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Association between weight-adjusted waist index and carotid atherosclerotic plaque in patients with type 2 diabetes in the Chinese population

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Abstract

Purpose The purpose of this study is to investigate the relationship between the weight-adjusted waist index (WWI) and the incidence of carotid atherosclerotic plaque in patients with type 2 diabetes (T2DM) in the Chinese population.

Methods A retrospective cross sectional analysis was conducted on data from 801 adult patients from May 2018 to January 2024. Spearman's correlation analysis was used to determine the correlation between WWI and carotid atherosclerotic plaque and analyzed the factors influencing carotid atherosclerotic plaque through binary logistic regression. Additionally, the area under the receiver operating characteristic (ROC) curve (AUC) was calculated to analyze the optimal cut-off point for WWI to predict carotid atherosclerotic plaque.

Results Compared with the non-carotid atherosclerotic plaque group, the incidence of hypertension, systolic blood pressure, in the carotid atherosclerotic plaque group were higher than in the non-carotid plaque group($P < 0.05$). Correlation analysis showed that WWI was positively correlated with carotid atherosclerotic plaque ($r = 0.263$)($P < 0.05$). Binary logistic regression analysis showed that WWI was an independent risk factor for carotid atherosclerotic plaque in patients with T2DM. The ROC curve analysis for the WWI yielded an AUC of 0.65 (95% CI = 0.611–0.69, $P < 0.05$) for predicting the presence of carotid atherosclerotic plaque.

Conclusion WWI was independently associated with the occurrence of carotid atherosclerotic plaque in patients with T2DM. Given its simplicity and widespread use, WWI emerges as a novel and practical predictor for assessing the risk of developing carotid atherosclerotic plaque in Chinese patients with T2DM.

Keywords Weight-adjusted waist index, Carotid atherosclerotic plaque, Type 2 diabetes

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Introduction

The prevalence of type 2 diabetes (T2DM) in China has gradually increased in recent years, reaching 11.2% between 2015 and 2017. However, the diabetes awareness rate (36.5%), treatment rate (32.2%), and control rate (49.2%) remain low [1]. Atherosclerotic cardiovascular disease (ASCVD) is a major macrovascular complication of T2DM that has become the leading cause of mortality and disability in patients with T2DM [2, 3], and its primary mechanism is atherosclerosis [4]. Carotid atherosclerotic plaque is considered a target in assessing ASCVD, and it has been proven to be an independent predictor of future cardiovascular risk [5]. Early detection and prediction of carotid plaque is therefore critical [6]. Previous studies have shown that obesity is a major risk factor for diabetes [7]. Relative to the risk of increased subcutaneous fat, an increase in visceral fat constitutes an independent risk factor for cardiovascular events [7, 8]. The weight-adjusted waist index (WWI), first proposed by Park et al. [9], is a novel body measurement index that normalizes waist circumference (WC) with respect to body weight. Compared with other body measurements, such as the visceral adiposity index (VAI), lipid accumulation product (LAP), and Chinese visceral adiposity index (CVAI) [10], WWI is simple and easy to obtain, allowing for a quantitative evaluation of fat and muscle mass [11]. Previous studies have shown that WWI is closely related to the risk of hypertension [12], osteoporosis [13], stroke [14], urinary protein [15], hyperuricemia [16], diabetes [17], cardiovascular disease [18], and non-alcoholic fatty liver disease (NAFLD) [19, 20]. However, a relationship between WWI and carotid atherosclerotic plaque in T2DM within the Chinese population has not been reported. Thus, we herein investigated the relationship between WWI and carotid atherosclerotic plaque in Chinese patients with T2DM.

Materials and methods

Study participants

We conducted a retrospective analysis on data from 801 adult patients with Type 2 diabetes mellitus (T2DM) admitted to the Department of Endocrinology at the Affiliated Hospital of Jiangsu University. Of these, 356 were males and 445 were females. To be eligible for inclusion, patients had to meet the diagnostic criteria established by the American Diabetes Association [21]. The exclusion criteria were individuals with (1) other types of diabetes; (2) acute and severe complications of diabetes; (3) severe chronic infection; (4) tumors; (5) severe liver or kidney disease; (6) autoimmune diseases; (7) smoking; and (8) cardiovascular and cerebrovascular diseases. In addition, T2DM patients on oral lipid-lowering drugs, anticoagulants, or antiplatelet drugs were excluded to reduce the influence of these medications on carotid atherosclerotic plaque.

Collection of clinical and biochemical data

Information was collected on patients regarding age, sex, hypertension, drinking history, medication history, and other primary clinical data.Patients were examined in light clothing, and systolic and diastolic blood pressure were measured after 15 min of rest. Professionally trained personnel then measured height, weight, neck circumference (NC), waist circumference (WC), and hip circumference (HC).

After fasting for eight hours, an oral glucose tolerance test (OGTT) test was performed to detect fasting blood glucose (FPG), fasting plasma insulin (FINS), C-peptide, 2-h postprandial plasma glucose (2hPG), 2-h postprandial insulin (2hINS), and 2-h C-peptide. We determined glucose levels with the glucose oxidase method. Chemiluminescence was employed to detect insulin and C-peptide. Liver and kidney function, blood lipids, and urinary microalbumin/creatinine were analyzed using a BECKMAN AU5800 automatic biochemical analyzer. Glycosylated hemoglobin (HbA1c) was evaluated through high-performance liquid chromatography (HPLC). Our calculations were as follows:

Homeostasis model assessment of insulin resistance (HOMA-IR) = FPG (mmol/L) × FINS (μIU/ml)/22.5;

WHtR = WC/height;

Body mass index (BMI) = weight (kg)/height $(m)^2$.

For males:

VAI = WC (cm) / (39.68 + 1.88 ×BMI)×(triacylglycerol [TG] [mmol/L]/1.03) × (1.31/high-density lipoprotein cholesterol [HDL-C][mmol/L]);

LAP = (WC [cm] – 65) × TG (mmol/L);

CVAI = -267.93 + 0.68 × year + 0.03 ×BMI (kg/m2) + 4.00 × WC (cm) + 22.00 × lgTG (mmol/L) – 16.32 × HDL-C (mmol/L).

For females:

VAI = WC (cm) / (36.58 + 1.89 ×BMI) × (TG [mmol/L]/0.81) × (1.52 /HDL-C [mmol/L]);

LAP = (WC [cm] – 58) × TG (mmol/L).

CVAI = -187.32 + 1.71 × year + 4.23 × BMI (kg/m2) + 1.12 × WC (cm) + 39.76 × lgTG (mmol/L) – 11.66 × HDL-C (mmol/L).

WWI was determined by dividing WC (cm) by the square root of body weight (kg).

Assessment of carotid atherosclerotic plaque

Experienced sonographers evaluated all patients in strict accordance with the instrument manual, using a Toshiba Apilo 500 and GE Logic 7 ultrasonic instruments were used. During the analysis, the subject was positioned supine with a pillow at the back of the neck and the head tilted back to expose the neck. A carotid atherosclerotic

plaque was defined as local thickening of cIMT > 0.5 mm or > 50% of the surrounding IMT value, or thickness from the media-adventitia interface to the intima-luminal interface > 1.5 mm [22].

Statistical analysis

We applied SPSS 27.0 statistical software for data analysis. For normally distributed data, continuous variables were expressed as mean ± SD. one-way ANOVA was used for multi-group comparison, with the LSD-t test used for post hoc two-group comparisons. Non-normally distributed data are presented as median (P25, P75), with the Kruskal-Wallis H rank-sum test adopted for multi-group comparisons and the Mann–Whitney U test used to compare two groups. Pearson or Spearman correlation analysis was used to investigate the correlation between WWI and carotid atherosclerotic plaque. We exploited binary logistic regression analysis to explore the relationship between carotid atherosclerotic plaque and WWI. The receiver operating characteristic (ROC) curve and the area under the ROC curve (AUC) were used to assess the predictive value of WWI for carotid atherosclerotic plaque. All significance tests were two-tailed, with $P < 0.05$ serving as the threshold for statistical significance.

Results

Comparison of general characteristics between the two groups

All participants ($n = 801$) were divided into a non-carotid atherosclerotic plaque group and a carotid atherosclerotic plaque group. The prevalence of carotid atherosclerotic plaque among participants was 34.46%, and the mean age of the carotid plaque group was significantly greater than that of the non-carotid plaque group, and the duration of diabetes was also longer in the carotid plaque group. Additionally, the incidence rates of hypertension and NAFLD were higher in the carotid plaque group, and the systolic blood pressure was higher than that of the non-carotid plaque group. Biochemical indices such as serum creatinine and urea nitrogen were higher in the carotid plaque group, while FPG, C-peptide, and alanine aminotransferase (ALT) were lower in the carotid plaque group. Although WHtR, CVAI, and WWI were higher in the carotid atherosclerotic plaque group, NC, WC, HC, VAI, and LAP did not differ significantly between groups (Table 1).

Baseline characteristics according to WWI index tertiles

Our study population was divided into three groups according to WWI values. As shown in Table 2, age, DM duration, incidence of hypertension, SBP, BMI, WC, HC, WHtR, FINS, 2hINS, HOMA-IR, HOMA-β, CVAI, VAI, and LAP all increased significantly with rising WWI values ($p < 0.05$), while Scr diminished. With the rise in WWI, the prevalence of carotid plaques increased gradually and commensurately ($p < 0.05$). The prevalence rates of carotid atherosclerotic plaques in the three groups were 19.85%, 36.33%, and 47.19%, respectively.

Correlations between WWI and other parameters

WWI was positively correlated with age, diabetes course, hypertension, SBP, BMI, WC, HC, WHtR, FINS, 2hINS, C-peptide, HOMA-IR, CVAI, LAP, and the presence of carotid atherosclerotic plaques. Conversely, WWI was negatively correlated with alcohol consumption and serum creatinine ($P < 0.05$) (Table 3).

Binary logistic regression analysis of carotid atherosclerotic plaque in T2DM patients

We exploited a binary logistic regression model to assess WWI's effects on the risk of carotid atherosclerotic plaque in T2DM, with the dependent variable designated as the presence of carotid atherosclerotic plaque (non-carotid atherosclerotic plaque = 0; carotid atherosclerotic plaque = 1). Independent variables included age, BMI, hypertension, SBP, FPG, FINS, 2hINS, C-peptide, BUN, WWI, and HOMA-IR, and we conducted a binary logistic regression analysis. Our results revealed that WWI, SBP, age, hypertension, C-peptide, and BUN were independent risk factors for carotid atherosclerotic plaque in patients with T2DM (Table 4).

Assessment of the predictive value of WWI for carotid atherosclerotic plaque in T2DM

Finally, we analyzed the diagnostic value of WWI for identifying carotid atherosclerotic plaque in T2DM. ROC curves yielded an AUC of 0.65 (95% CI = 0.611–0.69, $P < 0.05$), indicating that WWI offered diagnostic value in predicting carotid atherosclerotic plaque in patients with T2DM. The optimal cut-off for WWI was determined to be 10.72, with sensitivity and specificity values of 78.62% and 44.38%, respectively (Table 5,Fig. 1).

Discussion

This study is the first investigation of the relationship between the WWI and carotid atherosclerotic plaque in the Chinese population with T2DM. Our analysis accounted for potential confounding factors, excluding the effects of medications and smoking on carotid atherosclerotic plaque. We ascertained that WWI in the carotid atherosclerotic plaque group was significantly higher than in the non-carotid atherosclerotic plaque group, and regression analysis revealed that WWI was related to carotid atherosclerotic plaque and served as a better predictor than the atherosclerotic plaque than CVAI, VAI, LAP, BMI, or WHtR. These findings imply that WWI can be used as a straightforward indicator to

Table 1 Baseline clinical characteristics of all patients

	Non-carotid plaque	carotid plaque	$Z/\chi^2/t$	*P*
Gender	525	276		
Male	236	120		
Female	289	156	0.159[b]	0.69
Age, (y)	52(40.5, 58)	61(55, 68)	−11.48	<0.001
DM duration, (mouth)	7(0, 81)	63(0.25, 150.75)	−6.052	<0.001
Drinking, n (%)	130(24.76%)	57(20.65%)	1.707[b]	0.191
Hypertension, n (%)	184(35.05%)	155(56.16%)	33.03[b]	<0.001
antihypertensive drugs	166(31.62%)	138(50%)	25.954[b]	<0.001
NAFLD	190(36.19%)	75(27.17%)	6.643[b]	0.01
SBP (mmHg)	125(114,137)	132(118,143)	−4.288	<0.001
DBP (mmHg)	76(69,83)	75(67,83)	−1.206	0.228
BMI (kg/m^2)	24.86(22.61,27.55)	24.74(22.5,27.02)	−1.286	0.198
NC (cm)	37(35, 40)	37(35, 39)	−1.012	0.312
WC (cm)	89(84, 96)	91(85, 96)	−1.412	0.158
HC (cm)	97(92, 102)	97(92, 100)	−0.967	0.334
WHtR	0.54(0.51, 0.58)	0.55(0.52, 0.59)	−3.301	<0.001
HbA1c (%)	9.8(8.15, 11.4)	9.5(7.9, 11.1)	−0.792	0.428
FPG (mmol/L)	10.12(8.06, 12.99)	9.6(7.34, 12.51)	−2.061	0.039
2hPG (mmol/L)	19.17 ± 5.13	19.2 ± 5.99	9.118[a]	0.946
FINS (μIU/ml)	7.19(4.24, 11.16)	6.69(4.16, 12.4)	−0.255	0.799
2hINS (μIU/mL)	25.97(15.13, 43.38)	28.93(16.26, 47.79)	−1.679	0.093
C-peptide (ng/mL)	2.44(1.69, 3.12)	2.29(1.49, 2.98)	−2.378	0.017
2hC-peptide (ng/mL)	4.64(3.24, 7.47)	4.48(3.01, 6.52)	−1.708	0.088
HOMA-IR	3.08(1.91, 5.05)	2.77(1.79, 5.33)	−0.583	0.56
HOMA-β	0.24(0.12, 0.42)	0.26(0.12, 0.53)	−1.49	0.136
ALT(U/L)	24.1(15.25, 44)	19.95(13.45, 30.98)	−4.075	<0.001
AST(U/L)	18.9(14, 28.45)	18(13.3, 24.68)	−1.904	0.057
TC (mmol/L)	4.89(4.28, 5.64)	5.03(4.37, 5.79)	−1.758	0.079
TG (mmol/L)	1.79(1.29, 2.64)	1.83(1.26, 2.68)	−0.093	0.926
HDL-C (mmol/L)	1.09(0.92, 1.35)	1.14(0.95, 1.35)	−1.318	0.187
LDL-C (mmol/L)	2.85(2.31, 3.51)	2.96(2.4, 3.62)	−1.641	0.101
BUN (mmol/L)	4.96(4.08, 5.94)	5.5(4.5, 6.62)	−5.149	<0.001
SCr (μmol/L)	53.7(44.7, 65)	56.9(46.33, 67.78)	−2.687	0.007
UA (μmol/L)	279.7(215, 350)	284(225, 344)	−0.514	0.607
CVAI	112.93 ± 38.75	124.83 ± 33.08	4.833[a]	<0.001
VAI	2.82(1.75, 4.71)	2.82(1.73, 4.71)	−0.064	0.949
LAP	50.88(31.22, 85.59)	54(35.24, 84.22)	−0.728	0.466
WWI	10.84(10.4, 11.29)	11.18(10.76, 11.62)	−6.999	<0.001

a is the *t* value, b is the $\chi 2$ value, and the rest is the *Z* value

NAFLD: non-alcoholic fatty liver disease; SBP: systolic blood pressure; DBP: diastolic blood pressure; BMI: body mass index; NC: neck circumference; WC: waist circumference; HC: hip circumference; WHtR: waist-to-height ratio; HbA1c: glycosylated hemoglobin c; FPG: fasting plasma glucose;2hPG: 2-hour postprandial plasma glucose; FINS: fasting plasma insulin;2hINS: 2-hour postprandial plasma insulin; HOMR-IR: homeostasis model assessment-insulin resistance index; HOMA-β:homeostatic model assessment of β-cell function; ALT: alanine aminotransferase; AST: aspartate aminotransferase; TC: total cholesterol; TG: triglyceride; HDL-c: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; BUN: blood urea nitrogen; Scr: serum creatinine; UA: uric acid; CVAI: Chinese visceral adiposity index; VAI: visceral adiposity index; LAP: lipid accumulation product; WWI: weight-adjusted waist circumference index

assess the risk of carotid atherosclerotic plaque development in Chinese patients with type 2 diabetes.

Obesity is a chronic disease characterized by the accumulation of subcutaneous and visceral fat, which independently increases the risk of cardiovascular disease [23]. While BMI has traditionally been used as an indicator of obesity, studies show different conclusions concerning its association with carotid atherosclerotic plaque. A meta-analysis of prospective observational studies involving 900,000 adults showed an approximate log-linear relationship, with mortality rates from cardiovascular and cerebrovascular diseases significantly increasing with each standard deviation rise in BMI above 25 [24]. Gruberg et al. reported that patients with a BMI < 18.5 or normal BMI after coronary stent intervention exhibited a higher mortality rate within one year compared with overweight or obese patients, leading to the concept of an "obesity paradox" [25]. This area

Table 2 Baseline characteristics of study population across WWI tertiles

	9.11–10.67(267)	10.67–11.24(267)	11.24–13.34(267)	*P*
Gender	267	267	267	
Male	160	125	71	
Female	107	142	196	<0.001
Age, y	50(39,57)	55(46,61)	61(53,67)	<0.001
DM duration, mouth	13(0,90)	26(0,103)	36(0,126)	0.016
Drinking, n (%)	85(31.83%)	62(23.22%)	40(14.98%)	<0.001
Hypertension, n (%)	77(28.84%)	120(44.94%)	142(53.18%)	<0.001
antihypertensive drugs	65(24.34%)	104(38.95%)	135(50.56%)	<0.001
NAFLD	90(33.71%)	86(32.21%)	89(33.33%)	0.929
SBP (mmHg)	125(112,136)	128(116,138)	130(118,143)	<0.001
DBP (mmHg)	75(69,82)	76(69,84)	76(67,83)	0.319
BMI (kg/m^2)	24.33(22.15,26.87)	24.41(22.66,27.05)	25.56(23.07,27.68)	0.002
NC (cm)	37(35,40)	37(35,39)	37(34,40)	0.563
WC (cm)	85(80,91)	89(85,95)	95(90,100)	<0.001
HC (cm)	95(91,100)	97(93,101)	99(94,104)	<0.001
WHtR	0.51(0.48,0.54)	0.54(0.52,0.57)	0.59(0.56,0.63)	<0.001
HbA1c (%)	9.6(7.7,11.3)	9.8(8.1,11.3)	9.6(8.3,11.2)	0.38
FPG (mmol/L)	9.9(7.8,13.1)	10.3(7.8,12.8)	9.7(7.6,12.5)	0.368
2hPG (mmol/L)	18.8 ± 5.81	19.4 ± 5.14	19.4 ± 5.35	0.344
FINS (μIU/ml)	6.24(3.67, 10)	6.72(4.21, 10.86)	8.88(5.21,13.79)	<0.001
2hINS (μIU/mL)	21.74(13.37,37.6)	26.05(15.2,40.45)	37.34(20.71,56.04)	<0.001
C-peptide (ng/mL)	2.31(1.57,2.99)	2.37(1.62,3.06)	2.49(1.67,3.17)	0.145
2hC-peptide (ng/mL)	4.51(2.87,6.88)	4.49(3.28,6.83)	4.74(3.43,7.49)	0.223
HOMA-IR	2.85(1.55,4.47)	2.97(1.82,4.79)	3.48(2.14,6.17)	<0.001
HOMA-β	0.2(0.11,0.38)	0.22(0.11,0.41)	0.29(0.15,0.56)	<0.001
ALT(U/L)	21.6(14.1,40.5)	21.5(15,39.1)	22.5(14.2,38.8)	0.967
AST(U/L)	18.2(13.2,26.3)	17.9(13.7,27.1)	18.7(14.1,28)	0.276
TC (mmol/L)	4.79(4.25,5.6)	5(4.36,5.69)	5.01(4.4,5.75)	0.104
TG (mmol/L)	1.73(1.23,2.64)	1.77(1.26,2.68)	1.85(1.35,2.64)	0.716
HDL-C (mmol/L)	1.1(0.92,1.32)	1.08(0.9,1.36)	1.15(0.96,1.37)	0.193
LDL-C (mmol/L)	2.78(2.3,3.52)	2.95(2.33,3.56)	2.97(2.39,3.58)	0.311
BUN (mmol/L)	5(4.17,6.05)	5.23(4.29,6.24)	5.19(4.2,6.2)	0.447
SCr (μmol/L)	57.3(47.2,68.1)	54.9(44.9,66.1)	52.6(43.4,61.9)	0.001
UA (μmol/L)	282(229,351)	277(219,346)	285(212,345)	0.825
CVAI	100.31 ± 37.25	117.59 ± 32.65	133.2 ± 34.47	<0.001
VAI	2.51(1.47,4.6)	2.81(1.85,4.61)	3.26(1.97,4.93)	0.007
LAP	39.95(24.64,72.57)	50.49(32.24,80.29)	64.6(46.11,97.76)	<0.001
carotid plaque	53(19.85%)	97(36.33%)	126(47.19%)	<0.001

of research continued to expand, and Park, Galal, and others also subsequently confirmed this view [26–28]. Importantly, BMI does not distinguish between fat and lean body mass, while muscle mass and visceral fat exert opposite effects on mortality [29]. As a novel obesity-related indicator, WWI combines the advantages of WC while weakening the correlation with BMI. WWI is positively correlated with total fat mass and abdominal fat mass, but negatively correlated with the skeletal muscle mass of limbs [9, 30]. Thus, WWI can more accurately reflect "true obesity" that is metabolically unhealthy.

WWI was significantly higher in the carotid atherosclerotic plaque group in our study. According to our tripartite classification of WWI, we noted that as WWI increased in each tertile, the incidence of carotid atherosclerotic plaque increased significantly (19.85%, 36.33%, and 47.19%, respectively). Moreover, patient age, disease duration, and incidence of hypertension also increased gradually. Although there was no significant difference in the incidence of NAFLD [31, 32], obesity and insulin resistance-related indicators such as NC, WC, HC, WHtR, BMI, and HOMA-IR were elevated. Additionally, a study conducted by Abolnezhadian found that WWI was significantly higher in a metabolically unhealthy group compared to a metabolically healthy group, even when controlling for obesity levels [33]. Correlation analysis showed that WWI was positively correlated with carotid plaque, and ROC curve results suggested

Table 3 Correlation of WWI with other parameters

Variables	*r*	*P*
Gender	−0.313	<0.001
Age, y	0.384	<0.001
DM duration, mouth	0.144	<0.001
Drinking, n (%)	−0.201	<0.001
Hypertension, n (%)	0.207	<0.001
SBP (mmHg)	0.151	<0.001
DBP (mmHg)	−0.031	0.382
BMI (kg/m^2)	0.121	<0.001
NC (cm)	−0.033	0.356
WC (cm)	0.452	<0.001
HC (cm)	0.171	<0.001
WHtR	0.696	<0.001
HbA1c (%)	0.048	0.174
FPG (mmol/L)	−0.013	0.374
2hPG (mmol/L)	0.066	0.061
FINS (μIU/ml)	0.129	<0.001
2hINS (μIU/mL)	0.201	<0.001
C-peptide (ng/mL)	0.07	0.049
2hC-peptide (ng/mL)	0.052	0.138
HOMA-IR	0.119	<0.001
HOMA-β	0.054	0.126
ALT(U/L)	−0.039	0.276
AST(U/L)	0.017	0.628
TC (mmol/L)	0.056	0.115
TG (mmol/L)	−0.048	0.175
HDL-C (mmol/L)	0.065	0.066
LDL-C (mmol/L)	0.063	0.077
BUN (mmol/L)	0.016	0.657
SCr (μmol/L)	−0.097	0.006
UA (μmol/L)	−0.031	0.377
CVAI	0.402	<0.001
VAI	0.038	0.28
LAP	0.1	0.005
carotid plaque	0.263	<0.001
NAFLD	−0.013	0.708

an increased risk of carotid plaque for individuals with a WWI greater than 10.72.

The statistical results of our study demonstrated that the age, disease duration, incidence of hypertension, incidence of NAFLD [34] and systolic blood pressure of patients in the carotid plaque group were significantly higher than in the non-carotid plaque group, which aligned with the results of Chen et al. [35]. Long-term obesity, hypertension, hyperglycemia, and insulin resistance contribute to the onset and progression of atherosclerosis through various mechanisms, including vascular endothelium damage, oxidative stress, and inflammation [36]. In addition to these known mechanisms, recent research has recognized adipose tissue as a vital endocrine organ that produces "adipokines" that aggravate inflammation and accelerate the progression of atherosclerosis [37].

This study has several limitations. First, due to its cross-sectional nature, we could not establish a causal relationship between WWI and carotid plaque. Second, although we eliminated smoking, medication use, and several other influencing factors, we could not completely rule out the potential influence of other confounding factors. Third, the specificity of WWI is not high, which may be due to our limited sample size. Therefore, it is necessary to further collect more samples, expand the study, and design a prospective, multi-center study. Nevertheless, our results still showed that WWI was closely related to the occurrence and progression of carotid plaque. In addition, WWI is easily obtained and applicable to different populations. For individuals with high WWI, we recommend early assessment of target organ damage and timely intervention to reduce the risk of cardiovascular disease and improve prognosis.

Conclusions

WWI was independently associated with the occurrence of carotid atherosclerotic plaque in patients with type 2 diabetes. We posit that WWI can be used as a simple

Table 4 Linear regression analysis to investigate the influencing factors of WWI

Variables	β	SE	Wald χ2value	*P* value	OR	95%CI
WWI	0.596	0.134	19.761	<0.001	1.815	1.396–2.361
SBP	0.01	0.005	4.315	0.038	1.01	1.001–1.020
Age	1.077	0.179	36.164	<0.001	2.935	2.066–4.168
Hypertension	0.366	0.174	4.415	0.036	1.442	1.025–2.028
C-peptide	-0.192	0.073	6.833	0.009	0.826	0.715–0.953
BUN	0.195	0.053	13.73	<0.001	1.215	1.096–1.347
Constant	-9.696	1.611	36.201	<0.001	0.000	

Table 5 ROC curve analysis of WWI for risk assessment of carotid atherosclerotic plaque in T2DM

Variables	AUC	95% CI	Sensitivity (%)	Specifcity (%)	Cut-of value	Youden index
WWI	0.65	0.611–0.69	78.62	44.38	0.23	10.72
CVAI	0.596	0.556–0.637	44.93	71.43	0.16	131.49
WHtR	0.571	0.53–0.612	59.78	52.76	0.13	0.54

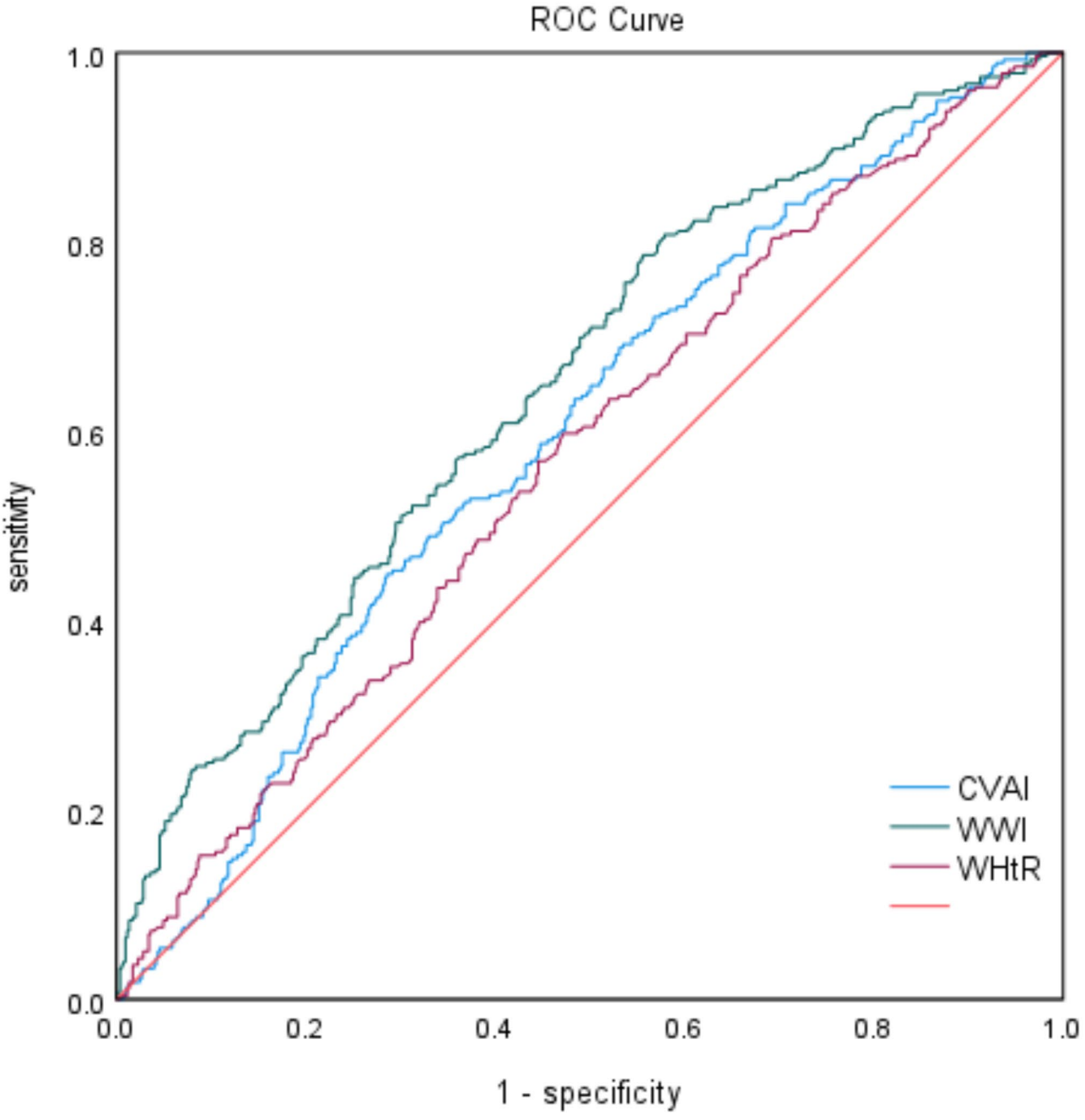

Fig. 1 ROC curves of WWI indices in predicting carotid atherosclerotic plaque risk in T2DM patients

indicator to assess the risk of developing carotid atherosclerotic plaque in Chinese patients with type 2 diabetes.

Author contributions
Y. Q. G.Y.Y.and D.X. conceived and designed the research, J.J. Y. AND X.N.W: collected the data, Y.X. adnd H.X.L: analyzed and interpreted the data, Y. Q. wrote the manuscript. L. Y: substantively revised it, G.Y.Y.and D.X. contributed to the discussion.All authors reviewed the manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

This study was approved by the Medical Ethics Committee of the Affiliated Hospital of Jiangsu University.Reference number: (SWYXLL20181217-1). All participants provided informed consent to participate.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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