Diabetes, Metabolic Syndrome and Obesity

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ORIGINAL RESEARCH

Association Between Visceral Obesity and Glycemic Control in Patients with Type 2 Diabetes Mellitus: A Retrospective Study

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Purpose: To investigate the association between visceral obesity and glycemic control in patients with type 2 diabetes mellitus.

Patients and Methods: A retrospective analysis involved 714 patients diagnosed with type 2 diabetes mellitus from the National Metabolic Management Center from November 2021 to February 2024. Medical data included sociodemographic data, lifestyle behaviors, and anthropometric and biochemical measurements. Multivariate logistic regression analysis was used to analyze their associations.

Results: Among the patients, 251 (35.2%) achieved good glycemic control (HbA1c < 7.0%). On univariate analysis, higher diastolic blood pressure, longer duration of type 2 diabetes mellitus, tobacco smoking, alcohol drinking, insulin treatment, higher levels of fasting plasma glucose, homeostasis model assessment of insulin resistance, triglyceride, total cholesterol, and low-density lipoprotein cholesterol, visceral obesity (visceral fat area ≥ 100cm^2) and diabetic peripheral neuropathy were all positively correlated with poor glycemic control; female, older age, higher levels of C peptide and serum uric acid were inversely associated with poor glycemic control (all $P < 0.05$). On multivariate logistic regression analysis, the results suggested that higher diastolic blood pressure [OR: 1.021, 95% CI (1.002, 1.040), $P = 0.030$], insulin treatment [currently used: OR = 2.156, 95% CI (1.249, 3.724), $P = 0.006$], higher level of fasting plasma glucose [OR: 1.819, 95% CI (1.598, 2.069), $P < 0.001$], and visceral obesity [OR: 1.876, 95% CI (1.158, 3.038), $P = 0.011$] were risk factors for poor glycemic control.

Conclusion: This study indicated that visceral obesity (visceral fat area ≥ 100cm^2) is positively associated with poor glycemic control, and serves as an independent risk factor for poor glycemic control (HbA1c ≥ 7.0%) in patients with type 2 diabetes mellitus. Screening for visceral obesity should be emphasized, and targeted interventions should be taken to improve glycemic control in patients with type 2 diabetes mellitus.

Keywords: type 2 diabetes mellitus, glycemic control, influencing factors, visceral obesity, visceral fat area

Introduction

Diabetes mellitus is a metabolic disease with a high prevalence characterized by chronic hyperglycemia. According to the International Diabetes Federation and the Global Burden of Disease (GBD), there were 529 million people with diabetes worldwide in 2021, with type 2 diabetes mellitus (T2DM) accounting for 96.0%.[1] The health burden caused by diabetes and its complications is increasing.[1,2] Long-term carbohydrate, as well as fat and protein metabolism disorders can cause chronic progressive lesions, functional decline and failure of the eyes, kidneys, heart, nerves, and other tissues and organs, which are the main causes of disability and death in diabetic patients.[3] Good glycemic control is essential for preventing diabetic complications.

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However, the current status of glycemic control of T2DM is not optimistic. The International Diabetes Management Practices Study (IDMPS) collected real-world data from 2005 to 2017 and indicated that <50% of participants attained the HbA1c goal [<53 mmol/mol (<7%)].[4] To further improve the blood glucose control of T2DM, many studies have explored the influencing factors of blood glucose control. Age, gender, region, education level, marital status, course of the disease, central obesity, poor lifestyle and dietary habits, comorbidity and poly-pharmacy have been identified as influencing factors for poor glycemic control.[5–9]

Visceral obesity, characterized by excessive accumulation of visceral adipose tissue, is strongly associated with a number of health-damaging diseases, including cardiovascular disease, metabolic disorders and cancer.[10,11] Visceral obesity has been reported as a strong predictor of (non-insulin-dependent) T2DM[12,13] and is correlated with insulin resistance.[14–16] A recent study[17] showed that visceral fat area (VFA) was significantly associated with HbA1c, and the relationship between the two variables was an inverted U-shaped association in T2DM patients. However, the association between visceral obesity and glycemic control remains elusive. Therefore, this study investigated the association between visceral obesity and glycemic control, so as to provide a theoretical basis for future targeted interventions to enhance blood glucose management.

Methods

Study Design and Participants

This retrospective study was conducted at the subcenter of the National Metabolic Management Center (MMC) at Fangshan Hospital of Beijing University of Traditional Chinese Medicine, spanning from November 2021 to February 2024 and involving 2111 patients (Figure 1). Exclusion criteria included: (a) patients < 18 years old; (b) patients with type 1 diabetes, gestational diabetes, and other specific types of diabetes; (c) glycated hemoglobin (HbA1c) < 7.0%; (d) patients with missing or incomplete data. A total of 714 patients were finally included for analysis. Baseline information encompassed questionnaire-sociodemographic data, lifestyle behaviors, anthropometric and biochemical measurements, including age, gender, level of education, diabetes medication, measurement of visceral fat area and liver and kidney function, etc. The data used in this study were retrieved from the electronic records of Fangshan Hospital of Beijing University of Traditional Chinese Medicine. Ethics approval was obtained from the Ethics Committee of Fangshan Hospital of Beijing University of Chinese Medicine (approval number: FZY LK-2024-036). Patient confidentiality was maintained, and informed consent was waived by the ethics committee, with no collection of personal patient information. All methods were performed following relevant guidelines and regulations.

Questionnaire-Sociodemographic Data and Lifestyle Behaviors

This study obtained questionnaire-sociodemographic data and lifestyle behaviors including age, gender, education level, occupation, annual income, tobacco smoking, alcohol drinking, duration, medication use, complications, and comorbidities of T2DM.

Anthropometric and Biochemical Measurements

This study collected anthropometric measurements including height, weight, waist, hip, systolic blood pressure (SBP), diastolic blood pressure (DBP), VFA, and the subcutaneous fat area (SFA). Biochemical indices included fasting plasma glucose (FPG), fasting insulin (FINS) and C peptide, alanine aminotransferase (ALT), aspartate aminotransferase (AST), γ-glutamyltransferase (γ-GT), albumin (ALB), blood urea nitrogen (BUN), serum creatinine (Cr), serum uric acid (UA), triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Body mass index (BMI) was calculated as BMI = weight (kg)/height $(m)^2$. Homeostasis model assessment of insulin resistance (HOMA-IR) was used to evaluate the individual level and was calculated as follows: HOMA-IR = FPG (mmol/L) * FINS (mU/L)/22.5.

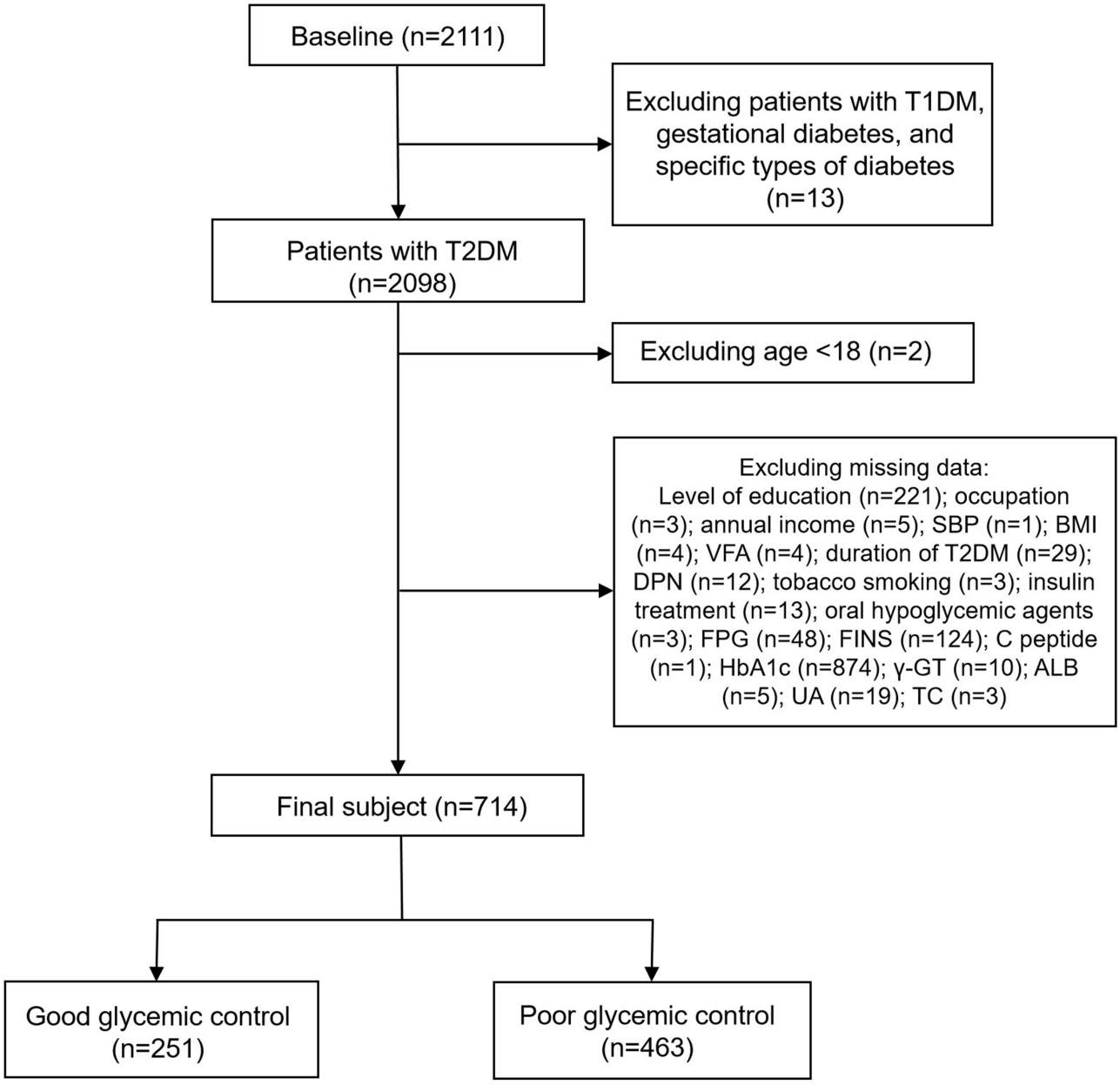

Figure 1 Flowchart of the study population.

Definitions of Visceral Obesity

VFA and SFA were measured using the DUALSCAN device (HDS-2000, OMRON, China). According to the cut-off value of VFA recommended by the Japan Society for the Study of Obesity,[18,19] VFA ≥ 100cm^2 was defined as visceral obesity.

HbA1c-Long Term Glycemic Control

HbA1c was measured by collecting venous blood from individuals after 10~12 h of fasting to evaluate glycemic control. According to the glycemic goals recommended by the American Diabetes Association,[20] HbA1c < 7% was considered as good glycemic control, whereas HbA1c ≥ 7% was defined as poor glycemic control.

Data Analysis

SPSS 24.0 software and R version 4.2.2 software were used for all analyses. GraphPad Prism 9.3.1 software was used for drawing. Variables conforming to a normal distribution were presented as mean ± SD and analyzed using an independent *t*-test, while variables conforming to a non-normal distribution were presented as medians and interquartile ranges and analyzed using the Mann–Whitney *U*-test. Categorical variables were described by frequency and percentage and analyzed using the Chi-square test. Univariate and multivariate analysis were performed to identify the variables affecting glycemic control in T2DM patients. Baseline variables that were considered clinically relevant or showed a univariate relationship with outcome were entered into the multivariable model. The odds ratio (OR) and related 95% confidence interval (CI) were evaluated. A two-tailed $p<0.05$ was considered statistically significant.

Results

General Characteristics of T2DM Patients

A total of 714 patients with T2DM were included in the study. Male patients accounted for 51.7%. 353 (49.4%) people had received a high school education or above. 172 (24.1%) patients were unemployed while 215 (30.1%) were managers. In terms of income, 407 (57.0%) patients had an annual income between ¥100,000 and ¥300,000. The majority of patients (70.9%) were non-smokers and 440 (61.6%) did not consume alcohol. Additionally, 441 (61.8%) patients had not received insulin treatment. 251 T2DM patients (35.2%) exhibited good glycemic control when they first joined the MMC management.

The research finding has shown that the good glycemic control prevalence of males was lower than females (χ^2 = 9.566, P = 0.002); tobacco smoking (χ^2 = 8.264, P = 0.016), alcohol drinking (χ^2 = 9.848, P = 0.007), and insulin treatment (χ^2 = 38.433, $P < 0.001$) were significantly associated with glycemic control. The median value of the duration of T2DM was significantly lower in patients with good glycemic control compared with those with poor glycemic control ($Z = -2.200$, P = 0.028). Interestingly, the age of patients with good glycemic control was higher than patients with poor glycemic control ($Z = -2.567$, P = 0.010) (Table 1).

Anthropometric and Biochemical Indicators of T2DM Patients

Patients with poor glycemic control exhibited significantly higher DBP, VFA, SFA, FPG, HOMA-IR, γ-GT, TG, TC, and LDL-c levels and lower C peptide and AST levels compared to those with good glycemic control (all $P < 0.05$). However, there were no significant differences in BMI, SBP, waist, hip, FINS, ALT, ALB, BUN, Cr, UA, or HDL-c between patients with poor glycemic control and those with good glycemic control (Table 2).

Table 1 General Characteristics of T2DM Patients

Characteristics	Good Glycemic Control (n = 251)	Poor Glycemic Control (n = 463)	Statistics	P value
Gender, n (%)			9.566	0.002*
Male	110 (29.8)	259 (70.2)		
Female	141 (40.9)	204 (59.1)		
Age (years old)	61 (53, 68)	58 (50, 66)	-2.567	0.010*
Level of education, n (%)			0.235	0.628
Below high school	130 (36.0)	231 (64.0)		
High school or above	121 (34.3)	232 (65.7)		

(*Continued*)

Table 1 (Continued).

Characteristics	Good Glycemic Control (n = 251)	Poor Glycemic Control (n = 463)	Statistics	P value
Occupation, n (%)			/	0.213
Unemployed	62 (36.0)	110 (64.0)		
Managers	88 (40.9)	127 (59.1)		
Clerical support	3 (37.5)	5 (62.5)		
Service and sales	36 (36.7)	62 (63.3)		
Craft & Related trade	1 (10.0)	9 (90.0)		
Agricultural	5 (23.8)	16 (76.2)		
Skilled professional	41 (28.7)	102 (71.3)		
Labourer	15 (31.9)	32 (68.1)		
Annual income (RMB), n (%)			/	0.888
<10,000	1 (50.0)	1 (50.0)		
10,000~	2 (22.2)	7 (77.8)		
30,000~	85 (36.6)	147 (63.4)		
100,000~	141 (34.6)	266 (65.4)		
300,000~	22 (34.4)	42 (65.6)		
Duration of T2DM (years)	65 (23, 147)	97 (33, 183)	-2.200	0.028*
Tobacco smoking, n (%)			8.264	0.016*
No	194 (38.3)	312 (61.7)		
Occasionally	19 (31.1)	42 (68.9)		
Daily or almost daily	38 (25.9)	109 (74.1)		
Alcohol drinking, n (%)			9.848	0.007*
No	174 (39.5)	266 (60.5)		
Occasionally	56 (27.5)	148 (72.5)		
Weekly or almost weekly	21 (30.0)	49 (70.0)		
Insulin treatment, n (%)			38.433	<0.001*
Never used	193 (43.8)	248 (56.2)		
Deactivated	6 (14.6)	35 (85.4)		
Currently used	52 (22.4)	180 (77.6)		
Oral hypoglycemic agents, n (%)			0.039	0.844
Not used	58 (35.8)	104 (64.2)		
Currently used	193 (35.0)	359 (65.0)		

Notes: Data are expressed as median (interquartile range), or n (%) as appropriate. *Denotes significance at a P value of <0.05.
Abbreviations: T2DM, type 2 diabetes mellitus.

Table 2 Anthropometric and Biochemical Indicators of T2DM Patients

Characteristics	Good Glycemic Control (n = 251)	Poor Glycemic Control (n = 463)	Statistics	P value
BMI (kg/m^2)	26.4 (24.2, 29.0)	26.4 (24.3, 29.3)	−0.290	0.772
SBP (mmHg)	136 (124, 150)	138 (124, 152)	−0.795	0.427
DBP (mmHg)	79 (72, 86)	81 (74, 88)	−3.079	0.002*
Waist (cm)	93.0 (87.5, 100.0)	94.0 (88.0, 100.0)	−1.394	0.163
Hip (cm)	99.5 (95.5, 104.0)	99.0 (94.5, 104.0)	−1.139	0.255
VFA (cm^2)	101 (82, 129)	112 (85, 139)	−2.628	0.009*
SFA (cm^2)	185 (155, 234)	186 (153, 239)	−5.232	<0.001*
FPG (mmol/L)	6.90 (6.00, 7.90)	9.15 (7.60, 11.67)	−13.164	<0.001*
FINS (μIU/mL)	12.64 (8.76, 18.95)	13.55 (8.51, 20.47)	−0.738	0.460
C peptide (ng/mL)	2.63 (1.83, 3.70)	2.18 (1.31, 3.19)	−4.450	<0.001*
HOMA-IR	3.87 (2.68, 5.85)	5.67 (3.19, 8.76)	−5.689	<0.001*
ALT (U/L)	21 (14, 30)	21 (15, 32)	−1.028	0.304
AST (U/L)	21.0 (17.0, 26.0)	20.0 (16.3, 25.6)	−2.018	0.044*
γ-GT (U/L)	22.0 (17.0, 32.2)	27.0 (19.0, 40.0)	−3.787	<0.001*
ALB (g/L)	43.9 (41.7, 46.0)	43.5 (40.6, 46.1)	−1.405	0.160
BUN (mmol/L)	5.42 (4.49, 6.44)	5.48 (4.54, 6.74)	−0.989	0.323
Cr (umol/L)	74.0 (62.9, 85.1)	72.0 (61.0, 85.0)	−1.187	0.235
UA (umol/L)	329.1 (276.0, 400.0)	323.8 (271.9, 380.8)	−1.735	0.083
TG (mmol/L)	1.41 (0.93, 2.01)	1.51 (1.04, 2.24)	−2.186	0.029*
TC (mmol/L)	4.37 (3.66, 5.07)	4.73 (3.82, 5.61)	−3.203	0.001*
HDL-c (mmol/L)	1.18 (1.00, 1.39)	1.19 (1.02, 1.38)	−0.158	0.874
LDL-c (mmol/L)	2.62 (2.11, 3.32)	2.99 (2.33, 3.70)	−4.068	<0.001*

Notes: Data are expressed as median (interquartile range). *Denotes significance at a P value of <0.05.

Abbreviations: ALB, albumin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; BUN, blood urea nitrogen; Cr, serum creatinine; DBP, diastolic blood pressure; FINS, fasting insulin; FPG, fasting plasma glucose; HDL-c, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; LDL-c, low-density lipoprotein cholesterol; SBP, systolic blood pressure; SFA, subcutaneous fat area; TC, total cholesterol; TG, triglyceride; T2DM, type 2 diabetes mellitus; UA, serum uric acid; VFA, visceral fat area; γ-GT, γ-glutamyltransferase.

Visceral Obesity, Other Comorbidities and Complications of T2DM Patients

The research finding has demonstrated that the good glycemic control prevalence of subjects with visceral obesity was lower than those without visceral obesity (χ^2 = 11.588, P < 0.001), and patients with diabetic peripheral neuropathy (DPN) lower than those without DPN (χ^2 = 8.997, P = 0.003). However, there were no significant differences in the comorbidities of hypertension, hyperlipidemia, hyperuricemia, coronary heart disease, stroke, peripheral arterial disease, and tumor between patients with good glycemic control and those with poor glycemic control (Table 3).

Table 3 Complications and Comorbidities of T2DM Patients

Characteristics	Good Glycemic Control (n = 251)	Poor Glycemic Control (n = 463)	Statistics	P value
Visceral obesity, n (%)			11.588	<0.001*
No	120 (42.7)	161 (57.3)		
Yes	131 (30.3)	302 (69.7)		
DPN, n (%)			8.997	0.003*
No	191 (38.7)	302 (61.3)		
Yes	60 (27.1)	161 (72.9)		
Hypertension, n (%)			2.569	0.109
No	85 (31.5)	185 (68.5)		
Yes	166 (37.4)	278 (62.6)		
Hyperlipidemia, n (%)			1.816	0.178
No	89 (32.1)	188 (67.9)		
Yes	162 (37.1)	275 (62.9)		
Hyperuricemia, n (%)			3.182	0.074
No	222 (34.2)	428 (65.8)		
Yes	29 (45.3)	35 (54.7)		
Coronary heart disease, n (%)			0.929	0.335
No	186 (34.2)	358 (65.8)		
Yes	65 (38.2)	105 (61.8)		
Stroke, n (%)			0.480	0.488
No	237 (35.5)	431 (64.5)		
Yes	14 (30.4)	32 (69.6)		
Peripheral arterial disease, n (%)			/	1.000
No	248 (35.2)	457 (64.8)		
Yes	3 (33.3)	6 (66.7)		
Tumor, n (%)			0.240	0.624
No	245 (35.3)	449 (64.7)		
Yes	6 (30.0)	14 (70.0)		

Notes: Data are expressed as n (%). *Denotes significance at a P value of <0.05.
Abbreviations: DPN, diabetic peripheral neuropathy; T2DM, type 2 diabetes mellitus.

Univariate Analysis of Risk Factors for Glycemic Control in T2DM Patients

Univariable logistic regression was employed to analyze significant clinical factors potentially associated with glycemic control. The results indicated that higher DBP, longer duration of T2DM, tobacco smoking, alcohol drinking, insulin treatment, higher levels of FPG, HOMA-IR, TG, TC, LDL-c, visceral obesity, and DPN were all positively correlated

with poor glycemic control; female, older age, higher levels of C peptide and UA were inversely correlated with poor glycemic control ($P < 0.05$) (Table 4).

Multivariate Logistic Regression Analysis of Risk Factors for Glycemic Control in T2D Patients

Multivariable logistic regression was employed to adjust for confounding factors. The research findings suggested that visceral obesity remained significantly associated with glycemic control after adjusting for gender, age, DBP, waist, duration of T2DM, tobacco smoking, alcohol drinking, insulin treatment, FPG, C peptide, HOMA-IR, UA, TG, TC, LDL-c and DPN. The results showed that higher DBP [OR: 1.021, 95% CI (1.002, 1.040), $P = 0.030$], insulin treatment [currently used: OR=2.156, 95% CI (1.249, 3.724), $P = 0.006$], higher level of FPG [OR: 1.819, 95% CI (1.598, 2.069), $P < 0.001$], and visceral obesity [OR: 1.876, 95% CI (1.158, 3.038), $P = 0.011$] were risk factors for poor glycemic control (Figure 2).

Table 4 Univariate Analysis of Key Characteristics and Glycemic Control

Characteristics	OR	95% CI	P value
Gender			
Male (Reference)			
Female	0.614	0.451–0.837	0.002*
Age (years old)	0.980	0.966–0.994	0.006*
DBP (mmHg)	1.023	1.009–1.038	0.002*
Waist (cm)	1.011	0.995–1.028	0.162
Duration of T2DM (years)	1.002	1.000–1.004	0.013*
Tobacco smoking			
No (Reference)			
Occasionally	1.374	0.777–2.432	0.275
Daily or almost daily	1.784	1.183–2.689	0.006*
Alcohol drinking			
No (Reference)			
Occasionally	1.729	1.204–2.483	0.003*
Weekly or almost weekly	1.526	0.884–2.634	0.129
Insulin treatment			
Never used (Reference)			
Deactivated	4.540	1.871–11.013	0.001*
Currently used	2.694	1.877–3.867	<0.001*
FPG (mmol/L)	1.815	1.626–2.026	<0.001*
C peptide (ng/mL)	0.830	0.753–0.916	<0.001*
HOMA-IR	1.105	1.060–1.151	<0.001*

(*Continued*)

Table 4 (Continued).

Characteristics	OR	95% CI	P value
UA (umol/L)	0.998	0.996–1.000	0.030*
TG (mmol/L)	1.161	1.036–1.302	0.010*
TC (mmol/L)	1.237	1.082–1.413	0.002*
LDL-c (mmol/L)	1.367	1.156–1.615	<0.001*
Visceral obesity			
No (Reference)			
Yes	1.718	1.257–2.350	0.001*
DPN			
No (Reference)			
Yes	1.697	1.199–2.402	0.003*

Notes: *Denotes significance at a P value of <0.05; OR: odds ratio; 95% CI = 95% confidence interval.

Abbreviations: DBP, diastolic blood pressure; DPN, diabetic peripheral neuropathy; FPG, fasting plasma glucose; HOMA-IR, homeostasis model assessment of insulin resistance; LDL-c, low-density lipoprotein cholesterol; TC, total cholesterol; TG, triglyceride; T2DM, type 2 diabetes mellitus; UA, serum uric acid.

Discussion

As a common chronic disease, T2DM can lead to a variety of serious complications and even premature death, causing a great economic burden to individuals and society. Recognizing and effectively managing factors associated with poor glycemic control are crucial for preventing diabetes-related complications. In this study, we investigated the association between visceral obesity and glycemic control and concluded that visceral obesity is an independent risk factor for poor glycemic control in T2DM patients.

We found that 35.0% of T2DM patients achieved good glycemic control when they first joined MMC management, exceeding the previously reported rate of 32.2% in China.[21] Furthermore, our study referred to the diagnostic criteria recommended by the Japan Society for the Study of Obesity,[18] which used 100 cm^2 as the diagnostic threshold for visceral obesity, and concluded that visceral obesity (VFA $\geq$ 100 cm^2) was an independent predictor of poor glycemic control (HbA1c $\geq$ 7.0%). Several studies have explored the association between VFA and glucose metabolism. A study[17] conducted at the MMC of People's Hospital of Yuxi, Yunnan Province showed an inverted U-shaped association between the VFA and HbA1c. Another study[22] conducted at Nanjing Drum Tower Hospital revealed a non-linear relationship between HbA1c and VFA, with inflection points observed around 7%. Abe et al[23] reported that VFA was independently associated with T2DM in Japanese adolescents. The accumulation of visceral fat is associated with the development of diabetes due to a decrease in adiponectin secretion by adipocytes.[24,25] Additionally, VFA shows a strong correlation with insulin resistance.[26] Excess visceral fat accumulation is related to various diabetogenic, atherogenic, and proinflammatory metabolic abnormalities, which together increase the risk of atherosclerotic cardiovascular disease.[27,28] Our findings indicated that particular attention should be given to the presence of visceral obesity in patients with T2DM, which can help them achieve good glycemic control.

In our study, T2DM patients with poor glycemic control also had higher levels of DBP and FPG. Previous research has demonstrated that diabetes and hypertension share several common pathogenic mechanisms, including activation of the renin-angiotensin system, oxidative stress, and proinflammatory cytokines.[29] Furthermore, preceding clinical studies have indicated that hypertension contributes to glucose dysregulation, and reducing blood pressure can attenuate the development of overt diabetes in patients with glucose intolerance.[30–32] These findings suggest the importance of controlling blood pressure in diabetic patients, which may be a key factor in improving blood glucose control.

Characteristics	OR	95% CI	P value
Gender			
Male (Reference)			
Female	0.955	0.570-1.600	0.863
Age (years old)	1.005	0.983-1.027	0.657
DBP (mmHg)	1.021	1.002-1.040	0.030*
Waist (cm)	0.981	0.956-1.007	0.157
Duration of T2DM (years)	1.001	0.999-1.004	0.370
Tobacco smoking			
No (Reference)			
Occasionally	1.213	0.558-2.634	0.626
Daily or almost daily	1.746	0.958-3.183	0.069
Alcohol drinking			
No (Reference)			
Occasionally	1.445	0.867-2.407	0.158
Weekly or almost weekly	0.855	0.402-1.820	0.685
Insulin treatment			
Never used (Reference)			
Deactivated	2.459	0.856-7.063	0.095
Currently used	2.156	1.249-3.724	0.006*
FPG (mmol/L)	1.819	1.598-2.069	<0.001*
C peptide (ng/mL)	0.848	0.710-1.014	0.071
HOMA-IR	0.990	0.932-1.052	0.750
UA (umol/L)	0.998	0.995-1.000	0.106
TG (mmol/L)	1.052	0.913-1.212	0.481
TC (mmol/L)	0.803	0.477-1.351	0.408
LDL-c (mmol/L)	1.499	0.810-2.777	0.198
Visceral obesity			
No (Reference)			
Yes	1.876	1.158-3.038	0.011*
DPN			
No (Reference)			
Yes	0.909	0.582-1.422	0.677

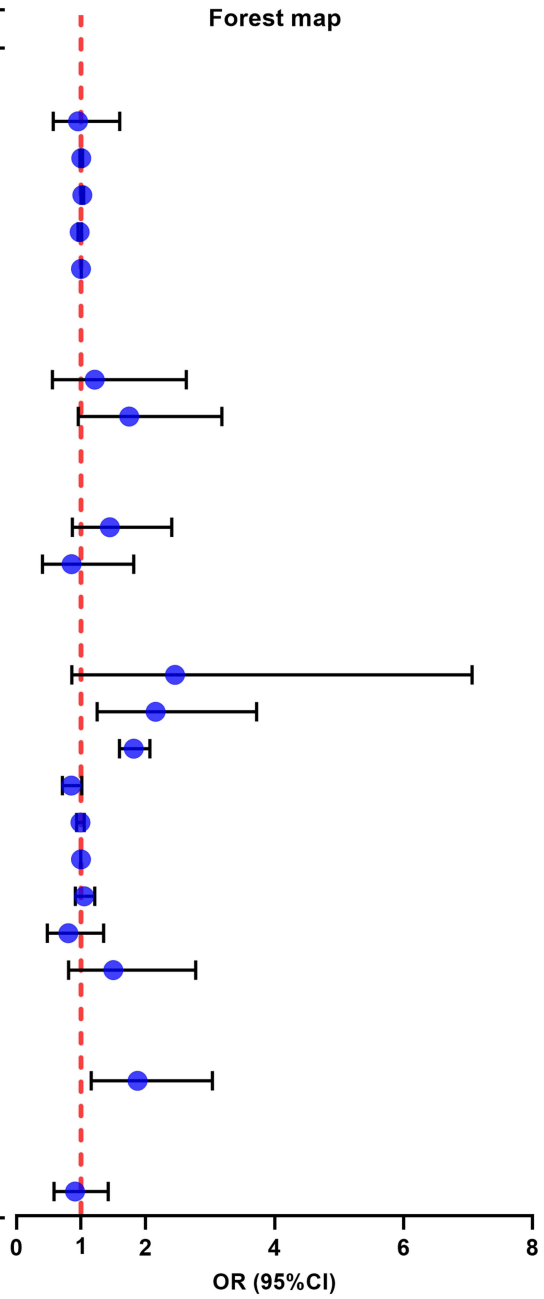

Figure 2 Multivariable logistic regression analysis of key characteristics and glycemic control.
Note: *Denotes significance at a P value of <0.05; OR: odds ratio; 95% CI = 95% confidence interval.

Additionally, our results showed that use of insulin therapy was associated with poor glycemic control. This is consistent with previous findings, which found a strong association between poor blood glucose control and receiving insulin therapy.[33] This could be a reverse cause effect where patients with higher blood glucose are more likely to receive insulin therapy or very stringent medication therapy.[33,34]

There are some limitations in this study. Firstly, this study was cross-sectional, and therefore the causal relationship could not be determined. Secondly, our data were derived exclusively from patients at a subcenter of the MMC in China, and the results cannot be generalized. Lastly, not all potential influencing factors were included, such as medications affecting HbA1c, medication adherence, and self- monitoring of blood glucose.

Conclusion

Our results discuss for the first time that visceral obesity is positively associated with poor glycemic control, and serves as an independent risk factor for poor glycemic control in patients with T2DM. The impact of visceral obesity on glycemic control should be emphasized, and appropriate measures should be implemented to reduce VFA to achieve optimal glycemic control, thereby reducing diabetes-related complications.

Abbreviations

ALB, albumin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; BUN, blood urea nitrogen; CI, confidence intervals; Cr, serum creatinine; DBP, diastolic blood pressure; DM, diabetes mellitus; DPN, diabetic peripheral neuropathy; FINS, fasting insulin; FPG, fasting plasma glucose; GBD, the International Diabetes Federation and the Global Burden of Disease; HbA1c, glycated hemoglobin; HDL-c, high-density lipoprotein cholesterol; HOMA-IR, homeostasis model assessment of insulin resistance; LDL-c, low-density lipoprotein cholesterol; MMC, the National Metabolic Management Center; OR, odds ratio; SBP, systolic blood pressure; SFA, subcutaneous fat area; T2DM, type 2 diabetes mellitus; TC, total cholesterol; TG, triglyceride; UA, serum uric acid; VFA, visceral fat area; γ-GT, γ-glutamyltransferase.

Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding authors on reasonable requests.

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Disclosure

The authors report no conflicts of interest in this work.

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