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Age and Cognitive Impairment in Dialysis Patients: A Cross-Sectional Analysis

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ABSTRACT

Background: Cognitive impairment is a frequent yet underrecognized complication in dialysis patients, adversely affecting quality of life and clinical outcomes. With the growing prevalence of chronic kidney disease (CKD), understanding the relationship between dialysis and cognitive decline is increasingly important.
Methods: This cross-sectional study was conducted in 3 major nephrology centers in Türkiye. Cognitive function was assessed using the AD8 Dementia Screening Interview. Demographic, clinical, laboratory data, and dialysis adequacy (Kt/V) were analyzed for associations with cognitive impairment.
Results: A total of 261 dialysis patients were included (54.4% male; median age: 56 years, range 18-89; 29.1% ≥65 years). Among them, 57.9% received hemodialysis and 42.1% peritoneal dialysis, with a median kidney replacement therapy duration of 3 years. Cognitive impairment was present in 63.6% of patients. These patients had a longer CKD duration (10 vs. 6 years, $P < .001$) but lower hypertension prevalence (65.2% vs. 84.4%, $P = .001$). Cognitive decline was more common in hemodialysis patients (66.3% vs. 43.2%, $P < .001$) and associated with lower hemoglobin ($P = .033$) and sodium levels ($P = .019$). In the multivariable logistic regression model, age was not an independent predictor ($P = .315$); correlates of cognitive impairment included longer CKD duration ($P = .001$), hemodialysis modality ($P = .003$), and lower sodium levels ($P = .014$).
Conclusion: Cognitive impairment was highly prevalent among dialysis patients, independent of age. Recognition of modifiable risk factors may help guide multidisciplinary strategies to improve cognitive and overall patient outcomes.
Keywords: Chronic kidney disease, cognitive impairment, hemodialysis, hyponatremia, peritoneal dialysis

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INTRODUCTION

Cognitive dysfunction in end-stage kidney disease (ESKD) has emerged as one of the most critical challenges in modern nephrology due to its profound socioeconomic implications and its impact on patient outcomes.[1] As the global prevalence of chronic kidney disease (CKD) continues to rise, the burden of cognitive impairment among patients in advanced stages of kidney disease has garnered increased attention.

Studies indicate a strong correlation between the decline in glomerular filtration rate and the severity of cognitive deficits, affecting domains such as memory, language, attention, comprehension, and executive function. Also, kidney replacement therapies (KRTs) in ESKD may contribute to cognitive impairment. In this patient group, additional risk factors such as hypoxemia, significant osmolar fluctuations, and intermittent exposure to uremic toxins exacerbate cognitive changes. These factors, combined with chronic systemic inflammation, oxidative stress, and vascular pathology, play a significant role in the development and progression of cognitive dysfunction. These mechanisms not only impair cognitive reserve but

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also exacerbate pre-existing neurological vulnerabilities, particularly in older adults and those with comorbidities such as diabetes or hypertension.[2-5] It is concluded that cognitive dysfunction in ESKD is associated with a higher risk of mortality, underscoring the urgent need for early identification, preventive strategies, and targeted interventions to mitigate these effects.[6]

This study aimed to evaluate the cognitive impairment in dialysis patients and to investigate its potential associations with clinical, demographic, and biochemical factors.

MATERIAL AND METHODS

This cross-sectional study was conducted at 3 nephrology centers in Türkiye: Hacettepe University Faculty of Medicine, Atatürk University Faculty of Medicine and Gazi University Faculty of Medicine. The aim was to evaluate the prevalence of cognitive impairment and its associated factors among dialysis patients.

The study protocol was approved by the Hacettepe University Ethics Committee (Approval Number: Sba 24/825, Date: September 26, 2024). Written informed consent was obtained from all participants before inclusion in the study. All procedures were conducted in accordance with the Declaration of Helsinki and relevant ethical guidelines.

The study included patients undergoing hemodialysis (HD) or peritoneal dialysis (PD) longer than 6 months. Patients with severe psychiatric illnesses, known neurodegenerative diseases, acute infections, or recent major surgeries were excluded.

MAIN POINTS

- In this study, cognitive impairment was common among dialysis patients and was not determined by age, suggesting that factors beyond natural aging are the main drivers of cognitive decline.
- The association of longer chronic kidney disease duration and lower serum sodium levels with impaired cognition indicates that metabolic and vascular disturbances are central mechanisms rather than chronological aging.
- The higher prevalence of cognitive impairment in hemodialysis compared with peritoneal dialysis points to the potential adverse cognitive effects of hemodynamic instability and rapid solute shifts during hemodialysis sessions.
- These results emphasize that routine cognitive screening should be integrated into the care of all dialysis patients, regardless of age, as an observational finding supporting early detection and monitoring strategies.
- As this was a cross-sectional study with a relatively limited sample size, causal inferences cannot be drawn; however, recognizing these non–age-related determinants may provide a basis for further research and tailored clinical approaches that could improve cognitive outcomes in this population.

Demographic data, including age, gender, and educational level, along with clinical parameters such as dialysis modality, duration of dialysis, and comorbid conditions, were collected. Laboratory parameters (blood urea nitrogen, serum creatinine, sodium, potassium, calcium, phosphorus, hemoglobin, leukocyte count, platelet count, ferritin, transferrin saturation, parathormone levels) were obtained from medical records.

In this study, the AD8 Dementia Screening Interview was used to assess cognitive impairment among dialysis patients. The AD8 is a brief, validated screening tool designed to detect early dementia through patient or informant reports.[7] The AD8 has not been validated specifically in dialysis populations and may be influenced by educational and cultural factors; however, it has been used effectively in large HD cohorts.[8] The AD8 was used as a screening instrument for cognitive impairment rather than for diagnostic classification of dementia. It consists of 8 yes/no questions evaluating changes in memory, problem-solving, judgment, and daily functioning. Each “Yes” response scores 1 point, while “No” or “No Change” responses receive zero points. A total score of ≥2 suggests cognitive impairment, while a score of 1 indicates slight cognitive impairment and 0 indicates no significant cognitive decline. The AD8 questionnaire was administered before the dialysis session to minimize post-dialysis cognitive fluctuation. The questionnaire was administered either directly to patients (self-report) or to an informant, such as a caregiver or close relative, when available. Patients scoring 2 or higher were classified as having suspected dementia and were assigned to the dementia group for statistical analysis. The AD8 was selected due to its high sensitivity and specificity in detecting mild cognitive impairment and early-stage dementia, making it a practical and reliable tool for screening in clinical settings.

Risk factors potentially influencing cognitive impairment, including anemia, blood pressure variability, uremic toxins, dialysis adequacy measures, including Kt/V, were evaluated. Patients’ adherence to treatment protocols was assessed through clinical interviews and dialysis records.

Baseline characteristics were summarized using descriptive statistics. Cognitive impairment was defined based on established cutoff scores for the AD8 score tools. Comparative analyses (e.g., *t*-tests, chi-square tests) were used to examine differences between groups with and without cognitive impairment. Multivariate logistic regression was applied to identify independent predictors of cognitive impairment, adjusting for potential confounders such as age, educational level, and dialysis duration. A *P*-value <.05 was considered statistically significant.

RESULTS

A total of 261 dialysis patients were included in the study (142 males (54.4%) and 119 females (45.6%)). The median age of the participants was 56.0 years (interquartile range (IQR): 43.0-67.0), with 29.1% of the patients aged 65 years or older. The median

duration of CKD was 7.0 years (IQR: 4.0-14.0 years), and diabetes mellitus (18.5%) and hypertension (22.3%) were identified as the most common underlying etiologies. The cohort included patients undergoing HD (57.9%) and PD (42.1%), with a median duration of KRT of 3.0 years (IQR: 1.0-7.0 years). The median Kt/V value, reflecting dialysis adequacy, was 1.7 (IQR: 1.4-2.0). Biochemical parameters revealed median blood urea nitrogen (BUN) levels of 55.9 mg/dL, creatinine levels of 7.9 mg/dL, and hemoglobin levels of 11.0 g/dL, ferritin level of 401.0 ng/mL and parathyroid hormone levels of 383.3 pg/mL (Table 1).

When the groups were evaluated using the AD8 score system, which monitors cognitive impairment, 58 patients (22.2%) scored 0 points, indicating no cognitive impairment, and 37 patients (14.2%) scored 1 point, while 166 patients scored ≥2 points. AD8 scores were correlated with BUN levels (r = 0.144, P = .020), Kt/V values (r = -0.176, P = .004), CKD duration (r = 0.303, P < .001), and serum sodium levels (r = −0.178, P = .004).

Gender distribution was similar between the patients with cognitive impairment and patients with normal cognition (female patients in cognitive impairment group: 47.0%, female patients in group with normal cognitive function: 43.2%, P = .606), indicating no significant difference in the proportion of male and female patients. The mean age was slightly higher in the cognitively impaired group (55.0 ± 7.6 vs. 53.5 ± 14.7 years), but this difference was not statistically significant (P = .452). The prevalence of elderly patients (>65 years) was not statistically different between groups (31.3% vs. 25.3%, P = .324). Regarding comorbid conditions, no significant differences were observed between the groups for diabetes mellitus (P = .199) or coronary artery disease (P = .219). However, the prevalence of hypertension was statistically significantly lower in the cognitive impairment group (65.2% vs. 84.4%, P = .001). The median duration of CKD was significantly longer in patients with cognitive impairment (10 vs. 6 years, 95% CI: 0.000-0.001). Analysis of KRT type revealed a significant association between dialysis modality and cognitive impairment with a higher proportion of HD patients in the cognitively impaired group (66.3% vs. 43.2%, P < .001). In contrast, PD was more common among patients without cognitive impairment (56.8% vs. 33.7%). Regarding PD type, there was no significant difference in the distribution of automated PD and continuous ambulatory PD between groups (P = .567). Regarding vascular access in the HD patients, the distribution of arteriovenous fistula, arteriovenous graft, and central venous catheter use did not differ significantly between the 2 groups (P = .329) (Table 2).

Among biochemical parameters, hemoglobin levels (10.8 ± 1.7 vs 11.3 ± 1.7, P = .033) and sodium levels (137.3 ± 3.7 vs 138.4 ± 3.2, P = .019) were significantly lower in the cognitive impairment group. No significant differences were found for BUN, creatinine, potassium, calcium, phosphorus, hemoglobin, leukocyte, thrombocyte, parathormone, ferritin, or transferrin saturation levels between groups (P > .05) (Table 3). When laboratory parameters were evaluated based on the targets recommended by Kidney Disease: Improving Global Outcomes (KDIGO CKD) guidelines[9] and laboratory reference values, categorizing them as within or outside the normal range, an abnormal serum sodium level was the only parameter significantly associated with cognitive impairment (26.5% vs. 8.42%, P < .001).

When the study population was divided into 2 groups based on age (>65 years and ≤65 years), there were no differences according to the frequency of cognitive impairment (≤65 years: patients with 114/185 (61.62%) vs >65 years patients with cognitive impairment: 52/76 (68.42%), P = .300). In the elderly

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population

	Study Population (n = 261)
Gender (female), n (%)	119 (54.4)
Age (years)	56 (45-67)
>65 years, n (%)	76 (29.1)
Weight (kg)	69.0 (58.4-80.0)
Number of comorbid diseases	2 (1-3)
Number of medications (tablets per day)	10 (6-15)
Chronic kidney disease duration (years)	7 (4-14)
Chronic kidney disease etiology, n (%)	
Hypertension	58 (22.3)
Glomerulonephritis	58 (22.3)
Diabetes mellitus	48 (18.5)
Kidney replacement therapy type, n (%)	
Hemodialysis	151 (57.9)
Peritoneal dialysis	110 (42.1)
Kt/V	1.69 (1.4-2.0)
Blood urea nitrogen (mg/dL)	55.9 (49.9-69.1)
Creatinine (mg/dL)	7.9 (5.9-9.6)
Sodium (mEq/L)	138.0 (136.0-140.0)
Potassium (mEq/L)	4.6 (4.2-5.2)
Calcium (mg/dL)	8.9 (8.5-9.5)
Phosphorus (mg/dL)	4.9 (4.2-5.9)
Hemoglobin (g/dL)	11.0 (9.8-12.1)
Leukocytes (×10^3/μL)	6.7 (5.4-8.0)
Platelets (×10^3/μL)	205.5 (165.0-259.3)
Parathyroid hormone (pg/mL)	383.3 (219.6-679.1)
Ferritin (ng/mL)	401.0 (221.0-599.0)
Transferrin saturation (%)	25.0 (19.0-35.0)

Variables are presented as median (IQR) for continuous data and as number (percentage) for categorical data.

Table 2. Comparison of Demographic Characteristics and Dialysis-Related Factors of the Study Group According to the Presence of Cognitive Dysfunction

	Patients with Cognitive Impairment (n = 166)	Patients Without Cognitive Impairment (n = 95)	*P*
Gender, n (%)			.606
Female	78 (47.0)	41 (43.2)	
Male	88 (53.0)	54 (56.8)	
Age (years)	57.0 (44.0-67.0)	55.0 (41.5-64.5)	.452
>65 years, n (%)	52 (31.3)	24 (25.3)	.324
Weight (kg)	70.0 (60.0-80.0)	67.5 (59.0-78.0)	.042
Number of comorbid diseases	2 (1-5)	2 (1-5)	.413
Comorbid diseases, n (%)			
Diabetes mellitus	53 (32.2)	22 (24.4)	.199
Hypertension	107 (65.2)	76 (84.4)	.001
Coronary artery disease	35 (21.3)	26 (28.9)	.219
Number of medications (tablets per day)	10 (6-15)	10 (5-14)	.059
Chronic kidney disease duration (years)	10.0 (6.0-15.0)	6.0 (4.0-12.0)	<.001
CKD etiology, n (%)			.812
Hypertension	35 (21.2)	23 (24.2)	
Glomerulonephritis	39 (23.6)	19 (20.0)	
Diabetes mellitus	33 (20)	15 (15.8)	
Kidney replacement therapy type, n (%)			<.001
Hemodialysis	110 (66.3)	41 (43.2)	
Peritoneal dialysis	56 (33.7)	54 (56.8)	
Peritoneal dialysis type, n (%)			.567
CAPD	30 (53.6)	25 (46.3)	
APD	26 (46.4)	29 (53.7)	
Hemodialysis vascular access, n (%)			.329
Arteriovenous fistula	69 (62.7)	25 (61.0)	
Arteriovenous graft	5 (4.5)	0 (0.0)	
Central venous catheter	36 (32.7)	16 (39.0)	
Kt/V	1.6 (1.4-1.9)	1.7 (1.4-2.1)	.351

Variables are presented median (IQR) for continuous data and as number (percentage) for categorical data.
APD, automated peritoneal dialysis; CAPD, continuous ambulatory peritoneal dialysis.

population, cognitive impairment was significantly associated with prolonged CKD duration (6.25 (1-46) vs 5.50 (1-13), *P* < .001), lower serum sodium levels (137.00 (130.00-146.00) vs. 139.50 (135.00-144.00), *P* = .013), and KRT type (HD: 75.00% vs 45.80%, *P* = .019). In the younger population (≤65 years), cognitive impairment was significantly associated with lower prevalence of hypertension (58.00% vs. 83.00%, *P* = .001), longer CKD duration (10.50 (0-46) vs. 6.00 (1-30), *P* = .001), KRT type (HD: 62.30% vs 42.30%, *P* = .008), and abnormal serum sodium levels (24.60% vs 11.30%, *P* = .035).

In the logistic regression analysis, prolonged CKD duration (Exp(B) = 1.065, *P* = .001, 95% CI: 1.025-1.106; Fig. 1), HD as the KRTmodality (Exp(B) = 0.439, *P* = .003, 95% CI: 0.254-0.759), and lower serum sodium levels (Exp(B) = 0.905, *P* = .014, 95% CI: 0.836-0.980; Fig. 2) were consistently associated with an increased likelihood of cognitive impairment.

In the multivariable logistic regression model, the overall model was significant (χ^2 = 37.788, *P* < .001), explaining approximately 18.6% of the variance (Nagelkerke R^2 = 0.186) with a correct classification rate of 68.3%. Age was not a significant predictor of cognitive impairment (*P* = .315; adjusted odds ratio (aOR) = 1.009, 95% CI: 0.991-1.027). Independent predictors included longer CKD duration (*P* = .001; aOR = 1.065, 95% CI: 1.025-1.106), HD modality (*P* = .003; aOR = 0.439, 95% CI: 0.254-0.759), and lower serum sodium levels (*P* = .014; aOR = 0.905, 95% CI: 0.836-0.980).

DISCUSSION

Our study examined 261 dialysis patients and evaluated cognitive function using the AD8 screening tool. The results indicate that several factors—specifically prolonged CKD duration, lower serum sodium levels, and the use of HD as KRT—are significantly associated with cognitive impairment. Also,

Table 3. Comparison of Laboratory Values of the Study Group According to the Presence of Cognitive Dysfunction

	Dialysis Patients with Cognitive Impairment (n = 166)	Dialysis Patients Without Cognitive Impairment (n = 95)	*P*
Blood urea nitrogen (mg/dL)	53.0 (45.0-66.9)	57.9 (47.7-71.0)	.169
Creatinine (mg/dL)	8.1 (6.1-9.9)	7.7 (5.5-9.1)	.325
Sodium (mEq/L)	137.0 (135.0-140.0)	139.0 (137.0-140.0)	.019
Potassium (mEq/L)	4.5 (4.1-5.2)	4.7 (4.3-5.3)	.228
Calcium (mg/dL)	9.1 (8.5-9.6)	8.8 (8.3-9.4)	.125
Phosphorus (mg/dL)	4.9 (4.1-6.0)	4.5 (3.8-5.4)	.859
Hemoglobin (g/dL)	10.8 (9.5-12.0)	11.4 (10.3-12.4)	.033
Leukocyte count (×10^3/μL)	6.7 (5.4-7.9)	6.7 (5.4-8.1)	.547
Platelet count (×10^3/μL)	203.0 (161.0-256.0)	210.0 (177.5-266.5)	.668
Parathyroid hormone (pg/mL)	240.0 (130.0-420.0)	210.0 (110.0-355.0)	.631
Ferritin (ng/mL)	200.0 (130.0-310.0)	190.0 (120.0-280.0)	.697
Transferrin saturation (%)	26.7 (19.0-38.4)	24.6 (19.0-30.0)	.210

Variables are presented as median (IQR) for continuous data and as number (percentage) for categorical data.

hypertension was more frequent in the group with better cognitive function. The prevalence of cognitive impairment was similarly high among both younger and older dialysis patients, suggesting that factors other than age may play a more critical role in its development.

In this study, 63.60% of the study population had cognitive impairment. Cognitive impairment is common in dialysis patients and can be up to 70%.[10,11] A longer duration of CKD was observed in patients with cognitive impairment, and logistic regression analysis confirmed that each additional year of CKD duration was associated with an increased likelihood of cognitive impairment in this study. These findings are also consistent with current literature.[12] This finding may be reflective of the cumulative burden of uremic toxins, vascular damage, and chronic inflammation that can adversely affect cerebral function over time. This relationship highlights the need for strategies aimed at mitigating the chronic effects of uremia, vascular damage, and inflammation. Also, early detection of cognitive deficits in the dialysis population, regardless of age, can facilitate timely interventions and help tailor patient management strategies. This strategy could also help to reduce the

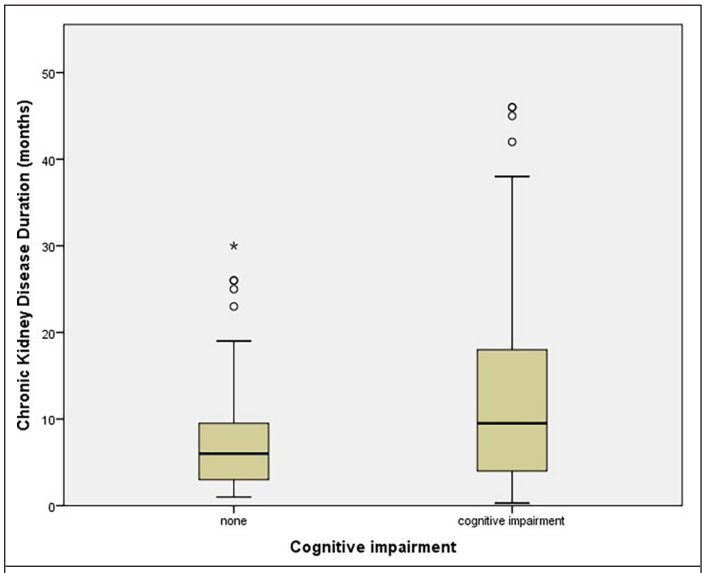

Figure 1. Relation of the chronic kidney disease duration with cognitive impairment in the study population.

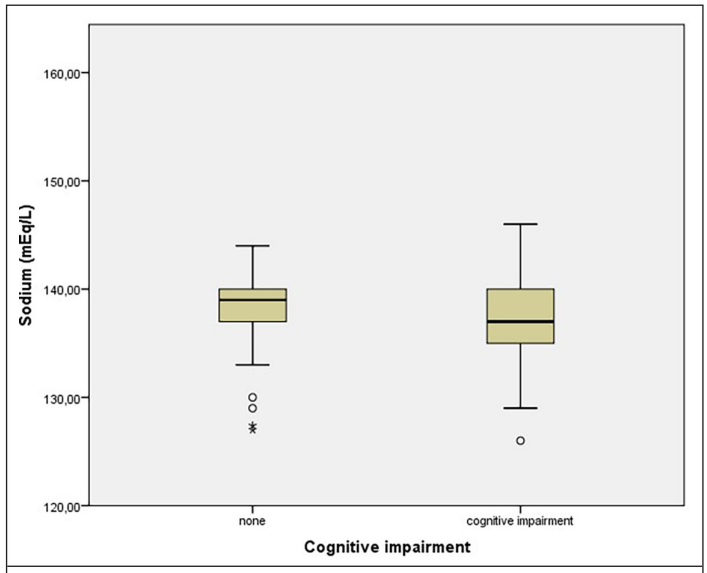

Figure 2. Relation of the serum sodium levels with cognitive impairment in the study population.

economic, social, and healthcare burdens associated with cognitive impairment.

The type of dialysis modality also emerged as a significant factor for cognitive impairment in this study. Patients undergoing HD were more likely to exhibit cognitive impairment compared to those on PD. Patients with PD are reported to have better cognitive function in different patient groups in previous studies.[5,13,14] Neumann et al[14] (2018) reported that PD patients maintained superior executive and attention functions compared with matched HD patients over 1 year, while Tian et al[5] (2019), in a Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA)-compliant meta-analysis, showed lower dementia risk among PD populations. This observation might be explained by the hemodynamic fluctuations and rapid shifts in solute levels inherent to HD, which can contribute to cerebral hypoperfusion or ischemic events. In contrast, the continuous nature of PD may provide a more stable metabolic environment that could potentially be protective against cognitive decline. Recent neuroimaging evidence suggests that intradialytic cerebral blood flow variation may contribute to cognitive decline among HDpatients.[16] Recent data have also implicated endothelial dysfunction as a potential contributor to cognitive decline in dialysis populations.[17] Weighing the potential cognitive risks when deciding on the modality of KRT, particularly in patients who already exhibit or are at high risk for cognitive decline, can be an important factor in avoiding progressive cognitive decline in the CKD population.

Electrolyte imbalance, specifically lower serum sodium levels, was another independent predictor of cognitive impairment. Hyponatremia has been linked in other studies to altered neuronal function and may serve as a marker of overall metabolic instability in patients with advanced CKD.[15,18] Experimental and clinical evidence suggests that chronic low sodium levels can disrupt osmotic balance in astrocytes and neurons, leading to cerebral edema, impaired neurotransmission, and attention or memory deficits. In dialysis populations, Shavit et al[15] (2014) reported that even mild hyponatremia was associated with reduced functional and cognitive performance among HD patients, while Xu et al[18] (2015) demonstrated a similar relationship in PD patients, identifying hyponatremia as an independent predictor of cognitive impairment. The consistent association between abnormal serum sodium levels and cognitive dysfunction emphasizes the need for close monitoring and management of electrolyte disturbances in this vulnerable population.

Based on the study results, although the cognitively impaired group had a slightly higher mean age, this difference was not statistically significant. Additionally, the proportion of elderly patients was similar between groups. Therefore, age by itself does not appear to be an independent risk factor for cognitive impairment in this cohort of dialysis patients. Model diagnostics indicated an adequate fit and confirmed that age, while related to comorbidity and disease duration, was not an independent determinant after adjustment. Longer CKD duration, HD modality, and lower serum sodium levels remained significant correlates. The current findings are broadly consistent with previous international reports examining cognitive outcomes in dialysis populations. In a large Taiwanese cohort, Chou et al[8] (2014) used the AD8 instrument in 1617 HD patients and demonstrated that longer dialysis duration—rather than age—was associated with lower dementia risk, emphasizing treatment-related rather than chronological determinants. Similarly, the study by Wolfgram et al[13] (2015) reported that dialysis modality and clinical variables were the principal determinants of cognitive outcomes. These findings indicate that the dialysis population can be vulnerable to cognitive impairment independent of age.

Interestingly, while hypertension is a well-established risk factor for cognitive decline in the general population, it has been observed that patients with cognitive impairment tend to have a lower prevalence of hypertension compared to those with preserved cognition.[19] This counterintuitive result suggests that in the context of dialysis, higher blood pressure might be a marker of better overall hemodynamic stability or less advanced systemic disease. For example, lower blood pressure in this population may reflect underlying cardiovascular compromise, malnutrition, or autonomic dysfunction, all of which could adversely affect cerebral perfusion and cognitive function. It is also possible that antihypertensive treatments, when properly managed, could have neuroprotective effects that help preserve cognitive function.[20] However, this paradoxical association requires further investigation to fully understand the complex interplay between blood pressure, its management, and cognitive outcomes in dialysis patients.

While this study provides valuable insights, its cross-sectional design limits causal interpretation. Therefore, these findings should be understood as observational associations rather than evidence of causation. Longitudinal studies are warranted to clarify whether these associations represent causal pathways. Single-time laboratory measures may not capture longitudinal variability; future studies should use serial data. Also, other potentially relevant clinical factors, such as intradialytic hypotension, nutritional status, and inflammatory markers, were not assessed in this analysis because these data were not uniformly available across participating centers. Nonetheless, previous studies have reported associations between recurrent hypotension, malnutrition, systemic inflammation, and cognitive decline in dialysis populations, suggesting that these parameters may contribute to neurocognitive vulnerability and merit inclusion in future longitudinal studies.

In summary, this study suggests that prolonged CKD duration, lower serum sodium levels, and the predominance of HD as a treatment modality were associated with a higher likelihood of cognitive impairment among dialysis patients. Cognitive

impairment was also observed across all age groups, suggesting that age alone may not fully explain these cognitive changes in this population. While causal inferences cannot be drawn, the consistent association between dialysis-related factors and cognitive impairment highlights potential targets for further investigation.

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

Artificial Intelligence Usage Statement: The authors declared that no Artificial Intelligence tool was used in the preparation of the manuscript.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of Hacettepe University (Approval No.: Sba 24/825; Date: 26.09.2024).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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