Article

Discovery Medicine 2026; 38(211): 2449–2456
https://doi.org/10.24976/Discov.Med.202638211.193

Obesity and Insulin Resistance: Clinical Relevance Analysis of Multiple Metabolic Indicators in Insulin Resistance in Patients With Type 2 Diabetes

Feifei Li[1,*], Qiaofang Ke[1], Gaoyan Dai[1]

[1]Department of Endocrinology, Xianju County People's Hospital, 317300 Taizhou, Zhejiang, China
*Correspondence: 13736636081@163.com (Feifei Li)
Submitted: 8 May 2026 Revised: 8 June 2026 Accepted: 25 June 2026 Published: 24 August 2026

Background: Type 2 diabetes mellitus (T2DM) is one of the most common chronic metabolic diseases worldwide, and insulin resistance is the core pathophysiological basis for its development. This study aims to explore the relationship between various metabolic-related indicators and insulin resistance in patients with T2DM, and to analyze the correlation between obesity indicators, glucose and lipid metabolism indicators, and serum uric acid with insulin resistance, providing a reference for clinical identification of insulin resistance.

Methods: This was a single-center retrospective study, consecutively enrolling 263 patients with type 2 diabetes who received treatment at our center between August 2022 and August 2025. Patients were divided into a non-insulin-resistant group (n = 211) and an insulin-resistant group (n = 52) based on the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR). Baseline clinical data and laboratory parameters were collected, including body mass index, waist-to-hip ratio, fasting blood glucose, 2-hour postprandial blood glucose, lipid profile, glycated hemoglobin, fasting C-peptide, and serum uric acid. The triglyceride-glucose (TyG) index was also calculated. Logistic regression analysis was performed to screen for metabolic indicators associated with insulin resistance, and receiver operating characteristic (ROC) curve analysis was then conducted to evaluate the discriminative ability of these indicators for insulin resistance.

Results: Patients in the insulin resistance group had significantly higher levels of body mass index (BMI), waist-to-hip ratio (WHR), triglycerides, fasting blood glucose, TyG index, fasting C-peptide, glycated hemoglobin (HbA1c), systolic blood pressure, diastolic blood pressure, and serum uric acid than the non-insulin resistance group (all $p < 0.05$), and a higher proportion of patients with hypertension (65.38% vs. 46.45%, $p = 0.014$). Multivariate logistic regression analysis showed that WHR, TyG index, fasting C-peptide, and serum uric acid were independently associated with insulin resistance. The area under the curve (AUC) for assessing insulin resistance using the combined indicators was 0.81 (95% CI: 0.74–0.88), with a sensitivity of 79.0% and a specificity of 71.0%.

Conclusion: In patients with type 2 diabetes, WHR and serum uric acid levels are significantly correlated with insulin resistance status. These indicators reflect various metabolic characteristics from different perspectives, including obesity, glucose and lipid metabolism, and pancreatic β-cell function, and may provide preliminary insights into the metabolic abnormalities associated with insulin resistance in patients with type 2 diabetes.

Keywords: type 2 diabetes; insulin resistance; triglyceride-glucose index; metabolic indicators

Introduction

Type 2 diabetes mellitus (T2DM) is one of the most common chronic metabolic diseases worldwide. Its incidence has been rising in recent years, posing a serious threat to public health [1]. The occurrence of T2DM is closely related to a variety of metabolic abnormalities, among which insulin resistance (IR) is considered to be one of its core pathophysiological bases [2]. IR refers to the weakening of the biological response of peripheral tissues to insulin, which leads to a reduction in glucose uptake and utilization, thereby resulting in glucose metabolic dysfunction [3]. Long-term insulin resistance not only elevates blood glucose levels but also increases the compensatory workload of pancreatic β cells, eventually leading to β-cell dysfunction and promoting the development and progression of T2DM [4]. In addition to playing a key role in the development and progression of diabetes, IR is also closely related to a variety of cardiovascular metabolic diseases [5,6]. IR can promote atherosclerosis underlying mechanisms such as inducing chronic low-grade inflammatory response, enhancing oxidative stress levels, promoting lipid metabolism disorders, and damaging vascular endothelial function, thereby increasing the risk of cardiovascular events [7]. Thus, IR is a fundamental pathophysiological mechanism underlying glucose dysregulation and a major risk factor for cardio-

vascular diseases, warranting increased clinical attention. Previous research indicates that insulin resistance is commonly associated with obesity, dyslipidemia, and hypertension, and these metabolic disturbances are implicated in the development of metabolic diseases [8]. Therefore, investigating multiple metabolic factors associated with insulin resistance is of great significance for a deeper understanding of its metabolic characteristics.

Obesity is considered one of the most important risk factors for insulin resistance [9]. Numerous studies have shown that abdominal obesity is particularly associated with insulin resistance. Adipose tissue is not only an energy storage organ, but also an active endocrine organ that can secrete a variety of adipokines and inflammatory factors, such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6). These inflammatory mediators can interfere with insulin signaling, thereby reducing the body's sensitivity to insulin [10,11,12]. Therefore, adipose tissue dysfunction in obesity is considered to be closely associated with insulin resistance. In addition to obesity-related indicators, a variety of conventional metabolic indicators are also considered to be related to insulin resistance. For instance, dyslipidemia is a key contributor to insulin resistance, typically characterized by elevated triglycerides and reduced HDL cholesterol [13,14]. In addition, some composite metabolic indicators, such as the triglyceride-glucose index (TyG index), have also been recognized as key contributors to insulin resistance in recent years and proposed as a simple alternative indicator [15]. TyG can reflect insulin sensitivity to a certain extent and is associated with the risk of various diseases such as metabolic syndrome, cardiovascular disease and diabetic complications [16].

Although existing research suggests possible associations between various obesity-related and metabolic indicators and insulin resistance, results remain inconsistent across different studies. Prior research has mainly concentrated on the general population or individuals with metabolic syndrome, while evidence specifically in patients with T2DM remains limited. Furthermore, the diversity of metabolic indicators used across studies has resulted in inconsistent clinical relevance. Thus, a systematic assessment of common metabolic indicators in relation to insulin resistance in T2DM patients is essential to better characterize their metabolic profile and to develop practical clinical biomarkers.

Based on this, this study enrolled patients with T2DM as research subjects and systematically analyzed the association between metabolic indicators and insulin resistance by collecting various clinical and metabolic-related indicators, including obesity-related indices and lipid metabolism profiles. The aim was to explore the clinical relevance of various common metabolic indicators in insulin resistance in T2DM patients and provide a basis for more convenient identification of insulin resistance in clinical practice.

Methods

Participants

This is a single-center retrospective study conducted at Xianju County People's Hospital. Consecutive clinical data of patients with type 2 diabetes who received outpatient or inpatient treatment in the Department of Endocrinology at this center between August 2022 and August 2025 were collected. The study protocol followed the ethical principles of the Declaration of Helsinki and was approved by the Ethics Committee of Xianju County People's Hospital (approval number: 2026-Y-39). As this was a retrospective observational study, only patients' past medical records and laboratory test results obtained from routine clinical treatment were collected. No additional interventions were performed, and no additional risk or burden was imposed on patients. Furthermore, all patient information was anonymized, ensuring that individual identities could not be traced, thus effectively protecting patient privacy. This study was reviewed by the Ethics Committee and met the relevant ethical requirements for waiver of informed consent.

The inclusion criteria were: (1) age ≥18 years; (2) diagnosed with type 2 diabetes per the American Diabetes Association (ADA) criteria [17]; (3) clinical data including fasting blood glucose, 2-hour postprandial blood glucose, lipid profile, glycated hemoglobin and C-peptide and other related laboratory tests; (4) having complete basic demographic data and clinical indicator records, including body mass index (BMI) and waist-to-hip ratio (WHR).

The exclusion criteria were as follows: (1) type 1 diabetes, gestational diabetes, or other special types of diabetes; (2) acute metabolic disorders such as severe acute infection, diabetic ketoacidosis, or hyperosmolar hyperglycemia; (3) a previous diagnosis of severe liver failure, end-stage renal disease, or malignant tumors, or other diseases that may significantly affect metabolic status; (4) long-term use of glucocorticoids or other drugs that may significantly affect glucose and lipid metabolism; and (5) incomplete clinical data or missing key laboratory indicators. Patients who met the inclusion criteria and had no exclusion criteria were ultimately included in the final analysis.

Data Collection

Relevant clinical data were obtained by reviewing the hospital's electronic medical record system and laboratory information management system. Collected baseline data comprised demographic and clinical variables: age, sex, BMI, WHR, diabetes duration, smoking, drinking, hypertension, and other comorbidities. Blood pressure, including systolic blood pressure (SBP) and diastolic blood pressure (DBP), was also recorded at admission or during outpatient visits.

All laboratory indicators were derived from patients' routine clinical test results. Patients fasted for at least 8–12 hours before blood collection, and venous blood samples were collected the following morning for relevant biochemical indicator testing. Fasting blood glucose (FBG) and 2-hour postprandial plasma glucose (2-hour PPG) were measured using the hexokinase method on a fully automated biochemical analyzer. Total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C) were measured enzymatically on a fully automated biochemical analyzer. Serum uric acid (UA) levels were measured using the uricase method. Glycosylated hemoglobin (HbA1c) was measured using high-performance liquid chromatography (HPLC). Fasting C-peptide (FCP) was detected using chemiluminescent immunoassay.

The homeostasis model assessment of insulin resistance (HOMA-IR) was used to assess the insulin resistance of patients. According to the modified Homa formula proposed by Li et al. [18], HOMA-IR was calculated as: HOMA-IR = 1.5 + fasting blood glucose (mmol/L) × fasting C-peptide (pmol/L) / 2800. In addition, the TyG index was also calculated in this study, with the formula: TyG index = ln [triglycerides (mg/dL) × fasting blood glucose (mg/dL)/2]. The relationship between various metabolic-related indicators and insulin resistance was further evaluated using the above indicators. According to previous study, patients were divided into the non-insulin resistance group (HOMA-IR <2.31) and the insulin resistance group (HOMA-IR ≥2.31) based on the HOMA-IR levels [19]. The cutoff value of 2.31 was selected based on previous epidemiological studies conducted in Chinese adults and has been adopted in several studies involving Chinese populations. Although this threshold was not specifically established in patients with T2DM, it provides a pragmatic reference for stratifying insulin resistance in the present study. In the calculation of HOMA-IR, the fasting C-peptide values were originally measured in nmol/L as presented in Table 1. These values were converted to pmol/L by multiplying by 1000 (1 nmol/L = 1000 pmol/L) before applying the formula. For the TyG index, the fasting blood glucose and triglyceride values were originally measured in mmol/L (as shown in Table 1). They were converted to mg/dL using standard conversion factors: 1 mmol/L of glucose = 18 mg/dL, and 1 mmol/L of triglycerides ≈ 88.57 mg/dL.

Data Statistics and Analysis Methods

All statistical analyses were performed using SPSS 26.0 statistical software (IBM Corp., Armonk, NY, USA). First, normality was tested for continuous variables. Normally distributed continuous data were presented as mean ± SD ($\bar{x} \pm s$), and non-normally distributed data as median (interquartile range). Categorical variables were summarized as frequency and percentage (n, %). Participants were classified into insulin-resistant and non-insulin-resistant groups according to insulin resistance-related indicators. Between-group comparisons used independent-samples *t*-tests or Mann-Whitney U tests for continuous variables (depending on normality), and χ^2 tests for categorical variables.

To assess variables associated with IR, logistic regression analysis was conducted. Variables from baseline data were included in univariate analysis. The TyG index, a composite indicator combining fasting blood glucose and triglycerides, integrates information on glucose and lipid metabolism and is more representative than single blood glucose or lipid indicators; therefore, it was included as a core metabolic variable in the analysis. For hypertension-related factors, this study selected hypertension comorbidities (presence/absence) as a categorical variable in univariate analysis, rather than including both systolic and diastolic blood pressure. This was primarily because hypertension comorbidities reflect a patient's long-term hypertension status, with clear and stable clinical significance, while single blood pressure measurements are subject to short-term fluctuations influenced by factors such as emotions and physical activity at the time of visit. Variables with $p < 0.05$ in the univariate analysis were further included in a multivariate logistic regression model. Backward stepwise regression (exclusion criterion: $p > 0.10$) was applied to select variables, identify factors independently associated with insulin resistance, and calculate the odds ratio (OR) and its 95% confidence interval (95% CI). To facilitate a more clinically meaningful interpretation of the OR, the WHR was multiplied by 100 before being included in the logistic regression analysis; that is, each 1-unit increase in WHR corresponds to a 0.01 increase in the original waist-to-hip ratio. All other continuous variables were included in the analysis in their original units. All statistical tests were two-tailed, with $p < 0.05$ considered statistically significant.

Results

Baseline Data

This study ultimately included 263 patients with type 2 diabetes, who were divided into a non-insulin resistance group (n = 211) and an insulin resistance group (n = 52) based on their HOMA-IR levels. The baseline data of the two groups are shown in Table 1.

Regarding demographic characteristics, there were no statistically significant differences between the two groups in age, sex composition, duration of diabetes, smoking history, alcohol consumption history, other comorbidities, TC, HDL-C, LDL-C, or 2 h PPG (all $p > 0.05$). Compared with the non-insulin resistance group, the insulin resistance group showed significantly higher levels of BMI, WHR, SBP, DBP, TG, FBG, TyG index, FCP, HbA1c, UA, and a higher proportion of patients with hypertension (all $p < 0.05$).

Table 1. Comparison of baseline data between the two groups.

Variables	Non-insulin resistant group (n = 211)	Insulin resistance group (n = 52)	Statistic	*p*
Age (years)	57.14 ± 11.96	54.63 ± 12.05	$t = 1.35$	0.177
BMI (kg/m^2)	26.64 ± 2.72	27.78 ± 3.31	$t = –2.60$	0.010
WHR	0.91 (0.87, 0.95)	0.93 (0.90, 0.97)	Z = –3.48	<0.001
Diabetes course (years)	8.36 ± 2.94	8.11 ± 2.77	$t = 0.55$	0.581
LDL (mmol/L)	2.54 ± 0.47	2.67 ± 0.50	$t = –1.77$	0.079
HDL (mmol/L)	1.07 (0.91, 1.26)	1.06 (0.99, 1.15)	Z = –0.27	0.791
TC (mmol/L)	4.48 ± 1.13	4.59 ± 1.14	$t = –0.61$	0.539
TG (mmol/L)	1.43 ± 0.44	1.63 ± 0.59	$t = –2.40$	0.019
FBG (mmol/L)	5.87 ± 1.53	7.38 ± 1.91	$t = –5.30$	<0.001
2 h PPG (mmol/L)	16.64 ± 2.84	16.78 ± 2.58	$t = –0.31$	0.758
TyG, Mean ± SD	8.72 ± 0.43	9.06 ± 0.50	$t = –4.95$	<0.001
FCP (nmol/L)	1.01 (0.79, 1.21)	1.36 (0.81, 2.28)	Z = -3.46	<0.001
HbA1c (%)	7.75 ± 1.83	8.43 ± 1.81	$t = –2.41$	0.017
SBP (mmHg)	137.64 ± 17.78	143.27 ± 15.59	$t = –2.09$	0.037
DBP (mmHg)	86.21 ± 6.56	88.48 ± 7.12	$t = –2.19$	0.029
UA (μmol/L)	361.32 ± 102.44	440.70 ± 104.93	$t = –4.98$	<0.001
Gender, n (%)			$\chi^2 = 0.27$	0.600
Male	110 (52.13)	25 (48.08)		
Female	101 (47.87)	27 (51.92)		
Smoking, n (%)			$\chi^2 = 1.33$	0.249
No	154 (72.99)	42 (80.77)		
Yes	57 (27.01)	10 (19.23)		
Drinking, n (%)			$\chi^2 = 0.68$	0.411
No	177 (83.89)	46 (88.46)		
Yes	34 (16.11)	6 (11.54)		
Hypertension, n (%)			$\chi^2 = 5.99$	0.014
No	113 (53.55)	18 (34.62)		
Yes	98 (46.45)	34 (65.38)		
Other complications, n (%)			$\chi^2 = 0.08$	0.784
No	166 (78.67)	40 (76.92)		
Yes	45 (21.33)	12 (23.08)		

BMI, Body Mass Index; WHR, Waist-to-hip ratio; LDL, Low-density lipoprotein cholesterol; HDL, High-density lipoprotein cholesterol; TC, Total cholesterol; TG, Triglycerides; FBG, Fasting blood glucose; 2h PPG, 2-hour postprandial plasma glucose; TyG, Triglyceride-glucose index; FCP, Fasting C-peptide; HbA1c, Glycosylated hemoglobin; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; UA, Uric acid.

Univariate Regression Analysis

Univariate logistic regression analysis showed that BMI, WHR, TyG index, FCP, HbA1c, hypertension, and UA were significantly associated with insulin resistance (all $p < 0.05$). See Table 2 for detailed univariate analysis results.

Multivariate Logistic Regression Analysis

Multivariate analysis showed that WHR, TyG index, FCP, and UA independently associated with insulin resistance (all $p < 0.05$). Specifically, elevated WHR, TyG index, FCP, and UA levels were all significantly correlated with insulin resistance. The results of the multivariate logistic regression analysis are detailed in Table 3.

Discriminative Ability Assessment

Receiver operating characteristic (ROC) curves were plotted based on the independent factors identified by multivariate analysis, and the area under the curve (AUC) was calculated (Fig. 1). The combined use of the above indicators yielded an AUC of 0.81 (95% CI: 0.74–0.88), indicating good discriminative ability. The optimal cutoff value was determined to be 0.162 based on the Youden index, corresponding to a sensitivity of 79.0% and a specificity of 71.0%.

Discussion

IR is a central pathophysiological driver of T2DM onset and progression, contributing not only to glucose dysregulation but also to lipid abnormalities, obesity, and mul-

Table 2. Results of univariate regression analysis.

Variables	β	SE	Z	*p*	OR (95% CI)
Gender					
Male					1.00 (Reference)
Female	0.16	0.31	0.52	0.600	1.18 (0.64~2.16)
Smoking					
No					1.00 (Reference)
Yes	–0.44	0.38	–1.15	0.251	0.64 (0.30~1.37)
Drinking					
No					1.00 (Reference)
Yes	–0.39	0.47	–0.82	0.413	0.68 (0.27~1.72)
Hypertension					
No					1.00 (Reference)
Yes	0.78	0.32	2.41	0.016	2.18 (1.16~4.10)
Other complications					
No					1.00 (Reference)
Yes	0.10	0.37	0.27	0.784	1.11 (0.54~2.28)
Age	–0.02	0.01	–1.35	0.177	0.98 (0.96~1.01)
BMI	0.13	0.05	2.53	0.011	1.14 (1.03~1.27)
WHR	0.12	0.03	3.47	<0.001	1.12 (1.05~1.20)
Diabetes course	–0.03	0.05	–0.55	0.580	0.97 (0.87~1.08)
LDL	0.56	0.32	1.75	0.080	1.76 (0.93~3.31)
HDL	–0.27	0.72	–0.37	0.709	0.76 (0.19~3.13)
TC	0.08	0.14	0.62	0.538	1.09 (0.83~1.42)
2 h PPG	0.02	0.06	0.31	0.757	1.02 (0.91~1.13)
TyG	1.78	0.39	4.74	<0.001	5.91 (2.74~12.75)
FCP	1.65	0.32	5.07	<0.001	5.19 (2.75~9.81)
HbA1c	0.21	0.09	2.36	0.018	1.23 (1.04~1.47)
UA	0.007	0.002	4.559	<0.001	1.007 (1.004~1.010)

Table 3. Results of multivariate analysis.

Variables	β	SE	Z	*p*	OR (95% CI)
Hypertension					
No					1.00 (Reference)
Have	0.63	0.38	1.66	0.100	1.88 (0.89~3.96)
WHR	0.10	0.04	2.57	0.010	1.10 (1.02~1.19)
TyG	1.35	0.44	3.10	0.002	3.85 (1.64~9.04)
FCP	1.30	0.36	3.67	<0.001	3.68 (1.83~7.40)
UA	0.01	0.00	3.55	<0.001	1.01 (1.01~1.01)

tiple cardiovascular risk factors [2,5,8,20]. Investigating the associations between various metabolic indicators and insulin resistance (IR) in T2DM patients is crucial for understanding their metabolic profiles and clinical management. This single-center retrospective study analyzed clinical and biochemical data from 263 T2DM patients. Compared with non-IR patients, the IR group showed significant differences in obesity, glucose-lipid metabolism, and other metabolic indicators. Multivariate logistic regression further identified WHR, TyG index, FCP, and UA as independently associated with IR, suggesting their potential clinical relevance to metabolic abnormalities in T2DM.

Obesity, especially abdominal obesity, is considered an important factor in the development of insulin resistance [21]. The results of this study showed that the BMI and WHR of patients in the insulin resistance group were significantly higher than those in the non-insulin resistance group, and WHR remained significantly associated with insulin resistance in the multivariate analysis. These results suggest that abdominal fat distribution may be more closely related to insulin resistance than overall obesity indicators. As an important indicator of abdominal obesity, an increase in WHR primarily suggests abdominal fat accumulation, particularly an increase in visceral adipose tissue. Previous studies have shown that visceral adipose tissue contributes to insulin resistance through the secretion of adipokines and free fatty acids that interfere with insulin signaling [22,23,24]. Therefore, an increase in WHR may indicate an increase in visceral fat accumulation, which is closely related to insulin resistance.

This study also suggests a significant correlation between the TyG index and insulin resistance. Previous studies have shown that the TyG index has good agreement with HOMA-IR and is considered one of the simple alternative indicators for assessing insulin resistance [25,26,27]. The results of this study are consistent with previous studies.

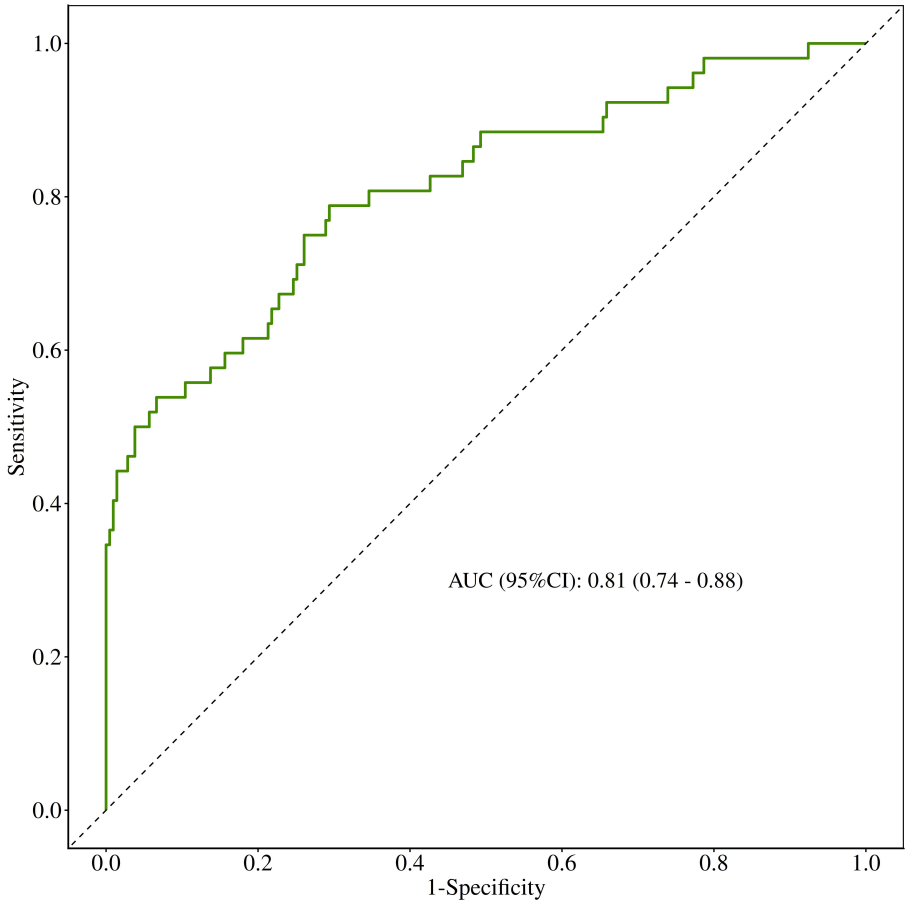

Fig. 1. ROC curve. ROC, receiver operating characteristic.

FCP is a byproduct of insulin secretion by pancreatic β cells, and its level can reflect the endogenous insulin secretion capacity [28]. In insulin resistance, the body often needs to compensate by increasing insulin secretion in order to maintain normal blood glucose levels, which is often accompanied by increased FCP levels [29]. In this study, FCP levels were significantly higher in the insulin resistance group than in the non-insulin resistance group and remained independently associated with insulin resistance in the multivariate analysis. This result suggests that in patients with T2DM, higher FCP may reflect a compensatory hyperinsulin secretion state, which is closely related to insulin resistance. Consistent with our findings, a study by Shajith Anoop et al. [30] also found that higher FCP levels were significantly associated with insulin resistance and visceral fat accumulation in patients with type 2 diabetes. Hyperuricemia is common in patients with obesity, metabolic syndrome and T2DM, and is closely related to insulin resistance [31]. Potential mechanisms include reduced renal uric acid excretion due to insulin resistance and uric acid-induced oxidative stress, which impairs insulin signaling [32,33,34]. Thus, our findings align with previous reports, indicating that serum uric acid may contribute to metabolic disturbances in T2DM patients. Regarding lipid indicators, triglyceride levels differed significantly between the two groups in this study, but no independent correlation was observed in the multivariate analysis. HDL-C, LDL-C, and TC also showed no significant differences between the groups. This may be because lipid levels are influenced by various factors, such as diet, medication use, and lifestyle. Therefore, a comprehensive analysis in the clinical context is still necessary when interpreting lipid indicator results.

Overall, this study suggests complex interrelationships among obesity-related indicators, glucose and lipid metabolism indicators, and metabolic-related indicators in patients with T2DM. Factors such as abdominal obesity, glucose and lipid metabolism disorders, and compensatory insulin secretion may be collectively associated with insulin resistance. A comprehensive assessment of these indicators provides a more complete understanding of the metabolic status of T2DM patients.

This study still has certain limitations. First, as a single-center retrospective study, selection bias and information bias may exist, limiting the generalizability of the findings. Second, the study did not include some indicators closely related to insulin resistance (such as fasting insulin and inflammatory factors), which may have affected the results. In addition, the HOMA-IR cutoff value of 2.31

was adopted from previous studies conducted in Chinese adult populations rather than being specifically validated in Chinese patients with T2DM. Therefore, the applicability of this threshold to all patients with T2DM should be interpreted with caution. Third, detailed information on glucose-lowering medications, statins, and urate-lowering therapy was not available in this retrospective study. Since such medications may influence levels of TG, UA, HbA1c, and insulin resistance, their potential confounding effects cannot be fully excluded. Future prospective studies with standardized medication documentation are warranted to further validate the findings. Fourth, detailed renal function markers (e.g., eGFR, Scr) were not collected in this retrospective study. Although patients with end-stage renal disease were excluded according to our criteria, mild-to-moderate renal impairment may influence uric acid levels and could potentially affect the observed associations. Future studies should incorporate renal function measures to further clarify the relationship between serum uric acid and insulin resistance. Finally, because this study was cross-sectional, causal relationships cannot be established. Prospective studies are therefore required to confirm these findings.

Conclusion

WHR, TyG index, FCP, and serum uric acid levels are significantly correlated with insulin resistance in patients with T2DM. These indicators reflect various metabolic characteristics, including obesity, glucose and lipid metabolism, and pancreatic β-cell function, and provide valuable insights into metabolic abnormalities in T2DM patients.

Availability of Data and Materials

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions

FFL designed the research study. FFL and GYD performed the research. QFK and FFL analyzed the data. FFL drafted the article. All authors contributed to important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study obtained approval from Xianju County People's Hospital (approval number: 2026-Y-39). All patient information was anonymized to protect privacy, and the study was approved by the Ethics Committee with a waiver of informed consent. The study protocol followed the ethical principles of the Declaration of Helsinki.

Acknowledgment

Not applicable.

Funding

This research received no external funding.

Conflict of Interest

The authors declare no conflict of interest.

References

[1] Rob A, Hoque A, Asaduzzaman MM, Khatoon MAA, Khalil R, Thomas D, et al. The global challenges of type 2 diabetes. Bangladesh Journal of Medicine. 2025; 36: 92–98.

[2] Accili D, Deng Z, Liu Q. Insulin resistance in type 2 diabetes mellitus. Nature Reviews. Endocrinology. 2025; 21: 413–426. https://doi.org/10.1038/s41574-025-01114-y

[3] Li M, Chi X, Wang Y, Setrerrahmane S, Xie W, Xu H. Trends in insulin resistance: insights into mechanisms and therapeutic strategy. Signal Transduction and Targeted Therapy. 2022; 7: 216. https://doi.org/10.1038/s41392-022-01073-0

[4] Berbudi A, Khairani S, Tjahjadi AI. Interplay Between Insulin Resistance and Immune Dysregulation in Type 2 Diabetes Mellitus: Implications for Therapeutic Interventions. ImmunoTargets and Therapy. 2025; 14: 359–382. https://doi.org/10.2147/ITT.S499605

[5] Kosmas CE, Bousvarou MD, Kostara CE, Papakonstantinou EJ, Salamou E, Guzman E. Insulin resistance and cardiovascular disease. The Journal of International Medical Research. 2023; 51: 3000605231164548. https://doi.org/10.1177/03000605231164548

[6] Ma W, Wang K, Sun L, Su F. Efficacy of Dapagliflozin in the Treatment of Type 2 Diabetes Mellitus Complicated by Coronary Heart Disease: A Meta-Analysis. British Journal of Hospital Medicine. 2025; 86: 1–15. https://doi.org/10.12968/hmed.2024.0336

[7] Szukiewicz D. Molecular Mechanisms for the Vicious Cycle between Insulin Resistance and the Inflammatory Response in Obesity. International Journal of Molecular Sciences. 2023; 24: 9818. https://doi.org/10.3390/ijms24129818

[8] Zhao X, An X, Yang C, Sun W, Ji H, Lian F. The crucial role and mechanism of insulin resistance in metabolic disease. Frontiers in Endocrinology. 2023; 14: 1149239. https://doi.org/10.3389/fendo.2023.1149239

[9] Gołacki J, Matuszek M, Matyjaszek-Matuszek B. Link between Insulin Resistance and Obesity-From Diagnosis to Treatment. Diagnostics (Basel, Switzerland). 2022; 12: 1681. https://doi.org/10.3390/diagnostics12071681

[10] Gupta A, Gupta P, Singh AK, Gupta V. Association of adipokines with insulin resistance and metabolic syndrome including obesity and diabetes. GHM Open. 2023; 3: 7–19. https://doi.org/10.35772/ghmo.2023.01004

[11] Fadaei R. Adipokines as a link between adipose tissue with inflammation and insulin resistance in cardiometabolic diseases. Acta Biochimica Iranica. 2023; 1: 112–118.

[12] Yan K. Recent advances in the effect of adipose tissue inflammation on insulin resistance. Cellular Signalling. 2024; 120: 111229. https://doi.org/10.1016/j.cellsig.2024.111229

[13] Ghani ZA, Qaddori H, Al-Mayah Q. Triglyceride/high-density lipoprotein ratio as a predictor for insulin resistance in a sample of healthy Iraqi adults. Journal of Medicine and Life. 2023; 16: 668–674. https://doi.org/10.25122/jml-2022-0239

[14] Lee YC, Lee JW, Kwon YJ. Comparison of the triglyceride glucose (TyG) index, triglyceride to high-density lipoprotein cholesterol (TG/HDL-C) ratio, and metabolic score for insulin resistance (METS-IR) associated with periodontitis in Korean adults. Therapeutic Advances in Chronic Disease. 2022; 13: 20406223221122671. https://doi.org/10.1177/20406223221122671

[15] Zheng Z, Yin X, Liu J, Zhang Y. Triglyceride-glucose index and its derivatives as emerging biomarkers of insulin resistance for prognostic evaluation of cardiovascular-kidney-metabolic syndrome: mechanisms and clinical applications. Frontiers in Pharmacology. 2026; 17: 1787130. https://doi.org/10.3389/fphar.2026.1787130

[16] Li HF, Miao X, Li Y. The Triglyceride Glucose (TyG) Index as a Sensible Marker for Identifying Insulin Resistance and Predicting Diabetic Kidney Disease. Medical Science Monitor : International Medical Journal of Experimental and Clinical Research. 2023; 29: e939482. https://doi.org/10.12659/MSM.939482

[17] American Diabetes Association Professional Practice Committee. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes-2024. Diabetes Care. 2024; 47: S20–S42. https://doi.org/10.2337/dc24-S002

[18] Li X, Zhou ZG, Qi HY, Chen XY, Huang G. Replacement of insulin by fasting C-peptide in modified homeostasis model assessment to evaluate insulin resistance and islet beta cell function. Zhong Nan Da Xue Xue Bao. Yi Xue Ban = Journal of Central South University. Medical Sciences. 2004; 29: 419–423.

[19] Chen L, Jia WP, Lu JX, Bao YQ, Wu YM, Jiang SY, et al. Epidemiological survey of metabolic syndrome among adults in Shanghai. Chinese Journal of Cardiology. 2003; 31: 284–286. (In Chinese)

[20] Tong Y, Xu S, Huang L, Chen C. Obesity and insulin resistance: Pathophysiology and treatment. Drug Discovery Today. 2022; 27: 822–830. https://doi.org/10.1016/j.drudis.2021.11.001

[21] Lu Wen, Cheng Chen, Keji Zhou. Relationship between Abdominal Obesity and Insulin Resistance, Growth Hormone, and Insulin-like Growth Factor-1 in Individuals with Type 2 Diabetes. Cellular and Molecular Biology (Noisy-le-Grand, France). 2022; 68: 36–41. https://doi.org/10.14715/cmb/2022.68.12.8

[22] Yalçın T, Oğuz SH, Bayraktar M, Rakıcıoğlu N. Anthropometric measurements and serum TNF-α, IL-6 and adiponectin in type 2 diabetes. Diabetology International. 2021; 13: 396–406. https://doi.org/10.1007/s13340-021-00553-y

[23] Rabe K, Lehrke M, Parhofer KG, Broedl UC. Adipokines and insulin resistance. Molecular Medicine (Cambridge, Mass.). 2008; 14: 741–751. https://doi.org/10.2119/2008-00058.Rabe

[24] Yazıcı D, Demir SÇ, Sezer H. Insulin Resistance, Obesity, and Lipotoxicity. Advances in Experimental Medicine and Biology. 2024; 1460: 391–430. https://doi.org/10.1007/978-3-031-63657-8_14

[25] Tahapary DL, Pratisthita LB, Fitri NA, Marcella C, Wafa S, Kurniawan F, et al. Challenges in the diagnosis of insulin resistance: Focusing on the role of HOMA-IR and Tryglyceride/glucose index. Diabetes & Metabolic Syndrome. 2022; 16: 102581. https://doi.org/10.1016/j.dsx.2022.102581

[26] Luo Y, Liu L, Liu M, Tang C, Liu H, Wang M, et al. Relationship Between Body Composition and Insulin Resistance Evaluated by the TyG Index: A Retrospective Study Among Chinese Population. Clinical Endocrinology. 2025; 102: 264–272. https://doi.org/10.1111/cen.15171

[27] Kim B, Kim GM, Huh U, Lee J, Kim E. Association of HOMA-IR Versus TyG Index with Diabetes in Individuals Without Underweight or Obesity. Healthcare (Basel, Switzerland). 2024; 12: 2458. https://doi.org/10.3390/healthcare12232458

[28] Chen J, Huang Y, Liu C, Chi J, Wang Y, Xu L. The role of C-peptide in diabetes and its complications: an updated review. Frontiers in Endocrinology. 2023; 14: 1256093. https://doi.org/10.3389/fendo.2023.1256093

[29] Jassim WE, Hussein AA. Insulin Resistance, Lipid Profile, C-Peptide, and Their Interplay in Type 2 Diabetes: A Basrah Population Study. Magazine of Al-Kufa University for Biology. 2024; 16.

[30] Anoop S, Misra A, Bhatt SP, Gulati S, Mahajan H. High fasting C-peptide levels and insulin resistance in non-lean & non-obese (BMI >19 to < 25 kg/m^2) Asian Indians with type 2 diabetes are independently associated with high intra-abdominal fat and liver span. Diabetes & Metabolic Syndrome. 2019; 13: 708–715. https://doi.org/10.1016/j.dsx.2018.11.041

[31] Asma Sakalli A, Küçükerdem HS, Aygün O. What is the relationship between serum uric acid level and insulin resistance?: A case-control study. Medicine. 2023; 102: e36732. https://doi.org/10.1097/MD.0000000000036732

[32] Liu R, Li Z, Zhang Y, Du M, Wang X, Zhang S, et al. Association of Serum Uric Acid with Indices of Insulin Resistance: Proposal of a New Model with Reference to Gender Differences. Diabetes, Metabolic Syndrome and Obesity : Targets and Therapy. 2024; 17: 3783–3793. https://doi.org/10.2147/DMSO.S481233

[33] Dikker O, Aktaş A, Şahin M, Doğan M, Dağ H. The Association of Serum Uric Acid Levels and Various Uric Acid-Related Ratios with Insulin Resistance and Obesity: A Preliminary Study in Adolescents. Children (Basel, Switzerland). 2023; 10: 1493. https://doi.org/10.3390/children10091493

[34] Sun H, Chang X, Bian N, An Y, Liu J, Leng S, et al. Adipose Tissue Insulin Resistance Is Positively Associated With Serum Uric Acid Levels and Hyperuricemia in Northern Chinese Adults. Frontiers in Endocrinology. 2022; 13: 835154. https://doi.org/10.3389/fendo.2022.835154