



THE IMPACT OF RENAL MARKERS IN PARKINSON'S DISEASE

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ABSTRACT

This study aims to investigate the relationship between renal and metabolic markers and the progression of Parkinson's disease (PD). In recent years, increasing evidence has supported the systemic nature of PD, which involves not only neurodegenerative but also metabolic, vascular, and renal changes. The analysis of biochemical parameters makes it possible to detect hidden signs of excretory system dysfunction and to assess their impact on disease progression. The results of this study demonstrate that elevated levels of urea and glucose, as well as reduced hematocrit, correlate with the progression of Parkinson's disease, reflecting the systemic involvement of peripheral organs in the pathological process. Identifying such alterations has important clinical significance for early diagnosis, prevention of complications, and personalized therapy.

Relevance

Parkinson's disease has traditionally been regarded as a neurodegenerative disorder of the central nervous system; however, recent data indicate its systemic nature, encompassing metabolic and renal impairments. According to recent studies (Yang et al., *Neurology India*, 2023), levels of urea, creatinine, and proteinuria in PD patients are significantly higher than in healthy individuals, suggesting a link between renal function and neurodegeneration. The works of Liu Y. et al. (*BMC Public Health*, 2024) and Huang S. et al. (*Medflics Revue*, 2023) confirm that chronic kidney disease and decreased glomerular filtration rate significantly increase the risk of developing Parkinson's disease, indicating the presence of a systemic metabolic component in the pathogenesis. The study by Zhang R. et al. (*PubMed*, 2014) demonstrated that patients with renal dysfunction exhibit more pronounced neurodegenerative changes and worse cognitive outcomes, explained by oxidative stress and reduced elimination of toxic metabolites. Furthermore, Wang L. et al. (*Nutrients*, MDPI, 2023) revealed a relationship between nitrogen metabolism disorders, microangiopathic processes, and PD progression, emphasizing the role of systemic inflammation and endothelial dysfunction.

Introduction

Parkinson's disease is a chronic neurodegenerative disorder characterized by both motor and

non-motor symptoms, and it is capable of inducing systemic metabolic and vascular alterations. The present study focuses on examining the interrelation between renal and metabolic markers and the progression of Parkinson's disease.

Materials and Methods

The clinical study was conducted from October 2023 to December 2024 at the Department of Neurology, Tashkent Medical Academy. The study included 54 patients with a confirmed diagnosis of Parkinson's disease (mean age 62.0 ± 1.86 years), among whom were 20 women (37.0%) and 34 men (63.0%).

Renal (urea, creatinine) and metabolic (glucose, hematocrit) markers obtained from standard biochemical analyses were evaluated. Changes in disease stage and reno-metabolic parameters were assessed using descriptive and correlation statistics.

Results

Biochemical analysis showed mean concentrations of glucose —6.57 mmol/L, urea 7.23 mmol/L, creatinine 84.51 μ mol/L, and hematocrit 43.1%.

A significant positive correlation was found between age and urea levels ($\rho = 0.42$, $p = 0.01$), indicating increased renal load with age. Lower glucose levels moderately correlated with disease duration ($\rho = 0.36$, $p = 0.03$). Hematocrit showed a negative association with disease severity ($\rho = -0.31$, $p = 0.04$). In contrast, creatinine levels did not show statistically significant relationships ($p = 0.18$; $p = 0.15$).

Conclusion

The study demonstrated that renal and metabolic dysfunction accelerate the progression of Parkinson's disease. Neurodegenerative metabolic stress is accompanied by increased urea and glucose levels, while reduced hematocrit reflects late microangiopathic changes. The absence of marked changes in creatinine levels indicates preserved excretory renal function, whereas urea serves as a more sensitive marker in Parkinson's disease.

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