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ORIGINAL RESEARCH

Triglyceride-Glucose Index Levels Positively Associated with Higher Risk of Low Muscle Mass in Patients with Type 2 Diabetes

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Aim: Although the interplay of insulin resistance (IR) and low muscle mass is increasingly recognized, it remains unknown whether the triglyceride-glucose (TyG) index, as an indicator of IR, is associated with low muscle mass in patients with type 2 diabetes (T2D). Our study aimed to investigate the association between TyG index and low muscle mass in hospitalized T2D patients.

Methods: This cross-sectional study involved 2687 hospitalized participants with T2D. The TyG index was calculated by fasting plasma glucose (FPG) and triglyceride levels. The outcome variables were defined as appendicular skeletal muscle mass index (ASMI) and low muscle mass. To explore the relationship between TyG index and low muscle mass, we conducted the multivariate linear regression, multivariate logistic regression, and subgroup analysis.

Results: In the fully adjusted multivariate linear regression, there was a negative correlation between TyG index (β=−0.10, 95% CI: −0.14, −0.06) and ASMI. TyG index (OR = 1.34, 95% CI: 1.08, 1.65) had a more significant association with low muscle mass compared to FPG (OR = 1.05, 95% CI: 1.01, 1.09) and glycated hemoglobin A1c (OR = 1.07, 95% CI: 0.99, 1.15). The statistical significance of the trend persisted among the TyG index quartile groups. Subgroup analysis revealed stronger positive associations between TyG index and low muscle mass in females, individuals aged 60 years or older, those with a body mass index of 28kg/m^2 or higher, and HbA1c levels of 6.5% or higher, as well as those with hypertension and dyslipidemia.

Conclusion: A higher TyG index level is positively associated with a higher risk of low muscle mass, suggesting that TyG index could be a potential biomarker of low muscle mass in hospitalized T2D patients.

Keywords: triglyceride-glucose index, low muscle mass, type 2 diabetes, insulin resistance, cross-sectional study

Introduction

Sarcopenia is a syndrome characterized by a progressive loss of muscle mass and function and determined by dual-energy X-ray absorptiometry (DEXA) or bioresistive spectroscopy.[1,2] It has been associated with an increased risk of body dysfunction, disability, falls, and mortality.[3] Although sarcopenia is commonly reported in the elderly, it can also affect patients with diabetes,[4] cancer,[1] metabolic disorders,[5] kidney dysfunction,[6] and other conditions. There is a growing perception that sarcopenia is associated with various complications and adverse clinical events in patients with diabetes.[7] Additionally, the interplay of insulin resistance (IR) and low muscle mass is increasingly recognized. Therefore, early identification and treatment of low muscle mass can help reduce complications and improve the quality of life for patients with diabetes.

IR, as an abnormal metabolic status, is characterized by impaired responsiveness of peripheral tissue to insulin and is related to increased complications and mortality.[8,9] Recently, researchers have found that the triglyceride-glucose (TyG) index, calculated by fasting plasma glucose (FPG) and triglyceride (TG), is strongly correlated with the results of the hyperinsulinemic-euglycemic (HIEC) clamp test.[10] The TyG index has been recognized as a more convenient method than HIEC and a more reliable surrogate than the homoeostasis model assessment of insulin resistance (HOMA-IR).[11]

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A cross-sectional study demonstrated that the prevalence of low muscle mass in the general population increased linearly with the quartiles of the TyG index, 2.5%, 4.2%, 5.6%, and 6.7% in Q1-Q4, respectively.[12] However, the relationship between TyG index and low muscle mass in type 2 diabetes (T2D) has not been established. Therefore, we aimed to investigate the association between TyG index levels and low muscle mass, as well as the potential of the TyG index as a biomarker for detecting the presence of low muscle mass in individuals with T2D.

Materials and Methods

Study Population

This cross-sectional study was conducted on hospitalized T2D patients with complete body composition data at the Department of Endocrinology and Metabolism of Tianjin Medical University General Hospital, from June 2013 to May 2018. The study was approved by the ethics committee of Tianjin Medical University General Hospital [Batch number: IRB2020-YX-027-01]. This study also followed the Declaration of Helsinki. Participants with the following conditions were included: (1) had complete body composition data; (2) met the World Health Organization (WHO) criteria for the diagnosis of T2D in 1999.[13] The exclusion criteria were as follows: (1) the repeated patients; (2) lack of clinical data on FPG, TG, and glycosylated hemoglobin (HbA1c). Our study included 3763 participants with complete body composition data. We excluded 851 individuals due to the deduplication for repeated hospitalization. Exclusions were made for participants with missing data on FPG, TG, and HbA1c. Finally, we analyzed a total of 2687 individuals with T2D. All participants were divided into four groups based on their TyG index quartile: group Q1 <2.90, group Q2 [2.90, 3.40), group Q3 [3.40, 3.92), and group Q4 ≥3.92 (see Figure 1).

Exposure Variables and Outcome Variables

The TyG index was used as the exposure variable, calculated as ln{[FPG (mmol/l)/18]×[TG (mmol/l)/0.0113]/2}[14] Besides, considering that FPG and HbA1c are common indicators to evaluate diabetes status, we further treated FPG and HbA1c as the exposure variable. The outcome variables included appendicular skeletal muscle mass index (ASMI) and the prevalence of low muscle mass. Body composition was measured by trained health technicians using a DEXA scanner (Hologic, Inc., Bedford, Massachusetts; software version Apex 3.2). The ASMI was calculated as appendicular skeletal muscle mass divided by height squared (kg/m^2).[15] Low muscle mass was defined as ASMI of <7.0 and <5.4 kg/m^2 for males and females, respectively.[15]

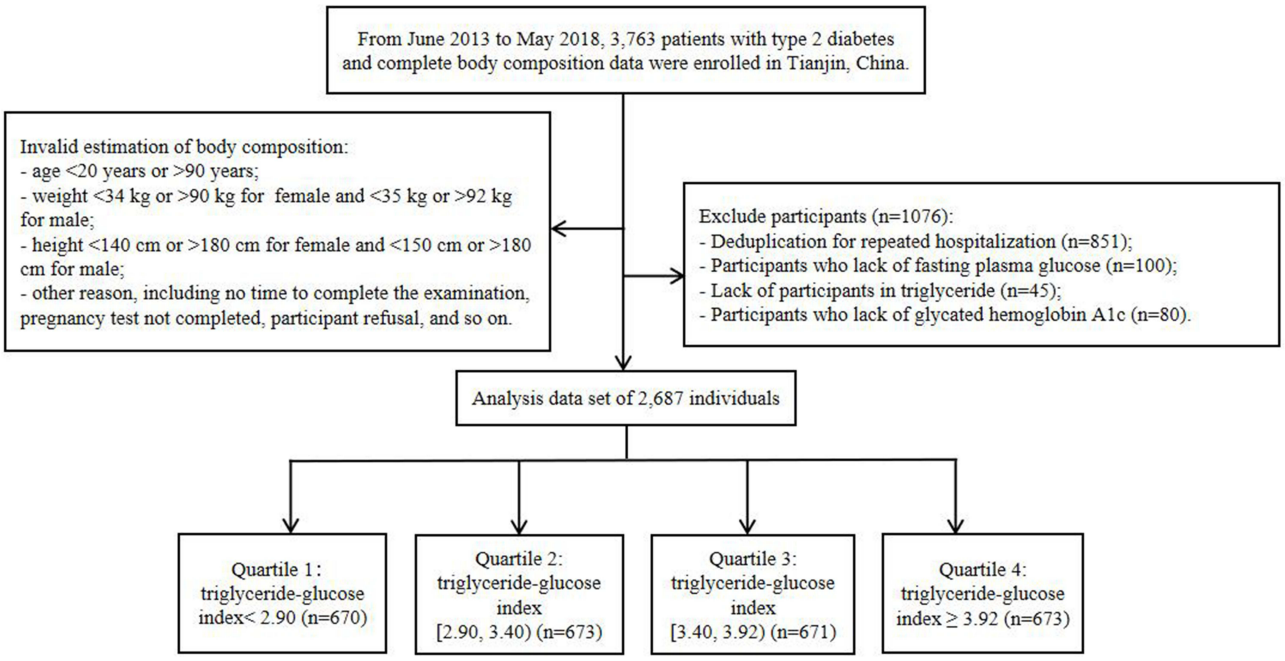

Figure 1 Flowchart of this study population.

Covariates

Demographic data comprised age, sex, diabetic duration, smoking status, and drinking status. Anthropometric measurements, including systolic blood pressure (SBP), diastolic blood pressure (DBP), and body mass index (BMI), were obtained from all participants. BMI was calculated as weight in kilograms divided by the square of height in meters. The laboratory indicators included FPG (mmol/L), HbA1c (%), total cholesterol (TC, mmol/L), triglyceride (TG, mmol/L), low-density lipoprotein cholesterol (LDL, mmol/L), high-density lipoprotein cholesterol (HDL, mmol/L), serum creatinine (SCr, umol/L), and the estimated glomerular filtration rate (eGFR, mL/min/1.73 m^2). Our study considered demographic data, anthropometric measurements (SBP and BMI), medication (use of oral hypoglycemic drugs, insulin, lipid-lowering agents, and anti-hypertensive drugs), and laboratory indicators (SCr and TC) that affect TyG index, FPG, HbA1c and muscular mass in clinical practice as covariates. FPG, HbA1c, TG, TC, LDL, and HDL were measured using an automatic analyzer (Model 7080; Hitachi, Tokyo, Japan). The estimated glomerular filtration rate (eGFR) was calculated using the MDRD study equation (for males, eGFR = 186 × serum creatinine$^{-1.154}$×year$^{-0.203}$; females, eGFR = 186×serum creatinine$^{-1.154}$×year$^{-0.203}$×0.724).[16] Hypertension is defined as SBP ≥140mmHg and/or DBP ≥90mmHg or self-reported hypertension with the use of anti-hypertensive drugs. Dyslipidemia[17] can be diagnosed when the TC level is 6.2mmol/L or higher, or the LDL level is 4.1mmol/L or higher, or the TG level is 2.3mmol/L or higher, or the HDL level is less than 1.0mmol/L, or self-reported dyslipidemia with the use of lipid-lowering drugs. This study included medication of oral hypoglycemic drugs, insulin, lipid-lowering and anti-hypertensive drugs.

Statistical Analysis

Continuous variables were presented as either mean with standard deviation (SD) or median with interquartile range. Differences in normally distributed data were identified using one-way ANOVA tests, while differences in non-normally distributed data were determined using Wilcoxon rank-sum tests. Categorical variables were reported as the number of patients (%). Chi-square tests were used to analyze categorical variables.

To evaluate the relationship between FPG, HbA1c, TyG index and muscular mass, we conducted multiple regression analyses in our study. We used three models: crude model, which was an unadjusted regression; model 1, which was adjusted for age, sex, and diabetic duration; and model 2, which was further adjusted for smoking status, drinking status, SBP, SCr, TC, BMI, use of oral hypoglycemic drugs, insulin, lipid-lowering agents, and anti-hypertensive drugs. Furthermore, the relationships between FPG, HbA1c, TyG index and ASMI were analyzed using multivariate linear regression models and the calculated β (95% CIs). Additionally, the associations of FPG, HbA1c, and TyG index with low muscle mass were evaluated using multivariate logistic regression models and calculated odds ratios (ORs (95% CIs)). To investigate whether the association between the TyG index and low muscle mass persists under different conditions, we conducted multivariate logistic regression models for subgroup analyses based on sex, age (≥60 years), BMI (≥28kg/m^2), HbA1c (≥6.5%), hypertension, and dyslipidemia. We considered the aforementioned associations reliable when the interaction P-value was not significant. Otherwise, there may be a special population. All statistical analyses in our study were conducted using R version 4.3.1 (http://www.r-project.org/). P value <0.05 was considered statistically significant.

Results

Table 1 shows the baseline characteristics of the population based on the quartile of the TyG index. Participants with higher TyG index levels were found to be younger and have a higher prevalence of current smoking and drinking, hypertension and dyslipidemia. They were also more likely to use oral hypoglycemic, insulin, and lipid-lowering drug compared to those with lower TyG index values. Subjects with higher TyG index values exhibited elevated levels of BMI, SBP, DBP, ASMI, FPG, HbA1c, TC, TG, LDL, and eGFR, while HDL levels significantly decreased (all $P < 0.05$). In addition, there were no differences in sex and SCr among the different groups.

Figure 2 shows the association between FPG, HbA1c, TyG index and ASMI. After fully adjusting for confounding factors in the multivariate linear regression models, significant negative associations were found between FPG (β=−0.01, 95% CI: −0.02, −0.01) and TyG index (β=−0.10, 95% CI: −0.14, −0.06) with ASMI. However, the relationship between HbA1c and ASMI was not statistically significant (β=−0.01, 95% CI: −0.02, 0). Meanwhile, Figure 3 shows that after adjusting for confounding variables (model 2), a per-SD increase in FPG was associated with a 5% increase in the odds

Table 1 Baseline Characteristics of Participants in This Study

Characteristics	Triglyceride-Glucose Index				P value
	Q1 <2.90 (n=670)	Q2 [2.90, 3.40) (n=673)	Q3 [3.40, 3.92) (n=671)	Q4 ≥3.92 (n=673)	
Age, years	60.1±12.5	60.3±12.0	57.0±11.8	53.7±13.4	<0.001
Male, n (%)	321 (47.9)	315 (46.8)	326 (48.6)	354 (52.6)	0.161
Diabetic duration, years	10.0 (3.0, 16.0)	9.0 (2.0, 15.0)	8.0 (2.0, 14.0)	7.0 (2.0, 14.0)	<0.001
Smoking status, n (%)	171 (25.5)	212 (31.5)	231 (34.4)	267 (39.7)	<0.001
Drinking status, n (%)	116 (17.3)	141 (21.0)	164 (24.4)	184 (27.3)	<0.001
BMI, kg/m^2	24.4 (22.1, 26.9)	26.0 (23.7, 28.6)	26.9 (24.5, 29.6)	27.1 (24.9, 29.7)	<0.001
SBP, mmHg	133±19	134±17	136±19	137±19	<0.001
DBP, mmHg	78±10	81±11	82±11	84±11	<0.001
ASMI	6.84 (6.11, 7.68)	7.05 (6.30,7.88)	7.41 (6.61, 8.26)	7.50 (6.69, 8.28)	<0.001
FPG, mmol/L	5.7 (4.7, 6.9)	7.1 (6.1, 8.3)	8.6 (7.1, 10.5)	10.8 (8.5, 13.4)	
HbA1c, %	7.3 (6.4, 8.8)	7.7 (6.7, 9.2)	8.3 (7.2, 9.8)	9.3 (7.9, 10.6)	<0.001
TC, mmol/L	4.29 (3.63, 5.04)	4.64 (4.05, 5.38)	4.95 (4.20, 5.75)	5.24 (4.56, 6.31)	<0.001
TG, mmol/L	0.87 (0.70, 1.07)	1.33 (1.10, 1.60)	1.80 (1.43, 2.23)	3.17 (2.33, 4.58)	
HDL, mmol/L	1.27 (1.03, 1.55)	1.09 (0.94, 1.31)	1.04 (0.87, 1.25)	0.93 (0.79, 1.13)	<0.001
LDL, mmol/L	2.55 (2.00, 3.15)	2.88 (2.33, 3.56)	3.02 (2.36, 3.74)	3.07 (2.36, 3.75)	<0.001
SCr, umol/L	66.37±23.95	68.60±30.65	67.76±23.92	67.92±25.95	0.475
eGFR, (mL/min/1.73 m^2)	95.72 (86.36, 104.96)	94.92 (83.84, 104.40)	97.89 (87.25, 106.15)	100.61 (88.00, 100.61)	<0.001
Hypertension	384 (57.3)	416 (61.8)	433 (64.5)	435 (64.6)	0.018
Hyperlipidemia	161 (24.0)	294 (43.7)	482 (71.8)	638 (94.8)	<0.001
Low muscle mass	114 (17.0)	84 (12.5)	52 (7.7)	52 (7.7)	<0.001
Oral hypoglycemic drugs	568 (84.8)	598 (88.9)	612 (91.2)	617 (91.7)	<0.001
Insulin	419 (62.5)	375 (55.7)	404 (60.2)	480 (71.3)	<0.001
Lipid-lowering drugs	221 (33.0)	268 (39.8)	310 (46.2)	413 (61.4)	<0.001
Anti-hypertensive drugs	316 (47.2)	334 (49.6)	380 (56.6)	338 (50.2)	0.004

Notes: Continuous variables are presented as mean ± SD if normally distributed and median (interquartile range) if not normally distributed. Categorical variables are presented as number of patients (%).

Abbreviations: BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; ASMI, appendicular skeletal muscle mass index; FPG, fasting plasma glucose; HbA1c, glycosylated hemoglobin; TC, total cholesterol; TG, triglyceride; HDL, high density lipoprotein; LDL, low density lipoprotein; SCr, serum creatinine; eGFR, estimated glomerular filtration rate.

of low muscle mass (OR = 1.05, 95% CI: 1.01, 1.09), and a per-SD increase in TyG index was associated with a 34% increase in the odds of low muscle mass (OR = 1.34, 95% CI: 1.08, 1.65). Nevertheless, there was no statistically significant association between HbA1c and low muscle mass (OR = 1.07, 95% CI: 0.99, 1.15).

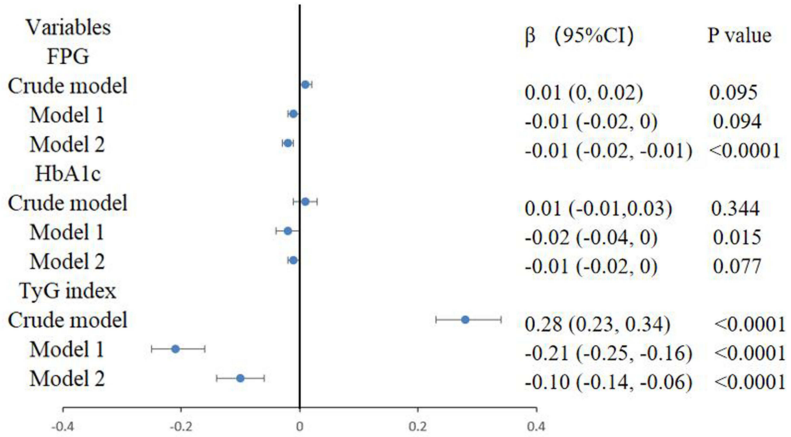

Figure 2 Multivariate linear analysis examined the association between FPG, HbA1c, TyG index, and ASMI. Crude model was an unadjusted regression. Model 1 was adjusted for age, sex, and diabetic duration. Model 2 was further adjusted for smoking status, drinking status, systolic blood pressure, serum creatinine, total cholesterol, body mass index, use of oral hypoglycemic drugs, insulin, lipid-lowering agents, and anti-hypertensive drugs.

Abbreviations: ASMI, appendicular skeletal muscle mass index; FPG, fasting plasma glucose; HbA1c, glycated hemoglobin A1c; TyG, triglyceride-glucose.

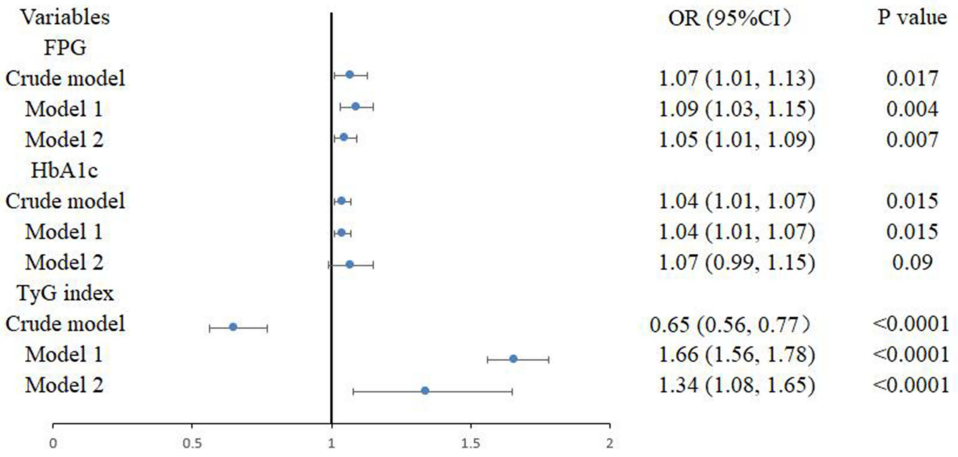

Figure 3 Multivariate logistic analysis examined the association between FPG, HbA1c, TyG index, and low muscle mass. Crude model was an unadjusted regression. Model 1 was adjusted for age, sex, and diabetic duration. Model 2 was further adjusted for smoking status, drinking status, systolic blood pressure, serum creatinine, total cholesterol, body mass index, use of oral hypoglycemic drugs, insulin, lipid-lowering agents, and anti-hypertensive drugs.
Abbreviations: FPG, fasting plasma glucose; HbA1c, glycated hemoglobin A1c; TyG, triglyceride-glucose.

To explore the independent effect of the TyG index on low muscle mass, we conducted the multivariate logistic regression analysis (Table 2). The OR of Q4 was 1.09 (95% CI: 1.06, 1.12), 1.08 (95% CI: 1.06, 1.10), and 1.05 (95% CI: 1.01, 1.09), respectively, in the crude model (no adjustment), model 1 (adjusted for age, sex, and diabetic duration), and model 2 (adjusted for age, sex, diabetic duration, smoking status, drinking status, SBP, SCr, TC, BMI, use of oral hypoglycemic drugs, insulin, lipid-lowering agents, and anti-hypertensive drugs). There was no significant linear trend among TyG index quartile groups (model 2: P for trend >0.05). To assess the robustness of the aforementioned findings across different groups, we conducted a subgroup analysis (Figure 4). We observed stronger positive associations between TyG index and low muscle mass was observed in females (OR = 1.47, 95% CI: 1.03, 2.10), individuals aged ≥60 years (OR = 1.35, 95% CI: 1.01, 1.84), those with BMI ≥28kg/m^2 (OR = 1.39, 95% CI: 1.12, 1.73), HbA1c ≥6.5% (OR = 1.40, 95% CI: 1.11, 1.77), hypertensive individuals (OR = 1.71, 95% CI: 1.23, 2.38), and hyperlipidemic (OR = 1.86, 95% CI: 1.26, 2.76) individuals. Additionally, BMI and hypertension were considered as the most prominent factors affecting the correlation between TyG index and low muscle mass (P for interaction < 0.05).

Discussion

To the best of our knowledge, our study is the first to investigate the relationship between TyG index and low muscle mass in hospitalized patients with T2D. The results demonstrated a negative association between the TyG index and the increase of ASMI, as well as a positive correlation with the elevated risk of low muscle mass. Moreover, TyG index

Table 2 Multivariate Logistic Analysis of the Association Between the TyG Index and Low Muscle Mass

TyG index (quartile)	Crude Model		Model 1		Model 2	
	OR (95% CI)	P value	OR (95% CI)	P value	OR (95% CI)	P value
Q1 <2.90	Reference		Reference		Reference	
Q2 [2.90, 3.40)	1.05 (1.01, 1.08)	0.008	1.04 (1.01, 1.07)	0.009	1.0 (0.99, 1.01)	0.578
Q3 [3.40, 3.92)	1.10 (1.06, 1.13)	<0.0001	1.09 (1.06, 1.12)	<0.0001	1.05 (1,01, 1.08)	0.011
Q4 ≥3.92	1.09 (1.06, 1.12)	<0.0001	1.08 (1.06, 1.10)	<0.0001	1.05 (1.01, 1.09)	0.013
P for trend	<0.0001		<0.0001		0.250	

Notes: Crude model was an unadjusted regression. Model 1 was adjusted for age, sex, and diabetic duration. Model 2 was further adjusted for smoking status, drinking status, systolic blood pressure, serum creatinine, total cholesterol, body mass index, use of oral hypoglycemic drugs, insulin, lipid-lowering agents, and anti-hypertensive drugs;
Abbreviation: TyG, triglyceride-glucose.

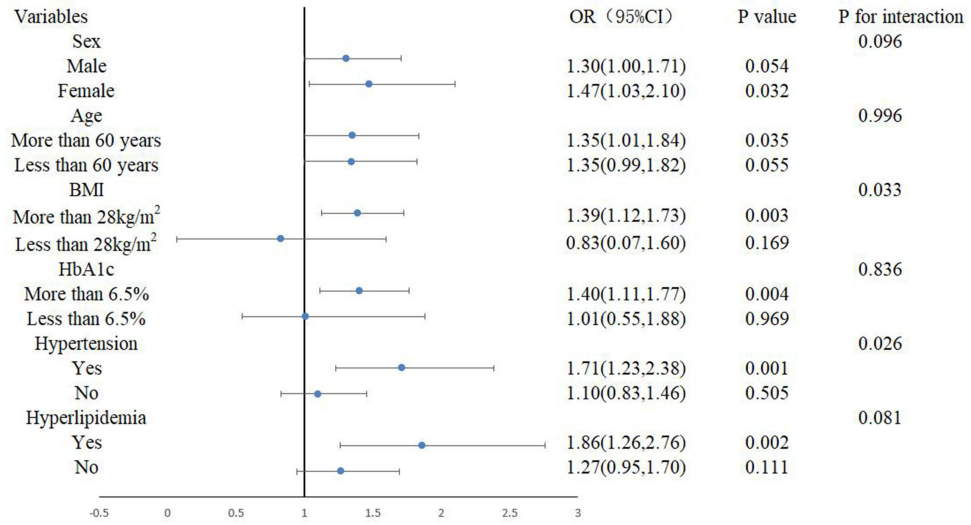

Figure 4 Forest plots of stratified analyses of the triglyceride-glucose index and low muscle mass. Adjusted for age, sex, diabetic duration, smoking status, drinking status, systolic blood pressure, serum creatinine, total cholesterol, body mass index, use of oral hypoglycemic drugs, insulin, lipid-lowering agents, and anti-hypertensive drugs, other than the variable for stratification.
Abbreviations: BMI, body mass index; HbA1c, glycated hemoglobin A1c.

showed a stronger correlation with low muscle mass compared to FPG and HbA1c. The subgroup analysis indicated that a stronger association between TyG index and low muscle mass in females, age ≥60 years, BMI ≥28kg/m^2, HbA1c ≥6.5%, hypertensive, and hyperlipidemic individuals. Besides, the interaction test showed that sex and hypertension may be the prominent factors influencing the association between TyG index and low muscle mass. The above results suggested that TyG index could potentially serve as a biomarker for low muscle mass in participants with T2D.

Recently, the interplay of IR and low muscle mass is increasingly recognized. IR is the main pathogenesis of T2D, which leads to dysglycemia and hyperinsulinemia. These conditions can cause increased protein degradation and decreased synthesis, resulting in low muscle mass.[18] In addition, low muscle mass can also lead to reduced muscle glucose uptake, hyperglycemia, hyperinsulinemia, and IR.[19] Kim TN, et al[20] found that individuals with T2D had an increased risk of sarcopenia after adjusting for confounders in the Korean population. Lee PG, et al[21] also found that sarcopenia has been the cause of IR in older adults with T2D in the US. Muscle mass, as the main insulin-sensitive tissue, accounts for 40% of the body weight, and its loss can be associated with IR.[22,23] Furthermore, patients with T2D often have low muscle mass, which may further increase the risk of metabolic and functional decline.[24,25] Therefore, early identification of the presence of low muscle mass is critical for the population with T2D.

TyG index, as a validated marker for assessing IR, has been reported to be strongly associated with HIEC.[10] It has been recognized as a more convenient method than HIEC and a more reliable surrogate than HOMA-IR.[11] A previous study found that after adjusting for confounders in the general population, the OR for low muscle mass was 1.13 (95% CI 1.07, 1.20) for per-SD increase in TyG index. The prevalence of low muscle mass increased linearly with the quartiles of the TyG index, 2.5%, 4.2%, 5.6%, and 6.7% in Q1-Q4, respectively.[12] In a study by Zhenzhen Liet al, which included 36,274 subjects who underwent health checks, the TyG index was found to be significantly correlated with low muscle mass (OR 1.87, 95% CI 1.75, 2.00).[26] Consistent with previous studies, our study demonstrated that, after adjusting for age, sex, diabetic duration, smoking status, drinking status, BMI, SBP, SCr, TC, use of oral hypoglycemic drugs, insulin, lipid-lowering drugs and antihypertensive drugs, the TyG index was negatively associated with ASMI, and positively correlated to the elevated risk of low muscle mass among the hospitalized participants with T2D. This trend remained statistically significant when TyG index was considered a categorical variable in quartiles. Moreover, we compared TyG index to the commonly used clinical metabolic markers, FPG and HbA1c, and found that TyG index was more effective in identifying the risk of low muscle mass. These results indicated that the TyG index levels positively correlate with low muscle mass, suggesting that TyG index could be a potential biomarker of low muscle mass in patients with T2D. To enhance the effectiveness of prevention strategies, subgroup analysis was conducted to identify high-risk individuals among patients with T2D. Our study found that in

hospitalized T2D patients, significant associations between TyG index and low muscle mass were present in females, age ≥60 years, BMI ≥28kg/m^2, HbA1c ≥6.5%, hypertensive, and hyperlipidemic patients. In this case, health providers may consider more assertive management of these high-risk populations.

Previous studies have explored the relationship between IR and low muscle mass, which may partially explain the mechanism behind the positive correlation between TyG index and low muscle mass. Firstly, insulin plays a crucial role in promoting muscle protein synthesis and inhibiting protein breakdown. IR can cause an imbalance between protein synthesis and degradation in muscle mass, resulting in low muscle mass among patients with T2D.[27] Secondly, IR can cause a decrease in the oxidative metabolism of type I slow muscle fibers and an increase in glycolysis metabolism of type II fast muscle fibers. This change may promote the occurrence of low muscle mass.[28] Lastly, IR leads to an increase in pro-inflammatory factors including interleukin-6, tumor necrosis factor-α, and C-reactive protein, which not only triggers muscle cell apoptosis but also muscle fiber atrophy, ultimately resulting in the prevalence of low muscle mass.[29]

Our study confirmed the predictive value of the TyG index for low muscle mass in participants with T2D. The TyG index showed a negative association with the increase of ASMI and a positive association with the higher risk of low muscle mass in patients with T2D. The article also benefited from a large sample size of hospitalized patients with T2D, a novel concept, and accurate evaluation of body composition. However, the study has potential limitations due to missing information on physical activity and dietary habits, and the observational nature of the study precludes making causal inferences. Furthermore, the participants included in this cross-sectional analysis were from a single center, so further investigation is needed to determine if the effects of the TyG index on low muscle mass are applicable to different populations and cohort studies.

Conclusion

In conclusion, we have demonstrated a significant correlation between higher TyG index levels and decreased levels of ASMI. Additionally, we observed a positive association between the TyG index and low muscle mass in participants with T2D. Further prospective and multi-center studies are necessary to confirm the causal relationship.

Data Sharing Statement

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

Ethical Approval

The study was authorized by the Tianjin Medical University General Hospital Institutional Review Board (approval number: IRB2020-YX-027-01).

Informed Consent

The requirement for informed consent was waived since the study used data from medical records with concealed identity.

Author Contributions

All authors conceived and designed the study and participated in the analysis and interpretation of the data. Qinying Zhao collected baseline characteristics of participants from the Department of Endocrinology and Metabolism of Tianjin Medical University General Hospital and analyzed the data. Qinying Zhao was a major contributor in writing the manuscript. Ziyue Zhang assisted in statistical analysis and manuscript writing. Shuo Li also helped in the collection of baseline data. Ming Liu is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. All authors took part in drafting, revising or critically reviewing the article; gave final approval for 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

Qinying Zhao and Ziyue Zhang are co-first authors for this study. The authors declared that they have no potential conflicts of interest in this work.

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