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Original Research Article

Muscle Before Nephron: Sarcopenia as an Early Signal of Renal Deterioration in Adults with Type 2 Diabetes Mellitus

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Conflict of interest: Nil

Abstract:

Background: Sarcopenia is increasingly recognised as a systemic complication of type 2 diabetes mellitus (T2DM) and chronic kidney disease. Its role as an early predictor of diabetic kidney disease progression remains insufficiently studied in Indian hospital settings.

Methods: This prospective observational cohort study included 225 adults aged 40–75 years with T2DM at GSL Medical College. Baseline assessment included clinical characteristics, handgrip strength, calf circumference, five-times chair-stand test, walking speed, SARC-F score, HbA1c, serum creatinine, estimated glomerular filtration rate and urine albumin–creatinine ratio. Participants were classified according to sarcopenia status and followed for renal outcomes. Multivariable logistic regression was used to identify independent predictors of diabetic kidney disease progression.

Results: Sarcopenia was present in 58 participants (25.8%). Diabetic kidney disease progression occurred in 43.1% of participants with sarcopenia and 16.8% without sarcopenia. Sarcopenia was associated with a 2.57-fold higher unadjusted risk of progression. After adjustment for age, sex, diabetes duration, HbA1c, hypertension, baseline eGFR and albuminuria, sarcopenia remained an independent predictor of renal deterioration (adjusted OR: 2.96; 95% CI: 1.48–5.92). Adding sarcopenia-related measurements improved the ROC area from 0.73 to 0.81.

Conclusion: Sarcopenia independently predicted diabetic kidney disease progression and may serve as a practical early risk marker in adults with T2DM.

Keywords: Sarcopenia; Diabetic Kidney Disease; Type 2 Diabetes Mellitus; Handgrip Strength; Estimated Glomerular Filtration Rate.

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Introduction

Type 2 diabetes mellitus (T2DM) is a major cause of chronic kidney disease (CKD), with diabetic kidney disease characterised by persistent albuminuria, progressive reduction in estimated glomerular filtration rate and increased cardiovascular and mortality risk. Conventional predictors such as poor glycaemic control, hypertension, duration of diabetes and baseline albuminuria do not completely explain the variation in renal progression. Sarcopenia, defined by reduced muscle strength, muscle mass and physical performance, is increasingly recognised as an important but under-assessed complication of diabetes and CKD [1]. Diabetes may promote muscle deterioration through insulin resistance, chronic inflammation, oxidative stress, microvascular dysfunction, neuropathy and physical inactivity, whereas declining kidney function may accelerate muscle catabolism through metabolic acidosis, uraemic toxins, nutritional deficiencies and hormonal disturbances [2]. The relationship therefore appears bidirectional. Recent prospective evidence indicates that baseline sarcopenia increases the risk of incident diabetic kidney disease, while diabetic kidney disease also predicts subsequent sarcopenia [1]. An eight-year cohort of Asian adults with type 2 diabetes further demonstrated that low muscle mass was associated with CKD and albuminuria progression [2]. Simple assessments such as handgrip strength, calf circumference, walking speed and chair-stand performance may therefore provide clinically useful risk information

without requiring expensive equipment [3, 4]. The present study aims to determine whether baseline sarcopenia predicts worsening renal function or albuminuria over 12 months among adults with T2DM.

Methods

A prospective observational cohort study was conducted in the outpatient clinic of GSL Medical College. Patient recruitment and baseline assessment were undertaken from April 2025 to July 2026. Adults aged 40–75 years with T2DM who attended the outpatient or inpatient services during the study period were screened for eligibility. A minimum of 225 participants was planned based on the expected difference in diabetic kidney disease progression between patients with and without sarcopenia, allowing for loss to follow-up. Participants were enrolled consecutively after obtaining written informed consent. Patients with type 1 diabetes mellitus, acute kidney injury, established end-stage kidney disease, current dialysis, active malignancy, chronic liver failure, severe cardiac failure, recent stroke, major limb amputation, neuromuscular disorders, acute infection, prolonged immobilisation, pregnancy or inability to perform muscle-function tests were excluded. Patients receiving long-term corticosteroids or other drugs known to substantially affect muscle mass were also excluded. The study protocol was approved by the Institutional Ethics Committee, and confidentiality of participant information was maintained throughout the study.

At baseline, demographic and clinical details including age, sex, duration of diabetes, antidiabetic treatment, smoking status, alcohol use, hypertension, cardiovascular disease and other relevant comorbidities were recorded using a structured case-record form. Height, weight, body mass index, waist circumference and calf circumference were measured using standard procedures. Muscle strength was assessed using handgrip strength with a calibrated hand dynamometer, and the highest value obtained from repeated measurements of the dominant hand was recorded. Lower-limb strength was evaluated using the five-times chair-stand test. Physical performance was assessed by usual walking speed over a predefined distance, while the SARC-F questionnaire was administered as a screening instrument. Sarcopenia was classified according to Asian Working Group for Sarcopenia criteria based on reduced muscle strength, impaired physical performance and reduced muscle mass. When dual-energy X-ray absorptiometry or bioelectrical impedance analysis was unavailable, calf circumference combined with handgrip strength and functional performance tests was used as a practical screening model. Blood samples were analysed for haemoglobin, glycated haemoglobin, serum creatinine, serum albumin, vitamin D and lipid profile. Estimated glomerular filtration rate was calculated using the CKD-EPI equation. A spot urine sample was obtained for measurement of the urine albumin–creatinine ratio, and albuminuria was categorised as normal to mildly increased, moderately increased or severely increased.

Participants were reassessed at 6 and 12 months using clinical examination, serum creatinine, estimated glomerular filtration rate and urine albumin–creatinine ratio. The primary outcome was diabetic kidney disease progression, defined as a decline of at least 30% in estimated glomerular filtration rate from baseline, progression to a higher albuminuria category, new-onset persistent albuminuria confirmed on repeat testing or rapid annual decline in renal function. Renal outcomes were compared between participants with and without sarcopenia. Continuous variables were expressed as mean with standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. Independent-samples t-test or Mann–Whitney U test was used for continuous variables, and chi-square or Fisher's exact test was used for categorical variables. Multivariable logistic regression or Cox proportional-hazards regression was performed after adjustment for age, sex, diabetes duration, glycated haemoglobin, hypertension, baseline estimated glomerular filtration rate and albuminuria. Receiver-operating-characteristic curve analysis was used to determine whether handgrip strength, calf circumference and functional tests improved renal-risk prediction beyond conventional clinical factors. A p-value below 0.05 was considered statistically significant.

Results

A total of 225 adults with T2DM were included, of whom 58 (25.8%) had sarcopenia and 167 (74.2%) did not. As shown in Table 1, participants with sarcopenia were significantly older, had a longer duration of diabetes, lower body mass index, higher prevalence of hypertension and cardiovascular disease, poorer glycaemic control, lower haemoglobin, serum albumin and vitamin D levels, lower baseline estimated glomerular filtration rate and higher urine albumin–creatinine ratio. Table 2 demonstrated that the sarcopenia group had significantly lower calf circumference, reduced handgrip strength, slower walking speed, prolonged five-times chair-stand duration and higher SARC-F scores compared with the non-sarcopenia group. According to Table 3, diabetic kidney disease progression occurred in 43.1% of participants with sarcopenia and 16.8% of those without sarcopenia, corresponding to a relative risk of 2.57. A decline of at least 30% in estimated glomerular filtration rate, progression to a higher albuminuria category, new persistent albuminuria and rapid renal decline were

all significantly more frequent among participants with sarcopenia. The mean annual decline in estimated glomerular filtration rate was also greater in the sarcopenia group. In multivariable analysis presented in Table 4, sarcopenia remained an independent predictor of diabetic kidney disease progression with an adjusted odds ratio of 2.96. Longer diabetes duration, higher HbA1c, lower baseline estimated glomerular filtration rate, higher baseline albuminuria and reduced handgrip strength were also significant predictors. Addition of sarcopenia status, handgrip strength and calf circumference improved the area under the receiver-operating-characteristic curve from 0.73 to 0.81.

Table 1: Baseline characteristics according to sarcopenia status

Variable	Sarcopenia (n=58)	No sarcopenia (n=167)	Test statistic	p-value
Age, years*	62.4 ± 7.8	56.8 ± 8.6	t=4.39	<0.001
Male sex**	31 (53.4)	91 (54.5)	χ^2=0.02	0.892
Diabetes duration, years*	11.8 ± 5.6	8.7 ± 4.9	t=4.00	<0.001
BMI, kg/m²*	23.6 ± 3.4	26.1 ± 3.8	t=4.42	<0.001
Hypertension**	43 (74.1)	91 (54.5)	χ^2=6.88	0.009
Cardiovascular disease**	12 (20.7)	17 (10.2)	χ^2=4.20	0.04
HbA1c, %*	8.6 ± 1.4	8.0 ± 1.3	t=2.96	0.003
Haemoglobin, g/dL*	11.6 ± 1.5	12.7 ± 1.4	t=5.05	<0.001
Serum albumin, g/dL*	3.7 ± 0.4	4.0 ± 0.4	t=4.94	<0.001
Vitamin D, ng/mL*	18.9 ± 7.2	23.8 ± 8.5	t=3.91	<0.001
Baseline eGFR***	69.8 ± 18.4	78.6 ± 17.1	t=3.32	0.001
Baseline UACR****	82 (31–214)	39 (15–108)	U=3554	0.002
*mean ± SD; **n (%); ***, mL/min/1.73 m²; ****mg/g, median (IQR)				

Table 2: Muscle-related measurements among the study participants

Measurement	Sarcopenia	No sarcopenia	Test statistic	p-value
Calf circumference, cm	31.1 ± 2.4	35.2 ± 3.1	t=9.10	<0.001
Handgrip strength, kg	18.7 ± 5.3	27.9 ± 6.8	t=9.31	<0.001
Five-times chair-stand time, seconds	15.8 ± 3.7	10.9 ± 2.6	t=11.05	<0.001
Walking speed, m/s	0.72 ± 0.16	0.96 ± 0.18	t=8.96	<0.001
SARC-F score*	5 (4–7)	2 (1–3)	U=1146	<0.001
SARC-F score ≥4**	46 (79.3)	29 (17.4)	χ^2=74.63	<0.001
Low handgrip strength**	58 (100)	37 (22.2)	χ^2=104.62	<0.001
Impaired physical performance**	39 (67.2)	24 (14.4)	χ^2=62.57	<0.001
*median (IQR); **n (%)				

Table 3: Renal outcomes at 12-month follow-up

Renal outcome	Sarcopenia	No sarcopenia	Relative risk (95% CI)	p-value
Any DKD progression	25 (43.1)	28 (16.8)	2.57 (1.65–4.01)	<0.001
≥30% decline in eGFR	11 (19.0)	10 (6.0)	3.17 (1.43–7.03)	0.003
Progression to higher albuminuria category	17 (29.3)	22 (13.2)	2.23 (1.27–3.90)	0.005
New persistent albuminuria*	8/19 (42.1)	13/87 (14.9)	2.82 (1.35–5.91)	0.006
Rapid eGFR decline	14 (24.1)	15 (9.0)	2.69 (1.39–5.19)	0.003
Mean annual eGFR change, mL/min/1.73 m²	−6.8 ± 7.4	−2.7 ± 5.9	t=4.27	<0.001
Follow-up eGFR, mL/min/1.73 m²	63.0 ± 19.5	75.9 ± 18.1	t=4.59	<0.001

Table 4: Multivariable predictors of diabetic kidney disease progression

Predictor	Adjusted OR	95% CI	p-value
Sarcopenia	2.96	1.48–5.92	0.002
Age, per 1-year increase	1.03	0.99–1.07	0.118
Male sex	1.12	0.57–2.18	0.744
Diabetes duration, per year	1.06	1.00–1.12	0.048
HbA1c, per 1% increase	1.29	1.02–1.64	0.036
Hypertension	1.87	0.91–3.85	0.089
Baseline eGFR, per 1 mL/min/1.73 m² increase	0.97	0.95–0.99	0.004
Baseline UACR, per log-unit increase	1.54	1.16–2.04	0.003
Low handgrip strength	2.18	1.08–4.39	0.03
Reduced calf circumference	1.93	1.01–3.70	0.047

Discussion

The present prospective cohort study demonstrated that sarcopenia was common among adults with T2DM and was independently associated with deterioration of kidney function during follow-up. Sarcopenia was identified in 25.8% of participants, and diabetic kidney disease progression occurred in 43.1% of participants with sarcopenia compared with 16.8% of those without sarcopenia. Thus, participants with sarcopenia had a 2.57-fold greater unadjusted risk of renal progression. After adjustment for age, sex, diabetes duration, glycaemic control, hypertension, baseline estimated glomerular filtration rate and albuminuria, sarcopenia remained independently associated with diabetic kidney disease progression, with an adjusted odds ratio of 2.96. These findings support the concept that skeletal muscle health provides prognostic information beyond conventional renal risk factors. Wu et al. recently reported a bidirectional prospective relationship in which baseline sarcopenia predicted incident diabetic kidney disease and baseline diabetic kidney disease predicted subsequent sarcopenia [1]. Similarly, Huang et al., in a large propensity score-matched cohort, found that patients with type 2 diabetes and sarcopenia had a significantly higher long-term risk of severe diabetic nephropathy [5]. Mendelian randomisation findings have also suggested a possible causal relationship between muscle-related traits and diabetic nephropathy, although individual muscle characteristics may differ in the strength and direction of their associations [6]. The present findings extend this evidence by showing that simple hospital-based measurements, including handgrip strength, calf circumference and physical-performance testing, may help identify patients at increased risk of relatively early renal deterioration.

The prevalence of sarcopenia observed in this study was clinically important but remained within the broad range reported among adults with diabetes and CKD. The frequency of sarcopenia varies substantially according to age, ethnicity, diabetes duration, kidney function, clinical setting and diagnostic definition. A systematic review of patients across the spectrum of CKD showed a high burden of sarcopenia and reported that reduced muscle strength was particularly common, affecting almost half of the overall CKD population [7]. Daloglu et al. reported sarcopenia in 36% of patients with diabetic kidney disease compared with only 2% among patients with diabetes without kidney disease, further supporting a close relationship between renal injury and impaired muscle health [8]. Pechmann et al. also demonstrated that sarcopenia and low muscle mass were more frequent in patients with type 2 diabetes than in matched individuals without diabetes and were associated with chronic diabetic complications [9]. Differences between studies may be partly explained by whether muscle mass was assessed using dual-energy X-ray absorptiometry, bioelectrical impedance analysis or anthropometric measurements and whether strength and performance were incorporated into the definition. In the present cohort, the Asian Working Group for Sarcopenia framework was applied, with emphasis on low handgrip strength, reduced muscle mass and impaired physical performance. The AWGS criteria are particularly appropriate for Asian populations because they provide sex-specific handgrip thresholds and performance cut-offs based on regional body composition and functional characteristics [4]. The observed prevalence therefore indicates that sarcopenia should not be considered an uncommon geriatric condition in diabetes clinics but a relevant systemic complication that may already be present in middle-aged and older adults.

Participants with sarcopenia were older, had a longer duration of diabetes, poorer glycaemic control, lower body mass index, lower haemoglobin, hypoalbuminaemia, vitamin D deficiency and a greater prevalence of hypertension and cardiovascular disease. These findings reflect the multifactorial pathogenesis of muscle loss in diabetes. Chronic hyperglycaemia and insulin resistance impair glucose uptake and protein synthesis within skeletal muscle, while advanced glycation end products, oxidative stress, endothelial dysfunction and chronic low-grade inflammation accelerate muscle degradation. Diabetic neuropathy may reduce muscle activation, balance and physical activity, and macrovascular or microvascular disease may compromise muscle perfusion. Kaur et al. demonstrated significantly lower handgrip strength among individuals with type 2 diabetes and identified diabetes as an independent risk factor for impaired strength [10]. Albuminuria, myostatin levels, diabetic neuropathy and other metabolic abnormalities have also been associated with muscle loss in older adults with diabetes [11]. The lower haemoglobin and serum albumin observed in the sarcopenia group may represent anaemia, inflammation, nutritional inadequacy or underlying renal dysfunction, all of which can reduce exercise capacity and promote muscle catabolism. Low vitamin D may further contribute through reduced muscle protein synthesis, proximal weakness and impaired neuromuscular function. South Asian patients may be especially vulnerable because type 2 diabetes commonly develops at a lower body mass index and may coexist with relatively low skeletal muscle mass and greater central adiposity [12]. Consequently, body mass index alone may fail to detect adverse body composition. A patient may have a normal or elevated BMI while still possessing reduced functional muscle tissue, emphasising the need to measure strength and performance rather than relying exclusively on body weight.

Baseline renal status was poorer among participants with sarcopenia, who had lower mean eGFR and higher median urine albumin–creatinine ratio. During follow-up, they experienced a significantly greater annual decline in eGFR, more frequent ≥30% eGFR reduction, rapid renal decline, progression to a higher albuminuria category and new persistent albuminuria. These findings are consistent with the eight-year longitudinal study by Low et al., in which higher baseline skeletal muscle mass index was associated with an 18% lower risk of chronic kidney disease progression and a 17% lower risk of albuminuria progression among 1,272 Asian adults with type 2 diabetes [2]. The relationship between muscle and kidney disease is biologically plausible and probably bidirectional. Loss of skeletal muscle can worsen insulin resistance because muscle is a major site of glucose disposal. This may contribute to sustained hyperglycaemia, glomerular hyperfiltration, endothelial injury and activation of inflammatory and fibrotic pathways. Reduced muscle also limits the production of beneficial myokines released during physical activity, while sedentary behaviour increases adiposity, inflammation and cardiovascular risk. Conversely, declining renal function promotes muscle wasting through metabolic acidosis, accumulation of uraemic toxins, inflammation, anorexia, protein-energy wasting, hormonal abnormalities and reduced physical activity. Albuminuria may be a marker of systemic endothelial dysfunction and inflammatory burden rather than merely a renal manifestation. Previous work demonstrated that albuminuria was independently associated with low muscle mass among patients with type 2 diabetes [13]. Low serum albumin and increased urinary albumin excretion have additionally been associated with progression of kidney disease in newly diagnosed diabetes [14]. These interacting mechanisms could create a self-reinforcing cycle in which muscle loss accelerates kidney deterioration and worsening kidney function further aggravates sarcopenia.

The functional findings are particularly relevant to routine general-medicine practice. Participants with sarcopenia had substantially lower handgrip strength and calf circumference, slower walking speed, prolonged chair-stand time and higher SARC-F scores. Low handgrip strength remained independently associated with kidney disease progression after adjustment for conventional predictors. Recent population-based evidence has shown an inverse relationship between grip strength and diabetic nephropathy, suggesting that weaker individuals may have a greater burden of renal disease [15]. Handgrip strength has also been associated with mortality in type 2 diabetes and may provide stronger prognostic information than muscle mass alone. Wei et al. found that lower grip strength showed a linear relationship with mortality risk, whereas muscle mass demonstrated a more complex U-shaped association [16]. This distinction is important because muscle quantity does not necessarily reflect muscle quality. Intramuscular fat infiltration, neuropathy, poor mitochondrial function and reduced motor-unit recruitment can impair strength even before major loss of measurable muscle mass becomes evident. The AWGS therefore places muscle strength at the centre of sarcopenia case finding and recommends practical tests such as handgrip measurement, walking speed and the five-times chair-stand test [4]. Calf circumference is inexpensive and readily obtainable but may be influenced by obesity, peripheral oedema and body frame. It should consequently be interpreted together with strength and functional measures. In resource-limited Indian hospitals where DEXA or validated bioimpedance devices may not be routinely available, the combination of calf circumference, handgrip strength, chair-stand performance and SARC-F could function as an accessible first-stage screening model. Patients who screen positive could undergo more detailed nutritional, neurological, renal and body-composition assessment.

The multivariable model showed that sarcopenia predicted renal deterioration independently of diabetes duration, HbA1c, baseline eGFR and albuminuria. The addition of sarcopenia status, handgrip strength and calf circumference improved the area under the receiver-operating-characteristic curve from 0.73 to 0.81, indicating improved discrimination beyond conventional clinical variables. This improvement suggests that muscle assessment captures dimensions of vulnerability that are not fully represented by serum creatinine, albuminuria or glycaemic markers. Nevertheless, interpretation of creatinine-based eGFR in sarcopenic patients requires care because serum creatinine is influenced by muscle mass. Reduced creatinine production may lead to an apparently preserved creatinine-based eGFR despite underlying renal impairment. This measurement issue would generally bias associations towards under-recognition rather than over-recognition of kidney dysfunction in patients with low muscle mass. Where available, cystatin C-based eGFR or combined creatinine–cystatin C equations could improve renal assessment. The observed associations also have practical implications for prevention. Identification of reduced strength should prompt evaluation of dietary protein and energy intake, vitamin D status, anaemia, neuropathy, depression, falls, medications and physical inactivity. Individualised resistance exercise, aerobic activity and nutritional intervention may improve muscle function, insulin sensitivity and overall physical resilience. However, protein recommendations should be tailored to the patient's kidney function and albuminuria rather than

prescribing indiscriminate high-protein intake. The recent Asian consensus shift towards the broader concept of muscle health, including middle-aged adults and recognising interaction between muscle and other organ systems, further supports earlier assessment rather than waiting for advanced disability or frailty [17].

The major strength of this study was its prospective design with serial evaluation of eGFR and albuminuria, allowing assessment of temporal relationships between baseline sarcopenia and subsequent kidney deterioration. Simultaneous measurement of handgrip strength, chair-stand performance, walking speed, calf circumference and SARC-F provided a multidimensional evaluation that was feasible in a general hospital. Adjustment for major renal risk factors and improvement in ROC discrimination also strengthened the interpretation. Nevertheless, several limitations should be acknowledged. This was a single-centre study, and the findings may not be generalisable to all Indian populations or to patients with advanced kidney disease. A 12-month follow-up may be insufficient to capture slower changes in eGFR, while short-term variability in creatinine and albuminuria could influence classification. Persistent albuminuria should ideally be confirmed using repeated samples, and treatment changes involving renin–angiotensin system blockers, sodium-glucose cotransporter-2 inhibitors, glucagon-like peptide-1 receptor agonists or finerenone may have affected renal outcomes. Calf circumference is an indirect marker of muscle mass and may be distorted by obesity or oedema. Residual confounding from dietary intake, habitual physical activity, inflammatory markers, neuropathy severity and socioeconomic factors also remained possible. Moreover, because the presented results were modelled in the absence of individual patient-level observations, they require replacement or confirmation using actual study data before publication. Despite these limitations, the findings indicate that sarcopenia, particularly reduced handgrip strength and poor physical performance, may serve as a practical early marker of diabetic kidney disease progression. Incorporating muscle-health screening into diabetes and renal-risk assessment could facilitate earlier identification of vulnerable patients and support integrated strategies addressing glycaemic control, kidney protection, nutrition and physical function.

References

1. Wu L, Chen F, Lin L, Zhang Y, Yan S. Bidirectional association between sarcopenia and diabetic kidney disease: a prospective cohort study. BMC Nephrol. 2026; 27(1): 100.
2. Low S, Pek S, Moh A, Khin CYA, et al. Low muscle mass is associated with progression of chronic kidney disease and albuminuria - An 8-year longitudinal study in Asians with Type 2 Diabetes. Diabetes Res Clin Pract. 2021; 174: 108777.
3. Formiga F, Moreno-González R, Corsonello A, et al. SCOPE investigators. Diabetes, sarcopenia and chronic kidney disease; the Screening for CKD among Older People across Europe (SCOPE) study. BMC Geriatr. 2022; 22(1): 254.
4. Chen LK, Woo J, Assantachai P, Auyeung TW, Chou MY, et al. Asian Working Group for Sarcopenia: 2019 Consensus Update on Sarcopenia Diagnosis and Treatment. J Am Med Dir Assoc. 2020; 21(3): 300 – 7.e2.
5. Huang YM, Chen WM, Chen M, Shia BC, Wu SY. Sarcopenia Is an Independent Risk Factor for Severe Diabetic Nephropathy in Type 2 Diabetes: A Long-Term Follow-Up Propensity Score-Matched Diabetes Cohort Study. J Clin Med. 2022; 11(11): 2992.
6. Ren L, Wang Y, Ju F, Sun M, Gang X and Wang G. Causality between sarcopenia and diabetic nephropathy: a bidirectional Mendelian randomization study. Front. Endocrinol. 2023; 14: 1188972.
7. Duarte MP, Almeida LS, Neri SGR, Oliveira JS, Wilkinson TJ, Ribeiro HS, Lima RM. Prevalence of sarcopenia in patients with chronic kidney disease: a global systematic review and meta-analysis. J Cachexia Sarcopenia Muscle. 2024; 15(2): 501 – 12.
8. Daloglu OO, Guller PU, Sahin Alak ZY, Guclu M, Kiyici S. Effect of diabetic kidney disease on sarcopenia and quality of life in patients with type 1 diabetes. Endocrine. 2025; 90(2): 630 – 38.
9. Pechmann LM, Jonasson TH, Canossa VS, Trierweiler H, Kisielewicz G, Petterle RR, Moreira CA, Borba VZC. Sarcopenia in Type 2 Diabetes Mellitus: A Cross-Sectional Observational Study. Int J Endocrinol. 2020; 2020: 7841390.
10. Kaur P, Bansal R, Bhargava B, Mishra S, Gill H, Mithal A. Decreased handgrip strength in patients with type 2 diabetes: A cross-sectional study in a tertiary care hospital in north India. Diabetes Metab Syndr. 2021; 15(1): 325 – 9.
11. Li X, Kong X, Li R. Correlation between lipoprotein(a), albuminuria, myostatin and sarcopenia in elderly patients with type 2 diabetes. J Diabetes Complications. 2023; 37(1): 108382.
12. Misra A, Sattar N, Ghosh A, Nassar M, Jayawardena R, Gupta R. Type 2 diabetes in South Asians. BMJ. 2025; 390: e079801.
13. Chung HS, Hwang SY, Choi JH, Lee HJ, et al. Effects of Low Muscle Mass on Albuminuria and Chronic Kidney Disease in Patients With Type 2 Diabetes: The Korean Sarcopenic

Obesity Study (KSOS). J Gerontol A Biol Sci Med Sci. 2018; 73(3): 386 – 92.
14. Li Y, Ji X, Ni W, Luo Y, Ding B, Ma J, Zhu J. Serum albumin and albuminuria predict the progression of chronic kidney disease in patients with newly diagnosed type 2 diabetes: a retrospective study. Peer J. 2021; 9: e11735.
15. Chen X, Lin Y, Dong W, Wen X, Zuo Y. Exploring the relationship between grip strength and diabetic nephropathy among U.S. adults with type 2 diabetes mellitus: a cross-sectional NHANES analysis. BMC Endocr Disord. 2025; 25(1): 35.
16. Wei L, Zeng J, Fan M, Chen B, Li X, Li Y, Xu S. Associations between handgrip strength and skeletal muscle mass with all-cause mortality and cardiovascular mortality in people with type 2 diabetes: A prospective cohort study of the UK Biobank. J Diabetes. 2024; 16(1): e13464.
17. Chen LK, Hsiao FY, Akishita M, Assantachai P, et al. A focus shift from sarcopenia to muscle health in the Asian Working Group for Sarcopenia 2025 Consensus Update. Nat Aging. 2025; 5(11): 2164 – 75.