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RESEARCH ARTICLE

Frequency of Sarcopenia and Associated Factors in Older Adults with Non-dialysis Dependent Chronic Kidney Disease: A Cross-sectional Study

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ABSTRACT

Objective: This cross-sectional study aimed to determine the prevalence of sarcopenia and investigate the associated clinical and biochemical factors in geriatric patients with non-dialysis chronic kidney disease (CKD).

Materials and Methods: Seventy-one pre-dialysis CKD patients followed in geriatric and nephrology outpatient clinics were enrolled. Sarcopenia was diagnosed according to the EWGSOP2 criteria using the skeletal muscle index (SMI; bioelectrical impedance analysis), handgrip strength (dynamometer), 4-metre gait speed, and five-times sit-to-stand test. Patients with probable sarcopenia were included in the sarcopenic group. The Mann-Whitney U test, Fisher's exact test, and univariate logistic regression analysis were applied.

Results: The median age was 71.8±4.4 years, and 45.1% of patients were male. The prevalence of sarcopenia was 22.5% (n=16), with defined sarcopenia at 14.1% and probable sarcopenia at 8.5%. BMI was significantly lower in patients with sarcopenia (26.2 vs. 31.9 kg/m^2; p=0.009). The SMI, fat-free mass index (FFMI), and handgrip strength were significantly lower in the sarcopenic group (all p<0.05). Hemoglobin showed a strong positive correlation with SMI (r=+0.524; p<0.001) and handgrip strength (r=+0.437; p<0.001). In univariate logistic regression analysis, BMI was the only significant independent predictor (OR=0.801; 95% CI: 0.677–0.947; p=0.009).

Conclusion: Sarcopenia prevalence was 22.5% in non-dialysis CKD patients, and low BMI was an independent risk indicator. The strong correlation between hemoglobin and muscle parameters supports the role of renal anemia in the pathophysiology of sarcopenia. Early sarcopenia screening and body composition assessment are recommended for CKD follow-up.

Keywords: Anemia, chronic kidney disease, older adults, sarcopenia

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INTRODUCTION

Sarcopenia, a multifactorial clinical syndrome, is characterised by progressive loss of skeletal muscle mass, muscle strength, and physical performance. It is associated with loss of functionality, increased risk of falls, hospitalisations, morbidity, and mortality. The 2018 updated consensus statement of the European Working Group on Sarcopenia in Older People (EWGSOP2) identifies low muscle strength as the primary diagnostic criterion; confirmation of low muscle mass is additionally required for a diagnosis of "sarcopenia," while impairment in all three components defines "severe sarcopenia." Patients with isolated reduced muscle strength are classified as having "probable sarcopenia," representing an important window of opportunity for intervention.[1,2]

Chronic kidney disease (CKD) has become a major public health problem globally, largely attributable to demographic ageing and the increasing incidence of hypertension and diabetes. Population-based studies indicate a CKD prevalence of

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7.2% among individuals aged 30 years and older, with this figure rising to between 23.4% and 35.8% in older adults.[3,4] In Türkiye, the CREDIT study identified CKD rates of 32.7% in the 60–69 age group, 41.3% in the 70–79 age group, and 54.7% in individuals over 80 years of age.[5] The pathophysiology of CKD contributes to sarcopenia through several converging mechanisms, including the accumulation of uraemic toxins, metabolic acidosis that accelerates muscle proteolysis, disorders in insulin and IGF-1 signalling leading to anabolic resistance, vitamin D deficiency with direct myotoxic effects, and systemic inflammation.[6,7] The independent roles of CKD-specific factors—such as proteinuria, renal anaemia, and secondary hyperparathyroidism—in the development and progression of sarcopenia remain incompletely understood.[8]

Comprehensive sarcopenia assessments using the EWGSOP2 criteria in predialysis CKD patients remain limited. Using varying definitions, the prevalence of sarcopenia ranges from 5.9% to 14% in patients with CKD not receiving renal replacement therapy and may reach up to 42.2% in haemodialysis patients.[9–14] Currently, there is a paucity of population-specific data from Türkiye employing the EWGSOP2 framework in predialysis CKD patients. Utilising the EWGSOP2 criteria, the present study investigated the prevalence of sarcopenia among predialysis CKD patients and examined associated clinical and biochemical factors, with particular emphasis on eGFR staging, type 2 diabetes mellitus (T2DM) aetiology, haemoglobin concentrations, and proteinuria.

MATERIALS and METHODS

Study Design and Patient Selection

This cross-sectional study was conducted at the Geriatrics and Nephrology Outpatient Clinics of İstanbul Kanuni Sultan Süleyman Training and Research Hospital from August 1, 2023, to November 1, 2023. Participants eligible for inclusion were individuals aged 65 years and older who had been under follow-up for a minimum of six months with a diagnosis of chronic kidney disease (CKD) and had provided written informed consent. The exclusion criteria included active dialysis treatment, a history of kidney transplantation, active malignancy, decompensated heart failure (NYHA Stage III–IV), active infection, advanced liver cirrhosis, and orthopaedic or neuromuscular disorders that precluded bilateral handgrip measurement. Of the 81 patients initially assessed, five declined participation, four were excluded due to significant oedema, and one was excluded due to missing data, resulting in the inclusion of 71 patients. The estimated glomerular filtration rate (eGFR) was calculated using the 2009 CKD-EPI creatinine equation, and chronic kidney disease (CKD) staging was conducted in accordance with the KDIGO 2012 guideline.[15,16] Approval from the Ethics Committee of the University of Health Sciences Kanuni Sultan Suleyman Training and Research Hospital was secured for this study (Decision No: 2023.07.89; Decision date: 13.07.2023). Furthermore, the consent process was conducted in writing for all participants, conforming to the Declaration of Helsinki guidelines.

Definitons of Sarcopenia

Body composition was assessed using an eight-electrode multifrequency bioelectrical impedance analysis (BIA) device (InBody 270, InBody Co., Ltd., Seoul, South Korea). Measurements were performed in the morning after at least eight hours of fasting; participants abstained from vigorous physical activity and alcohol for 24 hours prior to measurement. All measurements were conducted with participants wearing light clothing, without shoes or socks, standing on the foot electrodes and gripping the hand electrodes. Parameters recorded included the appendicular skeletal muscle index (ASMI), calculated as appendicular skeletal muscle mass (kg) divided by height squared (m^2); total body fat mass (kg); percent body fat; and BMI. Cut-off values for low appendicular muscle mass were ASMI<7.0 kg/m^2 for men and <5.5 kg/m^2 for women.[1]

Handgrip strength was measured on the dominant hand using a hydraulic hand dynamometer (Baseline Hydraulic Hand Dynamometer 12–024, Fabrication Enterprises, New York, USA); the mean of three measurements was recorded. Thresholds for low handgrip strength were <27 kg for men and <16 kg for women. For the five-times chair stand test, a time of ≥15 seconds indicated low muscle strength.[1] Gait speed was assessed over 4 metres; a value <0.8 m/s defined poor physical performance. Patients with isolated low muscle strength were classified as having probable sarcopenia; those with additional low ASMI were classified as having sarcopenia; and those with impairment in all three components were classified as having severe sarcopenia. Probable sarcopenia cases were included in the sarcopenic group for all analyses.

Clinical and Biochemical Assessment

Serum creatinine, BUN, albumin, hemoglobin (Hb), HbA1c, CRP, PTH, calcium, phosphorus, sodium, and potassium were measured from fasting morning blood samples. Proteinuria was assessed using a 24-hour urine collection. Body weight, height, and BMI were measured under standardised conditions.

Statistical Analysis

The Shapiro-Wilk test was employed to assess the normality of continuous variables. Variables with a normal distribution were expressed as mean±standard deviation (mean±SD),

Table 1. General characteristics and between-group comparison of patients

Variable	All patients (n=71)	Sarcopenic (n=16)	Non-sarcopenic (n=55)	p
Demographic characteristics				
Age (years)	71.8±4.4	71.2±3.9	71.9±4.5	0.588
Sex, male n (%)	32 (45.1)	4 (25.0)	28 (50.9)	0.089
BMI (kg/m²)	31.1 (27.8–34.5)	26.2 (23.6–30.8)	31.9 (28.7–35.0)	**0.009**
Renal function parameters				
eGFR (mL/min/1.73 m²)	42.1 (32.3–50.4)	37.9 (27.8–49.3)	43.2 (34.8–50.4)	0.285
eGFR <30, n (%)	16 (22.5)	6 (37.5)	10 (18.2)	0.170
Creatinine (mg/dL)	1.39 (1.13–1.83)	1.47 (1.20–1.89)	1.39 (1.13–1.83)	0.571
BUN (mg/dL)	24.7 (19.8–32.2)	28.5 (21.3–33.4)	24.7 (19.7–32.2)	0.359
Proteinuria (mg/day)	130.0 (60.0–450.0)	70.0 (40.0–155.0)	165.0 (80.0–525.0)	0.055
Biochemical parameters				
Haemoglobin (g/dL)	12.6 (11.5–13.7)	11.9 (11.4–12.6)	12.7 (11.6–13.9)	0.155
Albumin (g/L)	44.0 (42.0–46.0)	43.0 (42.0–44.0)	44.0 (42.0–46.0)	0.247
CRP (mg/L)	2.8 (1.1–6.2)	2.4 (1.0–7.8)	2.8 (1.2–6.1)	0.865
PTH (pg/mL)	64.0 (45.0–91.0)	80.5 (57.0–90.0)	62.0 (45.0–91.0)	0.397
Vitamin D (mg/L)	21.19.7	20,67.1	21.210.3	0.845
Phosphorus (mg/dL)	3.5 (3.2–4.0)	3.8 (3.5–4.4)	3.5 (3.2–4.0)	0.114
Potassium (mEq/L)	4.8 (4.4–5.0)	4.9 (4.6–5.1)	4.8 (4.4–5.0)	0.323
Body composition and performance				
ASMI (kg/m²)	7.3 (6.6–8.4)	5.9 (5.7–6.8)	8.0 (6.9–8.7)	**<0.001**
FFMI (kg/m²)	18.6 (17.3–20.7)	16.2 (15.7–16.6)	19.2 (17.7–20.8)	**<0.001**
Handgrip strength (kg)	25.0 (18.0–32.0)	16.0 (15.0–24.5)	28.0 (22.0–37.8)	**0.002**
Gait speed (m/s)	0.75 (0.67–0.92)	0.75 (0.63–0.83)	0.75 (0.67–0.92)	0.263
Chair stand time (s)	12.0 (10.0–14.0)	14.0 (11.2–16.0)	12.0 (10.0–13.8)	0.053
T2DM (aetiology), n (%)	35 (49.3)	11 (68.8)	24 (43.6)	0.094

Data are presented as mean±SD (normally distributed) and median (IQR) or n (%). Bold P-values indicate statistical significance (P<0.05). ASMI: Appendicular skeletal muscle index; FFMI: Fat-free mass index; BMI: Body mass index; eGFR: estimated glomerular filtration rate; BUN: Blood urea nitrogen; PTH: Parathyroid hormone; CRP: C-reactive protein; T2DM: Type 2 diabetes mellitus; HbA1c: Glycated haemoglobin A1c

while those not normally distributed were reported as median (interquartile range, IQR). For intergroup comparisons of continuous variables, the Mann-Whitney U test was utilised, and Fisher's exact test was applied to categorical variables. Univariate logistic regression was conducted to examine variables associated with sarcopenia, with results presented as odds ratios (ORs) and 95% confidence intervals (95% CIs). The Spearman correlation coefficient was used to analyse correlations between continuous variables. A p-value of less than 0.05 was considered statistically significant. A post-hoc power analysis conducted using G*Power 3.1.9.7 (Faul et al., 2007) indicated that, for the primary comparison, the achieved power with the current sample was 84.2% (α=0.05, two-tailed).[17] For the prevalence estimate of 22.5%, a minimum sample of n=67 was required to achieve a ±10% margin of error at 95% confidence, which the current cohort satisfies. All statistical analyses were conducted using SPSS version 31.0 (IBM Corp., Armonk, NY, USA).

RESULTS

General Characteristics

The mean age of the 71 included patients was 71.8±4.4 years, and 45.1% (n=32) were male. The median eGFR was 42.1 mL/min/1.73 m² (IQR 32.3–50.4). The most common CKD aetiology was T2DM (49.3%), followed by hypertension (38.0%). The median number of comorbidities was 3 (IQR 2–4). The median number of medications was 6 (IQR 4–7). Table 1 presented the demographic, biochemical, and clinical characteristics.

Table 2. Univariate logistic regression analysis for factors associated with sarcopenia

Variable	OR	95% CI	p
BMI (kg/m²)	0.801	0.677–0.947	0.009
T2DM (etiology)	2.842	0.870–9.283	0.084
Male sex	0.321	0.092–1.121	0.075
eGFR <30 vs ≥30 mL/min/1.73 m²	2.700	0.795–9.166	0.111
Haemoglobin (g/dL)	0.818	0.597–1.122	0.213
Proteinuria (mg/day)	0.999	0.997–1.001	0.236
eGFR (mL/min/1.73 m²)	0.979	0.942–1.017	0.271
CRP (mg/L)	1.048	0.970–1.132	0.237
Albumin (g/L)	1.014	0.928–1.108	0.751
Age (years)	0.963	0.845–1.097	0.568

P<0.05 indicates statistical significance. OR: Odds ratio; CI: Confidence interval; BMI: Body mass index; T2DM: Type 2 diabetes mellitus; eGFR: Estimated glomerular filtration rate; CRP: C-reactive protein

Sarcopenia Prevalence

The overall prevalence of sarcopenia was 22.5% (n=16). According to EWGSOP2 staging, 14.1% (n=10) met the criteria for defined sarcopenia and 8.5% (n=6) for probable sarcopenia. BMI was significantly lower in sarcopenic patients (26.2 vs 31.9 kg/m²; p=0.009). ASMI (5.9 vs 8.0 kg/m²; p<0.001) and FFMI (16.2 vs 19.2 kg/m²; p<0.001) were significantly lower in the sarcopenic group, as was handgrip strength (16.0 vs 28.0 kg; p=0.002). Although haemoglobin was numerically lower in the sarcopenic group, the difference did not reach statistical significance (11.9 vs 12.7 g/dL; p=0.155). Proteinuria levels showed a trend towards lower values in sarcopenic patients (70.0 vs 165.0 mg/day; p=0.055). Significant differences were not observed in eGFR, creatinine, albumin, or CRP values between groups.

Factors Associated with Sarcopenia

On univariate logistic regression, BMI was the only variable significantly associated with sarcopenia (OR=0.801; 95% CI: 0.677–0.947; p=0.009). T2DM aetiology (OR=2.842; 95% CI: 0.870–9.283; p=0.084) and male sex (OR=0.321; 95% CI: 0.092–1.121; p=0.075) showed borderline associations. eGFR, haemoglobin, proteinuria, and age were not significantly associated with sarcopenia in univariate analysis. The binary classification of eGFR as CKD eGFR<30 vs. ≥30 mL/min/1.73 m² also did not reach significance (OR=2.700; 95% CI: 0.795–9.166; p=0.111). Logistic regression results are presented in Table 2.

Correlation Analyses

Spearman correlation analysis revealed a strong positive correlation between haemoglobin and ASMI (r=+0.524; p<0.001) and a moderate positive correlation between haemoglobin and handgrip strength (r=+0.437; p<0.001). A strong positive correlation was also observed between BMI and ASMI (r=+0.498; p<0.001). A significant positive correlation was identified between proteinuria and ASMI (r=+0.276; p=0.033). Correlation findings are summarised in Table 3.

Table 3. Correlation analysis between haemoglobin, BMI, proteinuria and sarcopenia components

Variables	Spearman r	p
Haemoglobin –ASMI	+0.524	<0.001
Haemoglobin – Handgrip strength	+0.437	<0.001
Haemoglobin – FFMI	+0.282	0.032
Haemoglobin – Gait speed	+0.307	0.012
BMI – ASMI	+0.498	<0.001
BMI – Handgrip strength	+0.305	0.011
Proteinuria – ASMI	+0.276	0.033
Proteinuria – eGFR	−0.132	0.278

P<0.05 indicates statistical significance. ASMI: Appendicular skeletal muscle index; FFMI: Fat-free mass index; BMI: Body mass index; eGFR: Estimated glomerular filtration rate

DISCUSSION

In this cross-sectional study, sarcopenia prevalence among geriatric patients with predialysis CKD was found to be 22.5% based on the EWGSOP2 criteria. Low body mass index (BMI) emerged as the sole independent factor significantly linked to sarcopenia. Notably, a significant correlation was found between haemoglobin levels and both muscle mass and muscle strength.

The observed prevalence of 22.5% is consistent with the previously reported range of 5–40% in studies focusing on predialysis CKD patients. For example, Souza et al.[13] reported a prevalence of 28.3% using the EWGSOP criteria in a similar cohort. Furthermore, data compiled by Sabatino et al.[12] demonstrate that prevalence rates vary significantly based on measurement methods and patient characteristics. A comprehensive systematic review and meta-analysis by Duarte et al.[14] estimated the prevalence of sarcopenia in CKD patients to be approximately 21%. In Türkiye, Tufan et al.[18] identified low handgrip strength, potentially indicative of sarcopenia, in 29.7% of patients with eGFR<60 mL/min/1.73 m²—a finding consistent with our results and highlighting the clinical burden of muscle weakness in the Turkish CKD population. The relatively lower prevalence observed compared to some series is primarily due to the outpatient setting and the absence of a significant burden of advanced

comorbidities. When only defined sarcopenia cases are considered, prevalence falls to 14.1%, which is consistent with predialysis cohorts reported in the literature.

The lack of a significant association between eGFR level and sarcopenia prevalence is a recurrent finding in comparable studies.[12,19] Although the proportion of sarcopenic patients was numerically higher in those with eGFR<30 mL/min/1.73 m^2 (37.5%) than in those with eGFR≥30 (18.2%), the difference did not reach statistical significance, likely due to the limited number of Stage 4–5 patients. Importantly, muscle loss emerges from the combined effects of the uraemic environment, chronic inflammation, and nutritional disturbances that begin from the early stages of CKD, regardless of eGFR level; as a result, no sharp distinction in sarcopenia risk forms between CKD stages.[6,19]

The strong positive correlation between haemoglobin and both ASMI and handgrip strength is a clinically important finding. Several mechanisms may underlie this relationship. First, lower haemoglobin levels can impair aerobic muscle metabolism, leading to energy insufficiency and accelerated muscle fatigue. Hypoxia causes decreased oxygen delivery to the muscles, and this is more pronounced in anaemia associated with CKD.[20] Second, systemic inflammation, erythropoietin resistance, and iron deficiency contribute in parallel to both renal anaemia[21] and skeletal muscle loss. Third, emerging evidence suggests that correction of renal anaemia may be associated with improved exercise capacity and muscle function in CKD patients; however, the direct effect of erythropoiesis-stimulating agents on skeletal muscle mass and strength warrants further investigation in prospective studies.[22,23] These observations collectively suggest that optimal management of renal anaemia should be considered an integral component of sarcopenia prevention in CKD.

The aetiology of T2DM was observed at a marginally higher frequency within the sarcopenic group, indicating that diabetes-related mechanisms, such as insulin resistance, hyperglycaemic myotoxicity, and peripheral neuropathy, may collectively expedite muscle loss in this demographic. Numerous studies have documented elevated rates of sarcopenia in diabetic patients with CKD compared to their non-diabetic counterparts, with insulin resistance and impaired insulin-like growth factor 1 (IGF-1) signalling identified as principal contributors.[8,12] The SCOPE study reported a 13% prevalence of sarcopenia among diabetic patients diagnosed with chronic renal failure. Within this study, diabetic participants exhibited a higher incidence of adverse outcomes across all sarcopenia components compared to non-diabetic participants.[24]

The observation that proteinuria tends to be lower in sarcopenic patients initially appears paradoxical. One plausible explanation is that patients with elevated proteinuria in this cohort—particularly those with nephrotic syndrome or glomerulonephritis—may be relatively younger individuals with fewer comorbidities and greater capacity to preserve muscle mass. Moreover, concomitant oedema and fluid retention may cause measurement bias in lean mass estimates by BIA. The significant positive correlation between proteinuria and ASMI supports this interpretation. The limited number of patients with high-grade proteinuria in this cohort may have contributed to this counterintuitive finding, and it should be interpreted with caution until confirmed in larger prospective studies with specific proteinuria subgroups.[8]

Body Mass Index (BMI) emerged as the sole independent predictor of sarcopenia. An increased incidence of sarcopenia was observed in patients with a low BMI, which can be attributed to the effects of mechanical loading and adipokine-mediated anabolic signalling. The study underscores the importance of protein-energy malnutrition, frequently seen in CKD, in the pathogenesis of sarcopenia.[25] Furthermore, the possibility of sarcopenic obesity, defined by diminished muscle mass despite a maintained or elevated BMI, should be considered in this population. This is due to the fact that BMI alone may not adequately represent the risk of sarcopenia in overweight or obese CKD patients.

This study has several limitations. The cross-sectional design restricts the ability to infer causal relationships. The modest sample size of patients diminishes the statistical power to assess the association between eGFR and sarcopenia, as well as to separately analyse the three EWGSOP2 sarcopenia stages (probable, confirmed, severe). Potential confounding factors, such as dietary protein intake and physical activity levels, were not systematically recorded. The single-centre design limits the generalisability of the findings. Lastly, BIA-based muscle mass assessment is influenced by hydration status, presenting a methodological limitation, particularly in patients with significant proteinuria or oedema.

CONCLUSION

This study determined that the prevalence of sarcopenia among geriatric patients with non-dialysis-dependent CKD was 22.5% per EWGSOP2 criteria. Although eGFR stage, haemoglobin level, T2DM aetiology, and proteinuria were not independently associated with sarcopenia in univariate analysis, a strong correlation was observed between haemoglobin and both muscle mass and muscle strength, underscoring

the role of renal anaemia in the pathophysiology of sarcopenia. Low BMI emerged as a significant independent risk indicator, highlighting the necessity of early body composition assessment in CKD patients. These findings advocate for routine sarcopenia screening during CKD follow-up. Prospective multicentre studies—especially those examining the impact of anaemia management and diabetes control on muscle mass—are recommended.

Disclosures

Ethics Committee Approval: The study was approved by the University of Health Sciences Kanuni Sultan Suleyman Training and Research Hospital Ethics Committee (No: 2023.07.89, Date: 13/07/2023).

Informed Consent: Written informed consents were obtained from patients who participated in this study.

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