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Dynapenia in Patients with Diabetic Nephropathy: A Cross-Sectional Comparative Study

Hasan Kırmaz
Gaziosmanpaşa Eğitim Ve Araştırma Hastanesi
Emel Tatli
emeltat@yahoo.com
Gaziosmanpaşa Eğitim Ve Araştırma Hastanesi
Okcan Basat
Gaziosmanpaşa Eğitim Ve Araştırma Hastanesi
Abdullah Ozan Polat
Gaziosmanpaşa Eğitim Ve Araştırma Hastanesi

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Abstract

Background and objective:

Diabetic nephropathy is a frequent microvascular complication of type 2 diabetes mellitus (T2DM), and both conditions have been linked to loss of muscle strength (dynapenia). Whether the presence of nephropathy adds to the muscle-strength deficit already associated with diabetes remains incompletely defined. We compared handgrip strength, functional mobility and body composition among healthy controls, patients with isolated T2DM, and patients with diabetic nephropathy, restricting enrollment to individuals with a preserved skeletal muscle mass so that strength loss could be evaluated independently of muscle mass loss.

Methods

In this single-center, cross-sectional, descriptive-analytical study conducted between February and April 2024, 117 adults attending family medicine and nephrology outpatient clinics were allocated to three equal groups of 39: healthy controls, patients with T2DM without nephropathy, and patients with diabetic nephropathy (defined by a urine albumin-to-creatinine ratio > 300 mg/g). All participants underwent laboratory testing, bioelectrical impedance analysis, handgrip dynamometry and the Timed Up and Go (TUG) test. Dynapenia was defined as handgrip strength below 28.5 kg in men and 18.5 kg in women. Group comparisons used the Kruskal-Wallis test with Bonferroni post-hoc correction, chi-square or Fisher's exact tests, and Spearman correlation.

Results

Dynapenia was present in 0% of controls, 28.2% of patients with isolated T2DM, and 38.5% of patients with diabetic nephropathy. Mean handgrip strength was significantly lower in both diabetic groups than in controls (31.85 ± 8.58 kg in controls vs 26.25 ± 8.97 kg in isolated diabetes and 24.95 ± 7.17 kg in diabetic nephropathy; $p = 0.001$), with no significant difference between the two diabetic groups. TUG duration was significantly longer in the diabetic nephropathy group (13.73 ± 1.49 s) than in controls (10.48 ± 1.4 s) and isolated diabetes (11.31 ± 1.61 s; $p < 0.001$). Across the whole cohort, handgrip strength correlated inversely with body mass index, waist and hip circumference, TUG duration, body-fat percentage, fasting glucose and HbA1c, and positively with skeletal muscle percentage and total protein (all $p < 0.05$).

Conclusions

Diabetes mellitus was associated with reduced handgrip strength irrespective of nephropathy status, but the additional presence of diabetic nephropathy was associated with a higher prevalence of dynapenia

and a marked prolongation of TUG performance, indicating a greater impact on functional mobility once nephropathy develops. Simple, low-cost functional assessments may help identify diabetic patients at greatest risk of functional decline.

Introduction

Diabetic kidney disease is one of the most frequent and prognostically important microvascular complications of type 2 diabetes mellitus (T2DM), affecting a substantial proportion of patients worldwide and contributing heavily to the global burden of chronic kidney disease [1]. Alongside its renal and cardiovascular consequences, T2DM is increasingly recognized as a risk factor for musculoskeletal decline. Sarcopenia, the age- and disease-related loss of skeletal muscle mass and function, and its distinct sub-phenotype dynapenia—loss of muscle strength that is not proportional to, and may occur independently of, muscle mass loss—are now well documented in patients with diabetes [2–5].

Several mechanisms have been proposed to link diabetes and diabetic kidney disease to impaired muscle strength, including chronic low-grade inflammation, insulin resistance, accumulation of advanced glycation end-products, vitamin D deficiency, and activation of the renin-angiotensin system, which promotes muscle proteolysis and impairs muscle regeneration [4, 6]. In patients with chronic kidney disease specifically, reduced handgrip strength has been associated with lower glomerular filtration rate and higher albuminuria, suggesting that declining renal function may compound the muscle-strength deficits already present in diabetes [7]. However, most previous studies have examined either diabetes or chronic kidney disease in isolation, and comparative data directly contrasting diabetic patients with and without nephropathy against non-diabetic controls remain limited, particularly when muscle mass is accounted for so that strength loss (dynapenia) can be distinguished from mass loss [8, 9].

We therefore designed a cross-sectional study comparing handgrip strength, functional mobility (Timed Up and Go, TUG) and body composition among three groups—healthy controls, patients with T2DM without nephropathy, and patients with diabetic nephropathy—restricting enrollment to individuals with a preserved skeletal muscle mass index so that the specific contribution of diabetic nephropathy to dynapenia could be isolated from concurrent sarcopenia.

Material and Methods

Study design and setting

This single-center, cross-sectional, descriptive and analytical study was conducted between 1 February and 30 April 2024 among adults attending the family medicine and nephrology outpatient clinics of Gaziosmanpaşa Training and Research Hospital. The study was approved by the Clinical Research Ethics Committee of the hospital (approval date: 27.12.2023; decision no: 167) and was conducted in accordance with the Declaration of Helsinki; written informed consent was obtained from all participants.

Participants

A total of 117 adults were enrolled and allocated to three equal groups of 39 participants each: healthy controls without diabetes, patients with T2DM without nephropathy (isolated diabetes), and patients with T2DM and diabetic nephropathy. Diabetic nephropathy was defined by a spot urine albumin-to-creatinine ratio above 300 mg/g. Eligible participants were over 18 years of age and had a preserved skeletal muscle mass index (SMMI ≥ 10.76 kg/m² in men and ≥ 6.76 kg/m² in women, calculated from bioelectrical impedance analysis), so that strength loss could be evaluated independently of muscle mass loss. Individuals with a physical disability, neurological disease, malignancy or musculoskeletal pathology were excluded.

Sample size was calculated with G*Power 3.1.9.4 based on handgrip-strength data reported by Çeliker et al. for diabetic, diabetic-nephropathy and control groups (24.00 ± 11.2 kg, 19.70 ± 8.9 kg and 25.10 ± 9.2 kg, respectively) [10], yielding a large effect size (d = 0.76); with α = 0.05 and power (1 − β) of 0.95, a minimum of 21 participants per group was required. The final sample of 39 per group exceeded this requirement.

Assessments

Venous blood samples were obtained for glucose, urea, creatinine, alanine aminotransferase, aspartate aminotransferase, total protein, albumin, a full lipid panel (total cholesterol, LDL, HDL, triglycerides), electrolytes (calcium, magnesium, sodium, potassium), complete blood count, HbA1c and vitamin B12, together with spot urine protein-to-creatinine and albumin-to-creatinine ratios. Handgrip strength was measured by a single examiner using a Camry electronic hand dynamometer (model EH101, 2019), and body composition (skeletal muscle mass, body-fat percentage) was assessed by bioelectrical impedance analysis (InBody 270, Republic of Korea, 2016). Waist, hip, upper-arm and calf circumferences were measured with a tape measure, and functional mobility was assessed with the Timed Up and Go (TUG) test.

Dynapenia was defined, following the cut-off values reported by Wang and Chen, as handgrip strength below 28.5 kg in men and below 18.5 kg in women [11], applied only to participants who met the preserved-SMMI inclusion criterion, so that the diagnosis reflects loss of strength rather than loss of muscle mass.

Statistical analysis

Statistical analyses were performed with IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed with the Kolmogorov–Smirnov test; because most variables departed from a normal distribution, continuous variables are presented as mean±standard deviation and compared across the three groups with the Kruskal-Wallis test, with the Bonferroni-corrected test used for post-hoc pairwise comparisons. Categorical variables are presented

as n (%) and compared with the Pearson chi-square test or Fisher's exact test, as appropriate. Associations between handgrip strength and continuous clinical variables were assessed with Spearman's rank correlation. A two-sided p-value < 0.05 was considered statistically significant.

Results

Baseline characteristics

The 117 participants had a mean age of 58.76 ± 7.28 years; 73 (62.4%) were women and 44 (37.6%) were men, with no significant difference in age or sex distribution across the three groups (Table 1). Hypertension, coronary heart disease, chronic renal failure, retinopathy and hyperlipidemia were all significantly more frequent in the diabetic nephropathy and isolated diabetes groups than in controls (all p < 0.001); coronary heart disease and retinopathy were not observed at all in the isolated diabetes group, and none of the three groups differed significantly in the prevalence of hypothyroidism (Table 1).

Table 1

Baseline sociodemographic and clinical characteristics of the study population (n = 117). Continuous variable compared with the Kruskal-Wallis test; categorical variables compared with the Pearson chi-square test or Fisher's exact test. Note: the chronic-renal-failure count has been corrected to 16 (13.7%) for internal consistency with the subgroup counts and total sample size; the authors should verify this figure against the original statistical output before submission.

Variable	Total (n = 117)	Controls (n = 39)	Diabetic nephropathy (n = 39)	Isolated diabetes (n = 39)	p
Age, years, mean ± SD	58.76 ± 7.28	56.61 ± 6.73	60.23 ± 7.38	59.46 ± 7.39	0.070
Female sex, n (%)	73 (62.4)	22 (56.4)	24 (61.5)	27 (69.2)	0.501
Hypertension, n (%)	67 (57.3)	6 (15.4)	36 (92.3)	25 (64.1)	< 0.001
Coronary heart disease, n (%)	12 (10.3)	0 (0)	12 (30.8)	0 (0)	< 0.001
Chronic renal failure, n (%)	16 (13.7)	0 (0)	13 (33.3)	3 (7.7)	< 0.001
Retinopathy, n (%)	9 (7.7)	0 (0)	9 (23.1)	0 (0)	< 0.001
Hyperlipidemia, n (%)	39 (33.3)	2 (5.1)	15 (38.5)	22 (56.4)	< 0.001
Hypothyroidism, n (%)	15 (12.8)	3 (7.7)	6 (15.4)	6 (15.4)	0.502

Prevalence of dynapenia

No participant in the control group met criteria for dynapenia. In the isolated diabetes group, 11 of 39 patients (28.2%; 9 women, 2 men) had dynapenia, compared with 15 of 39 patients (38.5%; 7 women, 8 men) in the diabetic nephropathy group. Overall, dynapenia was present in 26 of 117 participants (22.2%).

Handgrip strength, functional mobility and body composition across groups

Mean handgrip strength was significantly higher in controls (31.85 ± 8.58 kg) than in both the diabetic nephropathy (24.95 ± 7.17 kg) and isolated diabetes (26.25 ± 8.97 kg) groups ($p = 0.001$), with no significant difference between the two diabetic groups. TUG duration was significantly longer in the diabetic nephropathy group (13.73 ± 1.49 s) than in controls (10.48 ± 1.4 s) and the isolated diabetes group (11.31 ± 1.61 s; $p < 0.001$), while controls and isolated diabetes did not differ significantly from one another. Skeletal muscle percentage differed significantly across all three groups (29.75 ± 4.75% in controls, 13.64 ± 4.42% in diabetic nephropathy, 25.36 ± 5.29% in isolated diabetes; $p < 0.001$ for all pairwise comparisons), whereas body-fat percentage did not differ significantly ($p = 0.087$). Glomerular filtration rate, total protein, fasting glucose and HbA1c also differed significantly across all three groups (all $p < 0.001$), consistent with the graded metabolic and renal severity of the three groups. Full between-group comparisons are shown in Table 2.

Table 2

Between-group comparison of functional, anthropometric, body-composition and laboratory parameters (Kruskal-Wallis test with Bonferroni-corrected post-hoc pairwise comparisons). C, controls; DN, diabetic nephropathy; ID, isolated diabetes; TUG, Timed Up and Go; eGFR, estimated glomerular filtration rate.

Variable	Controls (n = 39)	Diabetic nephropathy (n = 39)	Isolated diabetes (n = 39)	p	Post-hoc
Handgrip strength, kg	31.85 ± 8.58	24.95 ± 7.17	26.25 ± 8.97	0.001	C-DN, C-ID
Body mass index, kg/m²	30.98 ± 5.38	32.87 ± 6.23	33.06 ± 6.4	0.272	–
Waist circumference, cm	102.13 ± 8.6	108.18 ± 10	107.95 ± 13	0.030	C-ID
Hip circumference, cm	107.31 ± 17	110.05 ± 20	114.23 ± 13	0.212	–
TUG test, s	10.48 ± 1.4	13.73 ± 1.49	11.31 ± 1.61	< 0.001	DN-C, DN-ID
Urine protein/creatinine ratio	184.67 ± 11	2219.46 ± 1	165.5 ± 161	< 0.001	DN-C, DN-ID
Urine albumin/creatinine ratio	52.25 ± 38.8	775.74 ± 46	35.03 ± 47.7	< 0.001	DN-C, DN-ID
Skeletal muscle, %	29.75 ± 4.75	13.64 ± 4.42	25.36 ± 5.29	< 0.001	C-DN, C-ID, DN-ID
Body fat, %	35.85 ± 7.55	37.62 ± 11.35	41.53 ± 10.9	0.087	–
eGFR, mL/min/1.73m²	89.82 ± 11.8	54 ± 22.42	76.67 ± 22.6	< 0.001	C-DN, C-ID, DN-ID
Total protein, g/L	75.69 ± 3.77	72.74 ± 3.0	74.74 ± 3.46	< 0.001	DN-C, DN-ID
Fasting glucose, mg/dL	98.96 ± 9.62	176.74 ± 79	138.41 ± 44	< 0.001	C-DN, C-ID, DN-ID
HbA1c, %	5.56 ± 0.33	8.12 ± 1.68	7.12 ± 1.54	< 0.001	C-DN, C-ID, DN-ID
Triglycerides, mg/dL	144.36 ± 17.53	185.31 ± 20.42	154.54 ± 13.39	0.045	DN-C
Calcium, mg/dL	9.91 ± 3.08	9.55 ± 4.46	9.71 ± 4.15	0.001	C-DN

Correlates of handgrip strength

In the cohort as a whole, handgrip strength correlated inversely with body mass index ($r = -0.300$, $p = 0.001$), waist circumference ($r = -0.211$, $p = 0.022$), hip circumference ($r = -0.372$, $p < 0.001$), TUG duration ($r = -0.441$, $p < 0.001$), body-fat percentage ($r = -0.501$, $p < 0.001$), fasting glucose ($r = -0.229$, $p = 0.013$), HbA1c ($r = -0.202$, $p = 0.029$) and HDL cholesterol ($r = -0.246$, $p = 0.008$), and positively with skeletal muscle percentage ($r = 0.501$, $p < 0.001$) and total protein ($r = 0.202$, $p = 0.029$) (Table 3). No significant correlation was found between handgrip strength and the urine protein-to-creatinine ratio, the albumin-to-creatinine ratio, or eGFR in the cohort as a whole.

When examined separately by group, the correlation between handgrip strength and total protein remained significant only in the diabetic nephropathy group ($r = 0.433$, $p = 0.006$), while correlations with body mass index, hip circumference, TUG duration and body-fat percentage were significant in the control and isolated-diabetes groups but attenuated in the diabetic-nephropathy group, suggesting that once nephropathy is established, factors other than adiposity and anthropometry become more relevant to residual muscle strength. Within each group, handgrip strength was significantly higher in men than in women (controls: 39.45 ± 4.49 vs 25.96 ± 5.89 kg; isolated diabetes: 36.59 ± 6.73 vs 21.65 ± 5.22 kg; diabetic nephropathy: 29.95 ± 8.00 vs 21.82 ± 4.39 kg; all $p < 0.001$).

Table 3
Spearman correlations between handgrip strength and clinical, anthropometric and laboratory variables in the whole cohort (n = 117). TUG, Timed Up and Go; eGFR, estimated glomerular filtration rate.

Variable	r	p
Body mass index	−0.300	0.001
Waist circumference	−0.211	0.022
Hip circumference	−0.372	< 0.001
Upper-arm circumference	−0.148	0.110
Calf circumference	−0.159	0.087
TUG test duration	−0.441	< 0.001
Urine protein/creatinine ratio	−0.119	0.200
Urine albumin/creatinine ratio	−0.117	0.211
Skeletal muscle, %	0.501	< 0.001
Body fat, %	−0.501	< 0.001
eGFR	0.154	0.097
Total protein	0.202	0.029
Fasting glucose	−0.229	0.013
Vitamin B12	−0.126	0.176
HbA1c	−0.202	0.029
Total cholesterol	0.055	0.554
LDL cholesterol	0.106	0.253
HDL cholesterol	−0.246	0.008
Triglycerides	0.129	0.167
Calcium	0.101	0.279

Discussion

In this cross-sectional study comparing healthy controls, patients with isolated T2DM and patients with diabetic nephropathy—restricted to individuals with a preserved skeletal muscle mass index so that strength loss could be isolated from mass loss—dynapenia was absent in controls but present in more than a quarter of patients with isolated diabetes and in almost two out of five patients with diabetic nephropathy. Mean handgrip strength was significantly lower in both diabetic groups than in controls,

indicating that diabetes itself, independent of nephropathy, is associated with reduced muscle strength; the additional presence of nephropathy did not further reduce mean handgrip strength relative to isolated diabetes, but it was associated with a higher proportion of patients falling below the dynapenia threshold and with a substantially longer TUG duration, pointing to a disproportionate effect on functional mobility once renal impairment is superimposed on diabetes.

Grip-strength thresholds for functional impairment have been derived from several populations. Wang and Chen proposed cut-offs of 18.5 kg in women and 28.5 kg in men based on the strength required to perform heavy manual tasks in older adults [11], values adopted in the present study; other groups, using slow gait speed as the functional anchor, have reported comparable thresholds ranging from 15.9–22.0 kg in women and 23.2–39.0 kg in men [12, 13]. Using the Wang and Chen cut-offs, we found no dynapenia in the healthy control group, while 11 patients in the isolated diabetes group (9 women, 2 men) and 15 patients in the diabetic nephropathy group (7 women, 8 men) met criteria for dynapenia. Although absolute handgrip strength was somewhat lower in the diabetic nephropathy group than in the isolated diabetes group, this difference was not statistically significant, suggesting that the presence of nephropathy does not exert an additional negative effect on mean strength beyond that already attributable to diabetes itself.

TUG performance, in contrast, clearly distinguished the diabetic nephropathy group from the other two. Bohannon's meta-analysis of 21 studies and 4,395 healthy adults aged 60–99 years reported a mean TUG duration of 9.4 ± 0.5 seconds overall, rising from 8.1 ± 0.9 seconds at 60–69 years to 11.3 ± 1.3 seconds at 80–99 years [14]. Despite a considerably younger mean age in our cohort (58.76 ± 7.28 years), TUG duration was prolonged in both diabetic groups relative to Bohannon's age-matched reference values, and particularly so in the diabetic nephropathy group (13.73 ± 1.49 seconds), which differed significantly from both the control and isolated-diabetes groups. This indicates that diabetes alone impairs mobility to some extent, but that the addition of nephropathy amplifies this impairment considerably, restricting the physical activity of these patients more than diabetes alone.

Our finding that handgrip strength did not correlate significantly with eGFR in the cohort as a whole contrasts with a large Korean cross-sectional study (KNHANES 2014–2017) in adults aged 19–39 years, which reported a low-handgrip-strength prevalence of 6.2% in participants without chronic kidney disease versus 25.2% in those with chronic kidney disease, together with a significant positive correlation between handgrip strength and GFR [15]. The much younger age of that cohort, and its reliance on a reduced GFR alone (without regard to the underlying cause) to define chronic kidney disease, may explain the discrepancy; in our study, the diabetic nephropathy group—defined by macroalbuminuria rather than by GFR alone—had the highest dynapenia prevalence of the three groups, and reduced GFR was one of several features that characterized this group, together with lower handgrip strength and prolonged TUG duration. That this pattern of muscle impairment in chronic kidney disease emerges independently of chronological age, in both our study and that of Lee et al., supports the view that declining renal function is itself an important determinant of muscle strength and function, potentially via activation of the renin-angiotensin system: in experimental models, angiotensin II infusion

increases muscle proteolysis, while angiotensin-II type 1 receptor blockade attenuates transforming-growth-factor-beta-mediated failure of muscle regeneration [6].

The functional consequences of these deficits are clinically meaningful. In a cross-sectional study of 2,083 participants, Yang et al. found that lower handgrip strength and longer TUG duration were both significantly associated with poorer health-related quality of life (measured with the EQ-5D), an association that strengthened with advancing age and the presence of chronic disease [16]. In our cohort, the diabetic nephropathy group had both the longest TUG duration and the highest dynapenia prevalence, suggesting that these patients are likely to experience the greatest impact on quality of life among the three groups studied. Diabetes appears to impair muscle strength independently of nephropathy, but once nephropathy develops, functional mobility—as captured by the TUG test—deteriorates significantly further, underscoring the importance of comprehensive follow-up, treatment and management of diabetes and its renal complications for preserving patients' functional status and quality of life.

Consistent with previous reports linking diabetes to dynapenia and sarcopenia through mechanisms such as advanced glycation end-product accumulation [3–5], reduced vitamin D status [17, 18], and altered body composition [7, 19], our correlational analysis found handgrip strength to be inversely related to adiposity (body mass index, waist and hip circumference, body-fat percentage) and glycemic burden (fasting glucose, HbA1c), and positively related to skeletal muscle percentage and total protein, across the cohort as a whole. Notably, in the diabetic-nephropathy subgroup, most of these associations attenuated except for the correlation with total protein, which suggests that once nephropathy is established, protein status—plausibly reflecting nutritional state and protein-energy wasting—may become a more relevant correlate of residual muscle strength than adiposity per se, a pattern that merits confirmation in future, adequately powered studies.

Limitations

This study has several limitations. First, its cross-sectional, single-center design precludes causal inference and limits generalizability; the observed associations should be interpreted as hypothesis-generating. Second, although the sample size exceeded the a priori power calculation, the number of participants in each subgroup (n = 39) is nonetheless modest, particularly for the gender-stratified and per-group correlation analyses, which should be interpreted with caution. Third, the diagnosis of dynapenia relied on a single set of externally derived handgrip-strength cut-offs; other proposed thresholds exist and may classify participants differently. Fourth, information on physical activity level, nutritional intake, and diabetes duration—each of which could influence muscle strength independently of nephropathy—was not systematically collected and could not be adjusted for.

Conclusions

In this cross-sectional comparison of healthy controls, patients with isolated type 2 diabetes and patients with diabetic nephropathy, diabetes mellitus was associated with reduced handgrip strength irrespective of nephropathy status, while the additional presence of diabetic nephropathy was associated with a higher prevalence of dynapenia and a marked, clinically relevant prolongation of Timed Up and Go performance. These findings suggest that simple, low-cost functional assessments—handgrip dynamometry and the TUG test—could usefully complement metabolic and renal monitoring in diabetic patients, particularly once nephropathy develops, to identify those at greatest risk of functional decline. Larger, prospective, multicenter studies are needed to confirm these associations and to clarify their implications for clinical management.

Declarations

Ethical statement

This study was approved by the Clinical Research Ethics Committee of Gaziosmanpaşa Training and Research Hospital (approval date: 27.12.2023; decision no: 167) and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

Conflict of interest

The authors declare no conflict of interest.

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Author Contribution

H.K. conceived and designed the study, collected the data, and drafted the manuscript. E.T. contributed to study design, data analysis/interpretation, and critical revision of the manuscript. O.B. supervised the study as principal thesis advisor and critically revised the manuscript for important intellectual content. A.O.P. contributed to data curation, formal analysis, and critical review of the manuscript. All authors read and approved the final version.

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Data Availability

The datasets generated and/or analyzed during the current study are not publicly available due to patient confidentiality but are available from the corresponding author on reasonable request.

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