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Association Between Triglyceride–Glucose Body Mass Index and Mortality in Acute Ischemic Stroke Patients With Medium Vessel Occlusion: A Multicenter Retrospective Cohort Study

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Abstract

Background

The triglyceride-glucose body mass index (TyG-BMI), a composite indicator integrating lipid–glucose metabolism and obesity, is associated with insulin resistance and cardiovascular risk; however, its relationship with mortality in acute ischemic stroke (AIS) patients with medium vessel occlusion (MeVO) unclear.

Methods

This was a multicenter, retrospective cohort study that included patients with MeVO-related AIS from three comprehensive stroke centers between January 2021 and March 2025. Patients were stratified into high and low TyG-BMI groups according to the optimal cutoff value determined by receiver operating characteristic (ROC) analysis. Primary outcomes were 90-day and 1-year all-cause mortality. Survival was compared via Kaplan-Meier, and TyG-BMI–mortality associations were assessed using Cox regression and restricted cubic splines,.with interaction and subgroup analyses for robustness.

Results

Among 187 eligible AIS patients with MeVO (median age 69.0 years; 35.8% male), 18 (9.6%) and 35 (18.7%) died within 90 days and 1 year, respectively. In fully adjusted Cox models, TyG-BMI was independently associated with increased risks of 90-day mortality (HR = 1.027, 95% CI: 1.011–1.043, P = 0.001) and 1-year mortality (HR = 1.024, 95% CI: 1.012–1.037, P < 0.001). RCS analysis indicated a linear positive association between TyG-BMI and both mortality endpoints. Subgroup analyses showed generally consistent associations across strata, with a significant interaction observed for diabetes in relation to 90-day mortality (P for interaction = 0.043).

Conclusions

The TyG-BMI index is significantly and linearly associated with all-cause mortality in AIS patients with MeVO, suggesting its potential as a risk stratification tool in this population.

Introduction

Acute ischemic stroke (AIS) is a leading cause of death and disability worldwide[1]. Medium vessel occlusion (MeVO), typically defined as occlusion of the M2/M3 segments of the middle cerebral artery (MCA), A2/A3 segments of the anterior cerebral artery (ACA), or P2/P3 segments of the posterior cerebral artery (PCA)[2, 3], accounts for 25%-40% of all AIS cases[4, 5]. Although MeVO strokes cause milder clinical deficits and disability than large vessel occlusion (LVO) strokes, one-third of patients fail

to achieve functional independence within 90 days, with a substantial proportion dying within 90 days, highlighting the urgent need for reliable prognostic biomarkers [2].

Insulin resistance (IR) is a fundamental pathophysiological characteristic of metabolic syndrome and plays a key role in the development and progression of cerebrovascular diseases [6, 7]. The triglyceride-glucose (TyG) index, calculated as the product of fasting triglycerides and fasting blood glucose, has been recognized as a reliable surrogate marker of insulin resistance [8]. Previous studies have validated its predictive ability for cardiovascular disease outcomes, including acute myocardial infarction, coronary heart disease, heart failure, stroke, and hypertension (9–13). Notably, combining TyG index with obesity indicators, including body mass index (BMI), waist circumference, and waist-to-height ratio, significantly improves the accuracy of IR assessment[9, 10]. Studies have shown that TyG-BMI is closely associated with diabetes, hypertension, and non-alcoholic fatty liver disease, as well as cardiovascular outcomes (11–13).

Recent findings on TyG-BMI and stroke outcomes are inconsistent. Huang et al. reported that lower TyG-BMI was associated with increased long-term mortality in critically ill stroke patients based on the MIMIC-IV database [10], while Ouyang et al. reported a positive association between TyG-BMI and 28-day mortality in critically ill ischemic stroke patients using the eICU database[13]. Studies in general populations have also suggested that elevated TyG-BMI is an important risk factor for stroke occurrence. These observations suggest that the prognostic role of TyG-BMI may depend on the specific clinical context, thus necessitating disease-specific investigation. To date, the role of TyG-BMI in the MeVO patient population remains inadequately explored. Therefore, we conducted this multicenter retrospective cohort study to investigate the association between the TyG-BMI index and 90-day and 1-year all-cause mortality in patients with MeVO stroke.

Methods

Study Design and Population

This retrospective multicenter cohort study was conducted at three comprehensive stroke centers. A total of 187 patients with MeVO-related AIS admitted between January 2021 and March 2025 were included to investigate the association between the TyG-BMI index and 90-day and 1-year all-cause mortality. The Xiangyang No.1 People's Hospital Human Research Ethics Committee approved this study (No. 2025KY031), waiving the requirement for written informed consent.The research was con-ducted in accordance with the Declaration of Helsinki. MEVO was defined as occlusion of the M2 or M3 segment of the middle cerebral artery, A2 or A3 segment of the anterior cerebral artery, or P2 or P3 segment of the posterior cerebral artery, all confirmed by vascular imaging. The inclusion criteria required patients aged ≥ 18 years with a diagnosis of cerebral infarction with MEVO. Exclusion criteria included: (1) pre-stroke modified Rankin Scale (mRS) score ≥ 2; (2) missing data on fasting plasma glucose, triglycerides, or BMI; (3) loss to follow-up at 90 days or 1 year.The inclusion flowchart is shown in Fig. 1.

Data Collection

Demographic, clinical, and laboratory data were extracted from electronic medical records. Variables collected included: age, sex, hospital stay, pre-stroke mRS score[14], baseline National Institutes of Health Stroke Scale (NIHSS) score [15], smoking status, drinking status, history of hypertension, diabetes mellitus, atrial fibrillation, coronary heart disease, and previous use of antiplatelet agents, anticoagulants, or statins. Clinical parameters on admission comprised systolic and diastolic blood pressure. Laboratory measurements included albumin, hemoglobin, urea, total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), white blood cell count, neutrophil count, lymphocyte count, platelet count, total protein, albumin/globulin ratio, creatinine, and uric acid. Radiological assessment was performed using the Alberta Stroke Program Early Computed Tomography Score (ASPECTS) [16]. Stroke etiology was classified according to the Trial of Org 10172 in Acute Stroke Treatment (TOAST) criteria [17]. Occlusion sites were categorized as M2/M3, A2/A3, or P2/P3 segments. Treatment-related variables included intravenous thrombolysis (IVT), tirofiban use, and successful recanalization, which was defined as modified Thrombolysis in Cerebral Infarction (mTICI) grade 2b–3 [18]. Symptomatic intracranial hemorrhage (sICH) was documented based on the Heidelberg Bleeding Classification [19].

Fasting plasma glucose (FPG) and triglycerides (TG) were measured from venous blood samples obtained after an overnight fast within 24 hours of admission, using an automated biochemical analyzer. Height and weight were recorded on admission, and BMI was calculated as weight (kg) divided by height (m^2). The TyG-BMI index was computed using the following formula:TyG-BMI = Ln [TG (mg/dL) × FPG (mg/dL) / 2] × BMI (kg/m^2) [20].

The primary endpoints of interest were all-cause mortality at 90 days and 1 year after admission. Vital status was ascertained through hospital records or telephone follow-up interview.

Statistical Analysis

Statistical analyses were performed using R version 4.4.2 (R Foundation for Statistical Computing). Two-tailed P values < 0.05 were considered statistically significant. Missing baseline data were imputed by multiple imputation, as detailed in **the supplementary material 1.** Continuous variables with normal distribution are expressed as mean ± standard deviation (SD), while variables with non-normal distribution are expressed as median and interquartile range (IQR). Categorical variables are reported as counts and percentages. For univariate comparisons, the Kruskal-Wallis test or Mann-Whitney U test was used for continuous variables, and the chi-square (χ^2) test or Fisher's exact test was used for categorical variables.

Given the absence of a predefined cut-off for TyG-BMI, we used ROC curve analysis with the Youden index to identify an optimal threshold of 215.53, dividing participants into high (≥ 215.53) and low (< 215.53) groups. Kaplan–Meier curves illustrated the cumulative incidence of endpoints during follow-up. To evaluate the association between TyG-BMI and mortality, we fitted Cox proportional-hazards models

in three nested specifications: Model 1 was unadjusted; Model 2 adjusted for age and sex; and Model 3 further adjusted for endovascular treatment, prestroke status, baseline NIHSS score, smoking, hypertension, diabetes, atrial fibrillation, coronary heart disease, ASPECTS score, albumin, hemoglobin, urea, TOAST etiology, occlusion site, intravenous thrombolysis, successful recanalization, and symptomatic intracranial hemorrhage. Additionally, we performed interaction and subgroup analyses to assess the robustness of our findings, and we presented the results as forest plots.

Results

Baseline Characteristics of the Study Population

Among 282 screened patients, 87 were excluded for missing FPG/TG data and 8 were lost to follow-up, leaving 187 patients (median age 69 years; 35.8% male) for analysis (Fig. 1). Using the ROC-derived optimal cut-off of 215.53, patients were stratified into low (≤ 215.53, n = 95) and high (> 215.53, n = 92) TyG-BMI groups. Baseline characteristics are summarized in Table 1. The high TyG-BMI group had significantly higher proportions of hypertension (90.2% vs. 77.9%, P = 0.036) and diabetes (35.9% vs. 20.0%, P = 0.024), along with elevated fasting glucose (7.02 vs. 5.92 mmol/L, P = 0.001) and triglycerides (1.29 vs. 0.96 mmol/L, P < 0.001). No other significant between-group differences were observed for the remaining baseline variables (all P > 0.05).

Table 1
Baseline characteristics stratified by TyG-BMI index

Variable	Overall (n = 187)	Low TyG-BMI (n = 95)	High TyG-BMI (n = 92)	P value
Age (years)	69 (60–75)	69 (61–74.5)	68 (60–75)	*0.912*
Male (n, %)	67 (35.8)	33 (34.7)	34 (37.0)	*0.870*
Hospital stay (days)	10 (7–13)	10 (7–13)	11 (7.75–13.25)	*0.265*
Smoking (n, %)	56 (29.9)	29 (30.5)	27 (29.3)	*0.987*
Drinking (n, %)	45 (24.1)	24 (25.3)	21 (22.8)	*0.827*
Prestroke mRS ≥ 2 (n, %)	73 (39.0)	36 (37.9)	37 (40.2)	*0.861*
Hypertension (n, %)	157 (84.0)	74 (77.9)	83 (90.2)	*0.036*
Diabetes mellitus (n, %)	52 (27.8)	19 (20.0)	33 (35.9)	*0.024*
Atrial fibrillation (n, %)	36 (19.3)	19 (20.0)	17 (18.5)	*0.938*
Coronary heart disease (n, %)	39 (20.9)	20 (21.1)	19 (20.7)	*1.000*
Previous antiplatelet (n, %)	26 (13.9)	14 (14.7)	12 (13.0)	*0.902*
Previous anticoagulation (n, %)	8 (4.3)	4 (4.2)	4 (4.3)	*1.000*
Previous statin (n, %)	21 (11.2)	10 (10.5)	11 (12.0)	*0.938*
Systolic blood pressure (mmHg)	146 (136–162)	145 (136–162)	147 (134.75–162)	*0.808*
Diastolic blood pressure (mmHg)	86 (78.5–95)	86 (78–97)	86 (79.75–93)	*0.414*
Laboratory examination				
TyG-BMI index	212.47 (185.74–244.34)	186.15 (169.86–202.26)	245.64 (227.07–262.67)	*< 0.001*
Fasting plasma glucose (mmol/L)	6.7 (5.57–8.41)	5.92 (5.3–7.8)	7.02 (6.1–8.91)	*0.001*
Triglycerides (mmol/L)	1.12 (0.82–1.62)	0.96 (0.72–1.33)	1.29 (0.97–2.04)	*< 0.001*

Abbreviations: TyG-BMI, triglyceride-glucose body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; TG, triglyceride; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; WBC, white blood cell; NIHSS, National Institutes of Health Stroke Scale; ASPECTS, Alberta Stroke Program Early CT Score; IVT, intravenous thrombolysis; sICH, symptomatic intracranial hemorrhage; TOAST, Trial of Org 10172 in Acute Stroke Treatment; CHD, coronary heart disease; mRS, modified Rankin Scale.

Variable	Overall (n = 187)	Low TyG-BMI (n = 95)	High TyG-BMI (n = 92)	P value
Total cholesterol (mmol/L)	3.88 (3.3–4.62)	3.8 (3.26–4.44)	3.96 (3.38–4.74)	*0.132*
HDL-C (mmol/L)	1.03 (0.92–1.26)	1.06 (0.94–1.27)	0.99 (0.9–1.23)	*0.082*
LDL-C (mmol/L)	2.3 (1.7–2.91)	2.17 (1.63–2.81)	2.49 (1.81–3)	*0.068*
WBC (×10⁹/L)	7.02 (5.63–9.05)	7.08 (5.63–8.84)	7.01 (5.68–9.25)	*0.831*
Neutrophil count (×10⁹/L)	4.86 (3.7–6.74)	5.1 (3.8–6.68)	4.56 (3.62–6.83)	*0.640*
Lymphocyte count (×10⁹/L)	1.28 (0.95–1.79)	1.16 (0.94–1.68)	1.35 (0.97–1.85)	*0.252*
Platelet count (×10⁹/L)	205 (171.5–250)	205 (172.5–245)	205 (171.75–259.5)	*0.991*
Hemoglobin (g/L)	128 (116–142.5)	132 (116–144)	127 (116.5–137.25)	*0.335*
Total protein (g/L)	63.9 (60.8–68.55)	63.8 (61.1–69.05)	63.95 (60.22–68.2)	*0.524*
Albumin (g/L)	37.8 (34.95–40.95)	38.17 (35.4–41.1)	37.6 (34.85–40.55)	*0.198*
A/G ratio	1.45 (1.29–1.6)	1.5 (1.3–1.6)	1.44 (1.27–1.6)	*0.462*
Urea (mmol/L)	5.4 (4.4–7.15)	5.4 (4.4–7.36)	5.4 (4.47–7.03)	*0.727*
Creatinine (μmol/L)	70.8 (60–84.15)	70.5 (60–79.75)	72.65 (60–88.95)	*0.373*
Uric acid (μmol/L)	320 (255.5–381)	324 (264–384.2)	307.1 (250–367.5)	*0.444*
NIHSS at admission	7 (3–14)	6 (2.5–12.5)	8 (3–15)	*0.349*
ASPECTS score	9 (7–9)	9 (7–9)	9 (7–9)	*0.818*
TOAST classification (n, %)				**0.767**
Large artery atherosclerosis	147 (78.6)	73 (76.8)	74 (80.4)	

Abbreviations: TyG-BMI, triglyceride-glucose body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; TG, triglyceride; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; WBC, white blood cell; NIHSS, National Institutes of Health Stroke Scale; ASPECTS, Alberta Stroke Program Early CT Score; IVT, intravenous thrombolysis; sICH, symptomatic intracranial hemorrhage; TOAST, Trial of Org 10172 in Acute Stroke Treatment; CHD, coronary heart disease; mRS, modified Rankin Scale.

Variable	Overall (n = 187)	Low TyG-BMI (n = 95)	High TyG-BMI (n = 92)	P value
Cardioembolism	23 (12.3)	12 (12.6)	11 (12.0)	
Other/undetermined	17 (9.1)	10 (10.5)	7 (7.6)	
Hemisphere affected (n, %)				**0.487**
Left	89 (47.6)	43 (45.3)	46 (50.0)	
Right	91 (48.7)	47 (49.5)	44 (47.8)	
Bilateral/posterior	7 (3.7)	5 (5.3)	2 (2.2)	
Occlusion site (n, %)				**0.228**
M2/M3	119 (63.6)	66 (69.5)	53 (57.6)	
A2/A3	13 (7.0)	5 (5.3)	8 (8.7)	
P2/P3	55 (29.4)	24 (25.3)	31 (33.7)	
Treatment				
Intravenous thrombolysis (n, %)	41 (21.9)	24 (25.3)	17 (18.5)	*0.345*
Tirofiban (n, %)	15 (8.1)	7 (7.4)	8 (8.7)	*0.965*
Outcomes				
sICH (n, %)	2 (1.1)	0 (0.0)	2 (2.2)	*0.463*

Abbreviations: TyG-BMI, triglyceride-glucose body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; TG, triglyceride; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; WBC, white blood cell; NIHSS, National Institutes of Health Stroke Scale; ASPECTS, Alberta Stroke Program Early CT Score; IVT, intravenous thrombolysis; sICH, symptomatic intracranial hemorrhage; TOAST, Trial of Org 10172 in Acute Stroke Treatment; CHD, coronary heart disease; mRS, modified Rankin Scale.

Associations of the TyG-BMI Index with All-Cause Mortality

Among the 187 patients, 18 (9.6%) died within 90 days and 35 (18.7%) died within one year. Kaplan-Meier survival analysis was performed to compare all-cause mortality between the two patient groups based on the TyG-BMI index. The analysis showed significantly different survival curves for both 90-day (log-rank P = 0.039; Fig. 2A) and 1-year endpoints (log-rank P = 0.001; Fig. 2B) between the two groups. Patients in the high TyG-BMI group had consistently lower survival rates than those in the low TyG-BMI group (Fig. 2).

We conducted Cox proportional hazards regression analysis to evaluate the association between TyG-BMI and mortality, and the results are summarized in Table 2. When analyzed as a continuous variable,

TyG-BMI was not significantly associated with 90-day mortality in the unadjusted Model 1 (HR 1.007, 95% CI 0.998–1.016, P = 0.145) and Model 2 adjusted for age and sex (HR 1.009, 95% CI 0.999–1.018, P = 0.068). However, in the fully adjusted Model 3, the TyG-BMI index was significantly associated with 90-day mortality (HR 1.027, 95% CI 1.011–1.043, P = 0.001). For 1-year mortality, TyG-BMI as a continuous variable was significantly associated with mortality across all three models, with the strongest effect observed in Model 3 (HR 1.024, 95% CI 1.012–1.037, P < 0.001). When categorized using the cutoff of 215.53, the high TyG-BMI group had significantly elevated death risk. In the fully adjusted Model 3, the high TyG-BMI group had a 9.45-fold increased risk of 90-day mortality (95% CI 2.315–38.584, P = 0.002) and a 12.66-fold increased risk of 1-year mortality (95% CI 4.291–37.355, P < 0.001) (see Table 2). Furthermore, RCS analysis showed a linear positive association between the TyG-BMI index and the risk of 90-day and 1-year all-cause death (P-overall = 0.004 and < 0.001, respectively; P-nonlinear = 0.532 and 0.844, respectively) (Fig. 3).

Table 2
Multivariate Cox Regression Analyses for 90-Day and 1-Year All-Cause Mortality

Categories	**Model 1**		**Model 2**		**Model 3**	
	HR (95% CI)	**P value**	**HR (95% CI)**	**P value**	**HR (95% CI)**	**P value**
90-Day All-Cause Mortality (Events = 18/187)						
TyG-BMI continuous variable						
Per unit increase	1.007 (0.998–1.016)	*0.145*	1.009 (0.999–1.018)	*0.068*	1.027 (1.011–1.043)	*0.001*
TyG-BMI categorical variable						
Low TyG-BMI (reference)	1 (reference)	—	1 (reference)	—	1 (reference)	—
High TyG-BMI	2.818 (1.005–7.904)	*0.049*	2.730 (0.971–7.677)	*0.057*	9.450 (2.315–38.584)	*0.002*
1-Year All-Cause Mortality (Events = 35/187)						
TyG-BMI continuous variable						
Per unit increase	1.008 (1.002–1.014)	*0.014*	1.010 (1.003–1.016)	*0.003*	1.024 (1.012–1.037)	*< 0.001*
TyG-BMI categorical variable						
Low TyG-BMI (reference)	1 (reference)	—	1 (reference)	—	1 (reference)	—
High TyG-BMI	3.332 (1.561–7.114)	*0.002*	3.436 (1.605–7.356)	*0.001*	12.661 (4.291–37.355)	*< 0.001*

Subgroup Analysis

To further evaluate the association between TyG-BMI and mortality, we conducted subgroup analyses to assess the association between sex, diabetes, atrial fibrillation, baseline NIHSS (> 5 vs. ≤5), intravenous thrombolysis, and 90-day and 1-year all-cause mortality in MeVO patients (Fig. 4). Subgroup analysis showed that the association between TyG-BMI and increased mortality risk was consistent across all predefined subgroups, with no significant interaction (P for interaction > 0.05). Notably, the interaction between TyG-BMI and diabetes status approached statistical significance for 90-day mortality (*P* for interaction = 0.043), suggesting that the predictive effect might be stronger in diabetic patients (HR 4.946, 95% CI 4.728–5.173, *P* < 0.001). For 1-year mortality, the association was consistent across all strata, further confirming the robustness of the main findings. Notably, in patients with atrial fibrillation, the association was particularly strong (HR 16.725, 95% CI 15.976–17.509, P < 0.001).

Discussion

In this multicenter retrospective cohort study of 187 patients with MeVO-related acute ischemic stroke, we demonstrated that the TyG-BMI index was independently and linearly associated with 90-day and 1-year all-cause mortality. After comprehensive multivariable adjustment, each 1-unit increase in TyG-BMI corresponded to a 2.7% and 2.4% higher risk of 90-day and 1-year death, respectively, and patients with TyG-BMI above the optimal cutoff of 215.53 exhibited substantially elevated mortality risks. Restricted cubic spline analysis confirmed a monotonically increasing dose-response relationship without evidence of nonlinearity or a threshold effect, suggesting that TyG-BMI may serve as a practical risk stratification tool in this population.

Our results are consistent with extensive evidence on the role of insulin resistance (IR) in adverse cerebrovascular outcomes and further extend this evidence base[21]. IR promotes endothelial dysfunction, systemic inflammation, and hypercoagulable states—all of which can exacerbate cerebral ischemia and impair tissue reperfusion[22, 23]. The TyG index, as a surrogate of IR, has been associated with stroke occurrence and adverse functional outcomes in multiple cohorts [9, 24–26]. Recent studies have further demonstrated that TyG-BMI, which combines IR and obesity indicators, provides superior prognostic value compared to TyG index alone (10,19–21). Sun et al. [27]demonstrated in a multicenter cohort of ischemic stroke patients undergoing endovascular therapy that elevated TyG index was independently associated with all-cause mortality. Obesity amplifies IR and independently increases cardiovascular risk through several complementary mechanisms, including adipokine dysregulation, increased free fatty acid flux, and systemic low-grade inflammation[7, 23, 28]. The TyG-BMI index has previously been shown to have clinical value in predicting hypertension[29], non-alcoholic fatty liver disease[30], diabetes mellitus[31], and severe coronary artery disease[32]. In our study, the significant and independent linear association between the TyG-BMI index and death risk in MeVO patients suggests that this composite metabolic index may serve as a practical tool for early risk stratification in this population.

The relationship between TyG-BMI and stroke outcomes is not uniform across the existing literature. Huang et al. reported an inverse association between TyG-BMI and long-term all-cause mortality in critically ill stroke patients using the MIMIC-IV database [10], which may reflect the "obesity paradox" or "reverse epidemiology" commonly observed in ICU populations—where increased adiposity may provide short-term metabolic reserve to buffer acute physiological stress [33–35]. In contrast, Ouyang et al.[13] showed a positive association between TyG-BMI and 28-day mortality in critically ill AIS patients using the eICU database, consistent with our findings. The discrepancies among these studies likely stem from differences in patient populations (ICU hospitalization vs. EVT treatment), outcome definitions (in-hospital vs. 28-day vs. 90-day/1-year), and the specific clinical context of MeVO. Our cohort exclusively included patients with medium vessel cerebral infarction—a group in which outcomes may be more directly influenced by pre-existing metabolic abnormalities rather than acute physiological reserve. Furthermore, robust evidence from general stroke populations has consistently established that higher TyG-BMI serves as a significant predictor of poorer long-term prognosis, including stroke recurrence and cardiovascular mortality. [36–38], which aligns with the positive mortality association observed in our study.

Furthermore, this study conducted subgroup analyses to explore risk stratification. Although the association between the TyG-BMI index and all-cause mortality was largely consistent across subgroups, some differences were observed. The interaction between TyG-BMI and diabetes status was significant for 90-day mortality (P for interaction = 0.043). In patients with comorbid diabetes, the risk associated with elevated TyG-BMI was substantially increased. [39, 40]. Notably, the diabetes interaction effect attenuated and was no longer significant for 1-year mortality (interaction P = 0.102), suggesting that the acute-phase diabetes-IR synergistic effect may be most prominent, while long-term death risk is more driven by cumulative factors including comorbidities, functional status, and secondary prevention[41, 42]. However, given the limited number of diabetic patients with 90-day death events, the precision of this interaction estimate is limited and requires validation in larger prospective studies.

Several limitations must be acknowledged. First, as a retrospective observational study, it can only imply associations rather than causality. Despite comprehensive adjustment, residual confounding from unmeasured variables cannot be excluded. Second, TyG-BMI was measured only at admission; serial assessments could not be performed to evaluate its dynamic prognostic value. Third, BMI as a surrogate for visceral fat mass is imperfect; future studies incorporating waist circumference or imaging-based fat quantification may provide more accurate discriminatory ability. Finally, the moderate sample size (n = 187) limits statistical power, and extreme estimates warrant cautious interpretation. Larger prospective cohorts are needed to validate our findings.

Conclusions

Elevated TyG-BMI was independently and linearly associated with higher 90-day and 1-year all-cause mortality in AIS-MeVO patients, with high levels conferring greater short-and long-term risks. Derived from routine admission data, TyG-BMI is a practical, economical biomarker for early risk stratification.

These findings suggest that targeting insulin resistance and obesity may be a modifiable strategy to improve outcomes, though larger prospective validation is warranted.

Abbreviations

AIS
acute ischemic stroke
ASPECTS
Alberta Stroke Program Early CT Score
BMI
body mass index
CI
confidence interval
EVT
endovascular treatment
FPG
fasting plasma glucose
HR
hazard ratio
IR
insulin resistance
IVT
intravenous thrombolysis
LVO
large vessel occlusion
MeVO
medium vessel occlusion
mRS
modified Rankin Scale
NIHSS
National Institutes of Health Stroke Scale
RCS
restricted cubic spline
ROC
receiver operating characteristic
sICH
symptomatic intracranial hemorrhage
TG
triglyceride
TOAST

Trial of Org 10172 in Acute Stroke Treatment
TyG-BMI
triglyceride-glucose body mass index.

Declarations

Ethics approval and informed consent

The Xiangyang No.1 People's Hospital Human Research Ethics Committee approved this study (No. 2025KY031), waiving the requirement for written informed consent. The research was conducted in accordance with the Declaration of Helsinki.

Consent for publication

Not applicable.

Data availability

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

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Contributor

Z.B. and T.L. contributed equally to this work and share first authorship. Z.B. and T.L.: conceptualization, data curation, formal analysis, writing—original draft. Z.L., F.Z., and Y.L.: data collection, investigation, and validation. Y.S., J.H., and W.D.: methodology, supervision, and project administration. P.Z. and W.D.: conceptualization, funding acquisition, resources, writing—review and editing, and corresponding responsibility. All authors reviewed and approved the final manuscript.

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Disclosure

The authors report no conflicts of interest in this work.

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Figures

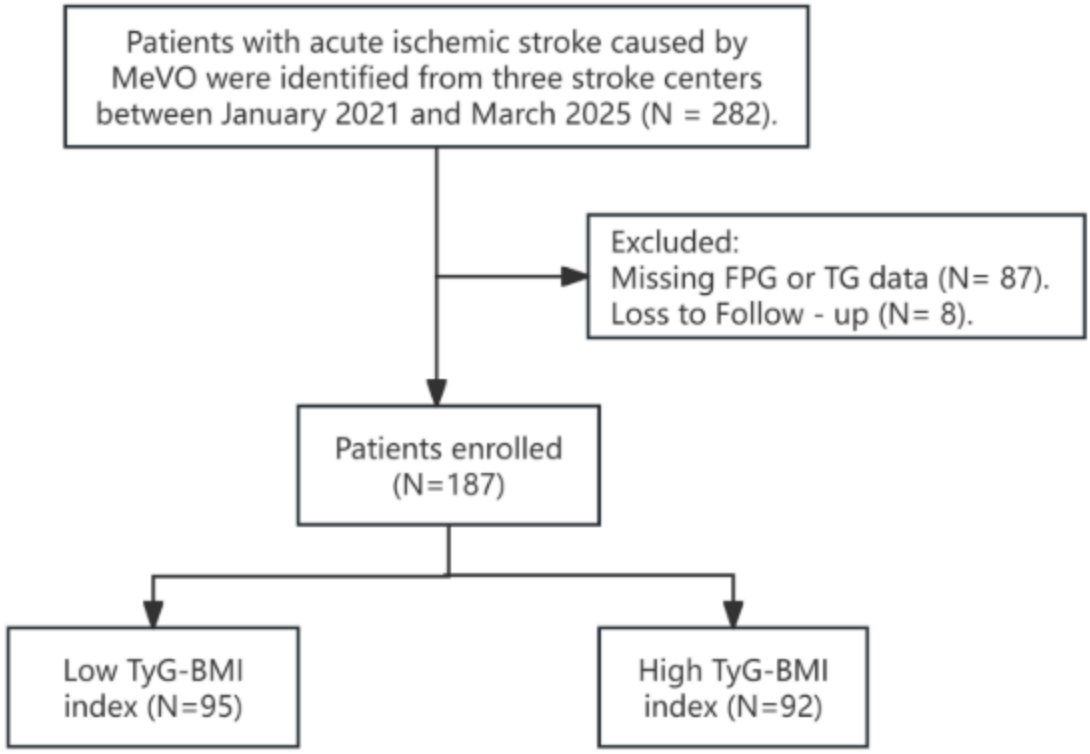


Figure 1

Study flow diagram. A total of 282 patients with acute ischemic stroke caused by medium vessel occlusion (MeVO) were initially screened. After excluding 87 patients for missing metabolic data and 8 for loss to follow-up, 187 patients were included in the final analysis. FPG, fasting plasma glucose; TG, triglyceride; BMI, body mass index; MeVO, medium vessel occlusion.

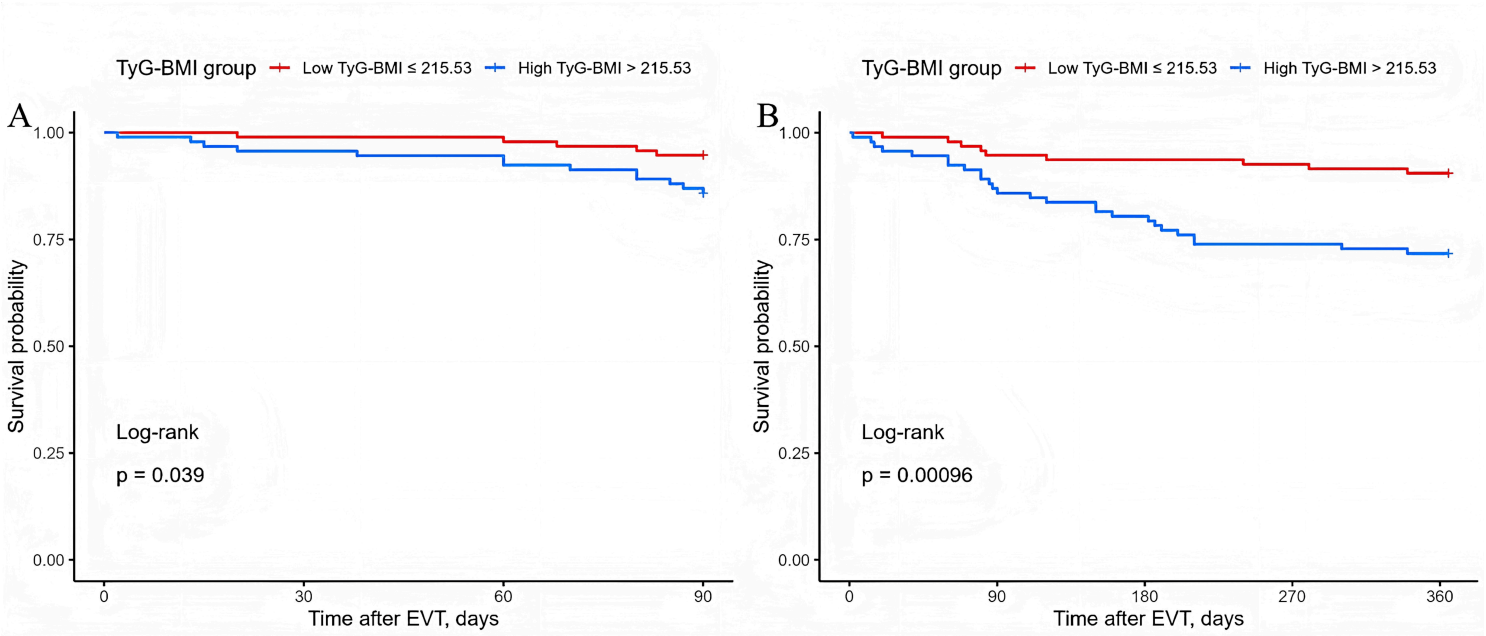


Figure 2

Kaplan–Meier survival curves stratified by TyG-BMI group. (A) 90-day all-cause mortality. (B) 1-year all-cause mortality. Patients were classified into the high TyG-BMI group (TyG-BMI >215.53, n=92) and low TyG-BMI group (TyG-BMI ≤215.53, n=95). Statistical comparisons were performed using the log-rank test. TyG-BMI, triglyceride-glucose body mass index.

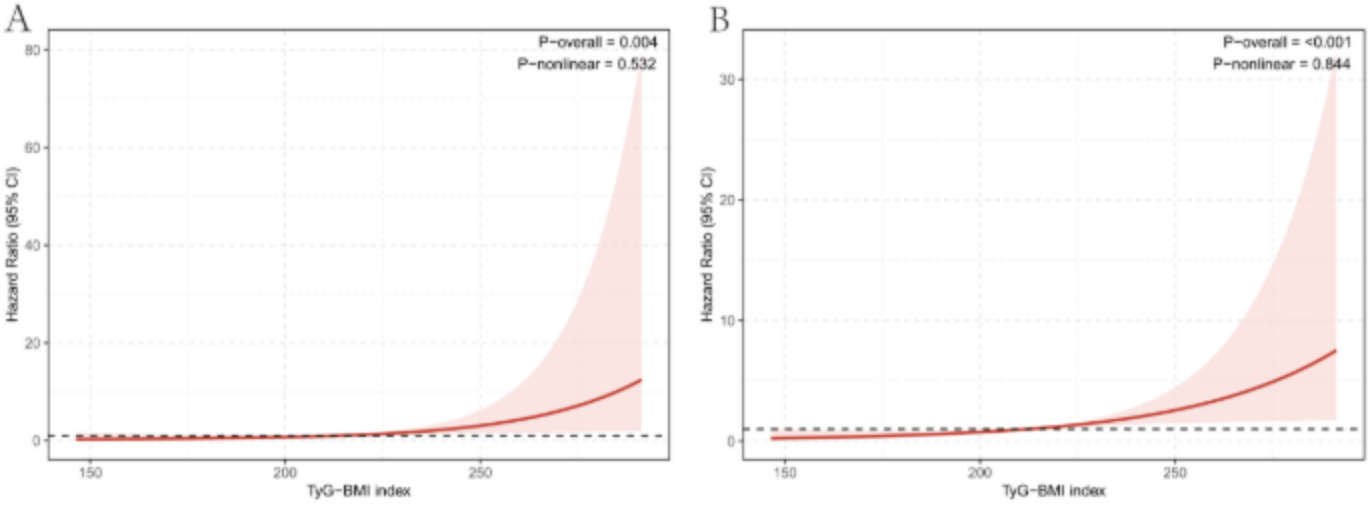


Figure 3

Restricted cubic spline curves depicting the dose–response relationship between TyG-BMI and mortality risk in the fully adjusted Cox model. (A) 90-day all-cause mortality; (B) 1-year all-cause mortality. The solid line represents the estimated hazard ratio, and the shaded area indicates the 95% confidence interval. The reference value is the median TyG-BMI (212.47). P overall and P for nonlinearity are shown for each outcome. TyG-BMI, triglyceride-glucose body mass index.

Supplementary Files

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- SupplementaryFigure.docx