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The relationship between the triglyceride–glucose index and functional outcomes in patients with aneurysmal subarachnoid hemorrhage: a retrospective cohort study

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Abstract

Background Aneurysmal subarachnoid hemorrhage (aSAH) is a life-threatening disease with high morbidity and mortality. The triglyceride–glucose (TyG) index, a marker of insulin resistance (IR), has been linked to adverse outcomes in cerebrovascular conditions; however, its influence on functional prognosis in aSAH remains unclear. This study aimed to elucidate the relationship between the TyG index and functional outcomes in aSAH patients.

Methods A retrospective cohort study included consecutive aSAH patients. Functional outcomes were assessed using the modified Rankin Scale (mRS) at 3 months and categorized as favorable (mRS 0–2) or unfavorable (mRS 3–6). Univariate and multivariate logistic regression analyzed the association between the TyG index and functional outcomes. Propensity score matching (PSM) was used to mitigate confounding. Non-linear relationships were explored with restricted cubic splines (RCS), and subgroup analyses were performed. A nomogram integrating the TyG index and traditional prognostic scales was developed, and model predictive performance was compared using the area under the curve (AUC) on a test set.

Results A total of 470 patients (61.7% female) were enrolled, with 154 experiencing unfavorable outcomes. Multivariate logistic regression showed a significant association between the TyG index and adverse outcomes (OR: 1.86, 95% CI 1.12–3.1, $P=0.017$). An optimal TyG index cutoff of 8.83 was identified. Patients with TyG index ≥ 8.83 had a higher risk of poor outcomes (48.7% vs. 24.8%; $P=0.015$). PSM confirmed these findings. RCS indicated a progressive association between elevated TyG index and increased risk of adverse functional outcome. Subgroup analyses showed consistent relationships. The enhanced model with the TyG index had a higher AUC (0.899) than the traditional model (0.889, DeLong test $P=0.048$).

Conclusions A high TyG index is significantly associated with an increased risk of unfavorable functional outcomes in patients with aSAH.

Keywords Triglyceride–glucose index, Aneurysmal subarachnoid hemorrhage, Functional outcomes

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Introduction

Spontaneous subarachnoid hemorrhage (SAH) constitutes the third most frequent subtype of stroke, a cerebrovascular disorder that can have a devastating impact on affected individuals [1]. A considerable number of SAH cases are attributable to the rupture of intracranial aneurysm (IA), a condition known as aneurysmal subarachnoid hemorrhage (aSAH) [2]. Patients suffering from aSAH exhibit a broad spectrum of prognostic outcomes. These outcomes encompass instances of full recovery, alongside cases of severe disability or even mortality. A noteworthy segment of survivors, amounting to at least 20% of the total, fails to achieve functional independence [3]. The prognosis of aSAH is influenced by several well-established factors, including clinical grading scales, such as the Hunt–Hess grade and the World Federation of Neurosurgical Societies (WFNS) classification, which assess the severity of neurological impairment upon admission [4, 5]. Radiological assessments, notably the modified Fisher (mFisher) scale, evaluate the extent of SAH and the presence of intraventricular hemorrhage (IVH), both correlated with vasospasm risk and overall outcomes [6, 7]. In addition, aneurysm characteristics, such as location and size, have been associated with patient prognosis. Despite the utility of these factors, they may not fully capture the complexity of individual patient outcomes, the utilization of additional biomarkers may enhance the overall prognostic accuracy in aSAH patients. This might be achieved by the identification of high-risk individuals and the subsequent guidance of appropriate treatment interventions. Current biomarkers employed in aSAH prognosis, such as neuron-specific enolase (NSE), glial fibrillary acidic protein (GFAP), and early S100 calcium binding protein B (S100B), present several challenges [8–11]. These include analytical complexities, temporal variability, and the need for invasive sampling methods. For instance, measuring NSE and GFAP requires specialized immunoassays with notable inter-laboratory variability, which can limit reproducibility. In addition, biomarkers, such as S100B often necessitate cerebrospinal fluid sampling, which may not be feasible in all patients. These factors underscore the need for additional biomarkers that are reliable, easily implementable, and minimally invasive to enhance prognostic accuracy in aSAH patients.

Insulin resistance (IR) is a metabolic disorder characterized by impaired tissue responsiveness to insulin stimulation, which ultimately leads to dysfunction in both glucose and blood lipid metabolism. Published literature has demonstrated the correlation between IR and a diverse array of vascular diseases, including atherosclerosis, stroke, and coronary artery disease [12–14]. Prior investigations have substantiated a correlation between the TyG index and the prognosis of individuals with arterial stiffness, ischemic stroke and coronary artery disease [15–17]. Nevertheless, there has been a notable dearth of research examining the relationship between the TyG index and prognosis in patients with hemorrhagic stroke, with particular emphasis on aSAH. Existing investigations examining the prognostic value of the TyG index in SAH patients exhibit some limitations. A study utilizing the Medical Information Mart for Intensive Care (MIMIC-IV) database failed to distinguish between SAH and intracerebral hemorrhage (ICH), thereby conflating two etiologically distinct stroke subtypes while omitting essential clinical prognosticators including Hunt–Hess grade and World Federation of Neurosurgical Societies (WFNS) classification [18]. Furthermore, a single-center retrospective study, though focused specifically on SAH, demonstrated inadequate statistical power due to restricted sample size and lacked critical neuroimaging parameters, such as aneurysm localization and modified Fisher scale (mFisher) scores [19]. These deficiencies collectively compromise the generalizability and clinical applicability of prior findings. In this study, we address these gaps by implementing several methodological advancements. First, we focus exclusively on aSAH by including only radiologically confirmed cases. Second, we incorporate a more comprehensive set of prognostic variables, including Hunt–Hess grade, WFNS classification, mFisher scores, and aneurysm location, to better capture the factors influencing patient outcomes. Third, we employ a larger cohort compared to partial previous studies, thereby enhancing the statistical power of our analysis. These methodological refinements collectively enable a more robust evaluation of the prognostic utility of the TyG index in predicting functional outcomes in the aSAH population.

In this study, we hypothesize that an elevated TyG index is independently associated with poorer functional outcomes at 90 days following aSAH, even after adjusting for established prognostic variables, such as the Hunt–Hess grade, WFNS grade, mFisher scores, and aneurysm location. This hypothesis underscores the potential of the TyG index to serve as an incremental prognostic marker, thereby providing additional insights for risk stratification and informing targeted clinical management strategies.

Materials and methods

Study population

This is a single-center, retrospective, observational cohort study based on all consecutive patients received treatment for aSAH from January 2018 to October 2023 at the Department of Neurosurgery, Tongji Hospital, Tongji Medical College, Huazhong University of

Science and Technology. The inclusion criteria were defined as follows: (1) SAH demonstrated by computed tomography (CT) or lumbar puncture; (2) specific diagnosis of IA confirmed by computed tomography angiography (CTA), digital subtraction angiography (DSA), or surgery; (3) subjects with aSAH treated with surgical clipping or endovascular coiling within 72 h; (4) age of patients >18 years; and (5) adequate laboratory parameters could be obtained prior to treatment. Participants were excluded if they had any of the following conditions: (1) pregnant or perinatal patients; (2) in the presence of traumatic brain injury or other cerebrovascular disease, such as arteriovenous malformation, dural arteriovenous fistula, inability to confirm the relationship between SAH and IA; (3) patient loss to follow-up within 3 months; (4) patients with hepatic or renal disease, malignancy, mental disorders, and severe heart or respiratory failure; and (5) pre-treatment triglyceride and glucose data were not available.

Data collection and definitions

Trained clinicians retrieved data from the electronic medical record system, including medical history, diagnoses, resident admission notes, discharge notes, and laboratory and imaging examination. Four main aspects of information were collected: (1) demographics including age, sex, current smoking and alcohol consumption status; (2) comorbidities existing including hypertension, diabetes, hyperlipidemia, ICH, IVH, pneumonia, and hydrocephalus; (3) clinical indicators, including WFNS grade, Glasgow Coma Scale (GCS) score [20], Hunt–Hess grade, mFisher score [21], and surgical methods; and (4) hematological and biochemical parameters, such as fasting blood glucose, triglycerides, serum creatinine, blood urea nitrogen, hemoglobin, blood cell counts, and coagulation function. All blood biochemical parameters were obtained within 24 h of admission to ensure consistency and accuracy in reflecting the patients'baseline biochemical status.

The TyG index was calculated as Ln [fasting triglyceride (mg/dL) × fasting glucose (mg/dL)/2]. All patients were re-evaluated by qualified neurosurgeons on admission and at 3 months following discharge. Neurological prognosis was assessed via the mRS score [22], measured by a semi-structured telephone interview or outpatient visit. Based on the mRS score, patients were categorized into two groups: favorable outcome (mRS score of 0–2) and unfavorable outcome (mRS score of 3–6).

We excluded patients for whom pre-treatment triglyceride and glucose data were unavailable, as these parameters are essential for calculating the TyG index. We confirmed that no other critical data were missing for the remaining patients. The patient selection flowchart (Fig. 1) illustrates the exclusion process, ensuring transparency in our methodology.

Statistical analysis

The subjects were stratified into quartiles based on their TyG index values (Q1–Q4) for the purposes of this study. Continuous variables were presented as mean ± standard deviation (SD) or median and interquartile range (IQR), as appropriate to their distribution. In accordance with the normality of the continuous variable distributions, either the independent samples *t* test or Mann–Whitney *U* test was employed. Categorical variables were expressed as frequency with percentage and analyzed by the Chi-square test or Fisher exact test, as appropriate. The optimal cutoff value for predicting unfavorable

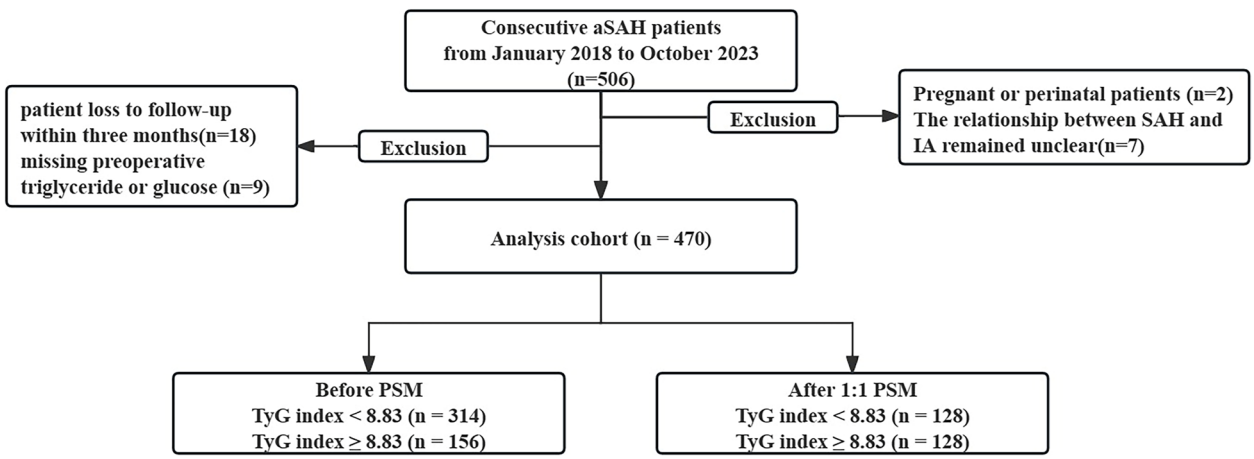

Fig. 1 Flowchart of participant selection. aSAH: aneurysmal subarachnoid hemorrhage; SAH: subarachnoid hemorrhage; IA: intracranial aneurysm; PSM: propensity score matching; TyG: triglyceride–glucose

outcomes following aSAH was determined through the application of the maximization of Youden's index, calculated via receiver operating characteristic (ROC) analysis. Based on the results of the optimal cutoff value of TyG index, patients were divided into two groups, high TyG index (≥ 8.83) and low TyG index (< 8.83). In additional, given the imbalance in baseline characteristics between groups, we utilized 1:1 propensity score matching (PSM) to create a balanced cohort of patients with high and low TyG index. This was achieved using the nearest neighbor matching method, with a caliper width of 0.20, to ensure that key variables were evenly distributed between the groups. Univariate and multivariate logistic regression models were constructed using the stepwise method to investigate the association between the TyG index and functional outcome among aSAH patients. To control for the effects of potential confounding factors, multivariable logistic regression involved multiple different models. In the crude model, no variates were adjusted. Model 1 adjusted for demographic parameters including age, sex, current smoking, and alcohol consumption status. Apart from demographic parameters, we adjusted for comorbidities and complications in model 2, which included hypertension, diabetes mellitus, hyperlipidemia, ICH, IVH, pneumonia, and hydrocephalus. Model 3, which was built on the foundation of Model 2, was further adjusted for factors that were related to the severity of aSAH, including GCS score, Hunt–Hess grade, WFNS grade, and mFisher score. We selected factors as confounders follow the following principles: (1) a factor had a change in the effect estimate of more than 10% and (2) a factor was significantly associated with the outcomes of interest.

The associations between levels of TyG index and functional outcomes were evaluated on a continuous scale with restricted cubic spline (RCS) curves based on logistic regression models. In an attempt to demonstrate the consistency of the results, a subgroup analysis was conducted. Subgroup analyses were conducted via logistic regression models defined by age (< 65 vs. ≥65 years), gender, hypertension, presence of ICH, presence of IVH, Hunt–Hess grade (< 4 vs. ≥4), and mFisher score (< 3 vs. ≥3). We developed a nomogram integrating the TyG index with traditional prognostic scales (WFNS, Hunt–Hess, and mFisher) to predict poor functional outcomes in aSAH patients. The data set was randomly divided into a training set (70%) and a testing set (30%) using the createDataPartition function from the caret package in R, ensuring a balanced distribution of outcomes in both sets. The nomogram was constructed using logistic regression analysis on the training set and visualized using the rms package in R. We compared the predictive performance of a traditional model (excluding the TyG index) and a TyG-enhanced model (including the TyG index) by evaluating the area under the curve (AUC) on the testing set. The DeLong test was used to assess the statistical significance of differences in AUC values between the two models. Statistical analyses were performed using R software version 4.3.2 (http://www.r-project.org). $P < 0.05$ was considered statistically significant.

Results

Patient characteristics

Following the application of a series of inclusion and exclusion criteria (Fig. 1), a cohort of 470 patients with aSAH was identified for further analysis in this study. The participants, with a mean age of 56.0 ± 9.6 years, comprised 61.7% women, and the mean TyG index among them was 8.6 (IQR: 8.1–9.0). As illustrated in Table 1, the baseline characteristics of the enrolled patients were presented in quartiles of the TyG index (quartile Q1: 6.40–8.13; Q2: 8.13–8.56; Q3: 8.56–9.02; Q4: 9.02–11.23). The median value of the TyG index for each quartile was found to be 8.26 (IQR: 8.09–8.41), 8.74 (IQR: 8.64–8.82), 9.09 (IQR: 8.99–9.21), and 9.76 (IQR: 9.53–10.03), respectively.

No statistically significant differences were found in age, gender, smoking status, or alcohol consumption between participants with higher TyG index and those in the lowest quartile. However, participants in the higher TyG index quartile had a significantly higher prevalence of comorbidities, such as hyperlipidemia, diabetes mellitus, IVH, pneumonia, and exhibited a worse clinical status compared to their counterparts in the lowest quartile. Furthermore, significant differences in laboratory parameters were observed between the groups. Specifically, individuals in the highest quartile showed markedly higher levels of glucose, triglycerides, serum creatinine, hemoglobin, and various blood cell counts, including erythrocytes, leukocytes, neutrophils, lymphocytes, and monocytes, as compared to those in the lowest quartile.

Association between TyG index and functional outcome post-aSAH

According to the results of the ROC analysis, TyG index of 8.83 was identified as the optimal cutoff value for predicting functional outcomes post-aSAH. To account for potential confounders and selection bias, 1:1 PSM was implemented to reinforce the reliability of the results. The propensity score distributions of aSAH patients before and after PSM are shown in Fig. 2. PSM yielded 128 well-matched pairs between the group with low TyG index (< 8.83) and the group with high TyG index (≥ 8.83), demonstrating a good balance in key parameters and making them suitable for further analysis. Table 2 demonstrates

Table 1 Characteristics and outcomes of participants according to the TyG index quartiles

Characteristics	Quartiles of TyG index					*P* value
	Overall	Q1 (6.40–8.13)	Q2 (8.13–8.56)	Q3 (8.56–9.02)	Q4 (9.02–11.23)	
N (%)	470	119 (25.3)	115 (24.5)	118 (25.1)	118 (25.1)	
Demographics						
Age, years, mean (SD)	56.0 ± 9.6	56.2 ± 9.7	56.5 ± 10.0	55.0 ± 8.2	56.3 ± 10.3	0.601
Gender, female, (%)	290 (61.7)	77 (64.7)	72 (62.6)	69 (58.5)	72 (61)	0.792
Smoking, *n* (%)	123 (26.2)	30 (25.2)	30 (26.1)	37 (31.4)	26 (22)	0.433
Alcohol, *n* (%)	141 (30.0)	30 (25.2)	35 (30.4)	38 (32.2)	38 (32.2)	0.603
Comorbidities						
Hypertension, *n* (%)	227 (48.3)	51 (42.9)	52 (45.2)	61 (51.7)	63 (53.4)	0.306
Hyperlipidemia, *n* (%)	85 (18.1)	11 (9.2)	10 (8.7)	17 (14.4)	47 (39.8)	< 0.001
Diabetes mellitus, *n* (%)	38 (8.1)	4 (3.4)	3 (2.6)	9 (7.6)	22 (18.6)	< 0.001
Presence of ICH, *n* (%)	109 (23.2)	24 (20.2)	23 (20.0)	26 (22.0)	36 (30.5)	0.179
Presence of IVH, *n* (%)	136 (28.9)	28 (23.5)	34 (29.6)	28 (23.7)	46 (39.0)	0.028
Hydrocephalus, *n* (%)	72 (15.3)	18 (15.1)	14 (12.2)	14 (11.9)	26 (22.0)	0.116
Pneumonia, *n* (%)	112 (23.8)	18 (15.1)	26 (22.6)	23 (19.5)	45 (38.1)	< 0.001
Clinical data						
GCS score, median (IQR)	15.0 (13.0–15.0)	15.0 (14.0–15.0)	15.0 (13.0–15.0)	15.0 (13.0–15.0)	14.0 (8.0–15.0)	< 0.001
WFNS grade, median (IQR)	1.0 (1.0–3.0)	1.0 (1.0–2.0)	1.0 (1.0–2.0)	1.0 (1.0–2.0)	2.0 (1.0–4.0)	< 0.001
Hunt–Hess grade, median (IQR)	2.0 (1.0–3.0)	1.0 (1.0–3.0)	1.0 (1.0–3.0)	1.0 (1.0–3.0)	3.0 (1.0–4.0)	< 0.001
mFisher score, median (IQR)	1.0 (1.0–3.0)	1.0 (1.0–3.0)	1.0 (1.0–2.0)	1.0 (1.0–3.0)	2.0 (1.0–3.0)	0.011
Treatment						0.033
Coiling, *n* (%)	171 (36.4)	44 (37)	54 (47)	38 (32.2)	35 (29.7)	
Clipping, *n* (%)	299 (63.6)	75 (63)	61 (53)	80 (67.8)	83 (70.3)	
Aneurysm locations						0.439
Middle cerebral artery, *n* (%)	103 (21.9)	26 (21.8)	24 (20.9)	28 (23.7)	25 (21.2)	
Anterior cerebral artery, *n* (%)	24 (5.1)	6 (5)	7 (6.1)	8 (6.8)	3 (2.5)	
AcomA, *n* (%)	128 (27.2)	30 (25.2)	34 (29.6)	25 (21.2)	39 (33.1)	
PcomA, *n* (%)	148 (31.5)	42 (35.3)	35 (30.4)	36 (30.5)	35 (29.7)	
Internal carotid artery, *n* (%)	33 (7.0)	7 (5.9)	8 (7)	14 (11.9)	4 (3.4)	
Posterior circulation, *n* (%)	34 (7.2)	8 (6.7)	7 (6.1)	7 (5.9)	12 (10.2)	
Laboratory data, median (IQR)						
Glucose, mg/dL	127.5 (108.4–154.2)	117.1 (102.4–136.4)	120.2 (104.2–142.9)	129.4 (108.3–157.2)	151.7 (126.7–185.0)	< 0.001
Triglyceride, mg/dL	80.6 (56.7–123.8)	41.6 (30.1–52.3)	70.0 (58.5–82.8)	100.1 (82.4–115.8)	160.4 (128.5–205.3)	< 0.001
TyG index	8.6 (8.1–9.0)	7.8 (7.6–8.0)	8.4 (8.2–8.5)	8.8 (8.7–8.9)	9.3 (9.1–9.7)	< 0.001
Blood urea nitrogen, mmol/L	4.3 (3.5–5.3)	4.1 (3.5–5.3)	4.3 (3.5–5.1)	4.3 (3.5–5.5)	4.7 (3.6–5.4)	0.412
Serum creatinine μmol/L/	58.0 (47.0–69.0)	51.0 (37.0–63.5)	56.0 (48.5–66.0)	62.0 (52.0–73.8)	63.5 (51.2–74.8)	< 0.001
Platelet count, $\times 10^9$/L	202.0 (161.0–243.0)	200.0 (156.5–234.5)	198.0 (154.0–228.0)	216.0 (170.0–255.2)	198.5 (163.0–251.2)	0.149
Hemoglobin, g/L	129.0 (118.2–140.0)	124.0 (113.0–133.0)	130.0 (120.0–139.5)	132.0 (121.2–141.0)	133.5 (122.0–143.0)	< 0.001
Erythrocyte count, $\times 10^9$/L	4.3 (3.9–4.6)	4.1 (3.8–4.5)	4.2 (3.9–4.6)	4.4 (4.0–4.7)	4.3 (4.0–4.7)	< 0.001
Leukocyte count, $\times 10^9$/L	10.9 (8.3–13.9)	9.8 (7.7–12.6)	10.0 (8.0–12.6)	11.0 (8.6–13.9)	12.8 (9.1–17.7)	< 0.001
Neutrophil count, $\times 10^9$/L	9.2 (6.6–12.3)	8.5 (6.4–11.3)	8.5 (6.4–11.3)	9.0 (6.8–12.2)	10.9 (7.4–15.5)	< 0.001
Lymphocyte count, $\times 10^9$/L	0.9 (0.7–1.3)	0.8 (0.6–1.1)	0.9 (0.7–1.2)	1.0 (0.7–1.4)	1.0 (0.7–1.3)	0.005
Monocyte count, $\times 10^9$/L	0.5 (0.4–0.7)	0.4 (0.3–0.6)	0.5 (0.3–0.7)	0.5 (0.4–0.8)	0.6 (0.4–0.9)	< 0.001
PT, sec	13.3 (12.9–13.8)	13.5 (13.1–14.0)	13.2 (12.9–13.9)	13.3 (12.9–13.8)	13.2 (12.7–13.7)	0.009
APTT, sec	33.2 (31.2–35.7)	33.4 (31.4–36.2)	33.4 (31.9–35.7)	32.9 (31.2–35.5)	32.7 (30.9–35.2)	0.247
TT, sec	16.4 (15.8–17.2)	16.4 (15.6–17.1)	16.4 (15.8–17.2)	16.4 (15.7–17.2)	16.4 (15.9–17.3)	0.833
FBG, g/L	3.1 (2.7–3.6)	3.0 (2.7–3.5)	3.1 (2.8–3.6)	3.1 (2.6–3.5)	3.2 (2.8–3.8)	0.081
Clinical outcomes						
3-month mRS score, *n* (%)						0.008

Table 1 (continued)

Characteristics	Quartiles of TyG index					*P* value
	Overall	Q1 (6.40–8.13)	Q2 (8.13–8.56)	Q3 (8.56–9.02)	Q4 (9.02–11.23)	
0	186 (39.6)	52 (43.7)	53 (46.1)	50 (42.4)	31 (26.3)	
1	85 (18.1)	27 (22.7)	17 (14.8)	21 (17.8)	20 (16.9)	
2	45 (9.6)	12 (10.1)	14 (12.2)	12 (10.2)	7 (5.9)	
3	35 (7.4)	6 (5.0)	5 (4.3)	10 (8.5)	14 (11.9)	
4	34 (7.2)	9 (7.6)	8 (7.0)	9 (7.6)	8 (6.8)	
5	62 (13.2)	9 (7.6)	14 (12.2)	11 (9.3)	28 (23.7)	
6	23 (4.9)	4 (3.4)	4 (3.5)	5 (4.2)	10 (8.5)	
Functional outcome, *n* (%)						< 0.001
Favorable	316 (67.2)	91 (76.5)	84 (73)	83 (70.3)	58 (49.2)	
Unfavorable	154 (32.8)	28 (23.5)	31 (27)	35 (29.7)	60 (50.8)	

TyG: triglyceride–glucose; SD: standard deviation; IQR: interquartile range; ICH: intracerebral hemorrhage; IVH: intraventricular hemorrhage; GCS: Glasgow Coma Scale; WFNS: World Federation of Neurosurgical Societies; mFisher: modified Fisher scale; AcomA: anterior communicating artery; PcomA: posterior communicating artery; PT: prothrombin time; APTT: activated partial thromboplastin time; TT: thrombin time; FBG: Fibrinogen; mRS: modified Rankin scale

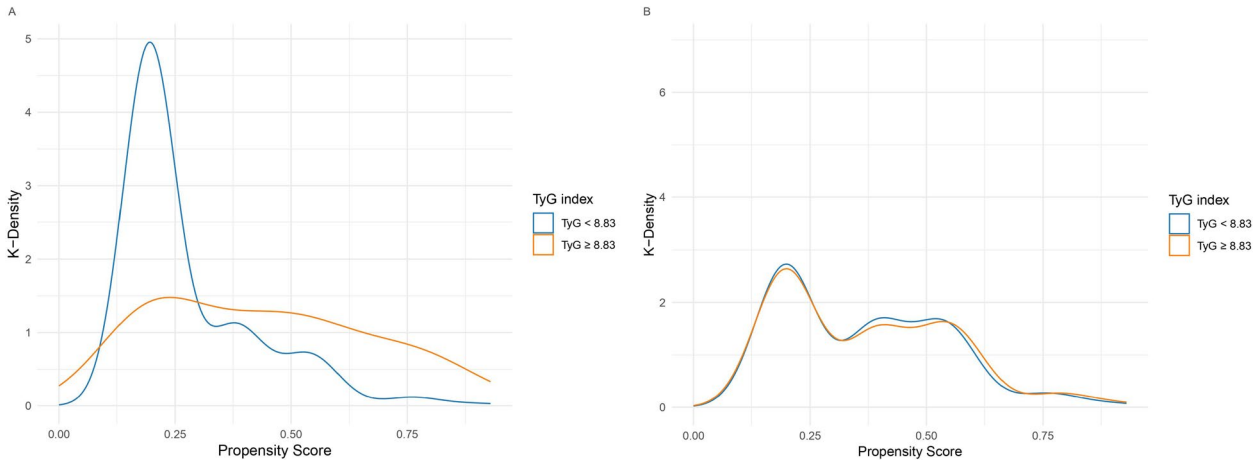

Fig. 2 Distribution of propensity scores before and after PSM. **A** Propensity score distribution before Matching. **B** Propensity Score Distribution After Matching. PSM: propensity score matching; TyG: triglyceride–glucose

that, following PSM, the imbalances in demographic and clinically parameters such as age, gender, smoking status, alcohol consumption, hypertension, hyperlipidemia, diabetes mellitus, hydrocephalus, pneumonia, presence of ICH and IVH, Hunt–Hess grade, WFNS grade, GCS score, and mFisher score between the two groups have been rectified. Remarkably, there were significant differences in poor functional outcome in patients with aSAH between the two groups, regardless of PSM (before PSM: 48.7% vs. 24.8%, $P < 0.001$; after PSM: 43.0% vs. 26.6%, $P = 0.006$).

To thoroughly assess the relationship between the TyG index and poor functional outcomes post-aSAH, univariate and multivariable logistic regression analyses were performed on both the pre- and post-PSM cohorts. In addition, Table 3 summarizes the results of logistic regression analyses. The univariate logistic analysis, referred to as the crude model, excluding adjustment for any parameters, suggested that TyG ≥ 8.83 was strongly correlated with the incidence of negative outcomes in patients affected by aSAH (before PSM: OR 2.87, 95% CI 1.92–4.31, $P < 0.001$; after PSM: OR 2.08, 95% CI 1.23–3.52, $P = 0.006$;). In model 1, the risk of unfavorable outcome post-aSAH was substantially elevated in the high-TyG group, even after adjustment for demographic parameters including age, sex, current smoking, and alcohol consumption status (OR 2.96, 95% CI 1.95–4.47, $P < 0.001$). In Model 2, adjusted for demographic parameters and incorporating comorbidities and complications such as pneumonia, hydrocephalus, hypertension,

Table 2 Baseline characteristics and outcomes of aSAH patients before and after PSM

Characteristic	Before PSM				After PSM			
	Total patients (*n* = 470)	**TyG < 8.83 (*n* = 314)**	**TyG ≥ 8.83 (*n* = 156)**	***P* value**	**Total patients (*n* = 256)**	**TyG < 8.83 (*n* = 128)**	**TyG ≥ 8.83 (*n* = 128)**	***P* value**
Demographics								
Age, years, mean (SD)	56.0 ± 9.6	55.9 ± 9.5	56.3 ± 9.7	0.693	55.4 ± 8.9	55.9 ± 8.7	55.0 ± 9.0	0.395
Gender, female, (%)	290 (61.7)	199 (63.4)	91 (58.3)	0.29	150 (58.6)	78 (60.9)	72 (56.2)	0.446
Smoking, *n* (%)	123 (26.2)	83 (26.4)	40 (25.6)	0.854	73 (28.5)	39 (30.5)	34 (26.6)	0.489
Alcohol, *n* (%)	141 (30.0)	89 (28.3)	52 (33.3)	0.266	82 (32.0)	41 (32)	41 (32)	1
Comorbidities								
Hypertension, *n* (%)	227 (48.3)	140 (44.6)	87 (55.8)	0.022	123 (48.0)	58 (45.3)	65 (50.8)	0.381
Hyperlipidemia, *n* (%)	85 (18.1)	31 (9.9)	54 (34.6)	< 0.001	67 (26.2)	31 (24.2)	36 (28.1)	0.477
Diabetes mellitus, *n* (%)	38 (8.1)	12 (3.8)	26 (16.7)	< 0.001	21 (8.2)	12 (9.4)	9 (7)	0.494
Presence of ICH, *n* (%)	109 (23.2)	63 (20.1)	46 (29.5)	0.023	61 (23.8)	28 (21.9)	33 (25.8)	0.463
Presence of IVH, *n* (%)	136 (28.9)	78 (24.8)	58 (37.2)	0.005	79 (30.9)	40 (31.2)	39 (30.5)	0.892
Hydrocephalus, *n* (%)	72 (15.3)	39 (12.4)	33 (21.2)	0.013	41 (16.0)	17 (13.3)	24 (18.8)	0.233
Pneumonia, *n* (%)	112 (23.8)	56 (17.8)	56 (35.9)	< 0.001	72 (28.1)	37 (28.9)	35 (27.3)	0.781
Clinical data								
GCS score, median (IQR)	15.0 (13.0, 15.0)	15.0 (13.0–15.0)	14.0 (9.0–15.0)	< 0.001	15.0 (12.0–15.0)	15.0 (13.0–15.0)	15.0 (10.0–15.0)	0.166
WFNS grade, median (IQR)	1.0 (1.0–3.0)	1.0 (1.0–2.0)	2.0 (1.0–4.0)	< 0.001	1.0 (1.0–4.0)	1.0 (1.0–2.0)	1.0 (1.0–4.0)	0.092
Hunt–Hess grade, median (IQR)	2.0 (1.0–3.0)	1.0 (1.0–3.0)	3.0 (1.0–4.0)	< 0.001	2.0 (1.0–3.0)	2.0 (1.0–3.0)	2.0 (1.0–4.0)	0.279
mFisher score, median (IQR)	1.0 (1.0–3.0)	1.0 (1.0–2.0)	2.0 (1.0–3.0)	< 0.001	1.0 (1.0–3.0)	1.5 (1.0–3.0)	1.0 (1.0–3.0)	0.911
Treatment				0.114				0.427
Coiling, n (%)	171 (36.4)	122 (38.9)	49 (31.4)		86 (33.6)	46 (35.9)	40 (31.2)	
Clipping, n (%)	299 (63.6)	192 (61.1)	107 (68.6)		170 (66.4)	82 (64.1)	88 (68.8)	
Aneurysm locations				0.281				0.54
Middle cerebral artery, *n* (%)	103 (21.9)	73 (23.2)	30 (19.2)		53 (20.7)	29 (22.7)	24 (18.8)	
Anterior cerebral artery, *n* (%)	24 (5.1)	19 (6.1)	5 (3.2)		10 (3.9)	7 (5.5)	3 (2.3)	
AcomA, *n* (%)	128 (27.2)	81 (25.8)	47 (30.1)		76 (29.7)	38 (29.7)	38 (29.7)	
PcomA, *n* (%)	148 (31.5)	97 (30.9)	51 (32.7)		78 (30.5)	34 (26.6)	44 (34.4)	
Internal carotid artery, *n* (%)	33 (7.0)	25 (8)	8 (5.1)		17 (6.6)	10 (7.8)	7 (5.5)	

Table 2 (continued)

Characteristic	Before PSM				After PSM			
	Total patients (*n* = 470)	**TyG < 8.83 (*n* = 314)**	**TyG ≥ 8.83 (*n* = 156)**	***P* value**	**Total patients (*n* = 256)**	**TyG < 8.83 (*n* = 128)**	**TyG ≥ 8.83 (*n* = 128)**	***P* value**
Posterior circulation, *n* (%)	34 (7.2)	19 (6.1)	15 (9.6)		22 (8.6)	10 (7.8)	12 (9.4)	
Laboratory data, median (IQR)								
Glucose, mg/dl	127.5 (108.4–154.2)	120.1 (104.2–143.3)	144.4 (120.2–179.1)	< 0.001	131.5 (111.7–157.2)	120.7 (108.7–147.5)	140.4 (118.8–172.9)	< 0.001
Triglyceride, mg/dl	80.6 (56.7–123.8)	63.8 (47.2–83.9)	148.0 (114.3–184.7)	< 0.001	97.5 (64.7–149.1)	64.7 (49.6–85.9)	148.0 (115.8–185.4)	< 0.001
TyG index	8.6 (8.1, 9.0)	8.3 (8.0, 8.6)	9.2 (9.0, 9.5)	< 0.001	8.8 (8.4–9.1)	8.4 (8.0–8.6)	9.1 (9.0–9.5)	< 0.001
Blood urea nitrogen, mmol/L	4.3 (3.5–5.3)	4.3 (3.5–5.3)	4.7 (3.6–5.4)	0.086	4.3 (3.5–5.3)	4.2 (3.5–5.3)	4.6 (3.6–5.3)	0.125
Serum creatinine, μmol/L/	58.0 (47.0–69.0)	56.0 (45.2–67.0)	63.0 (50.8–77.0)	< 0.001	59.0 (47.0–72.0)	57.5 (44.8–67.0)	63.0 (49.8–76.2)	0.002
Platelet count, $\times 10^9$/L	202.0 (161.0–243.0)	201.0 (158.2–237.5)	204.0 (166.8–261.0)	0.204	202.0 (167.8–252.2)	200.0 (161.0–235.2)	207.0 (172.8–262.5)	0.231
Hemoglobin, g/L	129.0 (118.2–140.0)	126.0 (117.0–137.0)	134.0 (122.0–143.0)	< 0.001	129.0 (119.0–142.0)	126.0 (116.8–140.2)	133.5 (122.0–143.0)	0.016
Erythrocyte count, $\times 10^9$/L	4.3 (3.9–4.6)	4.2 (3.9–4.6)	4.4 (4.0–4.7)	0.002	4.3 (4.0–4.7)	4.2 (3.8–4.6)	4.4 (4.0–4.7)	0.013
Leukocytes count, $\times 10^9$/L	10.9 (8.3–13.9)	10.1 (8.0–12.6)	12.5 (9.1–17.2)	< 0.001	11.1 (8.5–14.4)	10.3 (8.2–12.5)	12.4 (8.9–16.5)	< 0.001
Neutrophil count, $\times 10^9$/L	9.2 (6.6–12.3)	8.7 (6.4–11.2)	10.6 (7.5–15.2)	< 0.001	9.4 (6.7–12.9)	8.7 (6.5–11.1)	10.5 (7.0–14.7)	0.004
Lymphocyte count, $\times 10^9$/L	0.9 (0.7–1.3)	0.9 (0.7–1.2)	1.0 (0.7–1.4)	0.063	0.9 (0.7–1.3)	0.9 (0.6–1.2)	1.0 (0.7–1.4)	0.086
Monocyte count, $\times 10^9$/L	0.5 (0.4–0.7)	0.5 (0.3–0.7)	0.6 (0.4–0.9)	< 0.001	0.5 (0.4–0.8)	0.5 (0.4–0.7)	0.6 (0.4–0.9)	0.018
PT, sec	13.3 (12.9–13.8)	13.4 (12.9–13.9)	13.3 (12.8–13.8)	0.125	13.3 (12.8–13.8)	13.3 (12.9–13.9)	13.3 (12.8–13.8)	0.281
APTT, sec	33.2 (31.2–35.7)	33.4 (31.4–35.9)	32.7 (30.9–35.3)	0.066	33.1 (31.2–35.5)	33.3 (31.1–35.7)	32.8 (31.2–35.4)	0.49
TT, sec	16.4 (15.8–17.2)	16.4 (15.8–17.2)	16.4 (15.8–17.3)	0.431	16.4 (15.8–17.3)	16.4 (15.8–17.1)	16.5 (15.9–17.3)	0.367
FBG, g/L	3.1 (2.7–3.6)	3.1 (2.7–3.6)	3.2 (2.7–3.7)	0.27	3.2 (2.7–3.6)	3.2 (2.8–3.6)	3.2 (2.7–3.6)	0.851
Outcome characteristics								
3-month mRS score, *n* (%)				< 0.001				0.046
0	186 (39.6)	139 (44.3)	47 (30.1)		102 (39.8)	56 (43.8)	46 (35.9)	
1	85 (18.1)	61 (19.4)	24 (15.4)		41 (16.0)	21 (16.4)	20 (15.6)	
2	45 (9.6)	36 (11.5)	9 (5.8)		24 (9.4)	17 (13.3)	7 (5.5)	
3	35 (7.4)	20 (6.4)	15 (9.6)		17 (6.6)	4 (3.1)	13 (10.2)	
4	34 (7.2)	19 (6.1)	15 (9.6)		16 (6.2)	8 (6.2)	8 (6.2)	
5	62 (13.2)	27 (8.6)	35 (22.4)		43 (16.8)	16 (12.5)	27 (21.1)	
6	23 (4.9)	12 (3.8)	11 (7.1)		13 (5.1)	6 (4.7)	7 (5.5)	
Functional outcome-*n* (%)				< 0.001				0.006
Favorable	316 (67.2)	236 (75.2)	80 (51.3)		167 (65.2)	94 (73.4)	73 (57)	
Unfavorable	154 (32.8)	78 (24.8)	76 (48.7)		89 (34.8)	34 (26.6)	55 (43)	

aSAH: aneurysmal subarachnoid hemorrhage; PSM: propensity score matching; TyG: triglyceride–glucose; SD: standard deviation; IQR: interquartile range; ICH: intracerebral hemorrhage; IVH: intraventricular hemorrhage; GCS: Glasgow Coma Scale; WFNS: World Federation of Neurosurgical Societies; mFisher: modified Fisher; AcomA: anterior communicating artery; PcomA: posterior communicating artery; PT: prothrombin time; APTT: activated partial thromboplastin time; TT: thrombin time; FBG: Fibrinogen; mRS: modified Rankin scale

Table 3 Multivariate logistic regression analysis for functional outcome in aSAH patients before and after PSM

Characteristic	Crude		Model 1			Model 2		Model 3	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value		OR (95% CI)	*P* value	OR (95% CI)	*P* value
Before PSM									
TyG index	2.16 (1.6–2.92)	< 0.001	2.23 (1.64–3.04)	< 0.001	2.01 (1.27–3.19)		0.003	1.86 (1.12–3.1)	0.017
TyG < 8.83	1(Ref)		1(Ref)		1(Ref)			1(Ref)	
TyG ≥ 8.83	2.87 (1.92–4.31)	< 0.001	2.96 (1.95–4.47)	< 0.001	2.31 (1.25–4.27)		0.007	2.3 (1.17–4.5)	0.015
After PSM									
TyG index	1.8 (1.22–2.67)	0.003	1.98 (1.32–2.98)	0.001	3.11 (1.61–5.98)		0.001	2.89 (1.34–6.24)	0.007
TyG < 8.83	1(Ref)		1(Ref)		1(Ref)			1(Ref)	
TyG ≥ 8.83	2.08 (1.23–3.52)	0.006	2.3 (1.34–3.95)	0.003	4.86 (2.03–11.65)		< 0.001	4.96 (1.81–13.57)	0.002

Model 1: Adjusted for demographic parameters (age, gender, smoking and alcohol)

Model 2: Model 1 plus comorbidities and complications (pneumonia, hydrocephalus, hypertension, hyperlipidemia, diabetes mellitus, ICH and IVH)

Model 3: Model 2 plus variables related to disease severity (GCS score, WFNS grade, Hunt–Hess grade and mFisher score)

aSAH: aneurysmal subarachnoid hemorrhage; PSM: propensity score matching; OR: odds ratio; CI: confidence interval; TyG: triglyceride–glucose; ICH: intracerebral hemorrhage; IVH: intraventricular hemorrhage; GCS: Glasgow Coma Scale; WFNS: World Federation of Neurosurgical Societies; mFisher: modified Fisher

hyperlipidemia, diabetes mellitus, ICH, and IVH, the high-TyG group persisted in exhibiting a strong correlation with unfavorable outcomes post-aSAH (OR 2.31, 95% CI 1.25–4.27, $P = 0.007$). In model 3, adjustments were made for all variables from model 1 and model 2 as well as parameters related to disease severity including GCS score, WFNS grade, Hunt–Hess grade, and mFisher score. TyG ≥ 8.83 emerged as independent risk factor for unfavorable outcome in patients with aSAH (OR 2.25, 95% CI 1.16–4.38, $P = 0.017$).

The results of multivariable logistic regression analyses across the three models on the PSM cohort yielded consistent findings (Model 1: OR 2.3, 95% CI 1.34–3.95, $P = 0.003$; Model 2: OR 4.86, 95% CI 2.03–11.65, $P < 0.001$; Model 3: OR 4.95, 95% CI 1.83–13.44, $P = 0.002$). As depicted in Fig. 3, the RCS regression model indicated a linear increase in the risk of unfavorable outcomes post-aSAH with a rising TyG index (P for non-linearity = 0.38).

Subgroup analysis

Ultimately, subgroup analyses were conducted based on gender, age (< 65 and ≥ 65 years), hypertension, IVH, ICH, Hunt–Hess grade (< 4 and ≥ 4) and mFisher score (< 3 and ≥ 3) to comprehensively elucidate the correlation between the TyG index and unfavorable functional outcomes in patients with aSAH. (Fig. 4). The TyG index was markedly associated with increased risk of adverse functional outcomes in several subgroups of aSAH patients. These subgroups included: female (OR 2.69, 95% CI 1.05–6.86, $P = 0.039$), those without hypertension (OR 2.93, 95% CI 1.06–8.12, $P = 0.039$), those with ICH (OR 7.33, 95% CI 1.09–49.3, $P = 0.04$), those with IVH (OR 3.54, 95% CI 1.13–11.15, $P = 0.031$), those with Hunt–Hess grade ≥ 4 (OR 5.99, 95% CI 2.02–17.82, $P = 0.001$), and those with mFisher score ≥ 3 (OR 6.58, 95% CI 1.41–30.73, $P = 0.017$). No statistically significant interactions between the TyG index and functional outcome in aSAH patients were observed except for individuals with Hunt–Hess grade ≥ 4 (P for interaction = 0.021).

Model performance comparison

The predictive performance of the traditional model and the TyG-enhanced model was evaluated using the area under the receiver operating characteristic curve (AUC). The traditional model achieved an AUC of 0.889 on the testing set, while the TyG-enhanced model demonstrated a modest improvement in AUC to 0.899. The DeLong test indicated a statistically significant improvement in discrimination when incorporating the TyG index (DeLong Test P value: 0.048). The corresponding ROC curve is presented in Fig. 5.

Nomogram development

A nomogram integrating the TyG index with traditional prognostic scales (WFNS, Hunt–Hess, mFisher) was developed to predict poor functional outcomes in aSAH patients. The nomogram effectively visualizes the contribution of each variable to the prediction of poor outcomes and facilitates bedside risk stratification. The nomogram is depicted in Fig. 6.

Discussion

This retrospective cohort study was designed to investigate the relationship between the TyG index and functional outcomes in individuals who experienced aSAH. Employing multivariable analysis with adjustment for a range of potential confounding factors, we identified a significant correlation between an elevated TyG index

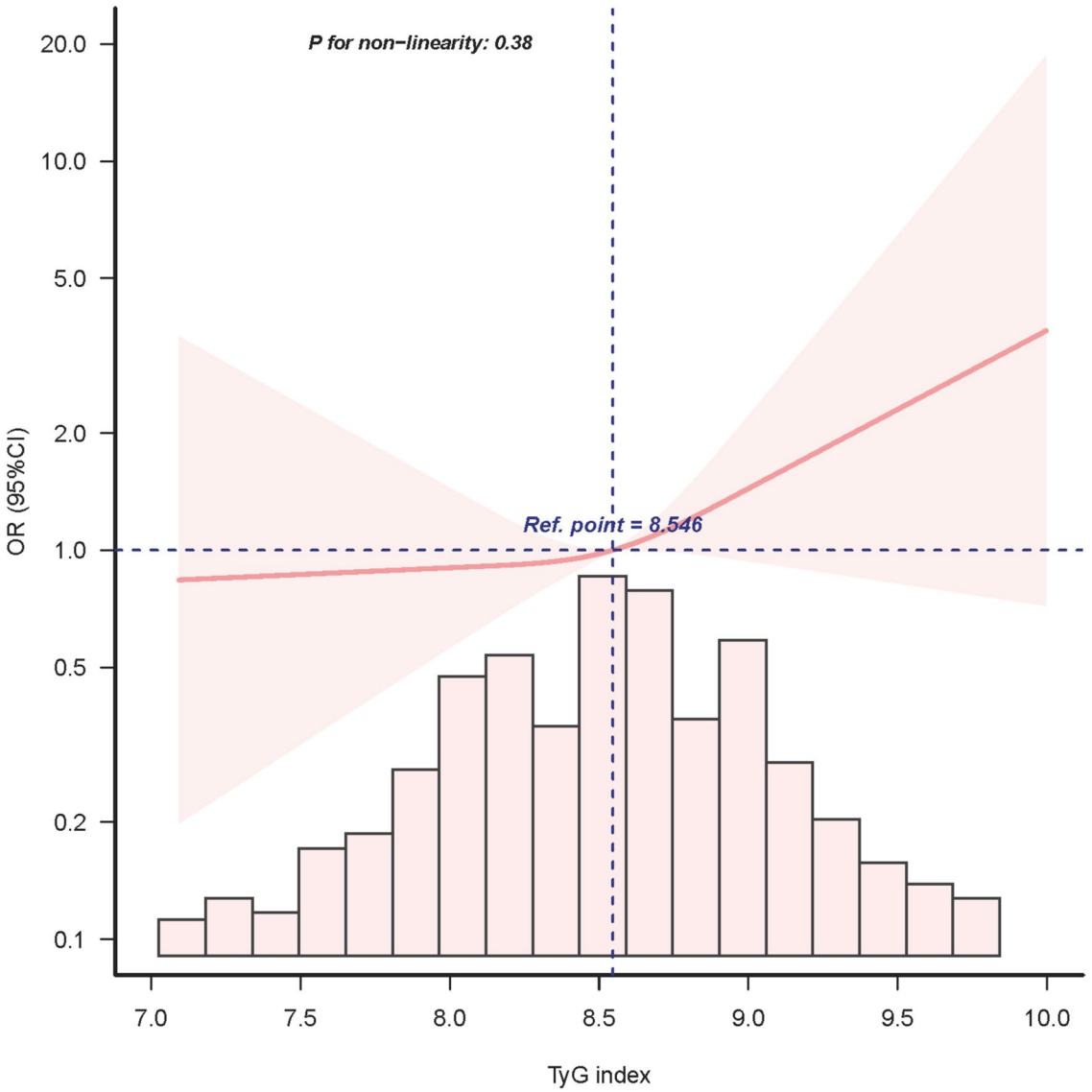

Fig. 3 Relationship between the TyG index and functional outcomes post-aSAH. OR: odds ratio; CI: confidence interval; TyG: triglyceride–glucose; aSAH: aneurysmal subarachnoid hemorrhage

and an increased risk of adverse functional outcomes. Notably, this association persisted even after the application of PSM, and subgroup analysis yielded consistent findings across the majority of subgroups. Building upon these findings, we further constructed a nomogram that integrates the TyG index and traditional prognostic scores (WFNS, Hunt–Hess, and mFisher), with the aim of facilitating bedside risk stratification. Evaluation of the predictive performance on an independent test set demonstrated that the enhanced model, which included the TyG index, exhibited a statistically significant improvement in the AUC (from 0.889 to 0.899, DeLong test $P = 0.048$) when compared to the traditional model. Therefore, the TyG index may prove to be a valuable adjunct for neurosurgeons in clinical decision-making, and it could potentially serve as an independent risk factor for functional outcome in patients with aSAH. The developed nomogram offers a practical means of integrating the TyG index into clinical practice for the purpose of risk stratification.

IR is characterized as a pathological condition, wherein the responsiveness of insulin-targeting tissues to high physiological insulin levels is diminished, ultimately resulting in a reduced biological efficacy of insulin [23].

As a novel and easily accessible biomarker, the TyG index is composed of fasting triglyceride and fasting

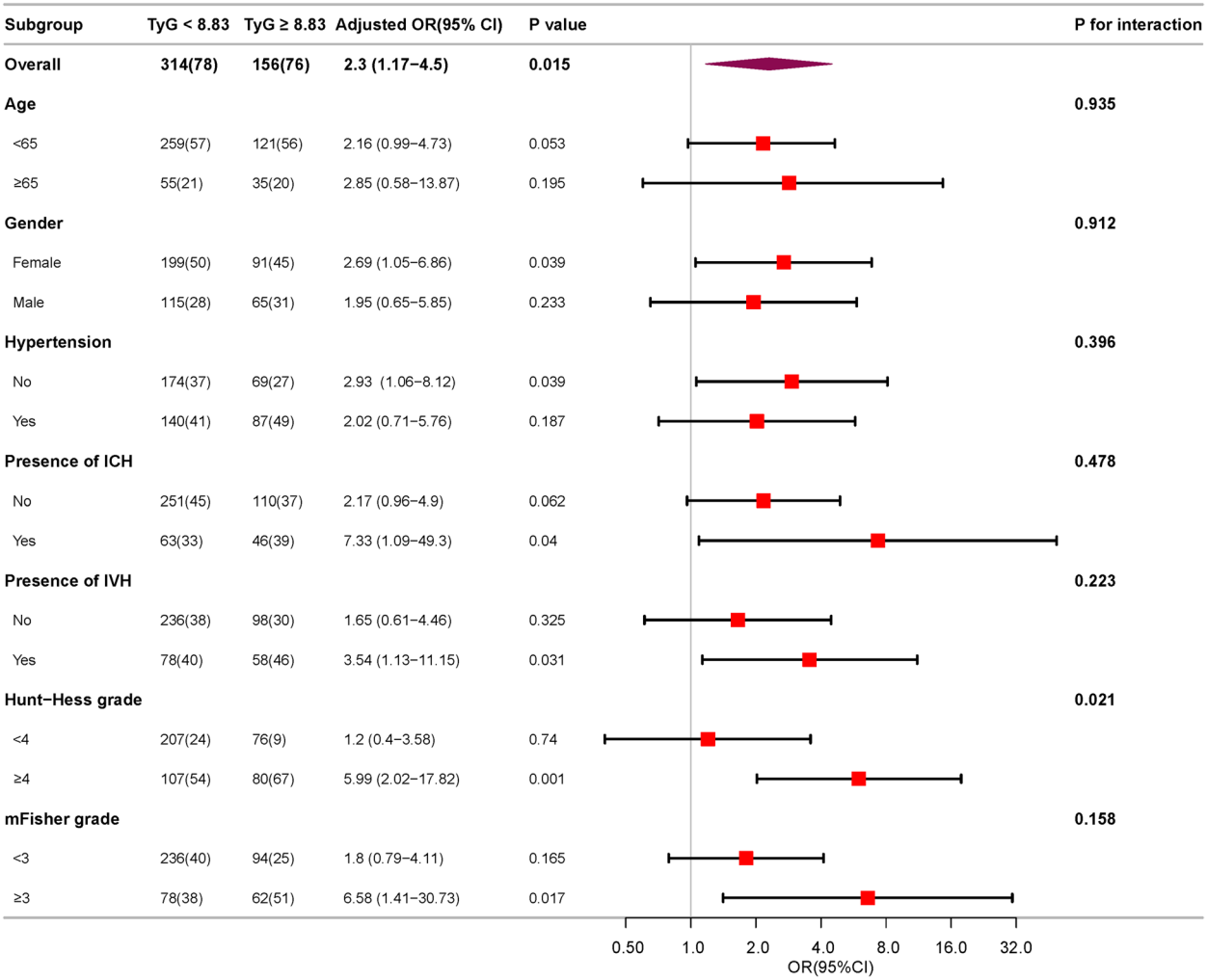

Fig. 4 Subgroup analyses of the association of TyG index and unfavorable outcomes post-aSAH. TyG: triglyceride–glucose; aSAH: aneurysmal subarachnoid hemorrhage; OR: odds ratio; CI: confidence interval; ICH: intracerebral hemorrhage; IVH: intraventricular hemorrhage; mFisher: modified Fisher

plasma glucose, and its reliability and validity in assessing IR have been confirmed in numerous previous studies [24–26]. Compared to traditional IR indicators, such as the homeostasis model assessment of IR (HOMA-IR), the TyG index offers distinct advantages. It requires only fasting triglyceride and glucose levels, which are routinely measured in clinical practice. This simplicity obviates the need for costly and labor-intensive insulin assays. Furthermore, the TyG index has demonstrated comparable or even higher accuracy than HOMA-IR in several studies [24, 27]. In the context of aSAH, we selected the TyG index due to its computational simplicity and clinical accessibility, which align well with the time-sensitive nature of aSAH management. In acute settings, complex laboratory tests can be challenging to implement, making the simplicity of the TyG index particularly valuable. In addition, the TyG index may be closely associated with key pathomechanisms of cerebrovascular diseases, such as endothelial dysfunction, which plays a central role in aSAH complications, such as vasospasm and delayed cerebral ischemia.

The detailed pathophysiological mechanisms linking the TyG index to the progression of cerebrovascular disease and poor clinical outcomes remain to be fully elucidated. Current evidence suggests IR may be a central mechanism. Prior studies indicate that glucose levels may reflect hepatic IR, while triglyceride levels may indicate adipose tissue IR [28]. Thus, the TyG index provides a composite measure of IR from dual perspectives.

IR significantly impacts endothelial function, which is critical in the pathophysiology of aSAH. Specifically, IR reduces endothelial nitric oxide synthase (eNOS) phosphorylation, leading to endothelial dysfunction and impaired vasodilation [29]. This dysfunction might

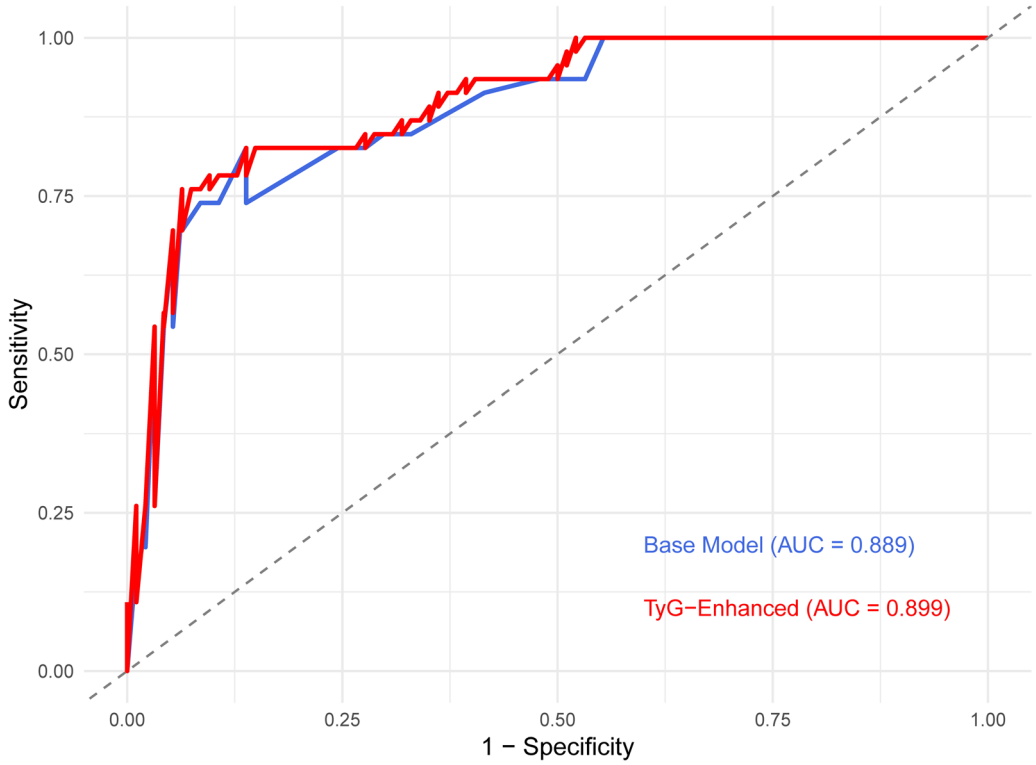

Fig. 5 ROC curve analysis comparing predictive performance of traditional and TyG-enhanced models. ROC: receiver operating characteristic; TyG: triglyceride–glucose

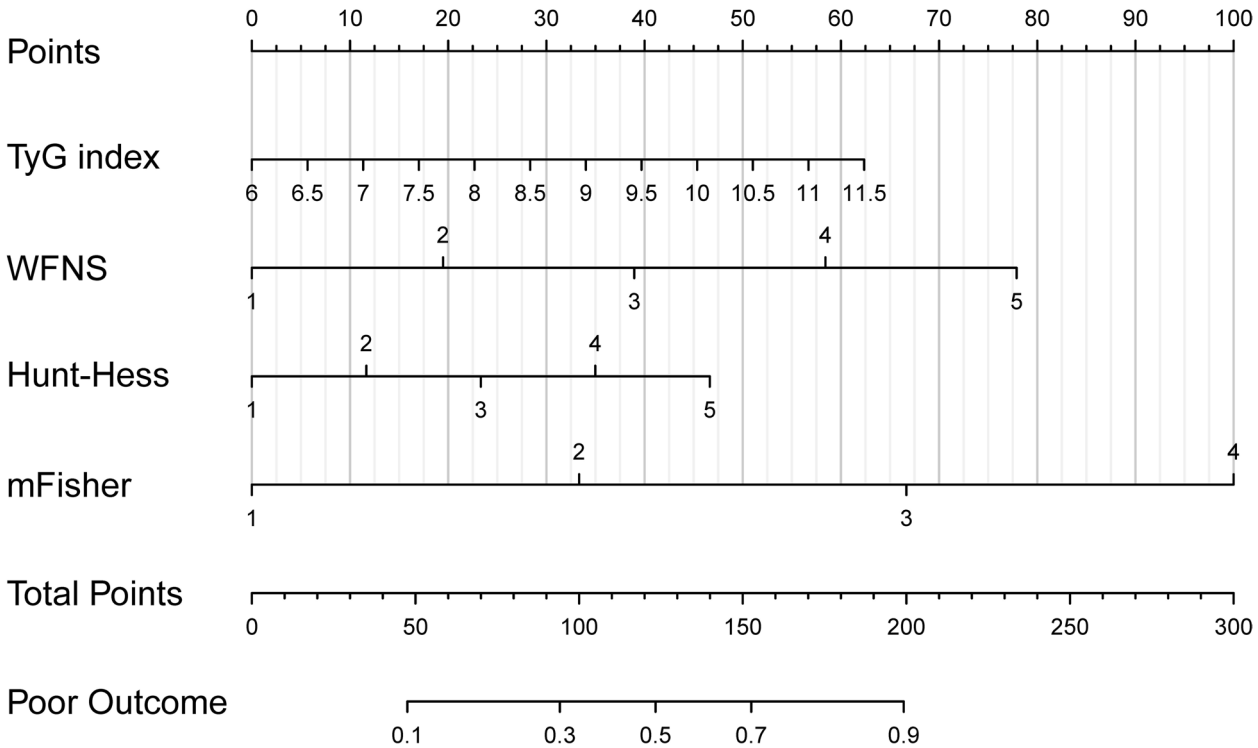

Fig. 6 Nomogram for predicting poor outcome probability in aSAH patients incorporating TyG Index and traditional prognostic scales. aSAH, aneurysmal subarachnoid hemorrhage; TyG: triglyceride–glucose

exacerbate cerebral vasospasm and delayed cerebral ischemia, hallmark complications of aSAH [30, 31].

Hyperglycemia following SAH, possibly induced by IR-related glucose metabolism impairment, may play a significant role in patient prognosis [32–34]. Hyperglycemia-induced oxidative stress could further compromise blood–brain barrier (BBB) integrity, potentially promoting immune cell infiltration and brain edema post-SAH [35]. This process is thought to be closely linked to the development of cerebral vasospasm and microthrombosis, critical determinants of neurological outcomes in aSAH patients [36]. In the context of dyslipidemia, IR may promote adipose tissue lipolysis and free fatty acid release into circulation. Elevated free fatty acids could impair insulin activity and glucose utilization in peripheral tissues, potentially perpetuating a cycle of IR and dyslipidemia [37, 38]. Lipid deposition might increase vascular endothelial permeability, thereby exacerbating endothelial damage and possibly promoting thrombotic events [39]. The TyG index could potentially indicate the metabolic processes involving glycation products and the reactivity of platelet, which may lead to endothelial cell-dependent vasodilation [37]. Furthermore, IR-induced endothelial dysfunction could disrupt vasomotor factor balance, with reduced nitric oxide (NO) activity and increased endothelin-I release possibly contributing to cerebral vasospasm [40]. Previous studies suggest the TyG index comprehensively reflects glucose metabolism, oxidative stress, and inflammation [16, 24]. IR may disrupt glial cell function, increasing neuronal apoptosis and impairing autophagy [41]. This neuroinflammatory cascade could further compromise BBB integrity, accelerating cerebral vasospasm progression [42]. Collectively, these pathophysiological disturbances underlie aSAH progression and adverse clinical outcomes. Maintaining an appropriate TyG index may offer a potential strategy to mitigate adverse prognosis in aSAH patients, though further research is needed to confirm this hypothesis.

While the predictive value of the TyG index for SAH prognosis has been previously reported, research specifically focusing on aSAH remains scarce. Existing studies have shown some utility of the TyG index in SAH, but these findings often come with limitations. For instance, Huang and colleagues, utilizing the MIMIC-IV database, explored the association between the TyG index and all-cause mortality in patients with hemorrhagic stroke [18]. However, this study combined SAH and ICH, potentially obscuring specific effects within the SAH population. Furthermore, due to inherent database limitations, critical clinical information, such as Hunt–Hess or WFNS grades, which are essential for stratifying SAH severity, could not be incorporated into their analysis.

Xie et al. conducted a single-center retrospective study investigating the TyG index and prognosis in SAH [19]. While their work provided valuable insights, the study was limited by its small sample size and the absence of key neuroimaging parameters, such as aneurysm location and mFisher score. Moreover, their study included all SAH patients, whereas our investigation specifically focuses on the aSAH population. Wang and colleagues explored the association between the TyG index and the risk of stroke over an 11-year follow-up period [43]. Although relevant to cerebrovascular events, their study's focus on stroke incidence differs from our investigation into the prognostic value of the TyG index specifically in the context of aSAH.

Ding et al. focused on the aSAH population and explored the predictive performance of various IR markers for prognosis [44]. However, similar to other studies, their analysis did not include crucial clinical information, such as mFisher score, Hunt–Hess, or WFNS grading systems. Moreover, their study had a smaller sample size compared to the present investigation.

Therefore, the current study aims to advance the field by specifically focusing on the prognostic value of the TyG index in patients with aSAH, addressing several limitations observed in previous research. By incorporating comprehensive clinical and neuroimaging data, including Hunt–Hess/WFNS grades and mFisher scores, and analyzing a larger cohort of aSAH patients, this research seeks to provide a more nuanced and generalizable understanding of the TyG index's role in predicting outcomes following aSAH. This detailed approach allows for a more direct comparison with existing literature focused on SAH and specifically addresses the gaps and limitations identified in prior studies, thereby contributing novel insights to the field.

While specific thresholds for "appropriate" TyG levels have not been universally established, prior studies have explored its association with metabolic and vascular outcomes. For instance, the TyG index has been linked to an increased risk of diabetic complications, such as retinopathy and nephropathy, with higher TyG values correlating with worse outcomes [45]. In addition, interventional studies in diabetes management have demonstrated that lifestyle modifications and pharmacological interventions targeting IR can positively influence the TyG index [46]. In previous study, lipid-lowering medications and glucose-lowering therapies have been shown to modulate the TyG index, suggesting potential avenues for intervention [47]. Future research could investigate whether targeted reductions in the TyG index improve functional recovery and clinical outcomes in specific patient populations.

To further explore the prognostic value of the TyG index across different clinical presentations of aSAH, we conducted subgroup analyses based on key demographic and clinical characteristics. Notably, interaction analyses revealed a statistically significant interaction between the TyG index and unfavorable functional outcome specifically in patients with a Hunt–Hess grade ≥ 4 (P for interaction $=0.021$). This finding suggests that the association between higher TyG levels and poorer outcomes is particularly pronounced in patients presenting with more severe initial clinical conditions. While significant main effects of the TyG index on functional outcome were observed in other subgroups including gender, hypertension status, presence of ICH or IVH, and mFisher score, the lack of significant interactions in these groups indicates a relatively consistent association across these characteristics. The enhanced prognostic impact of the TyG index in patients with higher Hunt–Hess grades underscores the potential for metabolic dysregulation, as reflected by the TyG index, to play a critical role in determining outcomes in the most severely affected aSAH patients. Future research should investigate the underlying mechanisms contributing to this intensified association and explore its implications for tailored management strategies in this high-risk population.

In addition, it should be acknowledged that the present study is subject to certain limitations. First of all, the retrospective nature of the study precludes the possibility of establishing a causal relationship. Notwithstanding the implementation of multivariate adjustments and subgroup analyses, the possibility of residual confounding remains. Moreover, the scope of this study was limited to analyzing the baseline TyG index. This study did not have access to data on the dynamic fluctuation of the TyG index in patients during their period of hospitalization. Accordingly, the predictive effect of changes in the TyG index over time requires further evaluation in subsequent studies. Due to the limitations inherent in our retrospective data, insulin levels required for HOMA-IR or metabolic score for IR (METS-IR) calculations were unavailable for our cohort. However, our findings establish an independent association between an elevated TyG index and poor aSAH prognosis. Future prospective studies should incorporate direct comparisons of the TyG index with HOMA-IR and METS-IR, utilizing ROC curves to evaluate their discriminative performance for aSAH outcomes.

Conclusion

In summary, there exists a robust correlation between the TyG index and the occurrence of adverse outcomes in patients with aSAH. This association highlights the potential utility of the TyG index as a prognostic tool for stratifying aSAH patients based on the risk of adverse outcomes. Monitoring TyG index may provide valuable insights for clinical decision-making and management. Further research is required to ascertain whether better management of the TyG index will enhance future clinical outcomes.

Abbreviations

aSAH	Aneurysmal subarachnoid hemorrhage
AUC	Area under the curve
BBB	Blood–brain barrier
CI	Confidence interval
CT	Computed tomography
CTA	Computed tomography angiography
DSA	Digital subtraction angiography
eNOS	Endothelial nitric oxide synthase
GCS	Glasgow Coma Scale
GFAP	Glial fibrillary acidic protein
HOMA-IR	Homeostasis model assessment of insulin resistance
IA	Intracranial aneurysm
ICH	Intracerebral hemorrhage
IR	Insulin resistance
IQR	Interquartile range
IVH	Intraventricular hemorrhage
mFisher	Modified Fisher Scale
mRS	Modified Rankin Scale
METS-IR	Metabolic score for insulin resistance
MIMIC-IV	Medical Information Mart for Intensive Care
NO	Nitric oxide
NSE	Neuron-specific enolase
OR	Odds ratio
PSM	Propensity score matching
ROC	Receiver operating characteristic
RCS	Restricted cubic splines
SAH	Subarachnoid hemorrhage
S100B	S100 calcium binding protein B
SD	Standard deviation
TyG	Triglyceride–Glucose Index
WFNS	World Federation of Neurosurgical Societies

Supplementary Information

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Supplementary Material 1.

Supplementary Material 2.

Supplementary Material 3.

Supplementary Material 4.

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None.

Author contributions
JY and XM contributed to the conception and design of the study, had full access to all the data in the study. YH and XG contributed to the acquisition of data. YH, XG, HL, RC and YH contributed to the analysis and interpretation of the data. All authors participated in manuscript writing, revision, and approved the submitted version.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
This study is a retrospective analysis utilizing anonymized data. It does not cause harm to human subjects, does not involve sensitive information, and does not involve commercial interests. Therefore, the need for ethics approval was waived by the Ethics Committee of Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology. Due to the retrospective nature of the study, informed consent was waived by the Ethics Committee of Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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