BMC Nephrology

RESEARCH

Open Access

Association of abdominal aortic calcification with cognitive impairment in peritoneal dialysis patients

Conghui Liu[1], Yanan Shi[1], Jiajie Cai[1], Tingting Bai[1], Jingjing Li[1], Lingju Yue[1] and Zhongxin Li[1*]

Abstract

Background Patients on peritoneal dialysis (PD) frequently have cognitive impairment, which is linked to a poor prognosis. The purpose of this study was to determine whether abdominal aortic calcification (AAC) may have an impact on PD patients' cognitive function.

Methods In this cross-sectional study of 110 PD patients, cognitive function was assessed using the Montreal Cognitive Assessment (MoCA), and AAC severity was quantified via lateral lumbar radiography (Kauppila method). Participants were stratified by AAC severity into high (HAAC; score ≥ 4) and low (LAAC; score < 4) groups.

Results Cognitive impairment (MoCA < 26) was present in 65.45% of patients. The HAAC group (71.8% of cohort) exhibited distinct metabolic profiles compared to LAAC: older age (63.2 ± 9.8 vs. 47.4 ± 12.1 years, $P < 0.001$), higher diabetes prevalence (68.4% vs. 22.6%, $P < 0.001$), elevated serum phosphorus (1.62 ± 0.45 vs. 1.30 ± 0.42 mmol/L, $P < 0.001$), and lower diastolic blood pressure (79.2 ± 10.8 vs. 86.6 ± 13.4 mmHg, $P = 0.005$). Notably, HAAC patients had reduced serum creatinine (898.4 ± 251.9 vs. 1190.7 ± 243.5 μmol/L, $P < 0.001$) and iPTH levels (142.5 vs. 218.0 pg/mL, $P = 0.011$), suggesting concurrent mineral bone disorder. Multivariate analysis identified AAC severity (OR = 1.28 per 1-point increase, 95%CI = 1.09–1.50) and age (OR = 1.12/year, 95%CI = 1.06–1.19) as independent predictors of cognitive impairment.

Conclusion AAC severity demonstrates a strong, dose-dependent association with cognitive dysfunction in PD patients, independent of traditional risk factors. The combination of elevated phosphorus and suppressed iPTH in high-AAC patients highlights the potential role of mineral metabolism dysregulation in both vascular calcification and neurocognitive decline.

Keywords Cognitive impairment, Vascular calcification, Peritoneal dialysis, Abdominal aortic calcification

*Correspondence:
Zhongxin Li
lhyy6806@ccmu.edu.cn
[1]Department of Nephrology, Beijing Luhe Hospital, Capital Medical University, NO.82 Xinhua South Road, Tongzhou District, Beijing 101100, China

Introduction

Cognitive impairment is the result of a deficit in one or more critical brain functions, such as memory, learning, focus, and decision-making.

Chronic kidney disease (CKD) significantly elevates the risk of cognitive impairment, particularly in dialysis-dependent patients [1–3]. According to a large body of research on the cognitive abilities of hemodialysis patients, the Montreal Cognitive Assessment (MoCA) found that 57.3% of patients had cognitive impairment (range: 31.9–69.4%) [4]. However, during the course of the three-year follow-up, 31% of hemodialysis patients saw a decrease in their MoCA scores of at least two points [5]. While peritoneal dialysis (PD) causes fewer hemodynamic fluctuations and minimal anticoagulation needs compared to hemodialysis, prior studies report that cognitive impairment affects 58.3–69.4% of PD patients [6–8]. This condition exacerbates poor clinical outcomes, including hospitalization, peritonitis, and mortality [7, 9–11], underscoring the urgency of identifying modifiable risk factors for early intervention.

Calcium phosphate buildup in the media or intima layers of arteries results in vascular calcification. Vascular calcification is more common in the dialysis population. In a cohort study, 1489 dialysis patients were included for a 4-year follow-up, and the prevalence of total VC was 90.7%, which represented a significant increase from the baseline of 77.1% [12]. Abdominal aortic calcification (AAC) and coronary artery calcification (CAC) are the two primary forms of arterial calcification. Numerous studies have linked CAC to cognitive outcomes [13–15]. The correlation between AAC and cognitive impairment has received far less research attention than the relationship between CAC and cognitive results [16], while the association among PD patients is unknown. Our objective was to investigate the connection between peritoneal dialysis patients' AAC and cognitive deterioration.

Materials and methods

Study population

All patients with end-stage renal illness who received PD at Beijing Luhe Hospital, which is affiliated with Capital Medical University, during June and July 2023 were included in our cross-sectional study. The inclusion criteria were: (1) patients must be at least 18 years old, (2) patients must have been on dialysis for at least three months, and (3) patients must have provided informed consent to participate in the study. The exclusion criteria were: (1) acute infection, (2) acute myocardial infarction, (3) acute strokes, (4) severe heart failure, (5) survival period less than 6 months, and (6) other impairments such as severe vision loss, linguistic incompatibility, illiteracy, mental disorder, or upper-limb handicap that made it difficult to participate in the study.

Prior to taking part in the trial, each participant signed an informed consent form. The clinical study was approved by the Ethics Committee of Beijing Luhe Hospital (2023-LHKY-012-02).

Clinical and laboratory characteristics

After admission, the age, gender, primary disease of ESRD, and dialysis duration of patients were recorded. We measured height, body weight, diastolic blood pressure (DBP), and systolic blood pressure (SBP). The body mass index (BMI) was calculated by dividing weight by height squared (kg/m2). Laboratory data collected at baseline included serum albumin, total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), potassium, sodium, total carbon dioxide (tCO2), glucose, urea, creatinine, uric acid, calcium, phosphorus, and C-reactive protein (CRP), intact parathyroid hormone (iPTH), urea clearance (Kt/V), leukocytes, hemoglobin, and platelets. To reduce mistakes, all samples were measured in the same batch using conventional techniques in the lab.

Assessment of abdominal aortic calcification

AAC was measured by lateral lumbar radiography. Four parts of the aorta's front and posterior walls were separated using Kauppila's method [17]. A score of "0" indicated no calcification, "1" indicated no more than one-third of the aortic wall in that segment was calcified, "2" indicated between one-third and two-thirds of the aortic wall, and "3" indicated more than two-thirds of the aortic wall. Aortic calcification was separated into eight segments. The anterior and posterior aortic wall scores were acquired independently, yielding a range of "0" to "3" for each segment, "0" to "6" vertebral levels, and "0" to "24" for the overall score.

If a patient's total score was ≥ 4, they were placed in the high AAC score (HAAC) group; if it was less than 4, they were placed in the low AAC score (LAAC) group [18].

Measurement of cognitive impairment

A skilled member of the study team administered the cognitive test in a calm testing environment. To assess cognitive function, MoCA tests were employed. The scale encompasses 7 areas of assessment: Language skills (sentence repetition: 2 points, verbal fluency: 1 point), delayed recall (5 points), abstract thinking (2 points), naming (3 points), attention (forward digit span: 1 point, backward digit span: 1 point, vigilance: 1 point, and serial 7 subtraction: 3 points), and visuospatial/executive functioning (trail-making test: 1 point, copy cube: 1 point, and clock drawing task: 3 points).

The MoCA maximum feasible score, when an educational adjustment is used, is 30. While others receive

no modification, those with less than 12 years of education receive an additional point toward their final score. Participants were categorized into two groups based on established criteria: MoCA screen-positive group: Total MoCA score < 26 (indicating increased likelihood of cognitive concerns requiring clinical evaluation). MoCA screen-negative group: Total MoCA score ≥ 26 (suggesting normal to mild cognitive status). Individuals with education levels < 12 years received a + 1 adjustment to their final score, aligning with MoCA's standardized implementation guidelines [19].

Statistical analysis

All analyses were performed using IBM SPSS 26.0 (IBM, USA). We performed normality checks for each parameter. Normally distributed quantitative data are presented as the mean ± standard deviation (SD), and nonnormally distributed quantitative data are presented as the median (interquartile range). For regularly distributed data, quantitative variables were compared using the Student's t-test. To compare the two sets of skewed distribution data, we used the Mann–Whitney U test. Categorical variables were compared using the $\chi 2$ test. To evaluate the association between abdominal aortic calcification (AAC) severity and cognitive function, we constructed three-tiered multivariable linear regression models: Model 1 (Unadjusted): Primary association between AAC and MoCA scores without covariate adjustment. Model 2 (Minimally Adjusted): Adjusted for demographic factors (age, sex) and dialysis vintage. Model 3 (Fully Adjusted): Further adjusted for diabetes mellitus, hypertension, body mass index, serum phosphorus, LDL cholesterol, and hemoglobin.To address the risk of multiple comparisons, we minimized exploratory analyses and focused solely on pre-specified variables based on prior clinical relevance and univariate statistical significance. In multivariate logistic regression, covariates were selected through forward stepwise likelihood ratio (LR) criteria with a threshold of $P < 0.05$. The independent influencing factors of cognitive impairment were identified using binary logistic analysis (using forward LR). Statistical significance is defined as a P value of less than 0.05.

Result

Characteristics of all the study participants

A total of 135 PD patients were screened. 3 patients had been on dialysis for less than 3 months, 4 patients had significant visual impairment, 2 patients had mental disturbance, 1 patient was suffering acute stroke, and 15 patients refused to participate in the study. Finally, the study included 110 PD patients. These patients' baseline characteristics are displayed in Table 1. The mean age was 57.78 ± 12.42 years, and 65.45% of them were male. The median dialysis duration was 37.5 (26, 67.25) months. 61 patients (55.45%) had diabetes mellitus. Hypertensive glomerulosclerosis (19 patients), diabetes (52 individuals), chronic glomerulonephritis (33 patients), and other conditions (6 patients) were the main renal disorders.

Clinical and biochemical characteristics stratified by AAC severity

Patients in the HAAC group exhibited distinct clinical profiles compared to those in the LAAC group. The HAAC group was significantly older (63.2 ± 9.8 vs. 47.4 ± 12.1 years, $P < 0.001$) and had a higher prevalence of diabetes mellitus (68.4% vs. 22.6%, $P < 0.001$). Additionally, HAAC patients demonstrated elevated serum phosphorus levels (1.62 ± 0.45 vs. 1.30 ± 0.42 mmol/L, $P < 0.001$), lower diastolic blood pressure (79.2 ± 10.8 vs. 86.6 ± 13.4 mmHg, $P = 0.005$), and reduced serum creatinine concentrations (898.4 ± 251.9 vs. 1190.7 ± 243.5 μmol/L, $P < 0.001$). Significant differences were also observed in lipid profiles, with higher total cholesterol (4.25 ± 1.12 vs. 3.85 ± 0.82 mmol/L, $P = 0.036$) and LDL-C levels (2.55 ± 0.85 vs. 2.28 ± 0.65 mmol/L, $P = 0.049$) in the HAAC group.

Notably, iPTH levels were inversely associated with AAC severity (142.5 vs. 218.0 pg/mL in HAAC vs. LAAC, $P = 0.011$). No significant differences were detected in systolic blood pressure, BMI, hemoglobin, inflammatory markers (CRP, leukocytes), nutritional parameters (albumin, urea), electrolyte levels (potassium, sodium, tCO_2), or dialysis adequacy (Kt/V) between the two groups (all $P > 0.05$). Comprehensive comparative data are presented in Table 1.

Comparisons of cognitive function parameters between the MoCA screen-positive and MoCA screen-negative groups

The average scores were 3.03 ± 1.49, 2.93 ± 0.28, 4.89 ± 1.41, 2.15 ± 1.00, 1.48 ± 0.73, and 5.64 ± 0.89 for visuospatial/executive functioning, naming, attention, language skills, abstract thinking, and, orientation respectively. While the median delayed recall score was 2.00 (0.00, 4.00).

Visuospatial/executive functioning, delayed recollection, attention, language skills, abstract thinking, and orientation scores were all poorer in the MoCA screen-positive group of PD patients than in the MoCA screen-negative group ($P < 0.05$). While there was no statistically significant difference in naming scores between groups. See Table 2.

Independent determinant factors for cognitive impairment by multiple logistic regression analysis in PD patients

A multivariate logistic regression model was used to identify the independent risk variables of cognitive

Table 1 Baseline demographics and clinical characteristics by AAC severity

Variable	All patients (*n*=110)	HAAC (≥4, *n*=79)	LAAC (<4, *n*=31)	*P* value
Male [n(%)]	72 (65.45%)	55 (69.6%)	17 (54.8%)	0.142
Age (years)	57.78±12.42	63.2±9.8	47.4±12.1	<0.001
Dialysis duration (months)	37.5 (26, 67.25)	40.0 (28, 70)	34.0 (22, 58)	0.201
BMI (kg/m^2)	24.79±4.12	24.6±4.0	25.2±4.3	0.463
Diabetes mellitus [n(%)]	61 (55.45%)	54 (68.4%)	7 (22.6%)	<0.001
SBP (mmHg)	129.06±19.12	130.5±18.6	126.8±20.1	0.327
DBP (mmHg)	81.53±12.10	79.2±10.8	86.6±13.4	0.005
Hemoglobin (g/L)	114.68±11.65	113.5±12.3	117.1±10.2	0.132
Leukocytes (×10^9/L)	7.57±2.48	7.6±2.5	7.5±2.4	0.875
Platelet (×10^9/L)	222.28±67.96	217.3±65.1	233.6±73.2	0.256
Serum CRP (mg/L)	3.23 (1.07, 9.35)	3.5 (1.1, 9.8)	2.9 (0.9, 8.2)	0.401
Serum Albumin (g/L)	37.09±3.14	36.8±3.2	37.6±2.9	0.196
Serum TG (mmol/L)	1.60 (1.04, 2.13)	1.65 (1.10, 2.20)	1.48 (0.98, 1.95)	0.067
Serum TC (mmol/L)	4.12±1.04	4.25±1.12	3.85±0.82	0.036
Serum HDL-C (mmol/L)	1.01±0.26	0.98±0.24	1.08±0.29	0.078
Serum LDL-C (mmol/L)	2.46±0.80	2.55±0.85	2.28±0.65	0.049
Serum Potassium (mmol/L)	4.44±0.62	4.42±0.60	4.49±0.67	0.601
Serum Sodium (mmol/L)	138.65±2.86	138.5±2.7	138.9±3.2	0.522
Serum tCO_2 (mmol/L)	25.21±2.08	25.0±2.1	25.6±2.0	0.155
Serum Creatinine (μmol/L)	981.79±277.87	898.4±251.9	1190.7±243.5	<0.001
Serum Urea (mmol/L)	21.81±6.36	22.3±6.5	20.9±6.1	0.289
Serum Uric Acid (μmol/L)	335.18±68.27	329.4±65.2	347.6±73.8	0.182
Serum Calcium (mmol/L)	2.31±0.18	2.29±0.19	2.35±0.15	0.089
Serum Phosphorus (mmol/L)	1.52±0.47	1.62±0.45	1.30±0.42	<0.001
Serum iPTH (pg/ml)	159.35 (56.62, 278.20)	142.5 (50.1, 265.3)	218.0 (89.5, 340.0)	0.011
Total Kt/V per week	1.80±0.29	1.78±0.28	1.84±0.31	0.334

Abbreviations: PD, Peritoneal dialysis; AAC, Abdominal aortic calcification; BMI, Body mass index; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; CRP, C-reactive protein; TG, Triglyceride; TC, Total cholesterol; HDL, High-density lipoprotein cholesterol; LDL-C, Low-density lipoprotein cholesterol; tCO2, Total carbon dioxide; iPTH, Intact parathyroid hormone

Table 2 Comparisons of cognitive function parameters between patients in the MoCA screen-positive group and MoCA screen-negative group

	All patients (*n*=110)	MoCA screen-positive (*n*=72)	MoCA screen-negative (*n*=38)	*P* value
MoCA score	22.92±5.20	20.40±4.69	27.68±1.36	<0.001
Visuospatial/executive functioning score	3.03±1.49	2.52±1.44	3.97±1.05	<0.001
Naming score	2.93±0.28	2.91±0.32	2.97±0.16	0.223
Attention score	4.89±1.41	4.45±1.55	5.71±0.46	<0.001
Language skills score	2.15±1.00	1.79±1.02	2.81±0.51	<0.001
Abstract thinking score	1.48±0.73	1.32±0.78	1.79±0.47	<0.001
Delayed recall score	2.00 (0.00, 4.00)	1.00 (0.00, 2.00)	4.00 (3.00, 4.00)	<0.001
Orientation score	5.64±0.89	5.49±1.05	5.92±0.27	0.001

Abbreviations: CI, The cognitive impairment group; NCI, The non-cognitive impairment group

impairment in PD patients in order to eliminate the influence of confounding factors on cognitive impairment.

The variables that significantly related to cognitive impairment in univariate analysis (age, with diabetes mellitus, AAC scores, DBP, serum creatinine, and phosphorus, $P<0.05$) and some previously reported variables (gender, dialysis vintage, hemoglobin, albumin, CRP, serum sodium, iPTH, and total Kt/V) entered the analysis as candidate variables. Age and AAC scores were found to be independently linked to cognitive impairment in PD patients. See Table 3.

Multivariable and age-stratified analysis of Aac severity and cognitive function

Stratified by AAC severity, patients in the HAAC group demonstrated significantly lower MoCA scores compared to the LAAC group across all analytical models. In unadjusted analysis (Model 1), HAAC was associated with MoCA scores (95% CI: -4.73 to -1.77, $P<0.001$). This association remained robust after minimal adjustment for age, sex, and dialysis vintage (Model 2: β=-2.89, $P<0.001$) and further attenuated but retained statistical significance in the fully adjusted model accounting

Table 3 Multivariate logistic regression analyses for predicting cognitive impairment in peritoneal dialysis patients

Variables	Univariate Logistic regression		Multivariate Logistic regression	
	Odd ratio (95% CI)	*P*-value	Odd ratio (95% CI)	*P*-value
Age, years	1.134 (1.077, 1.194)	< 0.001	1.125 (1.064, 1.190)	< 0.001
Male	0.460 (0.190 - 1.114)	0.085	Unentered	
Dialysis vintage (months)	0.994 (0.983 - 1.006)	0.346	Unentered	
With diabetes mellitus	2.292 (1.028 - 5.108)	0.043	Unentered	
AAC (HAAC vs. LAAC)	2.801 (1.454–5.402)	0.002	2.356 (1.122–4.933)	0.024
BMI (kg/m^2)	0.965 (0.877 - 1.062)	0.464		
SBP (mmHg)	0.998 (0.967 - 1.009)	0.256		
DBP (mmHg)	0.959 (0.925 - 0.994)	0.022	Unentered	
Hemoglobin (g/L)	1.014 (0.980 - 1.050)	0.419	Unentered	
Leukocytes (*10^9/L)	1.021 (0.870 - 1.199)	0.798		
Platelet (*10^9/L)	0.998 (0.992 - 1.004)	0.538		
Serum CRP (mg/L)	0.978 (0.950 - 1.006)	0.124	Unentered	
Serum albumin (g/L)	0.956 (0.841 - 1.086)	0.448	Unentered	
Serum TG (mmol/L)	1.029 (0.732 - 1.448)	0.868		
Serum TC (mmol/L)	0.936 (0.643 - 1.364)	0.731		
Serum HDL-C (mmol/L)	0.948 (0.203 - 4.421)	0.945		
Serum LDL-C (mmol/L)	1.174 (0.705 - 1.955)	0.539		
Serum potassium (mmol/L)	1.776 (0.410 - 1.468)	0.436		
Serum sodium (mmol/L)	0.975 (0.847 - 1.121)	0.718	Unentered	
Serum tCO2 (mmol/L)	1.099 (0.904 - 1.336)	0.344		
Serum creatinine (μmol/L)	0.997 (0.996 - 0.999)	0.001	Unentered	
Serum urea (mmol/L)	0.994 (0.935 - 1.058)	0.859		
Serum uric acid (μmol/L)	1.001 (0.995 - 1.007)	0.710		
Serum calcium (mmol/L)	2.498 (0.246 - 25.393)	0.439		
Serum phosphorus (mmol/L)	0.312 (0.122 - 0.795)	0.015	Unentered	
Serum iPTH (pg/ml)	0.999 (0.997 - 1.001)	0.486	Unentered	
Total Kt/V per wk	2.569 (0.597 - 11.049)	0.205	Unentered	

Abbreviations: PD, Peritoneal dialysis; AAC, Abdominal aortic calcification; BMI, Body mass index; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; CRP, C-reactive protein; TG, Triglyceride; TC, Total cholesterol; HDL, High-density lipoprotein cholesterol; LDL-C, Low-density lipoprotein cholesterol; tCO2, Total carbon dioxide; iPTH, Intact parathyroid hormone

Table 4 Association between AAC groups and cognitive function

Analysis	HAAC vs. LAAC β (95% CI)	*P*-value
Primary Models		
- Model 1 (Unadjusted)	-3.25 (-4.73, -1.77)	<0.001
- Model 2 (Minimally Adjusted)	-2.89 (-4.30, -1.48)	<0.001
- Model 3 (Fully Adjusted)	-2.41 (-3.85, -0.97)	0.001
Age Stratification		
- <65 years	-1.21 (-2.40, 0.18)	0.092
- ≥65 years	-3.92 (-5.80, -2.04)	<0.001

for diabetes, hypertension, and metabolic parameters (Model 3: β=-2.41, 95% CI: -3.85 to -0.97, $P=0.001$).

Age-stratified analyses revealed marked heterogeneity in this association. While HAAC patients aged < 65 years showed a non-significant MoCA reduction (95% CI: -2.40 to 0.18, $P=0.092$), those aged ≥ 65 years exhibited a pronounced deficit (95% CI: -5.80 to -2.04, $P<0.001$), indicating an age-dependent amplification of AAC-related cognitive impairment (Table 4).

AAC scores and cognitive function

79 patients (71.8%) were allocated to the HAAC group. The incidence of cognitive impairment was significantly higher in the HAAC group (73.42%) than in the LAAC group (45.16%) ($P=0.005$). In addition, the total MoCA score in the HAAC group was significantly lower than in the LAAC group (21.96 ± 5.51 vs. 25.35 ± 3.29, $P<0.001$) (Fig. 1A). There were significant differences in visuospatial/executive function score ($P=0.013$) (Fig. 1B), attention score ($P=0.004$) (Fig. 1D), language skills score ($P=0.046$) (Fig. 1E), delayed recall score ($P=0.012$) (Fig. 1G) and orientation score ($P=0.003$) (Fig. 1H) between groups. While there was no significant difference in naming score ($P=0.181$) (Fig. 1C) and abstraction score ($P=0.130$) (Fig. 1F).

Discussion

Our study showed that cognitive impairment and vascular calcification were common in PD patients. AAC score was higher in PD patients with CI than those without. We demonstrated that a higher AAC score was an

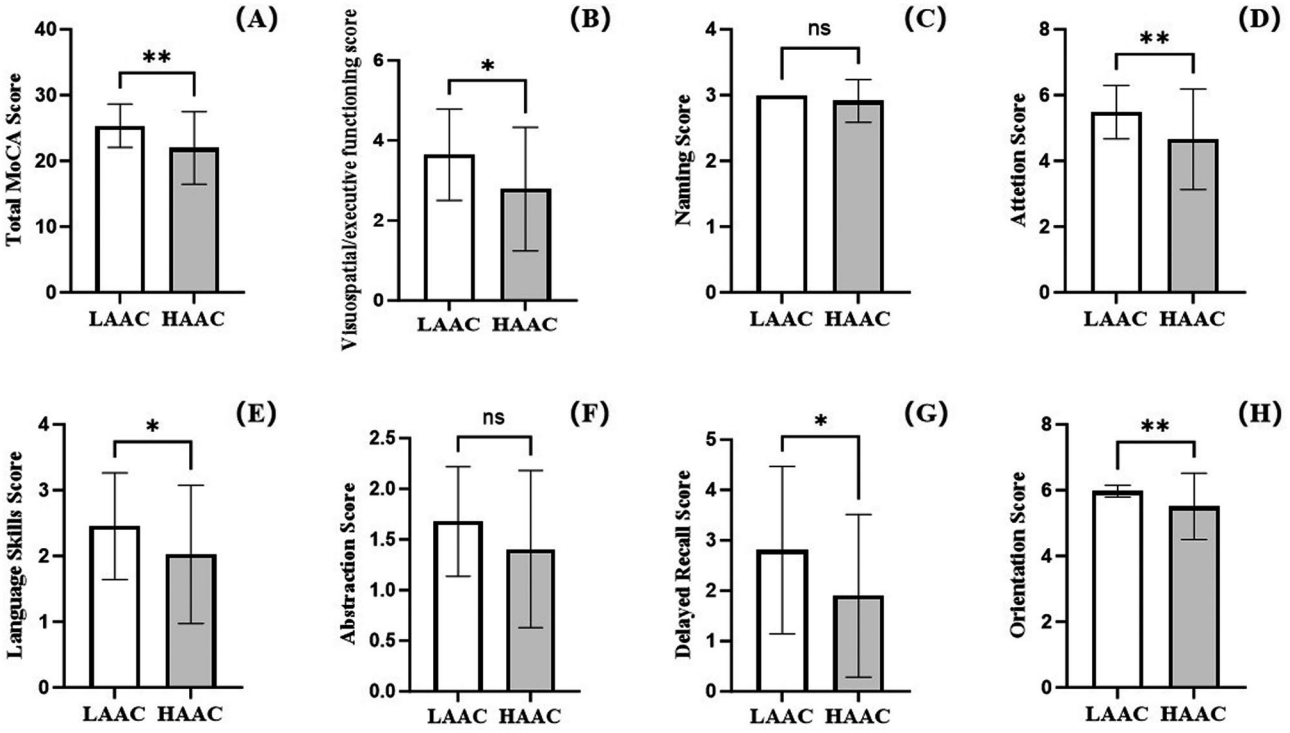

Fig. 1 Comparison of cognitive domains between LAAC and HAAC groups. (**A**: Total score; **B**: Visuospatial/executive function score; **C**: Naming score; **D**: Attention score; **E**: Language skills score; **F**: Abstraction score; **G**: Delayed recall score; **H**: Orientation score). Note: **=$P<0.01$, *=$P<0.05$, ns = no significance. Abbreviations: LAAC, low abdominal aortic calcification. HAAC, high abdominal aortic calcification

independent related factor of cognitive impairment in PD patients by a multivariate logistic regression.

Previous studies have demonstrated that the MoCA tests were found to have a higher sensitivity for detecting early stages of cognitive impairment than the Minimental State Examination (MMSE) [8]. Additionally, they showed both positive and negative predictive values, as well as strong specificity, in patients with automated PD [8]. So we used the MoCA test to screen PD patients. This PD sample had a prevalence of cognitive impairment of 65.45%, which is comparable to the percentages found in other studies [6, 7]. In our study, visuospatial/executive functioning, naming, attention, language skills, abstract thinking, delayed recall, and orientation scores declined in 88 cases (80.00%), 6 cases (0.05%), 61 cases (55.45%), 56 cases (51.91%), 42 cases (38.18%), 101 cases (91.82%) and 24 cases (21.82%). Similarly, in a different study, CAPD patients showed notable reductions in delayed recollection, language skills, executive and visuospatial abilities, and other cognitive impairment categories [6], indicating multidimensional impairment. We must pay more attention to cognitive impairment in PD patients because these changes may affect their capacity to reliably and safely complete the difficult activities of self-administering drugs and dialysis.

Patients receiving peritoneal dialysis have cognitive impairment through a variety of intricate pathophysiological pathways. The main risk factors encompassing broad demographic traits (age, gender, education level [8, 20, 21]), traditional cardiovascular risk factors (such as hypertension and diabetes mellitus [3, 20]), non-traditional kidney-related factors (such as hyponatremia, anemia, malnutrition, inflammation, secondary hyperparathyroidism [8, 21–24]), dialysis factors (such as dialysis vintage, dialysis adequacy [20, 25]) and other factors (such as depression [20]). Recently, Liao et al. [26] discovered a correlation between global cognitive decline and retinopathy, a disease common in PD patients. In addition, PD patients who have cognitive impairment and those who have normal cognition differ in the quantity and composition of their gut microbiomes [27]. According to our findings, older age, diabetes mellitus, and higher AAC scores were risk factors for cognitive impairment while higher DBP, serum creatinine, and serum phosphorus were protective factors in univariate logistic regression. However, in multivariate logistic regression, only older age and higher AAC scores were independent related factors of cognitive impairment. In our study, patients with cognitive impairment had lower levels of serum creatinine and phosphorus which was consistent with previous publications and could be explained by poorer nutritional status [8, 24]. In addition, Shoji et al. [28] shown that in hemodialysis patients, low DBP—rather than SBP—was substantially linked to

cognitive deterioration. These findings may be explained by the patients' poor cerebral blood flow and compromised brain blood pressure autoregulation. In our investigation, we also discovered that patients with cognitive impairment had lower DBPs than those without cognitive impairment. Although there was no statistical difference between DBP and cognitive function after multivariable adjustment, DBP still was a potential risk factor for cognitive impairment.

Our study's most significant finding—which has never before been shown in PD patients—was that AAC was independently linked to cognitive decline in these patients. Possible explanations about the connections between cognitive function and AAC are as follows. Firstly, numerous cardiovascular risk factors are linked to AAC, such as age, diabetes mellitus, hypertension, inflammation, nutritional status, and dyslipidemia [29–32], which are linked to cognitive function as well. Secondly, AAC, a marker of both subclinical atherosclerotic disease and arteriosclerosis, is associated with a more stiffened arterial system, and as a result, linked to the development of cardiovascular and cerebrovascular diseases. AAC was an independent predictor of all-cause mortality as well as significant adverse cardiac and cerebrovascular events in individuals with PD, according to earlier research [33]. From the perspective of the measurement methods, our findings highlight AAC's unique clinical relevance in PD populations. Unlike CACS (which requires specialized cardiac CT) or PWV (which reflects arterial stiffness but not localized calcification) [34, 35], lateral lumbar radiography for AAC assessment is low-cost, widely accessible, and routinely performed in dialysis clinics for other indications (e.g., vertebral fractures). This positions AAC as a practical screening tool for cognitive risk stratification in resource-limited settings.

There are various limitations to our investigation. First, because it was a single-center study, its generalizability might be limited by its small sample size. Second, because of the cross-sectional nature of this study, it was not possible to ascertain the cause-and-effect link between AAC and cognitive function in PD patients. In addition, we analyzed only AAC but did not focus on other vascular calcifications. In addition, although stepwise regression and prespecified variable selection reduced overfitting, we did not apply formal correction for multiple comparisons. Future studies with larger samples may benefit from such adjustments to further minimize Type I error risk. While our analysis adjusted for key covariates such as diabetes and hypertension, unmeasured confounders, including prior stroke, ischemic heart disease, smoking history, and longitudinal glycemic control may influence cognitive outcomes. Future studies incorporating comprehensive vascular and lifestyle risk profiling are warranted to refine risk stratification in PD populations. Also, we did not measure serum magnesium levels, which may modulate vascular calcification and cognitive function in dialysis patients. Further studies integrating magnesium and other uremic toxins are needed to refine the mechanistic understanding of these associations.

Conclusion

In summary, the MoCA analysis of this sample of PD patients revealed a multidimensional cognitive impairment prevalence of 65.45%. Our study provides preliminary clinical evidence supporting an independent association between abdominal aortic calcification (AAC) and cognitive impairment in peritoneal dialysis patients, expanding prior observations of vascular calcification's systemic effects in this population. Higher AAC scores and older age are independent risk factors for cognitive impairment in PD patients. Therefore, preventive therapeutic strategies in individuals with a high level of AAC score may be effective to impede the growth of cognitive decline or dementia in the future.

Abbreviations

LAAC Low abdominal aortic calcification
HAAC High abdominal aortic calcification

Acknowledgements

Not applicable.

Author contributions

Conghui Liu made significant contributions to research design, specific experimental process management, data analysis, and manuscript writing; Yanan Shi made significant contributions to research design and data analysis; Jiajie Cai contributed to research design; Tingting Bai and Jingjing Li contributed to experimental operation and data collection; Lingju Yue made significant contributions to research design and manuscript review process. Zhongxin Li made significant contributions to experimental technical support and results section. All authors reviewed the manuscript.

Funding

No funding was received for conducting this study.

Data availability

The data used to support the findings of this study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The clinical study was approved by the Ethics Committee of Beijing Luhe Hospital (2023-LHKY-012-02). Informed consent was obtained from all the participants. All methods were carried out in accordance with Declaration of Helsinki.

Consent for publication

Not applicable.

Clinical trial

Not applicable.

Competing interests

The authors declare no competing interests.

Received: 13 January 2025 / Accepted: 5 March 2025
Published online: 10 March 2025

References

1. Li T, Hu Z, Qiao L, Wu Y, Ye T. Chronic kidney disease and cognitive performance: NHANES 2011–2014. BMC Geriatr. 2024;24(1):351. https://doi.org/10.1186/s12877-024-04917-2.
2. Zhou T, Zhao J, Ma Y, He L, et al. Association of cognitive impairment with the interaction between chronic kidney disease and depression: findings from NHANES 2011–2014. BMC Psychiatry. 2024;24(1):312. https://doi.org/10.1186/s12888-024-05769-1.
3. Zhang J, Wu L, Wang P, Pan Y, et al. Prevalence of cognitive impairment and its predictors among chronic kidney disease patients: A systematic review and meta-analysis. PLoS One. 2024;19(6):e0304762. https://doi.org/10.1371/journal.pone.0304762.
4. Cao T, An R, Wang Y, Lei J, Liang J, Wan Q. Risk factors and prevalence of cognitive impairment in maintenance haemodialysis patients: A systematic review and meta-analysis of observational studies. J Adv Nurs. 2023;79(10):3691–706. https://doi.org/10.1111/jan.15746.
5. Wang L, Bian X, Liu L, He Q, et al. Association between cognitive function and skeletal muscle in patients undergoing maintenance Hemodialysis. Front Endocrinol (Lausanne). 2024;15:1324867. https://doi.org/10.3389/fendo.2024.1324867.
6. Shi C, Jia S, Wang X, Liu C, et al. Research on cognitive impairment and potential risk factors in peritoneal dialysis patients: an observational study. medicine. (Baltimore). 2024;103(28):e38374. https://doi.org/10.1097/MD.0000000000038374.
7. Wu C, Yu R, Li Q, Chen J, Wang W. Exploring the impact of cognitive impairments on treatment compliance and quality of life in patients with continuous ambulatory peritoneal Dialysis (CAPD). Medicine. (Baltimore). 2023;102(43):e35813. https://doi.org/10.1097/MD.0000000000035813.
8. Salazar-Félix NA, Martin-Del-Campo F, Cueto-Manzano AM, Romo-Flores ML, et al. Prevalence of mild cognitive impairment in automated peritoneal dialysis patients. Nephrol Dial Transpl. 2021;36(11):2106–11. https://doi.org/10.1097/MD.0000000000035813.
9. Zhang YH, Yang ZK, Wang JW, Xiong ZY, et al. Cognitive changes in peritoneal dialysis patients: A multicenter prospective cohort study. Am J Kidney Dis. 2018;72(5):691–700. https://doi.org/10.1053/j.ajkd.2018.04.020.
10. Liao JL, Zhang YH, Xiong ZB, Hao L, et al. The association of cognitive impairment with peritoneal dialysis-related peritonitis. Perit Dial Int. 2019;39(3):229–35. https://doi.org/10.3747/pdi.2018.00180.
11. Miyazaki S, Kitamura M, Hayashida M, Tamaki A, et al. Survival and cognitive deterioration in elderly patients undergoing hemodialysis. Geriatr Gerontol Int. 2023;23(2):111–6. https://doi.org/10.1111/ggi.14531.
12. Zhang H, Li G, Yu X, Yang J, et al. Progression of vascular calcification and clinical outcomes in patients receiving maintenance dialysis. JAMA Netw Open. 2023;6(5):e2310909. https://doi.org/10.1001/jamanetworkopen.2023.10909.
13. Gadde KM, Yin X, Goldberg RB, Orchard TJ, et al. Coronary artery calcium and cognitive decline in the diabetes prevention program outcomes study. J Am Heart Assoc. 2023;12(21):e029671. https://doi.org/10.1161/JAHA.123.029671.
14. Guo J, Nunley KA, Costacou T, Miller RG, et al. Greater progression of coronary artery calcification is associated with clinically relevant cognitive impairment in type 1 diabetes. Atherosclerosis. 2019;280:58-65. https://doi.org/10.1016/j.atherosclerosis.2018.11.003
15. Ma YN, Xie WX, Hou ZH, An YQ, et al. Association between coronary artery calcification and cognitive function in a Chinese community-based population. J Geriatr Cardiol. 2021;18(7):514–22. https://doi.org/10.11909/j.issn.1671-5411.2021.07.002.
16. Wei J, Ali MK, Wang T, Xu H. Abdominal aortic calcification and cognitive function among older adults: Cross-sectional analysis of National health and Nutrition Examination Survey, 2013–2014. Int J Geriatr Psychiatry. 2021;36(11):1778–84. https://doi.org/10.1002/gps.5599.
17. Kauppila LI, Polak JF, Cupples LA, Hannan MT, Kiel DP, Wilson PW. New indices to classify location, severity and progression of calcific lesions in the abdominal aorta: a 25-year follow-up study. Atherosclerosis. 1997;132(2):245–50. https://doi.org/10.1016/s0021-9150(97)00106-8.
18. Peeters MJ, van den Brand JA, van Zuilen AD, Koster Y, et al. Abdominal aortic calcification in patients with. CKD J Nephrol. 2017;30(1):109–18. https://doi.org/10.1007/s40620-015-0260-7.
19. Nasreddine ZS, Phillips NA, Bédirian V, Charbonneau S, et al. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. J Am Geriatr Soc. 2005;53(4):695–9. https://doi.org/10.1111/j.1532-5415.2005.53221.x.
20. Gamage I, Dhar A, Tregaskis P, Wilson S. Frequency and risk factors for cognitive dysfunction in peritoneal dialysis patients. Nephrol (Carlton). 2022;27(12):945–52. https://doi.org/10.1111/nep.14117.
21. Shea YF, Lee MC, Mok MM, Lam MF, et al. Self-care peritoneal dialysis patients with cognitive impairment have a higher risk of peritonitis in the second year. Perit Dial Int. 2019;39(1):51–8. https://doi.org/10.3747/pdi.2018.00048.
22. Duan LP, Zheng ZX, Zhang YH, Dong J. Association of malnutrition-inflammation-cardiovascular disease with cognitive deterioration in peritoneal dialysis patients. Beijing Da Xue Xue Bao Yi Xue Ban. 2019;51(3):510–8. https://doi.org/10.19723/j.issn.1671-167X.2019.03.020.
23. Mathur A, Ahn JB, Sutton W, Chu NM, et al. Secondary hyperparathyroidism (CKD-MBD) treatment and the risk of dementia. Nephrol Dial Transpl. 2022;37(11):2111–8. https://doi.org/10.1093/ndt/gfac167.
24. Yi C, Lin J, Cao P, Chen J, et al. Prevalence and prognosis of coexisting frailty and cognitive impairment in patients on continuous ambulatory peritoneal. Dialysis Sci Rep. 2018;8(1):17305. https://doi.org/10.1038/s41598-018-35548-4.
25. Shin DJ, Kim T, Jung DU, Moon JJ, et al. Association between dialysis adequacy and cognition in patients with. Perit Dialysis Psychiatry Investig. 2020;17(11):1143–8. https://doi.org/10.30773/pi.2020.0279.
26. Liao J, Wei Q, He Y, Liao Y, et al. Retinopathy is associated with impaired cognition in patients undergoing peritoneal dialysis. Ren Fail. 2023;45(2):2258989. https://doi.org/10.1080/0886022X.2023.2258989.
27. Wang J, Wu S, Zhang J, Li Y, Wu Y, Qi X. Correlation between gut microbiome and cognitive impairment in patients undergoing peritoneal dialysis. BMC Nephrol. 2023;24(1):360. https://doi.org/10.1186/s12882-023-03410-z.
28. Shoji T, Fujii H, Mori K, Nakatani S, et al. Associations of cardiovascular disease and blood pressure with cognition in hemodialysis patients: The Osaka dialysis complication study. Nephrol Dial Transpl. 2022;37(9):1758–67. https://doi.org/10.1093/ndt/gfab247.
29. Cao Q, Shi Y, Liu X, Yang F, Li X, Li Z. Analysis of factors influencing vascular calcification in peritoneal dialysis patients and their impact on long-term prognosis. BMC Nephrol. 2024;25(1):157. https://doi.org/10.1186/s12882-024-03582-2.
30. Dragoș D, Timofte D, Georgescu MT, Manea MM, et al. Cardiovascular calcifications are correlated with inflammation. Hemodial Patients Med (Kaunas). 2023;59(10):1801. https://doi.org/10.3390/medicina59101801.
31. Leal-Alegre G, Lerma C, Leal-Escobar G, Moguel-González B, Martínez-Vázquez KB, Cano-Escobar KB. Relationship between vascular calcification, protein-energy wasting syndrome, and sarcopenia in maintenance automated peritoneal dialysis. Life (Basel). 2021;11(7):666. https://doi.org/10.3390/life11070666.
32. Niu Q, Zhao H, Wu B, Tsai S et al. Study on the prevalence of vascular calcification in different types of arteries and influencing factors in maintenance peritoneal dialysis patients. Blood Purif. (2019);47 Suppl 1(Suppl 1):8–16. https://doi.org/10.1159/000496218
33. Ma D, Yan H, Yang X, Yu Z, Ni Z, Fang W. Abdominal aortic calcification score as a predictor of clinical outcome in peritoneal dialysis patients: a prospective cohort study. BMC Nephrol. 2020;21(1):151. https://doi.org/10.1186/s12882-020-01822-9.
34. Zhong W, Cruickshanks KJ, Schubert CR, et al. Pulse wave velocity and cognitive function in older adults. Alzheimer Dis Assoc Disord. 2014;28(1):44–9. https://doi.org/10.1097/WAD.0b013e3182949f06.
35. Ma YN, Xie WX, Hou ZH, et al. Association between coronary artery calcification and cognitive function in a Chinese community-based population. J Geriatr Cardiol. 2021;18(7):514–22. https://doi.org/10.11909/j.issn.1671-5411.2021.07.002.

Publisher's note