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ORIGINAL RESEARCH

Association Between Systemic Inflammation Response Index and Large Hemispheric Infarction: Development of a Predictive Diagnostic Model - A Retrospective Study

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Objective: Large hemispheric infarction (LHI) represents one of the most severe subtypes of ischemic stroke, associated with high rates of disability and mortality. This study aimed to examine the association between the systemic inflammation response index (SIRI) and LHI, identify independent risk factors, and develop a predictive model for clinical application.

Methods: A total of 152 patients diagnosed with LHI and admitted to Shaanxi Provincial People's Hospital between June 2020 and June 2023 were retrospectively selected based on defined inclusion and exclusion criteria. A control group comprising 153 healthy individuals from the same period was included for comparison. Clinical and laboratory data were collected, and statistical analyses were performed using SPSS version 26.0. Univariate and multivariate logistic regression analyses were conducted to determine independent risk factors. The predictive performance of these factors was evaluated using receiver operating characteristic curve analysis, and a nomogram-based predictive model was constructed.

Results: Multivariate logistic regression analysis identified a history of atrial fibrillation, coronary heart disease, prior stroke, elevated systolic blood pressure, increased fasting blood glucose (FBG), elevated homocysteine, and higher SIRI values as independent risk factors for LHI ($p < 0.05$). A nomogram predictive model based on these factors demonstrated satisfactory calibration and discriminatory capability.

Conclusion: SIRI has certain clinical value in predicting LHI. The developed nomogram-based predictive model incorporating SIRI exhibited robust predictive performance and may assist in guiding clinical decision-making.

Keywords: inflammation, large hemispheric infarction, nomogram, predictive model, risk factors, SIRI

Introduction

With China's aging population, the incidence of stroke has indicated a consistent upward trend. Globally, stroke remains the second leading cause of mortality.[1] Epidemiological data indicate that between 1990 and 2019, the number of patients affected by stroke increased by 85.0%, stroke-related deaths rose by 43.0%, and stroke-related disabilities grew by 32.0%.[2] In China, stroke has become the most common cause of death.[1,3] According to the World Health Organization clinical criteria, stroke is defined as the sudden onset of clinical signs of cerebral dysfunction—typically focal in nature—that persist for more than 24 hours or result in death.[4] Large hemispheric infarction (LHI) generally refers to total or partial anterior circulation infarction resulting from occlusion of the internal carotid artery or the proximal segment of the middle cerebral artery.[5] Common clinical manifestations include hemiplegia, severe hemisensory loss, global aphasia, gaze palsy, and head-eye deviation.[6] These neurological impairments substantially reduce quality of life, contribute to psychological distress among affected patients and their families, and impose significant economic burdens.

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LHI is influenced by a range of genetic predispositions and environmental exposures. Prior studies identified several major risk factors associated with the development of LHI, including advanced age, hypertension, diabetes mellitus, a history of atrial fibrillation, hyperlipidemia, frequent tobacco use, previous transient ischemic attacks or ischemic stroke, congestive heart failure, and coronary artery disease.[7,8] In recent years, significant attention has been directed toward the role of inflammation in the pathogenesis of stroke. Inflammatory mechanisms have been indicated to contribute to neuronal injury following AIS, and the presence of specific inflammatory biomarkers has demonstrated potential for predicting mortality risk and functional outcomes in affected patients.[9]

The systemic inflammation response index (SIRI), a novel inflammatory biomarker, has emerged as a more comprehensive indicator. It is calculated as the product of neutrophil and monocyte counts divided by lymphocyte count and reflects a broader spectrum of inflammatory activity.[10–12] SIRI offers advantages including ease of calculation, reproducibility, and low cost. Despite its potential, there is limited evidence available regarding the association between SIRI and large hemispheric infarction. The present study has to examine if SIRI functions as an independent risk factor for the development of LHI.

Furthermore, a nomogram-based predictive model was constructed to estimate the likelihood of LHI occurrence. This model is based on a fundamental statistical equation incorporating multiple independent variables. Each variable contributes to a cumulative score that is subsequently translated into a probability estimate for a specific clinical outcome. The nomogram format facilitates visual interpretation and clinical application, thereby enhancing its practical value. Accordingly, the present investigation preliminarily examined the association between SIRI and LHI during the acute phase and established a predictive model integrating relevant clinical risk factors to support targeted and individualized intervention strategies.

Materials and Methods

Study Participants and Grouping

Patients diagnosed with acute cerebral infarction and admitted to Shaanxi Provincial People's Hospital between June 2020 and June 2023 were retrospectively selected as study participants. From predefined inclusion and exclusion criteria, a total of 152 patients were assigned to the LHI group, while 153 healthy individuals from the same time period were included as the control group.

Inclusion and Exclusion Criteria

Inclusion Criteria

(1) Age: 18 years and above.

Specific to the LHI Group

(1) Diagnosis consistent with the Chinese Guidelines for the Diagnosis and Treatment of Acute Ischemic Stroke (2018).[13] (2) Time from symptom onset to admission≤48 hours. (3) Imaging findings consistent with LHI diagnosis.[14]

Specific to the Control Group

(1) No history of ischemic/hemorrhagic stroke, dementia, epilepsy, Parkinson's disease, liver or kidney disease, cancer, or autoimmune diseases. (2) No pregnancy or mental illness.

Exclusion Criteria

(1) Infection or inflammatory diseases within one week before admission. (2) Use of immunomodulatory medications within three months before admission, including glucocorticoids, antibiotics, biological agents, or monoclonal antibodies. (3) Presence of autoimmune diseases, hematologic disorders, history of malignant neoplasms, or severe cardiac, hepatic, renal, or other systemic diseases.

Specific to the LHI Group

(1) Diagnosis of cerebral hemorrhage, subarachnoid hemorrhage, hemorrhagic cerebral infarction, or posterior circulation infarction. (2) Receipt of thrombolysis, thrombectomy, or other endovascular interventions at the time of admission.

Specific to the Control Group

(1) History of ischemic/hemorrhagic stroke or significant stenosis of large vessels (eg,internal carotid artery).

Collection of General Clinical Data

Comprehensive general data were collected for all enrolled patients, including name, sex, age, medical history, body mass index (BMI), and personal history. The diagnostic criteria for cerebrovascular disease risk factors were defined as follows:

Hypertension: Diagnosed in accordance with the revised diagnostic criteria outlined in the *2018 Guidelines for the Prevention and Treatment of Hypertension*.[15]

Diabetes Mellitus: Diagnosed based on the criteria specified in the updated *2019 Guidelines for the Prevention and Treatment of Type 2 Diabetes Mellitus in China*.[16]

Heart Disease: Included conditions like atrial fibrillation and coronary atherosclerotic heart disease. Diagnosis was established by a qualified specialist using 24-hour dynamic electrocardiogram monitoring, coronary angiography, or a documented history of cardiac valve surgery or other relevant cardiac procedures.[17]

Hyperlipidemia: Diagnosed according to the *2016 Guidelines for the Prevention and Treatment of Dyslipidemia in Chinese Adults*, or based on a prior clinical diagnosis of hyperlipidemia.[18]

Collection of Laboratory Indicators

Fasting blood samples were collected in the morning on the day of admission or the following day by trained nursing staff. The laboratory assessments included complete blood count, liver and renal function tests, fasting blood glucose (FBG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), glycated hemoglobin (HbA1c), and homocysteine (HCY). All laboratory parameters were analyzed and reported by the central laboratory and radioimmunoassay center of the hospital.

Imaging Examinations

Cranial computed tomography and/or magnetic resonance imaging examinations—including T1-weighted imaging, T2-weighted imaging, fluid-attenuated inversion recovery, diffusion-weighted imaging, and magnetic resonance angiography—were performed within 48 hours of admission.

National Institute of Health Stroke Scale (NIHSS) Score

NIHSS scores at the time of admission were recorded for all patients. Scoring was independently conducted by one intermediate-level physician and one senior physician. In cases where discrepancies were observed between the two assessments, the final score was determined through consensus following discussion.

Statistical Analysis

Data analysis was conducted using SPSS version 26.0. After data aggregation, the proportion of missing data for all patients in each indicator was less than 1%. Missing values were imputed using the mean substitution method. Descriptive statistics were presented as follows: measurement data with a normal distribution were expressed as mean ± standard deviation ($\overline{X} \pm s$), while non-normally distributed measurement data were reported as median and interquartile range (Q1-Q3). Categorical variables were described as percentages (%).

Comparative analyses between two groups were conducted as follows: normally distributed measurement data were analyzed using independent samples *t*-tests, while the Mann–Whitney *U*-test was applied to non-normally distributed data. A *p*-value of less than 0.05 was considered indicative of statistical significance.

Variables indicating statistical significance in univariate analysis were included in binary logistic regression to identify independent risk factors for LHI, and odds ratios (ORs) with corresponding 95% confidence intervals (CIs) were calculated. A stepwise forward regression method based on the Akaike Information Criterion was applied to develop the diagnostic predictive model for LHI. R software version 4.3.2 was used to construct the nomogram-based predictive model.

Receiver operating characteristic (ROC) curves were generated to evaluate the predictive performance of individual indicators, and the Youden index was used to determine the optimal cutoff values. Calibration curves were plotted to assess the predictive accuracy and agreement of the constructed model.

Results

Description of General Data in the LHI Group and Control Group

From the predefined inclusion and exclusion criteria, a total of 152 patients diagnosed with LHI were included in the study group, while 153 healthy individuals were enrolled as the control group. Within the LHI group, 81 patients (53.3%) were male and 71 (46.7%) were female, with a median age of 70.50 years (interquartile range: 60.25–81.00), and an age range of 31 to 89 years. In the control group, 57 patients (37.3%) were male and 96 (62.7%) were female, with a median age of 68.00 years (interquartile range: 61.00–75.00), and an age range of 46 to 88 years.

Baseline Comparison Between the LHI Group, Control Group and Analysis of Risk Factors for Patients with LHI

Comparison of General Clinical Data Between the LHI Group and the Control Group

Independent samples *t*-tests and non-parametric tests were used to assess differences in general clinical characteristics between the LHI group and the control group. Categorical variables were analyzed using chi-square tests. The results are presented in Table 1.

Statistically significant differences were observed between the two groups in terms of sex distribution, systolic blood pressure, diastolic blood pressure, BMI, and medical history of hypertension, diabetes mellitus, coronary heart disease,

Table 1 Comparison of Clinical and Laboratory Data Between the LHI Group and the Control Group

Item	LHI Group (n = 152)	Control Group (n = 153)	$\chi 2$/t/Z value	*p* value
Age (years old)	70.50 (60.25, 81.00)	68.00 (61.00, 75.00)	−1.584	0.113
Gender (Male/Female)	81/71	57/96	7.913	<0.01*
Hypertension (n, %)	104 (68.40%)	55 (35.90%)	33.999	<0.01*
Diabetes (n, %)	49 (32.20%)	21 (13.70%)	14.776	<0.01*
Coronary heart disease (n, %)	47 (30.90%)	10 (6.50%)	29.837	<0.01*
Atrial fibrillation (n, %)	34 (22.40%)	2 (1.30%)	32.490	<0.01*
Stroke history (n, %)	40 (26.30%)	13 (8.5%)	16.863	<0.01*
Smoking history (n, %)	19 (12.50%)	17 (11.10%)	0.141	0.70
Alcohol consumption (n, %)	6 (3.90%)	6 (3.90%)	0.001	0.99
BMI (kg/m^2)	22.50 (20.50, 25.3)	23.90 (22.25, 25.35)	−3.650	<0.01*
Systolic blood pressure on admission (mmHg)	144.00 (130.00,160.00)	128.00 (120.00, 137.50)	−6.801	<0.01*
Diastolic blood pressure on admission (mmHg)	83.00 (76.00, 90.00)	77.00 (70.00, 83.00)	−4.462	<0.01*
Fasting blood glucose (mmol/L)	6.26 (5.17,8.42)	4.90 (4.35,5.70)	−6.674	<0.01*
ALT (U/L)	15.00 (11.00,21.00)	19.00 (12.00,27.00)	−2.738	0.006*
AST (U/L)	22.00 (18.00,23.00)	18.00 (15.00,23.00)	−5.251	<0.01*
Total protein (g/L)	64.65 (60.55,69.35)	65.50 (62.05,69.35)	−0.922	0.357
Albumin (g/L)	36.60 (34.13,39.68)	38.50 (36.80,40.65)	−4.301	<0.01*
Total cholesterol (mmol/L)	3.99±1.09	4.66±0.98	4.792	<0.01*
HDL-C (mmol/L)	1.06 (0.88,1.24)	1.15 (0.99,1.35)	−3.264	<0.01*
LDL-C (mmol/L)	2.26 (1.74,3.02)	2.51 (2.00,3.17)	−2.562	0.010*
Triglycerides (mmol/L)	1.23 (0.95,1.74)	1.36 (1.05,1.93)	−3.272	<0.01*
Homocysteine (umol/L)	24.40 (13.75,30.20)	13.50 (11.00,16.31)	−6.832	<0.01*
Glycated hemoglobin (%)	6.05 (5.60,6.80)	5.70 (5.40,6.10)	−4.245	<0.01*
Creatinine (umol/L)	61.00 (50.55,75.53)	54.00 (46.26,62.22)	−3.974	<0.01*
Serum potassium (g)	3.79 (3.50,4.10)	3.90 (3.70,4.10)	−1.368	0.171

Note: *p < 0.05 indicates statistical significance.

Table 2 Multivariate Logistic Regression Analysis of Variables Related to Acute LHI

Item	Regression Coefficient	Standard Error	Wald	P	OR	95% CI	
						Lower Limit	Upper Limit
Atrial fibrillation history	2.594	0.844	9.441	0.002*	13.381	2.558	69.992
Coronary heart disease history	1.507	0.588	6.578	0.010*	4.513	1.427	14.277
Stroke history	1.390	0.508	7.478	0.006*	4.016	1.483	10.879
Systolic blood pressure	0.028	0.009	9.033	0.003*	1.028	1.010	1.047
Fasting blood glucose	0.236	0.078	9.134	0.003*	1.266	1.086	1.474
Homocysteine	0.063	0.015	17.194	<0.001*	1.065	1.034	1.097
SIRI	1.164	0.205	32.195	<0.001*	3.204	2.143	4.790

Note: *p < 0.05 indicates statistical significance.

atrial fibrillation, and prior stroke (all $p < 0.05$). No significant differences were found in age, smoking history, or alcohol consumption ($p > 0.05$).

Regarding laboratory parameters, significant differences were identified in FBG, alanine aminotransferase, aspartate aminotransferase, TC, HDL-C, LDL-C, TG, HCY, HbA1c, creatinine, and SIRI ($p < 0.05$). No statistically significant difference was observed in serum potassium levels ($p > 0.05$).

Multivariate Logistic Regression Analysis of Variables Related to Acute LHI

Variables demonstrating statistically significant differences ($p < 0.05$) in the univariate analysis were included in the multivariate logistic regression analysis. To reduce the influence of potential confounders, sex was excluded from the model. Among the lipid-related variables, TC and LDL-C exhibited a strong correlation; therefore, LDL-C, HDL-C, and TG were selected for inclusion in the final model.

The selected variables were entered as independent variables, with the diagnosis of LHI defined as the dependent variable. Binary multivariate logistic regression analysis was performed using a stepwise forward selection method to identify independent risk factors associated with acute LHI.

As presented in Table 2, a history of atrial fibrillation, coronary heart disease, stroke, elevated systolic blood pressure, FBG, HCY, and SIRI were identified as independent risk factors for LHI. The Hosmer–Lemeshow goodness-of-fit test for the final logistic regression model yielded a chi-square value of 10.797 and a p-value of 0.213 (> 0.05), indicating an acceptable level of model calibration.

ROC Curves for Individual Independent Risk Factors

As presented in Figure 1 and detailed in Table 3, ROC curve analysis was conducted for each independent risk factor. When ranked by AUC values, the order was as follows: SIRI > HCY > systolic blood pressure > FBG > history of coronary heart disease > history of atrial fibrillation > history of stroke. These findings indicate that while each individual indicator provides some degree of predictive value, their discriminative performance is limited when used in isolation. Consequently, a nomogram-based predictive model was constructed by integrating the identified risk factors to enhance diagnostic accuracy.

Establishment of the Nomogram Predictive Model

Establishment of the Nomogram Predictive Model

The independent risk factors for LHI, as identified through multivariate logistic regression analysis, were incorporated into the construction of a nomogram-based predictive model, as presented in Figure 2. For each variable included in the model, a vertical line was drawn to the corresponding point axis to determine the individual score. The total score, obtained by summing the individual factor scores, was then mapped to a risk scale to estimate the probability of LHI. This nomogram provides a straightforward, visual, and efficient method for quantifying the risk of LHI in clinical settings.

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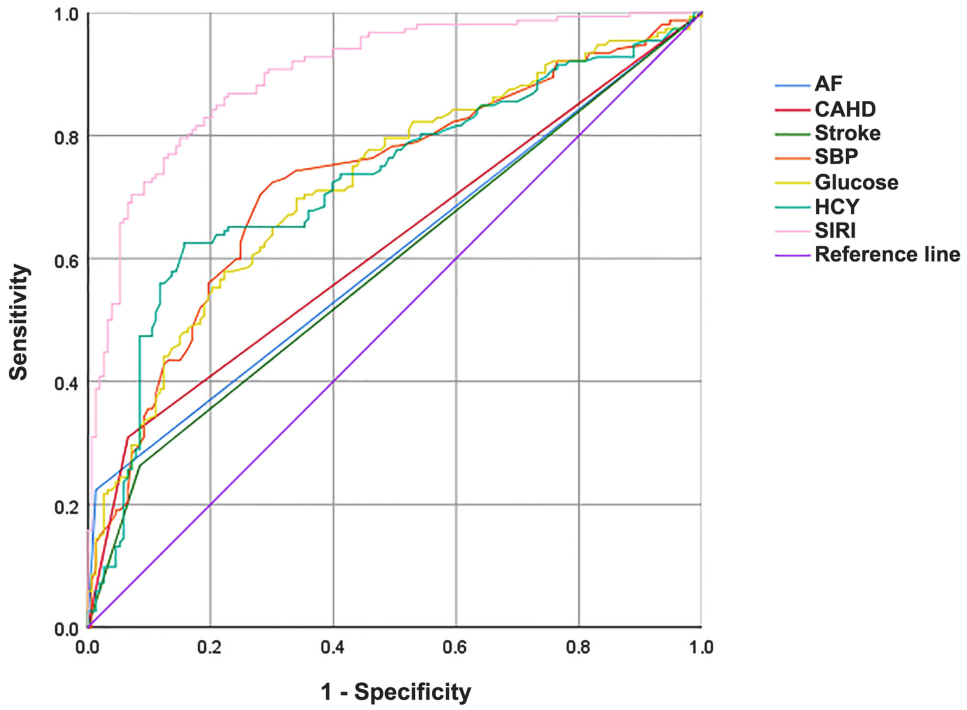

Figure 1 ROC curve analysis of individual independent risk factors.
Abbreviations: HCY, Homocysteine; SIRI, Systemic inflammatory response index; ROC, Receiver Operating Characteristic Curve; AF, Atrial Fibrillation; CAHD, Coronary Atherosclerotic Heart Disease; SBP, Systolic Blood Pressure.

Evaluation of the Predictive Model

The area under the ROC curve was used to assess the discriminative ability of the predictive model. The ROC analysis yielded an AUC of 0.950 (95% CI: 0.927–0.973), with a maximum Youden index of 0.790 and an optimal cutoff value of 0.4. At this threshold, the model demonstrated a sensitivity of 90.1% and a specificity of 88.9%, indicating excellent discriminative performance in distinguishing patients with LHI, as presented in Figure 3.

Internal validation of the nomogram predictive model was conducted using the bootstrap method, in which resampling was conducted from the original study group. A calibration curve was generated using R software to assess the agreement between predicted and observed probabilities (Figure 4). In this curve, the x-axis represents the predicted probability of LHI, and the y-axis represents the actual observed probability. The 45° diagonal line denotes perfect agreement between prediction and outcome. The short-dashed line represents the performance of the model in the entire group, while the solid line indicates the bias-corrected performance of the nomogram following bootstrap adjustment. The close alignment of these curves with the ideal reference line demonstrates good calibration and suggests that the nomogram model reliably estimates the probability of acute LHI.

Table 3 ROC Curve Results of Independent Risk Factors Identified by Multivariate Logistic Regression Analysis

Item	AUC	Cut-off value	95% CI	Sensitivity (%)	Specificity (%)	p value
Atrial fibrillation history	0.605	0.5	0.542–0.669	22.4%	98.7%	0.001*
Coronary heart disease history	0.622	0.5	0.559–0.685	30.9%	93.5%	<0.001*
Stroke history	0.589	0.5	0.525–0.653	26.3%	91.5%	0.007*
Systolic blood pressure	0.725	134	0.668–0.783	72.4%	69.9%	<0.001*
Fasting blood glucose	0.721	5.37	0.664–0.778	69.7%	66%	<0.001*
Homocysteine	0.726	18.05	0.668–0.784	62.5%	84.3%	<0.001*
SIRI	0.901	1.39	0.867–0.935	79.6%	85%	<0.001*

Note: *p < 0.05 indicates statistical significance.

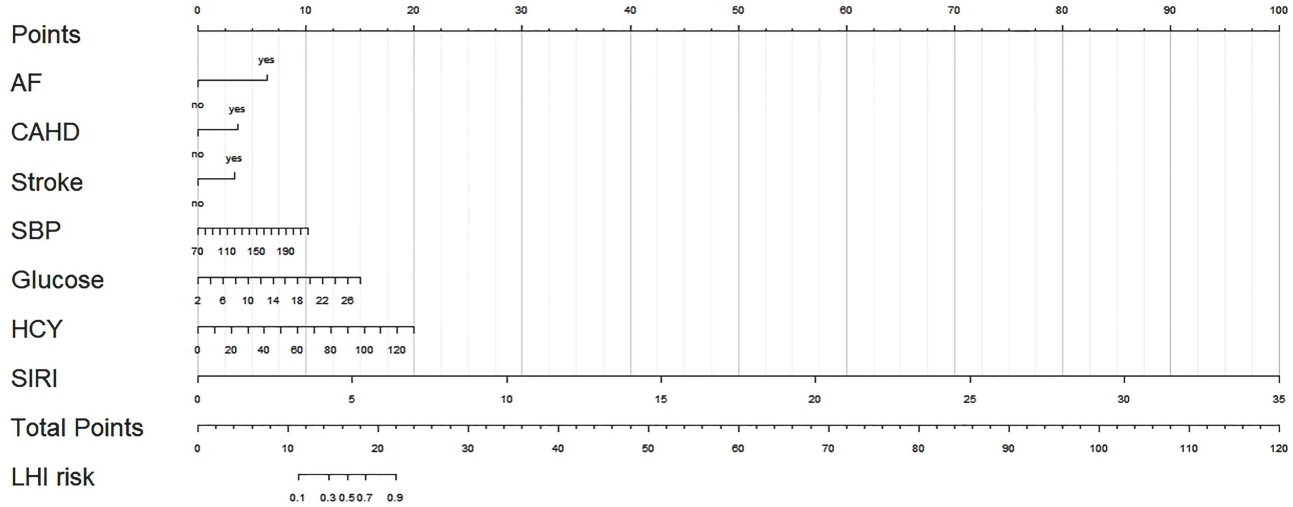

Figure 2 Nomogram predictive model for patients with acute LHI.
Abbreviations: LHI, Large Hemispheric Infarction; ROC, Receiver Operating Characteristic Curve; AF, Atrial Fibrillation; CAHD, Coronary Atherosclerotic Heart Disease; SBP, Systolic Blood Pressure.

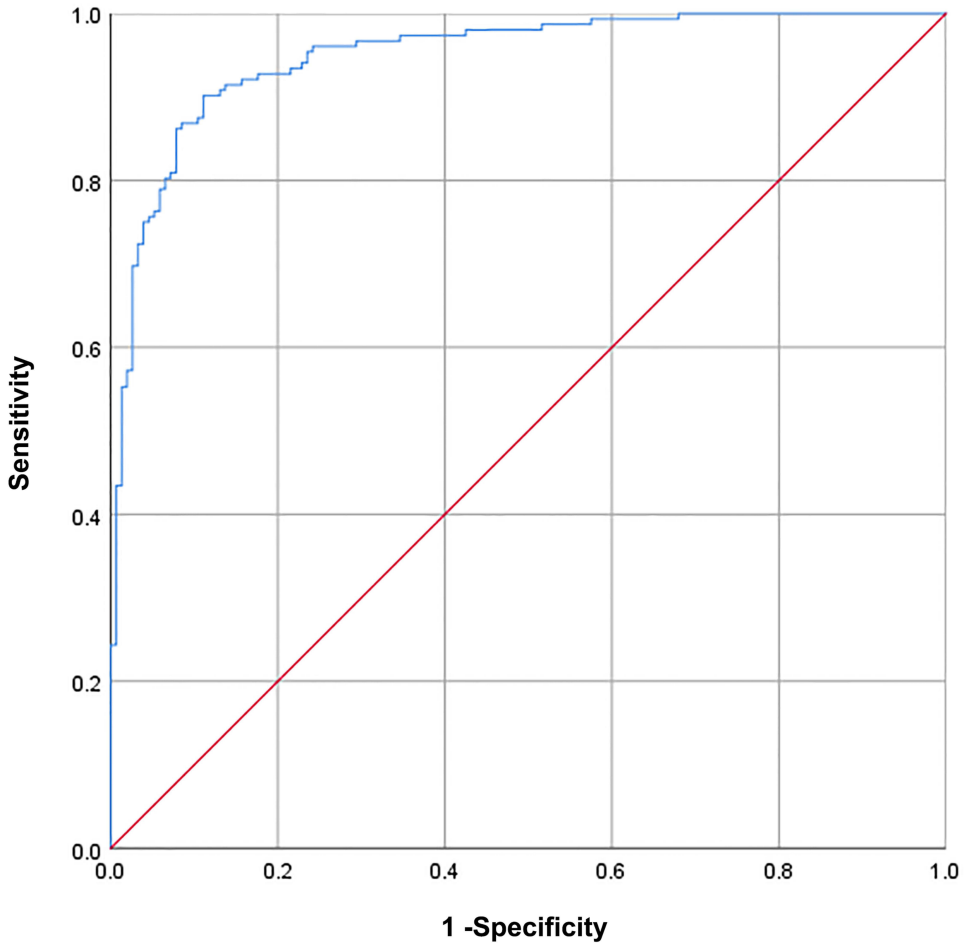

Figure 3 ROC curve of the nomogram model for predicting acute LHI.
Abbreviation: LHI, Large Hemispheric Infarction.

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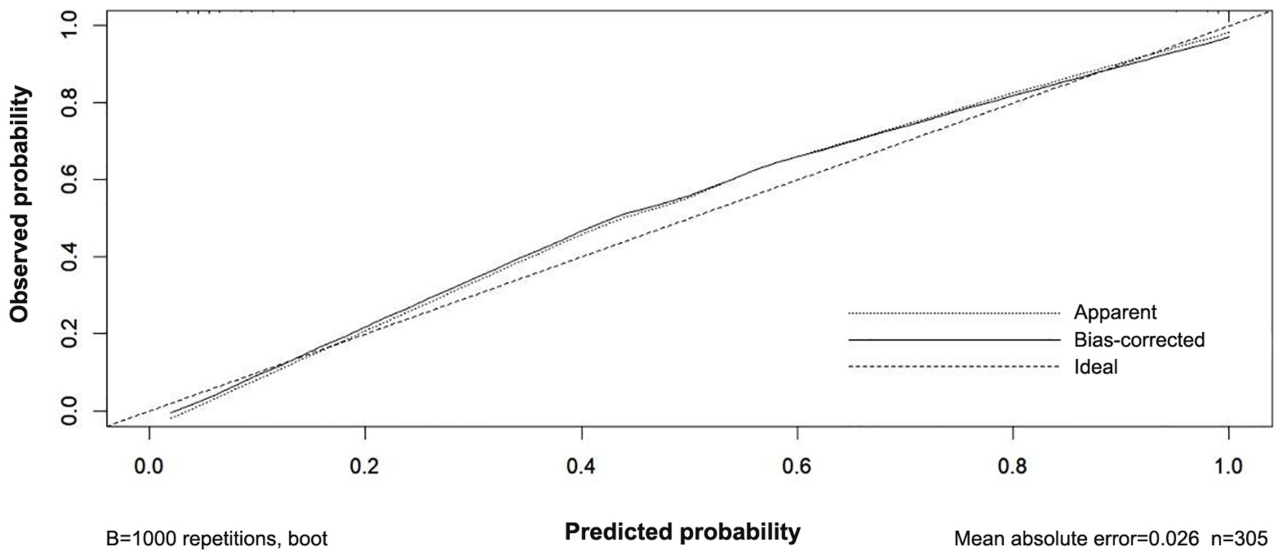

Figure 4 Calibration curve of the nomogram predictive model for acute LHI.
Abbreviation: LHI, Large Hemispheric Infarction.

Discussion

LHI is a neurological condition that predominantly affects older adults and is associated with high rates of incidence, disability, and mortality. In recent years, a trend toward earlier onset has been observed. Early identification of risk factors for LHI is critical for facilitating timely diagnosis, risk stratification, and the implementation of targeted therapeutic strategies aimed at improving clinical outcomes. It has been reported that neuroinflammatory responses not only play a key role in the pathophysiological process of ischemic stroke, but are also crucial in hemorrhagic stroke and traumatic brain injury, with more significant inflammatory activation observed in LHI cases.[19,20] Inflammatory responses and cell activation, neutrophil migration and recruitment, as well as the release of inflammatory mediators can lead to secondary damage, with neutrophils, monocytes, and lymphocytes playing different roles in this process.[21] However, due to the complexity and interrelatedness of inflammatory signaling pathways, it is difficult to generalize the overall inflammatory response. The presence of specific inflammatory biomarkers provides a potential basis for predicting the risk of death and functional prognosis in patients.[9,22,23] Existing inflammatory markers often lack either clinical practicality or the capacity to comprehensively represent the dynamic balance between pro-inflammatory and anti-inflammatory processes. Consequently, the identification of sensitive and reliable inflammatory biomarkers is essential for the early detection and clinical intervention of conditions like LHI, which carry substantial prognostic implications.

This study examined if SIRI could serve as an independent risk factor for LHI and further evaluated the diagnostic value of a predictive model incorporating SIRI alongside other independent risk factors. Over the past few years, substantial research has indicated the relevance of composite inflammatory indices in the context of inflammatory diseases. Common markers such as the neutrophil-to-lymphocyte ratio (NLR), the lymphocyte-to-platelet ratio (PLR), and the systemic immune-inflammation index (SII) have been proven to effectively reflect the overall inflammatory status. High NLR, low PLR,[24] and high SII[25] levels are associated with the severity of ischemic stroke, traumatic brain injury, functional outcomes, and poor prognosis of patient mortality. SIRI, as a novel composite marker, integrates neutrophil, monocyte, and lymphocyte counts and is hypothesized to reflect the magnitude of inflammatory activity in patients experiencing acute cerebrovascular events. Despite its potential, there has been limited research focusing specifically on the association between SIRI and early neurological impairment or disease severity in LHI.

Several prior studies have supported the clinical relevance of SIRI in stroke populations. Jin et al conducted a 10-year follow-up study involving 85,154 individuals and reported that, after adjusting for demographic, socioeconomic, and clinical confounders, individuals with SIRI levels $\geq 1.07 \times 10^9$/L had a significantly higher risk of total stroke, hemorrhagic stroke, and ischemic stroke compared to those with SIRI $< 0.13 \times 10^9$/L.[26] Kadir Arslan et al[22] conducted

a retrospective study on critically ill patients in the hospital's ICU wards. They found that SIRI could predict the functional status upon discharge, and it was significantly higher in the group with poor functional outcomes. Similarly, Zhang et al, in an analysis of 180 patients with ischemic stroke, identified a positive correlation between SIRI and NIHSS scores ($r = 0.340$, $p < 0.001$).[27] Huang et al stratified 234 ischemic stroke patients into mild (NIHSS ≤ 5) and moderate-to-severe (NIHSS > 5) groups, and found that SIRI levels were significantly higher in the moderate-to-severe group, with a significant positive correlation between SIRI and NIHSS scores.[28]

Consistent with these findings, the current study demonstrated that patients in the LHI group had significantly higher SIRI levels compared to those in the healthy control group. Furthermore, the diagnostic analysis indicated that SIRI had the highest predictive performance among evaluated biomarkers, with both high sensitivity and specificity. Multivariate logistic regression analysis confirmed that SIRI is an independent risk factor for LHI. From these findings, patients with neurological deficits and SIRI levels exceeding the identified threshold of 1.39 should be considered at high risk for LHI, and early intervention should be prioritized to prevent further clinical deterioration.

Among the independent risk factors included in the predictive model, atrial fibrillation—one of the most common arrhythmias—has been closely associated with the development of ischemic stroke. Patients with atrial fibrillation are at increased risk for large cerebral infarctions.[29,30] In this study, atrial fibrillation was independently associated with LHI, consistent with prior research.

Coronary heart disease shares common pathophysiological mechanisms with cerebral infarction, particularly atherosclerosis, which leads to arterial stenosis, plaque rupture, or embolic events. The analysis indicated that a history of coronary heart disease was associated with a significantly increased risk of LHI, identifying it as an independent risk factor.

A prior history of stroke was significantly associated with LHI. In the LHI group, 26.3% of patients ($n = 40$) had a history of stroke, compared to 8.5% ($n = 13$) in the control group. This finding aligns with the results of Zhang et al, who reported that a history of cerebral infarction is an independent predictor of poor prognosis in patients with LHI.[31]

Systolic and diastolic blood pressures were significantly elevated in the LHI group when compared to the control group, and multivariate logistic regression identified elevated systolic blood pressure as an independent risk factor for LHI. Prior studies established a positive correlation between blood pressure and infarct size, with higher systolic and diastolic pressures associated with larger infarcts.[32]

Hyperglycemia exacerbates endothelial dysfunction, increases blood viscosity, expands infarct size, and elevates the risk of hemorrhagic transformation.[33] Additionally, Ruan et al examined the relationship between HCY levels and infarct size in patients with acute cerebral infarction, finding a significant positive correlation.[34] Larger infarct sizes were associated with elevated serum HCY levels, indicating that HCY serves as a useful biomarker for the diagnosis and evaluation of cerebral infarction severity.

In this study, serum HCY levels were significantly higher in the LHI group than in the control group, and multivariate regression analysis identified HCY as an independent risk factor for LHI.

In summary, patients presenting with one or more of these independent risk factors—atrial fibrillation, coronary heart disease, prior stroke, elevated systolic blood pressure, hyperglycemia, and elevated serum HCY—should be closely monitored to enable early detection and prevention of large hemispheric infarction.

This study examined the relationship between SIRI, other common clinical risk factors, and the diagnosis of LHI. Atrial fibrillation, a history of coronary heart disease, prior stroke, elevated FBG, increased systolic blood pressure, HCY, and SIRI were identified as independent risk factors for LHI. The diagnostic value of these variables was assessed using ROC curve analysis, and a predictive model incorporating SIRI along with the six other independent risk factors was subsequently developed. A nomogram was constructed based on this model, and its discriminative and calibration capabilities were evaluated using ROC and calibration curves. The findings confirmed that the model demonstrated strong performance in estimating LHI risk.

The nomogram integrates multiple predictors derived from multivariate logistic regression into a visual format, providing a straightforward and accessible tool for estimating individual risk. This enables the early identification of individuals at high risk for LHI and supports the formulation of timely and targeted treatment strategies to mitigate adverse outcomes. The predictive model was based on routinely available laboratory and imaging parameters, facilitating

its application in clinical practice for efficient risk assessment and personalized treatment planning. These features contribute to its practical use and clinical relevance.

There were some limitations in our study as follows: 1) Considering this was a single-center respective study, our sample size was limited, which may lead to biased research results. 2) During our research, we failed to take into account more other risk factors, which limited the further analysis of the study. 3) This article is a retrospective study and may have recall bias. 4) This study only followed up for a short period of time to verify that even the effect lacks long-term observation effects.

Conclusion

SIRI has certain clinical value in predicting LHI. The developed nomogram-based predictive model incorporating SIRI exhibited robust predictive performance and may assist in guiding clinical decision-making.

Abbreviations

LHI, Large Hemispheric Infarction; SIRI, Systemic inflammatory response index; NIHSS, National Institutes of Health Stroke Scale; FBG, Fasting blood glucose; TC, Total Cholesterol; TG, Triglyceride; HDL-C, High Density Lipoprotein Cholessterol; LDL-C, Low Density Lipoprotein Cholessterol; HBA1C, Glycosylated hemoglobin; HCY, Homocysteine; AIS, Acute Ischemic Stroke; ICA, Internal cerebral artery; MCA, Middle cerebral artery; NLR, Neutrophil-to-Lymphocyte Ratio; PLR, Platelet-Lymphocyte Ratio; LMR, Lymphocyte-monocyte ratio; RDW, Red blood cell distribution width; CT, Computed Tomography; MRI, Magnetic Resonance Imaging; DWI, Diffusion Weighted Imaging; FLAIR, Fluid-attenuated inversion recovery; ASPECTS, Alberta Stroke Program Early CT Score; BBB, Blood brain barrier; ROS, Reactive Oxygen Species; NO, Nitric Oxide; BMI, Body Mass Index.

Data Sharing Statement

All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author.

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki (as was revised in 2013). The study was approved by Ethics Committee of the Shaanxi Provincial People's Hospital. Written informed consent was obtained from all participants.

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