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Research Article

Comparison of Sarcopenia in Diabetics and Non-Diabetics: Prevalence and Impact on Muscle Health.

Dr. Farhana Ahad[1], Dr. Mahrukh Malik[2], Dr. Masarat Nazeer[3]*.
[1]Professor and Head, Department of Physiology, SKIMS, MCH, Bemina.
[2]Senior Resident, Department of Physiology, GMC, Anantnag.
[3]Assistant Professor, Department of Physiology, Madhubani Medical College Bihar.

***Corresponding Author**
Dr. Masarat Nazeer.
musarrat669@gmail.com

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Abstract: A gradual reduction in muscle mass, strength and function over time, known as sarcopenia, is a growing concern among patients who are presenting with type 2 diabetes mellitus (T2DM). Rapid muscle loss may be associated with insulin resistance, chronic inflammation and oxidative stress that are associated with T2DM. This study aimed to assess the prevalence of sarcopenia in adult age group between 40-60 years and compare the association of sarcopenia in diabetic and non-diabetic adults. ***Methodology:*** The present comparative analysis was done at the Department of Physiology, SKIMS Medical College & Hospital, Srinagar, India for two years. About 136 participants were enrolled in this study, of them 68 had a diagnosis of T2DM (>5 years duration) and 68 were age matched controls. We assessed clinically relevant parameters such as body mass index, handgrip strength and gait speed. The criteria for the diagnosis of sarcopenia were applied. ***Results:*** The results revealed a significantly higher prevalence of sarcopenia among diabetics in comparison to non-diabetics (16.18% vs. 8.82%; p=0.039). The average BMI values were on higher side in the diabetics (27.15) as compared with the non-diabetics (25.46) with difference being statistically significant (p<0.05). Interestingly, the diabetics reflected significantly less handgrip strength than the non-diabetics (21.36 vs. 27.60) (p<0.01). Diabetics had a significantly higher gait speed (17.16 vs. 9.97) than non-diabetics (p<0.01). Women had higher prevalence rates, and there was a higher prevalence rate among those aged 51-60 years. ***Conclusion:*** Sarcopenia was associated with T2DM and changes in muscle strength and physical performance.

Keywords: Sarcopenia, T2DM, Muscle Strength, Insulin Resistance, Gait Speed, Handgrip Strength, Metabolic Dysfunction, Physical Performance, Aging, Neuromuscular Dysfunction.

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INTRODUCTION

Sarcopenia, the progressive decline in skeletal muscle mass, strength and function is a major public health problem in the middle and elderly ageedgroups. 1-3 Although muscle loss was thought to be a problem of ageing, recent studies have identified that sarcopenia is linked to chronic metabolic disorders including Type 2 Diabetes Mellitus (T2DM), a complex metabolic disease, with insulin resistance, chronic inflammation and glucose metabolism impairment representing key muscle-degrading factors associated with this disease.4-6 Insulin is also important for muscle protein synthesis and diabetes may cause insulin resistance, which leads to loss of muscle mass and muscle function. Also, continued hyperglycemia and oxidative stress promote further loss of muscle and eventually, persons with T2DM have a higher likelihood of sarcopenia than non-diabetic of the same age.

This vicious cycle between diabetes and sarcopenia ultimately results in worsening diabetes outcomes.7,8 In addition, the prevalence of sarcopenia in diabetics also puts them at higher risk of falls, fractures, hospitalization and a diminished quality of life, and therefore it is a very important condition to detect and treat early. Even with increasing awareness, there is little research on the prevalence of sarcopenia among middle aged adults (40-60 years) with diabetes and without diabetes.9 With this in mind, the present study was objectively conducted to evaluate the prevalence of sarcopenia in adults aging 40-60 and to determine any possible clinical differences between T2DM and non-diabetics. The identification of the magnitude of sarcopenia within these groups will lead to insights into the effects of diabetes on muscle health and highlight the need for targeted prevention and management strategies such as lifestyle interventions, exercise and nutrition.

MATERIALS AND METHODS

This comparative analytical study was conducted over two years in the Department of Physiology at SKIMS Medical College & Hospital, a tertiary care center in Srinagar, Jammu and Kashmir. The study population included patients referred from the Department of Medicine at SKIMS Medical College Hospital, Bemina, Srinagar. Participants were selected based on specific inclusion and exclusion criteria. Individuals aged 40–60 years attending the outpatient department of medicine who provided informed consent were included. Both male and female participants were considered, with the diabetic group comprising patients diagnosed with Type 2 Diabetes Mellitus (T2DM) for more than five years.Type 2 Diabetes Mellitus (T2DM) was specifically defined by reduced sensitivity to insulin action and an inability to compensate for this resistance

through sufficient insulin secretionExclusion criteria included individuals who did not consent, those with systemic illnesses such as severe hepatic, renal, or cardiac insufficiency, tuberculosis, or malignancy, and those who had undergone recent weight loss surgery or had a metal stent or pacemaker. Additionally, patients on medications affecting muscle mass, including NSAIDs, statins, steroids, and cytotoxic drugs, were excluded.

The sample size was determined using G-Power Sample Size Calculation Software, considering a sarcopenia prevalence of 13% in diabetics and 4% in non-diabetics, with a power of 90% and a permissible error of 2%. This calculation resulted in a required sample of 68 participants per group, making a total of 136. A consecutive non-probability sampling method was employed, wherein eligible participants were recruited sequentially. If a participant declined, the next available patient was invited to participate.

All participants who met the inclusion and exclusion criteria were approached for participation. The study's aim, objectives, and data confidentiality were explained to each participant, and written informed consent was obtained. Following this, a face-to-face interview was conducted, and relevant information was documented. Participants were then assessed for sociodemographic and clinical characteristics likesuch as age, sex, residence, education, occupation, marital status, socioeconomic status, diabetes duration, and family history of diabetesand subjected to a series of standardized tests to evaluate musculoskeletal integrity, mobility, strength, and balance.

Data collection was conducted in collaboration with the Department of Medicine at SKIMS Medical College & Hospital over a period of two years, ensuring a comprehensive assessment and analysis of sarcopenia in diabetics and non-diabetics. A comprehensive assessment of sarcopenia was conducted using objective tests to evaluate muscle mass, strength, and physical performance.

Body Mass Index (BMI): BMI was calculated based on the height and weight of each participant to provide a general measure of body composition.

Skeletal Muscle Mass Assessment: Dual-energy X-ray absorptiometry (DEXA) is the standard method for assessing skeletal muscle mass. However, due to its limited availability, alternative measurements such as mid-arm circumference and calf circumference were utilized, as they strongly correlate with skeletal muscle mass.

- Mid-Arm Circumference: Participants were instructed to raise their arm to shoulder level with the elbow bent at a 90º angle and flex the biceps. The circumference was measured at the point of maximum girth.
- Calf circumference was recorded at its widest point while the participant stood with feet shoulder-width apart.

Muscle mass was expressed as kg/m^2 after height adjustment. Low muscle mass was defined according to AWGS 2019 cutoffs: <7.0 kg/m^2 in men and <5.4 kg/m^2 in women by DEXA, and <7.0 kg/m^2 in men and <5.7 kg/m^2 in women by BIA. CT and MRI were also recognized methods for muscle mass assessment. Handgrip strength was measured in both hands using a dynamometer; three readings were taken with rest between attempts, and the highest value was recorded. Values <28 kg in men and <18 kg in women indicated reduced strength. For the chair stand test, participants rose from a standard chair five times without using their arms. A duration of ≥12 seconds indicated reduced lower-limb performance.

Gait speed was assessed over 6 metres; a speed <1.0 m/s was considered reduced. AWGS 2019 also recommends calf circumference, SARC-F, and SARC-CalF for initial screening. Cutoffs were <34 cm in men, <33 cm in women, SARC-F ≥4, and SARC-CalF ≥11.

The study was approved by the Institutional Ethics Committee of SKIMS Medical College & Hospital, Bemina. Participants provided informed consent after receiving an explanation in their native language and data were anonymized, checked, and stored securely in a password-protected file.

Statistical Analysis

The collected data were tabulated in an Excel spreadsheet under the supervision of a statistician. Statistical analysis was performed using SPSS version 22.0 (SPSS Inc., Chicago, USA). Means and standard deviations were calculated for continuous variables, while categorical variables were analyzed using frequency distributions. The differences between diabetic and non-diabetic groups were determined using the t-test and chi-square test, with statistical significance set at $p < 0.05$.

RESULTS

Table 1: Demographic and Occupational Characteristics of Study Groups

Variable	Diabetic (n = 68)	Non-Diabetic (n = 68)	p-value
Gender			
Male	31 (45.6)	34 (50.0)	**0.27**
Female	37 (54.4)	34 (50.0)	
Age Group (years)			
40-50	28 (41.2)	33 (48.5)	**0.21**
51-60	40 (58.8)	35 (51.5)	
Occupation			
Sedentary	54 (79.4)	63 (92.6)	**0.067**
Active	14 (20.6)	5 (7.4)	

The table 1presents a comparison of gender distribution, age groups, and occupation between diabetic and non-diabetic participants. Among the diabetic group, 31 (45.6%) were male, while 37 (54.4%) were female, compared to 34 (50.0%) males and 34 (50.0%) females in the non-diabetic group (p = 0.27). Age-wise, 28 (41.2%) diabetic individuals were in the 40–50 years group, while 40 (58.8%) were in the 51–60 years group. In contrast, 33 (48.5%) non-diabetics were aged 40–50 years, and 35 (51.5%) were 51–60 years old (p = 0.21). Regarding occupation, a sedentary lifestyle was more prevalent among diabetics (54, 79.4%) compared to non-diabetics (63, 92.6%), whereas an active occupation was reported by 14 (20.6%) diabetics and only 5 (7.4%) non-diabetics (p = 0.067).

Table 2: Comparison of Co-morbidities Between Diabetic and Non-Diabetic Groups

Co-morbidities	Diabetic n (%)	Non-Diabetic n (%)	p-value
HTN	36 (52.94)	24 (35.29)	0.42
HTx	14 (20.6)	6 (7.4)	

Table 2 presents the distribution of co-morbidities among diabetic and non-diabetic participants. Hypertension (HTN) was more prevalent among diabetics, observed in 52.94% of cases, compared to 35.29% in non-diabetics. Similarly, a history of transplant (HTx) was recorded in 20.6% of diabetic individuals and 7.4% of non-diabetics. However, the difference in comorbidities between the groups was not statistically significant (p=0.42). Among diabetic participants, the majority (79.41%) had a disease duration of 5–10 years, while 13.24% had diabetes for 11–15 years, and 7.35% for 16–20 years. Regarding medication history, 77.94% were on oral hypoglycemic agents, whereas 22.06% required insulin therapy.

Table 3: Comparison of Physical and Anthropometric Parameters Between Diabetic and Non-Diabetic Groups

Variable	Diabetic (Mean ± SD)	Non-Diabetic (Mean ± SD)	p-value
Gait Speed (seconds)	17.16 ± 6.15	9.97 ± 3.39	<0.01*
BMI (kg/m^2)	27.15 ± 3.85	25.46 ± 2.97	0.005*
Arm Circumference (cm)	27.35 ± 3.10	28.79 ± 2.92	0.006*
Calf Circumference (cm)	31.93 ± 3.20	34.15 ± 2.21	<0.01*
Hand Grip Strength (kg)	21.36 ± 7.66	27.60 ± 8.79	<0.01*
Chair Stand (seconds)	23.49 ± 5.74	15.25 ± 3.29	<0.01*

We observed that diabetic participants had a significantly lower mean gait speed (17.16 ± 6.15 seconds) compared to non-diabetic individuals (9.97 ± 3.39 seconds, p < 0.01). The mean BMI was higher in diabetics (27.15 ± 3.85 kg/m^2) than in non-diabetics (25.46 ± 2.97 kg/m^2, p = 0.005). Similarly, arm circumference was lower in diabetics (27.35 ± 3.10 cm) compared to non-diabetics (28.79 ± 2.92 cm, p = 0.006), and calf circumference was also significantly lower (31.93 ± 3.20 cm vs. 34.15 ± 2.21 cm, p < 0.01). Additionally, diabetics had reduced hand grip strength (21.36 ± 7.66 kg vs. 27.60 ± 8.79 kg, p < 0.01) and took longer to complete the chair stand test (23.49 ± 5.74 seconds vs. 15.25 ± 3.29 seconds, p < 0.01). These findings indicate that diabetic individuals exhibit poorer physical performance and reduced muscle strength compared to their non-diabetic counterparts.

Table 4: Comparison of Sarcopenia Status Between Diabetic and Non-Diabetic Groups

Sarcopenia Status	Diabetic n(%)	Non-Diabetic n(%)	p-value
Present	11 (16.18)	6 (8.82)	0.039*
Absent	57 (83.82)	62 (91.18)	
Total	68 (100)	68 (100)	

Sarcopenia was more prevalent among diabetic individuals (11 [16.18%]) compared to non-diabetic individuals (6 [8.82%]), with a statistically significant difference (p = 0.039). Conversely, the majority of participants in both groups did

not have sarcopenia, with 57 (83.82%) diabetics and 62 (91.18%) non-diabetics classified as sarcopenia-free. These findings suggest a potential association between diabetes and an increased risk of sarcopenia.

Table 5: Sarcopenia Distribution by Gender Among Diabetic and Non-Diabetic Groups

Sarcopenia Status	Diabetic Male n (%)	Diabetic Female n(%)	Non-Diabetic Male n(%)	Non-Diabetic Female n(%)
Present	4 (12.90)	7 (18.92)	2 (5.88)	4 (11.76)
Absent	27 (87.10)	30 (81.08)	32 (94.12)	30 (88.24)
Total	31 (100)	37 (100)	34 (100)	34 (100)

Sarcopenia was more prevalent among diabetic females (7 [18.92%]) compared to diabetic males (4 [12.90%]). Similarly, among non-diabetic participants, sarcopenia was observed in 4 (11.76%) females and 2 (5.88%) males. The majority of participants in all groups were free of sarcopenia, with diabetic males (27 [87.10%]) and non-diabetic males (32 [94.12%]) showing slightly lower prevalence compared to diabetic females (30 [81.08%]) and non-diabetic females (30 [88.24%]). These findings suggest a potential influence of gender and diabetes status on sarcopenia prevalence.

DISCUSSION

The present study included 136 participants, with 68 individuals in each group. The two study groups were comparable with respect to sex and age distribution. Most participants were aged 51–60 years, which is relevant because muscle strength and physical performance decline progressively with age.9-11 BMI, however, was significantly higher among diabetic participants (27.15 vs. 25.46 kg/m^2; $p<0.05$). A similar difference was reported by Sravya et al. (27.4 vs. 26.9 kg/m^2).12 Increased adiposity in T2DM may be accompanied by fat deposition within skeletal muscle, which can adversely affect muscle quality and function.13-15Thus, a higher body weight in patients with diabetes does not exclude the possibility of clinically important muscle impairment. The diabetic participants in this study had established disease, with 79.41% having a duration of 5–10 years, while only 7.25% had diabetes for 16–20 years and most patients (77.94%) were receiving oral hypoglycaemic agents and 22.06% were on insulin therapy. Although the present study was not designed to examine the relationship between treatment and sarcopenia, the duration of diabetes is of particular interest. Pechmann et al. reported a longer duration of diabetes, approximately 19 years, among patients with sarcopenia compared with those without sarcopenia.16 Interestingly, HbA1c levels were similar between the groups in that study.

This suggests that the cumulative effects of diabetes may be relevant to muscle health rather than glycaemic status at a single point in time. Persistent hyperglycaemia and insulin resistance can adversely affect skeletal muscle. Impaired insulin signalling reduces glucose utilisation and may interfere with normal muscle protein synthesis.14 Moreover, oxidative stress plus metabolic abnormalities associated with prolonged diabetes may further favour muscle protein breakdown. These processes provide a plausible explanation for the poorer muscle strength observed among diabetic participants in the present study. However, the present data do not allow the individual contribution of glycaemic control, diabetes duration or treatment to be determined. Hypertension was also more frequent among diabetic participants. This finding is consistent with the well-established coexistence of hypertension and T2DM. Pechmann et al. reported hypertension to be nearly three times more common among diabetic patients, while Brazilian epidemiological data showed a prevalence approximately 2.4 times higher in individuals with diabetes.16

The association between these conditions has been linked to insulin resistance, endothelial dysfunction and increased arterial stiffness.17 From the perspective of skeletal muscle, vascular abnormalities may have additional consequences because impaired perfusion can reduce the delivery of oxygen and nutrients to muscle tissue. This may be particularly relevant in patients with long-standing diabetes. The possible effect of antihypertensive treatment on muscle function is worth mentioning, although it cannot be assessed directly in the present study. Some studies have suggested that ACE inhibitors may have a favourable effect on muscle strength and may slow the decline in skeletal muscle mass.18,19 Whether such an effect has any clinical relevance in patients with T2DM and sarcopenia remains uncertain. Differences in antihypertensive treatment between participants could also influence muscle-related outcomes and therefore represent a potential confounding factor. A clear difference was observed in handgrip strength. Handgrip strength was markedly lower in the diabetic group than in controls (21.36 vs. 27.60; $p<0.01$), which is in consonance with the study of Sravya et al., who reported poorer handgrip strength among individuals with sarcopenia.20 Handgrip strength is a simple measure, but it provides useful information about general muscle strength and has an established role in the assessment of sarcopenia.

The lower values observed in the diabetic group suggest that diabetes may be associated with clinically relevant impairment in muscle strength even during middle age. Several factors may contribute to this reduction. Insulin resistance can impair glucose utilisation by skeletal muscle and interfere with anabolic signalling, while persistent hyperglycaemia and altered lipid metabolism may promote intramuscular lipid accumulation.14,16,21 Myosteatosis can adversely affect muscle quality and contractile function. The effect may therefore extend beyond a simple reduction in muscle quantity. Changes in myostatin, which negatively regulates skeletal muscle growth, have also been implicated in diabetes-related muscle loss. Dial et al. reported an association between altered myostatin activity and muscle abnormalities in diabetes.22 The type of muscle fibre affected may also be relevant. Jun et al. reported that muscle atrophy in diabetes may particularly involve type

II muscle fibres, which are important for rapid force production.23 Loss or dysfunction of these fibres could result in a considerable decline in strength even when the reduction in total muscle mass is less pronounced. This may explain why measures of muscle strength and physical performance can provide clinically useful information alongside assessment of muscle mass.

Sarcopenia is increasingly recognized in patients with type 2 diabetes mellitus (T2DM), particularly as the duration of diabetes increases. In the present study, sarcopenia was observed in 16.18% of diabetic participants compared with 8.82% of non-diabetic participants, and this difference was statistically significant ($p=0.039$). The higher prevalence among diabetic participants supports the association between T2DM and impaired skeletal muscle health. Similar findings have been reported previously. Pechmann et al. found sarcopenia in 12.9% of diabetic patients compared with 5.4% of controls, while Sravya et al. reported prevalence rates of 22% and 8.8%, respectively.16,20 The differences in prevalence between studies may be related to variation in age groups, diagnostic criteria, duration of diabetes and other characteristics of the study populations.

Gait speed also showed a statistically significant difference between the two groups, with mean values of 17.16 among diabetic participants and 9.97 among non-diabetic participants ($p<0.01$). The direction of this difference requires careful interpretation because the clinical meaning of the numerical values depends on the unit and method used for measuring gait speed. Sravya et al. reported poorer gait performance among sarcopenic individuals.20 Therefore, the present gait-speed finding should be interpreted in the context of the measurement protocol rather than assuming that the higher numerical value necessarily represents better or worse physical performance. Nevertheless, the significant difference indicates that physical performance differed between the two study groups. Sarcopenia was more frequent among females than males and was particularly common in participants aged 51–60 years. The age-related decline in muscle reserve may become more apparent in this period of life, particularly when accompanied by diabetes. The higher frequency among women may also reflect differences in body composition, muscle mass and hormonal factors, although the present study was not designed to determine the reasons for this sex difference. These findings nevertheless identify older adults and women with T2DM as groups in whom assessment of muscle strength and physical performance may be particularly relevant.

The association between T2DM and sarcopenia is likely to involve several processes acting together. Insulin resistance is central to this relationship. Reduced insulin action affects both glucose metabolism and the anabolic response of skeletal muscle. Kalyani et al. described the role of impaired insulin signalling in reduced muscle protein synthesis and increased muscle loss.14 Chronic hyperglycaemia may additionally increase oxidative stress and inflammatory activity, with effects on mitochondrial function and muscle cell integrity.16,24 These changes may gradually reduce both muscle quantity and muscle quality. Vascular dysfunction and diabetic neuropathy may add to this burden by affecting muscle perfusion and neuromuscular function.19,24,25 Repeated denervation and incomplete reinnervation may contribute to muscle weakness and atrophy. In patients with longer-standing diabetes, these factors may act together with the normal age-related decline in muscle mass and strength.

The result is a clinical picture in which reduced strength and impaired physical function may become evident before severe muscle wasting is recognized. The findings of the present study therefore support the need to consider muscle health as part of the clinical assessment of patients with T2DM. A higher BMI should not be taken as evidence of adequate muscle health, particularly when muscle strength is reduced. Handgrip strength and assessment of physical performance are relatively simple measures and may help identify patients who require further evaluation for sarcopenia. This may be particularly useful in middle-aged and older patients with longer disease duration. The present study has some limitations. The sample size was relatively small and the observational design does not permit a causal relationship between T2DM and sarcopenia to be established. Factors like; physical activity, nutritional intake, glycaemic control, diabetic complications and medication use may also influence muscle function and were not examined in sufficient detail to determine their independent effects. Despite these limitations, the higher prevalence of sarcopenia and the lower handgrip strength observed among diabetic participants provide clinically relevant evidence of impaired muscle health in this population.

CONCLUSION

The findings of the present study indicated that T2DM is associated with a greater burden of sarcopenia and reduced muscle strength in adults aged 40–60 years. The association is likely to reflect the combined effects of insulin resistance, chronic hyperglycaemia, metabolic abnormalities, vascular changes and neuromuscular impairment rather than a single pathological process. Early recognition of reduced muscle strength and physical performance may therefore be useful in patients with T2DM, particularly those with longer disease duration and those approaching the later part of middle age. Resistance exercise, appropriate nutritional management and good diabetes control remain reasonable components of care, although their specific effect on sarcopenia should be assessed in prospective studies.

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