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Relationships between obesity and prevalence of gout in patients with type 2 diabetes mellitus: a cross-sectional population-based study

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Abstract

Background The purpose of this study was to investigate the relationships between generalized, abdominal, and visceral fat obesity and the prevalence of gout in patients with type 2 diabetes mellitus (T2DM).

Methods Data were obtained from the electronic medical databases of the National Metabolic Management Center (MMC) of Yuhuan Second People's Hospital and Taizhou Central Hospital (Taizhou University Hospital) between September 2017 and June 2023. Four obesity indicators were analyzed: waist circumference (WC), waist-to-hip ratio (WHR), body mass index (BMI), and visceral fat area (VFA). The relationships between these parameters and gout prevalence were analyzed using multivariate logistic regression and restricted cubic spline (RCS) analyses. Receiver operating characteristic (ROC) curves were used to evaluate the diagnostic efficacy of the four parameters for gout.

Results This cross-sectional study enrolled 10,535 participants (600 cases and 9,935 controls). Obesity was more common in patients with gout, and the obesity indicators were markedly higher in this group. After adjustment for confounders, obesity, as defined by BMI, WC, WHR, and VFA, was found to be associated with greater gout prevalence, with odds ratios (OR) of 1.775, 1.691, 1.858, and 1.578, respectively ($P < 0.001$). The gout odds ratios increased markedly in relation to the obesity indicator quartiles (P-value for trend < 0.001), and the obesity indicators were positively correlated with gout prevalence, as shown using RCS. The area under the ROC curve values for BMI, WC, WHR, and VFA were 0.629, 0.651, 0.634, and 0.633, respectively.

Conclusion Obesity—whether general, abdominal, or visceral fat obesity—was positively linked with elevated gout risk. But uncovering the causality behind the relationship requires further prospective study. Obesity indicators (BMI, WC, WHR, and VFA) may have potential value for diagnosing gout in clinical practice.

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Keywords Obesity, Gout, Type 2 diabetes mellitus, Body mass index, Waist circumference, Waist-to-hip ratio, Visceral fat area

Background

Gout is an inflammatory disease affecting the joints [1]. Over the last few decades, gout has become increasingly prevalent in various regions and countries [2]. Between 2010 and 2020, the prevalence of gout increased globally from 0.08% to 2–4% [3]. A nationwide survey in China reported a gout prevalence of 3.2% between 2015 and 2017 (4.4% in men and 2.0% in women), translating into approximately 25.56 million people being affected [4].

Type 2 diabetes mellitus (T2DM) is a co-morbidity of gout. When T2DM coexists with gout, it can worsen the effects of the disease, lower the quality of life, and increase financial burdens on society [5]. Therefore, research on gout- and diabetes-related risk factors is crucial.

Obesity is a significant global health concern and is associated with gout [6–8]. Obesity is often thought to impact gout risk via elevated levels of serum uric acid. It has been associated with hyperuricemia possibly due to increasing production and reduced renal excretion of urate [9, 10].

However, most previous studies have chosen body mass index (BMI) as a measure of obesity, which is widely applied in clinical and epidemiological research [11]. Nevertheless, the accumulation of body fat, rather than weight, is the key factor determining obesity. Waist circumference (WC) [12], waist-to-hip ratio (WHR) [13], and visceral fat area (VFA) [14] are also indicators of obesity. These parameters reflect the adipose tissue distribution throughout the body. Both WC and WHR are indicators of abdominal obesity, while visceral fat obesity (VFO) is indicated by VFA.

This study evaluated the relationship between different obesity indicators (BMI, WC, WHR, and VFA) and gout prevalence in patients with T2DM using cross-sectional data from the National Metabolic Management Center (MMC) of The Second People's Hospital of Yuhuan and Taizhou Central Hospital (Taizhou University Hospital).

Methods

Study design and participants

In this cross-sectional population-based study, a total of 10,848 participants was recruited from the MMC electronic medical database [15] of The Second People's Hospital of Yuhuan and Taizhou Central Hospital (Taizhou University Hospital) between September 2017 and June 2023. Based on the idea of "One Center, One Step, and One Standard Model", MMC is a national project to manage patients with metabolic conditions. Previous publications provide a thorough introduction to the MMC program [15]. Every patient consented to a thorough physical examination, blood sample collection, and interviews using an oral questionnaire. All data were gathered at the time of recruitment at local MMCs by personnel who had received training and followed a standard protocol. Additionally, some indicators, including visceral fat area (VFA), were detected using the same machine model. Additional quality control procedures could be seen in the MMC protocol. The inclusion criterion was to satisfy the 1999 T2DM diagnostic criteria established by the World Health Organization [16]. Those who met the following criteria were excluded: (1) T1DM or other type diabetes patients, (2) age < 18y, and (3) missing any values of obesity parameters. After screening, 10,535 people were enrolled (Fig. 1).

Patient data collection

Using the MMC specialist electronic medical record system, we gathered patient data through standardized questionnaires and clinical and laboratory assessments. The following variable data were collected: age, sex (male or female), smoking and drinking status, height (H), weight (W), WC, hip circumference (HC), diastolic blood pressure (DBP), systolic blood pressure (SBP), urea nitrogen (UN), serum creatinine (Scr), uric acid (UA), fasting blood glucose (FBG), glycated hemoglobin (HbA1c), alanine transaminase (ALT), aspartate aminotransferase (AST), triglyceride (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and estimated glomerular infiltration rate (eGFR), VFA, hypertension (no/yes), and hyperlipidemia (no/yes). H, W, WC and HC were measured with a standard protocol, and BMI was calculated as W/H^2 (kg/m^2), while WHR was determined as WC/HC. VFA were measured at the level of umbilicus by a dual bioelectrical impedance analyzer (HDS2000, Omron Healthcare Co).

Variable definitions

Gout was diagnosed according to the American College of Rheumatology (ACR)/European League Against Rheumatism (EULAR) clinical classification criteria for gout [17]. Hypertension was described as an SBP or DBP value of ≥140 or ≥90 mmHg, respectively [18], or physician diagnosis. Hyperlipidemia was determined by at least one of the following: TC≥5.7 mmol/L, TG≥1.7 mmol/L, LDL-C≥3.6 mmol/L, and HDL-C < 1.29 mmol/L in women and < 1.03 mmol/L in men [19] or physician diagnosis. Those who smoked cigarettes were considered current smokers, whereas those who were currently drinking

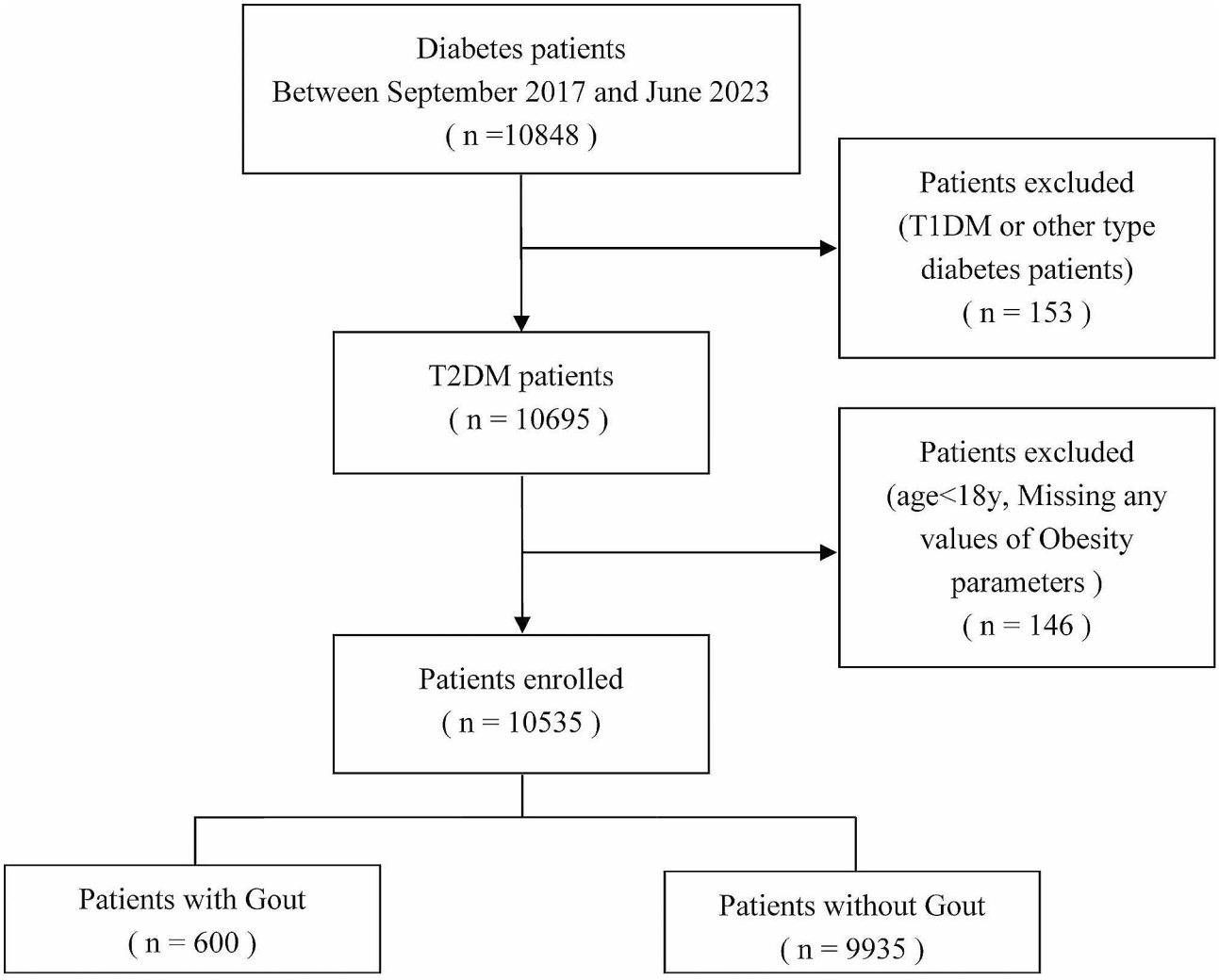

Fig. 1 Flowchart of the study

alcohol were considered current drinkers. The eGFR was determined as described [20].

Four obesity indicators were analyzed, namely, BMI, WC, WHR, and VFA. The obesity thresholds were: (1) general, BMI≥28.0 kg/m^2 [11], (2) abdominal, WC>90 cm for men or >85 cm for women [12], or WHR>0.90 for men or >0.85 for women [13], (3) VFO: VFA≥100 cm^2 [14].

Statistical analysis

Data are shown as numbers (%) or medians (interquartile range). Non-normally distributed continuous variables were evaluated using the Mann–Whitney U test, and categorical variables were analyzed with the Chi-squared test. Two-sided p-values<0.05 were deemed to indicate statistical significance. Statistical analysis was undertaken with SPSS, version 23.0, and R, version 4.1.3.

Multivariate logistic regression was used to assess the independent effects of general obesity, abdominal obesity, VFO, and obesity indicators (BMI, WC, WHR, and VFA) on the presence of gout. Obesity indicators were divided into quartiles; the lowest quartile was used as a reference. Analyses were adjusted for multiple variables. We considered the the baseline difference, clinical significance, and the results of previous studies to determine the adjusted variables [7, 21]. We performed a number of different statistical models to verify the stability of the results: Age and sex were adjusted in Model 1, with further adjustments for HbA1c, hyperlipidemia, hypertension, smoking status, drinking status, and eGFR in Model 2. We conducted Restricted cubic spline (RCS) models fitted for the logistic regression model to assess the potential nonlinear relationships between levels of obesity indicators and gout. In this model, obesity indicators was used as a continuous variable with four knots (5th,

35th, 65th and 95th) suggested by Harrell. The diagnostic efficacies of the indices for gout were analyzed with Receiver operating characteristic (ROC) curves.

Ethics statement

The study protocol conforms to STROBE guidelines [22], and was approved by the Ethics Committees of Yuhuan Second People's Hospital and Taizhou Central Hospital (Taizhou University Hospital). Written informed consent was obtained from all participants.

Results

Characteristics of study participants

Figure 1 illustrates a flowchart of the study cohort, and Table 1 lists the features of the cohort. The median (IQR) age of the participants was 55(47–63) years, and 6342(60.2%) of them were men. The median (IQR) obesity indicators(BMI, WC, WHR, VFA) was 25.2(23.07,27.55), 89.4(83.1,96), 0.94(0.9,0.98), 94(66,124), respectively. Out of 10,535 participants. 600 had gout, while 9,935 did not. The group with gout was predominately male (n=503, 83.8%) compared to the group without gout. The distribution of obesity indicators is shown in Figure S1 (Supplementary Fig. S1). Patients with gout had markedly higher values of BMI, WC, WHR, VFA, SBP, and DBP compared to those without gout. Additionally, they exhibited markedly higher levels of AST, ALT, UN, Scr, UA, and TG, and lower levels of FBG, HbA1c, HDL-C, and e-GFR. Moreover, patients with gout exhibited higher proportions of obesity (general obesity, abdominal

Table 1 Patient demographic and clinical parameters

Variables	Total(n=10535)	no Gout(n=9935)	Gout(n=600)	P value
Age (y)	55(47,63)	55(47,63)	55(47,64)	0.985
Male, n (%)	6342(60.2)	5839(58.8)	503(83.8)	< 0.001
SBP (mmHg)	75(69,83)	75(69,83)	78(71,85)	< 0.001
DBP (mmHg)	130(120,141)	130(120,141)	134(123,146)	< 0.001
BMI (kg/m^2)	25.2(23.07,27.55)	25.1(22.97,27.44)	26.6(24.4,29.1)	< 0.001
< 28, n (%)	8267(78.5)	7880(79.3)	387(64.5)	< 0.001
≥ 28, n (%)	2268(21.5)	2055(20.7)	213(35.5)	< 0.001
WC(cm)	89.4(83.1,96)	89(83,95.5)	93.6(88,100)	< 0.001
M < 90,F < 85, n (%)	4424(42)	4259(42.9)	165(27.5)	< 0.001
M ≥ 90,F ≥ 85, n (%)	6111(58)	5676(57.1)	435(72.5)	
WHR	0.94(0.9,0.98)	0.94(0.9,0.98)	0.97(0.93,1)	< 0.001
M < 0.9,F < 0.85, n (%)	1639(15.6)	1590(16.0)	49(8.17)	< 0.001
M ≥ 0.9,F ≥ 0.85, n (%)	8896(84.4)	8345(84.0)	551(91.8)	
VFA(cm^2)	94(66,124)	93(66,123)	115(85.5,146)	< 0.001
< 100, n (%)	5826(55.3)	5598(56.3)	228(38.0)	< 0.001
≥ 100, n (%)	4709(44.7)	4337(43.7)	372(62.0)	
FBG(mmol/L)	8.215(6.63,11.06)	8.23(6.64,11.07)	7.88(6.48,10.86)	0.026
HbA1c (%)	8.1(6.9,10.1)	8.1(6.9,10.1)	7.9(6.7,9.9)	0.037
ALT (IU/L)	22(16,35)	22(15,35)	27(18,41)	< 0.001
AST (IU/L)	19(15,26)	19(15,26)	21(17,29)	< 0.001
UN (mmol/L)	5.31(4.39,6.46)	5.3(4.37,6.41)	5.7(4.52,7.13)	< 0.001
Scr (mmol/L)	63(52,76)	62(52,74)	78(66,101)	< 0.001
e-GFR (mL/min per 1.73 m^2)	104.25(85.10,125.14)	105.03(86.29,125.94)	86.2(63.88,109.46)	< 0.001
UA (mmol/L)	329(270,398)	325(267,390)	437(354.5,526.5)	< 0.001
TG (mmol/L)	1.53(1.05,2.32)	1.5(1.04,2.28)	2.04(1.41,3.31)	< 0.001
TC (mmol/L)	5.08(4.29,5.91)	5.08(4.29,5.9)	5.08(4.25,5.96)	0.931
HDL-C (mmol/L)	1.12(0.94,1.33)	1.13(0.95,1.34)	1.01(0.86,1.19)	< 0.001
LDL-C (mmol/L)	2.86(2.23,3.53)	2.86(2.24,3.53)	2.82(2.105,3.46)	0.017
current smokers, n (%)	2602(24.7)	2399(24.1)	203(33.8)	< 0.001
current drinkers, n (%)	1450(13.8)	1306(13.1)	144(24.0)	< 0.001
History of hypertension, n (%)	4760(45.2)	4383(44.1)	377(62.8)	< 0.001
History of hyperlipidemia, n (%)	2772(26.3)	2482(25.0)	290(48.3)	< 0.001

Note: Data are presented as counts(%) or medians (interquartile ranges). Non-normally distributed continuous data were analyzed using the Mann–Whitney U test, and the Chi-squared test was used for comparing categorical data of baseline features between the patients with and without gout

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio, VFA, visceral fat area, FBG, fasting blood glucose; HbA1c, glycated hemoglobin; ALT, alanine transaminase; AST, aspartate aminotransferase; UN, urea nitrogen; Scr, serum creatinine; eGFR, estimated glomerular filtration rate; UA, uric acid; TG, triglycerides; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol

obesity, and VFO) and comorbidities such as hypertension and hyperlipidemia. Additionally, patients with gout had higher rates of smoking and alcohol consumption compared to those without gout.

Association between obesity indicators and gout prevalence

After gradually adjusting for all potential confounding factors, obesity defined by BMI, WC, WHR, and VFA was linked with a higher prevalence of gout, with ORs (95% CI) of 1.775 (1.468–2.145), 1.691 (1.394–2.053), 1.858 (1.367–2.524), and 1.578 (1.317–1.890), respectively (Table 2).

Table 2 illustrates the relationships between obesity parameters and gout. In the multivariate regression model, adjusting for sex, age, HbA1c, hyperlipidemia, hypertension, smoking status, drinking status, and eGFR, a significant increase in the ORs of gout from the lowest to the highest quartiles of obesity indicators (BMI, WC, WHR, and VFA) was observed (trend P-value < 0.001). The ORs (95% CI) of gout were 2.751 (2.034–3.720), 2.999 (2.185–4.118), 2.127 (1.561–2.896), and 2.259 (1.730–2.950) for the highest relative to the lowest quartiles, respectively.

Furthermore, the RCS analysis indicated positive correlations between the four obesity indices and gout prevalence in both males and females (P-value for nonlinearity = 0.1453, 0.1118, 0.0374, and 0.8171 in males, and 0.4902, 0.2890, 0.0072, and 0.3472 in females, for BMI, WC, WHR, and VFA, respectively) (Fig. 2).

RCS, restricted cubic spline; BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; VFA, visceral fat area; OR, odds ratio; CI, confidence interval; HbA1c, glycated hemoglobin; eGFR, estimated glomerular filtration rate.

ROC curve analysis of gout

The ability of obesity parameters to diagnose gout was investigated with ROC curves. The analysis showed a slightly higher area under the ROC curve (AUC) for WC (0.651, 95% CI: 0.510–0.707) and WHR (0.634, 95% CI: 0.417–0.785) than for BMI (0.629, 95% CI: 0.370–0.812) and VFA (0.633, 95% CI: 0.477–0.726) (Fig. 3). The optimum thresholds of BMI, WC, WHR, and VFA for gout were 24.055, 89.25, 0.925, and 118.5, respectively.

Table 2 Results of logistic regression analyses for the relationships between obesity indicators and gout risk

Variable	Unadjusted		Model 1		Model 2	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value	OR (95% CI)	*P* value
BMI	2.110(1.773–2.512)	< 0.001	2.209(1.848–2.641)	< 0.001	1.775(1.468–2.145)	< 0.001
Quartile1	1	< 0.001	1	< 0.001	1	< 0.001
Quartile2	20,219(2.642-3.000)	< 0.001	2.097(1.549–2.840)	< 0.001	1.888(1.380–2.582)	< 0.001
Quartile3	2.619(1.951–3.516)	< 0.001	2.408(1.791–3.238)	< 0.001	1.999(1.469–2.721)	< 0.001
Quartile4	3.923(2.960–5.200)	< 0.001	3.819(2.873–5.077)	< 0.001	2.751(2.034–3.720)	< 0.001
P for trend	< 0.001		< 0.001		< 0.001	
WC	1.978(1.646–2.377)	< 0.001	2.105(1.750–2.533)	< 0.001	1.691(1.394–2.053)	< 0.001
Quartile1	1	< 0.001	1	< 0.001	1	< 0.001
Quartile2	2.358(1.705–3.261)	< 0.001	2.056(1.484–2.849)	< 0.001	1.869(1.336–2.614)	< 0.001
Quartile3	3.211(2.348–4.392)	< 0.001	2.602(1.897–3.568)	< 0.001	2.139(1.539–2.966)	< 0.001
Quartile4	5.057(3.753–6.814)	< 0.001	4.051(2.998–5.473)	< 0.001	2.999(2.185–4.118)	< 0.001
P for trend	< 0.001		< 0.001		< 0.001	
WHR	2.143(1.592–2.884)	< 0.001	2.375(1.762–3.202)	< 0.001	1.858(1.367–2.524)	< 0.001
Quartile1	1	< 0.001	1	< 0.001	1	< 0.001
Quartile2	2.027(1.469–2.798)	< 0.001	1.669(1.206–2.309)	0.002	1.462(1.049–2.037)	0.025
Quartile3	2.877(2.120–3.904)	< 0.001	2.158(1.583–2.942)	< 0.001	1.834(1.335–2.520)	< 0.001
Quartile4	3.958(2.955–5.301)	< 0.001	3.212(2.556–4.037)	< 0.001	2.127(1.561–2.896)	< 0.001
P for trend	< 0.001		< 0.001		< 0.001	
VFA	2.106(1.778–2.495)	< 0.001	1.870(1.576–2.219)	< 0.001	1.578(1.317–1.890)	< 0.001
Quartile1	1	< 0.001	1	< 0.001	1	< 0.001
Quartile2	1.322(0.992–1.761)	0.056	1.389(1.041–1.853)	0.026	1.266(0.940–1.704)	0.12
Quartile3	1.627(1.235–2.144)	0.001	1.577(1.195–2.081)	0.001	1.360(1.020–1.815)	0.036
Quartile4	3.274(2.548–4.207)	< 0.001	2.863(2.224–3.686)	< 0.001	2.259(1.730–2.950)	< 0.001
P for trend	< 0.001		< 0.001		< 0.001	

Abbreviations: BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; VFA, visceral fat area; OR, odds ratio; CI, confidence interval

Model 1: adjustments for sex and age

Model 2: adjustments for Model 1 variables plus HbA1c, hyperlipidemia, hypertension, smoking status, drinking status, and eGFR

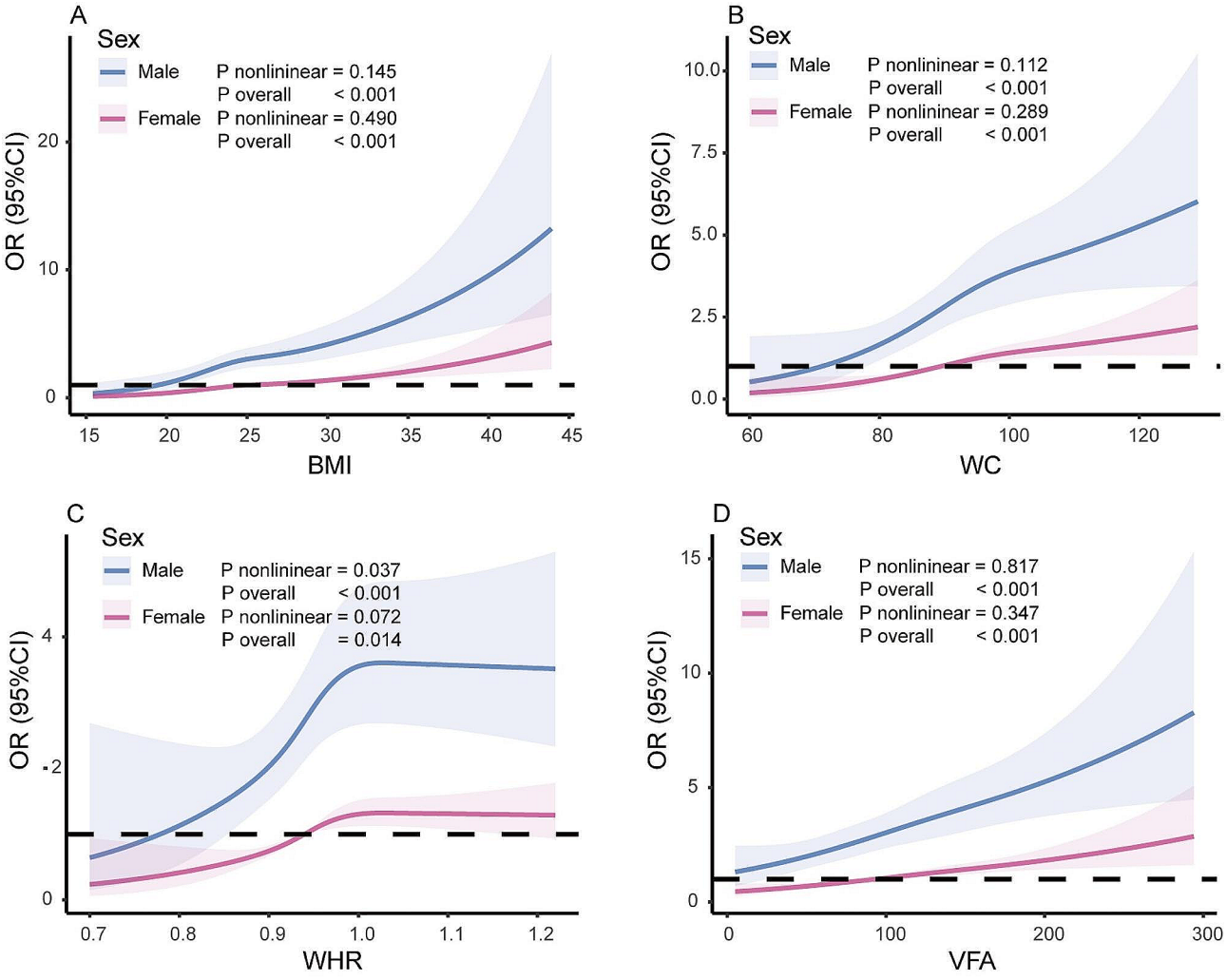

Fig. 2 RCS analysis of the relationships between obesity indices (BMI, WC, WHR, and VFA) and gout prevalence. The solid line represents the OR, with the shaded area representing the 95% CI. Adjustments were made to the model for age, HbA1c, hyperlipidemia, hypertension, smoking status, drinking status, and eGFR

Discussion

To our knowledge, this is the first study to investigate the relationship between different obesity indicators (BMI, WC, WHR, and VFA) and gout in T2DM. In this study, associations were observed between increased risk of gout and general obesity, abdominal obesity, and VFO in patients with T2DM. Levels of obesity indicators (BMI, WC, WHR, and VFA) were markedly higher in patients with gout compared to those without. Lastly, ROC curves indicated increased—although non-significantly—AUCs for indicators of abdominal obesity (WC and WHR) compared to indicators of general obesity (BMI) and VFO (VFA), suggesting similar predictive abilities of these parameters for the development of gout.

Significant associations have been reported between obesity, hyperuricemia, and gout incidence. A study of 29,310 participants reported a 5% increase in the incidence of gout for every unit increase in BMI [8]. According to a 2018 meta-analysis, BMI values of 30 kg/m^2 were linked to a 2.24-fold increased risk of developing gout [7]. A health professional follow-up study revealed that weight gain and adiposity are linked with elevated gout risk in male patients, while weight reduction was found to be preventive [6]. A study of patients with obesity in Sweden showed that, over 26 years of follow-up, gout incidence was reduced by 40% after bariatric surgery [23]. Guo et al.'s study [24] revealed an interesting finding that WHR and UA target achievement were correlated, although BMI was not. Furthermore, abdominal obesity has been found to be more common in patients with gout [25]. Choi et al. [26]. reported a gout risk of 1.82 (95% CI 1.39–2.39; P for trend < 0.001) in males in the top WHR quintile (0.98–1.39) relative to the bottom quintile (0.70–0.88). However, Takahashi et al. [27]. contended that increased visceral fat might be more detrimental to UA metabolism than BMI, suggesting

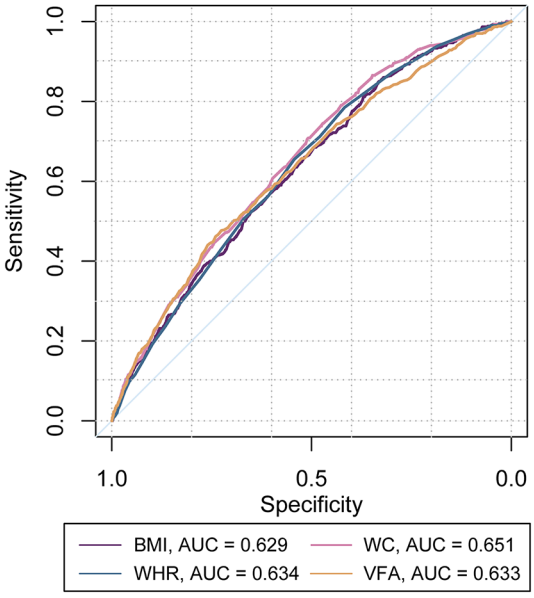

Fig. 3 ROC curves for obesity indices (BMI, WC, WHR, and VFA) for gout prediction. ROC, receiver operating characteristic; BMI, body mass index; WC, waist circumference; WHR, waist-to-hip ratio; VFA, visceral fat area

that visceral rather than subcutaneous fat is linked to metabolic anomalies and hyperuricemia in gout [28]. According to a health examination study, VFO was more common in individuals with gout compared to healthy controls [21]. However, the aforementioned studies may havecertain shortcomings. These include the comparatively smaller sample size, the fact that only the general population was taken into account, rather than the so-called highly exposed population groups, and the absence of a comparative study of several obesity indicators. In contrast to other research, the focus of this study was on the relationship between indicators of various types obesity with gout in patients with T2DM.

The exact underlying processes of obesity in gout development remain unknown. It has been found that obesity influences gout by augmenting serum UA levels. Obesity, especially that associated with abdominal fat, has been linked to hyperuricemia, possibly resulting from increased urate synthesis and decreased renal clearance [29]. Increased body fat leads to increased total nucleic acid metabolism, which in turn promotes UA production via purine metabolism [30]. Moreover, obesity may cause abnormalities in glomerular hemodynamics and hyperactivate the renin-angiotensin-aldosterone pathway, resulting in obesity-linked nephropathy. Long-term exposure to these effects may lower renal UA excretion by inducing glomerular atherosclerosis [31]. The excess adipose tissues of obesity produce pro-inflammatory cytokines that reduce antioxidant levels and raise reactive oxygen species (ROS) and nitrogen species produced by macrophages and monocytes. As a result, this exacerbates the impact of urate excretion dysfunction, leading to urate resorption disequilibrium and ultimately to hyperuricemia and gout risk [32]. Additionally, it has been shown that the onset of hyperuricemia is correlated with several adipocytokines linked to obesity, including adiponectin and leptin [33, 34].

Hyperuricemia and gouty arthritis are interrelated because white blood cells phagocytize MSU crystals [35]. MSU crystals can interact with Toll-like receptors and interleukin-1(IL-1) receptors on macrophage surfaces, causing the NOD-like receptor family protein 3 (NLRP3) inflammasome to produce IL-1 and promote neutrophil and macrophage influx [35]. It has been demonstrated that obesity increases proinflammatory molecules, such as interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α) [36, 37]. This intensifies inflammation brought on by obesity. Furthermore, previous studies have shown that soluble urate can promote NLRP3 inflammasome activation [38] and act as an alum to stimulate inflammation in an adjuvant capacity [35]. B cell antigen presentation to CD4 and CD8 T cells furthers the pathogenic function of IgM/IgG antibodies by facilitating urate crystallization, which in turn causes MSU crystals to be phagocytosed and the NLRP3 inflammasome to be sensed [39]. In addition, comorbidities related to obesity may raise the incidence of gout [40, 41].

This study has several limitations. First, causal links cannot be inferred owing to the cross-sectional design. Future studies should examine the longitudinal relationship between gout and obesity indicators. Second, by employing statistical adjustments, our study reduced other risk factors; however, unobserved confounders, such as alcohol intake and history of pertinent medication use, persisted. This limitation may have affected the robustness and reliability of our findings. Third, although we included multiple indicators of obesity for our study, there are also some better indicators associated with obesity and metabolic disease, such as body roundness index, triglyceride glucose index-waist circumference [42, 43]. Future study could be conducted on these indicators. Fourth, our study is the lack of data on specific endocrine factors such as plasma aldosterone concentrations. Recent research has highlighted the potential dual impact of elevated aldosterone levels on both hyperuricemia and gout risk in hypertensive patients [44]. Aldosterone, a hormone pivotal in blood pressure regulation, may contribute to the development of gout through its effects on uric acid metabolism and renal excretion. The exclusion of such endocrine data may have limited our ability to fully elucidate the mechanisms linking obesity with gout, particularly in the context of metabolic and cardiovascular comorbidities. Future studies should consider incorporating measurements of aldosterone and other related hormones to provide a more comprehensive

understanding of the underlying pathophysiology. Finally, our investigation was limited to Chinese populations, which may restrict the general extrapolation of our findings. Further research on various ethnic groups is needed to determine the relationships between obesity and gout risk.

Conclusions

Obesity, particularly abdominal fat, contributes to the occurrence of gout. Obesity and gout risk were found to be significantly and positively linked in patients with T2DM. But uncovering the causality behind the relationship requires further prospective study. Obesity indicators (BMI, WC, WHR, and VFA) show potential predictive ability for gout development. Avoiding excessive gain in weight, WC, and VFA maybe an effective approach to prevent gout in patients with T2DM.

Abbreviations

MSU	Monosodium urate
T2DM	Type 2 diabetes mellitus
BMI	Body mass index
WC	Waist circumference
WHR	Waist-to-hip ratio
VFA	Visceral fat area
VFO	Visceral fat obesity
H	Height
W	Weight
WC	Waist circumference
HC	Hip circumference
DBP	Diastolic blood pressure
SBP	Systolic blood pressure
UN	Urea nitrogen
Scr	Serum creatinine
UA	Uric acid
FBG	Fasting blood glucose
HbA1c	Glycated hemoglobin
ALT	Alanine transaminase
AST	Aspartate aminotransferase
TG	Triglyceride
TC	Total cholesterol
LDL	C-Low-density lipoprotein cholesterol
HDL	C-High-density lipoprotein cholesterol
eGFR	Estimated glomerular infiltration rate

Supplementary Information

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Supplementary Material 1

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Author contributions

YYW and NYC: Concept and design. PF and QDZ: Data acquisition and analysis. NYC and MDC: Interpretation of data and drafting of the manuscript. XPZ, SQY and ZBZ: Critical manuscript revision. All authors contributed to the manuscript, and read and approved the submitted version.

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Data availability

The datasets of the study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

The study protocol was approved by the Ethics Committees of Yuhuan Second People's Hospital and Taizhou Central Hospital (Taizhou University Hospital). All participants provided written informed consent.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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