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%BF, Rather Than BMI, is Associated with an Increased Risk of Sarcopenia in Hospitalized Postmenopausal Chinese Women with Type 2 Diabetes Mellitus

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Purpose: To investigate the relationship between obesity indices and sarcopenia in postmenopausal patients with type 2 diabetes mellitus (T2DM) at different body mass index (BMI) levels.

Patients and Methods: This retrospective cross-sectional study included 298 hospitalized postmenopausal women diagnosed with T2DM. We collected demographic, biochemical, and anthropometric data on each subject. Body composition was measured using dual-energy X-ray absorptiometry (DXA), and skeletal muscle mass index (SMI) and body fat percentage (%BF) were calculated. According to BMI stratification, the patients were divided into normal group A (18.5 $kg/m^2 \leq BMI < 24$ kg/m^2), overweight group B (24.0 $kg/m^2 \leq BMI < 28$ kg/m^2), and obesity group C (28.0 $kg/m^2 \leq BMI < 35$ kg/m^2).

Results: From group A to group C, SMI (5.21±0.56 vs 5.48±0.56 vs 6.03±0.69) increased gradually ($P < 0.05$). Logistic regression analysis indicated that for each 1-unit increase in BMI, the risk of sarcopenia decreased by 63.2% (OR=0.368, 95% CI 0.215–0.629, P=0.000) in group A. Age (OR=1.077, 95% CI 1.015–1.144, P=0.015) and %BF (OR=1.094, 95% CI 1.010–1.186, P=0.028) increased the risk of sarcopenia by 1.077 and 1.094 times, respectively, in group B. While every 1-unit increase in BMI, the risk of sarcopenia decreased by 35% (OR=0.650, 95% CI 0.430–0.983, P=0.041) in group B. %BF (OR=1.459, 95% CI 1.093–1.949, P=0.010) increased the risk factors of sarcopenia by 1.459 times in group C.

Conclusion: In postmenopausal patients with T2DM, BMI had a protective effect on the occurrence of sarcopenia within a certain range, and with the increase of BMI, the risk of sarcopenia was increasing by increased %BF levels in overweight and obese patients.

Keywords: postmenopausal women, T2DM, sarcopenia, %BF, BMI

Introduction

The incidence of type 2 diabetes mellitus (T2DM) in China had been increasing year by year. Studies had shown that the prevalence of sarcopenia in T2DM patients had reached 28%.[1] Sarcopenia has recently been identified as a complication of T2DM.[2] It is characterized by a progressive reduction in skeletal muscle mass associated with aging, accompanied by muscle strength and/or muscle function decline syndrome, which could lead to an increase in a series of adverse consequences, such as falls, fractures, bed rest, readmission, death, etc.[3] One of the metabolic complications caused by obesity was diabetes.[4] Adipose tissue played a key role in the development of insulin resistance,[5] and the increase of intermuscular fat (IMAT) puts the skeletal muscle system in a low-grade inflammation,[6] thus promoting the occurrence of sarcopenia. It could be seen that obesity, diabetes, and sarcopenia were inextricably linked.

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In recent years, sarcopenic obesity had received increasing attention in the industry, characterized by low muscle mass and high body fat, with an increasing prevalence with age,[7] which was associated with an increased risk of cardiovascular disease, metabolic disorders, cognitive impairment, arthritis, functional limitations, and lung disease.[8] And elderly patients were more common to experience a decrease in lean body mass due to excessive obesity. Therefore, sarcopenia often coexisted with overweight and obesity in the elderly population.[9] Although the international guideline consensus was to use body mass index (BMI) as a diagnostic indicator of overweight and obesity, BMI could not well interpret the body composition of fat mass and lean body mass in overweight and obese patients, especially the impact of BMI as an evaluation indicator of obesity on sarcopenia was still controversial. Recent studies indicate that overweight and obesity, as defined by BMI, may have a protective effect against sarcopenia in specific populations, though this relationship is still subject to debate.[10] Lu et al also found that overweight or obesity as defined by BMI could prevent sarcopenia.[11] It had also been reported that the increase of BMI was accompanied by the increase of body fat and the decrease of muscle mass, which increased the risk of sarcopenia.[12]

Different obesity parameter indicators were often used in clinical and scientific research to evaluate the risk and prognosis of related diseases. Common obesity indices included BMI, waist circumference (WC), waist to height ratio (WHtR), and body fat rate (%BF). In recent years, new obesity indices had also included visceral adiposity index (VAI) and lipid accumulation product (LAP).[13,14] The emergence of these obesity assessment indicators enriched the role of BMI, which previously relied solely on weight and height and could not distinguish body composition, in the diagnosis and prognosis of diseases. The introduction of abdominal obesity assessment indicators further approached the application of body composition assessment in obesity assessment.[15]

Compared to obesity, abdominal obesity was more prominent in Chinese T2DM patients.[16,17] BMI and other obesity evaluation indicators, which was more closely related to sarcopenia? Therefore, our study chose the special population of postmenopausal women, as their sudden decrease in estrogen levels and redistribution of fat increased the risk of T2DM and sarcopenia.[18,19] The aim of this study was to explore the relationship between different obesity indices and sarcopenia and its parameters in postmenopausal T2DM patients at different BMI levels, in order to seek more meaningful obesity indices to improve the early identification and prevention of sarcopenia in postmenopausal T2DM, a special group prone to sarcopenia.

Materials and Methods

Subjects

By using a retrospective cross-sectional study design, 298 postmenopausal inpatients diagnosed with T2DM in the Department of Endocrinology, the First Hospital of Qinhuangdao of Hebei from September 2019 to June 2023, were selected. The exclusion criteria included the following: 1) acute complications of diabetes mellitus such as diabetic ketoacidosis and hyperosmolar hyperglycemia; 2) acute myocardial infarction; acute cerebrovascular disease; acute inflammation; Gastrointestinal bleeding; Malignant tumor; 3) maintenance hemodialysis; 4) hepatic dysfunction (>3-fold elevation of alanine aminotransferase, aspartate aminotransferase); 5) severe osteoarthropathy or neuromuscular disease; 6) implantation of a pacemaker; 7) inability to understand/perform the exercise tests for this study. This study was approved by the Ethics Committee of the First Hospital of Qinhuangdao (Approval number: No.2020B004) in accordance with the ethical principles of the Declaration of Helsinki. All patients provided written informed consent for participation.

Data Collection

A pre-designed questionnaire was used to collect general data such as age and gender of the subjects, and anthropometric measurements were measured and recorded, including height, weight, calculated BMI (BMI = weight (kg)/height^2 (m2)), WC, and calculated WHtR (WHtR = WC(m)/height(m)). We measured grip strength with the Jamar dynamometer (Performance health supply, inc., Cedarburg, WI, USA). Subjects initially sat in a chair to maintain upper body straight, elbow bending 90 °, with both hands or dominant hand to squeeze the dynamometer for 3 s. Subjects were asked to squeeze twice, taking the maximum as the final figure A commonly used gait speed test is called the 6-m usual walking

speed test (6MWT), with speed measured manually with a stopwatch. Peripheral venous blood samples were taken at 8:00 AM after at least 8-hours of fasting and subjected to biochemical measurements, including triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), uric acid (UA), fasting blood glucose (FBG), and glycated hemoglobin (HbA1c). Body composition including total body fat and total body lean was assessed using dual-energy X-ray absorptiometry (DXA) (MEDILINK SARL., France), and skeletal muscle mass index (SMI) and body fat percentage (%BF) were subsequently calculated. (SMI = the sum of the lean amount of the bilateral upper limbs and the bilateral lower limbs (kg)/height^2 (m^2); %BF = total body fat mass (TFM) (kg)/weight (kg) *100%). The visceral fat index (VAI) and lipid accumulation product (LAP) were calculated using relevant anthropometric and biochemical measurements.[18] Calculation formula of VAI was shown:

$$VAI = \left(\frac{WC(cm)}{36.58+(1.89\times BMI)} \times \frac{TG(mmol/l)}{0.81} \times \frac{1.52}{HDL(mmol/l)}\right) in females.[18]$$

Calculation formula of LAP was shown:

$$\mathrm{LAP} = (WC(cm) - 58) \times TG\,(mmol/l)\, in females.[18]$$

Diagnosis and Groups

The cut-off points of BMI were recommended by the Working Group on Obesity in China.[20] The people were divided into three groups according to BMI: normal group A (18.5 kg/m^2≤BMI < 24 kg/m^2, n=101, 33.9%); overweight group B (24.0 kg/m^2≤BMI < 28 kg/m^2, n=141,47.3%); and obesity group C (28.0 kg/m^2 ≤BMI < 35 kg/m^2, n=56,18.8%). According to the recommended diagnostic algorithm of the Asian Working Group for Sarcopenia 2019 consensus (AWGS 2019).[21] Sarcopenia was defined as low SMI (<5.4 kg/m^2 in females) associated with either low handgrip strength (HGS) (<18 kg in females) or low gait speed (<1.0 m/s). The subjects were divided into sarcopenia group (SP group: n = 100, 33.6%) and non-sarcopenia group (non-SP group, n = 198,66.4%).

Statistical Analysis

Data were analyzed using SPSS (version 25.0 for Windows, SPSS Inc., Chicago, IL, USA). Baseline characteristics of the study participants are presented below. Continuous variables were expressed as mean (SD). Comparisons were conducted from different groups using Two-way ANOVA. We utilized Spearman correlation analysis to investigate the correlation between obesity indices and various parameters of sarcopenia at different BMI categories. Multivariate logistic regression analysis was performed to determine independent risk factors of sarcopenia in postmenopausal women with T2DM at different BMI levels. Statistical significance was established at $p < 0.05$.

Results

Comparison of Baseline Characteristics of Hospitalized Postmenopausal Women with T2DM Between SP Group and Non-SP Group

Compared with the non-SP group, the age of the SP group was significantly higher than that of the non-SP group, and UA, weight, height, BMI, WC, WHtR, LAP, HGS, 6MWT and SMI were lower than those of the non-SP group, with statistical significance (P < 0.05), Table 1.

Comparison of Obesity Indices in Different BMI Categories

From group A to group C, WC, WHtR, LAP, and %BF were gradually increased among the three groups, and the differences were statistically significant (P < 0.05). There was no significant difference in VAI among the three groups (P > 0.05), as shown in Table 2.

Table 1 Comparison of Baseline Characteristics of Hospitalized Postmenopausal Women with T2DM Between SP Group and Non-SP Group

	SP Group (n=100)	Non-SP Group (n=198)	t	P
Age (years)	67.32±7.61	63.23±7.86	−4.284	0.000★
HbA1c (%)	8.90±1.98	8.77±2.05	−0.492	0.623
FPG (mmo/L)	8.67±3.64	8.75±3.36	0.205	0.838
UA (mmo/L)	286.5±82.60	309.±84.47	2.213	0.028★
TG (mmo/L)	1.92±1.04	2.19±1.71	1.441	0.151
TC (mmo/L)	5.26±1.69	5.61±1.32	1.946	0.053
HDL-C (mmo/L)	1.10±0.28	1.13±0.39	0.704	0.482
LDL-C (mmo/L)	2.85±1.15	3.04±0.86	1.618	0.107
Weight (kg)	60.83±7.73	67.13±9.06	5.940	0.000★
Height (m)	1.58±0.05	1.59±0.05	2.073	0.039★
BMI (kg/ m^2)	24.25±2.90	26.31±3.37	5.233	0.000★
WC (cm)	88.37±8.40	91.46±8.66	2.940	0.004★
WHtR (%)	55.86±5.64	57.33±5.81	2.084	0.038★
%BF (%)	54.53±7.09	54.40±6.22	−0.162	0.872
VAI	3.83±2.69	4.25±4.31	0.896	0.371
LAP	59.49±37.18	73.9±61.42	2.158	0.032★
HGS (kg)	16.64±4.74	20.87±5.40	6.644	0.000★
6MWT (m/s)	0.87±0.15	1.00±0.21	5.840	0.000★

Notes: Values are expressed as means ± SD. SP group (sarcopenia group, n = 100) and non-SP group (non- sarcopenia group, n = 198).

Abbreviations: SP group, sarcopenia group; non-SP group, non- sarcopenia group; HbA1c, glycated hemoglobin; FPG, fasting plasma glucose; UA, uric acid; TG, triglycerides; TC, total cholesterol; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; BMI, body mass index; WC, waist circumference; WHtR, waist to height ratio; %BF, body fat rate; VAI, visceral adiposity index; LAP, lipid accumulation product; HGS, handgrip strength; 6MWT, the 6-m usual walking speed test. SD, standard deviation. ★P <0.05.

Table 2 Comparison of Obesity Indices in Different BMI Categories

	Group A (n=101)	Group B (n=141)	Group C (n=56)	F	p
WC (cm)	84.35±7.07◆★	90.83±6.04★	100.36±7.45	104.058	0.000
WHtR (%)	52.96±4.38◆★	56.98±4.28★	63.47±5.18	98.647	0.000
%BF (%)	51.32±6.42◆★	54.82±5.24★	59.14±6.60	31.909	0.000
VAI	3.44±2.82	4.44±4.74	4.49±2.59	2.362	0.096
LAP	47.45±33.85◆★	74.63±64.12★	94.01±46.13	15.833	0.000

Notes: Values are expressed as means ± SD. Group A: (normal; n=101); Group B: (overweight; n=141); Group C: (obesity; n=56). ◆compared with group B; ★compared with group C.

Abbreviations: WC, waist circumference; WHtR, waist to height ratio; %BF, body fat rate; VAI, visceral adiposity index; LAP, lipid accumulation product. SD, standard deviation.

Spearman Correlation Analysis of Sarcopenia Parameters and Obesity Indices in Different BMI Categories

In the whole sample, SMI was positively correlated with BMI, WC, WHtR, VAI and LAP, the difference was statistically significant (P < 0.05). There was no significant correlation between HGS and obesity indices (P > 0.05). The 6MWT was negatively correlated with WC and WHtR, and the difference was statistically significant (P < 0.05), Table 3.

In normal group A, SMI was positively correlated with BMI and negatively correlated with %BF, the difference was statistically significant (P < 0.05). There was no significant correlation between HGS and obesity indices (P > 0.05). The 6MWT was negatively correlated with WC and WHtR, and the difference was statistically significant (P < 0.05), Table 3.

Table 3 Spearman Correlation Analysis of Sarcopenia Parameters and Obesity Indices in Different BMI Categories

		BMI (kg/ m^2)		WC (cm)		WHtR (%)		VAI		LAP		%BF (%)	
		r	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*	*r*	*p*
The total sample	SMI	0.470	0.000	0.294	0.000	0.235	0.000	0.121	0.037	0.201	0.000	−0.103	0.076
	HGS	0.090	0.122	0.049	0.403	−0.050	0.385	−0.017	0.766	0.016	0.780	0.029	0.624
	6MWT	−0.095	0.101	−0.203	0.000	−0.250	0.000	0.024	0.678	−0.023	0.696	0.011	0.850
Group A	SMI	0.387	0.000	0.090	0.370	0.075	0.457	−0.028	0.785	−0.005	0.956	−0.298	0.002
	HGS	0.151	0.132	0.078	0.440	0.008	0.940	−0.152	0.129	−0.072	0.476	0.057	0.570
	6MWT	0.034	0.739	−0.347	0.000	−0.349	0.000	−0.066	0.510	−0.185	0.064	0.049	0.630
Group B	SMI	0.193	0.022	0.006	0.943	−0.112	0.188	0.197	0.019	0.180	0.033	−0.377	0.000
	HGS	0.166	0.049	0.047	0.577	−0.104	0.221	0.067	0.432	0.080	0.349	−0.023	0.785
	6MWT	0.087	0.305	−0.069	0.416	−0.148	0.080	0.079	0.352	0.087	0.307	0.073	0.390
Group C	SMI	0.115	0.397	−0.052	0.704	−0.135	0.322	−0.089	0.513	−0.067	0.622	−0.382	0.004
	HGS	−0.186	0.169	−0.244	0.070	−0.398	0.002	−0.171	0.207	−0.198	0.143	−0.079	0.565
	6MWT	−0.239	0.076	−0.205	0.130	−0.336	0.011	0.016	0.906	−0.050	0.717	0.015	0.910

Notes: the total sample: (n=298); Group A: (normal; n=101); Group B: (overweight; n=141); Group C: (obesity; n=56).
Abbreviations: BMI, body mass index; WC, waist circumference; WHtR, waist to height ratio; VAI, visceral adiposity index; LAP, lipid accumulation product; %BF, body fat rate; SMI, skeletal muscle mass index; HGS, handgrip strength; 6MWT, the 6-m usual walking speed test.

In the overweight group B, SMI was weak positively correlated with BMI, VAI and LAP, and negatively correlated with %BF, with statistical significance ($P < 0.05$). There was no correlation between HGS and 6MWT and obesity indices, with no statistical significance ($P > 0.05$), Table 3.

In the obesity group C, SMI was negatively correlated with %BF; WHtR was negatively correlated with HGS and 6MWT, and the difference was statistically significant ($P < 0.05$), as shown in Table 3.

Logistic Regression Analysis of Sarcopenia Risk Factors Across BMI Categories

In the overall study sample, whether it was sarcopenia (non-SP=0, SP=1) was the dependent variable, and age, BMI, WC, WHtR, %BF, LAP, and UA were the independent variables. Logistic regression analysis showed that: Age (OR=1.065, 95% CI 1.026–1.104, P=0.001), WHtR (OR=1.166, 95% CI 1.002–1.358, P=0.047), %BF (OR=1.060, 95% CI 1.011–1.113, P=0.017) was risk factors for sarcopenia. For every 1 unit increase in BMI, the risk of sarcopenia decreased by 26.4% (OR=0.736, 95% CI 0.635–0.853, P=0.000). Logistic analysis showed that each 1-unit increase in BMI decreased the risk of sarcopenia by 63.2% (OR=0.368, 95% CI 0.215–0.629, P=0.000) in group A. Age (OR=1.077, 95% CI 1.015–1.144, P=0.015) and %BF (OR=1.094, 95% CI 1.010–1.186, P=0.028) increased the risk of sarcopenia by 1.077 and 1.094 times, respectively, in group B. While each 1-unit increase in BMI, the risk of sarcopenia decreased by 35% (OR=0.650, 95% CI 0.430–0.983, P=0.041) in group B. %BF (OR=1.459, 95% CI 1.093–1.949, P=0.010) increased the risk factors of sarcopenia by 1.459 times in group C, as shown in Table 4.

Discussion

T2DM and sarcopenia were both age-related diseases.[22] Skeletal muscle was not only an athletic organ but also an endocrine and metabolic organ, which was responsible for about 80% of postpranpranal glucose utilization and played an important role in the regulation of glucose metabolism.[23] Sarcopenia had a complex etiology, the current etiological mechanisms of sarcopenia mainly focused on low grade chronic inflammation, insulin and anabolic resistance, mitochondrial dysfunction, oxidative stress, hormonal changes, malnutrition, inactivity, and chronic diseases.[24] The occurrence of sarcopenia was not only a part of aging but also strongly associated with obesity, insulin resistance, and T2DM.[25]

Postmenopausal women, a special population, were selected as the research object in our study, which was characterized by a sudden drop in estrogen levels. And estrogen plays a crucial role in human physiology, including

Table 4 Logistic Regression Analysis of Sarcopenia Risk Factors Across BMI Categories

		B	OR	95% CI	P
The total sample (n=298)	Age (years)	0.063	1.065	1.026–1.104	0.001
	BMI (kg/ m^2)	−0.306	0.736	0.635–0.853	0.000
	WHtR (%)	0.154	1.166	1.002–1.358	0.047
	%BF (%)	0.059	1.060	1.011–1.113	0.017
Group A (n=101)	BMI (kg/ m^2)	−1.001	0.368	0.215–0.629	0.000
Group B (n=141)	Age (years)	0.074	1.077	1.015–1.144	0.015
	BMI (kg/ m^2)	−0.431	0.650	0.430–0.983	0.041
	%BF (%)	0.090	1.094	1.010–1.186	0.028
Group C (n=56)	%BF (%)	0.378	1.459	1.093–1.949	0.010

Notes: The dependent variable: sarcopenia (non-SP =0, SP =1).The independent variables: age, BMI, WC, WHtR, %BF, LAP and UA.
Abbreviations: BMI, body mass index; WC, waist circumference; WHtR, waist to height ratio; %BF, body fat rate; LAP, lipid accumulation product; UA, uric acid.

glucose and lipid metabolism, bone metabolism, reproductive function and nervous function.[8] The decrease of circulating estrogen level directly or indirectly affects skeletal muscle mass and function, and accelerates the occurrence of sarcopenia.[26,27] At the same time, with the increase of age, the distribution of body fat also changes, and visceral obesity is obvious, and the proportion of muscle decreases year by year. Previous studies had shown that over the age of 50, leg muscle mass and strength declined by 1%–2% and 1.5–5% per year, respectively.[28] In our study of postmenopausal hospitalized T2DM patients, we found that the detection rate of sarcopenia was 33.6%, slightly higher than the prevalence of sarcopenia in T2DM patients reported in previous studies of 30.06%,[29] and significantly higher than the prevalence of sarcopenia in Chinese community population of 19.6%.[30] The reason was that the special population in our study had diabetes and postmenopausal estrogen decline at the same time, which affected fat distribution, lipid metabolism disorder and insulin's effect on anabolism.

Sarcopenic obesity has also been a hot topic in recent years. Previous studies had shown that the increase of BMI could effectively reduce the incidence of sarcopenia.[30] Subjects with a high BMI tended to have more lean body mass, regardless of body fat. A Chinese cohort study found a protective effect of high BMI against sarcopenia in elderly people after a 4-year follow-up.[31] Li conducted aerobic and strength training for 12 weeks on elderly people with sarcopenia obesity, which significantly reduced body fat, increased muscle mass, and improved physical function,[32] indicating that increased BMI and increased skeletal muscle mass might be more favorable factors for maintaining and improving physical function and reducing the risk of sarcopenia in elderly people.

However, it had been suggested that although BMI has been the most important indicator of evaluating obesity, it was the sum of fat mass and lean body mass, and might not be the best indicator of evaluating the relationship between obesity and sarcopenia.[9] Ectopic deposition of fat played a key role in the development of sarcopenia as well as a key factor in the poor prognosis of obesity.[33] A previous study in Australia found that the increase of BMI reflected a substantial increase in body fat mass and a decline in lean body mass, which may have adverse implications for future development of sarcopenia.[12] Cheng Li et al found that high visceral fat area could weaken the negative correlation between high BMI and sarcopenia.[34]

Our study circumvented the inconsistency between BMI and lean body mass by stratification of BMI. This study found that the BMI and abdominal obesity evaluation indicators WC and WHtR of postmenopausal T2DM patients with sarcopenia were significantly lower than those of non-sarcopenia patients, while there was no difference in %BF, suggested that postmenopausal T2DM patients with sarcopenia had a relatively higher %BF regardless of whether they were overweight and obese. Spearman correlation analysis showed that the negative correlation between SMI and % BF gradually increased with the increase of BMI, and there was no significant relationship between BMI and SMI in

obese people. It can be seen that obesity defined by BMI does not mean that the larger the BMI, the larger the SMI. There was no significant correlation between grip strength and obesity indices in normal weight and overweight people. While in obese people, grip strength and gait speed, which represent muscle function indicators, were significantly negatively correlated with abdominal obesity indices WHtR. At the same time, our study also found that VAT and LAP, which represent the new obesity indices of visceral fat, had a weak positive correlation with SMI in overweight. A similar positive correlation between increased visceral fat and sarcopenia had also been reported.[34,35] The possible reason was that adipose tissue was the main site of sex hormone storage and metabolism, and female abdominal fat stored high levels of sex hormones, which had a positive impact on skeletal muscle mass.[36] Therefore, different obesity phenotypes might have different effects on muscle mass and muscle function.

Logistic analysis of our study further confirmed that as BMI increased in postmenopausal T2DM patients, the protective effect of BMI on sarcopenia diminished, particularly in obese individuals, suggesting that the protective effect of BMI on the occurrence of sarcopenia appears within a certain range. Notably, %BF emerged as a significant risk factor for sarcopenia. Our study found that with the increase of BMI, especially in overweight and obese patients, %BF increased the risk of sarcopenia by 1.094 and 1.459 times, respectively. The possible mechanism was that in the normal progression of obesity, high BMI was usually accompanied by an increase in fat mass. In particular, the accumulation of visceral fat could cause systemic inflammatory response and insulin resistance, and then lead to skeletal muscle dysfunction and sarcopenia.[37] The adipose tissue of obese individuals had high levels of tumor necrosis factor-α (TNF-α), which promoted the production and secretion of several cellular inflammatory factors.[38] From the perspective of inflammation theory, fat tissue released large amounts of Interleukin-6 (IL-6) and TNF-α, leading to low-level chronic inflammation, reducing insulin sensitivity, impacting protein synthesis ability, promoting hydrolytic metabolic pathways, and ultimately damaging muscle function. The amount of fat increases with age and could gradually penetrate into skeletal muscle, resulting in changes in muscle fiber structure and contractile performance, resulting in loss of skeletal muscle mass, strength, and function.[39]

The present study also had some limitations. First, this study was a cross-sectional retrospective study, which precluded the establishment of causal relationships between events. Second, this study was a single-center study with a small sample size. Meanwhile, this study was limited to hospitalized postmenopausal T2DM patients, and no healthy people were collected as the control group. Therefore, the results need to be verified in multiple centers.

Conclusion

In postmenopausal women with T2DM, BMI exhibited a protective effect against sarcopenia within a specific range, although this protective effect diminishes as BMI increases, especially in the presence of high body fat percentage. However, the increasing %BF in overweight and obese individuals highlights the importance of comprehensive body composition assessments in early sarcopenia detection and prevention.

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Disclosure

The authors state that there are no conflict of interest in the publication of this article.

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