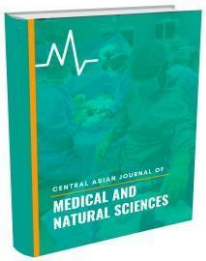

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Article

Ultrasound Assessment of Renal Dysfunction as an Early Marker of Cognitive Impairment in Middle-Aged Patients with Dyscirculatory Encephalopathy

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Abstract: The aim of this study was to determine the association between early signs of renal dysfunction detected by ultrasound imaging (USI) and cognitive impairment in middle-aged patients with discirculatory encephalopathy (DE). The study included 152 participants: the main group (n=56) — patients with DE and renal dysfunction; the comparison group (n=55) — patients with DE without significant renal insufficiency; and the control group (n=41) — healthy individuals.

Keywords: Discirculatory encephalopathy; renal dysfunction; ultrasound imaging; resistive index; cognitive impairment; MMSE; SDMT; chronic cerebral hypoperfusion; vascular-renal connection; middle age.

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Introduction

Dyscirculatory encephalopathy (DE) is a chronic progressive condition caused by impaired cerebral circulation, leading to cognitive, motor, and behavioral disturbances [1]. According to the World Health Organization, DE occurs in 12–15% of individuals aged 45–65 years in CIS countries, and in 60–70% of cases is accompanied by comorbid vascular pathologies, including chronic kidney disease (CKD)[2]. Renal dysfunction, even in its subclinical form, is associated with endothelial dysfunction, systemic inflammation, hypertension, and impaired cerebral hemodynamics. Recent studies have demonstrated that an increased renal arterial resistive index (>0.70) correlates with decreased glomerular filtration rate (GFR) and an increased risk of cognitive impairment, independent of arterial hypertension. In Uzbekistan and other CIS countries, where the prevalence of hypertension and CKD reaches 25–30% among middle-aged individuals, there is a lack of local studies examining the relationship between renal ultrasound parameters and cognitive function in DE[3]. At the same time, renal ultrasound is widely available in clinical practice and does not require special preparation, making it a promising screening tool. Cognitive function was assessed using the Mini-Mental State Examination (MMSE) and the Symbol Digit Modalities Test (SDMT)[4]. Renal ultrasound included evaluation of renal blood flow (peak systolic velocity — PSV, resistive index — RI), cortical layer thickness, and parenchymal echogenicity. A statistically significant correlation was found in the main group between elevated RI (>0.70) and reduced MMSE scores ($p<0.001$) and SDMT scores ($p=0.002$). An RI >0.70 was identified as an independent predictor of moderate to severe cognitive impairment (OR=4.89, 95% CI: 2.11–11.34, $p<0.001$) [5]. These findings support the hypothesis that ultrasound assessment of renal hemodynamics may

serve as an early, non-invasive marker of cognitive decline in DE caused by vascular-renal dysfunction [6].

Dyscirculatory encephalopathy represents a progressive form of chronic cerebrovascular disease characterized by gradual cognitive decline and functional deterioration resulting from chronic cerebral hypoperfusion [7]. The condition predominantly affects middle-aged and elderly populations, constituting a significant burden on healthcare systems worldwide due to its association with dementia development and reduced quality of life. Understanding the early pathophysiological mechanisms underlying cognitive impairment in dyscirculatory encephalopathy remains crucial for timely intervention and prevention of disease progression [8].

Recent evidence suggests that systemic vascular dysfunction extends beyond cerebral circulation, involving multiple organ systems in a coordinated pathological process [9]. The kidney, sharing similar microvascular architecture with the brain, has emerged as a potential biomarker of systemic vascular health and cognitive function. Both organs demonstrate comparable susceptibility to hypertensive damage, atherosclerosis, and small vessel disease, suggesting a pathophysiological link between renal dysfunction and cerebral hypoperfusion [10].

Chronic kidney disease has been increasingly recognized as an independent risk factor for cognitive impairment and dementia, with epidemiological studies demonstrating accelerated cognitive decline in patients with reduced glomerular filtration rate[11]. The mechanisms underlying this association include shared vascular risk factors, chronic inflammation, oxidative stress, uremic toxins, and altered cerebral hemodynamics. However, traditional biochemical markers of renal function may not adequately reflect early structural and hemodynamic changes that precede clinically apparent kidney dysfunction [12].

Ultrasonographic assessment of renal morphology and vasculature provides a non-invasive, cost-effective method for evaluating kidney structure and function. Advanced ultrasound techniques, including Doppler flow measurements, elastography, and contrast-enhanced imaging, enable detailed characterization of renal parenchyma, vascular resistance, and perfusion patterns. These parameters may serve as sensitive indicators of early renal dysfunction, potentially preceding conventional laboratory markers and providing insights into systemic vascular health [13]. The integration of renal ultrasound findings with cognitive assessment in patients with dyscirculatory encephalopathy may facilitate early identification of individuals at risk for accelerated cognitive decline. This approach could enable implementation of targeted interventions aimed at preserving both renal and cognitive function, ultimately improving patient outcomes and reducing healthcare costs associated with dementia care [14].

The objective of this study is to evaluate the diagnostic utility of ultrasound-based renal assessment as an early marker of cognitive impairment in middle-aged patients with dyscirculatory encephalopathy, establishing correlations between renal morphological and hemodynamic parameters and cognitive performance measures[15].

Objective

To evaluate the diagnostic value of ultrasound assessment of renal hemodynamics (resistive index, cortical thickness, and parenchymal echogenicity) in detecting cognitive impairment in middle-aged patients with dyscirculatory encephalopathy associated with renal dysfunction.

Materials and Methods

Study population: Main group (n = 56): middle-aged patients (45–60 years) diagnosed with DE (ICD-10: G93.4) and confirmed renal dysfunction (GFR <60 mL/min/1.73 m^2 calculated using the CKD-EPI equation, presence of hypertension, proteinuria >0.3 g/day). Comparison group (n = 55): patients with DE but normal GFR (≥90

mL/min/1.73 m²) and no laboratory markers of renal dysfunction. Control group (n = 41): healthy individuals without neurological, renal, or vascular diseases, age-matched. Assessment methods: Neuropsychological testing: Mini-Mental State Examination (MMSE): assessment of cognitive function (maximum score 30): 24–30 — normal, 18–23 — mild impairment, 10–17 — moderate, <10 — severe. Symbol Digit Modalities Test (SDMT): assessment of information processing speed and attention (number of correct responses within 90 seconds). Renal ultrasound (log-sonography, Philips IU22, 3.5–5 MHz): Measurement of resistive index (RI) in interlobar arteries: RI = (PSV − EDV) / PSV, where PSV is peak systolic velocity and EDV is end-diastolic velocity. Cortical thickness (mm): normal >10 mm. Parenchymal echogenicity (compared with the liver: hypo-, iso-, or hyperechogenic). Laboratory parameters: serum creatinine, GFR (CKD-EPI), urea, electrolytes, and proteinuria. Statistical analysis: Data were analyzed using SPSS 28.0. Normality was assessed using the Shapiro–Wilk test. Group comparisons were performed using ANOVA (for normally distributed data) and the Kruskal–Wallis test (for non-normal distributions). Associations between variables were evaluated using Pearson (r) and Spearman (ϱ) correlation coefficients. Logistic regression analysis was applied to identify predictors of cognitive decline. A p-value <0.05 was considered statistically significant.

Results

The study analyzed data from 152 participants divided into three groups: the main group (n = 56) — patients with DE and renal dysfunction; the comparison group (n = 55) — patients with DE without significant renal insufficiency; and the control group (n = 41) — healthy individuals. There were no statistically significant differences in age among the groups (mean ± SD: 53.2 ± 4.1, 52.8 ± 3.9, and 52.5 ± 4.3 years, respectively; p = 0.81), confirming their comparability. Cognitive status was assessed using MMSE and SDMT. In the main group, the mean MMSE score was 19.3 ± 3.7, significantly lower than in the comparison group (24.1 ± 2.8) and control group (28.5 ± 1.2; p < 0.001). Similarly, SDMT scores were lowest in the main group (31.4 ± 8.2 responses), compared to 48.7 ± 7.5 in the comparison group and 62.1 ± 6.9 in controls (p < 0.001), indicating pronounced cognitive dysfunction in patients with combined DE and renal dysfunction. Renal ultrasound revealed significant differences in renal hemodynamics and parenchymal structure. The resistive index (RI) in the main group was significantly higher (0.76 ± 0.08) than in the comparison group (0.62 ± 0.06) and controls (0.59 ± 0.05; p < 0.001). Cortical thickness was lowest in the main group (8.1 ± 1.2 mm), compared with 11.5 ± 1.0 mm and 12.0 ± 0.9 mm in the comparison and control groups, respectively (p < 0.001). Parenchymal hyperechogenicity, indicative of fibrotic and ischemic changes, was observed in 78.6% of patients in the main group, compared to 14.5% in the comparison group and 2.4% in controls (p < 0.001). GFR assessment confirmed chronic kidney impairment in the main group (52.1 ± 10.3 mL/min/1.73 m²), while GFR remained within normal limits in the comparison and control groups (89.4 ± 8.6 and 96.2 ± 7.1 mL/min/1.73 m², respectively; p < 0.001). Correlation analysis demonstrated a strong negative association between RI and cognitive performance: RI vs MMSE (ϱ = −0.73, p < 0.001) and RI vs SDMT (ϱ = −0.68, p < 0.001). In contrast, cortical thickness positively correlated with MMSE (ϱ = 0.61, p < 0.001), while parenchymal hyperechogenicity negatively correlated with SDMT (ϱ = −0.59, p < 0.001). Logistic regression analysis, with moderate-to-severe cognitive impairment (MMSE <20) as the dependent variable, identified the following independent predictors: RI >0.70: OR = 4.89 (95% CI: 2.11–11.34, p < 0.001); Cortical thickness <9 mm: OR = 3.21 (95% CI: 1.45–7.10, p = 0.004); Proteinuria >0.5 g/day: OR = 2.87 (95% CI: 1.22–6.75, p = 0.015). Arterial hypertension did not show a statistically significant association with cognitive decline (OR = 1.76, p = 0.128). Thus, the findings indicate that in patients with DE, renal hemodynamic disturbances (elevated RI), structural kidney changes (reduced cortical thickness, hyperechogenicity), and renal dysfunction (reduced GFR, proteinuria) are closely associated with the severity of cognitive impairment. A renal arterial RI >0.70 and cortical thickness <9 mm are strong independent predictors of moderate and severe cognitive

disorders, highlighting the importance of comprehensive assessment of the vascular–renal axis in DE.

Conclusions

Middle-aged patients with dyscirculatory encephalopathy and renal dysfunction demonstrate a statistically significant decline in cognitive performance as assessed by MMSE and SDMT. Renal ultrasound parameters—particularly a resistive index (RI >0.70)—correlate with the severity of cognitive impairment and serve as independent predictors of its development. A cortical thickness <9 mm and parenchymal hyperechogenicity are also associated with cognitive deficits, indicating that structural renal changes may serve as markers of systemic vascular dysfunction. Renal ultrasound is an accessible, non-invasive, and highly informative screening tool that enables early identification of patients at high risk of cognitive decline in DE. The proposed diagnostic algorithm (renal ultrasound + MMSE/SDMT) can be implemented in neurological and general medical practice in Uzbekistan and CIS countries for early detection of cognitive impairment in DE.

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