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ORIGINAL RESEARCH

Association Between Triglyceride-Glucose Index and Gout in Young Adults in China

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Objective: Gout is a common inflammatory arthritis with a rising prevalence among young adults in China, imposing a substantial burden on patients and society. This study aims to examine the association between the triglyceride-glucose (TyG) index and the presence of gout among young Chinese adults.

Methods: We conducted a retrospective, age- and sex-matched (1:4) case-control study at Meizhou People's Hospital (2019–2023). Continuous variables were summarized as mean ± SD or median (IQR) and compared with Student's *t* test or the Mann–Whitney *U*-test. The association between the TyG index and prevalent gout was examined using multivariable logistic regression; multicollinearity was assessed. The diagnostic performance of the TyG index was evaluated by receiver operating characteristic, and Spearman correlation assessed TyG vs uric acid (UA), white blood cell count (WBC), systemic immune-inflammation index (SII), and high-density lipoprotein cholesterol (HDL-C).

Results: The study included 447 gout patients and 1788 controls (average age 34.5 ± 6.9 years). Median TyG was higher in the gout group than in controls (4.86 [0.44] vs 4.63 [0.40]; $P < 0.001$). In multivariable logistic regression, higher TyG was independently associated with gout (adjusted OR = 2.454; 95% CI: 1.496–4.026, $P < 0.001$). TyG yielded an AUC of 0.709 (95% CI: 0.683–0.736), with an optimal cutoff of 4.735 (sensitivity 0.669; specificity 0.638). TyG correlated positively with UA ($r = 0.3393$), WBC ($r = 0.3250$), and SII ($r = 0.2069$) and inversely with HDL-C ($r = -0.4913$); all $P < 0.001$.

Conclusion: In this cross-sectional study of young Chinese adults, higher TyG was independently associated with the presence of gout and showed acceptable discrimination. As a routinely available metric, TyG may aid risk stratification and case-finding; prospective studies are needed to define actionable thresholds and clarify temporality.

Keywords: gout, young adults, triglyceride-glucose index, insulin resistance, epidemiology

Introduction

Gout is the most common form of inflammatory arthritis, characterized by acute pain, redness, swelling, and increased skin temperature. Recurrent attacks can lead to joint destruction and deformities[1] Recent studies indicate a continued rise in global gout prevalence, with approximately 55.8 million affected individuals worldwide and an age-standardized prevalence rate (ASPR) of 659.3 per 100,000.[2] In 2019, China had around 16.2 million gout patients, with prevalence rates above the global average and showing an upward trend. By 2029, ASPR in men and women is expected to reach 1170 and 400 per 100,000, respectively.[3] Gout prevalence among young adults in China is also concerning, with rates of 1000, 2300, and 3400 per 100,000 in the 18–29, 30–39, and 40–49 age groups, respectively, exceeding those of the same age groups in the United States.[3] Among young adults, the primary labor force—gout limits mobility and severely affects productivity,[4,5] leading to direct and indirect economic losses of $172 to $6179 per person,[6,7] imposing an increasing burden on patients and society. Identifying risk factors for gout in young populations and implementing early prevention strategies hold critical importance.

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The TyG index, calculated from triglyceride and glucose levels, was initially recognized as a novel indicator of insulin resistance (IR).[8] In recent years, research has increasingly linked the TyG index to multiple metabolic diseases like coronary heart disease, heart failure, ischemic stroke, and hypertension. Studies have identified the TyG index as an independent risk factor for heart failure and an independent predictor of coronary heart disease severity, adverse outcomes, and all-cause mortality.[9–12] Tao et al showed that higher TyG was linked to worse prognosis in hypertension, including increased stroke risk, especially ischemic stroke.[13] The data indicate that the TyG index holds promise as a biomarker for disease prognosis and evaluation.

The pathogenesis of gout is closely linked to metabolic syndrome. Biologically, the TyG index reflects triglyceride-glucose coupling and serves as a validated surrogate of insulin resistance,[14,15] a state that can elevate serum urate by reducing renal urate excretion and up-regulating urate transporters such as URAT1 (SLC22A12) and GLUT9 (SLC2A9).[16,17] TyG elevation also co-occurs with low HDL-C and systemic inflammation, both implicated in gout susceptibility and activity.[18,19] Prior studies have linked TyG to gout in general populations: Cao et al reported that lower TyG was associated with lower gout odds,[20] and Li et al suggested TyG may help flag early gout rather than establish prediction or causation.[21] However, most TyG-gout studies have focused on general or older populations, and it remains unclear whether those findings apply to young adults.[21,22] Compared with older individuals, young adults tend to develop insulin resistance earlier, have shorter exposure to metabolic abnormalities, and exhibit more clustered metabolic risks (eg, central adiposity and dyslipidemia with low HDL-C); these features may modify both the effect size and clinical utility of TyG in this age stratum.[23,24] Focusing on this group may therefore refine risk stratification and identify opportunities for earlier preventive strategies. Accordingly, we hypothesized that higher TyG would be independently associated with the presence of gout among young adults.

Methods

Study Population

This retrospective case-control study received approval from the Medical Ethics Committee of Meizhou People's Hospital (No.: MPH-2024-C-127) and was conducted in full compliance with the Declaration of Helsinki. Written informed consent was obtained from all participants. We consecutively collected data from all patients diagnosed with gout at Meizhou People's Hospital between 2019 and 2023, designating them as the case group. We obtained all variables from the Meizhou People's Hospital electronic medical record (EMR) and its linked laboratory information system for 2019–2023. Collected data included complete blood counts, assessments of liver and kidney function, fasting blood glucose, and lipid profile measurements. Inclusion criteria were: (1) diagnosis consistent with the 2015 ACR/EULAR gout classification criteria;[25] (2) age ranging from 18 to 44 years. Exclusion criteria included: (1) secondary gout diagnosis; (2) severe conditions such as liver, kidney, and heart diseases, or cancer; (3) missing data. Simultaneously, individuals undergoing routine health examinations at Meizhou People's Hospital were collected as the control cohort during the same time frame. Data included complete blood counts, liver and kidney function assessments, fasting blood glucose (FBG), and lipid profiles. Inclusion criteria required individuals aged 18–44 years, while exclusion criteria included (1) severe conditions such as liver, kidney, or heart diseases, or cancer, and (2) missing data. Cases and controls were frequency-matched 1:4 on age and sex using SPSS 26.0 (IBM Corp., Armonk, NY, USA).

Definitions

In this study, the TyG index is defined as the natural logarithm of the product of fasting triglycerides (TG) and FBG, divided by 2, represented as TyG = ln [fasting TG (mg/dL) * FBG (mg/dL)/2]. The Systemic immune-inflammation index (SII) is determined by the multiplying platelet and neutrophil counts and then dividing by the lymphocyte count, represented as SII = platelets ($*10^9$/L) * neutrophils ($*10^9$/L) / lymphocytes ($*10^9$/L).

Statistical Analysis

Statistical analysis was performed utilizing SPSS 26.0 software. The Shapiro–Wilk test was used to assess normality. For normally distributed variables such as age, data were presented as mean ± standard deviation and inter-group

comparisons were conducted utilizing the Student's *t*-test. Total cholesterol (TC), TyG index, uric acid (UA), TG, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), FBG, creatinine, alanine aminotransferase (ALT), glutamate aminotransferase (AST), white blood cell (WBC) count, lymphocyte count, neutrophil count, platelet count, and the SII did not follow a normal distribution. These variables were presented as medians (interquartile ranges) and compared between groups utilizing the Mann–Whitney *U*-test. Male sex, history of hypertension, and diabetes history were reported as frequencies (percentages) and compared between groups utilizing the chi-square test. For the multivariable logistic regression analysis, variables were categorized as follows: (1) known factors associated with gout outcomes, including age, hypertension, diabetes, and UA levels; and (2) variables showing $P < 0.05$ in the univariate regression, including HDL-C, creatinine, ALT, AST, WBC count, and the SII. Since the TyG index is derived from TG and FBG, and SII is calculated from neutrophil, lymphocyte and platelet counts, these individual components were excluded from the multivariate regression model to avoid collinearity. Because sex distribution was highly imbalanced by design (97% male in both groups), sex was handled through restriction/frequency matching and was not entered as a covariate in multivariable models. A multivariable logistic regression model (enter method: simultaneous forced entry of prespecified covariates in one block and retained regardless of univariable significance) was applied to assess the association between the TyG index and the presence of gout in young patients. We assessed multicollinearity among all covariates entered into the multivariable model using variance inflation factors (VIF) and tolerance; VIF < 5 and tolerance > 0.20 were considered acceptable The diagnostic performance of TyG index for gout was assessed using receiver operating characteristic (ROC) analysis. The diagnostic performance of TyG was quantified using the area under the ROC curve (AUC), and the optimal cutoff was determined by maximizing Youden's J. Spearman's rank correlation coefficient was utilized to investigate the relationships among the TyG index, UA, WBC, SII, and HDL-C. A *P*-value below 0.05 was regarded as indicative of statistical significance.

Results

Baseline Analysis of Gout and Healthy Control Groups

From 2,572 gout candidates, 102 with secondary gout and 463 with serious disease were excluded; an additional 1,560 were excluded for missing key variables (ALT, n=321; creatinine, n=389; hypertension/diabetes, n=289; SII, n=219; TyG, n=342), leaving 447 cases. Among 32,920 potential controls, 177 had serious disease and 8,634 had missing data (ALT, n=1,796; creatinine, n=2,125; hypertension/diabetes, n=1,569; SII, n=1,105; TyG, n=2,039), yielding 24,109 eligible controls, from which 1,788 were frequency-matched 1:4 to the cases by age and sex for analysis (Figure 1). Because TyG components were ordered selectively in routine care, TyG missingness may not have been completely at random; thus, potential selection bias is noted. The final analytic sample comprised 2,235 individuals (447 cases and 1,788 controls; mean age 34.5 ± 6.9 years). Males accounted for 97% (2175 individuals). The gout group consisted of 447 patients, averaging 34.6 ± 6.8 years in age, with 435 (97%) being male. The healthy control group included 1788 individuals, averaging 34.6 ± 6.8 years in age, with 1740 (97%) males.

At baseline, TG, TC, FBG, creatinine, ALT, AST, WBC count, platelet count, and the SII were notably elevated in the gout group versus the healthy control group. Conversely, individuals with gout exhibited significantly lower levels of HDL-C ($P < 0.05$). The median TyG index was 4.86 (0.44) in the gout cohort, compared to 4.63 (0.40) in the healthy control group ($P < 0.001$, Table 1).

Association and discriminative performance of the TyG index (logistic regression and ROC analyses) Univariate logistic regression analysis indicated a significant association between the TyG index and gout (OR = 8.841, 95% CI: 6.379–12.253, $P < 0.001$); HDL-C was inversely related to gout (OR = 0.061, 95% CI: 0.041–0.091, $P < 0.001$); and WBC count showed a positive association with gout (OR = 1.694, 95% CI: 1.594–1.799, $P < 0.001$). Additional factors - including hypertension, diabetes mellitus, UA, creatinine, ALT, AST, WBC count, and the SII - were significantly associated with gout (all $P < 0.001$, Table 2).

After adjustment for age, hypertension, diabetes mellitus, UA, HDL-C, creatinine, ALT, AST, WBC count, and the SII, multivariate logistic regression analysis identified the TyG index as an independent associated factor for gout among young adults (OR = 2.454, 95% CI: 1.496–4.026, $P < 0.001$). Other independent associated factors included hypertension

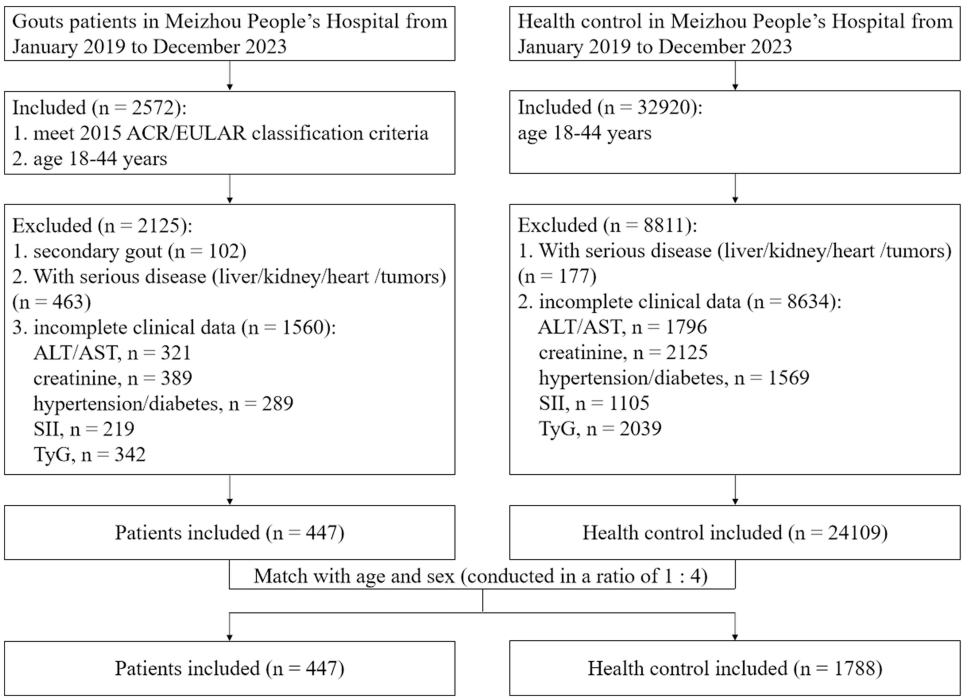

Figure 1 The flowchart of the study participants.

(OR = 1.886, 95% CI: 1.053–3.380, $P = 0.033$), HDL-C (OR = 0.247, 95% CI: 0.145–0.420, $P < 0.001$), WBC count (OR = 1.222, 95% CI: 1.129–1.324, $P < 0.001$), creatinine (OR = 1.020, 95% CI: 1.010–1.030, $P < 0.001$), ALT (OR = 1.012, 95% CI: 1.002–1.019, $P = 0.017$), UA (OR = 1.006, 95% CI: 1.004–1.007, $P < 0.001$), and the SII (OR = 1.003,

Table 1 Clinical Characteristics of Gout Individuals and Healthy Controls

Variables	Total (n = 2235)	Gout Individuals (n = 447)	Healthy Controls (n = 1788)	*P*-values
Age (years)	34.5 ± 6.9	34.6 ± 6.8	34.6 ± 6.8	1.000
Male, n (%)	2175 (97)	435 (97)	1740 (97)	1.000
Hypertension	103 (4.6)	44 (12)	59 (3.3)	< 0.001
Diabetes mellitus	38 (1.7)	19 (5.2)	19 (1.1)	< 0.001
TC (mmol/L)	4.87 (1.13)	5.15 (1.49)	4.82 (1.05)	< 0.001
TG (mmol/L)	1.46 (1.15)	2.05 (1.75)	1.38 (1.05)	< 0.001
LDL-C (mmol/L)	2.93 (0.89)	2.98 (1.03)	2.92 (0.87)	0.074
HDL-C (mmol/L)	1.45 (0.48)	1.21 (0.37)	1.48 (0.50)	< 0.001
FBG (mmol/L)	4.89 (0.83)	5.12 (0.98)	4.84 (0.82)	< 0.001
TyG index	4.67 (0.42)	4.86 (0.44)	4.63 (0.40)	< 0.001
UA (μmol/L)	419.20 (125.00)	522.25 (217.10)	407.40 (106.00)	< 0.001
Creatinine (μmol/L)	86.40 (16.00)	89.20 (25.60)	86.10 (15.00)	< 0.001
ALT (U/L)	26.00 (21.00)	36.00 (34.00)	24.00 (19.00)	< 0.001
AST (U/L)	22.00 (9.00)	23.00 (15.00)	22.00 (8.00)	< 0.001
WBC ($\times 10^9$/L)	6.80 (3.00)	8.90 (3.10)	6.60 (2.00)	< 0.001
Neutrophil count ($\times 10^9$/L)	3.78 (2.00)	5.47 (2.96)	3.58 (1.00)	< 0.001
Lymphocyte count ($\times 10^9$/L)	2.31 (1.00)	2.40 (1.05)	2.30 (1.00)	0.032
Platelet count ($\times 10^9$/L)	243.00 (75.00)	278.00 (105.00)	237.00 (71.00)	< 0.001
SII	395.04 (251.00)	625.86 (563.21)	380.86 (210.00)	< 0.001

Notes: Data are expressed as the mean ± standard deviation (SD), number (percentage) or median (interquartile range).
Abbreviations: TC, total cholesterol; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; FBG, fasting blood glucose; TyG index, triglyceride glucose index; UA, uric acid; ALT, alanine transaminase; AST, glutamate aminotransferase; WBC, white blood cell count; SII, systemic immune-inflammatory index.

Table 2 Logistic Regression Analysis Between TyG Index and Gout

Variables	Univariate		Multivariable*	
	OR (95% CI)	*P*	OR (95% CI)	*P*
Age	1.000 (0.985–1.015)	1.000	0.995 (0.973–1.018)	0.678
Hypertension	3.200 (2.134–4.798)	< 0.001	1.886 (1.053–3.380)	0.033
Diabetes mellitus	4.133 (2.169–7.875)	< 0.001	1.267 (0.441–3.641)	0.661
HDL-C	0.061 (0.041–0.091)	< 0.001	0.247 (0.145–0.420)	< 0.001
TyG	8.841 (6.379–12.253)	< 0.001	2.454 (1.496–4.026)	< 0.001
UA	1.009 (1.007–1.009)	< 0.001	1.006 (1.004–1.007)	< 0.001
Creatinine	1.031 (1.024–1.039)	< 0.001	1.020 (1.010–1.030)	< 0.001
ALT	1.027 (1.022–1.032)	< 0.001	1.011 (1.002–1.019)	0.017
AST	1.041 (1.030–1.052)	< 0.001	0.995 (0.975–1.015)	0.612
WBC	1.694 (1.594–1.799)	< 0.001	1.222 (1.129–1.324)	< 0.001
SII	1.004 (1.003–1.004)	< 0.001	1.003 (1.002–1.003)	< 0.001

Notes: *Collinearity checks: all variance inflation factors (VIFs) < 5 (maximum 2.565 for ALT) and all tolerances > 0.20 (minimum 0.390 for ALT); no problematic multicollinearity was detected.
Abbreviations: OR, odds ratio; CI, confidence interval; HDL-C, high-density lipoprotein cholesterol; TyG, triglyceride glucose index; UA, uric acid; ALT, alanine transaminase; AST, glutamate aminotransferase; WBC, white blood cell count; SII, systemic immune-inflammatory index.

95% CI: 1.002–1.003, $P < 0.001$, Table 2). In multivariable models, collinearity checks showed no problematic multicollinearity (all VIFs < 5, all tolerances > 0.20; Table S1).

We also evaluated the diagnostic performance of TyG in gout using receiver operating characteristic (ROC) analysis. TyG yielded an AUC of 0.709 (95% CI, 0.683–0.736), with an optimal cutoff of 4.735 (sensitivity 0.669; specificity 0.638; Figure 2).

Correlation Analysis Between TyG and Clinical Baseline Data

We performed a correlation analysis to investigate the associations among the TyG index, UA, WBC, SII, and HDL-C. The results revealed that the TyG index exhibited significant positive correlations with UA ($r = 0.3393$, $P < 0.001$), WBC

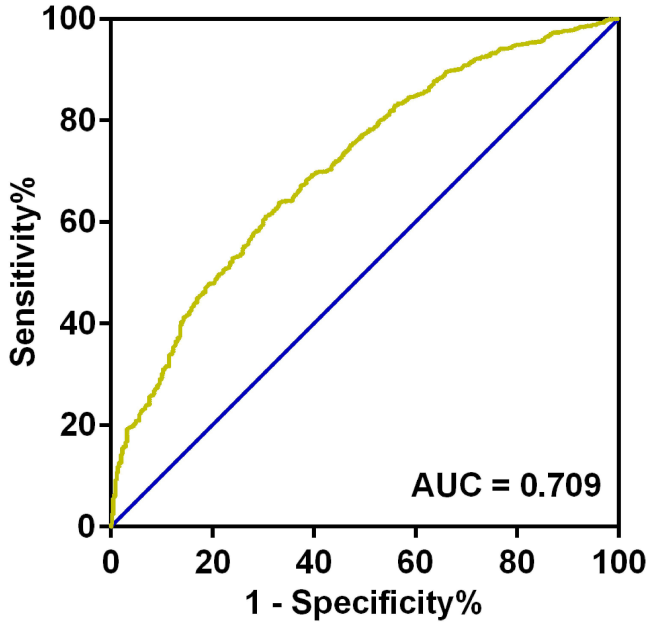

Figure 2 ROC curve of the TyG index for discriminating gout among young adults.

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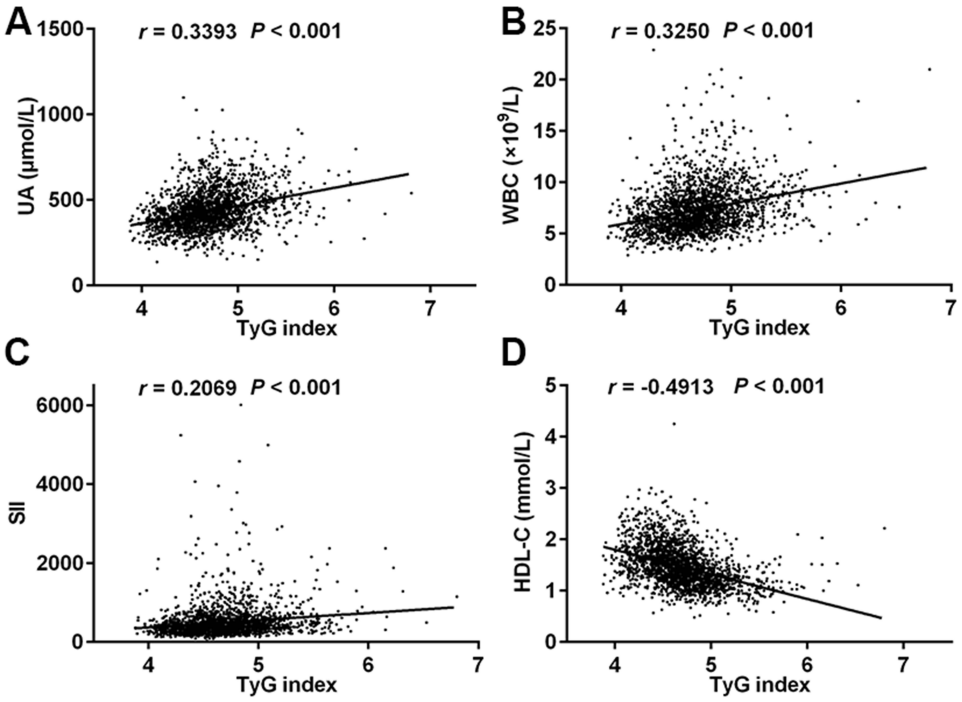

Figure 3 Correlations between the TyG index and clinical variables. (**A**) TyG vs UA; (**B**) TyG vs WBC; (**C**) TyG vs SII; (**D**) TyG vs HDL-C.

($r = 0.3250$, $P < 0.001$), and SII ($r = 0.2069$, $P < 0.001$; Figure 3A–C). Conversely, a significant negative correlation was observed between the TyG index and HDL-C ($r = -0.4913$, $P < 0.001$, Figure 3D).

Discussion

In this single-center case-control study, young patients with gout at Meizhou People's Hospital had higher TyG values than controls. In multivariable logistic regression, higher TyG was independently associated with the presence of gout. Beyond association, TyG showed diagnostic value with an AUC of 0.709. TyG correlated positively with UA, WBC, and SII and inversely with HDL-C, supporting its role as a readily available metabolic marker to aid risk stratification and case-finding in young adults.

A study based on the KNHANES database reported that among gout patients over the age of 19, males accounted for 90.7% (733,915/808,778), with 54.9% having comorbid hypertension (444,290/808,778) and 18.4% having diabetes (149,184/808,778).[26] Another study based on the NHANES database found that males comprised 69.4% (504/726) of gout patients, with 73.0% having hypertension (530/726) and 34.7% having diabetes (236/726).[16] In our study of 447 young gout patients, 12% had hypertension and 5.2% had diabetes. The mean UA level among young gout patients was 522.25 (217.10) μmol/L. Because the sex distribution was highly imbalanced by design (97% male in both groups), internal comparability was improved. However, this limits generalizability to women; future studies with more balanced sex distributions are warranted. Given that our study focused on young adults aged 18–45, the percentage of gout patients with hypertension and diabetes was relatively lower. Overall, our study population demonstrates a certain level of representativeness.

In our study, multivariable logistic regression showed that higher TyG was independently associated with the presence of gout (adjusted OR = 2.454). Similarly, NHANES-based analyses in US adults reported an association between higher TyG and prevalent gout, with adjusted ORs ranging from 1.43 to 1.64.[14–16] The larger effect size in our young Chinese cohort may reflect differences in age structure and ethnicity. Beyond association, TyG also demonstrated acceptable discriminative performance for case-finding. These results suggest that the TyG-gout association may be stronger in young adults than in the general population, potentially due to (i) population differences between China and the US; (ii) greater work-related stress and irregular schedules in younger individuals;[27,28] and (iii) unhealthy dietary

patterns, including higher consumption of sugar-sweetened beverages.[29,30] While the observed discrimination supports potential case-finding value, prospective work is needed to define actionable thresholds and assess incremental clinical utility.

The potential connection between the TyG index and gout likely centers around IR, as the TyG index serves as an important marker for this condition. IR is characterized by a diminished response to insulin, influenced by various factors, which leads to compensatory and secondary metabolic adaptations. This condition is frequently associated with metabolic disorders like obesity and metabolic syndrome, both of which are closely linked to gout.[31] Natalie McCormick et al found a causal relationship between IR, hyperuricemia (HUA), and gout.[32] Sun et al highlighted a bidirectional relationship between IR and HUA.[33] There are several theoretical mechanisms linking IR with gout. Yoo et al suggested that HUA observed in gout patients may be due to increased adiposity associated with IR.[34] Additionally, research has shown that IR enhances sodium reabsorption in renal tubules, thus reducing UA excretion.[35,36] Kottgenet et al proposed that IR elevates serum UA levels by promoting the generation of intermediates, such as pyrophosphate and ribose-5-phosphate, in glycolysis and the pentose phosphate pathway.[37] Conversely, elevated UA levels can worsen IR by reducing nitric oxide availability, intensifying mitochondrial oxidative stress, and initiating inflammation through multiple pathways.[35,36] Chang JB et al reported that overproduction of uric acid and reactive oxygen species is implicated in IR.[38] Further research is required to determine whether additional pathways, beyond those mediated by IR, link the TyG index with gout.

Interestingly, this study also suggests a potential association between HDL-C levels and the incidence of gout in young adults. A Mendelian randomization study by Yang et al revealed a significant causal link between elevated HDL-C and a reduced risk of gout.[39] Another Mendelian randomization study reported that each standard deviation increase in HDL-C was linked to an approximately 25% reduction in gout risk and a reduction in serum UA by 0.09 mg/dL.[40] Mendelian randomization analyses suggest a causal link consistent with a protective role of higher HDL-C. Mechanistically, HDL exerts anti-inflammatory and antioxidant actions relevant to gout pathophysiology: experimental work shows that HDL attenuates monosodium urate (MSU) crystal-induced inflammation by limiting leukocyte recruitment and chemokine (eg, CCL2) production in synovial cells and in vivo models; HDL can also dampen crystal-triggered cytokine responses.[41,42] In parallel, HDL particles possess inflammasome-modulating and antioxidant capacities (eg, via ApoA-I-dependent signaling and paraoxonase-1 activity), which may reduce oxidative stress and blunt downstream NLRP3 activation central to gout flares.[43] Taken together with our data, these observations are consistent with HDL-C acting as an associated protective marker in this cross-sectional design.

To further characterize the clinical milieu associated with TyG, we conducted a correlation analysis. The findings revealed a positive correlation between the TyG index and UA, which is closely associated with gout.[44] Furthermore, the TyG index demonstrated a positive correlation with WBC count and the SII. Yu et al reported higher WBC counts in acute gout compared with remission, while Yi et al observed higher SII in acute gouty arthritis than in healthy controls.[45,46] Conversely, the TyG index demonstrated a negative correlation with HDL-C levels. Prior research has indicated that the anti-inflammatory properties of HDL-C are diminished in individuals with gout.[47] Therefore, we hypothesized that the TyG index may have a potential association with gout and the precise underlying mechanisms connecting TyG to gout require further investigation.

The strength of our research: this study included 447 young patients diagnosed with gout over a five-year span, matched by sex and age, with a 1:4 ratio between case and control groups, resulting in 1788 healthy individuals as controls. The study design is robust, enhancing the reliability of the findings. This study represents one of the few investigations exploring the link between the TyG index and gout, specifically within a young Chinese population.

This study has several limitations. First, case ascertainment and control selection may introduce selection bias: cases were hospital patients, whereas controls were health-checkup attendees who may differ in health-seeking behavior and metabolic profiles. In addition, TyG availability depended on routine test ordering, making missingness unlikely to be completely at random; bias in either direction is possible. We therefore interpret the TyG-gout association with appropriate caution. Second, we did not capture lifestyle and anthropometric variables (eg, BMI/waist circumference, alcohol intake, consumption of purine- or fructose-rich foods, overall diet quality, physical activity). These factors are biologically linked to both TyG and serum urate and may introduce residual confounding. Third, this single-center study -

predominantly male (97%)-limits generalizability to women and to other settings or regions. Future work should include multicenter, community-based cohorts and prospective designs to validate thresholds, assess incremental clinical utility, and clarify temporality.

Conclusion

Our findings indicate that the TyG index is independently associated with the presence of gout among young adults, tracks a pro-inflammatory/low-HDL milieu, and exhibits moderate discriminative value for case-finding in this demographic. As a routinely available metric, TyG may support clinical risk stratification; further research is needed to establish actionable thresholds, assess incremental clinical utility, and clarify temporality and causal pathways in prospective studies, while interpreting the present results in light of missing TyG data and unmeasured lifestyle factors.

Abbreviations

ASPR, age-standardized prevalence rate; IR, insulin resistance; VIF, variance inflation factors; ROC, receiver operating characteristic; AUC, area under the ROC curve; TG, triglycerides; FBG, fasting blood glucose; SII, systemic immune-inflammation index; TC, total cholesterol; UA, uric acid; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; ALT, alanine aminotransferase; AST, glutamate aminotransferase; WBC, white blood cell.

Data Sharing Statement

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

The research presented in this work strictly adheres to the ethical standards outlined in the Declaration of Helsinki as endorsed by the World Medical Association. This study is granted by the Medical Ethics Committee of Meizhou People's Hospital (No.: MPH-2024-C-127). Written informed consent was obtained from all participants.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; 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 in this work.

References

1. Dalbeth N, Gosling AL, Gaffo A, Abhishek A. Gout. *Lancet*. 2021;397(10287):1843–1855. doi:10.1016/s0140-6736(21)00569-9

2. Cross M, Ong KL, Culbreth GT, et al. Global, regional, and national burden of gout, 1990-2020, and projections to 2050: a systematic analysis of the global burden of disease study 2021. *Lancet Rheumatol*. 2024;6(8):e507–e517. doi:10.1016/S2665-9913(24)00117-6
3. Song J, Jin C, Shan Z, Teng W, Li J. Prevalence and risk factors of hyperuricemia and gout: a cross-sectional survey from 31 provinces in mainland China. *J Transl Internal Med*. 2022;10(2):134–145. doi:10.2478/jtim-2022-0031
4. ten Klooster PM, Vonkeman HE, van de Laar MA. Disability due to gouty arthritis. *Curr Opin Rheumatol*. 2012;24(2):139–144. doi:10.1097/BOR.0b013e32834ff59d
5. Sigurdardottir V, Drivelegka P, Svard A, Jacobsson LTH, Dehlin M. Work disability in gout: a population-based case-control study. *Ann Rheum Dis*. 2018;77(3):399–404. doi:10.1136/annrheumdis-2017-212063
6. Safiri S, Kolahi AA, Cross M, et al. Prevalence, incidence, and years lived with disability due to gout and its attributable risk factors for 195 countries and territories 1990-2017: a systematic analysis of the global burden of disease study 2017. *Arthritis Rheumatol*. 2020;72(11):1916–1927. doi:10.1002/art.41404
7. Rai SK, Burns LC, De Vera MA, Haji A, Giustini D, Choi HK. The economic burden of gout: a systematic review. *Semin Arthritis Rheum*. 2015;45(1):75–80. doi:10.1016/j.semarthrit.2015.02.004
8. Simental-Mendia LE, Rodriguez-Moran M, Guerrero-Romero F. The product of fasting glucose and triglycerides as surrogate for identifying insulin resistance in apparently healthy subjects. *Metab Syndr Relat Disord*. 2008;6(4):299–304. doi:10.1089/met.2008.0034
9. Wang X, Xu W, Song Q, et al. Association between the triglyceride-glucose index and severity of coronary artery disease. *Cardiovasc Diabetol*. 2022;21(1):168. doi:10.1186/s12933-022-01606-5
10. Tao S, Yu L, Li J, et al. Association between the triglyceride-glucose index and 1-year major adverse cardiovascular events in patients with coronary heart disease and hypertension. *Cardiovasc Diabetol*. 2023;22(1):305. doi:10.1186/s12933-023-02018-9
11. Wang J, Huang X, Fu C, Sheng Q, Liu P. Association between triglyceride glucose index, coronary artery calcification and multivessel coronary disease in Chinese patients with acute coronary syndrome. *Cardiovasc Diabetol*. 2022;21(1):187. doi:10.1186/s12933-022-01615-4
12. Zhang R, Shi S, Chen W, et al. Independent effects of the triglyceride-glucose index on all-cause mortality in critically ill patients with coronary heart disease: analysis of the MIMIC-III database. *Cardiovasc Diabetol*. 2023;22(1):10. doi:10.1186/s12933-023-01737-3
13. Huang Z, Ding X, Yue Q, et al. Triglyceride-glucose index trajectory and stroke incidence in patients with hypertension: a prospective cohort study. *Cardiovasc Diabetol*. 2022;21(1):141. doi:10.1186/s12933-022-01577-7
14. Vasques ACJ, Novaes FS, de Oliveira Mda S, et al. TyG index performs better than HOMA in a Brazilian population: a hyperglycemic clamp validated study. *Diabet Res Clin Pract*. 2011;93(3):e98–e100. doi:10.1016/j.diabres.2011.05.030
15. Kurniawan LB. Triglyceride-glucose index as a biomarker of insulin resistance, diabetes mellitus, metabolic syndrome, and cardiovascular disease: a review. *EJIFCC*. 2024;35(1):44–51.
16. Toyoki D, Shibata S, Kuribayashi-Okuma E, et al. Insulin stimulates uric acid reabsorption via regulating urate transporter 1 and ATP-binding cassette subfamily G member 2. *Am J Physiol Renal Physiol*. 2017;313(3):F826–F834. doi:10.1152/ajprenal.00012.2017
17. Mandal AK, Leask MP, Estiverne C, Choi HK, Merriman TR, Mount DB. Genetic and physiological effects of insulin on human urate homeostasis. *Front Physiol*. 2021;12:713710. doi:10.3389/fphys.2021.713710
18. Adams-Huet B, Jialal I. An increasing triglyceride-glucose index is associated with a pro-inflammatory and pro-oxidant phenotype. *J Clin Med*. 2024;13(13):3941. doi:10.3390/jcm13133941
19. Han Y, Zhou Z, Zhang Y, Zhao G, Xu B. The association of surrogates of insulin resistance with hyperuricemia among middle-aged and older individuals: a population-based nationwide cohort study. *Nutrients*. 2023;15(14):3139. doi:10.3390/nu15143139
20. Cao S, Hu Y. Interpretable machine learning framework to predict gout associated with dietary fiber and triglyceride-glucose index. *Nutr Metab*. 2024;21(1):25. doi:10.1186/s12986-024-00802-2
21. Li T, Zhang H, Wu Q, Guo S, Hu W. Association between triglyceride glycemic index and gout in US adults. *J Health Popul Nutr*. 2024;43(1):115. doi:10.1186/s41043-024-00613-4
22. Dai Y, Zhang Y, Wang B, Cao L, Wang Z. The association between triglyceride glucose index and gout: a cross-sectional analysis based on NHANES 2007–2018. *BMC Endocr Disord*. 2024;24(1):218. doi:10.1186/s12902-024-01747-6
23. Eun Y, Han K, Lee SW, et al. Increased risk of incident gout in young men with metabolic syndrome: a nationwide population-based cohort study of 3.5 million men. *Front Med Lausanne*. 2022;9:1010391. doi:10.3389/fmed.2022.1010391
24. Amatucci AJ, Padnick-Silver L, LaMoreaux B, Bulbin DH. Comparison between early-onset and common gout: a systematic literature review. *Rheumatol Ther*. 2023;10(4):809–823. doi:10.1007/s40744-023-00565-x
25. Neogi T, Jansen TLTLTA, Dalbeth N, et al. Gout classification criteria: an American college of rheumatology/european league against rheumatism collaborative initiative. *Ann Rheum Dis*. 2015;74(10):1789–1798. doi:10.1136/annrheumdis-2015-208237
26. Jeong H, Chang Y-S, Jeon CH. Gout comorbidities: results from the Korean national health and nutrition examination survey. *Adv Rheumatol*. 2024;64(1):76. doi:10.1186/s42358-024-00413-8
27. Gwinnutt JM, Wieczorek M, Balanescu A, et al. EULAR recommendations regarding lifestyle behaviours and work participation to prevent progression of rheumatic and musculoskeletal diseases. *Ann Rheum Dis*. 2023;82(1):48–56. doi:10.1136/annrheumdis-2021-222020
28. FitzGerald JD, Dalbeth N, Mikuls T, et al. American college of rheumatology guideline for the management of gout. *Arthritis Care Res*. 2020;72(6):744–760. doi:10.1002/acr.24180
29. Li QH, Zou YW, Lian SY, et al. Sugar-sweeten beverage consumption is associated with more obesity and higher serum uric acid in Chinese male gout patients with early onset. *Front Nutr*. 2022;9:916811. doi:10.3389/fnut.2022.916811
30. Towiwat P, Li ZG. The association of vitamin C, alcohol, coffee, tea, milk and yogurt with uric acid and gout. *Int J Rheum Dis*. 2015;18(5):495–501. doi:10.1111/1756-185X.12622
31. Thottam GE, Krasnokutsky S, Pillinger MH. Gout and metabolic syndrome: a tangled web. *Curr Rheumatol Rep*. 2017;19(10):60. doi:10.1007/s11926-017-0688-y
32. McCormick N, O'Connor MJ, Yokose C, et al. Assessing the causal relationships between insulin resistance and hyperuricemia and gout using bidirectional mendelian randomization. *Arthritis Rheumatol*. 2021;73(11):2096–2104. doi:10.1002/art.41779
33. Sun J, Sun M, Su Y, et al. Mediation effect of obesity on the association between triglyceride-glucose index and hyperuricemia in Chinese hypertension adults. *J Clin Hypertens*. 2022;24(1):45–57. doi:10.1111/jch.14405

34. Yoo HG, Lee SI, Chae HJ, Park SJ, Lee YC, Yoo WH. Prevalence of insulin resistance and metabolic syndrome in patients with gouty arthritis. *Rheumatol Int*. 2011;31(4):485–491. doi:10.1007/s00296-009-1304-x
35. Muscelli E, Natali A, Bianchi S, et al. Effect of insulin on renal sodium and uric acid handling in essential hypertension. *Am J Hypertens*. 1996;9(8):746–752. doi:10.1016/0895-7061(96)00098-2
36. Ter Maaten JC, Voorburg A, Heine RJ, Ter Wee PM, Donker AJ, Gans RO. Renal handling of urate and sodium during acute physiological hyperinsulinaemia in healthy subjects. *Clin Sci*. 1997;92(1):51–58. doi:10.1042/cs0920051
37. Kottgen A, Albrecht E, Teumer A, et al. Genome-wide association analyses identify 18 new loci associated with serum urate concentrations. *Nat Genet*. 2013;45(2):145–154. doi:10.1038/ng.2500
38. Chang JB, Chen YL, Hung YJ, et al. The role of uric acid for predicting future metabolic syndrome and type 2 diabetes in older people. *J Nutr Health Aging*. 2017;21(3):329–335. doi:10.1007/s12603-016-0749-3
39. Yang Y, Xian W, Wu D, et al. The role of obesity, type 2 diabetes, and metabolic factors in gout: a Mendelian randomization study. *Front Endocrinol*. 2022;13:917056. doi:10.3389/fendo.2022.917056
40. Yu X, Wang T, Huang S, Zeng P. Evaluation of the causal effects of blood lipid levels on gout with summary level GWAS data: two-sample Mendelian randomization and mediation analysis. *J Hum Genet*. 2021;66(5):465–473. doi:10.1038/s10038-020-00863-0
41. Scanu A, Oliviero F, Gruaz L, et al. High-density lipoproteins downregulate CCL2 production in human fibroblast-like synoviocytes stimulated by urate crystals. *Arthritis Res Ther*. 2010;12(1):R23. doi:10.1186/ar2930
42. Scanu A, Luisetto R, Oliviero F, et al. High-density lipoproteins inhibit urate crystal-induced inflammation in mice. *Ann Rheum Dis*. 2015;74(3):587–594. doi:10.1136/annrheumdis-2013-203803
43. Thacker SG, Zarzour A, Chen Y, et al. High-density lipoprotein reduces inflammation from cholesterol crystals by inhibiting inflammasome activation. *Immunology*. 2016;149(3):306–319. doi:10.1111/imm.12638
44. Zhang WZ. Uric acid en route to gout. *Adv Clin Chem*. 2023;116:209–275. doi:10.1016/bs.acc.2023.05.003
45. Yu H, Xue W, Yu H, Gu H, Qin L, Peng A. Joint application of multiple inflammatory cytokines in diagnosis of gout flare. *J Inflamm Res*. 2023;16:1771–1782. doi:10.2147/JIR.S408929
46. Jiang Y, Tu X, Liao X, et al. New inflammatory marker associated with disease activity in gouty arthritis: the systemic inflammatory response index. *J Inflamm Res*. 2023;16:5565–5573. doi:10.2147/JIR.S432898
47. Wang Y, Wang Y, Jia X, et al. The anti-inflammatory properties of HDLs are impaired in gout. *Clin Rheumatol*. 2021;40(4):1525–1531. doi:10.1007/s10067-020-05374-z