathogenic links. A significant correlation was found between DE severity and white matter lesions on MRI ($r = 0.64$; $p < 0.01$), suggesting that clinical symptoms are closely related to morphological changes.

A negative correlation was also observed between blood pressure and MMSE score ($r = -0.57$; $p < 0.05$), emphasizing the importance of strict blood pressure control in DE management. Combined therapy with antihypertensive, nootropic, antioxidant, and angioprotective agents led to improved neuronal activity, reduced oxidative stress, and restored metabolic balance. Notably, patients treated with a combination of citicoline, piracetam, and vinpocetine achieved cognitive improvement 1.6 times faster.

Patients receiving additional physiotherapy and reflexotherapy showed a significant reduction in emotional instability and insomnia, underscoring the importance of psychosomatic factors in DE. According to the European Academy of Neurology (EAN, 2023), complex therapy can reduce DE symptoms by 25–35%. The findings of this study align with those data, confirming that personalized neuroprotective strategies tailored to age, disease stage, and comorbidities play a key role in improving treatment outcomes for DE patients.

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