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ORIGINAL RESEARCH

Predictive Value and Correlation Study of HOMA2 IR-CP and TyG-BMI for Metabolic Dysfunction-Associated Steatotic Liver Disease in Patients with Type 2 Diabetes Mellitus

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Background and Objectives: To investigate the associations of C-peptide-based insulin resistance index (HOMA2 IR-CP) and triglyceride-glucose-body mass index (TyG-BMI) with metabolic dysfunction-associated steatotic liver disease (MASLD) in patients with type 2 diabetes mellitus (T2DM), identify independent risk factors for MASLD, construct a clinical predictive model, and evaluate its predictive performance.

Methods: A total of 311 T2DM patients were enrolled and randomly assigned to a derivation cohort (218 cases) and a validation cohort (93 cases) at a 7:3 ratio. Predictive factors were screened via LASSO regression, with results integrated with those from logistic regression analysis. A nomogram was developed, and model performance was assessed using receiver operating characteristic (ROC) curves, calibration curves, and decision curve analysis (DCA). Restricted cubic spline (RCS) regression was used to explore dose-response relationships, and stratified analyses and interaction analyses were performed.

Results: HOMA2 IR-CP and TyG-BMI as independent risk factors for MASLD in T2DM patients. The area under the curve (AUC) was 0.805 in the derivation cohort and 0.730 in the validation cohort. Calibration curves showed good agreement between predicted probabilities and actual outcomes, and DCA verified the model's substantial clinical net benefit. RCS analysis revealed a non-linear association between HOMA2 IR-CP and MASLD (P for non-linearity < 0.05), whereas TyG-BMI exhibited a linear association with MASLD (P for non-linearity = 0.139). No significant interaction effects were observed across subgroups stratified by gender, age, BMI, or HbA1c level; however, the associations were more prominent in patients with BMI < 28 kg/m^2 and those with HbA1c ≥ 7%.

Conclusion: HOMA2 IR-CP and TyG-BMI are independent risk factors for MASLD in T2DM patients. The nomogram model constructed based on these two factors exhibits good predictive performance and can provide a reference for MASLD risk assessment in T2DM patients.

Keywords: type 2 diabetes mellitus, metabolic dysfunction-associated steatotic liver disease, HOMA2 IR-CP, TyG-BMI, predictive model

Introduction

In recent years, the global prevalence and incidence of metabolic dysfunction-associated steatotic liver disease (MASLD) have been on a yearly upward trend, with the prevalence surging to as high as 37.8% post-2016.[1] Specifically, among overweight and obese populations, the prevalence rates of MASLD are remarkably elevated, standing at 70.0% and 75.3%,[2] respectively. Epidemiological data indicate that the global prevalence of MASLD in patients with type 2 diabetes mellitus (T2DM) is 65.0%,[3] and these patients present a higher incidence of significant liver fibrosis compared to non-diabetic counterparts.[4] As the liver functions as a central hub for regulating energy metabolism and glucose-lipid homeostasis, unhealthy lifestyle factors—including high-oil and high-fat dietary patterns and insufficient physical

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activity—can induce conditions such as obesity,[5] T2DM,[6] and metabolic syndrome (MetS),[7] all of which are major risk factors for MASLD. Cumulative evidence from multiple studies has confirmed that patients comorbid with MASLD and T2DM face an increased risk of developing cardiovascular and cerebrovascular diseases,[8] renal disorders,[9] and malignancies.[10] Consequently, the early detection and identification of MASLD are pivotal for its effective management and treatment. However, the lack of overt clinical symptoms in patients with early-stage MASLD, coupled with widespread patient reluctance to undergo invasive diagnostic modalities (such as liver biopsy) or costly examinations (such as magnetic resonance imaging), renders the accurate assessment of MASLD risk in T2DM patients a challenging endeavor. Thus, the exploration of non-invasive biomarkers for MASLD diagnosis—characterized by clinical feasibility, reliability, and practicality—holds substantial clinical significance and value.

Currently, a diverse range of anthropometric and metabolic parameters have been put forward as potential predictors for MASLD.[5,6,11] Among these, insulin resistance (IR) emerges as the most prominent predictive factor for MASLD.[11] The hyperinsulinemic-euglycemic clamp (HEC) technique is internationally endorsed as the gold standard for IR evaluation; however, its operational complexity renders it impractical for routine clinical application. Accordingly, in 1985, Matthews et al proposed the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), a steady-state model designed for the indirect assessment of IR.[12] Numerous studies have demonstrated that HOMA-IR possesses the advantages of operational simplicity and cost-effectiveness, yet it exhibits substantial computational inaccuracies when applied to patients with T2DM undergoing insulin treatment. As an optimized modification of HOMA-IR, the C-peptide-based insulin resistance index (HOMA2 IR-CP) serves as a widely utilized and readily implementable clinical metric for indirect IR assessment, which is not interfered with by insulin antibodies or exogenous insulin administration. In recent years, accumulating studies have indicated that the triglyceride-glucose index (TyG) also constitutes a reliable indicator for IR evaluation.[13] To further explore the diagnostic potential of TyG-related metrics in MASLD, some researchers have integrated TyG with obesity-related parameters. Notably, the triglyceride-glucose-body mass index (TyG-BMI) has emerged as a promising diagnostic biomarker for MASLD, exhibiting superior diagnostic performance in female MASLD patients.[14]

However, there is currently a paucity of data regarding whether the HOMA2 IR-CP and TyG-BMI indices can function as predictive biomarkers for MASLD in patients with T2DM. In response to this knowledge gap, the present study intends to assess the diagnostic accuracy of HOMA2 IR-CP and TyG-BMI for MASLD in T2DM patients.

Methods

Study Design and Populations

This study adopts a cross-sectional design. A total of 400 patients with T2DM admitted to the Department of Endocrinology, Chongqing Jiangjin District Hospital of Chinese Medicine, between January 2024 and May 2025 were finally enrolled. All participants met the diagnostic criteria specified in the China Guideline for the Prevention and Treatment of Type 2 Diabetes (2020 Edition), were confirmed to have T2DM, and were receiving exogenous insulin injections for glycemic control.[15] The exclusion criteria were defined as follows: (1) Patients with severe chronic diseases, including cardiovascular, hepatic, renal, cerebrovascular, hematological, or malignant diseases; (2) Patients with a current or past history of infections with communicable pathogens, such as hepatitis viruses, Mycobacterium tuberculosis, Treponema pallidum (syphilis), or human immunodeficiency virus (HIV); (3) Pregnant or lactating women; (4) Patients with a family history of genetic disorders; (5) Patients with psychiatric or cognitive impairments; (6) Patients aged < 18 years.

The sample size was calculated using the pmsampsize package in R software (version 3.6.1). A binary classification prediction model was specified (type = "b"), with an anticipated C-statistic of 0.8, indicating good model discrimination. The model included 4 parameters (including the intercept), and the estimated event prevalence was 65%. This yielded a requirement of 150 events, corresponding to a total sample size of 230 participants. To account for an estimated 10% loss to follow-up or withdrawal, the minimum number of required events was adjusted to 167, resulting in a final total sample size of 256.

This study was approved by the Ethics Committee of the corresponding author's hospital (Approval No. ZYY2025012). Written informed consent was obtained from all participating patients prior to their enrollment in the study.

Diagnostic Criteria for MASLD

Diagnostic criteria were referenced from the Guidelines for the Prevention and Treatment of Metabolic Dysfunction-Associated (Non-Alcoholic) Fatty Liver Disease (2024 Version), requiring fulfillment of the following three criteria: (1) Imaging-diagnosed hepatic steatosis and/or histological evidence of ≥5% macrovesicular steatosis on liver biopsy; (2) Presence of at least one component of MetS, defined as: ① Overweight/Obesity: Body Mass Index (BMI) ≥ 24.0 kg/m^2, OR waist circumference ≥90 cm (male) / 85 cm (female), OR excess body fat content/percentage. ② Elevated blood pressure/Hypertension: Blood pressure ≥130/85 mmHg, OR ongoing antihypertensive drug therapy. ③ Prediabetes or T2DM: Fasting plasma glucose ≥6.1 mmol/L, OR 2-hour postprandial glucose ≥7.8 mmol/L OR HbA1c ≥5.7%, OR a history of T2DM, OR HOMA-IR ≥2.5. ④ Elevated serum triglycerides: Fasting serum triglycerides (TG) ≥1.70 mmol/L, OR ongoing lipid-lowering therapy. ⑤ Reduced HDL-cholesterol: Serum HDL-cholesterol ≤1.0 mmol/L (male) / 1.3 mmol/L (female), OR ongoing lipid-lowering therapy.(3) Exclusion of other known causes of hepatic steatosis, including but not limited to: significant alcohol consumption (defined as the weekly ethanol intake is ≥ 210 g for males and ≥ 140 g for females), malnutrition, Wilson's disease, etc.

Data Collection

Demographic and laboratory data were extracted from the electronic medical record system within 24 hours of patient admission, including the following categories. General information: Age, gender, height, and weight. Laboratory parameters: White blood cell count (WBC), platelet count (PLT), prothrombin time (PT), activated partial thromboplastin time (APTT), prothrombin time activity (PTA), glycated hemoglobin (HbA1c), random blood glucose (Gluc), total cholesterol (TC), TG, alanine aminotransferase (ALT), aspartate aminotransferase (AST), total bilirubin (TBIL), blood urea nitrogen (BUN), creatinine (CREA), and uric acid (UA). Additionally, fasting blood glucose (0-hour glucose) and fasting C-peptide (0-hour C-peptide) levels were documented based on oral glucose tolerance test (OGTT) results. All enrolled patients had undergone at least one magnetic resonance imaging examination, with results indicative of hepatic steatosis. All data were independently collected and verified by two or more authors. Missing data were supplemented through review of clinical records; data that remained incomplete were excluded, and the analysis was performed using a complete case dataset.

Calculation of Clinical Parameters

The following formulas were used to calculate the clinical parameters: (1) TyG = ln [triglycerides (mg/dl) × glucose (mg/dl)/2]; (2) BMI = body mass (kg)/height2(m2); (3) TyG-BMI = TyG × BMI; (4) HOMA2 IR-CP: Calculated using the HOMA2 Calculator v2.2.3 developed by the University of Oxford Diabetes Trials Unit (https://www.dtu.ox.ac.uk/homacalculator/);[16] (5)Triglyceride/High-Density Lipoprotein Cholesterol Ratio (TG/HDL-C) = Triglycerides (TG, mg/dL) / High-density lipoprotein cholesterol (HDL-C, mg/dL); (6) Hepatic Steatosis Index (HSI) = BMI × 1.2 + (ALT/AST ratio) × 0.06 − Age × 0.07 + Gender coefficient (Male = 0; Female = 6).

Grouping Criteria

In this study, the dataset was randomly split into a derivation cohort and a validation cohort at a ratio of 7:3 using a random sampling approach. Additionally, patients with T2DM were categorized into two groups: the MASLD group and the control group, based on the presence or absence of concurrent MASLD.

Statistical Analysis

All statistical analyses were performed using R software (version 4.2.2), MSTATA software (www.mstata.com) and SPSS software (version 27.0). For quantitative data adhering to a normal distribution, intergroup comparisons were conducted via independent-sample *t*-tests. Non-normally distributed data were presented as medians (Q1, Q3), with

intergroup comparisons performed using the Mann–Whitney *U*-test. Qualitative data were expressed as case counts (%), and intergroup comparisons were carried out using the chi-square (χ^2) test or Fisher's exact test, as appropriate. Given the high dimensionality of factors included in this study, Lasso regression was employed for variable selection, followed by binary logistic regression to identify independent predictors of MASLD in patients with T2DM. Cutoff values for HOMA2 IR-CP and TyG-BMI were determined using the derivation cohort (70% of total subjects) via the Youden's J statistic method; the validation cohort (30% of total subjects) was used to validate the diagnostic efficacy of these cutoff values. Then, based on the cutoff values, convert these parameters into categorical variables. A nomogram prediction model was constructed using the rms package in R 4.2.2. The discriminative ability and calibration of the model were evaluated using receiver operating characteristic (ROC) curves, calibration curves, and decision curve analysis (DCA). Furthermore, restricted cubic spline (RCS) regression analysis was performed to explore potential non-linear associations between HOMA2 IR-CP, TyG-BMI, and MASLD. To validate the robustness of the study findings, subgroup analyses were stratified by sex, age, HbA1c, and BMI levels. Interaction P-values were calculated to assess heterogeneity across subgroups. All statistical tests were two-tailed, and a P-value < 0.05 was considered statistically significant.

Results

Baseline Characteristics

A total of 311 patients with T2DM were finally enrolled in this study (Figure 1). According to the presence or absence of concurrent MASLD, the participants were stratified into the MASLD group and the control group. Compared with the control group, patients in the MASLD group were younger, had a shorter PT, and marginally lower HbA1c levels,

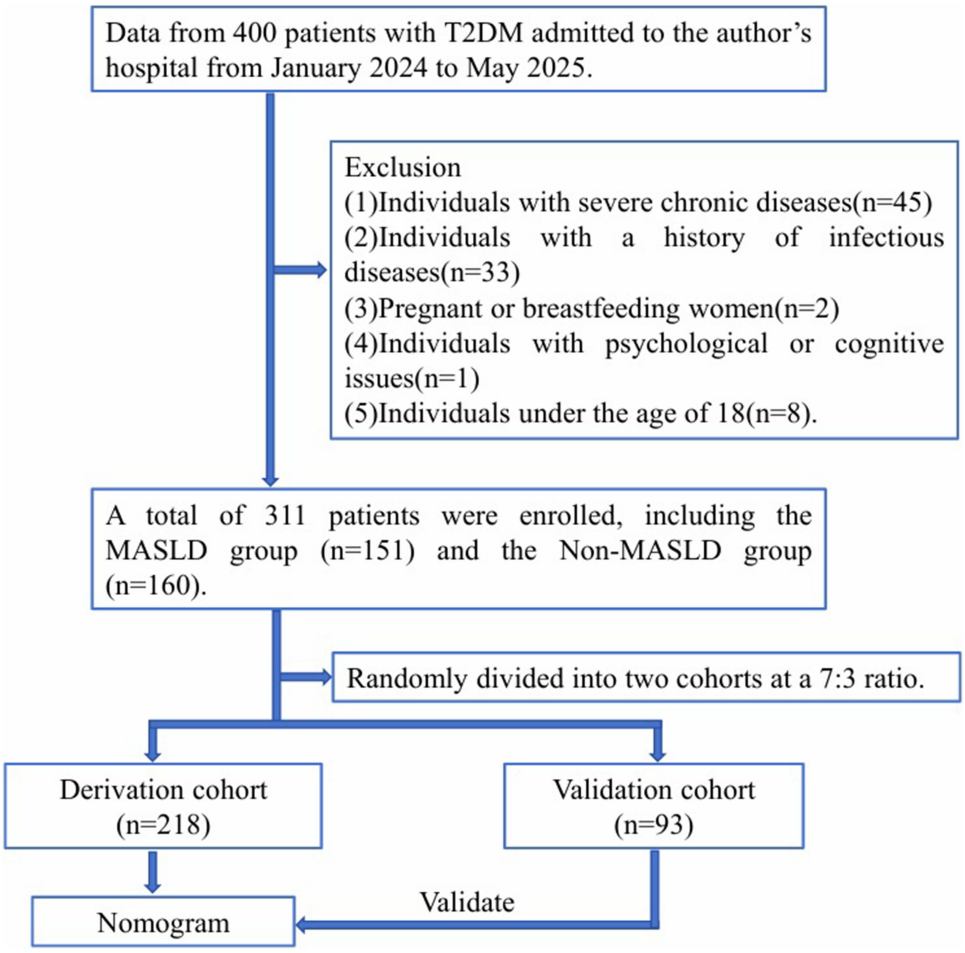

Figure 1 Study flow chart.

accompanied by significantly elevated levels of TC, TG, ALT, AST, TBIL, UA, BMI, HOMA2 IR-CP, TyG-BMI, TG/HDL-C, and HSI (Table 1). The baseline characteristics of the derivation cohort (218 cases) and an internal validation cohort (93 cases), divided at a 7:3 ratio, are detailed in Table S1; the distribution of key variables between the two sets was balanced (all P>0.05), confirming the rationality of the grouping strategy. In the derivation cohort, compared with the control group, MASLD patients had significantly lower age, PT, and BUN levels ($P < 0.05$), as well as significantly higher levels of TC, TG, ALT, AST, TBIL, UA, BMI, HOMA2 IR-CP, TyG-BMI, TG/HDL-C, and HSI ($P < 0.05$); no significant differences were observed for the remaining indicators ($P > 0.05$) (Table 2). In the validation cohort, MASLD

Table 1 Baseline Characteristics of T2DM

Characteristic	Overall (N=311)	Control (N=160)	MASLD (N=151)	*p*-value
Gender, n (%)				1.000
Male	140 (45.016%)	72 (45.000%)	68 (45.033%)	
Female	171 (54.984%)	88 (55.000%)	83 (54.967%)	
Age (year)	58 (51, 66)	60 (54.75, 67)	56 (49, 61.5)	<0.001
WBC (×10^9/L)	6.70 (5.54, 8.33)	6.85 (5.30, 8.36)	6.48 (5.59, 8.32)	0.698
PLT (×10^9/L)	203.00 (171.00, 250.00)	209.50 (173.50, 254.75)	199.00 (169.50, 249.00)	0.445
PT (Sec)	12.70 (12.30, 13.40)	12.80 (12.40, 13.60)	12.60 (12.20, 13.20)	0.008
APTT (Sec)	26.65 (24.18, 28.79)	26.75 (24.30, 28.82)	26.27 (24.00, 28.69)	0.300
HbA1c (%)	10.80 (8.75, 12.10)	10.95 (9.28, 12.80)	10.60 (8.60, 11.80)	0.035
Gluc (mmol/l)	12.68 (9.14, 16.55)	12.45 (8.67, 16.92)	12.81 (9.78, 16.39)	0.688
TC (mmol/l)	5.070 (4.37, 6.03)	4.86 (4.13, 5.86)	5.33 (4.45, 6.43)	0.005
TG (mmol/l)	1.910 (1.09, 3.35)	1.31 (0.89, 2.25)	2.77 (1.65, 4.85)	<0.001
ALT (U/L)	20.10 (13.75, 34.15]	16.95 (12.48, 30.45)	21.90 (16.15, 38.80)	<0.001
AST (U/L)	20.40 (16.35, 28.30]	19.05 (15.65, 24.83)	22.10 (17.25, 28.80)	0.002
TBIL (μmol/L)	12.90 (9.80;16.05)	11.85 (9.05;15.40)	13.40 (11.15;16.65)	0.004
BUN (mmol/l)	5.91 (4.82, 7.65)	6.37 (4.96, 8.59)	5.49 (4.77, 6.65)	0.001
CREA (U/L)	60.40 (49.85, 73.30)	62.10 (49.45, 78.83)	58.40 (50.20, 71.05)	0.147
UA (μmol/L)	285.00 (221.00, 364.00)	260.00 (201.25, 329.00)	313.00 (251.50, 385.50)	<0.001
BMI (kg/m^2)	24.66 (22.46;27.05)	23.44 (21.61, 25.82)	25.81 (23.66, 28.68)	<0.001
HOMA2 IR-CP	1.33 (0.69, 2.34)	0.92 (0.43, 1.65)	1.87 (1.02, 2.76)	<0.001
TyG-BMI	238.88 (201.66, 272.95)	212.94 (187.09, 247.10)	264.78 (233.82, 301.72)	<0.001
TG/HDL-C	3.68 (1.20, 6.55)	2.38 (1.47, 4.21)	5.04 (3.37, 8.55)	<0.001
HSI	34.98 (31.84, 39.53)	33.02 (30.46, 36.14)	37.47 (33.79, 41.75)	<0.001

Notes: WBC, white blood cell count; PLT, platelet; PT, prothrombin time; APTT, activated partial thromboplastin time; HbA1c, hemoglobin A1c; Gluc, glucose; TC, total cholesterol; TG, triglycerides; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TBil, total bilirubin; BUN, blood urea nitrogen; CR, creatinine; UA, uric acid; BMI, body mass index; HOMA2 IR-CP, homeostatic model assessment 2 of insulin resistance-C peptide; TyG-BMI, triglyceride-glucose-body mass index; TG/HDL-C, triglyceride to high-density lipoprotein cholesterol ratio; HSI, hepatic steatosis index.

Table 2 Baseline Characteristics of the Control Group and MASLD Group within Both the Derivation and Validation Cohorts

Characteristic	Derivation Cohort, N = 218			Validation Cohort, N = 93		
	ControlN = 117	MASLD N = 101	*p*-value	Control N = 43	MASLD N = 50	*p*-value
Gender, n (%)			0.686			0.576
Male	64 (54.7%)	58 (57.4%)		24 (55.8%)	25 (50.0%)	
Female	53 (45.3%)	43 (42.6%)		19 (44.2%)	25 (50.0%)	
Age (year)	60 (55, 67)	56 (48, 60)	<0.001	60 (54, 67)	57 (50, 67)	0.127
WBC (×10^9/L)	6.75 (5.28, 7.89)	6.72 (5.76, 8.39)	0.384	7.1 (6.0, 8.9)	6.2 (5.5, 8.1)	0.033

(*Continued*)

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Table 2 (Continued).

Characteristic	Derivation Cohort, N = 218			Validation Cohort, N = 93		
	ControlN = 117	MASLD N = 101	*p*-value	Control N = 43	MASLD N = 50	*p*-value
PLT (×10^9/L)	209 (177, 254)	199 (168, 249)	0.374	216 (151, 261)	199 (179, 249)	0.991
PT (Sec)	12.90 (12.40, 13.70)	12.60 (12.30, 13.20)	0.010	12.70 (12.20, 13.30)	12.50 (12.10, 13.20)	0.411
APTT (Sec)	27.0 (24.5, 28.9)	26.4 (24.3, 28.8)	0.460	26.63 (23.62, 28.51)	25.78 (23.12, 28.49)	0.505
HbA1c (%)	10.70 (9.00, 12.70)	10.80 (8.60, 11.90)	0.502	11.50 (9.90, 13.00)	9.85 (8.30, 11.50)	0.004
Gluc (mmol/l)	12.3 (8.6, 16.5)	13.2 (10.1, 16.5)	0.125	14.7 (9.4, 18.4)	11.9 (8.5, 16.0)	0.129
TC (mmol/l)	4.85 (4.28, 5.79)	5.33 (4.51, 6.41)	0.018	4.88 (3.86, 5.89)	5.32 (4.42, 6.64)	0.125
TG (mmol/l)	1.2 (0.9, 2.1)	2.9 (1.8, 5.4)	<0.001	1.57 (0.90, 2.45)	2.51 (1.45, 4.42)	0.002
ALT (U/L)	18 (12, 30)	22 (15, 40)	0.007	16 (13, 33)	22 (18, 35)	0.017
AST (U/L)	19 (16, 25)	22 (18, 29)	0.018	19 (15, 25)	22 (17, 29)	0.035
TBIL (μmol/L)	12 (9, 15)	14 (12, 17)	0.006	13 (9, 16)	13 (11, 16)	0.277
BUN (mmol/l)	6.01 (4.77, 7.94)	5.41 (4.62, 6.92)	0.032	7.24 (5.83, 9.91)	5.64 (4.82, 6.49)	<0.001
CREA (U/L)	60 (48, 73)	58 (51, 70)	0.932	71 (60, 90)	58 (50, 73)	0.005
UA (μmol/L)	245 (195, 309)	311 (249, 374)	<0.001	300 (243, 372)	327 (263, 400)	0.142
BMI (kg/m^2)	23.1 (21.5, 25.7)	26.5 (23.7, 29.1)	<0.001	24.4 (22.5, 26.0)	25.2 (23.3, 27.1)	0.160
HOMA2 IR-CP	0.85 (0.41, 1.65)	1.87 (1.12, 2.86)	<0.001	1.15 (0.67, 1.86)	1.87 (0.88, 2.67)	0.026
TyG-BMI	209 (185, 246)	272 (241, 306)	<0.001	224 (189, 254)	250 (227, 281)	<0.001
TG/HDL-C	2 (1, 4)	5 (3, 9)	<0.001	3.2 (1.5, 4.9)	5.0 (2.5, 7.6)	<0.001
HSI	32.8 (30.4, 36.1)	38.6 (34.1, 42.1)	<0.001	33.3 (31.3, 36.3)	37.0 (33.7, 40.0)	<0.001

Notes: WBC, white blood cell count; PLT, platelet; PT, prothrombin time; APTT, activated partial thromboplastin time; HbA1c, hemoglobin A1c; Gluc, glucose; TC, total cholesterol; TG, triglycerides; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TBil, total bilirubin; BUN, blood urea nitrogen; CR, creatinine; UA, uric acid; BMI, body mass index; HOMA2 IR-CP, homeostatic model assessment 2 of insulin resistance-C peptide; TyG-BMI, triglyceride-glucose-body mass index; TG/HDL-C, triglyceride to high-density lipoprotein cholesterol ratio; HSI, hepatic steatosis index.

patients exhibited significantly higher levels of TG, ALT, AST, HOMA2 IR-CP, TyG-BMI, TG/HDL-C, and HSI ($P < 0.05$), and significantly lower levels of WBC, HbA1c, BUN, and CR ($P < 0.05$) compared with the control group; the other indicators showed no statistically significant differences ($P > 0.05$) (Table 2).

Independent Risk Factors for MASLD in Patients with T2DM

LASSO regression was applied to select predictors with non-zero coefficients from variables that differed between T2DM patients with MASLD and the control group (Figure 2A). The optimal λ value was determined via 10-fold cross-

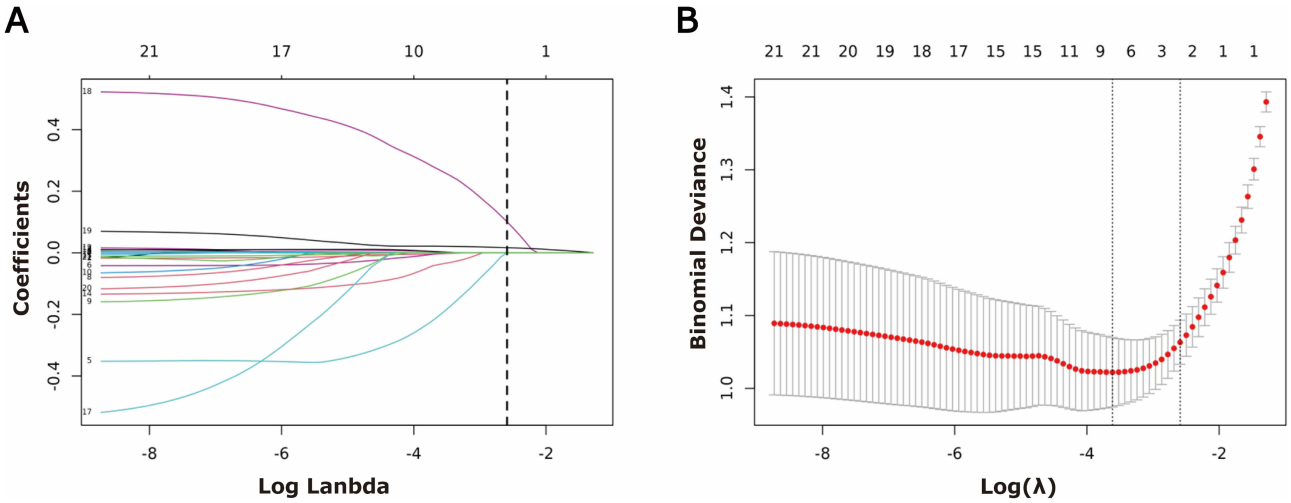

Figure 2 Screening of predictors for MASLD in T2DM patients via LASSO regression analysis. (**A**) Coefficients Path diagram; (**B**) Relationship diagram between binomial deviance and λ.

Table 3 Investigation of Risk Factors for MASLD in T2DM Patients via Logistic Regression Analysis

Characteristic	Univariable			Multivariable		
	OR	95% CI	*p*-value	OR	95% CI	*p*-value
HOMA2 IR-CP	1.78	1.39, 2.28	<0.001	1.47	1.12, 1.92	0.005
TyG-BMI	1.03	1.02, 1.04	<0.001	1.03	1.02, 1.04	<0.001

Table 4 Prediction of T2DM-Associated MASLD by HOMA2 IR-CP and TyG-BMI

Characteristic	Cutpoint	Youden	Sensitivity	Specificity	PPV	NPV	Accuracy
HOMA2 IR-CP	1.38	0.330	64.2%	68.8%	66.0%	67.1%	66.6%
TyG-BMI	223.33	0.431	88.1%	55.0%	64.9%	83.0%	71.1%

validation, leading to the identification of two predictive variables with non-zero coefficients (Figure 2B), namely HOMA2 IR-CP and TyG-BMI. Subsequent univariate and multivariate logistic regression analyses were performed, and the results demonstrated that HOMA2 IR-CP (OR: 1.47, 95% CI: 1.12–1.92) and TyG-BMI (OR: 1.03, 95% CI: 1.02–1.04) were independent risk factors for MASLD in patients with T2DM (Table 3). Clinically, each one-unit increase in HOMA2 IR-CP was associated with a 47% higher likelihood of MASLD, while each one-unit increase in TyG-BMI corresponded to a 3% increase in risk.

Model Construction and Performance Analysis

From the aforementioned analysis results, the optimal predictive cut-off values for HOMA2 IR-CP and TyG-BMI were determined using the Youden index derived from ROC curves of the derivation cohort (Table 4), and a nomogram prediction model was constructed accordingly (Figure 3). This model integrated two parameters: HOMA2 IR-CP (cut-off value: 1.38) and TyG-BMI (cut-off value: 223.33). In the derivation cohort, the area under the curve (AUC) of the model was 0.805 (95% CI: 0.749–0.860; Figure 4A), whereas in the validation cohort, it was 0.730 (95% CI: 0.632–0.829; Figure 5A). Model calibration was evaluated via calibration curves: the bias-corrected line was closely aligned with both the ideal line and the apparent line, indicating strong consistency between predicted and actual values (Figures 4B and 5B). DCA was performed to assess the model's clinical utility. Results showed that, compared with the strategies of intervening in all patients or no patients, the nomogram-based prediction and subsequent intervention for MASLD in T2DM patients resulted in a higher net benefit (Figures 4C and 5C).

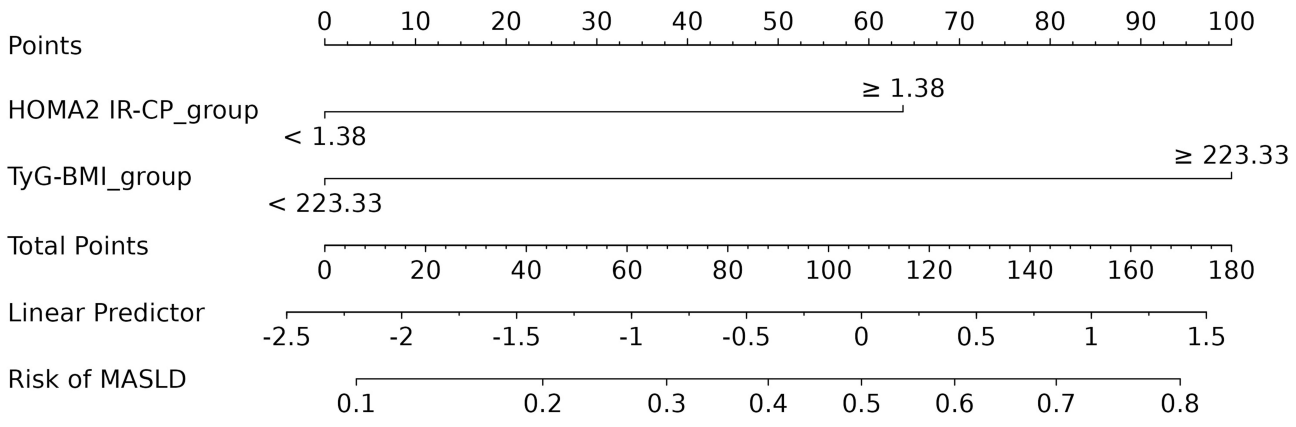

Figure 3 Nomogram for predicting MASLD in T2DM patients.

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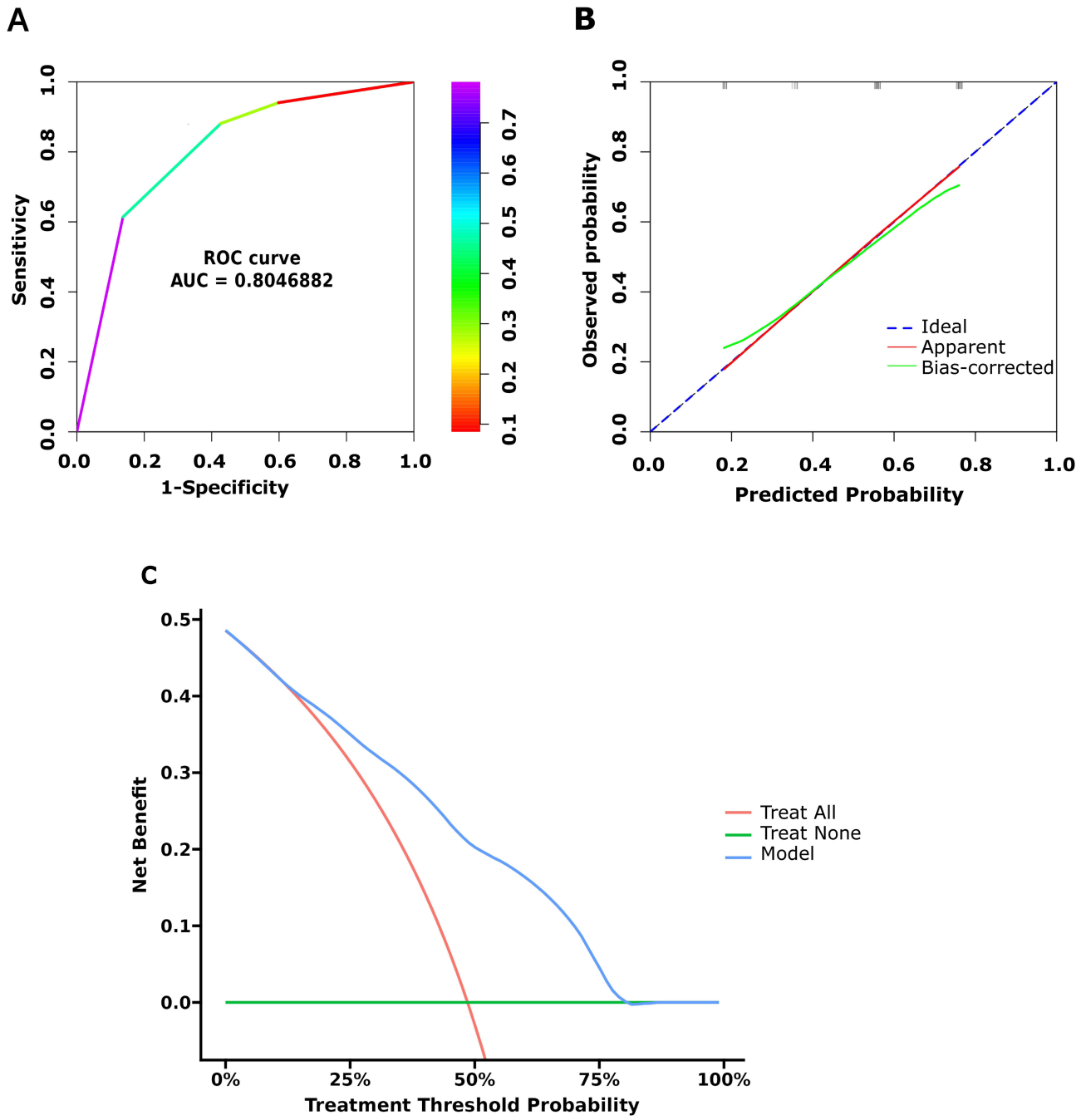

Figure 4 Diagnostic performance evaluation of the nomogram derived from the derivation cohort. (**A**) ROC curve of the nomogram; (**B**) calibration curve of the nomogram; (**C**) DCA curve of the nomogram.

RCS Analysis of HOMA2 IR-CP, TyG-BMI, and MASLD in T2DM Patients

Furthermore, RCS regression models were utilized to explore the dose-response relationship between HOMA2 IR-CP, TyG-BMI, and the risk of MASLD in T2DM patients. After adjusting for potential confounding factors, the RCS curves demonstrated a non-linear association between HOMA2 IR-CP and MASLD (P for non-linearity < 0.05) (Figure 6A). In contrast, TyG-BMI showed a linear association with MASLD (P for non-linearity = 0.139) (Figure 6B).

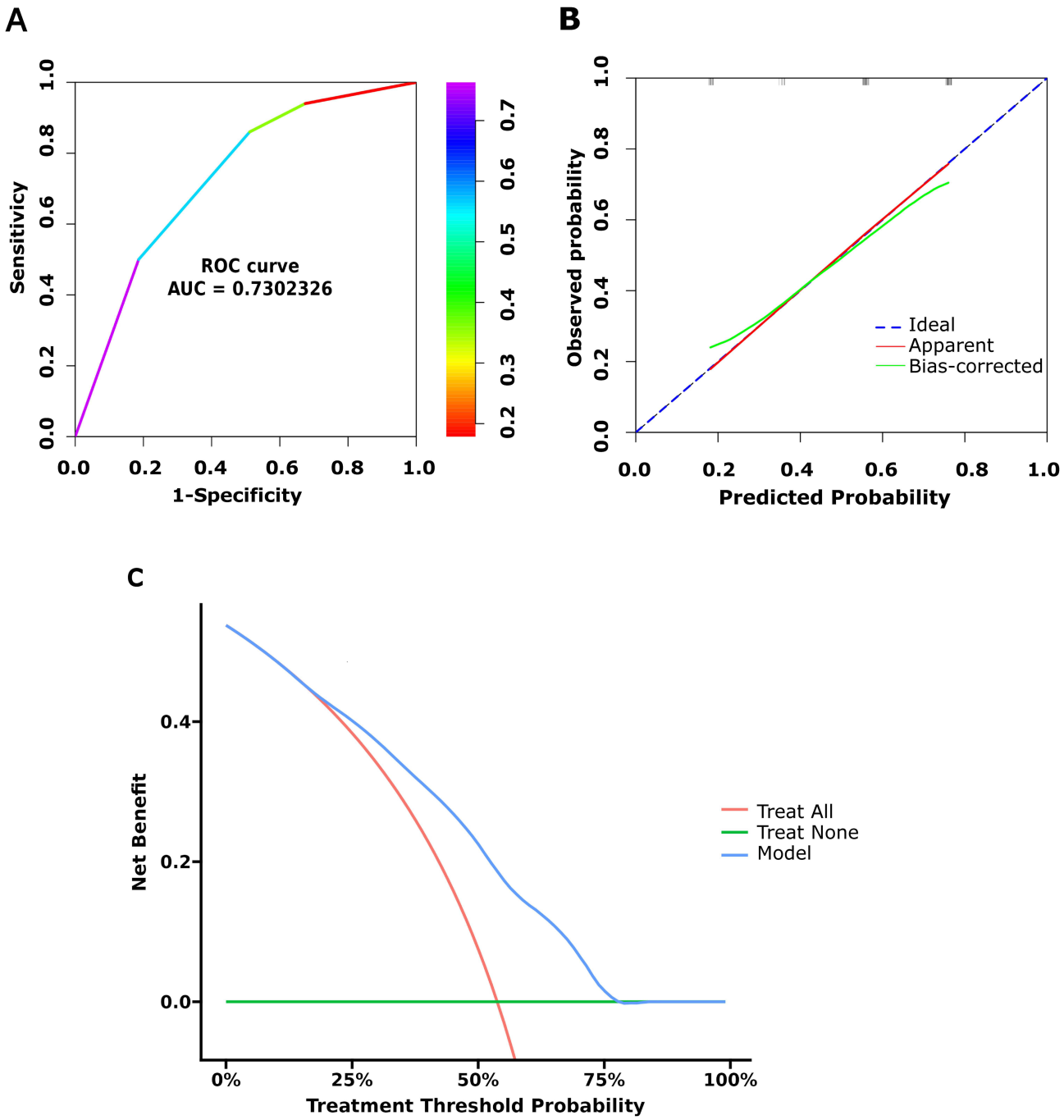

Figure 5 Diagnostic performance evaluation of the nomogram derived from the validation cohort. (**A**) ROC curve of the nomogram; (**B**) calibration curve of the nomogram; (**C**) DCA curve of the nomogram.

Analysis of the Association and Stratified Interaction Between HOMA2 IR-CP, TyG-BMI, and MASLD

After adjusting for potential confounding factors including age, gender, HbA1c, and BMI, stratified and interaction analyses were performed to examine the relationships between HOMA2 IR-CP, TyG-BMI, and MASLD. Key findings are summarized as follows:

Figure 7A illustrates a significant positive association between HOMA2 IR-CP and MASLD risk in the total population (OR=1.68, 95% CI: 1.37–2.06, P<0.001). Subgroup analyses revealed no significant interactions with gender

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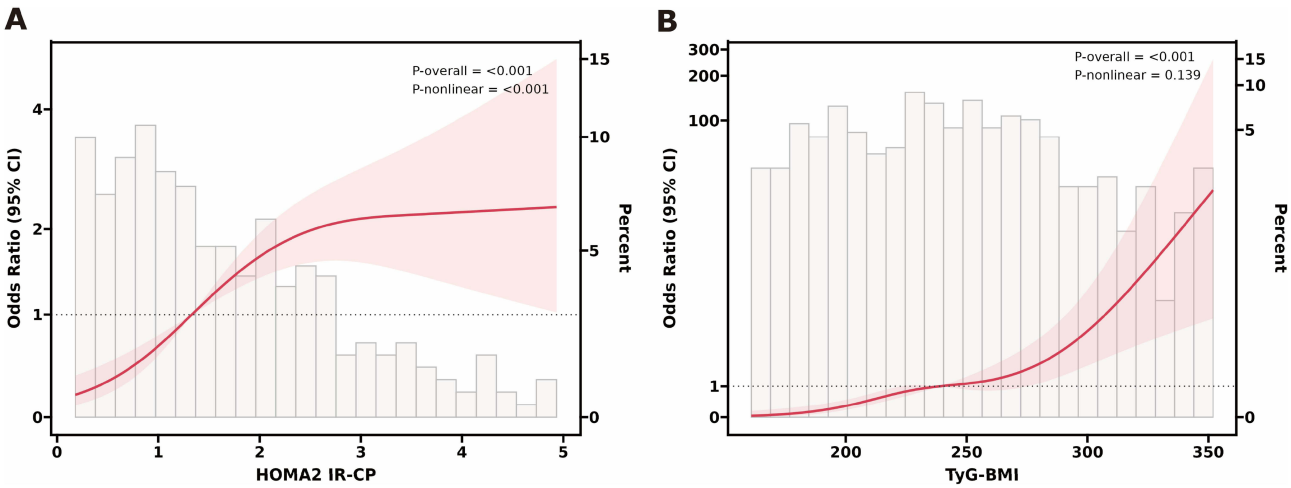

Figure 6 RCS analysis of HOMA2 IR-CP, TyG-BMI, and MASLD in T2DM patients; (**A**) HOMA2 IR-CP; (**B**) TyG-BMI.

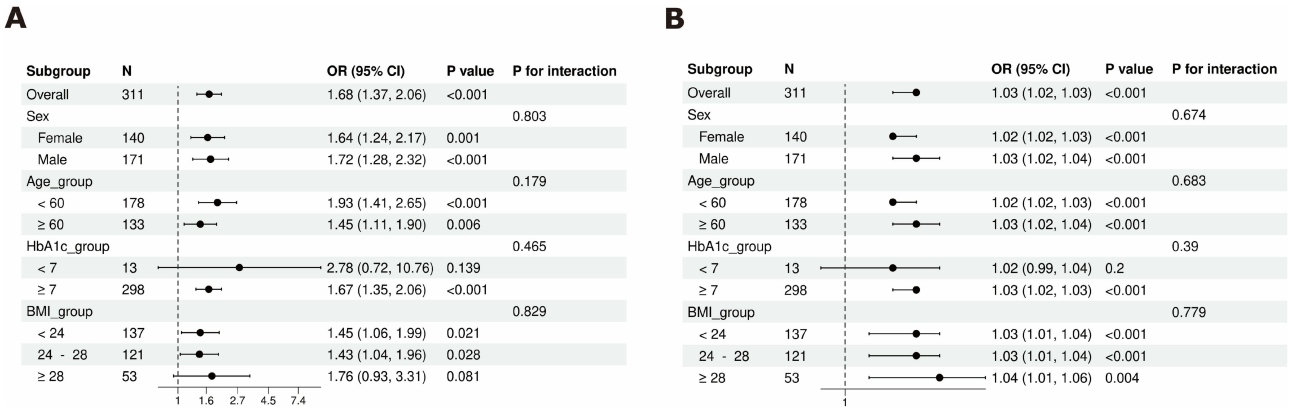

Figure 7 Association and stratified interaction between HOMA2 IR-CP, TyG-BMI, and MASLD; (**A**) HOMA2 IR-CP; (**B**) TyG-BMI.

(P for interaction=0.803) or age groups (P for interaction=0.179). The association strength was marginally higher in males than in females (OR=1.72 vs 1.64) and in the <60 years group compared with the ≥60 years group (OR=1.93 vs 1.45). No significant interaction was observed across HbA1c subgroups (P for interaction=0.465), with a significant association restricted to the HbA1c≥7% (poor glycemic control) subgroup (P<0.001). Similarly, no significant interaction was detected across BMI subgroups (P for interaction=0.829), while significant associations were noted in the BMI<24 and BMI 24–28 subgroups (P<0.05 for both).

Figure 7B demonstrates a significant positive association between TyG-BMI and MASLD risk in the total population (OR=1.03, 95% CI: 1.02–1.03, P<0.001). Subgroup analyses indicated that this association remained consistent across gender, age, and BMI subgroups, but was not significant in individuals with HbA1c<7% (good glycemic control) (P>0.05).

Discussion

This study aims to explore the associations between HOMA2 IR-CP, TyG-BMI, and MASLD in patients with T2DM, identify independent risk factors, construct a predictive model, and evaluate its predictive performance. Based on the above objectives, the following key findings were obtained: (1) Compared with the control group, patients in the MASLD group were younger, had a shorter PT and slightly lower HbA1c levels, whereas the levels of TC, TG, ALT, AST, TBIL, UA, BMI, HOMA2 IR-CP, TyG-BMI, TG/HDL-C ratio, and HSI were significantly elevated. (2) LASSO regression and logistic regression analyses revealed that HOMA2 IR-CP and TyG-BMI were independent risk factors for MASLD in

patients with T2DM. (3) The constructed predictive model exhibited good discriminatory power, with an AUC of 0.805 in the derivation cohort and 0.730 in the validation cohort. Calibration curves demonstrated strong agreement between predicted probabilities and actual outcomes, and DCA confirmed that the model provided substantial clinical net benefit. (4) RCS analysis indicated a non-linear association between HOMA2 IR-CP and MASLD (P for non-linearity < 0.05), while TyG-BMI showed a linear association with MASLD (P for non-linearity = 0.139). No significant interaction effects were observed across subgroups stratified by gender, age, BMI, or HbA1c levels (all P for interaction > 0.05); however, the associations were more prominent in subgroups with BMI < 28 and HbA1c ≥ 7% (indicating poor glycemic control).

The observed differences in clinical indicators between the two patient groups are physiologically consistent and unlikely to be incidental. As summarized in Table 1, patients with T2DM and comorbid MASLD exhibit significantly higher values in IR indices (HOMA2 IR-CP, TyG-BMI), lipid profiles (TC, TG), and markers of liver injury (ALT, AST, TBIL) compared to those with T2DM alone. This phenomenon may be attributed to the following mechanisms: hepatic adipocytes in MASLD patients secrete large amounts of resistin, which reduces the insulin sensitivity of adipocytes, muscle cells, and hepatocytes, exacerbates hepatic IR, promotes endogenous glucose production, and enhances hepatic glycogen synthesis. Meanwhile, resistin contributes to hepatic fibrinogen formation via its pro-inflammatory effects, triggering liver fibrosis, causes and aggravates liver damage, and thereby accelerating MASLD progression.[17] Additionally, IR is frequently accompanied by hyperinsulinemia, which promotes the breakdown of peripheral adipose tissue, elevates blood free fatty acid levels, and subsequently increases hepatic transport burden. This leads to increased TG synthesis and hepatic lipid deposition, ultimately resulting in hepatic steatosis.[18] Thus, IR, blood lipids, and MASLD form a vicious cycle of mutual reinforcement. Previous epidemiological studies have indicated that the prevalence of MASLD increases with age, peaks in the 50–59 years age group, and declines after 60 years.[19] In the present study, the median age of T2DM patients complicated with MASLD was 56 years, with a primary distribution of 49–61.5 years, which was younger than that of patients with T2DM alone—consistent with previous findings. This suggests a rapid increase in the prevalence of MASLD among young T2DM patients in mainland China. Notably, PT tends to decrease in T2DM patients complicated with MASLD, which aligns with the findings of Min-ran Li et al.[20] The underlying mechanism may involve the following: although hepatic synthetic function remains unimpaired in T2DM patients with MASLD, long-term stimulation by inflammatory factors and IR induces the liver to synthesize various endogenous coagulation factors, damages vascular endothelial cells, and promotes the release of procoagulant factors. Concurrently, the endogenous coagulation system is abnormally activated, leading to a hypercoagulable state of the blood. This state is manifested as shortened PT and a significant increase in the patient's risk of thrombosis. Therefore, for young T2DM patients complicated with MASLD, early PT shortening should raise concerns about their risk of thrombosis and cardio-cerebrovascular diseases, and targeted screening for these conditions should be conducted as early as possible.

MASLD is currently one of the most prevalent chronic liver diseases globally. Over the past decade, the prevalence of MASLD in China has increased sharply, with an onset trend toward younger populations, imposing a substantial economic burden on society. Patients with T2DM have twice the risk of developing MASLD compared to the general population and an even higher risk of progressing to severe liver diseases.[21] However, in routine clinical practice, most T2DM patients typically focus only on changes in blood glucose and glycated hemoglobin levels, neglecting screening for the potential risk of MASLD. Currently, liver biopsy remains the internationally recognized gold standard for MASLD diagnosis. Nevertheless, its high cost and invasive nature result in low patient acceptance, with most patients reluctant to undergo this procedure. Non-invasive diagnostic methods for MASLD recommended in various clinical guidelines include magnetic resonance imaging, liver ultrasound, and FibroScan, among others.[21,22] That said, primary care facilities or community hospitals in economically underdeveloped regions may lack essential equipment or fail to meet the needs of large-scale population screening. Therefore, this study uses commonly available clinical laboratory indicators to construct a clinical prediction model for T2DM complicated with MASLD. The objective is to enable early prediction of MASLD risk, facilitate earlier intervention and treatment, and thereby reduce the risk of progression to severe liver diseases.

This study found that HOMA2 IR-CP and TyG-BMI levels were significantly higher in T2DM patients complicated with MASLD than in the control group, and both were identified as independent risk factors. HOMA2 IR-CP is a homeostasis model designed to assess and diagnose IR. It differs from the conventional insulin resistance index

HOMA-IR, which is calculated based on fasting insulin levels and is one of the most widely used methods for evaluating IR in epidemiological studies. However, HOMA-IR may yield inaccurate results in T2DM patients requiring exogenous insulin: long-term insulin injections can induce the production of insulin antibodies, which interfere with the measurement of insulin levels via radioimmunoassay. In contrast, HOMA2 IR-CP—developed by a research team at the University of Oxford based on HOMA-IR—uses C-peptide to establish a homeostasis model. C-peptide is a peptide hormone secreted in equimolar amounts with insulin from pancreatic β-cells during the cleavage of proinsulin molecules; thus, it can be used to quantify insulin secretion. Furthermore, C-peptide levels in peripheral blood are higher and more stable than insulin levels, enabling it to replace fasting insulin for calculating the HOMA-IR. Previous studies have primarily focused on the correlation between HOMA-IR and MASLD, with few exploring the relationship between HOMA2 IR-CP and MASLD. For example, Gala Gutierrez-Buey et al enrolled 56 T2DM patients to evaluate the accuracy of HOMA-IR in predicting MASLD, demonstrating that HOMA-IR exhibited good diagnostic performance (AUC = 0.807) with an optimal cutoff value of 4.5. In the present study, we investigated the role of HOMA2 IR-CP in T2DM complicated with MASLD, and the results showed an AUC of up to 0.805 and an optimal cutoff value of 1.38.[23] This indicates that HOMA2 IR-CP is also a robust indicator for predicting MASLD in T2DM patients.

The TyG index is a metric that integrates blood glucose and triglyceride levels. Multiple population-based studies have confirmed that the TyG index is an ideal indicator for assessing IR.[24] Globally, research on the application of the TyG index in MASLD evaluation has also advanced significantly, revealing a strong correlation between elevated TyG index levels and increased MASLD risk in the general population.[25] Numerous studies have demonstrated that simple anthropometric measures—including BMI, waist circumference (WC), and waist-to-height ratio (WHtR)—are independently associated with MASLD, with BMI acting as a predictive indicator for MASLD.[26] TyG index-related parameters refer to combinations of the TyG index with WC, BMI, or WHtR, first reported by Ko et al.[27] In previous studies investigating the correlation between TyG-related parameters and MASLD, most results indicated that TyG-BMI outperforms the TyG index in MASLD prediction. For instance, in a study involving T2DM patients receiving non-insulin therapy with good glycemic control, TyG-BMI was identified as the optimal predictor of MASLD, exhibiting the highest AUC (0.738).[28] However, research on the potential predictive role of TyG-BMI in MASLD patients with concurrent diabetes remains limited, and its utility as an alternative biomarker for managing T2DM patients with MASLD remains unclear. Therefore, this study focused on the diagnostic performance of the TyG-BMI index for MASLD in T2DM patients. ROC curve analysis showed that TyG-BMI achieved the highest AUC (0.805) (Figure 4A), consistent with previous findings.[28] This suggests that TyG-BMI can effectively predict the risk of T2DM complicated with MASLD, which may be attributed to its calculation methodology. These parameters (blood glucose, triglycerides, and BMI) are key components of glucose and lipid metabolism; by integrating metabolic and anthropometric parameters, TyG-BMI enables a more comprehensive assessment of metabolic function than any single indicator alone. Additionally, previous studies have shown that hyperglycemia, obesity, and elevated triglyceride levels are closely linked to hepatic lipid deposition. This is because prolonged exposure to "lipotoxicity and glucotoxicity" ultimately induces IR, impairs normal adipocyte function, and ultimately drives the development and progression of MASLD.[29]

This study further constructed a predictive model based on HOMA2 IR-CP and TyG-BMI. The results showed that this model achieved a maximum AUC of 0.805 (95% CI: 0.749–0.860), indicating good discriminatory ability and calibration performance (Figures 4 and 5). The data required for calculating HOMA2 IR-CP and TyG-BMI are common, easily accessible clinical laboratory or measurement indicators. This not only reduces the economic burden on patients but also simplifies the complexity of clinical operations. Even in medical institutions with limited examination equipment—such as primary care hospitals or community health centers—these indicators can be used to assess and predict the risk of MASLD in T2DM patients. This expands the scope of the population eligible for screening and confirms the model's strong clinical applicability.

Meanwhile, this study further demonstrated via RCS analysis that HOMA2 IR-CP exhibited a non-linear association with MASLD (P for non-linearity < 0.05), whereas TyG-BMI showed a linear association (P for non-linearity = 0.139). No significant interaction effects were observed across subgroups stratified by gender, age, BMI, or HbA1c level (all P for interaction > 0.05); however, the associations were more prominent in patients with BMI < 28 kg/m^2 and those with

HbA1c ≥ 7% (indicating poor glycemic control). Regardless of age, gender, glycemic status, or weight category (normal weight/obesity), T2DM patients with elevated HOMA2 IR-CP or TyG-BMI had a significantly increased risk of MASLD. This finding expands the model's applicability, as it eliminates the need to adjust predictive thresholds across different subgroups and simplifies clinical operations—making it particularly suitable for "one-stop screening" in primary care hospitals. Notably, the stratified analysis revealed a higher MASLD risk in patients with BMI < 28 kg/m^2, suggesting that liver health should be prioritized even in non-obese T2DM populations. A meta-analysis reported that the incidence of MASLD in non-obese individuals in mainland China was 30.5%, significantly higher than the 15.7% incidence in the broader Asian population; this discrepancy may be associated with the high prevalence of central obesity among Chinese individuals.[19] For T2DM patients, long-term insulin therapy can lead to increased waist circumference, accelerating the development of central obesity. Additionally, the stratified analysis showed that patients with poor glycemic control (HbA1c ≥ 7.0%) had a higher MASLD risk, which contradicts the findings of Xinyu Ha et al,[30] who observed a weakened association between HbA1c and MASLD when HbA1c exceeded 8.0%. This inconsistency may be attributed to differences in the medication status of study participants. All patients in the present study were T2DM patients receiving insulin for glycemic control, and long-term insulin therapy often leads to weight gain—particularly visceral fat accumulation. Visceral fat acts as a source of inflammatory cytokines and free fatty acids, which interfere with insulin signaling in muscle and liver tissues, directly or indirectly exacerbating insulin resistance and promoting the development and progression of MASLD. In contrast, the study by Xinyu Ha et al did not account for medication use in diabetic patients; many commonly used antidiabetic drugs can reduce body weight or insulin resistance, thereby lowering the incidence of MASLD. Therefore, in the long-term management of T2DM patients, clinical practice should not only focus on glycemic control but also consider the potential risk of obesity and insulin resistance associated with certain medications. Furthermore, it is critical to correct the misconception that only obese T2DM patients require MASLD screening; non-obese T2DM patients should also be prioritized for MASLD screening to reduce the rate of missed diagnoses.

Therefore, we propose that in clinical practice, T2DM patients with "BMI < 28 kg/m^2, HbA1c ≥ 7%, and HOMA2 IR-CP≥1.38, TyG-BMI≥223.33" should be categorized as an "MASLD ultra-high-risk group" and prioritized for confirmation via hepatic imaging or FibroScan or liver biopsy, this provides clinicians with an actionable threshold to prioritize high-risk patients. Meanwhile, intervention strategies should simultaneously target "glycemic control" and "improvement of insulin resistance/glucose-lipid metabolism"—for example, by using glucagon-like peptide-1 receptor agonists (GLP-1 RAs), which not only lower blood glucose but also reduce hepatic fat. This avoids overemphasis on blood glucose management while neglecting liver protection.

Limitations

This study has several limitations. First, the diagnosis of MASLD relied on imaging modalities rather than the histological gold standard of liver biopsy, which may result in under detection of early-stage or mild steatohepatitis. Second, the data were derived from a single-center Asian cohort; given known variations in genetic background, lifestyle, and dietary habits across regions and ethnic groups, the generalizability of our findings to other populations requires further validation. Furthermore, the modest sample size may constrain the robustness of the statistical models and the reliability of the estimates. Future studies involving larger, multi-center cohorts with diverse demographic and clinical profiles are needed to validate the applicability and predictive performance of HOMA2 IR-CP and TyG-BMI across various healthcare settings.

Conclusions

Our study demonstrates that in T2DM patients receiving exogenous insulin for glycemic control, TyG-BMI and HOMA2 IR-CP are independent risk factors for MASLD. The nomogram model constructed based on these two indicators shows that both exhibit good predictive performance, with TyG-BMI demonstrating superior predictive ability and achieving the highest AUC (0.805). Therefore, TyG-BMI and HOMA2 IR-CP can serve as simple and effective predictive markers in clinical practice.

Data Sharing Statement

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

Ethics Approval and Consent to Participate

The study protocol was reviewed and approved by the Ethics Committee of Chongqing Jiangjin District Hospital of Chinese Medicine (Approval No. ZYY2025012). Written informed consent was obtained from all participants prior to their enrollment in the study.

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Author Contributions

Qiumei Cao: Conceptualization, Investigation, Writing – Original Draft, Funding Acquisition; Qiuhui Tian: Methodology, Software, Formal Analysis, Data Curation, Writing – Review & Editing; Yu Liu: Validation, Investigation, Writing – Review & Editing; Yuzhu Cheng: Methodology, Formal Analysis, Writing – Review & Editing; Ting Luo: Resources, Data Curation, Writing – Review & Editing; Xiaolin Zhu: Supervision, Writing – Review & Editing. All authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no competing interests.

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