Research Square

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Cholesterol, high-density lipoprotein, and glucose index and stroke risk in elderly hypertensive patients in China: a prospective cohort study

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Abstract

Objective

The relationship between the Cholesterol, High-density lipoprotein, and Glucose (CHG) index and stroke has been investigated in only a limited number of studies, and the findings remain inconsistent. This prospective cohort study aims to provide further evidence on the association between the CHG index and stroke risk in patients with hypertension.

Methods

A total of 6725 elderly subjects (≥ 60 years) were enrolled in this prospective cohort analysis. The CHG index was calculated, and incident stroke was identified as the outcome. Cox proportional hazards models were employed to examine the association between the CHG index and stroke risk. Restricted cubic splines (RCS) were used to evaluate the dose-response relationship.

Results

Over a mean follow-up of 3.92 (0.58) years, 319 (4.7%) patients developed new-onset stroke. After multivariate adjustment, each 1-unit increase of CHG index was associated with a 268% higher risk of stroke (HR = 3.68, 95% CI: 2.03, 6.65). Compared with participants in the lowest quartile (Q1) of the CHG index, those in Q2, Q3, and Q4 had fully adjusted HRs (95% CI) for stroke of 1.89 (1.35, 2.65), 2.20 (1.52, 3.18), and 3.11 (2.03, 4.76), respectively. RCS analysis revealed a positive linear association between the CHG index and stroke risk, and this association remained consistent across subgroups, with no significant interactions.

Conclusions

An elevated CHG index is significantly and linearly associated with increased stroke risk in hypertensive patients. The CHG index may serve as a useful early indicator for stroke risk assessment in this population.

Trial registration

: the China Hypertension Registry Study (registration number: ChiCTR1800017274; registered on July 20, 2018; available at http://www.chictr.org.cn/).

Introduction

Stroke is a leading cause of death and disability in adults, remains a huge disease burden worldwide and in China[1–2]. Hypertension, as the most important risk factor for stroke in China, has a large patient population, approaching 300 million[3]. Compared with people with normal blood pressure, the risk of stroke in patients with hypertension is approximately 2.5 to 3.5 times higher[4]. This trajectory presents important challenges to public health and healthcare resources[5–6]. Implementing primary prevention strategies can alleviate patient suffering and reduce economic burdens[7]; therefore, identifying biomarkers for stroke risk stratification is crucial.

Studies have confirmed that lipid metabolism disorders significantly increase the risk of cardiovascular and cerebrovascular events in individuals[8–9]. Elevated total cholesterol (TC) levels or decreased high-density lipoprotein cholesterol (HDL-C) levels are independently associated with a significant increase in stroke incidence. Additionally, the ratio of TC to HDL-C has been demonstrated to be closely linked to the risk of cardiovascular events and stroke[10–12]. Abnormal glucose metabolism is considered a common risk factor for stroke and metabolic syndrome[13]. Multiple studies have shown that higher fasting plasma glucose (FPG) levels are positively correlated with stroke risk[14]. The above evidence suggests single biomarkers, such as fasting blood glucose (FBG) and blood cholesterol, can currently predict the stroke risk, the integration of multiple biomarkers, including TC, HDL-C, and FPG, may provide a more comprehensive assessment[15–16].

A novel index of CHG index, composed of TC, HDL-C, and FPG, has been proposed as a biomarker for metabolic disorders[17]. Studies have demonstrated that the CHG index holds significant value in predicting the risk of diabetes mellitus and its complications, as well as in assessing cardiovascular events[17–18]. However, very few studies currently evaluates the association between CHG and stroke risk, and the conclusions are inconsistent[19–20]. Therefore, to fill the gap with definitive evidences, we conducted a prospective cohort study to assess the relationship between CHG and stroke risk in hypertensive patients.

Methods

Study participants

This study was a subset of the China Hypertension Registry Study (registration number: ChiCTR1800017274; registered on July 20, 2018; available at http://www.chictr.org.cn/), a real-world, prospective, and observational study performed in Wuyuan county of China, from March to August 2018. The follow-up was completed from June to August in 2022. The methodology for data acquisition, and inclusion and exclusion criteria had been described in previous studies[21–22]. This study adhered to the Declaration of Helsinki and was approved by the Ethics Committee of the Second Affiliated Hospital of Nanchang University (No. 2018019) and the Institute of Biomedical Sciences, Anhui Medical University (No. CH1059). Written informed consent was obtained from all enrolled participants.

From the initial cohort of 14234 hypertensive patients recruited at baseline, we excluded individuals with a history of stroke (n = 983), a history of diabetes or use of antidiabetic medications (n = 2401), a history of dyslipidemia or use of lipid-lowering drugs (n = 1252), missing data on TG and Hcy (n = 5), ≥60 years old (n = 2866) and those lost to follow-up (n = 2). Ultimately, a total of 6725 participants were included in the final analysis. The detailed process of participant selection is illustrated in Fig. 1.

Data collection and variable definition

Comprehensive baseline information was gathered by trained interviewers through standardized questionnaires physical examinations, and laboratory measurements. Data collection encompassed four major domains: (1) demographic and lifestyle characteristics (gender, age, current smoking and current drinking[22]); (2) physical assessments, such as height, weight, systolic blood pressure (SBP) ,diastolic blood pressure (DBP), pulse; (3) medical history, including hypertension, diabetes, dyslipidemia, atrial fibrillation (AF), coronary heart disease (CHD), and relevant medication use; and (4) biochemical indicators, specifically fasting blood glucose (FBG), homocysteine (Hcy), total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), total protein (Tp), albumin (Alb), aspartate minotransferase (AST), alanine aminotransferase (ALT). The body mass index (BMI) was calculated as weight (kg)/height (m^2). The estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaboration equation [23]. The formula for calculating the CHG index was Ln [TC (mg/dL) × FBG (mg/dL) / 2 × HDL-C (mg/dL)][17]. Blood pressure was measured on the right arm positioned at the heart level using an electronic sphygmomanometer (Omron HBP-1300; OMRON, Japan) after a 5-min rest, with a 30-s interval between measurements, and three measurements averaged as values for the analysis.

Assessment of incident stroke

The primary outcome was defined as the first occurrence of a stroke event, with subarachnoid hemorrhage and silent strokes excluded. Stroke events were identified based on self-reported physician diagnoses and confirmed through a rigorous, multistep protocol established in the literature[24–25]. Although recurrent strokes were documented, they were not considered primary outcomes. The follow-up period extended from the date of baseline survey completion to August 15, 2022.

Statistical analysis

Participants were categorized into quartiles based on CHG or two groups according to the occurrence of stroke event. Continuous variables were presented as mean ± standard deviation (SD) for normal distribution. Categorical variables were expressed as frequencies (N) and percentages (%). Comparisons utilized t test, one-way ANOVA, or Chi-squared test as appropriate. Cox proportional hazards models estimated hazard ratios (HRs) and 95% confidence intervals (CIs) through three sequential models. Covariates adjusted in Cox regression were selected based on clinical relevance, prior literature, and

changes in effect estimates > 10%. The proportional hazards assumption was verified using Schoenfeld residual tests, which demonstrated no violations, indicating that the assumption of proportionality was satisfied. Multiple collinearity screening between covariates showed that all variance inflation factors (VIF) were less than 5. The dose-response relationship was examined using restricted cubic splines (RCS) analysis, in which the reference value (HR = 1.00) was set at the median. Stratified analyses and interaction tests were conducted to identify potential effect modifiers influencing the association between the CHG index and stroke risk.

All statistical analyses were performed using R software (http://www.r-project.org) and EmpowerStats (http://www.empowerstats.com). A two-sided p-value < 0.05 was considered statistically significant.

Results

Participant characteristics

The basic characteristics of 6725 participants stratified by the CHG index quartiles were shown in Table 1. The overall mean (SD) age of the study cohort was 69.0 (6.3) years old, with males accounting for 48.8% of the participants. During an average follow-up duration of 3.92 (0.58) years, 319 patients (4.7%) experienced stroke events. Participants with higher CHG index were more likely to be younger, females, and current smokers, had higher BMI, DBP, pulse, TG, LDL-C, Tp, Alb, and ALT, and lower AST, eGFR, and proportion of current drinking (all $P < 0.05$). There were no significant differences in variables such as DBP, Hcy, CHD and AF among the four quartiles (all P > 0.05).

Table 2 revealed that the patients in the stroke group exhibited the following characteristics: older age, increased SBP, pulse and Hcy, higher rates of males, current smoking, CHD and AF, lower BMI, TG, Tp, Alb, ALT and eGFR (all $P < 0.05$). There were no significant differences between the two groups in terms of other variables, such as DBP, LDL-C, AST, and current drinking (all $P > 0.05$).

Table 1
Baseline characteristics of participants stratified by the CHG index quartiles

Characteristics	Total	Quartiles of CHG index				*P* value
		Q1 (3.80–4.90)	Q2 (4.91–5.09)	Q3 (5.10–5.28)	Q4 (5.29–5.99)	
N	6725	1774	1716	1649	1586	
Male, n (%)	3285 (48.8)	1085 (61.2)	821 (47.8)	720 (43.7)	659 (41.6)	< 0.001
Age, n (%)	69.0 ± 6.3	69.7 ± 6.4	69.1 ± 6.4	68.8 ± 6.1	68.4 ± 6.1	< 0.001
BMI, kg/m^2	22.6 ± 3.3	21.1 ± 3.2	22.3 ± 3.2	23.1 ± 3.1	24.0 ± 3.2	< 0.001
SBP, mmHg	150.0 ± 18.3	149.4 ± 18.7	150.1 ± 18.1	150.3 ± 17.8	150.3 ± 18.7	0.392
DBP, mmHg	86.9 ± 10.6	86.3 ± 10.8	86.9 ± 10.6	86.9 ± 10.2	87.4 ± 10.6	0.037
Pulse, bpm	75.3 ± 14.3	73.8 ± 14.9	74.6 ± 14.0	76.1 ± 13.9	76.8 ± 14.2	< 0.001
Hcy, μmol/L	18.6 ± 11.3	18.6 ± 10.9	18.4 ± 10.8	18.7 ± 11.8	18.8 ± 11.6	0.851
TG, mmol/L	1.5 ± 0.9	1.0 ± 0.4	1.3 ± 0.6	1.6 ± 0.7	2.3 ± 1.3	< 0.001
LDL-C, mmol/L	2.9 ± 0.8	2.4 ± 0.6	2.8 ± 0.6	3.1 ± 0.7	3.4 ± 0.8	< 0.001
Tp, g/L	73.5 ± 6.7	72.0 ± 7.2	73.2 ± 6.6	74.0 ± 6.3	75.2 ± 6.3	< 0.001
Alb, g/L	46.0 ± 3.9	45.1 ± 4.2	46.0 ± 3.9	46.3 ± 3.8	46.8 ± 3.6	< 0.001
AST, U/L	26.4 ± 12.3	27.6 ± 13.7	25.7 ± 9.1	26.1 ± 14.4	26.0 ± 11.2	< 0.001
ALT, U/L	17.8 ± 12.3	16.4 ± 9.8	16.9 ± 9.8	18.3 ± 14.8	19.8 ± 13.9	< 0.001
eGFR, mL/min/1.73 m^2	84.4 ± 18.9	84.8 ± 19.9	85.5 ± 18.1	84.9 ± 18.5	82.3 ± 19.1	< 0.001
Current smoking, n (%)	1982 (29.5)	682 (38.4)	506 (29.5)	392 (23.8)	402 (25.3)	< 0.001
Current drinking, n (%)	1574 (23.4)	592 (33.4)	389 (22.7)	319 (19.3)	274 (17.3)	< 0.001

Characteristics	Total	Quartiles of CHG index				*P* value
		Q1 (3.80–4.90)	Q2 (4.91–5.09)	Q3 (5.10–5.28)	Q4 (5.29–5.99)	
AF, n (%)	205 (3.0)	62 (3.5)	52 (3.0)	50 (3.0)	41 (2.6)	0.503
CHD, n (%)	333 (5.0)	91 (5.1)	79 (4.6)	73 (4.4)	90 (5.7)	0.349

Data are presented as mean ± SD, or n (%)

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Hcy, homocysteine; TG, triglycerides; LDL-C, low-density lipoprotein cholesterol; Tp, total protein; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; eGFR, estimated glomerular filtration rate; AF, atrial fibrillation; CHD, coronary heart disease.

Table 2
Baseline characteristics of participants with and without stroke

Characteristics	Total	Non-stroke	Stroke	*P* value
N	6725	6406	319	
Male, n (%)	3285 (48.8)	3079 (48.1)	206 (64.6)	< 0.001
Age, n (%)	69.0 ± 6.3	68.9 ± 6.2	71.3 ± 7.0	< 0.001
BMI, kg/m^2	22.6 ± 3.3	22.6 ± 3.3	21.6 ± 3.2	< 0.001
SBP, mmHg	150.0 ± 18.3	149.9 ± 18.2	152.8 ± 19.8	0.005
DBP, mmHg	86.9 ± 10.6	86.8 ± 10.5	87.5 ± 11.1	0.258
Pulse, bpm	75.3 ± 14.3	75.2 ± 14.2	77.2 ± 16.7	0.015
Hcy, μmol/L	18.6 ± 11.3	18.4 ± 11.0	22.0 ± 15.9	< 0.001
TG, mmol/L	1.5 ± 0.9	1.5 ± 0.9	1.4 ± 0.9	0.030
LDL-C, mmol/L	2.9 ± 0.8	2.9 ± 0.8	2.9 ± 0.9	0.883
Tp, g/L	73.5 ± 6.7	73.6 ± 6.7	72.8 ± 6.8	0.036
Alb, g/L	46.0 ± 3.9	46.1 ± 3.9	45.3 ± 4.0	< 0.001
AST, U/L	26.4 ± 12.3	26.4 ± 12.1	26.2 ± 15.8	0.846
ALT, U/L	17.8 ± 12.3	17.9 ± 12.3	16.5 ± 10.6	0.003
eGFR, mL/min/1.73 m^2	84.4 ± 18.9	84.6 ± 18.9	81.2 ± 18.6	< 0.001
Current smoking, n (%)	1982 (29.5)	1854 (28.9)	128 (40.1)	< 0.001
Current drinking, n (%)	1574 (23.4)	1487 (23.2)	87 (27.3)	0.095
AF, n (%)	205 (3.0)	178 (2.8)	27 (8.5)	< 0.001
CHD, n (%)	333 (5.0)	314 (4.9)	19 (6.0)	0.043

Data are presented as mean ± SD, or n (%)

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Hcy, homocysteine; TG, triglycerides; LDL-C, low density lipoprotein cholesterol; Tp, total protein; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; eGFR, estimated glomerular filtration rate; AF, atrial fibrillation; CHD, coronary heart disease.

Association of CHG with stroke

For the CHG index, each 1-unit increase was associated with a 268% increase in stroke risk (HR = 3.68, 95% CI: 2.03, 6.65). When the CHG index was assessed as quartiles, in the fully adjusted model (Model 3), the HR (95% CI) values of CHG on stroke for patients in quartiles 2, 3, and 4 were 1.89 (1.35, 2.65), 2.20 (1.52, 3.18), and 3.11 (2.03, 4.76), respectively, compared with those in quartile 1. The quartiles of the CHG index (Q2, Q3, and Q4) demonstrated a progressively increasing trend in stroke risk (*P* for trend < 0.001) (Table 3). Additionally, the adjusted RCS curve in Fig. 2 revealed a significant linear association between the CHG index and stroke risk (*P* for overall < 0.001, and *P* for nonlinear = 1.000).

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Hcy, homocysteine; TG, triglycerides; LDL-C, low density lipoprotein cholesterol; Tp, total protein; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; eGFR, estimated glomerular filtration rate; AF, atrial fibrillation; CHD, coronary heart disease.

Table 3
Cox regression models for the association between the CHG index and stroke risk

CHG index	**Events (%)**	**Stroke, HR (95% CI)**		
		Model 1	**Model 2**	**Model 3**
Per 1 unit increment	319 (4.7)	1.40 (0.93, 2.10)	1.92 (1.28, 2.86)	3.68 (2.03, 6.65)
Quartile				
Q1 (3.80–4.90)	66 (3.7)	1.00	1.00	1.00
Q2 (4.91–5.09)	86 (5.0)	1.37 (0.99, 1.88)	1.54 (1.11, 2.12)	1.89 (1.35, 2.65)
Q3 (5.10–5.28)	82 (5.0)	1.37 (0.99, 1.90)	1.64 (1.19, 2.28)	2.20 (1.52, 3.18)
Q4 (5.29–5.99)	85 (5.4)	1.54 (1.11, 2.12)	1.94 (1.40, 2.69)	3.11 (2.03, 4.76)
P for trend		0.013	< 0.001	< 0.001
Model 1: adjusted for none				
Model 2: adjusted for sex, age				
Model 3: adjusted for sex, age, BMI, SBP, DBP, pulse, Hcy, TG, LDL-C, Tp, Alb, AST, ALT, eGFR, current smoking, current drinking, AF, CHD				

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Hcy, homocysteine; TG, triglycerides; LDL-C, low density lipoprotein cholesterol; Tp, total protein; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; eGFR, estimated glomerular filtration rate; AF, atrial fibrillation; CHD, coronary heart disease.

Subgroup analysis

Stratified analyses were performed to evaluate the effect of the CHG index (per 1 unit increment) on stroke risk in various subgroups (Fig. 3). No significant interaction existed in subgroups of gender (male vs. female), age (<65 vs. ≥ 65 years), BMI (<24 vs. ≥ 24 kg/m^2), current smoking (no vs. yes), current drinking (no vs. yes), CHD (no vs. yes), and AF (no vs. yes), and the consistent positive associations were observed across all subgroups (all P for interaction > 0.05).

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Hcy, homocysteine; TG, triglycerides; LDL-C, low density lipoprotein cholesterol; Tp, total protein; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; eGFR, estimated glomerular filtration rate; AF, atrial fibrillation; CHD, coronary heart disease.

Conclusion

An elevated CHG index is significantly correlated with an increased stroke risk, demonstrating a linear relationship. The CHG index may serve as an early marker to assess the risk of stroke in patients with hypertension.

Discussion

In this prospective cohort study, we investigated the association between the CHG index and the risk of incident stroke among Chinese adults with hypertension. Our findings demonstrated that a higher CHG index was significantly associated with an increased risk of stroke, and this association remained consistent across diverse subgroups. Furthermore, restricted cubic spline (RCS) analysis revealed a significant linear relationship between the CHG index and stroke risk.

As a novel composite biomarker integrating lipid and glucose metabolism disorders, the CHG index was first proposed in the MASHAD (Mashhad stroke and heart atherosclerotic disorder) study[17], and has gradually attracted attention in recent years. Multiple large-scale cohort studies based on China Health and Retire Tracking Survey (CHARLS) data have validated the association between CHG index and stroke risk. Focusing on the research population aged 45 years and above, Zeng et al.[19] found a linear positive correlation between the CHG index and stroke risk. Using quartile 1 group of the CHG index as the reference, the stroke risk in quartile 2 (HR = 1.19, 95% CI: 0.95–1.49), quartile 3 (HR = 1.38; 95% CI: 1.09–1.75), and quartile 4 (HR = 1.57, 95% CI: 1.18–2.10) groups showed a significant stepwise increase (P for trend = 0.001). In contrast, Wang et al.[20] observed a nonlinear relationship, with an inflection point of CHG at 4.556. On the left side of that point, each unit increase in CHG was associated with a no significant increase of 12.4% in stroke risk (HR = 1.126, 95% CI: 0.691–1.835), while on the right side of the point, it was linked to a 58.0% increase in risk (HR = 1.580, 95% CI: 1.097–2.274). Ke et al.[26] conducted a study involving 6,836 patients with early-onset cardiovascular-kidney-metabolic (CKM) syndrome, demonstrating an approximately linear association between the CHG index and stroke risk. For every one standard deviation increase in the CHG index, the stroke risk increased by 28% (HR = 1.28, 95% CI: 1.17–1.40). Xiao et al.[27] evaluated the predictive value of the CHG index for stroke risk among

adults with varying glucose regulation. The results indicated that for each 1 standard deviation increase in the CHG index, the risk of stroke increased by 16% (HR = 1.16, 95% CI: 1.09–1.24). Qur results were consistent with these findings, collectively supporting the reliability of the CHG index as a predictor of stroke risk. However, previous studies predominantly focused on the general middle-aged and elderly hypertensive population or individuals with metabolic abnormalities, while our study specifically targeted hypertensive patients, a high-risk group for stroke.

Multiple underlying mechanisms may explain the association. First, the CHG index comprises three key metabolic markers—total cholesterol, high-density lipoprotein cholesterol (HDL-C), and fasting blood glucose—that synergistically contribute to vascular injury. Elevated total cholesterol facilitates lipid deposition within arterial walls, whereas reduced HDL-C compromises reverse cholesterol transport and diminishes its anti-inflammatory and antioxidant capacity, thereby accelerating atherosclerosis [28–29]. Concurrently, chronic hyperglycemia promotes endothelial dysfunction, oxidative stress, and glycation-induced vascular stiffening, collectively increasing cerebrovascular vulnerability[30–31]. Second, the CHG index serves as a reliable proxy for insulin resistance, a condition known to potentiate oxidative stress, systemic inflammation, endothelial dysfunction, reduced nitric oxide bioavailability, and prothrombotic alterations, all of which drive atherosclerosis and heighten stroke risk [32–35]. Third, individuals with insulin resistance frequently exhibit concomitant cardiovascular risk factors, including obesity, diabetes, and dyslipidemia, further amplifying stroke susceptibility[36–39]. In the present study, patients with a higher CHG index were more likely to be obese, as reflected by elevated BMI. Obesity-related systemic inflammation and dysregulated adipokine secretion may exacerbate dyslipidemia and insulin resistance, thereby establishing a positive feedback loop of metabolic disturbance that perpetuates vascular risk.

This study presents several notable strengths. First, it is the first to assess the association between the CHG index and stroke risk among hypertensive patients.

Given that hypertension frequently coexists with conditions such as insulin resistance and dyslipidemia, the cumulative burden of these comorbidities may amplify the metabolic dysregulation captured by the CHG index. To enhance the validity of the exposure assessment, we excluded individuals with pre-existing diabetes or dyslipidemia at baseline. Second, the prospective design and large sample size substantially strengthen the reliability of the findings. Third, subgroup analyses demonstrated consistent associations across various demographic and clinical subgroups, offering critical insights for the development of targeted intervention strategies.

Nevertheless, several limitations should be taken into consideration. Firstly, only the baseline CHG was calculated as the exposure factor, limiting the evaluation of temporal trend. Secondly, due to the lack of detailed clinical imaging data, some stroke events cannot be fully and accurately classified as either hemorrhagic or ischemic strokes. Therefore, the relationship between the CHG index and stroke risk in stroke subtypes was not analyzed. Thirdly, although multivariate adjustments were made, there might

still be residual confounding factors. Finally, since the study subjects were restricted to the Chinese hypertensive population, which limits the applicability of the findings to other populations.

Declarations

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Author contributions

C.Y. and W.Z. conducted the statistical analysis and wrote the manuscript. L.Z. and T.W. contributed to data collection and result interpretation. W.Z., H.B., and X.C. conceived and designed the study, and critically reviewed the manuscript. All authors read and approved the final manuscript.

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Data availability

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

Ethics approval and consent to participate

Ethical approval for this study was conducted by the Second Affiliated Hospital of Nanchang University (No. 2018019) and the Institute of Biomedical Sciences, Anhui Medical University (No. CH1059). The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. All participants were informed about the purpose of the study, assured of confidentiality, and provided written consent prior to participation. Participation was voluntary, and respondents could withdraw at any time without consequence.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests

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Figures

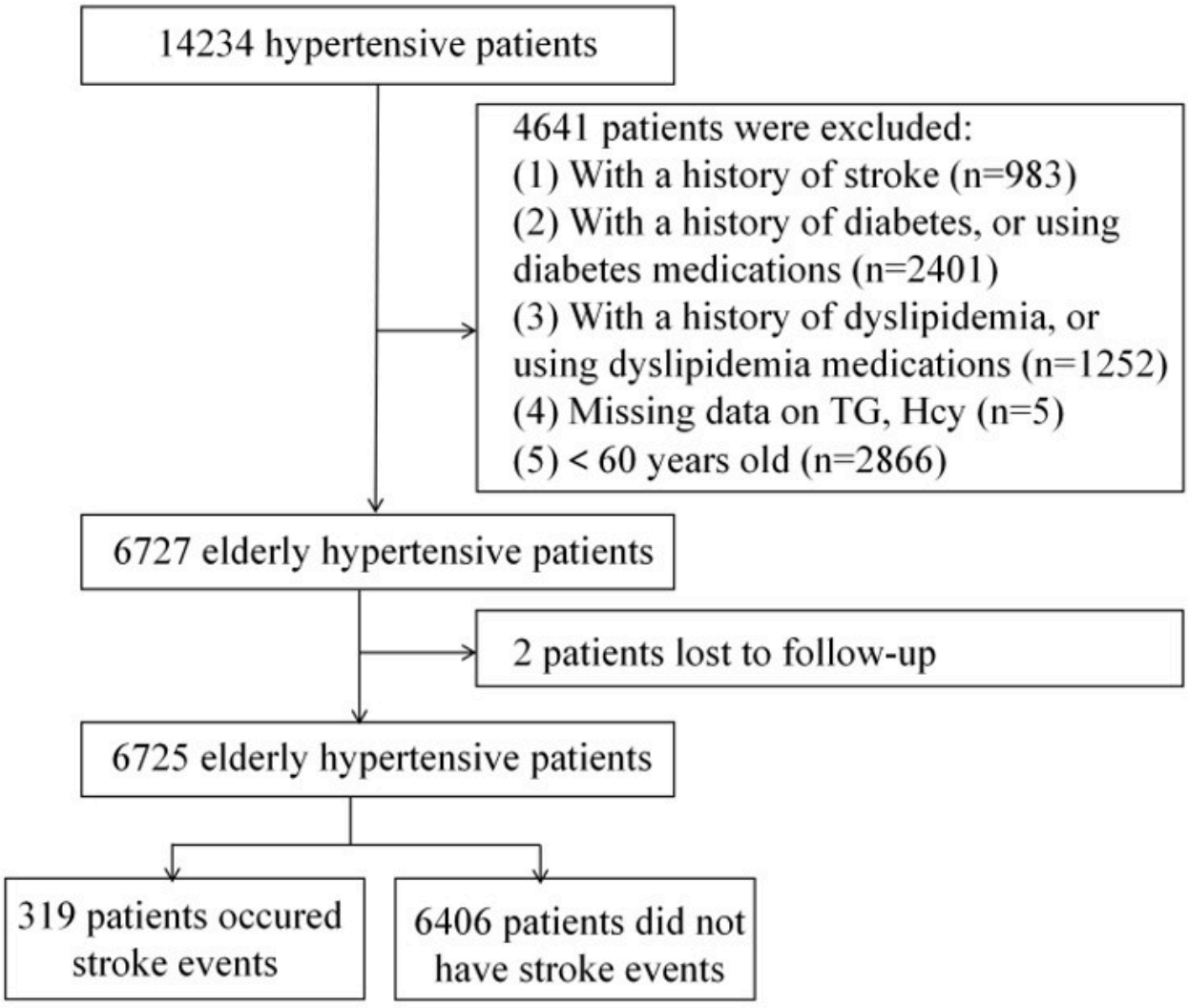


Figure 1

Flow chart of participants.

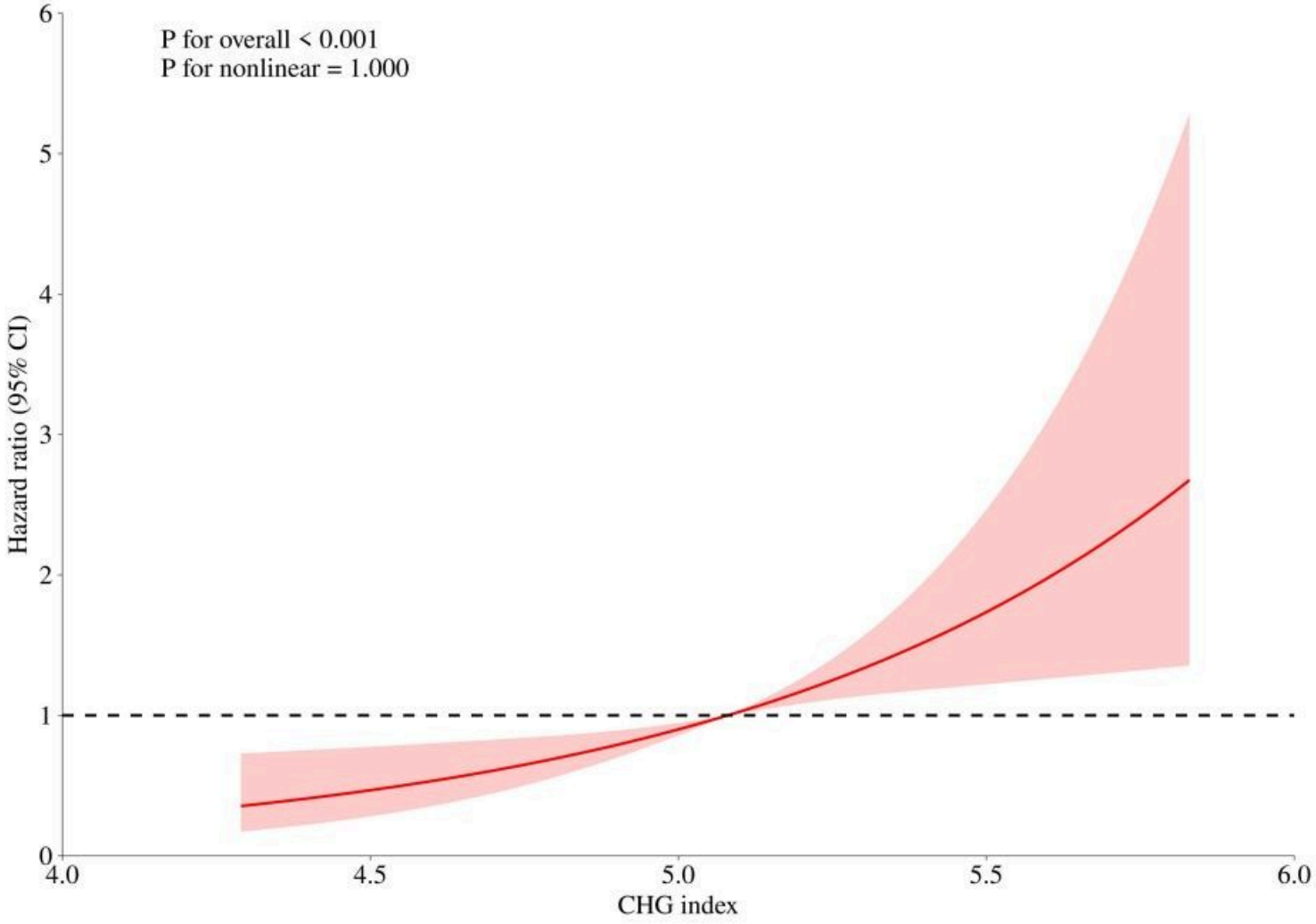


Figure 2

Dose-response relationship of the CHG index with stroke risk using restricted cubic splines. The model was adjusted for sex, age, BMI, SBP, DBP, pulse, Hcy, TG, LDL-C, Tp, Alb, AST, ALT, eGFR, current smoking, current drinking, AF, CHD.

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Hcy, homocysteine; TG, triglycerides; LDL-C, low density lipoprotein cholesterol; Tp, total protein; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; eGFR, estimated glomerular filtration rate; AF, atrial fibrillation; CHD, coronary heart disease.

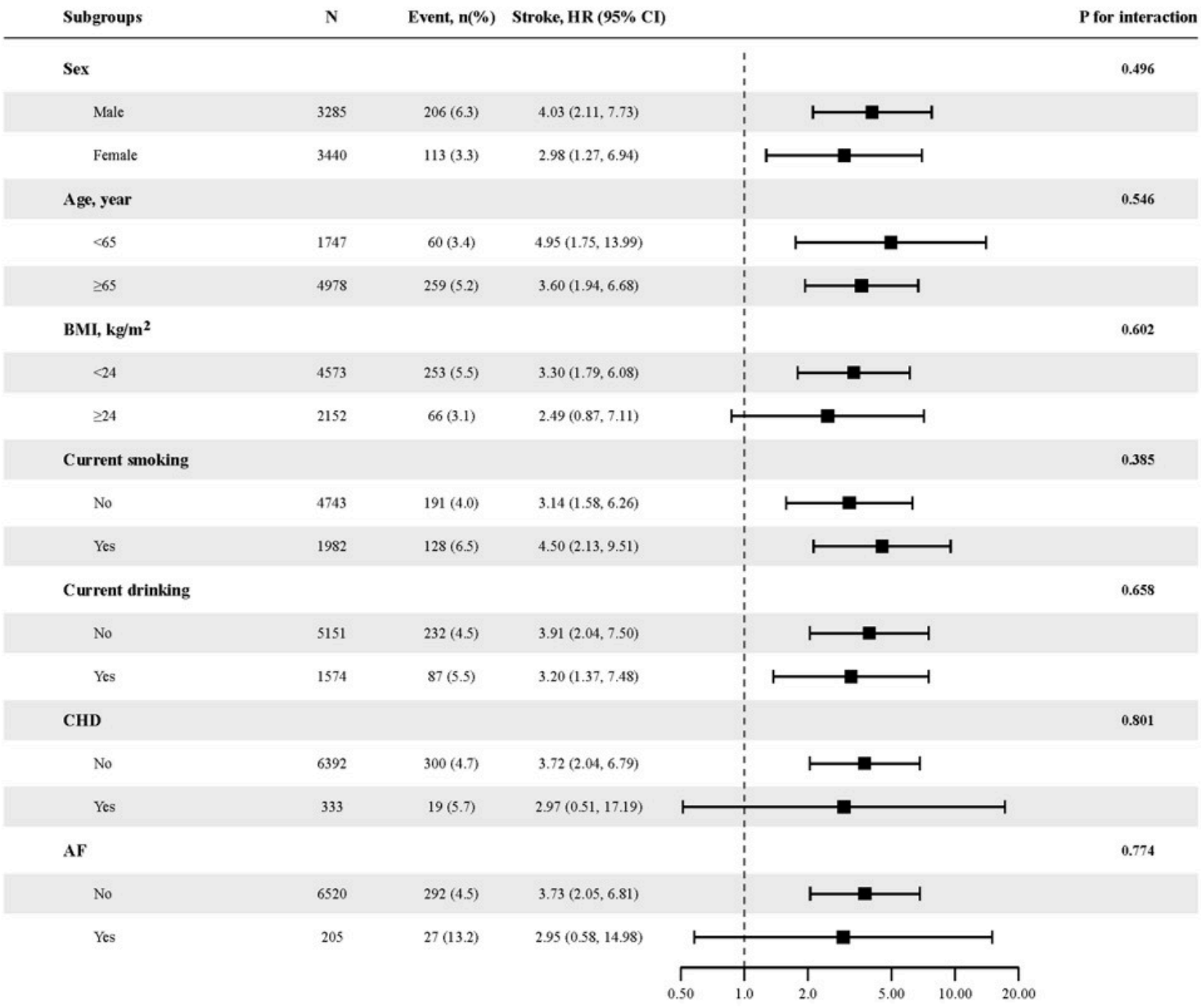


Figure 3

Subgroup analyses of the effect of the CHG index on stroke risk. Each subgroup analysis was adjusted, if not stratified, for sex, age, BMI, SBP, DBP, pulse, Hcy, TG, LDL-C, Tp, Alb, AST, ALT, eGFR, current smoking, current drinking, AF, CHD.

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Hcy, homocysteine; TG, triglycerides; LDL-C, low density lipoprotein cholesterol; Tp, total protein; Alb, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; eGFR, estimated glomerular filtration rate; AF, atrial fibrillation; CHD, coronary heart disease.