

Original Article

Risk factors and preventive strategies for postoperative cerebral infarction in patients with hypertensive intracerebral hemorrhage

Lixia Yu, Ping Xuan, Zifang Li, Hongmin Chen

Rehabilitation Medicine, Yiwu Central Hospital, No. 699 Jiangdong Road, Yiwu 322000, Zhejiang, China

Received March 23 2025; Accepted July 28 2025; Epub September 15, 2025; Published September 30, 2025

Abstract: Objectives: To identify the risk factors and evaluate preventive strategies for postoperative cerebral infarction (CI) in patients with hypertensive intracerebral hemorrhage (HICH). Methods: This retrospective study included 268 patients who underwent surgery for HICH at Yiwu Central Hospital between November 2022 and November 2024. Among them, 50 developed postoperative CI (CI group), while 218 patients did not (non-CI group). Logistic regression analysis was used to identify independent risk factors for postoperative CI, and receiver operating characteristic (ROC) curves were generated to assess the predictive performance of the identified variables. Results: Among the 268 HICH patients, 50 patients developed postoperative CI, resulting in an incidence of 18.66%. Compared to the non-CI group, patients in the CI group had notably lower Glasgow Coma Scale (GCS) scores, and larger hematomas and higher brain edema volumes (all $P < 0.001$). Logistic regression analysis identified systolic blood pressure ($P < 0.001$; OR: 1.169; CI: 1.099-1.244), diastolic blood pressure ($P = 0.027$; OR: 1.094; CI: 1.010-1.185), GCS score ($P = 0.017$; OR: 0.753; CI: 0.596-0.951), and hematoma volume ($P = 0.004$; OR: 1.229; CI: 1.067-1.414) as independent risk factors for postoperative CI in HICH patients. The combined predictive model yielded an area under the ROC curve (AUC) of 0.867. Conclusions: Elevated systolic and diastolic blood pressure, lower GCS scores, and larger hematoma volume are independent risk factors for postoperative CI in HICH patients.

Keywords: Hypertensive intracerebral hemorrhage, GCS score, cerebral infarction, hematoma volume, risk factor

Introduction

Hypertension is a leading cause of spontaneous intracerebral hemorrhage, and hypertensive intracerebral hemorrhage (HICH) is not only common in the elderly but also increasingly prevalent in middle-aged and young individuals [1]. Chronic elevation of blood pressure leads to arteriosclerosis in cerebral arteries, characterized by deposition of amorphous material between the intima and elastic lamina, which reduces vascular elasticity and increases fragility [2]. Sustained hypertension may promote the formation of microaneurysms at sites exposed to high hemodynamic stress. Additionally, blood infiltration into the vessel wall can further contribute to aneurysm formation. A sudden surge in blood pressure increases the risk of aneurysm rupture, resulting in intracranial hemorrhage and subsequent hemorrhagic stroke [3, 4].

HICH is the most common form of non-traumatic intracerebral hemorrhage, characterized by rapid onset and high rates of disability and mortality [5]. After HICH, hematoma formation in brain parenchyma compresses adjacent arteries and veins, leading to ischemia, hypoxia, and tissue necrosis. Additionally, the degradation of erythrocytes within the hematoma releases hemoglobin and other toxic metabolites, exacerbating secondary brain injury and perihematomal edema [6]. In severe cases, brain herniation can occur [7].

Craniotomy for hematoma evacuation remains the primary treatment for many HICH patients; however, postoperative complications are common, with cerebral infarction (CI) being a major concern [8]. Particularly, large-area infarction induces extensive ischemic necrosis or softening of brain tissue in patients, severely diminishing their quality of life and prognosis [8, 9].

The occurrence of postoperative CI significantly increases the disability and mortality rates in patients with HICH, affecting patient safety and prognosis [10]. Therefore, identifying risk factors for postoperative CI is crucial in clinical practice, as it enables the implementation of protective measures to prevent postoperative CI [9]. However, effective early warning indicators for postoperative CI following HICH remain lacking in current clinical practice, limiting the ability to implement proactive strategies and reduce its incidence. Therefore, this study aimed to investigate the predictors and independent risk factors for postoperative CI in HICH patients, to enable prevention.

Patients and methods

Data source

A retrospective analysis was conducted on the clinical data of 300 patients who underwent surgical treatment for HICH at Yiwu Central Hospital between November 2022 and November 2024. This retrospective study was approved by the ethics committee of Yiwu Central Hospital. Due to the anonymized nature of the data and the study's retrospective design, the requirement for informed consent was waived in compliance with institutional guidelines and applicable regulatory standards for minimal-risk retrospective research.

Inclusion criteria: Diagnosis of HICH confirmed by brain computed tomography (CT) and in accordance with the diagnostic criteria of the *Chinese Guidelines for Cerebral Hemorrhage* [11]; Admission within 24 hours of symptom onset; patients who underwent surgical intervention with evident clinical efficacy and no other postoperative complications; Availability of complete clinical data; age >18 years.

Exclusion criteria: patients with cerebral hemorrhage caused by other diseases; patients with concurrent dysfunction of vital organs such as the lungs, kidneys, or liver; patients with hemorrhage localized to the brainstem, cerebellum, or infratentorial regions; patients with a history of psychiatric disorders or cognitive impairment; patients with previous CI; pregnant women.

Based on the above criteria, a total of 268 patients who underwent surgical treatment for

HICH were ultimately included in the study. Postoperative cranial CT scans performed within 2 weeks of surgery were reviewed to assess the occurrence of CI. Patients were then categorized into two groups based on the occurrence of postoperative CI: 50 patients who developed postoperative CI were included in the CI group, while the remaining 218 patients were included in the non-CI group.

Research method

General information and clinical data of the patients were collected by reviewing electronic medical records and imaging data. The collected information mainly included age, gender, body mass index (BMI), time from symptom onset to hospital admission, length of hospital stay, history of hypertension, systolic and diastolic blood pressure at admission, history of diabetes, place of residence, Glasgow Coma Scale (GCS) score, hematoma volume, brain edema volume, and adherence to standardized surgical procedures during operation.

Outcome measures

Primary outcome measures: (1) The preoperative GCS scores in the CI group and non-CI group were analyzed and compared. One day before surgery, each patient's level of consciousness was evaluated using the GCS, which assesses eye-opening, verbal response, and motor response. Total scores of these three components indicates the degree of consciousness impairment: mild impairment (13-15), moderate impairment (9-12), and severe impairment (3-8) [12]. (2) The hematoma and brain edema volumes were measured one day preoperatively. Based on CT images, hematoma volume was estimated using the following formula: Hematoma volume = 0.5 × maximum long axis (cm) × maximum short axis (cm) × number of slices, assuming a slice thickness of 1 cm [13]. For brain edema, the area of edema was delineated on each CT slice using an image analysis system, and total edema volume was calculated by summing the edema areas and multiplying by slice thickness [14]. (3) To identify independent risk factors for postoperative CI, a univariate analysis was first conducted on each collected variable. Variables with statistical significance were then included in a multivariate logistic regression model to determine

Postoperative CI risk factors in HICH

Table 1. Baseline data of included patients

Factor	CI group (n=50)	Non-CI group (n=218)	χ^2/t	P
Age (year)	52.65±3.61	53.03±3.31	0.728	0.468
BMI (kg/m ²)	22.97±1.92	23.08±1.95	1.575	0.117
Sex			2.396	0.122
Male	29	100		
Female	21	118		
Time from onset to hospital admission (h)	12.38±2.76	12.85±2.78	1.087	0.278
Length of hospital stay (days)	23.51±5.83	22.98±8.21	1.409	0.644
History of hypertension (Year)	10.74±2.39	9.64±1.86	3.871	0.001
Systolic blood pressure (mmHg)	157.25±7.34	149.27±6.33	7.802	<0.001
Diastolic blood pressure (mmHg)	99.52±5.86	95.28±4.32	5.829	<0.001
History of diabetes			2.357	0.125
Yes	19	59		
No	31	159		
Place of incidence			1.329	0.249
Rural areas	34	129		
Urban areas	16	89		

Note: BMI: Body mass index.

independent predictors of postoperative CI in HICH patients.

Secondary outcome measures: (1) The clinical baseline data of both groups were collected, including age, body mass index (BMI), gender, time from symptom onset to hospital admission, length of hospital stay, history of hypertension, systolic blood pressure, diastolic blood pressure, history of diabetes, and place of residence. (2) The overall incidence of CI among the included patients was calculated.

Statistical analysis

Statistical analysis was conducted using SPSS 20.0 (IBM Corp, Armonk, NY, USA), and graph plotting was done by GraphPad Prism 7 (GraphPad Software, San Diego, USA). Counted data were presented as [n (%)], and inter-group comparisons were conducted using the chi-square test. Measured data were normally distributed and expressed as ($\bar{x} \pm s$). Comparisons of measured data between the two groups were performed using independent sample t-tests, and within-group comparisons using paired t-tests. Logistic regression analysis was used to identify independent risk factors for postoperative CI in patients with HICH. $P < 0.05$ was considered a significant difference.

Results

Clinical baseline data

A comparison of baseline characteristics between the two groups revealed no significant differences in terms of age, BMI, gender, time from symptom onset to hospital admission, history of diabetes, or place of residence ($P > 0.05$). However, differences were observed between the two groups regarding hypertension history, systolic blood pressure, and diastolic blood pressure ($P \leq 0.001$, **Table 1**).

Incidence and timing of postoperative CI

Among the 268 included HICH patients, 50 developed postoperative CI, yielding an incidence of 18.66%. The onset of CI occurred between 1 and 14 days postoperatively, with an average of (5.10 ± 2.01) days. Specifically, 17 cases occurred within 1-4 days, 31 cases within 5-9 days, and 2 cases within 10-14 days postoperatively, as detailed in **Table 2**.

GCS scores

Preoperative GCS score were significantly lower in the CI group (10.61 ± 1.80) compared to the non-CI group (12.28 ± 1.73) ($P < 0.001$, **Figure 1**).

Table 2. Incidence and timing of postoperative CI

Item	Statistics
Total number of cases	268
Number of patients with cerebral infarction (incidence rate)	50 (18.66%)
Time of occurrence of cerebral infarction (days)	5.10±2.01
Distribution of occurrence time of cerebral infarction	
1-4 days postoperatively	17
5-9 days postoperatively	31
10-14 days postoperatively	2

Note: CI: cerebral infarction.

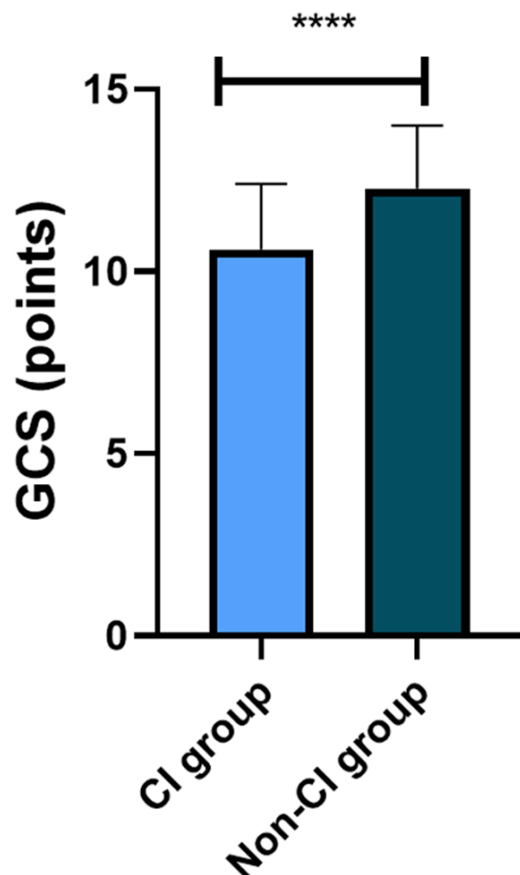


Figure 1. Comparison of GCS scores between the two groups. ****P<0.0001. GCS: Glasgow coma scale.

Hematoma volume and cerebral edema volume

Hematoma and cerebral edema volumes were compared between the two groups. In the CI group, the mean hematoma volume and cerebral edema volume were 25.57±1.78 mL and 25.64±8.72 cm³, respectively. In the non-CI group, these volumes were 22.49±1.78 mL and 23.73±6.29 cm³, respectively. The CI group

showed significantly higher hematoma volume and brain edema volume than the non-CI group (both P<0.05, **Figure 2**).

Univariate analysis of postoperative CI in HICH patients

Based on the comparative results, univariate analysis was conducted to explore factors associated with the occurrence of postoperative CI in HICH patients.

Differences were observed between the two groups in terms of a history of hypertension, systolic blood pressure, diastolic blood pressure, GCS score, hematoma volume, and cerebral edema volume (P<0.050). These factors were identified as potential influencing factors for the occurrence of postoperative CI (**Table 3**).

Multivariate logistic regression analysis of postoperative CI in HICH patients

In the multivariate logistic regression analysis, postoperative CI (occurred =1, not occurred =0) was set as the dependent variable, with a history of hypertension, systolic blood pressure, diastolic blood pressure, GCS score, hematoma volume, and cerebral edema volume as the independent variables. Logistic regression analysis identified systolic blood pressure (P<0.001; OR: 1.169; CI: 1.099-1.244), diastolic blood pressure (P=0.027; OR: 1.094; CI: 1.010-1.185), GCS score (P=0.017; OR: 0.753; CI: 0.596-0.951), and hematoma volume (P=0.004; OR: 1.229; CI: 1.067-1.414) as independent risk factors for postoperative CI in HICH patients (**Table 4**).

Predictive efficacy of independent risk factors and their combined detection

To evaluate the predictive performance of each independent risk factor and their combined detection, ROC curves were generated. The combined model yielded an AUC of 0.867, which was superior to the predictive performance of any individual variable (**Figure 3**; **Table 5**).

Discussion

Hypertension is a common chronic condition in clinical practice and a well-established risk

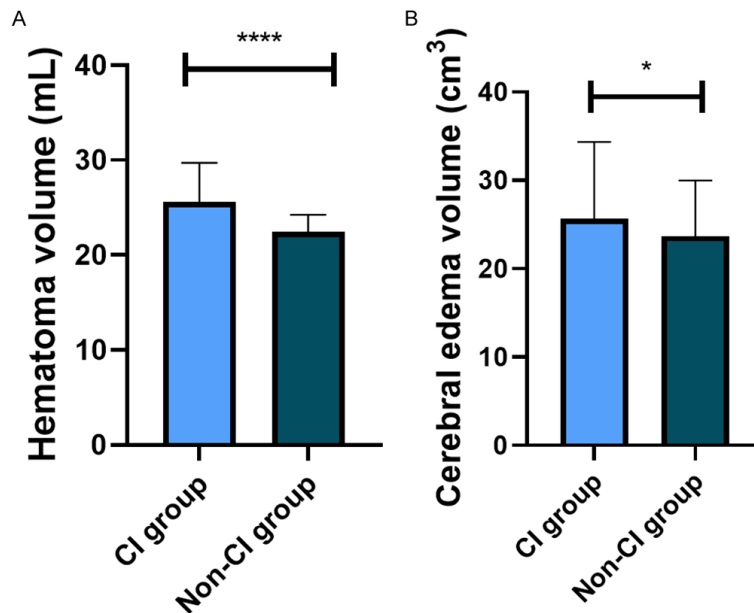


Figure 2. Comparison of hematoma volume and cerebral edema volume between the two groups. A: Comparison of hematoma volume between the two groups; B: Comparison of cerebral edema volume between the two groups. * $P < 0.05$; **** $P < 0.0001$.

factor for stroke, which can trigger hypertensive intracerebral hemorrhage (HICH) [15]. Surgical evacuation of hematoma remains the mainstay of treatment for HICH. However, surgery is a traumatic therapeutic approach that may lead to postoperative CI [16, 17]. This study investigated the incidence and independent risk factors for postoperative CI in patients with HICH, and explored potential preventive measures.

In this study, out of the 268 patients included, 50 patients developed postoperative CI, resulting in an incidence rate of 18.66%, which is notably higher than 11.1% reported by Lin et al. [18]. The relative high incidence in this study may be caused by the limited sample size and a higher proportion of patients with more severe presentations. In this study, the majority of postoperative CI cases occurred between 5-9 days postoperatively. Likewise, a study by Lin et al. [19] defines early postoperative cerebral infarction (ePCI) as occurring within 72 hours post-surgery, and delayed-type CI occurring between 3-14 days. This distinction likely reflects different pathologic mechanisms: ePCI primarily results from intraoperative mechanical traction or acute hypoperfusion, whereas delayed CI (DCI) is more likely associated with hypertensive vasculopathy [20]. These findings

underscore the need for time window-specific prevention strategies in clinical practice.

This study identified a history of hypertension, systolic and diastolic blood pressure, GCS score, hematoma volume, and cerebral edema volume as influencing factors for postoperative CI in patients with HICH. Elevated systolic and diastolic blood pressure, lower GCS score, and higher hematoma volume were independent risk factors for postoperative CI in patients with HICH. Possible reasons for this observation may be multifactorial. The arterial blood vessel wall is composed of three layers, with the innermost intima being relatively smooth and tightly bound under normal

conditions [21]. As indicated in a study by Totoń-Żurańska et al. [22], chronic uncontrolled hypertension activates the Rho/ROCK signaling pathway and pro-inflammatory factors, inducing vascular endothelial cell apoptosis and extracellular matrix degradation, compromising microstructural integrity of the vascular wall. Under physiologic conditions, cholesterol participates in the normal metabolism [23]; However, vessel endothelium damage caused by hypertension facilitates cholesterol infiltration and plaque formation on vessel walls [24]. Without timely intervention, these plaques gradually accumulate and rupture under continued hypertensive stress, leading to thrombosis, cerebral vessel occlusion, ischemia, and subsequent CI [25, 26]. Xu et al. [27] identified hypertension as one of the primary risk factors for HICH, which aligns with the conclusion of this study.

Lower GCS scores reflect decreased neurological function and consciousness levels, indicative of severe neurological injury [28]. In HICH patients, lower GCS scores may correlate with the severity of bleeding, compromised cerebral perfusion pressure, and deterioration of overall health, all of which increase CI susceptibility. Therefore, intensified monitoring and care,

Postoperative CI risk factors in HICH

Table 3. Univariate analysis of postoperative CI in HICH patients

Factor	CI group (n=50)	Non-CI group (n=218)	χ^2/t	P
History of hypertension (Year)	10.74±2.39	9.64±1.86	3.871	0.001
Systolic blood pressure (mmHg)	157.25±7.34	149.27±6.33	7.802	<0.001
Diastolic blood pressure (mmHg)	99.52±5.86	95.28±4.32	5.829	<0.001
GCS score	10.61±1.80	12.28±1.73	6.100	<0.001
Hematoma volume (mL)	25.57±1.78	22.49±1.78	8.233	<0.001
Cerebral edema volume (cm ³)	25.64±8.72	23.73±6.29	2.181	0.034

Notes: CI: Cerebral infarction; HICH: Hypertensive intracerebral hemorrhage; GCS: Glasgow coma scale.

Table 4. Multivariate logistic regression analysis of postoperative CI in HICH patients

Factor	B	S.E.	Wals	df	Sig.	Exp (B)	95% C.I. For EXP(B)	
							Lower limit	Upper limit
History of hypertension	0.152	0.104	2.130	1	0.144	1.164	0.949	1.426
Systolic blood pressure	0.156	0.031	24.701	1	<0.001	1.169	1.099	1.244
Diastolic blood pressure	0.090	0.041	4.880	1	0.027	1.094	1.010	1.185
GCS	-0.284	0.119	5.650	1	0.017	0.753	0.596	0.951
Hematoma volume	0.206	0.072	8.228	1	0.004	1.229	1.067	1.414
Cerebral edema volume	0.056	0.030	3.554	1	0.059	1.058	0.998	1.121

Notes: CI: Cerebral infarction; HICH: Hypertensive intracerebral hemorrhage; GCS: Glasgow coma scale.

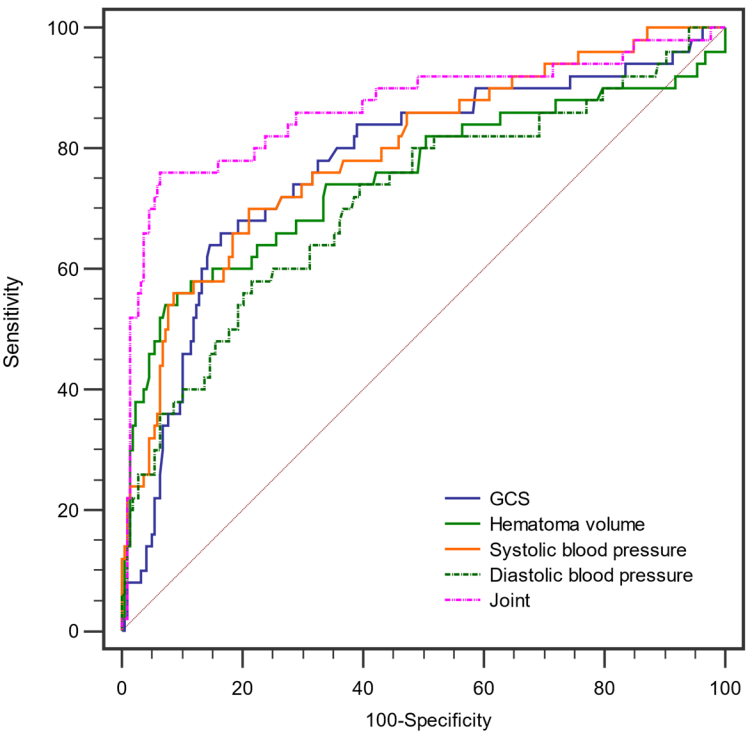


Figure 3. ROC curves of independent risk factors and their combination for predicting postoperative CI in HICH patients. Notes: CI: Cerebral infarction; HICH: Hypertensive intracerebral hemorrhage; GCS: Glasgow coma scale.

patients with lower GCS scores to mitigate CI risk.

Hematoma volume plays a critical role in CI risk stratification. Larger hematomas mechanically compress surrounding brain tissue and exacerbate cerebral edema, leading to intracranial hypertension and cerebral perfusion deficits [29]. This dual pathophysiology underscores the importance of timely hematoma evacuation in eligible patients. Supporting this, Wenz et al. [30] demonstrated that decompressive craniectomy combined with hematoma evacuation reduced DCI-related infarctions by 60% in aneurysmal subarachnoid hemorrhage patients (OR=0.2), despite not improving functional outcome. Their findings, along with our data, suggest that timely surgical intervention may mitigate secondary ischemic injury caused by mass effect. However, high complication rates (51% in their cohort) necessitate careful patient selection.

including respiratory management and strict blood pressure control, should be provided for

high complication rates (51% in their cohort) necessitate careful patient selection.

Table 5. ROC data of independent risk factors and their combination for predicting postoperative CI in HICH patients

	AUC	Standard deviation	Asymptotic 95% confidence interval	
			Upper limit	Lower limit
GCS	0.771	0.040	0.693	0.849
Hematoma volume	0.751	0.046	0.660	0.841
Systolic blood pressure	0.792	0.037	0.719	0.866
Diastolic blood pressure	0.714	0.044	0.627	0.800
Joint	0.867	0.035	0.798	0.935

Notes: ROC: Receiver operating characteristic; AUC: Area-under-the-curve; CI: Cerebral infarction; HICH: Hypertensive intracerebral hemorrhage; GCS: Glasgow coma scale.

IN summary, this study explored the combined predictive efficacy of systolic blood pressure, diastolic blood pressure, GCS score, and hematoma volume for postoperative CI in patients with HICH. The model achieved an AUC of 0.867, outperforming the predictive accuracy of each individual factor alone. This finding indicates that the combined use of these four independent risk factors significantly enhances the prediction of postoperative CI in HICH patients. Our CT-based predictive model offers clinically meaningful accuracy while maintaining practical advantages in cost-effectiveness and accessibility compared to MRI-dependent approaches, particularly valuable for resource-limited clinical settings. This pragmatic tool enables clinicians to assess infarction risk promptly using routinely available preoperative data, facilitating timely clinical decision-making and individualized preventive strategies. Methodologically, this study complements the work of Lee et al. [31] on postoperative CI in moyamoya disease. Their PCT-RAPID analysis identified Tmax >6 s volume >59.5 mL as an independent predictor of post-bypass infarction (AUC=0.811). Despite differences in disease pathology, both studies underscore the predictive value of quantitative perfusion indices and volumetric parameters for postoperative ischemic events. This convergence of findings suggests future potential for integrating perfusion CT-derived metrics into HICH risk prediction models to further refine CI risk stratification.

This study has some limitations. First, as a retrospective analysis, our findings may be susceptible to biases from unmeasured confounding factors inherent in observational designs; Second, the restricted scope of variables might have influenced the robustness of our conclu-

sions; Third, by focusing exclusively on supratentorial HICH cases, the results cannot be generalized to hemorrhages in the brainstem or cerebellum, which have distinct pathophysiologic characteristics. Fourth, the lack of formal inter-rater reliability assessment for hematoma volume measurements may have introduced measurement variability. Therefore, future prospective multi-center studies with broader inclusion criteria are warranted.

Conclusion

Elevated systolic and diastolic blood pressure, lower GCS score, and larger hematoma volume were independent risk factors for postoperative CI in HICH patients. Early identification and preoperative intervention should be actively pursued to reduce the risk of postoperative CI and improve patient outcomes.

Disclosure of conflict of interest

None.

Address correspondence to: Hongmin Chen, Rehabilitation Medicine, Yiwu Central Hospital, No. 699 Jiangdong Road, Yiwu 322000, Zhejiang, China. E-mail: 15805799302@163.com

References

[1] Kitada K. Hypertension research 2024 update and perspectives: basic research. *Hypertens Res* 2024; 47: 3304-3309.

[2] Chen F, Zhang S, Li B, Zhang J, Ran M and Qi B. A review of invasive intracranial pressure monitoring following surgery for hypertensive cerebral hemorrhage. *Front Neurol* 2023; 14: 1108722.

[3] Yamada SM, Tomita Y, Iwamoto N, Takeda R, Nakane M, Aso T and Takahashi M. Subcortical hemorrhage caused by cerebral amyloid

- angiopathy compared with hypertensive hemorrhage. *Clin Neurol Neurosurg* 2024; 236: 108076.
- [4] Jiang L, Tian J, Guo C, Zhang Y, Qian M, Wang X, Wang Z and Chen Y. Comparison of the efficacy of neuronavigation-assisted intracerebral hematoma puncture and drainage with neuro-endoscopic hematoma removal in treatment of hypertensive cerebral hemorrhage. *BMC Surg* 2024; 24: 86.
- [5] Xie Q, Zhang X, Ding Y and Zhang L. Assessing the clinical efficacy of stereotactic puncture in combination with postoperative rehabilitation training for hypertensive cerebral hemorrhage. *Altern Ther Health Med* 2024; 30: 122-127.
- [6] Xiao Y and Wang X. Omaha system-based extended nursing care in hypertensive cerebral hemorrhage: a randomized study. *J Trauma Nurs* 2024; 31: 319-329.
- [7] Okamoto T, Kotsugi M, Kakehi Y, Sasaki H, Morisaki Y, Maeoka R, Yokoyama S and Nakagawa I. Thalamic hemorrhage due to ruptured aneurysm at the feeder of a tentorial dural arteriovenous fistula in the Galen region mimicking a hypertensive hemorrhage. *J Stroke Cerebrovasc Dis* 2025; 34: 108166.
- [8] Nakamura M and Aihara M. Initial treatment of cerebral hemorrhage: determining the cause without preconceived notions. *No Shinkei Geka* 2023; 51: 1022-1032.
- [9] Sekiguchi Y, Nishitani M, Takizawa H, Iizuka H, Mitsumori T, Tomita S, Izumi H, Okubo M, Miyake K, Osawa T, Sawada T, Yoshikawa S, Okura H and Noguchi M. Low-risk essential thrombocythemia who presented with recurrent episodes of cerebral hemorrhage during pregnancy and developed cerebral infarction during puerperium. *Gan To Kagaku Ryoho* 2021; 48: 725-730.
- [10] Anthofer J, Bele S, Wendl C, Kieninger M, Zeman F, Bruendl E, Schmidt NO and Schebesch KM. Continuous intra-arterial nimodipine infusion as rescue treatment of severe refractory cerebral vasospasm after aneurysmal subarachnoid hemorrhage. *J Clin Neurosci* 2022; 96: 163-171.
- [11] Zheng WJ, Shi SW and Gong J. The truths behind the statistics of surgical treatment for hypertensive brainstem hemorrhage in China: a review. *Neurosurg Rev* 2022; 45: 1195-1204.
- [12] Ansari A, Zoghi S, Khoshbooei A, Mosayebi MA, Feili M, Yousefi O, Niakan A, Kouhpayeh SA, Taheri R and Khalili H. Development of a novel neurological score combining GCS and FOUR scales for assessment of neurosurgical patients with traumatic brain injury: GCS-FOUR scale. *World Neurosurg* 2024; 182: e866-e871.
- [13] Cao L, Liu M, Wang M, Ding J, Mao K, Liu K and Li S. 3D slicer-based calculation of hematoma irregularity index for predicting hematoma expansion in intracerebral hemorrhage. *BMC Neurol* 2022; 22: 452.
- [14] Choi HA, Bajgur SS, Jones WH, Savarraj JP, Ko SB, Edwards NJ, Chang TR, Hergenroeder GW, Dannenbaum MJ, Chen PR, Day AL, Kim DH, Lee K and Grotta JC. Quantification of cerebral edema after subarachnoid hemorrhage. *Neurocrit Care* 2016; 25: 64-70.
- [15] Xu X, Zhang H, Zhang J, Luo M, Wang Q, Zhao Y, Gan Z, Xu B and Chen X. Minimally invasive surgeries for spontaneous hypertensive intracerebral hemorrhage (MISICH): a multicenter randomized controlled trial. *BMC Med* 2024; 22: 244.
- [16] Ai M, Zhang H, Feng J, Chen H, Liu D, Li C, Yu F and Li C. Research advances in predicting the expansion of hypertensive intracerebral hemorrhage based on CT images: an overview. *PeerJ* 2024; 12: e17556.
- [17] Zhou Z, Yang Z, Jiang S, Zhuo J, Zhu T and Ma S. Surgical navigation system for hypertensive intracerebral hemorrhage based on mixed reality. *J Digit Imaging* 2022; 35: 1530-1543.
- [18] Lin K, Gao C, Lin Z, Tang Y, Lin Y and Jiang R. Characteristics, predictors and outcomes of early postoperative cerebral infarction on computed tomography in spontaneous intracerebral hemorrhage. *Sci Rep* 2024; 14: 19526.
- [19] Lin K, Zhan ZY, Tong YX, Lin ZC, Tang YH and Lin YX. Development and external validation of a prediction model for early postoperative cerebral infarction on computed tomography in spontaneous intracerebral hemorrhage. *Neurocrit Care* 2025; 43: 298-307.
- [20] Kumar A, Brown R, Dhar R, Sampson T, Derdeyn CP, Moran CJ and Diringner MN. Early vs. delayed cerebral infarction after aneurysm repair after subarachnoid hemorrhage. *Neurosurgery* 2013; 73: 617-623.
- [21] Kulkarni S and Wilkinson IB. Adrenoceptors and hypertension. *Handb Exp Pharmacol* 2024; 285: 297-332.
- [22] Totoń-Żurańska J, Mikołajczyk TP, Saju B and Guzik TJ. Vascular remodelling in cardiovascular diseases: hypertension, oxidation, and inflammation. *Clin Sci (Lond)* 2024; 138: 817-850.
- [23] Luo J, Yang H and Song BL. Mechanisms and regulation of cholesterol homeostasis. *Nat Rev Mol Cell Biol* 2020; 21: 225-245.
- [24] Su X, Rao H, Zhao C, Wu J, Zhang X and Li D. Association between the non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio and mortality among hypertension patients. *Sci Rep* 2025; 15: 6012.
- [25] Zheng D, Li X and Fu Y. Risk factors of acute cerebral infarction in patients with primary hy-

- pertension. *Ir J Med Sci* 2023; 192: 2441-2445.
- [26] Jaroonpipatkul C, Onwanna J, Tunvirachaisakul C, Jittapiromsak N, Rakvongthai Y, Chutinet A, Supasitthumrong T and Maes M. Depressive symptoms due to stroke are strongly predicted by the volume and location of the cerebral infarction, white matter hyperintensities, hypertension, and age: a precision nomothetic psychiatry analysis. *J Affect Disord* 2022; 309: 141-150.
- [27] Xu L, Wang Z, Wu W, Li M and Li Q. Global, regional, and national burden of intracerebral hemorrhage and its attributable risk factors from 1990 to 2021: results from the 2021 global burden of disease study. *BMC Public Health* 2024; 24: 2426.
- [28] Aguilar-Fuentes V, Orozco-Puga P and Jiménez-Ruiz A. The glasgow coma scale: 50-year anniversary. *Neurol Sci* 2024; 45: 2899-2901.
- [29] Chihi M, Darkwah Oppong M, Pierscianek D, Dinger TF, Ahmadipour Y, Gembruch O, Deuschl C, Li Y, Schmidt B, Wrede KH, Sure U and Jabbarli R. Analysis of brain natriuretic peptide levels after traumatic acute subdural hematoma and the risk of post-operative cerebral infarction. *J Neurotrauma* 2021; 38: 3068-3076.
- [30] Wenz F, Ziebart A, Hackenberg KAM, Rinkel GJE, Etminan N and Abdulazim A. Primary decompressive craniectomy in patients with large intracerebral hematomas due to aneurysmal subarachnoid hemorrhage. *Acta Neurochir (Wien)* 2024; 166: 332.
- [31] Un Lee S, Lee Y, Won Choi T, Hyo Lee S, Kim YD, Pil Ban S, Kwon OK, Wan Oh C and Seung Bang J. Prediction of postoperative cerebral infarction after combined bypass surgery in adult moyamoya disease: combining quantitative parameters on RAPID perfusion CT with clinically related factors. *J Neurosurg* 2024; 141: 966-974.