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Non-linear relationship between the triglyceride glucose-a body shape index and stroke risk: a prospective cohort study from the China health and retirement longitudinal study

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Abstract

Objective Recent evidence suggests a strong association between triglyceride glucose-a body shape index (TyG-ABSI) and cardiovascular mortality. However, the association between TyG-ABSI and incident stroke has not been explored. Our study utilized a prospective cohort design to explore the potential association between TyG-ABSI and incident stroke within a Chinese population.

Methods A total of 8,715 subjects from the China Health and Retirement Longitudinal Study were included in this analysis, categorized into stroke ($n=805$) and non-stroke ($n=7,910$) groups. To assess the association between TyG-ABSI and incident stroke, this study employed Cox proportional hazards regression, restricted cubic spline models, subgroup analyses, and sensitivity analyses.

Results The mean age of participants was significantly higher in the stroke group (61.26 ± 8.42 years) compared to the non-stroke group (59.14 ± 9.27 years). Multivariate analysis revealed that higher TyG-ABSI was significantly associated with increased stroke risk, with an HR of 1.22 (95% CI: 1.09–1.37, $P=0.0004$). Notably, this association between TyG-ABSI and incident stroke was non-linear, and a significant inflection point was identified at a TyG-ABSI of 6.42. The HR to the left and right of this inflection point were 2.44 (95%CI: 1.19–5.02, $P=0.0151$) and 1.16 (95%CI: 1.02–1.31, $P=0.0239$), respectively. Subgroup and sensitivity analyses further validated the robustness of the association between TyG-ABSI and incident stroke.

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Conclusion Our analysis revealed a non-linear positive association between TyG-ABSI and incident stroke. These findings emphasize the necessity of incorporating TyG-ABSI monitoring into clinical practice to identify individuals at greater risk for stroke.

Keywords Nonlinearity, A body shape index, Triglyceride glucose-a body shape index, Stroke, Triglyceride, Glucose

Introduction

Stroke is the leading cause of mortality and the second most significant contributor to disability among adults, imposing substantial economic and societal burdens on both families and society [1–3]. A large-scale survey revealed that, in 2020, the Chinese population aged 40 years and older experienced approximately 3.4 million new stroke cases, 17.8 million prevalent stroke cases, and 2.3 million deaths attributable to stroke [4]. Lifestyle modifications—particularly dietary interventions that modulate intestinal inflammation—have been shown to improve cardiometabolic risk profiles [5–7]. Therefore, the identification and effective management of stroke risk factors, together with the implementation of such lifestyle modifications, are essential for preventing stroke and mitigating its associated economic and societal impacts.

Currently, the triglyceride-glucose (TyG) index is widely recognized as a simple and non-invasive indices for evaluating insulin resistance (IR) [8, 9]. In addition, the TyG index is more commonly utilized due to its independence from insulin therapy and its well-established association with cardiovascular disease [10, 11]. A multitude of studies have demonstrated a significant association between the TyG index and an elevated risk of metabolic and cardiovascular conditions, such as hypertension, coronary artery disease, atherosclerosis, stroke, and diabetes [12–16]. These findings highlight its potential as a comprehensive marker for cardiometabolic health.

Obesity, a major global health concern, is widely recognized as a significant predictor of cardiovascular disease and is closely linked to IR [17]. Studies have shown that excess adiposity contributes to systemic inflammation, dyslipidemia, and impaired glucose metabolism, all of which exacerbate cardiovascular risk [18–20]. Traditional obesity-related parameters, such as body mass index (BMI), have been extensively utilized to gauge health risks associated with obesity [21–23]. However, BMI alone does not account for fat distribution, which plays a critical role in cardiovascular outcomes [24]. To address this limitation, indices such as a body shape index (ABSI) have been developed to better capture the impact of body composition and fat distribution on cardiovascular health [25, 26].

Because insulin resistance and central obesity act through complementary pathophysiological pathways to increase cardiovascular risk, composite indices

that combine metabolic and anthropometric information have been proposed to improve risk stratification. Examples include TyG index combined with BMI (TyG-BMI) and TyG index combined with waist circumference (TyG-WC); these composite measures have demonstrated improved predictive performance for metabolic syndrome, incident diabetes, and cardiovascular outcomes compared with TyG or anthropometric measures alone [27–31]. The triglyceride glucose–body shape index (TyG-ABSI), which integrates TyG index with ABSI, may further enhance prognostic accuracy for cardiovascular risk. By simultaneously capturing systemic metabolic dysfunction (via TyG) and adverse body shape or central adiposity (via ABSI), TyG-ABSI better reflects the combined burden of metabolic and anthropometric risk factors. Moreover, recent studies suggest that TyG-ABSI has been shown to outperform not only the traditional TyG index but also other composite measures, including TyG-BMI and TyG-WC, in predicting cardiovascular outcomes [32–34]. Despite the growing evidence for composite TyG-based indices in cardiovascular disease and diabetes, there is a notable gap in the literature regarding TyG-ABSI and cerebrovascular outcomes. To our knowledge, no large prospective study has specifically examined the relationship between TyG-ABSI and incident stroke, nor has the potential for a non-linear or threshold association been thoroughly explored. Addressing this gap is important because detecting threshold effects could have direct clinical relevance for risk stratification and prevention strategies.

Therefore, using the China Health and Retirement Longitudinal Study (CHARLS) database, the present study aims to evaluate the association between TyG-ABSI and incident stroke, including assessment of potential non-linear relationships and inflection points, thereby clarifying whether TyG-ABSI provides incremental prognostic information over existing indices.

Methods

Data source

This investigation employed longitudinal data derived from the CHARLS, a nationally representative cohort study spanning 2011–2020 that systematically monitors health and socioeconomic indicators among populations with ≥ 45 years across China's diverse geographical regions [35]. The survey encompasses participants from 28 provinces, 150 counties, and 450 communities, providing comprehensive data on socioeconomic status,

health conditions, and biomarkers [35]. Data collection was performed through structured interviews, physical examinations, and laboratory assessments.

Study population

The research protocol received approval from the Peking University Ethics Committee [35]. Before participant enrollment, informed written consent was carefully obtained from all individuals, ensuring full adherence to the fundamental ethical principles articulated in the Declaration of Helsinki.

The baseline survey (Wave 1, 2011–2012) enrolled 17,708 participants, collecting comprehensive data on covariates and exposure variables. Four additional waves of follow-up surveys were conducted biennially from 2013 to 2020. Participants who participated in any of these follow-up waves were included in the present study. Exclusion criteria included participants lost to follow-up, those with missing information on stroke status, individuals with a history of stroke at baseline, age below 45 years, and those with missing baseline data on triglycerides (TG), fasting plasma glucose (FPG), waist circumference (WC), weight, or height. Additionally, participants with outlier values for TyG-ABSI were excluded. After applying these criteria, the final cohort consisted of 8,715 subjects. Figure 1 presents a comprehensive outline of the participant selection process employed in this study.

Variables

Formula for TyG-ABSI

TyG index = $\ln[\text{TG (mg/dL)} \times \text{FPG (mg/dL)} / 2]$ [9], ABSI = $\text{WC (cm)} / (\text{BMI (kg/m}^2)^{2/3} \times \text{Height (cm)}^{1/2})$ [25], TyG-ABSI = TyG index $\times$ ABSI [25].

Diagnosis of stroke

The outcome variable of our study was the incident stroke, which was determined based on self-reported data [35]. Incident stroke cases were identified among subjects who were stroke-free at the initial assessment and subsequently reported a new stroke event during the longitudinal monitoring period. The follow-up duration was defined as the time from baseline to either the self-reported date of stroke occurrence or the date of the last follow-up, whichever came first. Stroke diagnosis was ascertained through self-reported questionnaires during follow-up interviews, which represents a methodological limitation of our study design. The CHARLS database does not include clinical validation through medical records, neuroimaging, or physician confirmation of stroke events.

Data collection

In this study, variables were selected on the basis of evidence from prior research and clinical expertise to reduce

the impact of confounding factors [36–40]. Comprehensive data were collected from participants by healthcare professionals and investigators, encompassing demographic information (e.g., gender, age), anthropometric measurements (e.g., height, weight, blood pressure, WC), chronic disease status, lifestyle behaviors (e.g., physical activity, drinking, and smoking), and laboratory findings. The presence of heart disease, chronic lung disease (CLD), and liver disease was determined through self-reported information and participants' medication use. Diabetes mellitus (DM) and hypertension were identified using a combination of self-reported data, medication history, clinical assessments, and blood test results. Laboratory data, including high-density lipoprotein cholesterol (HDL-C), white blood cell (WBC), uric acid (UA), FPG, LDL-C, platelet (PLT), blood urea nitrogen (BUN), fasting plasma glucose (FPG), TG, serum creatinine (Scr), C-reactive protein (CRP), hemoglobin, cystatin C, and total cholesterol (TC), were obtained from fasting blood samples.

Missing data processing

Our dataset exhibited some missing values across clinical parameters, with most variables demonstrating high data completeness. The following instances of missing data were recorded: hypertension in 53 subjects (0.61%), WBC in 174 subjects (2.00%), BUN in 1 subject (0.01%), DM in 88 subjects (1.01%), smoking in 5 subjects (0.06%), CLD in 45 subjects (0.52%), heart disease in 53 subjects (0.61%), and liver disease in 72 subjects (0.83%), drinking in 4 subjects (0.05%), HDL-C in 2 subjects (0.02%), PLT in 171 subjects (1.96%), physical activity in 5061 subjects (58.08%), Scr in 4 subjects (0.05%), LDL-C in 17 subjects (0.20%), hemoglobin in 172 subjects (1.97%), and cystatin C in 2120 subjects (24.32%). To mitigate potential bias and maximize use of available data, we performed multiple imputation using the fully conditional specification approach via chained equations [41, 42]. Multiple imputation was conducted to handle missing data, using the fully conditional specification approach with 10 imputations. The imputation model contained a comprehensive set of covariates, encompassing HDL-C, liver disease, cystatin C, age, drinking, TC, smoking, hemoglobin, physical activity, UA, gender, liver disease, heart disease, WBC, CLD, BMI, Scr, hypertension, CRP, DM, PLT, LDL-C, and BUN. The imputation process was conducted under the assumption that the data were missing at random, ensuring the validity of the approach. This method allowed for the preservation of statistical power and minimized bias in the final analyses [42].

Statistical analysis

Normally distributed data were analyzed using the ANOVA test and expressed as mean $\pm$ standard deviation

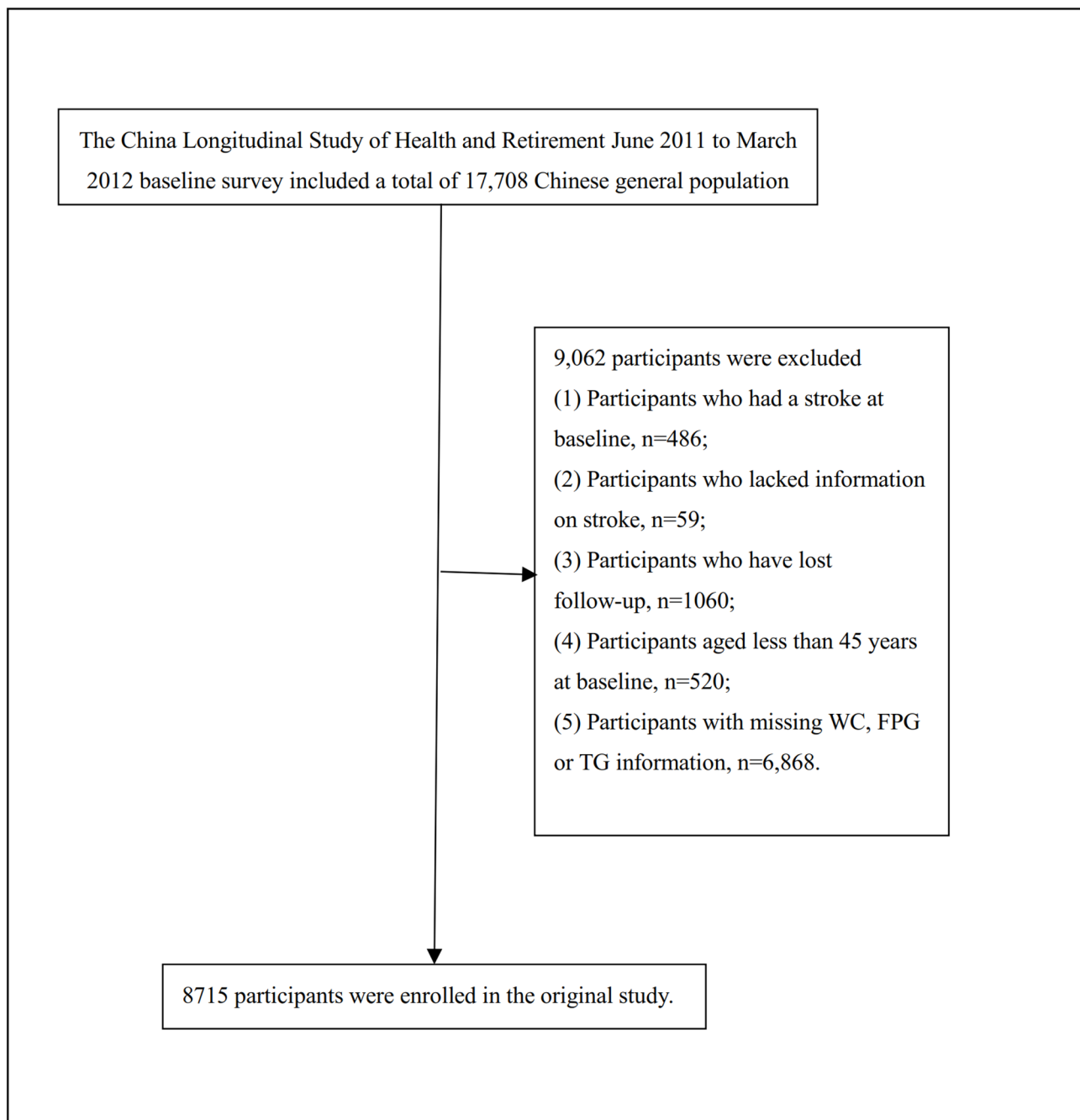


Fig. 1 Study population

(SD). Categorical variables were presented as numbers and percentages, and the chi-square test was used. Skewed distribution data were analyzed using the Kruskal-Wallis test and expressed as median and interquartile range. Multivariable Cox proportional hazard models were employed to evaluate the association between TyG-ABSI and incident stroke. To improve the stability of the relationship between TyG-ABSI and stroke, this investigation performed sensitivity analyses by excluding subjects with diagnoses of DM ($n=7,300$), heart disease

($n=7702$), or hypertension ($n=5154$). Furthermore, restricted cubic spline analyses were performed to test potential nonlinear relationships between TyG-ABSI and stroke occurrence. Subgroup analyses were also conducted to examine the interaction effects of confounding categorical variables. All results were reported in accordance with the STROBE guidelines. Empower-Stats software was used for all statistical analyses, and the significance threshold was set at a two-tailed P value of less than 0.05.

Results

Characteristics of participants

A total of 8,715 participants were included, among whom 805 (9.24%) experienced stroke events during the follow-up period. Table 1 summarizes baseline characteristics overall and by stroke status. Participants in the stroke cohort exhibited a significantly greater average age than those in the non-stroke cohort (61.26 ± 8.42 years vs. 59.14 ± 9.27 years). BMI and ABSI were markedly elevated in the stroke group (24.40 ± 4.06 kg/m² vs. 23.38 ± 3.71 kg/m²; 0.84 ± 0.06 vs. 0.83 ± 0.06 , respectively). The cohort of stroke patients exhibited a greater proportion of former drinkers and current smokers and a lower proportion of participants engaging in physical activity. Clinical comorbidities, such as heart disease, hypertension, and DM, were more prevalent in the stroke group than in the non-stroke group. Participants in the stroke group had higher levels of CRP, triglycerides, PLT, LDL-C, WBC, and FPG, while HDL-C levels were lower. Notably, both TyG (8.82 ± 0.65 vs. 8.66 ± 0.65 ; $P < 0.001$) and the composite TyG-ABSI index (7.40 ± 0.75 vs. 7.19 ± 0.74 ; $P < 0.001$) were significantly higher in the stroke group.

The incidence rate of stroke

Among the 8,715 participants, 805 stroke events occurred during the follow-up period, yielding an overall incidence rate of 9.24% (95% CI: 8.63–9.85%) or 11.43 per 1,000 person-years (Table 2). The incidence rate of stroke demonstrated a progressive increase across the TyG-ABSI quartiles. Stroke incidence increased monotonically across TyG-ABSI quartiles: from 6.29% (95% CI: 5.27–7.31%; 7.62 per 1,000 person-years) in Q1 to 8.49% (95% CI: 7.32–9.67%; 10.41 per 1,000 person-years) in Q2, 9.64% (95% CI: 8.40–10.88%; 11.90 per 1,000 person-years) in Q3, and 12.53% (95% CI: 11.14–13.92%; 16.08 per 1,000 person-years) in Q4.

Association between TyG-ABSI and stroke

The association between TyG-ABSI and stroke was evaluated through multivariable Cox proportional hazards models, and the results are presented in Table 3. TyG-ABSI levels were strongly linked to an elevated risk of stroke, with an HR of 1.46 (95% CI: 1.33–1.59, $P < 0.0001$) in the unadjusted model (Model 1). After accounting for gender, smoking, age, drinking, and physical activity (Model 2), the association remained significant, with an HR of 1.37 (95% CI: 1.25–1.51, $P < 0.0001$). In the fully adjusted model (Model 3), which further accounted for additional covariates such as Scr, WBC, BMI, heart disease, UA, BUN, CLD, hypertension, HDL-C, DM, liver disease, PLT, LDL-C, CRP, Cystatin C, and hemoglobin, the association persisted, with an HR of 1.22 (95% CI: 1.09–1.37, $P = 0.0004$).

Consistent results were observed in quartile-based analyses. Compared with Q1, the fully adjusted HRs were 1.17 (95% CI: 0.93–1.46; $P = 0.1784$) for Q2, 1.18 (95% CI: 0.94–1.48; $P = 0.1572$) for Q3, and 1.41 (95% CI: 1.11–1.80; $P = 0.0045$) for Q4, with a significant trend across quartiles (P for trend = 0.0063). Together, these findings indicate that TyG-ABSI is independently associated with stroke risk, particularly at higher levels.

Sensitivity analyses

Sensitivity analyses enhanced the stability of the relationship between TyG-ABSI and stroke (Table 4). After excluding participants with hypertension (Model 4; $n = 5,154$), TyG-ABSI remained significantly associated with stroke (HR: 1.38; 95% CI: 1.15–1.65; $P = 0.0005$). Similar associations were observed when excluding participants with diabetes (Model 5; $n = 7,300$; HR: 1.23; 95% CI: 1.08–1.40; $P = 0.0015$) and those with heart disease (Model 6; $n = 7,702$; HR: 1.26; 95% CI: 1.11–1.43; $P = 0.0003$).

The analyses of the non-linear association

The nonlinear relationship between TyG-ABSI and incident stroke was detected after adjusting for age, gender, smoking, drinking, physical activity, BMI, heart disease, CLD, hypertension, DM, liver disease, WBC, PLT, HDL-C, LDL-C, CRP, Scr, UA, BUN, Cystatin C, and hemoglobin by using restricted cubic spline analyses (P for non-linearity < 0.05) (Fig. 2). The solid red line represents the estimated log-transformed relative risk (Log RR), and the green shaded areas indicate the 95%CI. The inflection point was calculated with a recursive algorithm at 6.42. The inflection point was calculated with a recursive algorithm at 6.42. Below this threshold, the HR was 2.44 (95% CI: 1.19–5.02, $P = 0.0151$), while above the threshold, the HR was 1.16 (95% CI: 1.02–1.31, $P = 0.0239$) (Table 5).

Subgroup analysis

The subgroup analysis identified confounders that could influence the association between TyG-ABSI and stroke (Table 6). Stratification was performed according to relevant factors, including age, smoking, BMI, physical activity, gender, and drinking. The results revealed that none of the examined confounders had a significant impact on the observed association between TyG-ABSI and stroke, suggesting the stability of this association.

Discussion

Our longitudinal study revealed that elevated TyG-ABSI levels are linked to stroke in the Chinese population, exhibiting a non-linear association. In addition, subgroup analysis and sensitivity analyses provided additional evidence for this conclusion. These findings provide new insights into the role of TyG-ABSI as a potential predictor

Table 1 The baseline characteristics of participants

Baseline characteristic	Overall	Non-stroke	Stroke	P-value
Participants	8715	7910	805	
Gender				0.438
Female	4671 (53.60%)	4250 (53.73%)	421 (52.30%)	
Male	4044 (46.40%)	3660 (46.27%)	384 (47.70%)	
Age (years)	59.33 ± 9.22	59.14 ± 9.27	61.26 ± 8.42	< 0.001
BMI (kg/m ²)	23.47 ± 3.75	23.38 ± 3.71	24.40 ± 4.06	< 0.001
ABSI	0.83 ± 0.06	0.83 ± 0.06	0.84 ± 0.06	< 0.001
Drinking				0.007
Never drinkers	5146 (59.05%)	4691 (59.30%)	455 (56.52%)	
Former drinkers	714 (8.19%)	625 (7.90%)	89 (11.06%)	
Current drinkers	2855 (32.76%)	2594 (32.79%)	261 (32.42%)	
Smoking				< 0.001
Never smokers	5315 (60.99%)	4859 (61.43%)	456 (56.65%)	
Former smokers	754 (8.65%)	658 (8.32%)	96 (11.93%)	
Current smokers	2646 (30.36%)	2393 (30.25%)	253 (31.43%)	
Physical activity				< 0.001
No	910 (10.44%)	796 (10.06%)	114 (14.16%)	
Yes	7805 (89.56%)	7114 (89.94%)	691 (85.84%)	
Liver disease				0.155
No	8422 (96.64%)	7651 (96.73%)	771 (95.78%)	
Yes	293 (3.36%)	259 (3.27%)	34 (4.22%)	
Heart disease				< 0.001
No	7702 (88.38%)	7055 (89.19%)	647 (80.37%)	
Yes	1013 (11.62%)	855 (10.81%)	158 (19.63%)	
CLD				0.730
No	7868 (90.28%)	7144 (90.32%)	724 (89.94%)	
Yes	847 (9.72%)	766 (9.68%)	81 (10.06%)	
Hypertensive				< 0.001
No	5154 (59.14%)	4837 (61.15%)	317 (39.38%)	
Yes	3561 (40.86%)	3073 (38.85%)	488 (60.62%)	
DM				0.002
No	7300 (83.76%)	6657 (84.16%)	643 (79.88%)	
Yes	1415 (16.24%)	1253 (15.84%)	162 (20.12%)	
WBC (10 ⁹ /L)	6.24 ± 1.88	6.22 ± 1.87	6.42 ± 1.99	0.005
PLT (10 ⁹ /L)	211.99 ± 73.62	211.30 ± 72.82	218.73 ± 80.82	0.006
TC (mg/dL)	193.74 ± 38.23	193.37 ± 38.20	197.33 ± 38.31	0.005
TG (mg/dL)	105.32 (74.34–153.10)	104.43 (74.34–151.34.34.34)	116.82 (84.96–166.38.96.38)	< 0.001
HDL-C (mg/dL)	51.41 ± 15.26	51.66 ± 15.29	48.91 ± 14.75	< 0.001
LDL-C (mg/dL)	116.86 ± 34.94	116.50 ± 34.86	120.34 ± 35.57	0.003
CRP (mg/dL)	1.02 (0.55–2.14)	0.98 (0.54–2.09)	1.33 (0.68–2.64)	< 0.001
Scr (mg/dL)	0.78 ± 0.22	0.78 ± 0.23	0.80 ± 0.19	0.003
UA (mg/dL)	4.45 ± 1.24	4.44 ± 1.24	4.57 ± 1.32	0.005
BUN (mg/dL)	15.74 ± 4.50	15.74 ± 4.51	15.71 ± 4.40	0.872
Cystatin C (mg/L)	1.01 ± 0.27	1.00 ± 0.27	1.03 ± 0.24	0.003
Hemoglobin (g/L)	14.39 ± 2.20	14.36 ± 2.19	14.64 ± 2.28	< 0.001
FPG (mg/dL)	109.52 ± 34.22	109.01 ± 33.62	114.57 ± 39.28	< 0.001
TyG	8.68 ± 0.65	8.66 ± 0.65	8.82 ± 0.65	< 0.001
TyG-ABSI	7.21 ± 0.75	7.19 ± 0.74	7.40 ± 0.75	< 0.001

Values are n (%) or mean ± SD or median (quartile)

BMI: body mass index; ABSI: a body shape index; CLD: chronic lung diseases; DM: diabetes mellitus; WBC: white blood cell; PLT, platelet; TG: triglycerides; LDL-C: low-density lipoprotein cholesterol; TC: total cholesterol; HDL-C: high-density lipoprotein cholesterol; CRP: C-reactive protein; Scr: serum creatinine; UA: uric acid; BUN: blood urea nitrogen; FPG: fasting plasma glucose; TyG, triglyceride-glucose; TyG-ABSI: triglyceride glucose-a body shape index; FPG, fasting plasma glucose

Table 2 Incidence rate of incident stroke based on TyG-ABSI quartiles

TyG-ABSI	Partici- pants (n)	Stroke events (n)	Incidence rate (95% CI) (%)	Per 1,000 person-year
Total	8715	805	9.24 (8.63–9.85)	11.43
Q1	2179	137	6.29 (5.27–7.31)	7.62
Q2	2178	185	8.49 (7.32–9.67)	10.41
Q3	2179	210	9.64 (8.40–10.88,40.88)	11.90
Q4	2179	273	12.53 (11.14–13.92)	16.08

TyG-ABSI: triglyceride glucose-a body shape index; CI: confidence interval

Table 3 Relationship between TyG-ABSI and the incident stroke in different models

Exposure	Model 1 (HR, 95% CI, P)	Model 2 (HR, 95% CI, P)	Model 3 (HR, 95% CI, P)
TyG-ABSI	1.46 (1.33, 1.59) < 0.0001	1.37 (1.25, 1.51) < 0.0001	1.22 (1.09, 1.37) 0.0004
TyG-ABSI (quartile)			
Q1	Ref	Ref	Ref
Q2	1.37 (1.10, 1.71) 0.0051	1.29 (1.03, 1.61) 0.0245	1.17 (0.93, 1.46) 0.1784
Q3	1.57 (1.27, 1.95) < 0.0001	1.44 (1.16, 1.80) 0.0009	1.18 (0.94, 1.48) 0.1572
Q4	2.15 (1.75, 2.64) < 0.0001	1.89 (1.53, 2.34) < 0.0001	1.41 (1.11, 1.80) 0.0045
P for trend	< 0.0001	< 0.0001	0.0063

Model 1: we did not adjust for other covariants

Model 2: we adjusted for age, gender, smoking, drinking, and physical activity

Model 3: we adjusted for age, gender, smoking, drinking, physical activity, BMI, heart disease, CLD, hypertension, DM, liver disease, WBC, PLT, HDL-C, LDL-C, CRP, Scr, UA, BUN, Cystatin C, and hemoglobin

HR: hazard ratios; CI: confidence interval; Ref: reference; TyG-ABSI: triglyceride glucose-a body shape index

of stroke risk and highlight its clinical relevance in stroke prevention strategies.

The TyG index, a marker of IR derived from triglyceride and FPG levels, has been extensively studied in relation to cardiovascular and cerebrovascular diseases [14, 16, 43]. In a cohort study encompassing over 10,000 subjects performed in the United States, an increase of 1 unit in the TyG index was linked to a 32.1% rise in stroke risk after adjusting for potential confounders [44]. Similarly, a study from China reported that each standard deviation increase in the TyG index was linked to a 34% higher risk of stroke, with an adjusted HR of 1.34 (95% CI: 1.11–1.61) [45]. Moreover, the relationship between visceral obesity and increased stroke risk has been well-documented in the literature. ABSI, a commonly used marker of visceral fat content, has been shown to have a significant positive association with stroke incidence in several recent studies [46]. The recently developed TyG-ABSI combines the ABSI and TyG index, providing a more comprehensive measure of metabolic health and body composition

Table 4 Relationship between TyG-ABSI and stroke risk in different sensitivity analyses

Exposure	Model 4 (HR, 95%CI, P)	Model 5 (HR, 95%CI, P)	Model 6 (HR, 95%CI, P)
TyG-ABSI	1.38 (1.15, 1.65) 0.0005	1.23 (1.08, 1.40) 0.0015	1.26 (1.11, 1.43) 0.0003
TyG-ABSI (quartile)			
Q1	Ref	Ref	Ref
Q2	1.30 (0.94, 1.81) 0.1172	1.16 (0.91, 1.46) 0.2277	1.19 (0.93, 1.53) 0.1737
Q3	1.43 (1.02, 2.01) 0.0378	1.14 (0.90, 1.45) 0.2846	1.27 (0.99, 1.64) 0.0620
Q4	1.81 (1.26, 2.60) 0.0013	1.43 (1.10, 1.85) 0.0071	1.50 (1.15, 1.97) 0.0027
P for trend	0.0015	0.0129	0.0028

Model 4 was a sensitivity analysis in participants without hypertension (n=5154). We adjusted for age, gender, smoking, drinking, physical activity, BMI, heart disease, CLD, DM, liver disease, WBC, PLT, HDL-C, LDL-C, CRP, Scr, UA, BUN, Cystatin C, and hemoglobin

Model 5 was a sensitivity analysis in participants without diabetes (n = 7,300). We adjusted for age, gender, smoking, drinking, physical activity, BMI, heart disease, CLD, hypertension, liver disease, WBC, PLT, HDL-C, LDL-C, CRP, Scr, UA, BUN, Cystatin C, and hemoglobin

Model 6 was a sensitivity analysis in participants without heart disease (n=7702). We adjusted for age, gender, smoking, drinking, physical activity, BMI, CLD, hypertension, DM, liver disease, WBC, PLT, HDL-C, LDL-C, CRP, Scr, UA, BUN, Cystatin C, and hemoglobin

HR: hazard ratios; CI: confidence, Ref: reference; TyG-ABSI: triglyceride glucose-a body shape index

[40]. This index has been closely associated with cardiovascular mortality [40]. Notably, the TyG-ABSI has been shown to exceed the TyG index, both the conventional TyG index and other modifications in the prediction of cardiovascular mortality risk [40]. Given the strong associations between TyG, ABSI, and stroke risk, it was hypothesized that TyG-ABSI would also exhibit a positive association with stroke risk. Our analysis confirmed this hypothesis, demonstrating a significant association between TyG-ABSI and incident stroke. This study is the first to examine this relationship in a Chinese population. By analyzing TyG-ABSI as both a categorical and continuous variable, we enhanced the precision of the relationship quantification and minimized potential information loss. Furthermore, sensitivity analyses conducted on participants without heart disease, DM, or hypertension validated the robustness of the observed associations. In additional analyses, TyG-ABSI demonstrated slightly superior discrimination for incident stroke compared with established indices (AUROC: TyG-ABSI 0.5805 vs. TyG index 0.5714, BMI 0.5787, ABSI 0.5467; Table S1). Although the absolute AUROC values are modest—as expected for single-timepoint, non-imaging risk markers—the comparative advantage supports TyG-ABSI as a pragmatic screening metric.

While the precise mechanisms linking TyG-ABSI to stroke risk remain unclear, the relationship may

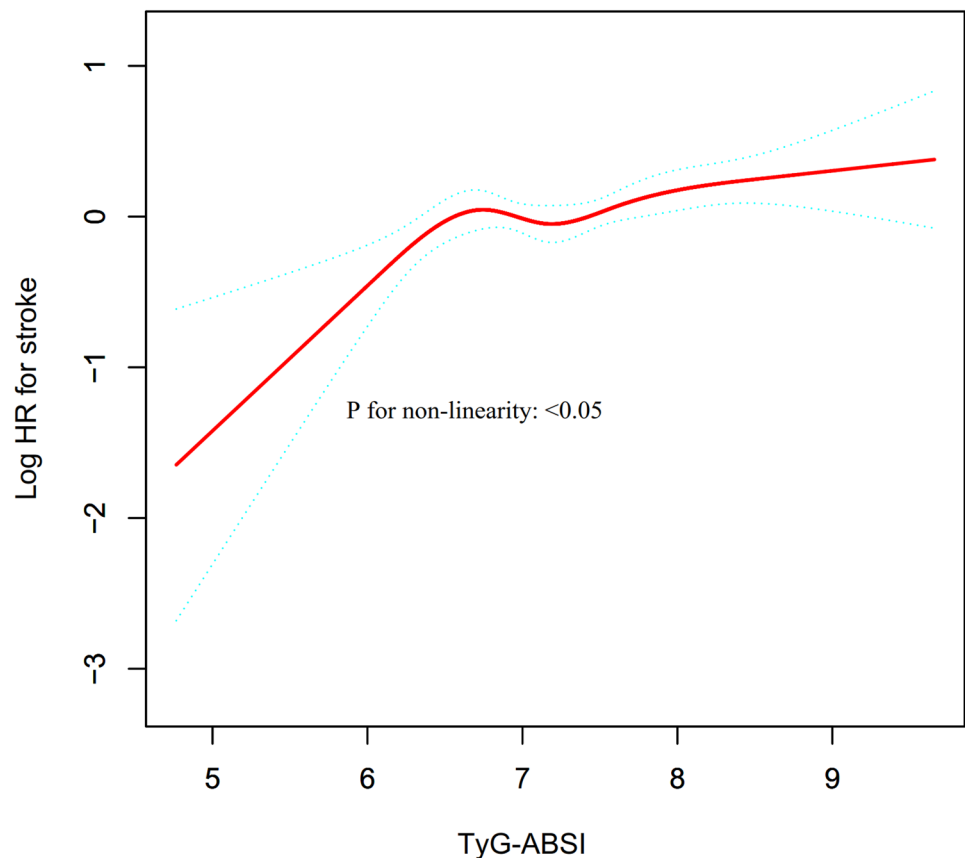


Fig. 2 The non-linear relationship between TyG-ABSI and incident stroke. Restricted cubic spline models showing the relationship between TyG-ABSI and log-transformed relative risk (Log RR) for adverse outcomes. The solid red line represents the estimated Log RR, and the green shaded areas indicate the 95% confidence intervals. The nonlinear relationship between TyG-ABSI and incident stroke was detected after adjusting for age, gender, smoking, drinking, physical activity, BMI, heart disease, CLD, hypertension, DM, liver disease, WBC, PLT, HDL-C, LDL-C, CRP, Scr, UA, BUN, Cystatin C, and hemoglobin by using restricted cubic spline analyses (P for non-linearity <0.05). The inflection point was calculated with a recursive algorithm at 6.42

Table 5 The result of the two-piecewise Cox proportional hazards regression model

Incident stroke	HR (95% CI)	P
Fitting model by standard linear regression	1.22 (1.09, 1.37)	0.0004
Fitting model by two-piecewise Cox proportional hazards		
The inflection point of the TyG-ABSI	6.42	
≤ 6.42	2.44 (1.19, 5.02)	0.0151
> 6.42	1.16 (1.02, 1.31)	0.0239
P for the log-likelihood ratio test	0.039	

We adjusted for age, gender, smoking, drinking, physical activity, BMI, heart disease, CLD, hypertension, DM, liver disease, WBC, PLT, HDL-C, LDL-C, CRP, Scr, UA, BUN, Cystatin C, and hemoglobin

HR: hazard ratios; CI: confidence; TyG-ABSI: triglyceride glucose-a body shape index

primarily be attributed to its strong connection with IR. TyG-ABSI integrates FPG, triglycerides, and ABSI, all of which are well-established indicators of IR. FPG reflects hepatic insulin sensitivity and pancreatic insulin production [47], while triglycerides and ABSI have been validated as reliable markers for identifying IR in previous studies [48–50]. IR plays a pivotal role in the development of atherosclerosis by promoting foam cell

formation, endothelial dysfunction, and the destabilization of atherosclerotic plaques [51, 52]. Additionally, IR is often associated with chronic low-grade inflammation, which can accelerate the progression of atherosclerosis [53]. Insulin resistance may also influence platelet function, enhancing platelet activation, aggregation, and adhesion. These changes can lead to arterial constriction or obstruction, thereby increasing the risk of stroke [54, 55]. In addition, the constituent elements of TyG-ABSI—triglycerides, FPG, and ABSI—may independently contribute to elevated stroke risk. Elevated triglycerides and hyperglycemia are associated with endothelial dysfunction through increased oxidative stress and the accumulation of advanced glycation end-products, thereby accelerating atherogenesis and plaque progression [56–58]. The adiposity distribution reflected by ABSI correlates with visceral adipose tissue activity and the secretion of pro-inflammatory cytokines (e.g., IL-6, TNF-α), which may destabilize atherosclerotic plaques and heighten cerebrovascular vulnerability [59–61]. Hypertriglyceridemia and insulin resistance are linked

Table 6 Effect size of TyG-ABSI on stroke in prespecified and exploratory subgroups

Characteristic	No of participants	HR (95%CI) <i>P</i> value	<i>P</i> for interaction
Age (years)			0.0894
< 60	4765	1.36 (1.16, 1.58) < 0.0001	
≥ 60	3950	1.15 (1.00, 1.32) 0.0440	
Gender			0.8991
Female	4671	1.21 (1.05, 1.40) 0.0085	
Male	4044	1.23 (1.05, 1.44) 0.0098	
BMI (kg/m ²)			0.6142
< 25	6038	1.18 (1.03, 1.35) 0.0177	
≥ 25	2677	1.24 (1.04, 1.48) 0.0143	
Smoking			0.9507
Never smokers	5315	1.23 (1.07, 1.42) 0.0046	
Former smokers	754	1.17 (0.86, 1.59) 0.3300	
Current smokers	2646	1.21 (1.00, 1.47) 0.0497	
Drinking			0.5699
Never drinkers	5146	1.28 (1.10, 1.48) 0.0010	
Former smokers	714	1.04 (0.73, 1.49) 0.8246	
Current drinkers	2855	1.23 (1.02, 1.48) 0.0328	
Physical activity			0.8305
No	910	1.19 (0.92, 1.55) 0.1895	
Yes	7805	1.23 (1.09, 1.39) 0.0008	

Note 1: The above model was adjusted for age, gender, smoking, drinking, physical activity, BMI, heart disease, CLD, hypertension, DM, liver disease, WBC, PLT, HDL-C, LDL-C, CRP, Scr, UA, BUN, Cystatin C, and hemoglobin

Note 2: The model is not adjusted for the stratification variable in each case

to enhanced platelet activation and upregulated coagulation pathways (e.g., increased PAI-1), promoting thrombus formation and, consequently, increasing ischemic stroke risk [62]. Broader metabolic dysregulation may also attenuate endothelial progenitor cell function and impair fibrinolysis, exacerbating ischemic injury following vascular occlusion [58]. Finally, ABSI—as a marker of central body shape—is associated with greater arterial stiffness and higher pulse pressure, both of which are independent determinants of cerebrovascular events [63]. Thus, the observed association between TyG-ABSI and stroke risk may be attributed to the combined effects of triglycerides, ABSI, and FPG on IR and its downstream consequences.

Our study identified a nonlinear association between TyG-ABSI and stroke, with an inflection point at a TyG-ABSI value of 6.42. Below this threshold, each 1-unit rise in TyG-ABSI corresponded to a 144% increase in the risk of stroke. In contrast, above the threshold, each standard deviation increase corresponded to a more modest 16% increase in stroke risk. The findings highlight the critical need for monitoring and, when necessary, reducing TyG-ABSI levels in clinical practice. Furthermore, the results provide valuable insights to support healthcare providers in effectively managing patients' TyG-ABSI levels to mitigate stroke risk.

The strengths of this investigation lie in the use of multiple sensitivity analyses, its substantial sample size, and thorough adjustment for various potential confounders to enhance the robustness of the relationship between TyG-ABSI and stroke. Furthermore, the identification of a nonlinear association between TyG-ABSI and stroke offers novel insights into the complex interplay between metabolic health and cerebrovascular outcomes.

Nonetheless, it is important to recognize several limitations inherent in the study. First, the observational study restricts our ability to infer causal association. Second, TyG-ABSI was calculated based on a single measurement of triglycerides, fasting glucose, and anthropometric parameters, which may not adequately reflect long-term metabolic changes. Third, residual confounding by unmeasured factors—such as family history of stroke, inflammatory biomarkers, and dietary habits—might have influenced the association between TyG-ABSI and stroke risk. In future studies, we will collect comprehensive data encompassing family history of stroke, inflammatory biomarkers, dietary habits, and longitudinal TyG-ABSI measurements to provide a more robust evaluation of the TyG-ABSI-stroke relationship. Fourth, stroke diagnosis in our study relies on self-reporting, which may introduce several potential biases: (1) Recall bias, particularly for minor strokes or transient ischemic attacks, (2) Misclassification bias due to confusion with other neurological conditions, and (3) Underreporting of asymptomatic strokes. Our data came from <http://www.issp.pku.edu.cn/cfps/>, and there was no information on stroke-related symptoms or imaging findings. We were therefore unable to systematically exclude TIAs or verify events using clinical documentation. Notwithstanding these constraints, prior validation studies indicate that self-reported stroke demonstrates reasonably high accuracy, with sensitivity ranging from 78% to 96% and specificity from 96% to 98% [64–66]. This evidence suggests that self-reported stroke is an acceptable proxy for event ascertainment in large-scale population surveys, although some degree of non-differential misclassification may persist. Consequently, effect estimates in our analysis may be biased toward the null, and residual

misclassification cannot be entirely excluded. To enhance diagnostic precision in future work, we plan to integrate self-report with clinical symptom inventories and neuroimaging confirmation (e.g., CT/MRI) and, where feasible, linkage to hospital discharge records or disease registries. Such enhancements should reduce misclassification, improve the validity of stroke ascertainment, and enable more nuanced analyses of the association between TyG-ABSI and stroke subtypes. Finally, as our research included only Chinese subjects aged ≥ 45 years, further validation is required in different age groups and diverse ethnic groups to enhance the generalizability of our findings.

Conclusion

Our study demonstrates that TyG-ABSI is significantly linked to incident stroke, with a nonlinear relationship observed between them. These findings suggest that TyG-ABSI may be a novel and comprehensive marker for stroke risk stratification, integrating metabolic and anthropometric factors.

Abbreviations

IR	Insulin resistance
HOMA-IR	Homeostatic assessment of insulin resistance
ABSI	Body shape index
TyG index	Triglyceride-glucose index
TyG-ABSI	Triglyceride glucose-a body shape index
CHARLS	China Health and Retirement Longitudinal Study
BMI	Body mass index
FPG	Fasting plasma glucose
TC	Total cholesterol
TG	Triglyceride
HDL-C	High-density lipoprotein cholesterol
LDL-C	Low-density lipid cholesterol
CRP	C-reactive protein
Scr	serum creatinine
BUN	Blood urea nitrogen
UA	Uric acid
CLD	Chronic lung disease
DM	Diabetes mellitus
HR	Hazard ratios
CI	Confidence intervals
Ref	Reference

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

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Author contributions

Dongpo He, Xintao Zhang, and Changchun Cao contributed to the study concept and design, researched and interpreted the data, and drafted the manuscript. Yong Han, Haofei Hu, and Xiaodan Zheng analyzed the data,

reviewed the manuscript, oversaw the project's progress, contributed to the discussion, and reviewed the manuscript. Zhenzhen Fan provided invaluable assistance in addressing the reviewers' comments and made significant contributions to enhancing the language quality, grammar, and syntax of the manuscript. All writers reviewed and approved the manuscript.

Data availability

Data are available from <http://www.issp.pku.edu.cn/cfps/>. Follow the prompts to register as a user and download the data once it has been reviewed and approved. The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Each participant in the CHARLS cohort conducted under Peking University's ethics review committee gave their informed permission before participating (IRB00001052-11015). In addition, the Declaration of Helsinki was followed during our research. The necessary standards and legislation were followed in executing all procedures, including the declarations in the Declarations section.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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