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Association between stress hyperglycemia ratio and functional outcomes in patients with acute ischemic stroke

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Abstract

Objective This study aimed to evaluate the association between stress hyperglycemia ratio (SHR) and poor functional outcomes at 90 days in patients with acute ischemic stroke (AIS).

Methods This study retrospectively collected 1988 AIS patients admitted to two hospitals in the Shenzhen area between January 2022 and March 2023. A total of 1255 patients with Fasting Blood-glucose (FBG) and hemoglobin A1c (HbA1C) values at admission were included in this analysis. SHR, measured by FBG/HbA1C, was evaluated as both a tri-categorical variable (Tertile 1: ≤ 0.83; Tertile 2: 0.84 -0.95; Tertile 3: ≥ 0.96). The outcome was poor functional outcomes (modified Rankin Scale [mRS] score 2–6) at 90 days. We performed univariate analysis, multiple equation regression analysis, stratified analysis, and interactive analysis.

Results Compared with patients in the lowest tertile of SHR, the highest tertile group had significantly lower odds of achieving poor functional outcomes (adjusted odds ratio, OR = 2.84, 95% CI: 2.02–3.99, P < 0.0001) at 90 days after adjusting for potential covariates. Similar results were observed after further adjustment for white blood cell count, neutrophil count, lymphocyte count, fasting blood glucose, stroke type, intravenous thrombolytic therapy, baseline Glasgow score, and baseline NIHSS score.

Conclusion SHR, as measured by the FBG/HbA1C, was associated with an increased odds of achieving poor functional outcomes in patients with AIS at 90 days.

Keywords Acute ischemic stroke, Stress hyperglycemia ratio, Functional outcomes, Cohort

Introduction

Stroke is the first cause of death and the second leading cause of disability in adults, and it imposes a serious economic and social burden on families and society [1]. GBD showed that The annual number of strokes and deaths due to stroke increased substantially from 1990 to 2019 [2]. It was estimated that among the Chinese population aged 40 years and older in 2020, there were 3.4 million incident cases of stroke, and 2.3 million deaths from stroke. Ischemic stroke constituted 15.5 million (86.8%) of all incident strokes in 2020 [3, 4]. The incidence of poor functional outcomes at 90 days after acute ischemic stroke is 13.7–55.6% [5–7]. Poor functional outcomes

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mean decreased patient quality of life and increased burden on society and family, even death.

In major diseases, such as stroke and myocardial infarction, patients experience a stress response in which plasma glucose levels are often acutely elevated, a phenomenon known as stress hyperglycemia [8]. This stress state can aggravate the progression of stroke disease, aggravate the damage and edema of the patient's brain tissue, expand the infarct size, and further increase disability and mortality. Stress hyperglycemia ratio (SHR) indicator considering both glucose and HbA1C to quantify this stress condition [9]. The degree of stress hyperglycemia can be accurately identified and quantified, and the predictive value of SHR is better than other indicators [10].

Some previous studies have data collection a respective cohort of post-discharge poor functional outcomes and mortality ranging from 3 to 12 months and found that SHR is associated with poor outcomes, but its evidence in the Chinese population still needs further validation [11]. Previous studies have shown that variables such as diabetes mellitus and intravenous thrombolytic therapy affect the relationship between SHR and poor functional outcomes in patients with AIS, and the relationship between SHR and poor functional outcomes between different subgroups is unclear and still needs to be further explored and validated [12–14].

Hence, using the data from double center hospitals for patients with AIS, we aimed to evaluate the association between SHR, measured by glucose/HbA1C, and 90-day functional outcomes in patients with AIS. To use stratified analysis to further explore the association between SHR and poor functional outcomes among different subgroups.

Material and methods

Study design and participants

This reprospective cohort study was collected of 1255 patients with AIS admitted to two hospitals in the Shenzhen area between January 2022 and March 2023. Inclusion criteria were met: (1) Age $\geq$ 18 years; (2) Onset of stroke within 72 h of hospital admission; (3) Meeting the diagnostic criteria of ICD-10 for diagnosis of stroke [15]. Exclusion criteria were met: (1) Transient Ischemic Attacks (TIA); (2) Severe cardiac, hepatic, renal, or other systemic diseases; (3) Intracerebral hemorrhage or mass lesions; (4) Missing data for the main observational and outcome indicators. This study was approved by the ethics committees of two hospitals, the ethical batch number was [Ethics Approval No. 2022 No. (150)], and (Ethics Approval No. 2022ECPJ161). Informed consent has been obtained from the participants, their parents and legally authorized representatives in this study.

Data collection

This was a double-center retrospective cohort study. Data collection was based on patients' hospitalized cases. The following data were collected: (1) Demographic data included age, gender, and education level; (2) Vital signs consisting of temperature, pulse, respiration, systolic blood pressure (SBP), diastolic blood pressure (DBP) measured at admission. (3) Stroke risk factors: history of stroke, hypertension, diabetes mellitus, hyperlipidemia, atrial fibrillation, smoke status, drink status; (4) Stroke characteristics: the severity of stroke was assessed using National Institutes of Health Stroke Scale (NIHSS) at admission, and the subtype of stroke was classified according to the Trial of ORG 10172 in Acute Stroke Treatment (TOAST) criteria [16]; (5) Laboratory data: white blood cells (WBC), low-density lipoprotein cholesterol (LDL-C), Neutrophil count, lymphocyte count, Alanine transaminase, hemoglobin A1c (HbA1c), and Fasting Blood glucose within 24 h of admission.

Assessment of stress hyperglycemia radio

Relevant data were obtained by reviewing patients' electronic medical records. Blood glucose values and HbA1c levels were measured from serum specimens at the time of admission. SHR was assessed using the following formula: glucose (mmol/L)/HbA1C (%) [17]. The patients were then categorized into three groups and further statistical analyses were performed by tertiles of glucose/HbA1C (Tertile 1: $\leq$ 0.83; Tertile 2: 0.84 -0.95; Tertile 3: $\geq$ 0.96).

Outcome measures

At 90 days after stroke onset, all patients were evaluated by modified Rankin Scale (mRs) score by telephone follow-up, we dichotomized patient outcome into two groups. The first group consisted of patients considered able to live independently, defined by mRS 0–1 upon 90 days after stroke [18]. The second group included all patients with varying degrees of dependency (including death), i.e., mRS 2–6 upon 90 days after AIS, the two groups were referred to as patients with favorable vs. poor functional outcomes, respectively.

Statistical analyses

All the normally distributed and skewed continuous variables were described as mean (standard deviation, SD) or median (interquartile range, IQR), and

categorical variables were described as frequencies (%). The baseline characteristics of the different functional outcomes groups were analyzed using a one-way analysis of variance (normal distribution), the Kruskal–Wallis H (skewed distribution), and the chi-square test (categorical variables). The effects of different SHR values on poor functional outcomes (mRS score of 2–6) were assessed using a binary logistic regression model. In the multivariable logistic regression analysis, two models were included in the analysis. The final factors incorporated included those with significance ($p < 0.05$) across tertiles of SHR and those associated with favorable functional outcomes in univariate analysis. In the first model, we adjusted variables including age, gender, Education, drinking, and smoking. In the second model, we further adjusted for age, gender, education, drinking, smoking, diabetes mellitus, stroke, white blood cell count, neutrophil count, lymphocyte count, fasting blood glucose, hemoglobin A1c, stroke type, intravenous thrombolytic therapy, baseline Glasgow score, baseline NIHSS score. Subsequently, subgroup analyses were conducted to examine the consistency between SHR and poor functional outcomes in patients with different baseline characteristics. The odds ratios (OR) with 95% confidence intervals (CIs) were also reported. This study used a generalized additive model (GAM) to investigate the dose–response relationship between the HbA1c and 90-day functional outcomes. All statistical analyses were performed using the statistical packages R (The R Foundation; http://www.r-project.org; version 3.6.3) and EmpowerStats (https://www.empowerstats.net, X&Y solutions, Inc. Boston, MA). Results were considered statistically significant at two-tailed $p < 0.05$.

Results

Study participants and baseline characteristics

A total of 1988 patients with AIS were enrolled from two hospitals in the Shenzhen area, of those, 733 were excluded. The inclusion and exclusion processes of the study were shown in Fig. 1. Finally, 1255 patients were included in this analysis, including 336 females (26.77%) and 919 males (73.23%). The median (IQR) age, and baseline NIHSS score were 58.23 (26–95) years, and 3 (2–6), respectively. The incidence of 90-day poor functional outcomes in patients with AIS was 23.82%. The baseline characteristics of the patients included by tertiles of SHR (Tertile 1: ≤ 0.83; Tertile 2: 0.84 -0.95; Tertile 3: ≥ 0.96) were shown in Table 1.

Predictive values of stress hyperglycemia ratio for function outcomes

The results of univariate regression analysis and multiple regression equation analysis were shown in Tables 2 and 3, respectively. As shown, the higher the SHR of a patient, the higher the probability of achieving a poor functional outcome. In the unadjusted model, SHR was positively correlated with 90-day poor functional outcomes (T1 vs. T3: *OR*: 2.84, 95% *CI*: 2.02–3.99, $P < 0.0001$). After adjusting for confounding factors, this positive correlation still exists in Model 1 (T1 vs. T3: *OR*: 3.11, 95% *CI*: 2.17–4.46, $P < 0.0001$) and Model 2 (T1 vs. T3: *OR*: 2.69, 95% *CI*: 1.57–4.61, $P < 0.0005$),

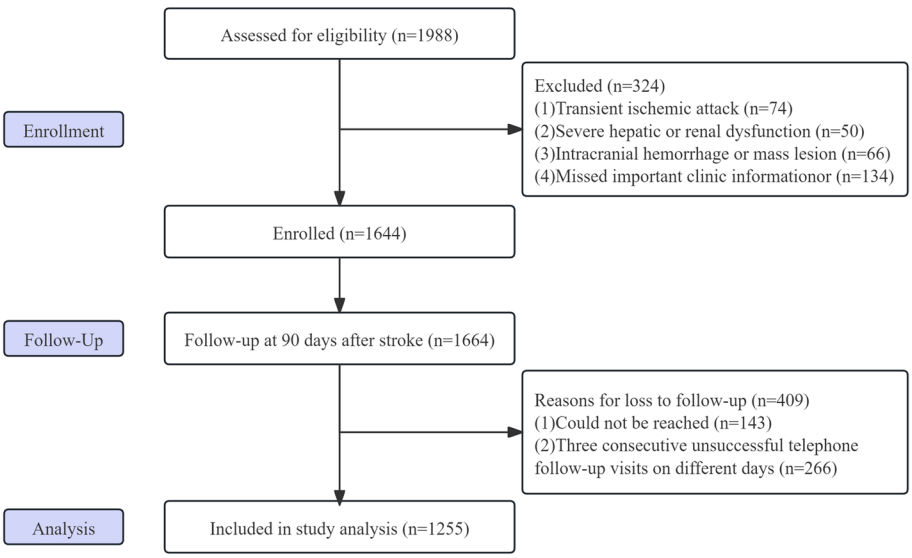

Fig. 1 Flow chart of study sample

Table 1 Baseline characteristics and functional outcomes of AIS patients according to SHR (*n*=1255)

Parameters	All patients (*n*= 1255)	Tertiles of SHR			*P*-value
		Tertile 1 ≤ 0.83 (*n*=418)	Tertile 2 (0.84-0.95) (*n*=418)	Tertile 3 ≥ 0.96 (*n*=419)	
Demographics					
Age, years, mean±SD	58.23 ± 12.82	58.02 ± 12.20	58.12 ± 12.81	58.55 ± 13.45	0.821
Sex, n (%)					0.749
Female	336 (26.77)	107 (25.60)	112 (26.79)	117 (27.92)	
Male	919 (73.23)	311 (74.40)	306 (73.21)	302 (72.08)	
Education, n (%)					0.009*
Illiteracy	37 (2.95)	10 (2.39)	12 (2.87)	15 (3.58)	
Primary or junior high school	666 (53.07)	233 (55.74)	230 (55.02)	203 (48.45)	
High school or junior college	413 (32.91)	116 (27.75)	134 (32.06)	163 (38.90)	
Bachelor degree or above	139 (11.08)	59 (14.11)	42 (10.05)	38 (9.07)	
Vital signs, mean±SD					
Temperature, ℃	36.55 ± 0.31	36.54 ± 0.31	36.55 ± 0.30	36.55 ± 0.32	0.729
Respiratory rate, bpm	19.37 ± 2.44	19.38 ± 1.21	19.33 ± 3.87	19.40 ± 1.22	0.908
Pulse Rate, /min	80.57 ± 14.78	79.48 ± 14.82	79.55 ± 13.85	82.67 ± 15.44	0.002*
Baseline SBP, mmHg	151.50 ± 24.50	149.86 ± 21.43	151.01 ± 23.99	153.61 ± 27.59	0.076
Baseline DBP, mmHg	91.54 ± 15.97	90.35 ± 14.75	91.71 ± 16.21	92.54 ± 16.84	0.134
Risk factors, n (%)					
Smoking	528 (42.07)	202 (48.33)	182 (43.54)	144 (34.37)	<0.001*
Drinking	377 (30.04)	124 (29.67)	126 (30.14)	127 (30.31)	0.978
Hypertension	803 (63.98)	259 (61.96)	269 (64.35)	275 (65.63)	0.532
Diabetes mellitus	265 (21.12)	70 (16.75)	62 (14.83)	133 (31.74)	<0.001*
Hyperlipidemia	44 (3.51)	17 (4.07)	18 (4.31)	9 (2.15)	0.177
Atrial fibrillation	39 (3.11)	10 (2.39)	13 (3.11)	16 (3.82)	0.493
Stroke	256 (20.40)	94 (22.49)	82 (19.62)	80 (19.09)	0.423
Stroke characteristics					
TOAST classification, n (%)					0.114
Large artery atherosclerosis	413 (32.91)	132 (31.58)	134 (32.06)	147 (35.08)	
Cardiogenic embolism	473 (37.69)	161 (38.52)	173 (41.39)	139 (33.17)	
Small vessel occlusion	94 (7.49)	30 (7.18)	22 (5.26)	42 (10.02)	
Other determined etiology	119 (9.48)	45 (10.77)	39 (9.33)	35 (8.35)	
Undetermined cause	156 (12.43)	50 (11.96)	50 (11.96)	56 (13.37)	
Baseline NIHSS, median (IQR)	3.00 (2.00-6.00)	3.00 (1.00-5.00)	3.00 (1.00-6.00)	4.00 (2.00-6.50)	<0.001*
Intravenous thrombolytic therapy, n (%)	228 (18.17)	68 (16.27)	70 (16.75)	90 (21.48)	0.097
Baseline GCS, mean±SD	14.62 ± 1.59	14.69 ± 1.55	14.84 ± 0.87	14.33 ± 2.07	<0.001*
Laboratory data					
WBC, 10^9/L, mean±SD	8.12 ± 2.73	8.05 ± 2.48	7.83 ± 2.64	8.48 ± 3.02	0.002*
LDL-C, mmol/L, mean±SD	3.00 ± 0.92	2.96 ± 0.90	2.97 ± 0.95	3.07 ± 0.90	0.163
Neutrophil count, 10^9/L, median (IQR)	5.00 (3.92-6.40)	4.80 (3.98-6.15)	4.90 (3.67-6.20)	5.40 (4.20-6.75)	<0.001*
Lymphocyte count, 10^9/L, median (IQR)	1.80 (1.35-2.30)	1.90 (1.40-2.33)	1.77 (1.36-2.20)	1.78 (1.30-2.40)	0.032*
Alanine transaminase, U/L, median (IQR)	18.00 (13.00-25.00)	18.00 (13.00-25.00)	17.00 (13.00-24.00)	19.00 (13.00-26.00)	0.041*
HbA1c, %, mean±SD	6.59 ± 1.75	6.46 ± 1.45	6.24 ± 1.32	7.06 ± 2.23	<0.001*
Fasting Blood-glucose, mmol/L, median (IQR)	5.30 (4.70-6.50)	4.60 (4.30-4.90)	5.21 (4.90-5.75)	6.94 (5.81-9.54)	<0.001*
Functional outcome, n (%)					<0.001*
Favorable functional outcome	956 (76.18)	357 (85.41)	317 (75.84)	282 (67.30)	
Poor functional outcome, n ()	299 (23.82)	61 (14.59)	101 (24.16)	137 (32.70)	

Abbreviations: AIS Acute ischemic stroke, *SHR* Stress hyperglycemia ratio, *SBP* Systolic blood pressure, *DBP* Diastolic blood pressure, *TOAST* Trial of ORG 10172 in Acute Stroke Treatment, *NIHSS* National Institutes of Health Stroke Scale, *GCS* Glasgow Coma Scale, *WBC* White blood cell count, *LDL-C* Low-Density Lipoprotein Cholesterol, *HbA1c* Hemoglobin A1c, *SD* Standard deviation, *IQR* Interquartile range

*$P < 0.05$

Table 2 The unadjusted association between baseline variables and 90-day poor functional outcome (*n*=1255)

Exposure	Favorable functional outcome (*n*=956)	Poor functional outcome (*n*=299)	OR (95% CI)	*P*-value
Demographics				
Age, years, mean±SD	56.41 ± 11.86	64.06 ± 13.99	1.05 (1.04, 1.06)	<0.001*
Gender, n (%)				0.235
Female	248 (25.94)	88 (29.43)	Reference	
Male	708 (74.06)	211 (70.57)	0.84 (0.63, 1.12)	
Education, n (%)				
Illiteracy	17 (1.78)	20 (6.69)	Reference	
Primary or Junior high school	476 (49.79)	190 (63.55)	0.34 (0.17, 0.66)	0.002*
High school or Junior college	345 (36.09)	68 (22.74)	0.17 (0.08, 0.34)	<0.001*
Bachelor degree or above	118 (12.34)	21 (7.02)	0.15 (0.07, 0.34)	<0.001*
Vital signs, mean±SD				
Temperature, ℃	36.53 ± 0.31	36.59 ± 0.32	1.87 (1.25, 2.79)	0.002*
Respiratory rate, bpm	19.40 ± 2.68	19.25 ± 1.40	0.95 (0.86, 1.05)	0.338
Pulse Rate, /min	80.29 ± 14.28	81.45 ± 16.29	1.01 (1.00, 1.01)	0.235
Baseline SBP, mmHg	150.46 ± 24.30	154.82 ± 24.88	1.01 (1.00, 1.01)	0.007*
Baseline DBP, mmHg	91.49 ± 16.42	91.68 ± 14.47	1.00 (0.99, 1.01)	0.864
Risk factors, n (%)**)**				
Smoking	412 (43.10)	116 (38.80)	0.84 (0.64, 1.09)	0.188
Drinking	287 (30.02)	90 (30.10)	1.00 (0.76, 1.33)	0.979
Hypertension	600 (62.76)	203 (67.89)	1.25 (0.95, 1.65)	0.107
Diabetes mellitus	189 (19.77)	76 (25.42)	1.38 (1.02, 1.88)	0.037*
Hyperlipidemia	37 (3.87)	7 (2.34)	0.60 (0.26, 1.35)	0.214
Atrial fibrillation	19 (1.99)	20 (6.69)	3.54 (1.86, 6.72)	0.001*
Stroke	164 (17.15)	92 (30.77)	2.15 (1.59, 2.89)	<0.001*
Stroke characteristics				
TOAST classification, n (%)				
Large artery atherosclerosis	288 (30.13)	125 (41.81)	Reference	
Cardiogenic embolism	385 (40.27)	88 (29.43)	0.53 (0.39, 0.72)	<0.001*
Small vessel occlusion	59 (6.17)	35 (11.71)	1.37 (0.86, 2.18)	0.191
Other determined etiology	92 (9.62)	27 (9.03)	0.68 (0.42, 1.09)	0.108
Undetermined cause	132 (13.81)	24 (8.03)	0.42 (0.26, 0.68)	<0.001*
Baseline NIHSS, median (IQR)	3.00 (1.00-5.00)	6.00 (4.00-9.00)	1.26 (1.21, 1.31)	<0.001*
Intravenous thrombolytic therapy, n (%)	778 (81.38)	249 (83.28)	0.88 (0.62, 1.24)	0.458
Baseline GCS, mean±SD	14.81 ± 1.19	14.02 ± 2.37	0.77(0.72,0.84)	<0.001*
Laboratory data				
WBC, 10^9/L, mean±SD	8.02 ± 2.54	8.43 ± 3.25	1.05 (1.01, 1.10)	0.026*
LDL-C, mmol/L, mean±SD	3.00 ± 0.91	3.00 ± 0.96	1.00 (0.87, 1.15)	0.959
Neutrophil count, 10^9/L, median (IQR)	4.90 (3.90-6.20)	5.40 (4.09-6.81)	1.07 (1.02, 1.12)	0.003*
Lymphocyte count, 10^9/L, median (IQR)	1.89 (1.42-2.35)	(1.20-2.10)	0.80 (0.68, 0.95)	0.009*
Alanine transaminase, U/L, median (IQR)	18.00 (13.00-26.00)	17.00 (12.00-23.00)	0.99 (0.98, 1.00)	0.148
HbA1c, %, mean±SD	6.51 ± 1.63	6.83 ± 2.06	1.10 (1.03, 1.18)	0.006*
Fasting Blood-glucose, mmol/L, median (IQR)	5.19 (4.70-6.25)	5.83(5.07-7.50)	1.12 (1.07, 1.17)	<0.001*
SHR, n (%)				
Low	357 (37.34)	61 (20.40)	Reference	
Middle	317 (33.16)	101 (33.78)	1.86(1.31, 2.65)	<0.001*
High	282 (29.50)	137 (45.82)	2.84(2.02, 3.99)	<0.001*

Abbreviations: AIS Acute ischemic stroke, *SHR* Stress hyperglycemia ratio, *SBP* Systolic blood pressure, *DBP* Diastolic blood pressure, *TOAST* Trial of ORG 10172 in Acute Stroke Treatment, *NIHSS* National Institutes of Health Stroke Scale, *GCS* Glasgow Coma Scale, *WBC* White blood cell count, *LDL-C* Low-Density Lipoprotein Cholesterol, *HbA1c* Hemoglobin A1c, *SD* Standard deviation, *IQR* Interquartile range, *OR* Odds Ratio, *CI* Confidence interval

**P < 0.05*

Table 3 Relationship between SHR and 90-day poor functional outcome (n=1255)

Exposure		Event, n (%)	Crude Model		Model 1		Model 2	
			Unadjusted OR (95% CI)	*P*-value	Adjusted OR (95% CI)	*P*-value	Adjusted OR (95% CI)	*P*-value
SHR	Tertile 1	61(14.59)	Reference	Reference	Reference	Reference	Reference	Reference
	Tertile 2	101(24.16)	1.86 (1.31-2.65)	<0.001*	1.94 (1.35-2.80)	<0.001*	1.97 (1.28-3.03)	0.002*
	Tertile 3	137(32.70)	2.84 (2.02-3.99)	<0.001*	3.11 (2.17-4.46)	<0.001*	2.69 (1.57-4.61)	<0.001*

The crude model is not adjusted. Model 1 adjust for: age, gender, education, drinking, smoking. Model 2 adjust for: age, gender, education, drinking, smoking, diabetes mellitus, stroke, white blood cell count, neutrophil count, lymphocyte count, fasting blood-glucose, hemoglobin A1c, stroke type, intravenous thrombolytic therapy, baseline Glasgow score, baseline NIHSS score

Abbreviations: OR Odds Ratio, *CI* Confidence interval, *SHR* Stress hyperglycemia ratio

P for the trend is less than 0.05. Furthermore, as illustrated in Figure S1, the probability of estimating a poor functional outcome increased as the SHR value increased.

Subgroup analyses

This study also stratified by continuous variables and categorical variables to perform subgroup analyses to further explore the association between SHR values and functional outcomes in different subgroups. There was a consistent effect of SHR on 90-day poor functional outcomes across different subgroups and interaction analysis also showed no heterogeneity among patients with different baseline characteristics, results were shown in Fig. 2. More information was shown in Table S1.

Discussion

In this retrospective double-center cohort study, we evaluated the association between the stress hyperglycemia ratio and 90-day poor functional outcomes in patients with AIS. This study found that the SHR calculated from the ratio of fasting blood glucose and HbA1c was associated with a 90-day poor functional outcome in patients with AIS, higher SHR is associated with a higher risk of a 90-day poor functional outcome. In addition, associations between SHR and poor functional outcomes were observed in different subgroups.

A Previous study have shown that a higher SHR implies a poorer functional outcome, mortality, neurological deficits, HT, and infectious complications in stroke patients [11]. However, the mechanism by which stress hyperglycemia ratio affects poor functional outcomes has not been fully explored, but there are several explanations for this association between stress hyperglycemia and increased risk of poor clinical outcomes; first, hyperglycemia promotes the activation and release of inflammatory factors, amplifies the inflammatory response, damages the vascular endothelium, and exacerbates neuronal injury after cerebral infarction [19]. Second, stress hyperglycemia may cause brain tissue acidosis and lactic acid production, leading to intracellular acidosis, accelerating oxidative stress by enhancing lipid and free radical peroxidation, further aggravating brain tissue hypoxia and hypoxia, and promoting nerve cell destruction [20]. Third, stress hyperglycemia may lead to abnormal platelet aggregation, which may also enhance vascular permeability, aggravate nerve cell damage and brain tissue edema, and further deteriorate neurological functions [21].

Subgroup analyses revealed an independent correlation between SHR and 90-day poor functional outcomes in different baseline characteristics subgroups, with no significant interaction noted. This study found that the association between SHR and 90-day poor functional outcomes is not influenced by diabetic status,. These findings align partially with prior research. A recently study revealed an independent correlation between SHR and poor prognosis in non-diabetic patients, whereas no similar correlation was identified in diabetic patients [22]. Merlino et al. [23] showed that SHR was associated with poor clinical prognosis at 90-day after onset which focus on non-diabetic patients with AIS. The risk of 90-day poor functional outcomes was higher in patients with diabetic than without[*OR*: 6.55 (2.98, 14.41) vs *OR*: 3.06 (1.28, 7.30)]. This was contrary to previous study, which demonstrated a correlation between SHR and all-cause mortality among diabetic patients compared to non-diabetic ones [24]. Consistent with this study, the interaction test lacked statistical significance. This may be because diabetic patients' cells are chronically exposed to hyperglycemia and have a more blunted response to hypo- and hyperglycemia and changes in blood glucose levels [25]. This study found whether thrombolytic or not. SHR is associated with poor outcomes in AIS patients, but patients receiving IVT have a significantly higher risk than those who do not receive IVT [*OR*: 15.52 (4.25, 56.72) vs *OR*: 3.74 (1.88, 7.43)]. Previous studies have confirmed the relationship between hyperglycemia or diabetes and

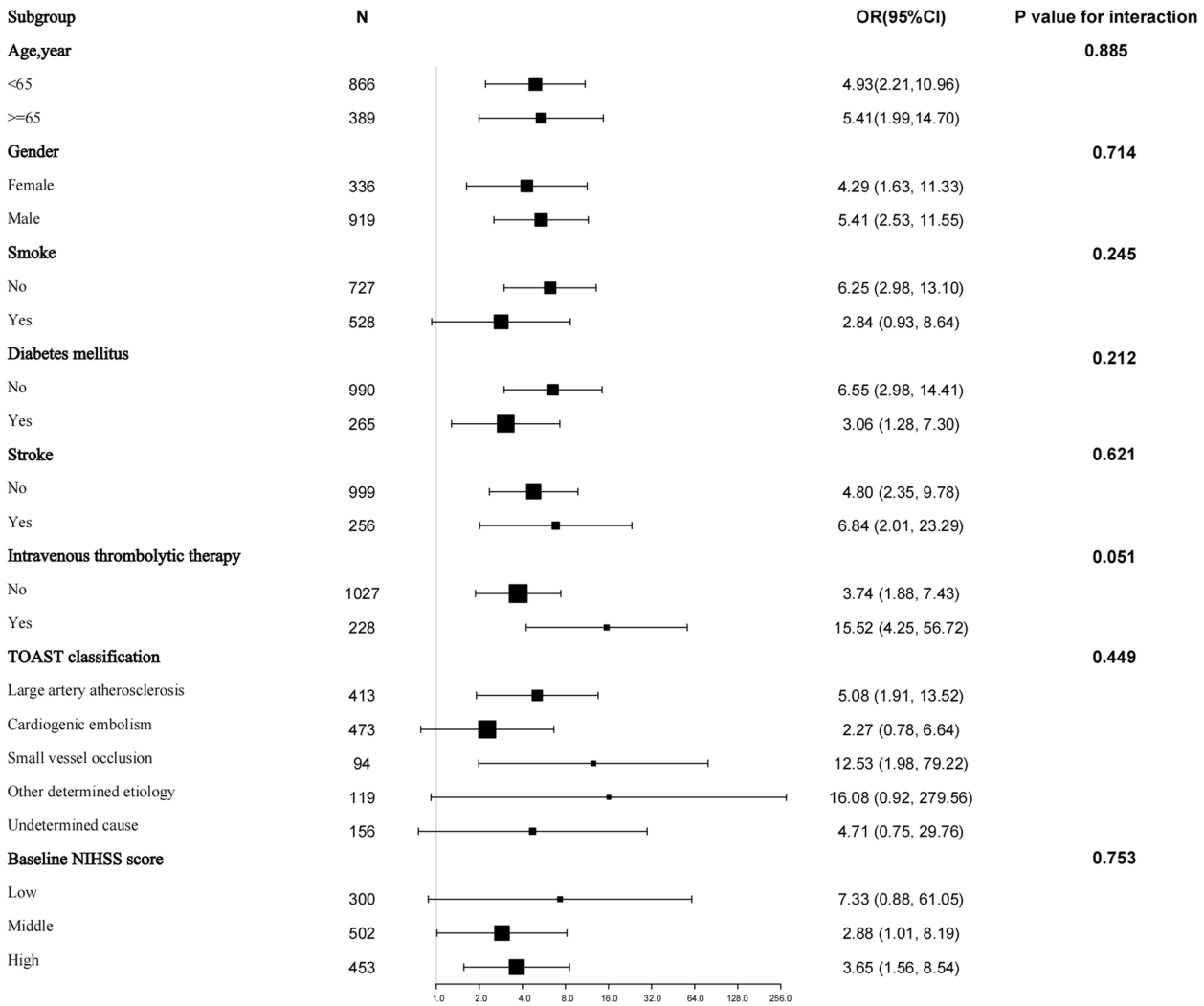

Fig. 2 Subgroup analyses of poor functional outcome

recurrence rate and prognosis in patients with IVT [26, 27]. This may be because acute hyperglycemic states may impede the fibrinolysis process and delay the reperfusion of ischemic penumbra [28].

To author's knowledge, this was the first study to investigate the association between SHR and 90-day poor functional outcome in China by stratified analysis and interaction of double hospital data from patients with AIS, and curve fitting of SHR to 90-day poor functional outcome. This study has some limitations. Firstly, since this study was a respective cohort, we excluded patients without admission glucose, HbA1C values, and 90-day mRS from this study, there may exist selection bias. Secondly, the participants included in this study were patients with AIS in China, considering potential differences in cultural and social conditions, one should be cautious when generalizing this findings to patients with AIS in other countries. Finally, this study only collected the SHR of patients at admission, and did not follow up the change of SHR. Future studies could conduct long-term follow-up of patients with SHR, and dynamically observe the impact of SHR on the functional outcomes of patients. It is necessary to be a large sample multicenter prospective longitudinal study to achieve generalizability.

In conclusion, the SHR, as measured by the glucose/HbA1C, increase was associated with increased odds of achieving a poor functional outcome in patients with AIS at 90 days. Therefore, early identification of SHR is essential to improve patient outcomes. However, further randomized controlled trials are needed to confirm the efficacy of SHR based glycemic control in improving patients.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12883-024-03795-w.

Supplementary Material 1

Authors' contributions

Xiao and Gao wrote the main manuscript text and Hu, Cao, Teng prepared figures 1-3. Xie responsible for paper guidance and article proofreading, All authors reviewed the manuscript.

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Availability of data and materials

The data of this study were collected from two hospitals in the Shenzhen area.

Declarations

Ethics approval and consent to participate

This study was approved by the ethics committees of two hospitals, the ethical batch number is [Ethics Approval No. 2022 No. (150)], and (Ethics Approval No. 2022ECPJ161).Informed consent has been obtained from the participants, their parents and legally authorized representatives in this study.

Consent for publication

Accepted for publication.

Competing interests

All authors in this study declare that they have no competing interests.

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