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Type 2 diabetes: is obesity for diabetic retinopathy good or bad? A cross-sectional study

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Abstract

Background The relationship between obesity and diabetic retinopathy (DR) remains controversial, and the relationship between sarcopenic obesity and DR is still unclear. The purpose of this study is to investigate the relationship between obesity, sarcopenic obesity, and DR in patients with type 2 diabetes mellitus (T2DM).

Methods A cross-sectional study was conducted on patients with T2DM. Obesity was assessed by body mass index (BMI), fat mass index (FMI), android fat mass, gynoid fat mass, and visceral adipose tissue (VAT) mass. Sarcopenia was defined according to the criteria of Consensus of the Asian Working Group for Sarcopenia (AWGS 2019). Sarcopenic obesity was defined as the coexistence of sarcopenia and obesity. The association between obesity, sarcopenic obesity, and DR was examined using univariable and multivariable logistic regression models.

Results A total of 367 patients with T2DM (mean age 58.3 years; 57.6% male) were involved in this study. The prevalence of DR was 28.3%. In total patients, significant adverse relationships between obesity and DR were observed when obesity was assessed by BMI (adjusted odds ratio [aOR] 0.54, 95% confidence interval [CI] 0.31 to 0.96, $p=0.036$), FMI (aOR 0.49, 95% CI 0.28 to 0.85, $p=0.012$), android fat mass (aOR 0.51, 95% CI 0.29 to 0.89, $p=0.019$), gynoid fat mass (aOR 0.52, 95% CI 0.30 to 0.91, $p=0.021$) or VAT mass (aOR 0.45, 95% CI 0.25 to 0.78, $p=0.005$). In patients with T2DM and obesity, the prevalence of sarcopenic obesity was 14.8% ($n=23$) when obesity was assessed by BMI, 30.6% ($n=56$) when assessed by FMI, 27.9% ($n=51$) when assessed by android fat mass, 28.4% ($n=52$) when assessed by gynoid fat mass, and 30.6% ($n=56$) when assessed by VAT mass. Sarcopenic obesity was associated with DR when obesity was assessed by BMI (aOR 2.61, 95% CI 1.07 to 6.37, $p=0.035$), android fat mass (aOR 3.27, 95% CI 1.37 to 7.80, $p=0.007$), or VAT mass (aOR 2.50, 95% CI 1.06 to 5.92, $p=0.037$).

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Conclusions Patients with T2DM showed a substantial inverse relationship between DR and obesity, and sarcopenic obesity was considerably favorably associated with DR. Detection of sarcopenia in patients with T2DM, especially in obese T2DM, is essential to guide clinical intervention in DR.

Keywords Obesity, Sarcopenia, Sarcopenic obesity, Diabetic retinopathy, Type 2 diabetes mellitus

Background

Diabetic retinopathy (DR) is a common microvascular complication in patients with diabetes. In people aged 20–74, DR is the primary cause of preventable blindness and visual loss [1], which has a substantial negative impact on the quality of life for diabetic patients. Therefore, it is imperative to carry out research to find modifiable risk factors for DR, which will inform clinical practice and help prevent the development and progression of DR.

Several prior studies have indicated that a higher body mass index (BMI) is associated with a higher risk of DR [2,3], although contrasting findings have suggested a potential protective influence [4–6]. Furthermore, the association between markers of central or abdominal obesity, such as waist circumference (WC), and DR exhibited similar inconsistencies [4,7,8]. The association between obesity and DR has not been consistently established. It appears that anthropometric indicators, such as BMI and WC, inadequately capture the relationship between obesity and DR well. Several previous studies have utilized regional adiposity indicators, assessed through dual-energy X-ray absorptiometry (DXA), to investigate the association between obesity and cardiometabolic risk. These studies found that lower body fat (gynoid and leg fat) was associated with lower risk factors, while upper body fat (android and visceral fat) was associated with higher risk factors [9–11]. Therefore, this study aims to evaluate obesity by integrating BMI and DXA-measured adiposity metrics, including total obesity indicators (BMI, fat mass index [FMI]), and regional adiposity indicators (android fat mass, gynoid fat mass, and visceral adipose tissue [VAT] fat mass), to investigate the correlation between DR and obesity.

Recent studies have demonstrated that sarcopenic obesity (SO), defined as a functional and clinical disorder characterized by a loss of skeletal muscle mass and function coexisting with an excess of adipose tissue [12], represents a specific obesity phenotype. The combination of sarcopenia and obesity may pose greater health risks, including disability, metabolic impairments, and mortality, compared to either condition alone [13,14]. Previous research has indicated that sarcopenic obesity has been linked in the past to an increased risk of cardiovascular disease (CVD) and chronic kidney disease (CKD) in individuals with type 2 diabetes mellitus (T2DM) [15,16]. A previous study has established an independent association between sarcopenia with DR in patients with T2DM [17]. It's unknown if sarcopenic obesity raises the risk of developing DR. As such, the present study explores further the potential for a higher correlation between DR and sarcopenia in obese T2DM.

Materials and methods

Subjects

This cross-sectional study included patients with T2DM (age > 18 years) who were admitted to the Department of Endocrinology at Longyan First Affiliated Hospital of Fujian Medical University in Fujian, China, between December 2021 and December 2022. The T2DM was defined according to the criteria set by the American Diabetes Association (ADA). Patients were eligible if they had undergone a fundus examination and a DXA scan during the study period. Participants were excluded if they met the following criteria: (1) diagnosis of other types of diabetes; (2) presence of acute complications such as diabetic ketoacidosis, hyperosmolar status, or acute infection; (3) presence of severe infection; (4) presence of liver cirrhosis, end-stage kidney disease, undergoing renal replacement therapy, or post-renal transplantation; (5) pregnancy or lactation; (6) presence of malignant tumor or life expectancy of less than 1 year. Finally, 367 patients were enrolled. This study adhered to the principles outlined in the Declaration of Helsinki and has received approval from the Institutional Ethics Research Committee of Longyan First Affiliated Hospital of Fujian Medical University. Informed consent was obtained from all participants enrolled in the study.

Data collection and clinical definition

Trained interviewers utilized standardized questionnaires to collect demographic information and health data, including age, gender, smoking and drinking habits, medication use, and history of diagnosed diseases. The smoking status was categorized as either "no current smoker" or "current smoker". The classification of drinking was based on the categories of "no current drinking" or "current drinking". Self-reported health conditions comprised physician-diagnosed hypertension, CVD, and dyslipidemia. Clinical characteristics and biochemical test results, including Hemoglobin A1C (HbA1c), serum creatinine (SCr), and uric acid (UA), were retrieved from the electronic medical record system. Upon admission, healthcare professionals conducted assessments of the patient's height, weight, and blood pressure (BP) using standardized documentation forms. Venous blood was

obtained in the early morning following an overnight fast.

The estimated glomerular filtration rate (eGFR) was calculated based on creatinine using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) 2009 equation. Diabetic nephropathy (DN) was diagnosed based on the urinary albumin/creatinine ratio (ACR) ≥ 30 mg/mmol or eGFR < 60 mL/min/1.73m^2 according to the Kidney Disease Improving Global Outcomes (KDIGO) organization [18,19]. CVD was defined as stroke and/or coronary artery disease (CAD).

Assessment of DR

The diagnosis of DR was established using a non-mydriatic fundus camera (Optos Daytona p200T) or fundus fluorescein angiography (FFA) conducted by qualified ophthalmologists. The diagnosis of DR was established by identifying one or more clinical features in the fundus, such as microaneurysms, hemorrhage, hard exudates, cotton wool spots, venous dilation and beading, intraretinal microvascular abnormalities, retinal neovascularization, fibrous hyperplasia, and photocoagulation scar [20]. Patients without DR were characterized as exhibiting no abnormalities in fundus photographs.

Definition of sarcopenia

The patient was positioned in the supine posture. The Skeletal Muscle Index (SMI) was assessed using the Hologic Discovery Wi dual-energy X-ray bone densitometer (Hologic Discovery QDR Series) by certified radiological technologists. The SMI was computed by dividing the appendicular skeletal muscle mass (ASM) by the square of the height (kg/m^2). The thresholds for low muscle mass were defined as an SMI of less than 7.0 kg/m^2 in men and less than 5.4 kg/m^2 in women [21].

Trained physicians conducted the handgrip strength measurement and 6-meter walk test. Handgrip strength was assessed with the Camry electronic grip strength meter (model EH101; Camry Scale). The patient was positioned either standing or sitting with the elbow fully extended. A maximal isometric contraction was then executed using the dominant hand, and at least two measurements were obtained, with the highest value being documented [21]. Low muscle strength was defined as a handgrip strength of less than 28.0 kg for men and less than 18.0 kg for women. The 6-meter walk was assessed by measuring the time taken to cover the distance at a normal speed without slowing down, and the average result of at least two trials was recorded. Low physical performance was characterized by a walking speed of less than 1.0 m/s over a distance of 6 m [21].

Sarcopenia was defined as having low muscle mass combined with either low muscle strength or low physical performance, according to the Consensus of the Asian Working Group for Sarcopenia (AWGS 2019) [21].

Definition of obesity and sarcopenic obesity

The BMI was calculated by dividing the body weight by the square of the height (kg/m^2). Fat indicators, such as FMI, android fat mass, gynoid fat mass, and VAT mass, were evaluated using DXA according to previously established protocols. The FMI was calculated by dividing the total fat mass (FM) by the square of the height (kg/m^2). The android region is approximately defined as the area around the waist, spanning from the mid-point of the lumbar spine to the top of the pelvis, while the gynoid region is situated between the head of the femur and the mid-thigh [22]. The estimation of VAT fat was derived from the measurements of VAT mass and volume in the android region [23].

To assess the correlation between various forms of obesity and diabetic retinopathy, our study employed five distinct definitions of obesity: a. BMI ≥ 25 kg/m^2 [24]; b. FMI exceeding the sex-specific median in the study (> 7.32 kg/m^2 for men, > 8.83 kg/m^2 for women); c. android fat mass exceeding the sex-specific median in the study (> 1.96 kg for men, > 1.77 kg for women); d. gynoid fat mass exceeding the sex-specific median in the study (> 2.68 kg for men, > 2.99 kg for women); e. VAT mass exceeding the sex-specific median in the study (> 0.67 kg for men, > 0.59 kg for women).

Further subgroup analysis was conducted in obese T2DM. Sarcopenic obesity was defined as the coexistence of sarcopenia and obesity, while simple obesity was defined as the absence of sarcopenia.

Statistical analysis

Continuous variables were presented as means ± standard deviation (SD) or as the median and interquartile range based on the distribution of the data. Categorical variables were displayed as either frequencies or percentages. For group comparisons, the student's t-test or Wilcoxon rank-sum test was employed for continuous variables, while the $\chi 2$ test was utilized for categorical variables.

Among all the participants, univariable and multivariable logistic regression models were employed to evaluate the correlation between obesity and DR. Obesity-related indicators were incorporated into the model as continuous and categorical variables, respectively. Logistic regression analysis was also employed to evaluate the association between sarcopenia and DR in total patients. Furthermore, among patients with obesity, univariable and multivariable logistic regression models were employed to assess the association between sarcopenic obesity and DR. Obesity-related indicators were incorporated into the model as categorical variables in this step.

Statistical analysis was conducted using the SPSS V.25.0 statistical software package. The data were summarized using odds ratios or regression coefficients with a 95% confidence interval (CI). All statistical tests were conducted as two-tailed, and findings with a significance level of $p<0.05$ were seemed to be statistically significant.

Results

Characteristics of study participants

The study included 367 patients diagnosed with type 2 diabetes, with an average age of 58.3 years, and 57.6% being male. The median duration of diabetes was 6 years, and the HbA1c level was 9.7% ± 2.3. Ninety-seven individuals (26.4%) were identified as current smokers. Nearly half of the patients presented with hypertension (44.6%; $n=171$), 11.2% ($n=41$) exhibited CVD, and 54.8% ($n=201$) were diagnosed with dyslipidemia (Table 1). The prevalence of DR was 28.3% ($n=104$), and the prevalence of sarcopenia was 31.1% ($n=114$). As shown in Table 1, patients with DR were older and had a longer duration of diabetes, higher systolic blood pressure, elevated levels of UA, and lower eGFR compared to patients without DR. Additionally, they exhibited a higher incidence of hypertension, DN, and CVD compared to patients without DR. The use of insulin and sodium-glucose cotransporter-2 (SGLT2) inhibitors was higher in patients with DR compared to those without DR. The grading of the diagnosed DR of all participants. were presented in Additional file 1: Tables S1.

Associations between obesity and DR

Patients with DR exhibited a lower gynoid fat mass compared to those without DR ($p=0.017$). A similar trend was observed for BMI, FMI, android fat mass, and VAT mass, although the difference was not statistically significant. The prevalence of obesity, as classified by gynoid fat mass or FMI, was lower in patients with DR compared to non-DR patients ($p=0.040$, $p=0.022$, respectively) (Table 1). The prevalence of DR was found to be lower in patients with obesity compared to those without obesity, as illustrated in Fig. 1.

In univariable models, a statistically significant negative correlation was found between gynoid fat mass as a continuous variable and DR (OR 0.76, 95% CI 0.60 to 0.97, $p=0.028$). Similar inverse findings were observed in the association between obesity, as indicated by higher gynoid fat mass or higher FMI, and DR. The ORs were 0.62 (95% CI 0.39 to 0.98, $p=0.041$) and 0.59 (95% CI 0.37 to 0.93, $p=0.023$), as shown in Table 2. In multivariable models, the continuous variables BMI (adjusted odds ratio [aOR] 0.92, 95% CI 0.84 to 0.99, $p=0.043$), FMI (aOR 0.88, 95% CI 0.79 to 0.99, $p=0.027$), and gynoid fat mass (aOR 0.68, 95% CI 0.50 to 0.92, $p=0.012$) showed a significant inverse association with DR. When assessing obesity using BMI (aOR 0.54, 95% CI 0.31 to 0.96, $p=0.036$), FMI (aOR 0.49, 95% CI 0.28 to 0.85, $p=0.012$), android fat mass (aOR 0.51, 95% CI 0.29 to 0.89, $p=0.019$), gynoid fat mass (aOR 0.52, 95% CI 0.30 to 0.91, $p=0.021$) or VAT mass (aOR 0.45, 95% CI 0.25 to 0.78, $p=0.005$), a significant inverse association was found between obesity and DR (Table 2). Variables adjusted for include age, duration of diabetes, presence of hypertension, dyslipidemia, history of CVD, eGFR, insulin usage, angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs), and SGLT2 inhibitors use.

Associations between sarcopenia and DR

Patients with DR showed decreased handgrip strength. The prevalence of sarcopenia in patients with DR was higher than in those without DR (42.3% vs. 26.6%, $p=0.003$) (Table 1). Table 3 shows that individuals with sarcopenia showed an increased risk of DR (OR 2.02, 95% CI 1.26 to 3.25, $p=0.004$) in a univariate model. After adjusting for various covariates, including age, duration of diabetes, hypertension, dyslipidemia, history of CVD, eGFR, insulin usage, ACEI/ARBs usage, and SGLT2 inhibitors usage, the association remained statistically significant (OR 2.31, 95% CI 1.23 to 4.33, $p=0.009$).

Associations between sarcopenic obesity and DR

Subsequent subgroup analysis was conducted among individuals with obesity. The baseline characteristics of patients with simple obesity and sarcopenic obesity, classified according to each obesity indicator, were presented in Additional file 1: Tables S2–S6. The prevalence of sarcopenic obesity was 14.8% ($n=23$) when obesity was assessed by BMI, 30.6% ($n=56$) when assessed by FMI, 27.9% ($n=51$) when assessed by android fat mass, 28.4% ($n=52$) when assessed by gynoid fat mass, and 30.6% ($n=56$) when assessed by VAT mass. The prevalence of DR was higher in patients with sarcopenic obesity compared to those with simple obesity, regardless of the obesity indicator used to assess obesity (Fig. 2).

Sarcopenic obesity showed a significant positive association with DR in univariable logistic models, regardless of the obesity indicator used to define obesity (Table 3). When assessing obesity using BMI (aOR 4.60, 95% CI 1.57 to 13.48, $p=0.005$), android fat mass (aOR 3.27, 95% CI 1.37 to 7.80, $p=0.007$), and VAT mass (aOR 2.50, 95% CI 1.06 to 5.92, $p=0.037$), sarcopenic obesity consistently and significantly associated with DR even after adjusting for variables such as age, duration of diabetes, hypertension, and eGFR (Table 3).

Table 1 Baseline characteristics of all participants

	Total	No DR(*n* = 263)	DR(*n* = 104)	*p*-value
Demographic characteristics				
Age (years)	58.3 ± 12.4	57.3 ± 13.1	60.8 ± 10.1	0.007
Male (%)	211 (57.6)	153 (58.2)	58 (55.8)	0.674
Duration of diabetes (year)	6.0 (1.0, 10.0)	5.0 (0.4, 10.0)	10.0 (5.0, 17.0)	< 0.001
Smoking, n (%)	97 (26.4)	75 (28.5)	22 (21.2)	0.149
Drinking, n (%)	28 (7.6)	19 (7.2)	9 (8.7)	0.642
SBP (mmHg)	133 ± 18	132 ± 17	137 ± 19	0.007
DBP (mmHg)	82 ± 16	82 ± 12	83 ± 12	0.698
Clinical condition				
Hypertension, n (%)	171 (46.6)	108 (41.1)	63 (60.6)	0.001
CVD, n (%)	41 (11.2)	24 (9.1)	17 (16.3)	0.048
DN, n (%)	46 (12.5)	21 (8.0)	25 (24.0)	< 0.001
Dyslipidemia, n (%)	201 (54.8)	150 (57.0)	51 (49.0)	0.165
Laboratory examination				
HbA1c (%)	9.8 ± 2.3	9.7 ± 2.3	10.1 ± 2.3	0.154
SCr (umol/L)	67 (55, 84)	67 (54, 80)	71 (58, 97)	0.006
eGFR (ml/min/1.73m^2)	91.51 ± 23.30	94.68 ± 22.07	83.58 ± 24.50	< 0.001
UA (umol/L)	340 ± 96	331 ± 89	363 ± 110	0.003
TG (mmol/L)	1.68 (1.08,2.57)	1.64 (1.08,2.59)	1.60 (1.00,2.28)	0.752
TC (mmol/L)	5.10 ± 1.42	5.09 ± 1.48	5.13 ± 1.26	0.790
LDL-C (mmol/L)	3.18 ± 0.98	3.17 ± 0.99	3.19 ± 0.94	0.860
HDL-C (mmol/L)	1.12 ± 0.38	1.10 ± 0.42	1.17 ± 0.28	0.139
Medical history				
Insulin, n (%)	49 (13.4)	27 (10.3)	22 (21.2)	0.006
Sulfonylureas, n (%)	54 (14.7)	38 (14.6)	16 (29.6)	0.742
Glinides, n (%)	121 (33.0)	83 (31.9)	38 (38.0)	0.274
Alpha-GIs, n (%)	172 (46.9)	119 (45.8)	53 (53.0)	0.219
Metformin, n (%)	36 (9.8)	26 (10.0)	10 (10.0)	0.898
TZDs, n (%)	43 (11.7)	35 (13.5)	8 (8.0)	0.203
GLP1-RAs, n (%)	35 (9.5)	24 (9.2)	11 (11.0)	0.691
SGLT-2 inhibitors, n (%)	32 (8.7)	14 (5.4)	18 (18.0)	0.001
DPP-4 inhibitors, n (%)	17 (4.6)	10 (3.8)	7 (7.0)	0.265
ACEI/ARBs, n (%)	87 (23.7)	56 (22.7)	31 (32.6)	0.058
Sarcopenia related indicators				
SMI (kg/m^2)	6.11 (5.43,7.23)	6.24 (5.44,7.30)	5.93 (5.48,6.93)	0.196
low muscle mass	177 (48.2)	123 (46.8)	54 (51.9)	0.373
handgrip (kg)	27.7 ± 10.3	28.8 ± 10.8	24.9 ± 8.5	< 0.001
low muscle strength	127 (37)	79 (32.4)	48 (48.5)	0.005
6-metre walk (m/s)	1.03 ± 0.20	1.04 ± 0.20	1.01 ± 0.19	0.418
low physical performance	63 (36.6)	44 (34.6)	19 (42.2)	0.365
Sarcopenia	114 (31.1)	70 (26.6)	44 (42.3)	0.003
Obesity-related indicators				
BMI (kg/m^2)	24.60 ± 3.55	24.77 ± 3.65	24.16 ± 3.24	0.140
BMI ≥ 25 kg/m^2, n (%)	155 (42.2)	116 (44.1)	39 (37.5)	0.248
FMI (kg/m^2)	8.12 ± 2.56	8.23 ± 2.62	7.82 ± 2.41	0.164
High FMI, n (%)	183 (49.9)	141 (53.6)	42 (40.4)	0.022
Android fat mass (kg)	2.00 ± 0.82	2.02 ± 0.81	1.93 ± 0.85	0.312
High Android fat mass, n (%)	183 (49.9)	138 (52.5)	45 (43.3)	0.112
Gynoid fat mass (kg)	2.90 ± 0.97	2.97 ± 1.01	2.73 ± 0.84	0.017
High Gynoid fat mass, n (%)	183 (49.9)	140 (53.2)	43 (41.3)	0.040

Table 1 (continued)

	Total	No DR($n=263$)	DR($n=104$)	*p*-value
VAT mass (kg)	0.67 ± 0.28	0.68 ± 0.28	0.66 ± 0.29	0.524
High VAT mass, n (%)	183 (49.9)	138 (52.5)	45 (43.3)	0.112

Abbreviations DR: diabetic retinopathy; SBP: systolic blood pressure; DBP: diastolic blood pressure; CVD: cardiovascular disease; DN: diabetic nephropathy; HbA1c: Hemoglobin A1C; SCr: serum creatinine; eGFR: estimated glomerular filtration rate; UA: uric acid; TG: triglyceride; TC: total cholesterol; LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol; TZDs: thiazolidinediones; GLP1-RA, glucagon-like peptide-1 receptors agonist; SGLT-2: sodium-glucose cotransporter-2; DPP-4: Dipeptidyl peptidase-4; ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin receptor blocker; SMI: skeletal muscle index; BMI: body mass index; FMI: fat mass index; VAT: visceral adipose tissue

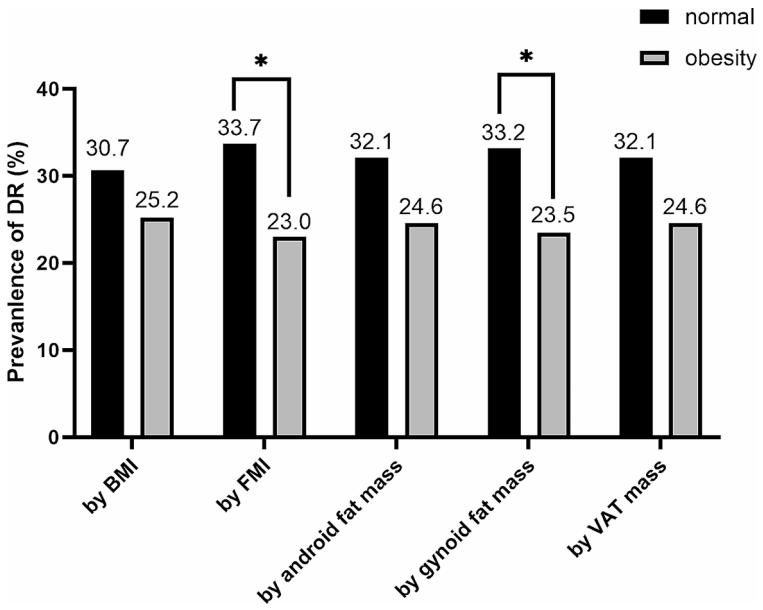

Fig. 1 Prevalence of DR in patients with T2DM in normal versus obese status. *Note* by BMI means obesity was defined as BMI ≥ 25 kg/m^2; by FMI/by android fat mass/by gynoid fat mass/by VAT mass means obesity was defined as FMI/android fat mass/gynoid fat mass/VAT mass was separately higher than the sex-specific median; *means p-value<0.05; *Abbreviations* DR: diabetic retinopathy; T2DM: type 2 diabetes mellitus; BMI: body mass index; FMI: fat mass index; VAT: visceral adipose tissue

Table 2 Associations between obesity, sarcopenia, and DR in patients with T2DM

	Univariable		Multivariable	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
BMI (kg/m^2)	0.95(0.89–1.02)	0.141	0.92(0.84–0.99)	0.043
BMI ≥ 25 kg/m^2	0.76(0.48–1.21)	0.249	0.54(0.31–0.96)	0.036
FMI (kg/m^2)	0.94(0.86–1.03)	0.164	0.88(0.79–0.98)	0.027
High FMI	0.59(0.37–0.93)	0.023	0.49(0.28–0.85)	0.012
Android fat mass (kg)	0.69(0.44–1.09)	0.312	0.73(0.52–1.02)	0.067
High Android fat mass	0.69(0.44–1.09)	0.113	0.51(0.29–0.89)	0.019
Gynoid fat mass (kg)	0.76(0.60–0.97)	0.028	0.68(0.50–0.92)	0.012
High Gynoid fat mass	0.62(0.39–0.98)	0.041	0.52(0.30–0.91)	0.021
VAT mass (kg)	0.76(0.33–1.75)	0.523	0.43(0.16–1.17)	0.099
High VAT mass	0.69(0.44–1.09)	0.113	0.45(0.25–0.78)	0.005
SMI (kg/m^2)	0.94(0.84–1.05)	0.298	0.87(0.74–1.02)	0.087
Low muscle mass	1.23(0.78–1.94)	0.373	1.23(0.71–2.13)	0.461
Sarcopenia	2.02(1.26–3.25)	0.004	2.31(1.23–4.33)	0.009

Adjusted for age, diabetes duration, hypertension, dyslipidemia, history of CVD, eGFR, insulin usage, ACEI/ARBs, and SGLT2 inhibitors usage

Abbreviations DR: diabetic retinopathy; T2DM: type 2 diabetes mellitus; BMI: body mass index; FMI: fat mass index; VAT: visceral adipose tissue. SMI: skeletal muscle in-dex

Table 3 Associations between sarcopenia and DR in T2DM patients with obesity

	Univariable		Multivariable	
	OR (95% CI)	***p*-value**	**OR (95% CI)**	***p*-value**
BMI ≥ 25 kg/m^2				
SMI (kg/m^2)	0.78(0.60-1.015)	0.065	0.79(0.62–1.02)	0.064
Low muscle mass	3.13(1.43–6.82)	0.004	3.02(1.26–7.29)	0.014
Sarcopenia	5.30(2.09–13.42)	<0.001	4.60(1.57–13.48)	0.005
High FMI				
SMI (kg/m^2)	0.97(0.83–1.13)	0.697	0.98(0.83–1.64)	0.852
Low muscle mass	2.26(1.11–4.58)	0.024	1.97(0.88–4.42)	0.101
Sarcopenia	3.03(1.48–6.19)	0.002	1.99(0.85–4.68)	0.114
High android fat mass				
SMI (kg/m^2)	0.95(0.81–1.11)	0.527	0.95(0.80–1.13)	0.572
Low muscle mass	2.64(1.32–5.26)	0.006	2.39(1.07–5.31)	0.033
Sarcopenia	4.11(2.01–8.41)	<0.001	3.27(1.37–7.80)	0.007
High gynoid fat mass				
SMI (kg/m^2)	0.90(0.75–1.07)	0.236	0.88(0.73–1.07)	0.204
Low muscle mass	2.64(1.30–5.38)	0.008	2.49(1.10–5.63)	0.029
Sarcopenia	2.94(1.43–6.01)	0.003	2.11(0.88–5.02)	0.093
High VAT mass				
SMI (kg/m^2)	0.88(0.73–1.06)	0.180	0.94(0.80–1.11)	0.482
Low muscle mass	1.95(0.98–3.85)	0.056	2.04(0.92–4.53)	0.082
Sarcopenia	2.58(1.28–5.19)	0.008	2.50(1.06–5.92)	0.037

Adjusted for age, diabetes duration, hypertension, and eGFR

Abbreviations DR: diabetic retinopathy; T2DM: type 2 diabetes mellitus; BMI: body mass index; SMI: skeletal muscle index; FMI: fat mass index; VAT: visceral adipose tissue

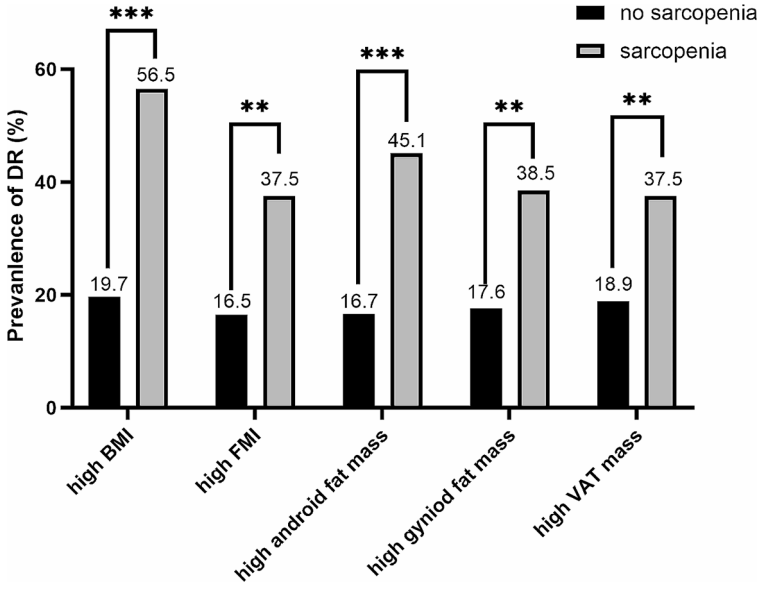

Fig. 2 Prevalence of DR in patients with T2DM and obesity in the absence of sarcopenia and the sarcopenia state. *Note* high BMI means BMI ≥ 25 kg/m^2; high FMI/high android fat mass/high gynoid fat mass/high VAT mass means FMI/android fat mass/gynoid fat mass/VAT mass was separately higher than the sex-specific median. **means p-value < 0.01; *** means p-value < 0.001; *Abbreviations* DR: diabetic retinopathy; T2DM: type 2 diabetes mellitus; BMI: body mass index; FMI: fat mass index; VAT: visceral adipose tissue

Discussion

This study investigated the association between obesity, as measured by BMI, and various fat indicators obtained through DXA, and DR in patients with T2DM. We found a lower prevalence of DR in patients with obesity compared to those without obesity. A significant negative association between obesity and DR. Further analysis showed that sarcopenic obesity had a higher prevalence of DR than simple obesity. There was a significant positive correlation between sarcopenic obesity and DR.

The association between obesity status and DR as investigated in previous studies using basic assessment metrics such as BMI, waist circumference, and waist-to-hip ratio proved equivocal. Several studies have demonstrated an inverse association between BMI and DR in patients with diabetes [4–6,25]. This is consistent with our findings. Conversely, a multicenter randomized trial involving patients aged 40–70 years with T2DM and an 8-year follow-up, conducted as part of the Japan Diabetic Complications Study, revealed that a higher BMI was linked to the development of DR [2]. A cross-sectional study involving 1,952 Chinese patients diagnosed with T2DM arrived at comparable findings [3]. Recent studies have suggested that abdominal obesity may serve as a more clinically relevant DR risk than systemic obesity in patients with T2DM [5,7,8]. Nevertheless, the association between central obesity, as measured by waist circumference and waist-to-hip ratio (WHR), and DR exhibited inconsistency in several studies [4,5,7,26]. The observed inconsistency may arise from differences in racial demographics, population characteristics, research methodology, and thresholds. It may also be ascribed to the limits of BMI, WC, and WHR as crude measures for obesity, as they cannot accurately represent the association between obesity and DR.

Therefore, additional indicators need to be used to assess the relationship between obesity and DR comprehensively. In our investigation, obesity was evaluated using a range of indicators, including total obesity indicators (BMI, FMI), and regional adiposity indicators (android fat mass, gynoid fat mass, and VAT fat mass). A study that included 1130 participants with T2DM from the Korean National Health and Nutrition Examination Survey showed that higher total body fat was significantly associated with a lower risk of Vision-Threatening Diabetic Retinopathy (VTDR) [27]. Similar results were obtained in our study using adjusted height squared total body fat (FMI), which is recommended for diagnosing obesity, similar to BMI. The advantage is that it is a direct measurement parameter for obesity [28]. Excess fat in the android region, mainly around the trunk and upper body, may lead to "apple-shaped" or central obesity, characterized by the dominance of visceral adipose tissue. Excessive visceral adipose tissue has been associated with heightened vulnerability to a range of metabolic disorders, such as impaired glucose and lipid metabolism, as well as insulin resistance [29,30]. Gynoid fat predominantly accumulates around the buttocks and thighs. Several studies have indicated a potential protective effect of subcutaneous fat in the lower body, including leg fat mass and gynoid fat mass, against CVD and T2DM [9,11,31]. Our results showed that, even after adjusting for hypertension, dyslipidemia, and CVD, obesity, as measured by either total obesity indicators (FMI) or regional adiposity indicators (android fat mass, gynoid fat mass, or VAT mass), was significantly inversely linked with DR. This implies that obesity may potentially act as a protective factor against DR. Additional cohort studies are required to investigate the causal relationship between obesity and DR.

In addition to adipose tissue, muscle, as the primary location for insulin-stimulated systemic glucose uptake, can impact systemic glucose regulation and insulin sensitivity [32]. Sarcopenia, a degenerative skeletal muscle condition characterized by the accelerated loss of muscle mass and function, has been increasingly acknowledged as a prevalent chronic complication in individuals with T2DM [33]. Research has demonstrated a significant association between sarcopenia and DN, DR, and diabetic neuropathy [17,34,35]. Our investigation also validated the significant association between sarcopenia and DR in individuals with T2DM, aligning with findings from previous research [17,36].

Both sarcopenia and obesity can independently increase the risk of mortality, and metabolic disorders, and impact quality of life. When these two conditions are concomitantly present, there may be a synergistic amplification of risks [13,14]. Patients with sarcopenic obesity may have a normal body shape due to reduced muscle mass and surplus fat, and therefore they are often ignored. Studies have shown that sarcopenic obesity is associated with higher waist circumference, elevated fasting blood sugar, insulin resistance, increased blood pressure, and abnormal blood lipid levels [37–39]. An increasing body of evidence indicates that diabetes is linked to a heightened risk of sarcopenic obesity [14,40]. It is hypothesized that there exists a bidirectional interaction involving obesity, low-grade inflammation, insulin resistance, and sarcopenia [14,41]. Cross-sectional studies have shown a significant positive association between sarcopenic obesity and CKD in diabetic patients [42,43], and a cohort study conducted in South Korea found an increased risk of CKD in patients with sarcopenic obesity over an average follow-up period of 8.9 ± 3.5 years [16]. Several studies have shown a significant correlation between DR and DN in individuals with T2DM [44,45]. DN has been identified as an independent risk factor for DR [46]. Our study revealed a significant positive association between sarcopenic obesity and DR in patients with T2DM and obesity even after adjusting for eGFR. As mentioned earlier, a substantial inverse link between obesity and DR has been found in this study. Additional subgroup analysis showed that DR and sarcopenic obesity were positively correlated. It appears that obesity is "good" for DR as obese individuals are less likely to develop DR. Sarcopenic obesity is "bad" for DR as patients with sarcopenic obesity are more likely to develop DR. We speculate that the protective impact

of fat on DR may be offset by sarcopenia, and further cohort studies are required to verify the findings. Given the impact of sarcopenia and sarcopenic obesity on DR, it is crucial to identify sarcopenia in T2DM patients, especially with obesity. We recommend that patients with T2DM engage in suitable resistance exercise, limit caloric intake, increase protein consumption, and take micronutrient supplements, particularly those with obesity [14,47].

There were several restrictions on our research. First, even though we have shown a correlation between obesity, sarcopenic obesity, and DR in T2DM patients, the cross-sectional design of the study makes it impossible to conclude causality. In the future, more longitudinal studies are required. Second, because all of the patients in our study came from the same medical facility and had similar features, the generalizability of our findings might be restricted. Third, the findings are based on a sample from China, meaning that the conclusions may not universally apply to other racial population. Fourth, only 367 patients finished all of the assessments because there were variations in participant compliance. Not every participant had their grip strength and 6-meter walking speed assessed. Future studies with larger sample sizes are required.

Conclusions

This study found an inverse association between obesity and DR in patients with T2DM. Sarcopenic obesity was significantly positively associated with DR in obese T2DM patients. Detecting sarcopenia in T2DM patients, especially those who are obese, is crucial for guiding direct clinical intervention. Additional prospective cohort studies are required in the future to establish a causal association.

Supplementary Information

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Supplementary Material 1

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Author contributions
Substantial contributions to the conception and design of the study were made by WW and MT; data collection was handled by ZYC, RYL, SLL, HC, HJC and BZC; data analysis and/or interpretation of data for the work was made by ZYC and XJZ; drafting of the work or revising it critically for important intellectual content was made by ZYC and WW; and all the authors gave final approval of the version to be published.

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Data availability
The data used to support the findings of this study have not been made available because of patient privacy. The dataset supporting the conclusions of this article is included within the article and its additional files.

Declarations

Ethics approval and consent to participate
The study was approved by the Institutional Ethics Research Committee of Longyan First Affiliated Hospital of Fujian Medical University. All patients gave written informed consent for participation in the study.

Consent for publication
All authors support the submission to this journal.

Competing interests
The authors declare that they have no competing interests.

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