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ORIGINAL RESEARCH

Analysis of Risk Factors for Hemorrhagic Transformation of Cerebral Infarction and Recurrence of Cerebral Infarction: A Retrospective Study on Cerebral Infarction Patients with or without Hemorrhagic Transformation

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Background: Acute ischemic stroke, especially hemorrhage cerebral infarction (HCI), resulted in the leading causes of mortality and long-term disability across populations. However, fewer researches have focused on the risk factors of first admission and recurrence of HCI.

Methods: The study included 1857 patients who underwent cerebral infarction with or without hemorrhagic transformation. Clinical characteristics were collected, and univariate and multivariate analysis were performed to explore the risk factors. The subgroup analysis of cerebral infarction recurrence was performed. ROC analysis was utilized, and AUCs were showed the diagnostic values of the risk factors.

Results: Compared to the patients with non-hemorrhage cerebral infarction, the patients with hemorrhage cerebral infarction were older and had higher Neutrophil infiltration, AST expression, globulin and BUN, while had lower ALT expression, triglyceride, PT, APTT, homocysteine, d-dimer, CRP and glycosylated hemoglobin. Utilizing univariate and multivariate analysis, age, thrombolytic, Hb, AST and glycosylated hemoglobin were the risk factors between the patients with hemorrhagic cerebral infarction and non-hemorrhagic cerebral infarction. ROC analysis was performed to demonstrate that glycosylated hemoglobin was a diagnostic biomarker for the patients with hemorrhagic cerebral infarction and non-hemorrhagic cerebral infarction (AUC = 0.808). Utilizing univariate and multivariate analysis, age, hypertension history, LDL and MRS Score on admission were the risk factors between non-hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence. ROC analysis was performed to demonstrate MRS Score on admission was a diagnostic biomarker for recurrence of cerebral infarction in patients with non-hemorrhagic cerebral infarction (AUC = 0.708). Utilizing univariate and multivariate analysis, only hypertension history was the risk factors between hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence.

Conclusion: In conclusion, age, hypertension history, LDL and MRS Score on admission were the risk factors between cerebral infarction patients with first admission or the cerebral infarction recurrence.

Keywords: acute ischemic stroke, hemorrhage cerebral infarction, LDL, MRS Score, ROC

Introduction

Acute ischemic stroke, especially hemorrhage cerebral infarction (HCI), is a substantial global health concern, resulting in the leading causes of mortality and long-term disability across populations.[1] HCI is a destructive complication

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associated with acute ischemic stroke, with the incidence of HCI being 15%.[2,3] The utilization of anticoagulants has shown efficacy in preventing stroke; however, it could also increase the risk of hemorrhagic complications following ischemic stroke.[4] Vitamin K antagonists were found to be an independent risk factor for HCI.[4] The patients experiencing HCI had a higher mortality rate.[5] The incidence of HCI after acute ischemic stroke varies significantly. Early identification of these risk factors may enable the prediction and prevention of this condition.

Studies have shown that early disruption of the blood–brain barrier plays a key role in the hemorrhagic transformation during acute ischemic stroke.[6] The development of HCI after cerebral infarction involves multiple interconnected pathological processes from peripheral blood cells to neurovascular units, such as hyperactive ischemic cascades with elevated matrix-metalloproteinase (MMP) levels, excessive reactive oxygen species (ROS) levels, cocopathy, BBB breakdown, and reperfusion injury.[7,8] Increased Neutrophil-to-lymphocyte Ratios are Associated with Greater Risk of Hemorrhagic Transformation in Patients with Acute Ischemic Stroke.[9] Low level of low-density lipoprotein cholesterol (LDL) was related with increased hemorrhagic transformation after acute ischemic cerebral infarction.[10] Research indicated some other risk factors for HCI, including age, stroke severity, baseline hypertension, high NIH Stroke Scale (NIHSS) scores, hyperglycemia, cardioembolic infarction, poor collaterals, early infarction on brain imaging, low platelet count, use of antithrombotic drugs, and reperfusion therapy.[11,12]

Through extensive clinical practice, in addition to the aforementioned risk factors, whether intravenous thrombolysis was initiated within the thrombolysis time window after the onset of this disease,[12] whether there was a history of using atrial fibrillation, whether small anti-hypertensive drugs were taken before the onset of the disease[13] and whether oral anticoagulants were taken for some reason before the onset of the disease[14] all have certain predictive effects on the occurrence of this disease. For example, cerebral vascular occlusion caused by atrial fibrillation has been repeatedly validated in numerous clinical studies, but the occurrence of hemorrhagic cerebral infarction is rarely mentioned.

However, fewer researches have focused on the risk factors of first admission and recurrence of HCI. In this study, we analyzed the risk factors of first admission and recurrence of HCI and ROC analysis was performed to show the diagnostic value, which may be used for the treatment to HCI patients and prevent the HCI recurrence.

Methods

Study Design

This retrospective cohort study was conducted at Tianjin Huanhu Hospital. We collected the medical information of 5738 patients. Based on strict inclusion criteria and exclusion criteria, we finally included 1857 patients, in which 433 underwent cerebral infarction with hemorrhagic transformation and 1424 underwent cerebral infarction without hemorrhagic transformation. All study protocol was approved by Tianjin Huanhu Hospital. This study was conducted in accordance with the Declaration of Helsinki and approved by the ethics committee of Tianjin Huanhu Hospital (2024–194). Written informed consent was obtained from all participants.

Inclusion criteria were as follows: (a) age ≥30 years; (b) completion of cranial CT or MRI imaging upon admission, with repeated head CT scans during hospitalization confirming hemorrhagic transformation of ischemic stroke; (c) signed informed consent. Exclusion criteria were: (a) patients with severe dementia; (b) patients with a history of brain surgery; (c) patients who did not undergo cranial imaging upon admission or did not show hemorrhagic transformation on repeated head CT scans after admission. All participants or their authorized representatives provided informed consent for clinical analysis. All analyses were conducted in accordance with the Declaration of Helsinki and local ethical policies. All the patients were treated according to the guidelines.

Data Collection

Upon admission, the baseline characteristics of patients were examined and collected, including demographic information (eg, gender, age, smoking), comorbidities (eg, stroke, hypertension, diabetes, coronary artery disease, atrial fibrillation), laboratory tests (eg, complete blood count, coagulation function, renal function, liver function, lipid profile, blood glucose, glycated hemoglobin), and stroke-related scoring scales (NIHSS scoring system, GCS scoring and mRS scoring).

Statistical Analysis

Data are presented as mean ± SD, median (Q1–Q3), or frequency (percentage). Statistical analyses were performed using SPSS 23. 0. Shapiro–Wilk normality test, Welch's *t* test (2 groups) and one- or two-way ANOVA with Bonferroni post-hoc analyses were used for comparisons between different groups. Univariate and multivariate analysis were performed to explore the risk factors. ROC analysis was utilized, and AUCs were showed the diagnostic values of the risk factors. Multiple comparison P values less than 0. 05 were considered to be statistically significant.

Results

The Baseline Characteristics of Patients with Hemorrhagic Cerebral Infarction or Non-Hemorrhagic Cerebral Infarction

A total of 1857 patients underwent cerebral infarction with or without hemorrhagic transformation. Hemorrhagic transformation occurred in 433 patients. Compared to the patients with non-hemorrhage cerebral infarction, the patients with hemorrhage cerebral infarction were older and had higher neutrophil infiltration, AST expression, globulin and BUN, while had lower ALT expression, triglyceride, PT, APTT, homocysteine, d-dimer, CRP and glycosylated hemoglobin (Table 1). Utilizing univariate and multivariate analysis, age, thrombolytic, Hb, AST and glycosylated hemoglobin

Table 1 The Baseline Characteristics of Patients with Hemorrhagic Cerebral Infarction or Non-Hemorrhagic Cerebral Infarction

Characteristics	**Hemorrhagic (n=433)**	**Non-Hemorrhagic (n=1424)**	**P Value**
Demographics			
Age, median (IQR)	66 (58, 73)	64 (57, 71)	**0.003**
Gender, n (%) Female Male	 124 (28.6%) 309 (71.4%)	 374 (26.3%) 1050 (73.7%)	0.329
Smoke, n (%)	225 (60.0%)	764 (53.7%)	0.537
Comorbidities			
Hypertension history, n (%)	305 (70.4%)	1078 (75.7%)	**0.028**
Coronary artery disease history, n (%)	112 (25.9%)	235 (16.5%)	**< 0.001**
Atrial fibrillation history, n (%)	63 (14.5%)	81 (5.9%)	**< 0.001**
Diabetes history, n (%)	142 (32.8%)	526 (36.9%)	0.116
Stroke history, n (%)	145 (33.5%)	453 (31.8%)	0.513
Patients on anticoagulants			
Use anticoagulants, n (%)	18 (4.2%)	280 (19.7%)	**< 0.001**
Acute recanalization therapy			
Thrombolytic, n (%)	156 (36.0%)	104 (7.3%)	**< 0.001**
Laboratory data			
Hb (g/L), median (IQR) Platelet (x10^9/L), median (IQR) Neutrophil absolute value, median (IQR) Neutrophil (%), median (IQR)	139 (127, 150) 215 (178.25, 259) 5.56 (3.8825, 8.7825) 71.7 (63.3, 81.7)	142 (132, 153) 220.5 (184, 262) 4.61 (3.6, 6.12) 65.4 (59.1, 72.2)	**< 0.001** 0.205 **< 0.001** **< 0.001**

(*Continued*)

Table 1 (Continued).

Characteristics	Hemorrhagic (n=433)	Non-Hemorrhagic (n=1424)	P Value
ALT (U/L), median (IQR)	17 (12, 25)	18 (13, 28)	**0.038**
AST (U/L), median (IQR)	21 (17, 27.25)	20 (16, 24)	**< 0.001**
Total protein (g/L), median (IQR)	69.1 (64.8, 73.2)	66.7 (63.3, 70.3)	**< 0.001**
Albumin (g/L), median (IQR)	39.2 (36.2, 41.9)	38.8 (36.7, 40.9)	0.165
Globulin (g/L), median (IQR)	29.345 (26.9, 33.1)	28.3 (26, 31.1)	**< 0.001**
Total cholesterol (mmol/L), median (IQR)	4.405 (3.5, 5.2375)	4.44 (3.77, 5.19)	0.087
Triglyceride (mmol/L), median (IQR)	1.12 (0.7925, 1.6)	1.34 (1, 1.82)	**< 0.001**
Creatinine (umol/L), median (IQR)	69.3 (58.7, 83.5)	70.95 (60.225, 83.3)	0.190
Prothrombin time (sec), median (IQR)	11.5 (10.9, 12)	11.6 (11.1, 12.1)	**0.002**
Activated partial thromboplastin time (sec), median (IQR)	23.7 (21.4, 26.2)	24.3 (22, 27.175)	**< 0.001**
LDL (mmol/L), median (IQR)	2.75 (2.08, 3.37)	2.74 (2.26, 3.28)	0.431
HDL (mmol/L), median (IQR)	1.07 (0.88, 1.2975)	1.04 (0.9, 1.21)	0.216
Homocysteine (umol/L), median (IQR)	0 (0, 13.13)	12.75 (9.88, 16.78)	**< 0.001**
ALP (U/L), median (IQR)	82 (68, 101)	83 (70, 98)	0.659
Uric Acid (umol/L), median (IQR)	291 (233, 369)	303.5 (244, 368)	0.083
BUN (mmol/L), median (IQR)	5.7 (4.5, 6.8)	5.1 (4.2, 6.3)	**< 0.001**
d-dimer (mL/L FEU), median (IQR)	0 (0, 0.77)	0.4 (0.24, 0.7975)	**< 0.001**
Fibrinogen (g/L), median (IQR)	3.03 (2.44, 3.68)	2.92 (2.49, 3.51)	0.161
CRP (mg/L), median (IQR)	0 (0, 3.32)	1.82 (0.61, 5.3)	**< 0.001**
Fasting blood-glucose (mmol/L), median (IQR)	5.89 (4.94, 8.33)	5.73 (4.92, 7.59)	0.222
Glycosylated hemoglobin (%), median (IQR)	0 (0, 6.3)	7.1 (6.1, 8.5)	**< 0.001**
Score			
NIHSS score on admission, median (IQR)	6 (2, 12)	5 (2, 8)	**< 0.001**
GCS score, median (IQR)	15 (14, 15)	15 (15, 15)	**< 0.001**
MRS Score on admission, median (IQR)	3 (1, 4)	0 (0, 0)	**< 0.001**

Notes: P-values below 0. 05, indicating statistical significance, are shown in bold.
Abbreviations: IQR, interquartile range; Hb, hemoglobin; ALT, glutamic-pyruvic transaminase; AST, glutamic oxalacetic transaminase; LDL, low-density lipoprotein; HDL, high-density lipoprotein; ALP, alkaline phosphatase; BUN, blood urea nitrogen; CRP, C-reactive protein; NIHSS, National Institutes of Health Stroke Scale; GCS, Glasgow score; MRS, Modified Rankin Scale.

were the risk factors between the patients with hemorrhagic cerebral infarction and non-hemorrhagic cerebral infarction (Table 2). ROC analysis was performed to demonstrate that glycosylated hemoglobin was a diagnostic biomarker for the patients with hemorrhagic cerebral infarction and non-hemorrhagic cerebral infarction (AUC = 0.808, Figure 1).

Table 2 Univariate and Multivariate Analysis of Patients with Hemorrhagic Cerebral Infarction or Non-Hemorrhagic Cerebral Infarction

Characteristics	Univariate Analysis		Multivariate Analysis	
	Odds Ratio (95% CI)	P Value	Odds Ratio (95% CI)	P Value
Age	0.985 (0.976–0.995)	**0.002**	0.972 (0.950–0.994)	**0.012**
Hypertension history	1.308 (1.029–1.661)	**0.028**	0.927 (0.546–1.573)	0.779
Coronary artery disease history	0.566 (0.438–0.732)	**< 0.001**	1.258 (0.681–2.326)	0.464
Atrial fibrillation history	0.354 (0.250–0.502)	**< 0.001**	0.414 (0.143–1.200)	0.104
Use anticoagulants	5.643 (3.458–9.208)	**< 0.001**	1.348 (0.498–3.650)	0.557

(*Continued*)

Table 2 (Continued).

Characteristics	Univariate Analysis		Multivariate Analysis	
	Odds Ratio (95% CI)	P Value	Odds Ratio (95% CI)	P Value
Thrombolytic	0.140 (0.106–0.185)	**< 0.001**	0.224 (0.109–0.459)	**< 0.001**
Hb (g/L)	1.014 (1.009–1.019)	**< 0.001**	1.012 (1.002–1.023)	**0.023**
Neutrophil absolute value	0.877 (0.847–0.908)	**< 0.001**	0.913 (0.812–1.025)	0.124
Neutrophil (%)	0.973 (0.964–0.982)	**< 0.001**	0.996 (0.973–1.019)	0.714
ALT (U/L)	1.005 (1.000–1.011)	0.072	1.017 (0.997–1.038)	0.089
AST (U/L)	0.988 (0.982–0.995)	**0.001**	0.955 (0.920–0.991)	**0.014**
Total protein (g/L)	0.992 (0.978–1.006)	0.269		
Globulin (g/L)	0.974 (0.953–0.996)	**0.019**	0.985 (0.949–1.024)	0.450
Triglyceride (mmol/L)	1.565 (1.328–1.844)	**< 0.001**	1.183 (0.926–1.511)	0.179
Prothrombin time (sec)	1.143 (1.076–1.215)	**< 0.001**	0.994 (0.858–1.151)	0.933
Activated partial thromboplastin time (sec)	1.043 (1.022–1.066)	**< 0.001**	1.031 (0.986–1.078)	0.185
Homocysteine (umol/L)	1.070 (1.052–1.087)	**< 0.001**	1.005 (0.980–1.030)	0.718
BUN (mmol/L)	0.888 (0.846–0.931)	**< 0.001**	0.943 (0.832–1.069)	0.357
d-dimer (mL/L FEU)	0.973 (0.946–1.000)	0.050	0.979 (0.887–1.079)	0.664
CRP (mg/L)	1.000 (0.996–1.004)	0.933		
Glycosylated hemoglobin (%)	1.485 (1.420–1.554)	**< 0.001**	1.107 (1.039–1.180)	**0.002**
NIHSS score on admission	0.949 (0.933–0.964)	**< 0.001**	0.990 (0.935–1.049)	0.741
GCS score	1.215 (1.159–1.272)	**< 0.001**	1.239 (0.963–1.593)	0.096

Notes: P-values below 0.05, indicating statistical significance, are shown in bold.
Abbreviations: CI, confidence interval; Hb, hemoglobin; ALT, glutamic-pyruvic transaminase; AST, glutamic oxalacetic transaminase; BUN, blood urea nitrogen; CRP, C-reactive protein; NIHSS, National Institutes of Health Stroke Scale; GCS, Glasgow score.

The Baseline Characteristics of Patients with or without First Admission

There were differences at the baseline characteristics of the patients with and without first admission, including age, hypertension history, coronary artery disease history, diabetes history, thrombolytic, Hb, neutrophil infiltration, total protein, albumin, total cholesterol, triglyceride, creatinine, PT, APTT, LDL, HDL, homocysteine, BUN, fibrinogen, CRP and glycosylated hemoglobin (Table 3). Utilizing univariate and multivariate analysis, age and hypertension history were the risk factors of the cerebral infarction recurrence (Table 4).

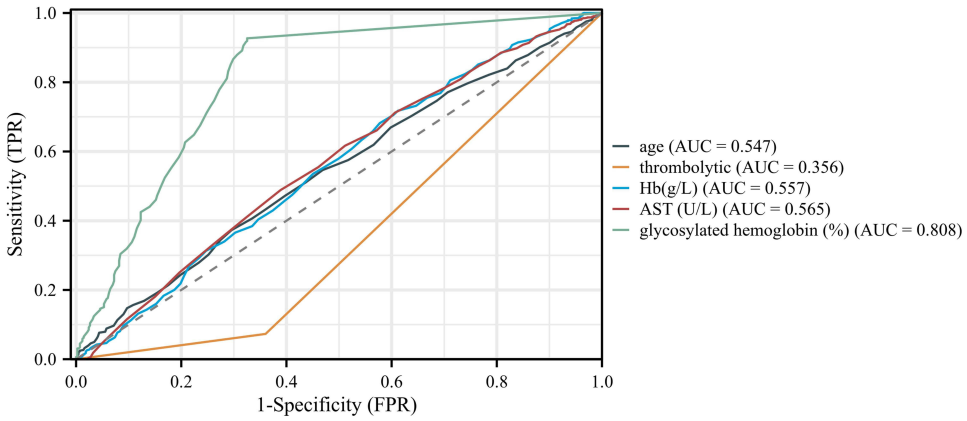

Figure 1 The diagnostic value between patients with hemorrhagic cerebral infarction and non-hemorrhagic cerebral infarction.

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Table 3 The Baseline Characteristics of Patients with or without First Admission

Characteristics	Ctrl (n=1259)	First Admission (n=598)	P Value
Demographics			
Age, median (IQR)	63 (56, 70)	67 (61, 73)	**< 0.001**
Gender, n (%) Female Male	 345 (27.4%) 914 (72.6%)	 153 (25.6%) 445 (74.4%)	0.409
Smoke, n (%)	660 (52.4%)	329 (55.0%)	0.295
Comorbidities			
Hypertension history, n (%)	891 (70.8%)	492 (82.3%)	**< 0.001**
Coronary artery disease history, n (%)	195 (15.5%)	152 (25.4%)	**< 0.001**
Atrial fibrillation history, n (%)	92 (7.3%)	52 (8.7%)	0.296
Diabetes history, n (%)	398 (31.6%)	270 (45.2%)	**< 0.001**
Patients on anticoagulants			
Use anticoagulants, n (%)	205 (16.3%)	93 (15.6%)	0.688
Acute recanalization therapy			
Thrombolytic, n (%)	192 (15.3%)	68 (11.4%)	**0.024**
Laboratory data			
Hb (g/L), median (IQR)	143 (131, 153)	140 (130, 151)	**0.003**
Platelet (x10^9/L), median (IQR)	221 (184.5, 261)	215 (179, 262)	0.188
Neutrophil absolute value, median (IQR)	4.72 (3.6, 6.475)	5.11 (3.83, 7.18)	**0.005**
Neutrophil (%), median (IQR)	65.9 (59.2, 74.65)	68.6 (61.8, 77.7)	**< 0.001**
ALT (U/L), median (IQR)	18 (13, 27)	18 (13, 27)	0.529
AST (U/L), median (IQR)	20 (17, 25)	20 (17, 25)	0.868
Total protein (g/L), median (IQR)	67.3 (63.7, 71.1)	66.6 (63.1, 70.6)	**0.014**
Albumin (g/L), median (IQR)	39 (36.9, 41.35)	38.3 (35.975, 40.725)	**< 0.001**
Globulin (g/L), median (IQR)	28.4 (26.2, 31.575)	28.8 (26.4, 31.7)	0.213
Total cholesterol (mmol/L), median (IQR)	4.525 (3.8375, 5.2525)	4.19 (3.45, 5.005)	**< 0.001**
Triglyceride (mmol/L), median (IQR)	1.3 (0.97, 1.8125)	1.23 (0.92, 1.66)	**0.011**
Creatinine (umol/L), median (IQR)	69.5 (59.3, 81.3)	73 (60.575, 87.6)	**< 0.001**
Pro$thrombin time (sec), median (IQR)	11.5 (11, 12)	11.6 (11.2, 12.2)	**< 0.001**
Activated partial thromboplastin time (sec), median (IQR)	23.9 (21.7, 26.6)	24.55 (22.4, 27.6)	**< 0.001**
LDL (mmol/L), median (IQR)	2.84 (2.34, 3.36)	2.55 (2.035, 3.155)	**< 0.001**
HDL (mmol/L), median (IQR)	1.06 (0.91, 1.24)	1.02 (0.865, 1.21)	**0.001**
Homocysteine (umol/L), median (IQR)	11.26 (0, 15.445)	10.01 (0, 14.91)	**0.023**
ALP (U/L), median (IQR)	82 (69, 99)	83 (69, 98)	0.958
Uric Acid (umol/L), median (IQR)	301 (243, 371)	297 (238, 364)	0.255
BUN (mmol/L), median (IQR)	5.2 (4.2, 6.3)	5.4 (4.4, 6.7)	**0.006**
d-dimer (mL/L FEU), median (IQR)	0.33 (0.19, 0.725)	0.415 (0.22, 0.91)	**< 0.001**
Fibrinogen (g/L), median (IQR)	2.87 (2.45, 3.44)	3.05 (2.545, 3.8375)	**< 0.001**
CRP (mg/L), median (IQR)	0.971 (0, 3.86)	1.46 (0, 7.14)	**0.018**
Fasting blood-glucose (mmol/L), median (IQR)	5.675 (4.92, 7.6125)	6.005 (4.94, 7.895)	0.093
Glycosylated hemoglobin (%), median (IQR)	6.3 (0, 7.9)	6.8 (0, 8)	**0.025**

(*Continued*)

Table 3 (Continued).

Characteristics	Ctrl (n=1259)	First Admission (n=598)	P Value
NIHSS score on admission, median (IQR)	5 (2, 8)	6 (3, 10)	**< 0.001**
GCS score, median (IQR)	15 (15, 15)	15 (15, 15)	0.321
MRS Score on admission, median (IQR)	0 (0, 1)	1 (0, 3)	**< 0.001**

Notes: P-values below 0.05, indicating statistical significance, are shown in bold.
Abbreviations: IQR, interquartile range; Hb, hemoglobin; ALT, glutamic-pyruvic transaminase; AST, glutamic oxalacetic transaminase; LDL, low-density lipoprotein; HDL, High-density lipoprotein; ALP, alkaline phosphatase; BUN, blood urea nitrogen; CRP, C-reactive protein; NIHSS, National Institutes of Health Stroke Scale; GCS, Glasgow score; MRS, Modified Rankin Scale.

Table 4 Univariate and Multivariate Analysis of Patients with or without First Admission

Characteristics	Univariate Analysis		Multivariate Analysis	
	Odds Ratio (95% CI)	P Value	Odds Ratio (95% CI)	P Value
Age	1.032 (1.022–1.041)	**< 0.001**	1.021 (1.001–1.041)	**0.035**
Hypertension history	1.917 (1.504–2.443)	**< 0.001**	1.873 (1.187–2.956)	**0.007**
Coronary artery disease history	1.860 (1.464–2.362)	**< 0.001**	1.162 (0.739–1.825)	0.516
Diabetes history	1.781 (1.458–2.175)	**< 0.001**	1.202 (0.721–2.004)	0.480
Thrombolytic	0.713 (0.531–0.958)	**0.025**	0.747 (0.464–1.201)	0.228
Hb (g/L)	0.996 (0.992–1.001)	0.101		
Neutrophil absolute value	1.047 (1.014–1.082)	**0.006**	0.999 (0.931–1.071)	0.973
Neutrophil (%)	1.019 (1.010–1.028)	**< 0.001**	1.003 (0.985–1.021)	0.729
Total protein (g/L)	0.992 (0.980–1.005)	0.222		
Albumin (g/L)	0.974 (0.954–0.994)	**0.011**	0.985 (0.950–1.021)	0.405
Total cholesterol (mmol/L)	0.852 (0.788–0.921)	**< 0.001**	1.075 (0.685–1.686)	0.753
Triglyceride (mmol/L)	0.884 (0.781–1.001)	0.053	1.011 (0.790–1.294)	0.932
Creatinine (umol/L)	1.009 (1.005–1.013)	**< 0.001**	1.007 (0.997–1.017)	0.193
Prothrombin time (sec)	1.065 (1.008–1.126)	**0.025**	1.012 (0.917–1.116)	0.817
Activated partial thromboplastin time (sec)	1.028 (1.009–1.048)	**0.004**	1.030 (0.993–1.069)	0.117
LDL (mmol/L)	0.733 (0.654–0.822)	**< 0.001**	0.700 (0.382–1.281)	0.248
HDL (mmol/L)	0.673 (0.484–0.936)	**0.019**	1.333 (0.619–2.868)	0.463
Homocysteine (umol/L)	0.981 (0.967–0.995)	**0.010**	0.987 (0.965–1.009)	0.252
BUN (mmol/L)	1.095 (1.047–1.146)	**< 0.001**	1.074 (0.966–1.193)	0.186
d-dimer (mL/L FEU)	1.018 (0.992–1.044)	0.177		
Fibrinogen (g/L)	1.211 (1.103–1.330)	**< 0.001**	1.067 (0.957–1.190)	0.244
CRP (mg/L)	1.003 (0.998–1.008)	0.192		
Glycosylated hemoglobin (%)	1.036 (1.002–1.070)	**0.038**	1.013 (0.947–1.083)	0.706
NIHSS score on admission	1.036 (1.021–1.052)	**< 0.001**	0.992 (0.949–1.037)	0.729
MRS Score on admission	1.364 (1.280–1.453)	**< 0.001**	1.036 (0.853–1.259)	0.722

Notes: P-values below 0.05, indicating statistical significance, are shown in bold.
Abbreviations: CI, confidence interval; Hb, hemoglobin; LDL, low-density lipoprotein; HDL, High-density lipoprotein; BUN, blood urea nitrogen; CRP, C-reactive protein; NIHSS, National Institutes of Health Stroke Scale; MRS, Modified Rankin Scale.

The Subgroup Analysis About the Cerebral Infarction Recurrence

There were differences at the baseline characteristics of the non-hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence, including age, hypertension history, coronary artery disease history, diabetes history, thrombolytic, Hb, neutrophil infiltration, total protein, albumin, total cholesterol, creatinine, PT, LDL, HDL, d-dimer, fibrinogen and CRP (Table 5). Utilizing univariate and multivariate analysis, age, hypertension history,

Table 5 The Baseline Characteristics of Non-Hemorrhagic Cerebral Infarction Patients with or without First Admission

Characteristics	Ctrl (n=971)	First Admission (n=453)	P Value
Demographics			
Age, median (IQR)	63 (55, 70)	67 (60, 72)	**< 0.001**
Gender, n (%) Female Male	 255 (26.3%) 716 (73.7%)	 119 (26.3%) 334 (73.7%)	0.998
Smoke, n (%)	517 (53.2%)	247 (54.5%)	0.652
Comorbidities			
Hypertension history, n (%)	700 (72.1%)	378 (83.4%)	**< 0.001**
Coronary artery disease history, n (%)	128 (13.2%)	107 (23.6%)	**< 0.001**
Atrial fibrillation history, n (%)	51 (5.3%)	30 (6.6%)	0.298
Diabetes history, n (%)	314 (32.3%)	212 (46.8%)	**< 0.001**
Patients on anticoagulants			
Use anticoagulants, n (%)	192 (19.8%)	88 (19.4%)	0.878
Acute recanalization therapy			
Thrombolytic, n (%)	84 (8.7%)	20 (4.4%)	**0.004**
Laboratory data			
Hb (g/L), median (IQR)	144 (133, 154)	141 (130, 151)	**0.001**
Platelet (x10^9/L), median (IQR)	221 (187, 261.5)	217 (181.5, 263)	0.480
Neutrophil absolute value, median (IQR)	4.5 (3.515, 5.985)	4.9 (3.785, 6.45)	**0.005**
Neutrophil (%), median (IQR)	64.8 (58, 71.35)	67.2 (60.8, 75.8)	**< 0.001**
ALT (U/L), median (IQR)	19 (13, 28)	18 (13, 26)	0.152
AST (U/L), median (IQR)	20 (17, 24)	19.5 (16, 24.25)	0.363
Total protein (g/L), median (IQR)	67 (63.5, 70.5)	65.85 (62.7, 69.925)	**0.006**
Albumin (g/L), median (IQR)	38.9 (36.9, 41.05)	38.3 (36.05, 40.5)	**< 0.001**
Globulin (g/L), median (IQR)	28.2 (26, 30.9)	28.5 (26, 31.3)	0.342
Total cholesterol (mmol/L), median (IQR)	4.52 (3.86, 5.23)	4.235 (3.4875, 5.06)	**< 0.001**
Triglyceride (mmol/L), median (IQR)	1.36 (1.01, 1.86)	1.3 (0.98, 1.74)	0.127
Creatinine (umol/L), median (IQR)	70.2 (60, 81.35)	72.6 (60.6, 87.25)	**0.010**
Prothrombin time (sec), median (IQR)	11.5 (11.1, 12)	11.6 (11.2, 12.1)	**0.030**
Activated partial thromboplastin time (sec), median (IQR)	24.2 (21.9, 27)	24.5 (22.3, 27.575)	0.095
LDL (mmol/L), median (IQR)	2.82 (2.35, 3.33)	2.56 (2.07, 3.1575)	**< 0.001**
HDL (mmol/L), median (IQR)	1.05 (0.91, 1.21)	1.01 (0.88, 1.18)	**0.004**
Homocysteine (umol/L), median (IQR)	12.75 (10.1, 16.85)	12.665 (9.005, 16.6)	0.321
ALP (U/L), median (IQR)	82 (70, 98)	83 (71, 97)	0.546
Uric Acid (umol/L), median (IQR)	305 (244, 373.25)	301.5 (243, 364)	0.360
BUN (mmol/L), median (IQR)	5.1 (4.2, 6.2)	5.3 (4.2, 6.4)	0.094
d-dimer (mL/L FEU), median (IQR)	0.37 (0.22, 0.73)	0.49 (0.29, 0.91)	**< 0.001**
Fibrinogen (g/L), median (IQR)	2.87 (2.45, 3.36)	3.09 (2.58, 3.84)	**< 0.001**
CRP (mg/L), median (IQR)	1.515 (0.565, 4.295)	2.58 (0.849, 13.22)	**< 0.001**
Fasting blood-glucose (mmol/L), median (IQR)	5.685 (4.9275, 7.4125)	5.9 (4.91, 7.74)	0.288
Glycosylated hemoglobin (%), median (IQR)	7.1 (6, 8.4)	7.2 (6.5, 8.6)	0.079

(*Continued*)

Table 5 (Continued).

Characteristics	Ctrl (n=971)	First Admission (n=453)	P Value
NIHSS score on admission, median (IQR)	5 (2, 8)	6 (3, 9)	**< 0.001**
GCS score, median (IQR)	15 (15, 15)	15 (15, 15)	**0.026**
MRS Score on admission, median (IQR)	0 (0, 0)	1 (0, 2)	**< 0.001**

Notes: P-values below 0.05, indicating statistical significance, are shown in bold.
Abbreviations: IQR, interquartile range; Hb, hemoglobin; ALT, glutamic-pyruvic transaminase; AST, glutamic oxalacetic transaminase; LDL, low-density lipoprotein; HDL, high-density lipoprotein; ALP, alkaline phosphatase; BUN, blood urea nitrogen; CRP, C-reactive protein; NIHSS, National Institutes of Health Stroke Scale; GCS, Glasgow score; MRS, Modified Rankin Scale.

LDL and MRS Score on admission were the risk factors between non-hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence (Table 6). ROC analysis was performed to demonstrate MRS Score on admission was a diagnostic biomarker for recurrence of cerebral infarction in patients with non-hemorrhagic cerebral infarction (AUC = 0.708, Figure 2).

There were differences at the baseline characteristics of the hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence, including age, hypertension history, diabetes history, total cholesterol,

Table 6 Univariate and Multivariate Analysis of Non-Hemorrhagic Cerebral Infarction Patients with or without First Admission

Characteristics	Univariate Analysis		Multivariate Analysis	
	Odds Ratio (95% CI)	P Value	Odds Ratio (95% CI)	P Value
Age	1.034 (1.023–1.045)	**< 0.001**	1.020 (1.001–1.039)	**0.041**
Hypertension history	1.951 (1.468–2.594)	**< 0.001**	2.470 (1.491–4.093)	**< 0.001**
Coronary artery disease history	2.037 (1.531–2.710)	**< 0.001**	1.236 (0.767–1.991)	0.384
Diabetes history	1.841 (1.465–2.313)	**< 0.001**	1.435 (0.964–2.134)	0.075
Thrombolytic	0.488 (0.296–0.805)	**0.005**	0.504 (0.165–1.537)	0.228
Hb (g/L)	0.990 (0.983–0.996)	**0.003**	0.998 (0.986–1.010)	0.725
Neutrophil absolute value	1.079 (1.027–1.133)	**0.003**	1.036 (0.925–1.161)	0.541
Neutrophil (%)	1.028 (1.015–1.041)	**< 0.001**	1.009 (0.982–1.038)	0.511
Total protein (g/L)	0.970 (0.950–0.990)	**0.004**	0.969 (0.930–1.010)	0.140
Albumin (g/L)	0.942 (0.912–0.973)	**< 0.001**	1.021 (0.947–1.102)	0.582
Total cholesterol (mmol/L)	0.810 (0.726–0.904)	**< 0.001**	1.701 (0.945–3.062)	0.076
Creatinine (umol/L)	1.007 (1.002–1.012)	**0.003**	1.004 (0.997–1.010)	0.254
Prothrombin time (sec)	1.067 (0.957–1.190)	0.241		
LDL (mmol/L)	0.692 (0.595–0.805)	**< 0.001**	0.390 (0.177–0.858)	**0.019**
HDL (mmol/L)	0.428 (0.261–0.704)	**< 0.001**	0.559 (0.207–1.512)	0.252
d-dimer (mL/L FEU)	1.084 (1.002–1.174)	**0.046**	1.018 (0.885–1.172)	0.798
Fibrinogen (g/L)	1.344 (1.178–1.533)	**< 0.001**	1.120 (0.970–1.293)	0.124
NIHSS score on admission	1.051 (1.031–1.072)	**< 0.001**	0.978 (0.932–1.027)	0.378
GCS score	0.903 (0.844–0.966)	**0.003**	0.912 (0.773–1.075)	0.273
MRS Score on admission	2.295 (2.026–2.600)	**< 0.001**	1.755 (1.498–2.057)	**< 0.001**

Notes: P-values below 0.05, indicating statistical significance, are shown in bold.
Abbreviations: CI, confidence interval; Hb, hemoglobin; LDL, low-density lipoprotein; HDL, high-density lipoprotein; NIHSS, National Institutes of Health Stroke Scale; GCS, Glasgow score; MRS, Modified Rankin Scale.

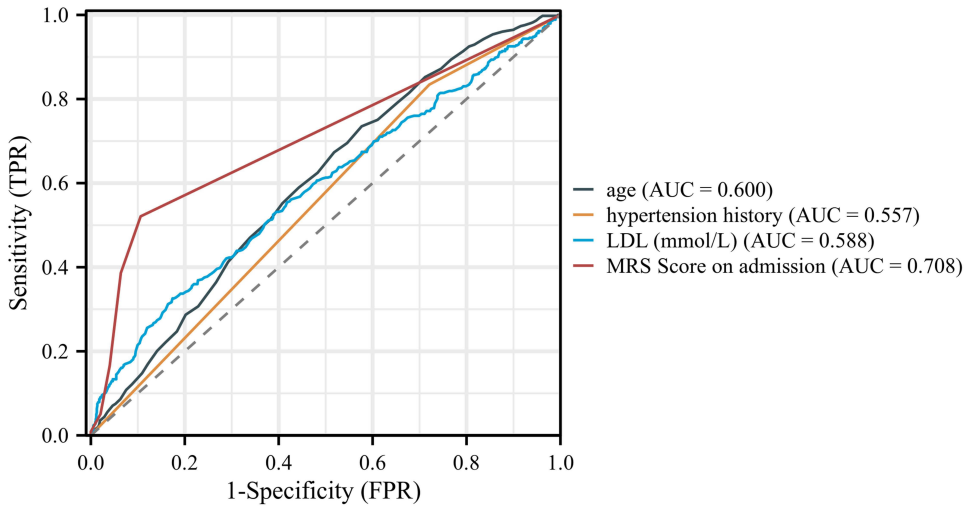

Figure 2 The diagnostic value between non-hemorrhagic cerebral infarction patients with or without first admission.

triglyceride, creatinine, PT, APTT, LDL and BUN (Table 7). Utilizing univariate and multivariate analysis, only hypertension history was the risk factors between hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence (Table 8).

Table 7 The Baseline Characteristics of Hemorrhagic Cerebral Infarction Patients with or without First Admission

Demographic Information			
Characteristics	**Ctrl (n=288)**	**First Admission (n=145)**	**P Value**
Demographics			
Age, median (IQR)	65 (57, 72)	68 (62, 73)	**0.019**
Gender, n (%) Female Male	 90 (31.3%) 198 (68.7%)	 34 (23.4%) 111 (76.6%)	0.090
Smoke, n (%)	143 (49.7%)	82 (56.6%)	0.175
Comorbidities			
Hypertension history, n (%)	191 (66.3%)	114 (78.6%)	**0.008**
Coronary artery disease history, n (%)	67 (23.3%)	45 (31.0%)	0.081
Atrial fibrillation history, n (%)	41 (14.2%)	22 (15.2%)	0.794
Diabetes history, n (%)	84 (29.2%)	58 (40.0%)	**0.023**
Patients on anticoagulants			
Use anticoagulants, n (%)	13 (4.5%)	5 (3.4%)	0.600
Acute recanalization therapy			
Thrombolytic, n (%)	108 (37.5%)	48 (33.1%)	0.368

(*Continued*)

Table 7 (Continued).

Demographic Information			
Characteristics	**Ctrl (n=288)**	**First Admission (n=145)**	**P Value**
Laboratory data			
Hb (g/L), median (IQR)	140 (126, 150.25)	138 (128, 150)	0.640
Platelet (x10^9/L), median (IQR)	219 (181, 260.25)	208 (168.25, 259)	0.224
Neutrophil absolute value, median (IQR)	5.49 (3.8625, 8.59)	5.64 (3.895, 8.9775)	0.386
Neutrophil (%), median (IQR)	71.45 (62.675, 80.45)	72.85 (63.75, 84.6)	0.208
ALT (U/L), median (IQR)	16 (12, 24)	17 (12, 29)	0.254
AST (U/L), median (IQR)	21 (17, 27)	22 (18, 28)	0.276
Total protein (g/L), median (IQR)	68.8 (65.05, 73.95)	69.2 (64.7, 72.8)	0.519
Albumin (g/L), median (IQR)	39.35 (36.3, 42.225)	38.1 (35.8, 41.3)	0.055
Globulin (g/L), median (IQR)	29.3 (26.8, 33.1)	29.7 (27.2, 33.1)	0.545
Total cholesterol (mmol/L), median (IQR)	4.57 (3.705, 5.32)	4.05 (3.16, 4.875)	**< 0.001**
Triglyceride (mmol/L), median (IQR)	1.16 (0.83, 1.645)	1.03 (0.76, 1.44)	**0.037**
Creatinine (umol/L), median (IQR)	67.9 (57.375, 81.125)	75 (60.5, 88.8)	**0.003**
Prothrombin time (sec), median (IQR)	11.4 (10.7, 12)	11.6 (11, 12.3)	**0.004**
Activated partial thromboplastin time (sec), median (IQR)	23.2 (20.65, 25.65)	24.75 (22.875, 27.6)	**< 0.001**
LDL (mmol/L), median (IQR)	2.89 (2.315, 3.485)	2.46 (1.92, 3.12)	**< 0.001**
HDL (mmol/L), median (IQR)	1.08 (0.9, 1.31)	1.06 (0.805, 1.27)	0.187
Homocysteine (umol/L), median (IQR)	0 (0, 13.52)	0 (0, 12.318)	0.119
ALP (U/L), median (IQR)	83 (68, 101)	81 (67, 101)	0.434
Uric Acid (umol/L), median (IQR)	292 (238, 369)	290 (225, 368)	0.558
BUN (mmol/L), median (IQR)	5.5 (4.4, 6.6)	6 (4.7, 7.4)	**0.013**
d-dimer (mL/L FEU), median (IQR)	0 (0, 0.6375)	0 (0, 0.83)	0.373
Fibrinogen (g/L), median (IQR)	3.04 (2.4275, 3.67)	2.955 (2.47, 3.7525)	0.806
CRP (mg/L), median (IQR)	0 (0, 3.22)	0.1715 (0, 3.385)	0.742
Fasting blood-glucose (mmol/L), median (IQR)	5.635 (4.8975, 8.2)	6.37 (5.14, 8.55)	0.184
Glycosylated hemoglobin (%), median (IQR)	0 (0, 6)	0 (0, 6.6)	0.374
NIHSS score on admission, median (IQR)	6 (2, 12)	7 (2, 13)	0.276
GCS score, median (IQR)	15 (14, 15)	15 (15, 15)	0.275
MRS Score on admission, median (IQR)	3 (1, 4)	3 (1, 4)	0.615

Notes: P-values below 0.05, indicating statistical significance, are shown in bold.

Abbreviations: IQR, interquartile range; Hb, hemoglobin; ALT, glutamic-pyruvic transaminase; AST, glutamic oxalacetic transaminase; LDL, low-density lipoprotein; HDL, high-density lipoprotein; ALP, alkaline phosphatase; BUN, blood urea nitrogen; CRP, C-reactive protein; NIHSS, National Institutes of Health Stroke Scale; GCS, Glasgow score; MRS, Modified Rankin Scale.

Table 8 Univariate and Multivariate Analysis of Hemorrhagic Cerebral Infarction Patients with or without First Admission

Characteristics	**Total(N)**	**Univariate Analysis**		**Multivariate Analysis**	
		Odds Ratio (95% CI)	**P value**	**Odds Ratio (95% CI)**	**P value**
Age	433	1.023 (1.004–1.043)	0.018	1.014 (0.993–1.035)	0.195
Hypertension history	433				
No	128	Reference		Reference	
Yes	305	1.868 (1.172–2.977)	0.009	1.758 (1.068–2.893)	**0.026**
Diabetes history	433				
No	291	Reference		Reference	
Yes	142	1.619 (1.066–2.460)	0.024	1.466 (0.928–2.315)	0.101
Total cholesterol (mmol/L)	426	0.902 (0.806–1.010)	0.073	1.160 (0.796–1.691)	0.439
Triglyceride (mmol/L)	426	0.837 (0.652–1.073)	0.159		

(*Continued*)

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Table 8 (Continued).

Characteristics	Total(N)	Univariate Analysis		Multivariate Analysis	
		Odds Ratio (95% CI)	P value	Odds Ratio (95% CI)	P value
Creatinine (umol/L)	433	1.012 (1.005–1.019)	< 0.001	1.006 (0.996–1.016)	0.237
Prothrombin time (sec)	427	1.068 (1.002–1.139)	0.044	1.028 (0.934–1.132)	0.572
Activated partial thromboplastin time (sec)	427	1.034 (1.006–1.062)	0.018	1.024 (0.984–1.066)	0.248
LDL (mmol/L)	433	0.797 (0.669–0.949)	0.011	0.629 (0.352–1.124)	0.118
BUN (mmol/L)	430	1.125 (1.045–1.212)	0.002	1.054 (0.946–1.173)	0.341

Notes: P-values below 0.05, indicating statistical significance, are shown in bold.
Abbreviations: LDL, Low-density lipoprotein; BUN, blood urea nitrogen.

Discussion

In the present study, records of 1857 consecutive treated stroke patients were reviewed, of whom 23.3% were diagnosed with HCI. In this study, we analyzed the risk factors of first admission and recurrence of HCI and ROC analysis was performed to show the diagnostic value. Utilizing univariate and multivariate analysis, age, hypertension history, LDL and MRS Score on admission were the risk factors between non-hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence. ROC analysis was performed to demonstrate that glycosylated hemoglobin was a diagnostic biomarker for the patients with hemorrhagic cerebral infarction and non-hemorrhagic cerebral infarction (AUC = 0.808). Utilizing univariate and multivariate analysis, only hypertension history was the risk factors between hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence.

Disruption of the BBB is a common pathway for hemorrhagic transformation after acute ischemic stroke.[7,8] Cytokines in an inflammatory state can accelerate endothelial damage and pathological changes in blood vessel walls, thereby increasing the risk of vessel rupture and cerebral hemorrhage.[8,15] In the present study, patients with hemorrhagic cerebral infarction were older and had higher neutrophil infiltration, AST expression, globulin, and BUN compared with patients with non-hemorrhagic cerebral infarction. Albumin, as the most abundant protein in plasma, plays a crucial role in maintaining colloid osmotic pressure within blood vessels.[16] When albumin levels are low, the colloid osmotic pressure decreases, causing water to leak from the blood vessels into surrounding tissues, thereby increasing the pressure on the vessel walls.[17] In the brain, this imbalance in osmotic pressure can lead to cerebral edema, exacerbating existing damage such as HCI. The specific mechanism is that edema not only increases intracranial pressure but may also trigger or worsen cerebral hemorrhage by increasing the tension on blood vessel walls and the risk of rupture.[18] Secondly, the relationship between albumin levels and systemic inflammation status indicates its role in the pathophysiological processes.[19] Low albumin levels are often associated with increased inflammation, as under inflammatory conditions, the synthesis of albumin by the liver decreases, while the activity of inflammatory cytokines increases. Finally, the antioxidant properties of albumin are crucial in preventing oxidative stress-induced damage to brain cells. Albumin can neutralize free radicals, reduce oxidative damage, and protect cells from harm.[20] In the context of HCI, low levels of albumin weaken this protective mechanism, making brain cells more susceptible to attacks from free radicals and increasing the risk of cell death and tissue damage. Additionally, oxidative stress can further promote inflammatory reactions, exacerbating brain tissue damage and creating a vicious cycle.[21] Therefore, maintaining appropriate levels of albumin is crucial for protecting brain health and preventing HCI.

In the present study, thrombolysis was a risk factor in patients with both hemorrhagic and nonhemorrhagic cerebral infarction. Reperfusion with alteplase (recombinant tissue plasminogen activator) or endovascular therapy (EVT) commonly exacerbates hemorrhagic transformation of cerebral infarction.[22] For patients at high risk of bleeding, it is important to carefully assess the indications and contraindications of thrombolytic therapy and monitor changes in patients after treatment to promptly detect and manage potential bleeding complications.[23] Current research has started to utilize advanced imaging techniques and biomarkers to assess the risk of bleeding after thrombolytic therapy. For example, MRI and CT imaging can help evaluate the condition of intracranial vessels and identify potential areas of hemorrhage.[24]

Platelets play a crucial role in the blood coagulation process, maintaining vascular integrity, and promoting wound healing.[25] However, in certain pathological conditions, an imbalance in platelet function may lead to increased bleeding or a tendency towards clotting, thereby affecting the risk of HCI.[26] A decrease in platelets weakens the blood's ability to clot, increasing the tendency to bleed, including intracerebral hemorrhage.[27] Abnormal platelet activation and aggregation are one of the main mechanisms leading to HCI.[28]

Anticoagulants are considered a significant risk factor for HCI.[29] Anticoagulants, particularly oral anticoagulants such as warfarin and novel oral anticoagulants, are highly effective in preventing thrombus formation, but they increase the potential risk of HCI.[30] This risk is closely related to dose management, patient-specific coagulation parameters, and individual bleeding tendencies. Particularly in the presence of other hemorrhagic risk factors such as hypertension history, cerebral hemorrhage history, renal insufficiency and advanced age, this risk is further increased.[31] The development of these models has provided better guidance for reducing hemorrhagic events in patients with AF while optimizing stroke prevention strategies.[32] To optimize treatment strategies, physicians need to use precise risk assessment tools, such as the CHA2 DS2-VASc scoring system, to evaluate the stroke risk in patients with AF, and compare it against the HAS-BLED score to assess bleeding risk, in order to devise personalized anticoagulation treatment plans.[33]

In the pathophysiology of HCI, the dysregulation of low-density lipoprotein (LDL) plays a significant role. This dysregulation may impact the stability and integrity of cerebral blood vessels through various mechanisms.[34] The dysregulation of LDL may lead to endothelial damage. Elevated levels of LDL can promote inflammation reactions in arterial endothelial cells, increasing the fragility of the vascular endothelium and thereby raising the risk of cerebral vascular rupture.[35] LDL particles can become oxidized to form oxidized low-density lipoprotein (ox-LDL). This form of LDL has a high degree of cytotoxicity and can stimulate the aggregation and activation of inflammatory cells, leading to endothelial damage and changes in vascular wall structure.[36] Additionally, the dysregulation of LDL may also affect the activation and aggregation processes of platelets.[37] In the context of HCI, this increased platelet activity may lead to an increased risk of microvascular rupture and local bleeding. Simultaneously, the dysregulation of LDL may affect the fibrinolysis process, reducing fibrinolytic activity and leading to difficulty in clot dissolution. This can result in the formation of persistent obstruction in cerebral blood vessels.[38] In addition, dysregulation of LDL may accelerate vascular sclerosis by promoting lipid deposition and fibrosis in the vascular wall, reducing vascular elasticity.[39] These hardened vessels are more susceptible to rupture under conditions of hypertension or other stressors, leading to hemorrhagic events.[40] Therefore, maintaining LDL within normal levels may be of significant importance in preventing HCI.

Hypertension has also been shown to increase the risk of HCI.[41] Hypertension acts through a number of different mechanisms, including increased inflammation, vascular remodeling with implications for collateral and autoregulation, and direct stress on the cerebrovascular system.[42] It may also lead to disruption of the BBB.[8,43] In addition, we found that a history of hypertension was an independent risk factor for recurrent cerebral infarction in patients with HCI. Therefore, it is essential for the management of blood pressure.

Conclusion

In conclusion, age, hypertension history, LDL and MRS Score on admission were the risk factors between non-hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence, while only hypertension history was the risk factors between hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence. ROC analysis was performed to demonstrate that glycosylated hemoglobin was a diagnostic biomarker for the patients with hemorrhagic cerebral infarction and non-hemorrhagic cerebral infarction (AUC = 0.808).

Highlights

- Glycosylated hemoglobin was a diagnostic biomarker for the patients with hemorrhagic cerebral infarction and non-hemorrhagic cerebral infarction.
- MRS Score on admission was a diagnostic biomarker for recurrence of non-hemorrhagic cerebral infarction.
- Only hypertension history was the risk factors between hemorrhagic cerebral infarction patients with first admission or the cerebral infarction recurrence.

- Age, hypertension history, LDL and MRS Score on admission were the risk factors between cerebral infarction patients with first admission or the cerebral infarction recurrence.

Data Sharing Statement

The datasets analyzed in this study are available from the corresponding author upon reasonable request.

Consent for Publication

All the authors have agreed to the submission and publication of this paper.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas, took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published, have agreed on the journal to which the article has been submitted, and agree to be accountable for all aspects of the work.

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Disclosure

All authors report no conflicts of interest in this work.

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