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Association Between TCBI (Triglycerides, Total Cholesterol, and Body Weight Index) and Hyperuricemia：a retrospective cross-sectional study

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Abstract

Background: The triglycerides, total cholesterol, and body weight index (TCBI) is a novel biomarker that integrates nutritional and metabolic status. Its potential association with hyperuricemia (HUA) remains unclear. This study aimed to investigate the independent association between TCBI and HUA.

Methods: We conducted a retrospective cross-sectional study of 3,775 participants (708 HUA cases and 3,067 controls). The association of standardized TCBI with HUA was assessed using univariable and multivariable logistic regression (adjusting for age, sex, BMI, glucose, and LDL-C), quartile analysis, and restricted cubic splines (RCS). Stratified analyses and interaction tests were performed by sex and BMI. Predictive utility was evaluated by the area under the curve (AUC), net reclassification improvement (NRI), and integrated discrimination improvement (IDI).

Results: After full adjustment, each standard deviation increase in TCBI was independently associated with a 13.5% higher HUA risk (OR = 1.135, 95% CI: 1.033 - 1.271, P=0.017). A significant, non-linear dose-response relationship was observed ($P < 0.001$). Stratified analyses revealed a stronger association in women and a significant modifying effect of BMI ($P = 0.03$). Adding TCBI to a base model modestly improved discrimination ($\Delta AUC = 0.005$, $P < 0.001$) and significantly enhanced risk reclassification (NRI = 14.97%, P = 0.019).

Conclusion: TCBI is an independent, non-linear risk factor for HUA, with effect modification by sex and BMI. Its ability to significantly improve risk reclassification positions TCBI as a simple and clinically valuable indicator for refining HUA risk

stratification, warranting validation in prospective cohorts.

Introduction

Hyperuricemia (HUA) is a global common metabolic disease characterized by elevated serum uric acid levels. It is considered to be a major cause of gout and gouty arthritis [1]. Beyond this classical association, evidence is accumulating that HUA is an independent risk factor for a range of chronic diseases, including hypertension, chronic kidney disease, and cardiovascular diseases [2]. The pathophysiology involves uric acid-induced endothelial dysfunction, activation of the renin-angiotensin system, and chronic low-grade inflammation [3,4]. The incidence of HUA is on the rise, which is in line with the global trend of metabolic syndrome[5]. Therefore, there is an urgent need for comprehensive indicators that can reflect the potential nutritional status and accumulated metabolic burden. This will greatly refine the risk stratification of HUA and enhance prevention and treatment strategies.

The triglycerides, total cholesterol, and body weight index (TCBI) are composite nutritional indicators calculated as TG (mg/dL) × TC (mg/dL) × body weight (kg) / 1000 [6,7]. Unlike traditional indices such as body mass index (BMI), which mainly reflects overall adiposity, the TCBI combines information on body weight and circulating lipid profiles (triglycerides and total cholesterol). This unique component reflects a state of integrated metabolic overload. Therefore, the TCBI has been proposed and validated in recent years as a practical surrogate for assessing overall metabolic and nutritional status, with predictive value for the outcome of cardiovascular and other diseases [8].

There is a biological relationship between TCBI and HUA. Obesity is a key component of TCBI, which leads to hyperuricemia through multiple mechanisms, including increased uric acid production due to accelerated purine cycling and decreased renal excretion due to insulin resistance [9]. At the same time, the elevation of triglycerides, another core factor of TCBI, is also frequently observed in patients with HUA and is a marker of the insulin-resistant, a state that impairs renal uric acid excretion [10]. The enzyme xanthine oxidase, which is involved in purine metabolism, is also intricacies linked to lipid metabolism pathways [11,12]. Thus, by simultaneously capturing these two interrelated risk factors, TCBI may provide more comprehensive and effective insight into HUA risk than any single parameter.

Despite the established roles of its components and its evaluation in other metabolic contexts, the specific relationship between TCBI and hyperuricemia remains unexplored. A comprehensive investigation is warranted to determine whether this integrated marker holds independent and significant value in the context of HUA. Given that previous studies on other metabolic risk factors have suggested potential variations in effect size by sex and BMI levels, we have conducted further exploration in this regard.

Methods

Study Design and Population

This cross-sectional study was conducted using electronic medical records of patients admitted to the Department of Endocrinology and Metabolism, Tianjin Medical University General Hospital, between October 2021 and March 2023.

Inclusion criteria were: (1) Age ≥ 18 years; (2) Complete laboratory and anthropometric data required for calculating TCBI and defining HUA; (3) Availability of key covariates (age, sex, BMI, and glucose parameters). Exclusion criteria included: (1) Severe renal dysfunction (estimated glomerular filtration rate < 30 mL/min/1.73 m^2); (2) Use of diuretics, uricosuric agents, or other medications affecting uric acid levels [13]; (3) Pregnancy or lactation; (4) Patients who failed to meet any of the aforementioned inclusion criteria.

Through this systematic screening process (***Figure 1***), a total of 3,775 eligible inpatients were included in the final analysis. Participants were definitively classified into HUA (N = 708) and non-HUA (N = 3,067) groups based on serum uric acid levels, using the standardized diagnostic criteria defined by the Chinese Multidisciplinary Expert Consensus on Hyperuricemia[14].

Data Collection

Demographic information, anthropometric measurements, and laboratory data were extracted from the hospital electronic medical record system. Height and weight measured at admission were used to calculate BMI. Laboratory parameters were obtained from the first fasting blood sample after admission (≥ 8-hour fast) and analyzed in the central laboratory using standardized procedures.

The laboratory parameters related to this study included blood uric acid (UA), lipid profile (total cholesterol [TC], triglycerides [TG], low-density lipoprotein cholesterol [LDL-C]), and fasting plasma glucose (FPG).

The TCBI was calculated as a composite nutritional marker using the validated

formula: TCBI = TG (mg/dL) × TC (mg/dL) × body weight (kg) / 1000, as established in previous methodological literature [6].

Statistical Analysis

All statistical analyses were performed using R software (version 4.4.1). Continuous variables were presented as mean ± standard deviation or median (interquartile range) based on their distribution, and compared using Student's t-test or Mann-Whitney U test as appropriate. Categorical variables were expressed as numbers (percentages) and compared using Chi-square test.

The association between TCBI and HUA was evaluated using two primary logistic regression approaches: (1) univariable analysis; (2) multivariable analysis fully adjusted for age, sex, BMI, glucose, and LDL-C. TCBI was analyzed as a continuous variable (per 1-standard deviation increase) and results were expressed as odds ratios (ORs) with 95% confidence intervals (CIs).

For dose-response analysis, TCBI was categorized into quartiles and tested for trend. Nonlinearity was assessed using restricted cubic splines (RCS) with four knots (rms package). Stratified analyses were performed by sex and BMI (median-based), and interactions were tested using multiplicative terms.

Predictive performance was evaluated using the area under the receiver operating characteristic curve (AUC). Incremental predictive value was assessed using net reclassification improvement (NRI) and integrated discrimination improvement (IDI) with 1,000 bootstrap resamples. Nomograms and calibration curves were constructed and internally validated using bootstrap resampling (1,000 iterations). Two-sided P <

0.05 was considered statistically significant.

Results

Baseline Characteristics of the Study Population

A total of 3,775 participants were included in the final analysis cohort, which was divided into the HUA group (N = 708, 18.8%) and the non-HUA group (N = 3067, 81.2%) according to serum uric acid level. The baseline characteristics of the study population are shown in ***Table 1***. The two groups differed significantly on multiple demographic and clinical variables, reflecting different metabolic profiles.

Compared with the non-HUA group, the HUA group was on average younger and had a higher proportion of males. In addition, they exhibited a metabolic phenotype consistent with known risk factors for uric acid dysregulation: significantly higher BMI and LDL-C levels (both $P < 0.001$). Importantly, the core independent variable TCBI was also significantly higher in the HUA group than in the non-HUA group ($P < 0.001$).

Univariate correlations between TCBI and other variables

Spearman correlation analysis showed that TCBI was significantly correlated with multiple clinical and laboratory parameters (***Table S1***). TCBI was strongly positively correlated with triglycerides ($r = 0.881$, $P < 0.001$) and moderately correlated with BMI ($r = 0.534$, $P < 0.001$). A moderate positive correlation was also observed with serum uric acid ($r = 0.404$, $P < 0.001$), whereas HDL-C was negatively correlated ($r = -0.405$, $P < 0.001$).

TCBI showed weaker but significant positive correlations with LDL-C, fasting

plasma glucose, and several hematological parameters, including RBC and hemoglobin (all $P < 0.05$). Age was negatively correlated with TCBI ($r = -0.362$, $P < 0.001$). No significant correlation was observed between TCBI and HbA1c ($P = 0.108$).

Association Between TCBI and HUA

Logistic regression analyses were performed to evaluate the association between TCBI and hyperuricemia, and full model results are presented in ***Table 2***. The initial univariate analysis revealed a strong, unadjusted relationship. For every one standard deviation (SD) increase in the TCBI value, the odds of having HUA increased by 63% (OR = 1.63, 95% CI: 1.447 - 1.841, $P < 0.001$). Logistic regression models were used to assess the association between TCBI and HUA.

To ascertain whether TCBI was an independent risk factor distinct from conventional metabolic parameters, we constructed a multivariate logistic regression model. This model adjusted for a predefined set of potential confounders: age, sex, BMI, fasting glucose, and LDL-C. After accounting for the influence of these variables, TCBI remained a statistically significant and independent predictor of HUA. The adjusted analysis demonstrated that each SD increase in TCBI was associated with a 13.5% increase in the odds of HUA (OR = 1.135, 95% CI: 1.033 - 1.271, $P = 0.017$). The results of this analysis are visually summarized in ***Figure 2***.

Dose-Response Relationship Between TCBI and HUA

To further elucidate the nature of this association, we examined the risk of HUA at different TCBI levels. The subjects were grouped according to the quartile value of

TCBI, and the first quartile (Q1) represented the lowest TCBI value and served as the reference group.

A clear dose-response relationship was observed. Compared with Q1, the adjusted risk of HUA in Q2 (OR = 1.54), Q3 (OR = 2.59) and Q4 (OR = 3.73) increased gradually. The p value for trend was significant (P for trend < 0.001) (***Figure S1***).

Non-Linear Association Between TCBI and HUA

Considering the complexity of the physiological mechanisms of metabolic disorders, we used RCS analysis to explore whether there was a nonlinear relationship between TCBI and HUA risk. Analyses showed that the association did exist (P < 0.0001 for a nonlinear test). As shown in ***Figure 3***, as TCBI increased from a low level to 0.98, the risk of HUA increased rapidly, and the peak of the whole curve was reached when TCBI was about 1. The curve then showed a slight downward trend, with a slight decrease in the predicted risk as the TCBI value increased, but it remained relatively stable overall.

Restricted cubic spline analysis demonstrated a significant nonlinear association between TCBI and HUA (P for nonlinearity < 0.001). The risk of HUA increased rapidly with increasing TCBI and reached an inflection point at a standardized TCBI value of approximately 0.98. The corresponding original TCBI value was 7273.44, after which the curve showed a plateau with a slight downward trend.

Stratified and Interaction Analyses

To assess the consistency of the association of TCBI with HUA across subgroups,

we performed stratified and interaction analyses by sex and BMI category (***Table 3***). In the sex-stratified analysis, the point estimates suggested an underlying trend: Each standard deviation increase in TCBI was associated with an OR of 1.18 (95%CI: 1.011 - 1.487) in women, but not in men (OR = 1.09, 95%CI: 0.977 - 1.249). The interaction analysis between TCBI and gender was not statistically significant (P = 0.70 for interaction).

In contrast, there was a significant interaction between TCBI and BMI (P = 0.03 for interaction). Stratified analysis showed that the positive correlation between TCBI and HUA was more significant in the low BMI group (OR = 1.30, 95%CI: 1.063 - 1.681) than in the high BMI group (OR = 1.14, 95%CI: 1.017 - 1.314).

Predictive Performance of TCBI

We evaluated the potential clinical utility of adding TCBI to the model to predict HUA. The base model included age, gender, BMI, glucose, and LDL-C, and its AUC was 0.7241. When the standardized TCBI was included in the model, the AUC increased to 0.7292. The difference was statistically significant (P < 0.001), but the improvement in the discrimination ability of the model was limited (***Figure S2***).

Further evaluation using reclassification index showed that the NRI was 14.97% (P = 0.019), indicating that the inclusion of TCBI significantly improved the risk reclassification ability. However, the IDI was small and not statistically significant (IDI = 0.0015, P = 0.5329).

To translate the model into a practical tool, we developed a nomogram to visualize the contribution of each predictor to the estimated risk of individual HUA

(***Figure 4***). Finally, the calibration curve of the final model showed a high degree of agreement between the predicted probability of HUA and the observed frequency (***Figure 5***).

Discussion

In this large cross-sectional study, we found a significant association between TCBI and HUA, and this association persisted even after adjusting for known metabolic risk factors (including age, gender, BMI, glucose, and LDL-C). This finding positions TCBI as an independent metabolic marker for HUA [15]. After eliminating the influence of BMI, the association still existed, suggesting that TCBI captures metabolic risks beyond simple obesity, reinforcing the complex interrelationships between dyslipidemia, body composition, and purine metabolism [16].

Unlike traditional adiposity indicators, TCBI integrates circulating lipid levels and body mass simultaneously, which may better reflect the metabolic load imposed on hepatic lipid handling and systemic energy metabolism. Emerging evidence indicates that hyperuricemia is not only a purine metabolic disorder but also a manifestation of systemic metabolic stress characterized by ectopic lipid deposition and mitochondrial dysfunction [17,18]. Therefore, the persistence of the association after BMI adjustment suggests that metabolic quality, rather than body size alone, may be the key determinant linking TCBI and uric acid metabolism.

One of the detailed findings in our study is that there is a significant nonlinear association between standardized TCBI and the risk of HUA. This association does

not follow a typical U-shaped pattern but rather shows a rapid increase in the risk of HUA at low TCBI levels, followed by a clear peak, and then a slight decrease but overall stability. This indicates that the metabolic pathways between the two are more complex than a simple linear dose-response relationship. Importantly, the nonlinear curve revealed a clear threshold effect, with the risk peak occurring at a TCBI value of approximately 7273. This finding suggests that the relationship between metabolic burden and uric acid homeostasis may not increase indefinitely but instead reaches a saturation state once metabolic overload exceeds a certain level. The elevated risk observed in the mid-range of TCBI may reflect the coexistence of hypertriglyceridemia, increased adiposity, and insulin resistance—conditions known to enhance renal urate reabsorption and increase uric acid production [19,20].

Mechanistically, hyperinsulinemia stimulates sodium–hydrogen exchanger activity in the renal proximal tubule and upregulates urate transporters (URAT1 and GLUT9), thereby reducing uric acid excretion [21]. In addition, elevated triglycerides reflect hepatic de novo lipogenesis, which increases ATP turnover and accelerates purine degradation, ultimately leading to excessive uric acid generation [22]. These pathways together may explain why the HUA risk rises steeply once TCBI reaches the metabolic overload range.

Conversely, the high HUA risk observed at low TCBI values may represent a relative metabolic or nutritional weakness state, reflecting a decrease in muscle mass [23]. Because skeletal muscle is the main organ for glucose metabolism and metabolic homeostasis. Lower muscle mass has an independent association with decreased

insulin sensitivity, which may cause the kidneys to have difficulty excreting uric acid substances even in the absence of significant obesity [24]. Additionally, skeletal muscle acts as an important reservoir for glucose disposal and lactate metabolism. Reduced muscle mass may promote compensatory hepatic gluconeogenesis and lactate accumulation, both of which compete with uric acid excretion through renal organic anion transporters [25,26]. This provides a plausible explanation for the paradoxical elevation of uric acid even in individuals without overt obesity but with low metabolic reserve. However, this explanation should be treated with caution as TCBI is not a direct measure of muscle mass. In summary, there is metabolic overload at moderate TCBI levels, and potential metabolic deficiency at lower levels, both of which may lead to elevated uric acid levels. Further assessment of body composition and detection of metabolic markers will help clarify the underlying mechanisms. Future studies incorporating dual-energy X-ray absorptiometry or bioimpedance analysis are necessary to distinguish fat mass from lean mass contributions to the observed nonlinear pattern [27].

Our stratified analyses provided further insights into the association's modifiers. Contrary to the initial unstratified view, the association was not significantly modified by sex, as indicated by the non-significant interaction term ($p=0.70$). Therefore, while point estimates differed, we cannot conclusively state that the association is stronger in females. This finding underscores the importance of formally testing for interaction rather than relying solely on stratified point estimates. However, a significant interaction was confirmed with BMI. The stronger association between TCBI and

HUA in individuals with a low BMI is particularly noteworthy. This suggests that in leaner individuals, TCBI may potentially capture a crucial aspect of adverse metabolic health—such as high triglyceride levels relative to their body size (i.e., "normal-weight obesity" or low muscle mass)—that is not reflected by BMI alone [28]. In individuals with high BMI, where obesity is already an overt and dominant risk factor, the additional information provided by TCBI, while still significant, may be relatively less impactful.

This phenomenon supports the concept of metabolically unhealthy normal weight, in which ectopic fat deposition and lipid overflow exist despite a normal BMI and are strongly associated with cardiometabolic disorders including hyperuricemia [29,30].

From a clinical perspective, these predictive performance indicators have enhanced the potential application value of TCBI. Although the absolute increase in the AUC is not significant, it is statistically significant, which confirms that TCBI adds discriminatory ability to the baseline model of conventional risk factors [31]. More importantly, the statistically significant NRI indicates that TCBI effectively improves the ability to correctly reclassify individuals into their true risk categories, especially for HUA [32]. This is a key indicator for clinical application, as it indicates that TCBI can refine risk stratification and potentially identify high-risk individuals who cannot be detected through traditional indicators such as BMI. The developed nomogram, as a practical and intuitive tool, converts these statistical results into clinical practice [33], and can quantify an individual's risk of HUA based on the

combined contribution of TCBI and other easily accessible factors.

Given that triglyceride and cholesterol measurements are routinely available in clinical practice, TCBI may serve as a low-cost screening indicator for metabolic hyperuricemia risk, particularly in general endocrinology outpatient settings [34].

This study has several limitations. Firstly, the cross-sectional design cannot make causal inferences about the relationship between TCBI and HUA. A prospective cohort study is needed to confirm whether TCBI at baseline can predict the development of HUA in the future [9]. Secondly, the research subjects come from a single center, and its generalizability needs to be verified through external studies to determine if it is applicable to other races or groups with different genetic backgrounds and lifestyles. Finally, this study lacks mechanistic research to explore the proposed hypotheses, especially regarding the role of muscle mass and insulin sensitivity in the observed non-linear peak relationship.

Conclusion

In conclusion, this large-scale cross-sectional study confirmed a significant association between TCBI and HUA, even after adjusting for risk factors. There is also a non-linear relationship between the two. When the TCBI level is low, the risk of HUA rises rapidly and reaches a peak before stabilizing. This association is more pronounced in individuals with a low BMI, suggesting that TCBI has a unique role in identifying adverse metabolic risks. It may reflect factors that BMI alone cannot capture, such as visceral fat or low muscle mass. Although this cross-sectional design cannot make causal inferences, our research results demonstrate that TCBI is a

promising comprehensive indicator. It is necessary to conduct prospective studies in different populations and further clarify its underlying mechanisms.

Abbreviations

Abbreviation	Full name
ATP	Adenosine Triphosphate
AUC	Area Under the Curve
BMI	Body Mass Index
CI	Confidence Interval
FPG	Fasting Plasma Glucose
GLUT9	Glucose Transporter 9
HbA1c	Hemoglobin A1c
HDL-C	High-Density Lipoprotein Cholesterol
HGB	Hemoglobin
HUA	Hyperuricemia
IDI	Integrated Discrimination Improvement
LDL-C	Low-Density Lipoprotein Cholesterol
NRI	Net Reclassification Improvement
OR	Odds Ratio
PLT	Platelet Count
RBC	Red Blood Cell Count
RCS	Restricted Cubic Splines
SD	Standard Deviation
TC	Total Cholesterol
TCBI	Triglycerides, Total Cholesterol, and Body Weight Index

Abbreviation	Full name
TG	Triglycerides
UA	Uric Acid
URAT1	Urate Transporter 1
WBC	White Blood Cell

Declarations

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Competing Interests

No potential conflict of interest relevant to this article was reported.

Availability of data and materials

Data were obtained from the corresponding author.

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of Tianjin Medical University General Hospital (approval number: IRB2020-YX-027-01) in accordance with the Declaration of Helsinki. Informed consent was not obtained because the patient data were obtained from the electronic medical records of the Department of Endocrinology and Metabolism and the patients' identities were anonymized except for the date of birth.

Consent to publication

All authors have consented to the publication of this article.

Authors' contributions

Wang Meng: Formal analysis; Investigation; Methodology; Writing-review & editing.

Cui Qianhui: Data curation; Writing - original draft; Software.

Cui Jingqiu: Conceptualization; Funding acquisition; Project administration.

All authors read and approved the final manuscript.

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AI Disclosure

No generative Artificial Intelligence (AI) tools were used in the preparation of the written or visual content of this manuscript.

Clinical trial number

Not applicable.

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Table 1. Comparison of Baseline Data Between With_HUA Group and Without_HUA Group.

Variables	With_HUA(N = 708)	Without_HUA(N = 3,067)	P value
Male	446 (63%)	1473 (48%)	<0.001
Age	44 (32,61)	59 (46,68)	<0.001
BMI (kg/m²)	28.58 (25.3,33.8)	25.62 (23.05,28.6)	<0.001
WBC (10^9/L)	6.81 (5.8,8.16)	6.24 (5.19,7.47)	<0.001
RBC (10^{12}/L)	4.745 (4.34,5.11)	4.47 (4.09,4.86)	<0.001
HGB(g/L)	140 (128,152)	133 (122,145)	<0.001
PLT (10^9/L)	239 (198,283)	226 (187,269)	<0.001
TC (mmol/L)	5.93 (4.39,6.32)	6.1 (4.82,6.32)	0.381
TG (mmol/L)	2.01 (1.49,2.91)	1.53 (1.12,2.2)	<0.001
HDL-C(mmol/L)	0.97 (0.86,1.11)	1.06 (0.9,1.25)	<0.001
LDL-C(mmol/L)	3.08 (2.54,3.67)	2.89 (2.29,3.52)	<0.001
TCBI	3453.06 (2265.81,5045.59)	2087.46 (1359.27,3309.19)	<0.001
Urea(mmol/L）	5 (3.9,6.2)	5 (4.1,6.3)	0.173
Scr (umol/L)	67.5 (54,84)	59 (48,71)	<0.001
UA (umol/L)	456 (414,509)	306 (250,363)	<0.001
FPG (mmol/L)	5.2 (4.5,6.77)	5.9 (4.8,7.9)	<0.001
HbA1c(%)	6.4 (5.8,7.9)	7.3 (6.2,9)	<0.001

Abbreviations: BMI, Body Mass Index; WBC, White Blood Cell count; RBC, Red Blood Cell count; HGB, Hemoglobin; PLT, Platelet count; TC, Total Cholesterol; TG, Triglycerides; HDL-C, High-Density Lipoprotein Cholesterol; LDL-C, Low-Density Lipoprotein Cholesterol; TCBI, Triglycerides, total cholesterol, and body weight index; Scr, Serum Creatinine; UA, Uric Acid; FPG, Fasting Plasma Glucose; HbA1C, Hemoglobin A1c.

Table 2. Logistic regression analyses for Hyperuricemia.

Variable	Univariable OR (95% CI)	P value	Multivariable OR (95% CI)	P value
TCBI (per SD)	1.63 (1.45,1.84)	<0.001	1.14 (1.03,1.27)	0.017
LDL-C (mmol/L)	-	-	1.11 (1.00,1.22)	0.042
BMI (kg/m^2)	-	-	1.07 (1.06,1.09)	<0.001
Age	-	-	0.98 (0.97,0.98)	<0.001
Glucose (mmol/L)	-	-	0.89 (0.85,0.92)	<0.001
Female (vs male)	-	-	0.51 (0.42,0.61)	<0.001

Abbreviations: OR, Odds Ratio; CI, Confidence Interval; SD, Standard Deviation; TCBI, Triglycerides, total cholesterol, and body weight index; LDL-C, Low-Density Lipoprotein Cholesterol; BMI, Body Mass Index; HUA, Hyperuricemia.

Table 3. Stratified analysis of the association between TCBI and Hyperuricemia.

Subgroup	OR	95% CI	P value	P for interaction
Sex				0.70
Male	1.09	0.98,1.25	0.17	
Female	1.18	1.01,1.49	0.09	
BMI category				0.03
Low BMI	1.30	1.06,1.68	0.04	
High BMI	1.14	1.02,1.31	0.05	

Abbreviations: BMI, Body Mass Index; CI, Confidence Interval; OR, Odds Ratio.

Table S1. Spearman correlations between TCBI and clinical variables.

Variable	Spearman r	P value
TG (mmol/L)	0.881	<0.001
BMI (kg/m²)	0.534	<0.001
HDL-C (mmol/L)	-0.405	<0.001

Variable	Spearman r	P value
UA (umol/L)	0.404	<0.001
Age	-0.362	<0.001
RBC (10^{12}/L)	0.341	<0.001
HGB (g/L)	0.313	<0.001
TC (mmol/L)	0.295	<0.001
WBC (10^9/L)	0.231	<0.001
LDL-C (mmol/L)	0.225	<0.001
Gender	0.138	<0.001
FPG (mmol/L)	0.114	<0.001
PLT (10^9/L)	0.089	<0.001
Urea (mmol/L）	-0.087	<0.001
Scr (umol/L)	0.046	0.016
HbA1c (%)	0.031	0.108

485 Abbreviations: TG, Triglycerides; BMI, Body Mass Index; HDL-C, High-Density Lipoprotein
486 Cholesterol; UA, Uric Acid; RBC, Red Blood Cell count; HGB, Hemoglobin; TC, Total
487 Cholesterol; WBC, White Blood Cell count; LDL-C, Low-Density Lipoprotein Cholesterol; FPG,
488 Fasting Plasma Glucose; PLT, Platelet count; Scr, Serum Creatinine; HbA1c, Hemoglobin A1c.
489

Figure 1. Flowchart of Patient Screening and Enrollment.

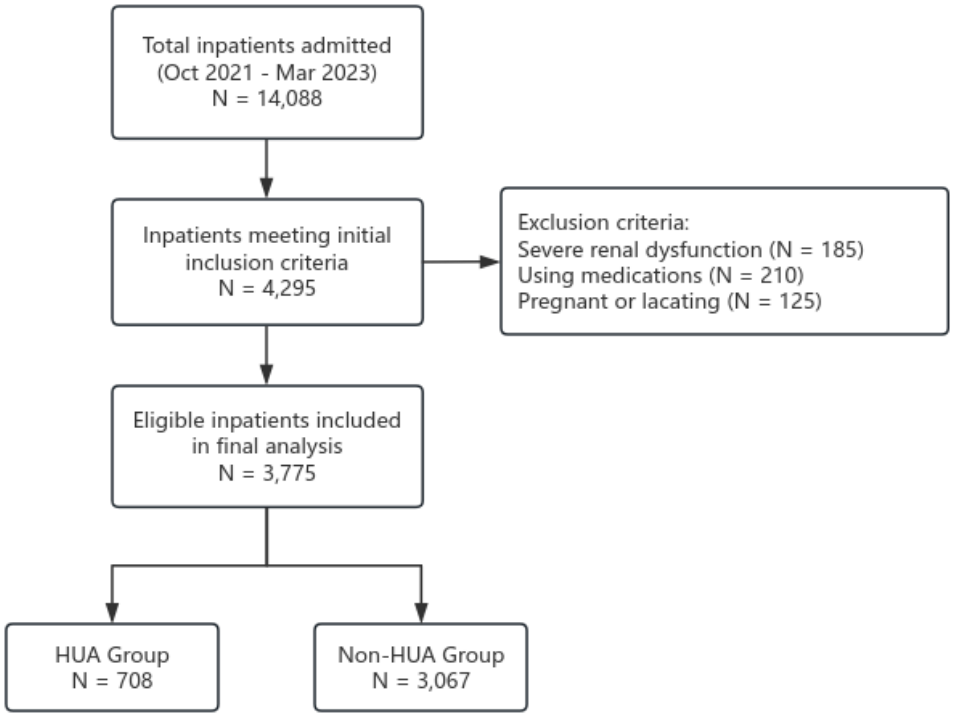


Figure 2. Multivariable logistic regression analysis of HUA risk factors.

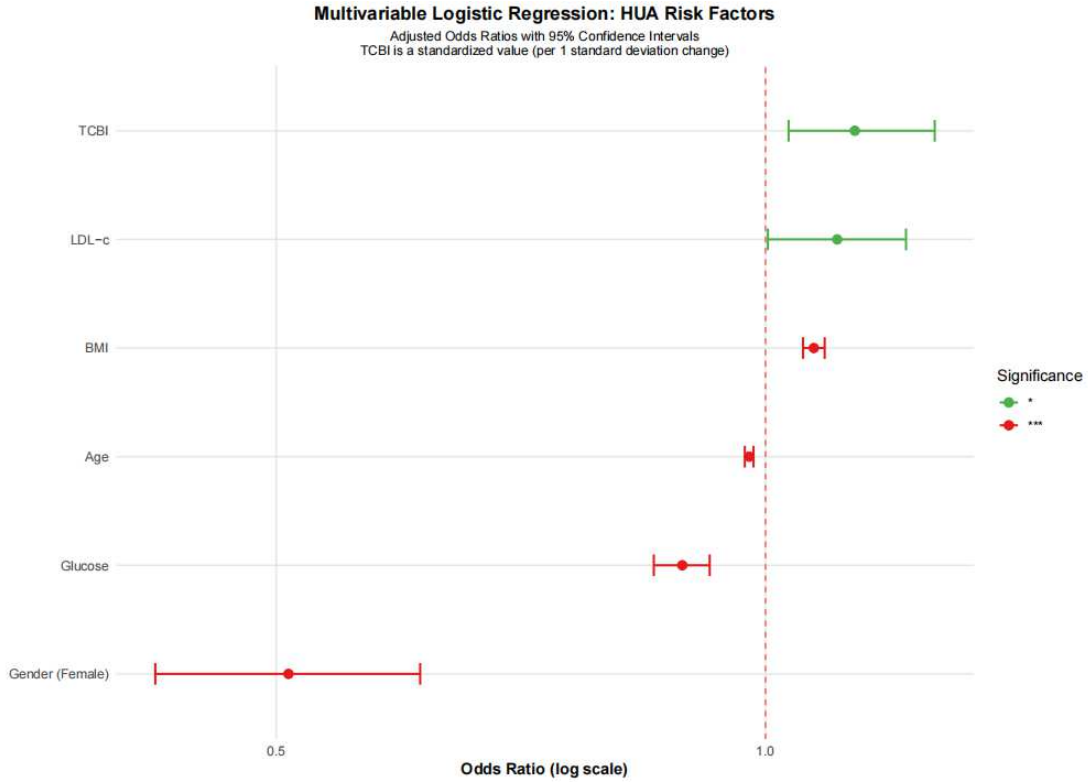


Figure 3. Dose–response relationship between TCBI and HUA risk using restricted cubic spline analysis.

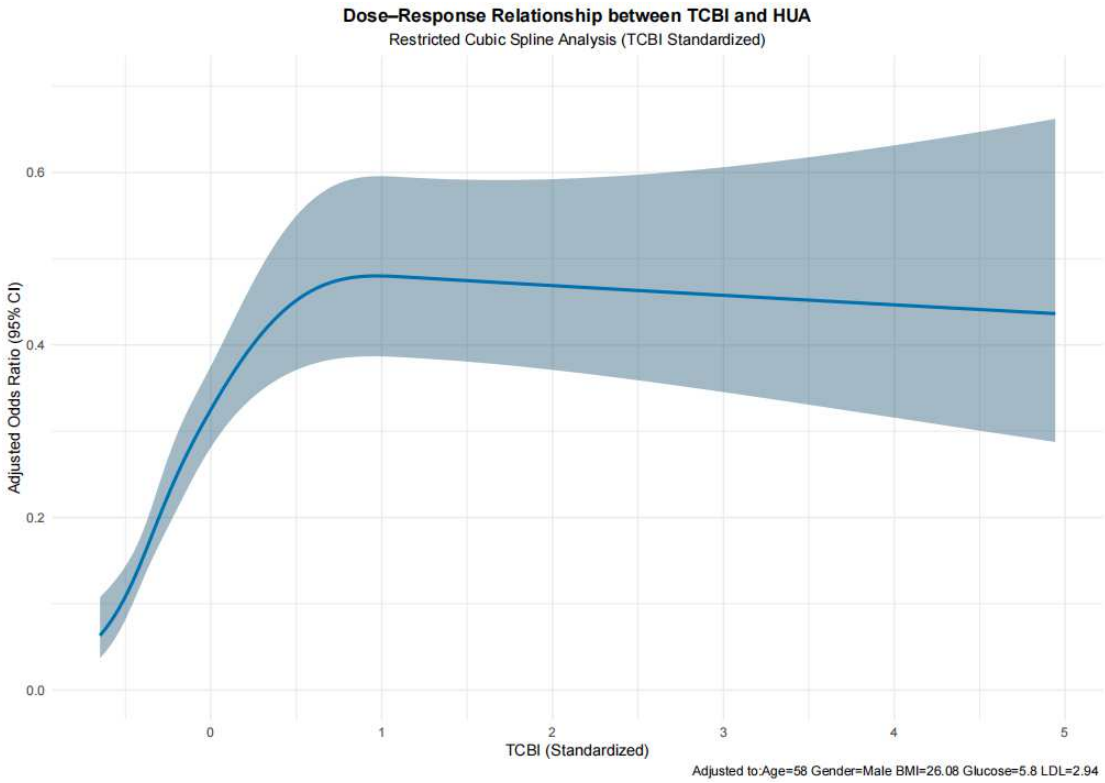
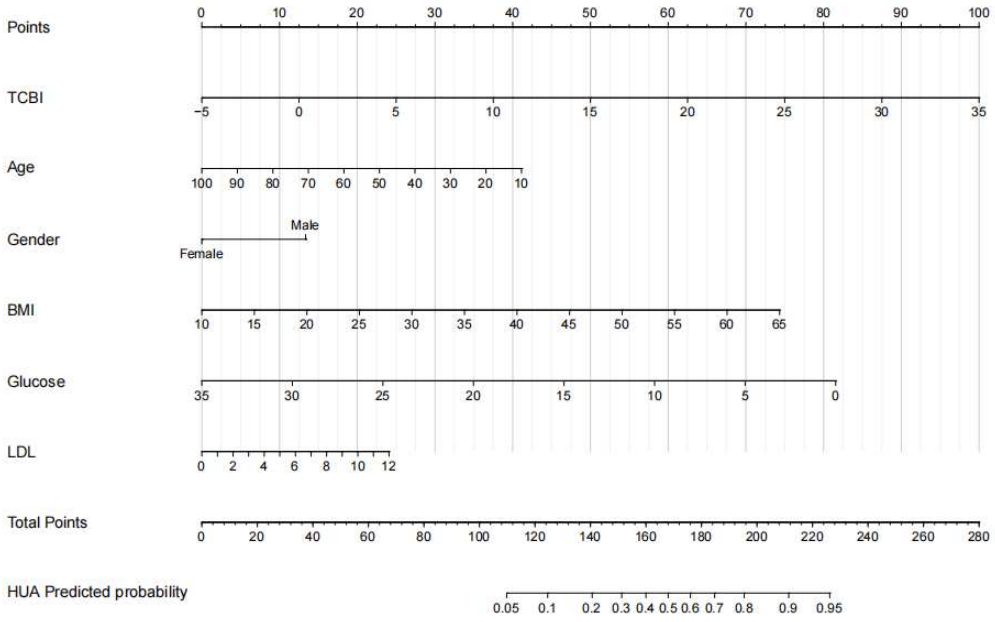
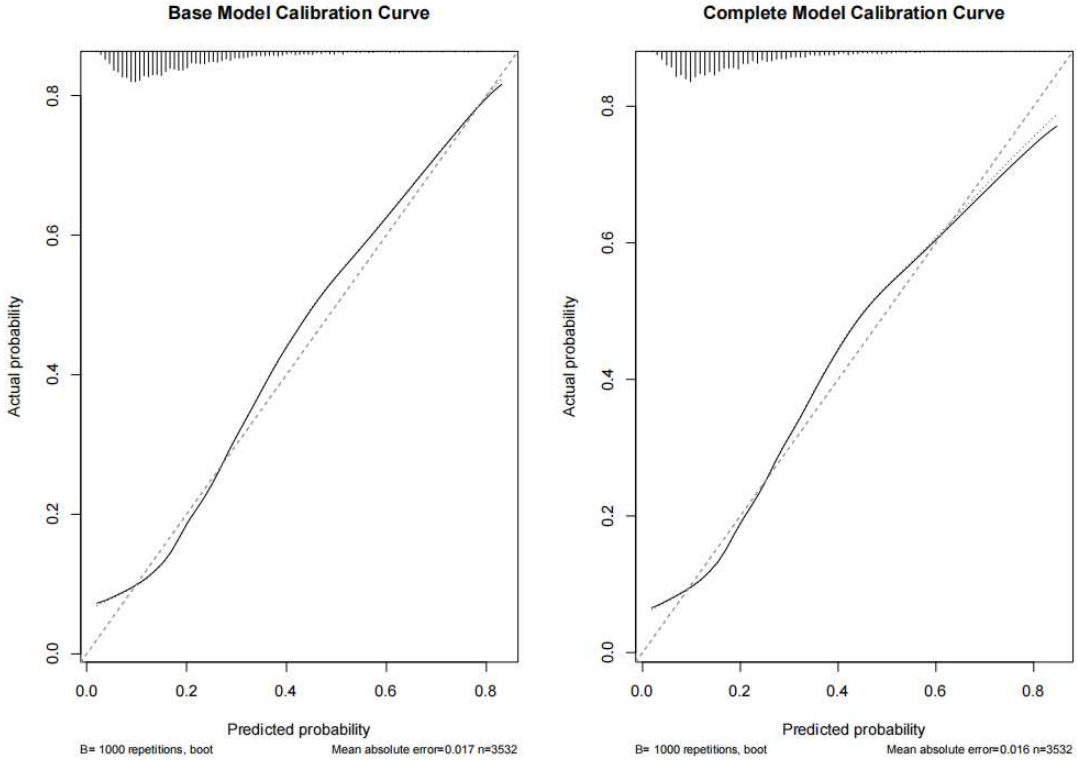


Figure 4. Nomogram for predicting the probability of HUA.



500 Figure 5. Calibration curves for the base model and the complete model (with TCBI).



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Supplementary Files

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- supplementaryfile.docx