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ORIGINAL RESEARCH

Clinical and Imaging Predictors of Hematoma Expansion in Spontaneous Intracerebral Hemorrhage: Development of a Prognostic Model

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Background: Identifying risk factors associated with hematoma expansion following spontaneous intracerebral hemorrhage (ICH) is essential for improving early intervention strategies. We hope to use this predictive model in the future to comprehensively score the risk factors of hospitalized patients with cerebral hemorrhage and evaluate the possibility of hematoma enlargement. Being able to identify high-risk patients with hematoma enlargement early and take intervention measures to save their lives.

Methods: A retrospective analysis was conducted on clinical data from 226 individuals diagnosed with spontaneous ICH between December 29, 2023, and August 29, 2024. Multiple logistic regression analysis was performed to identify risk factors associated with hematoma expansion. Predictive performance of the model was assessed using ROC curve analysis and receiver operating characteristic curve analysis. Mortality rates were calculated for each group following a 7-day follow-up period.

Results: Hematoma expansion was associated with diabetes mellitus, a low Glasgow Coma Scale (GCS) score at admission, elevated systolic blood pressure at admission, coagulation abnormalities, and specific computed tomography (CT) imaging findings, such as heterogeneous density, black hole sign, swirl sign, lobulation sign, and blend sign. A prognostic model incorporating these factors demonstrated robust predictive performance, achieving an area under the curve of 0.771 (95% CI: 0.628–0.915, $p = 0.002$). The model yielded a maximum Youden index of 0.489, with an optimal cutoff score of 38, a sensitivity of 54.5%, and a specificity of 94.4%. Mortality among individuals with coagulation abnormalities was 53.3%.

Conclusion: Coagulation abnormalities, GCS score, systolic blood pressure at admission, CT imaging findings, and diabetes mellitus were identified as predictors of hematoma expansion in spontaneous ICH. Individuals with coagulopathy and elevated systolic blood pressure at admission exhibited the poorest prognoses.

Keywords: cerebral hematoma, hematoma expansion, mortality, predictive model, risk factors

Introduction

Spontaneous intracerebral hemorrhage (ICH) represents the second most prevalent form of stroke and is associated with high mortality and morbidity.[1] Although it primarily affects middle-aged and older adults, its incidence among younger individuals has risen in recent years. Mortality rates for ICH range from 28% to 55%, with spontaneous ICH accounting for 10% to 20% of all stroke cases in Europe and the United States. In China, approximately 30% of stroke cases involve spontaneous ICH.[2]

Diagnosis of cerebral hemorrhage is primarily conducted using head computed tomography (CT), a non-invasive and rapid imaging technique. Hematoma expansion following ICH has been associated with an increased risk of mortality

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and poor clinical outcomes.[3] Therefore, strategies aimed at preventing hematoma expansion are critical for improving treatment efficacy and patient prognosis.

Multiple studies have examined risk factors associated with hematoma expansion following spontaneous ICH.[4] Elevated blood glucose levels have been suggested to increase the risk of hematoma expansion, potentially due to glucose-induced vascular injury. Coagulopathy, characterized by an increased international normalized ratio (INR) or a reduced platelet count, has also been correlated with a higher likelihood of hematoma expansion.[5] Additionally, chronic uncontrolled hypertension is a major risk factor for spontaneous ICH and may contribute to hematoma expansion through vascular injury.[6] Evidence from one study indicated that individuals with an initial hematoma volume exceeding 30 mL exhibited a significantly greater risk of hematoma expansion, with larger initial hematomas more likely to undergo unilateral enlargement.[7]

In clinical practice, there are many risk factors associated with the expansion of cerebral hemorrhage. To address this issue, we collected specific clinical factors (such as age, gender, coagulation function, Glasgow Coma Scale (GCS) score, systolic blood pressure at admission, initial hematoma volume, and head CT imaging characteristics) and hypothesized that these factors are independent predictors of spontaneous intracerebral hemorrhage hematoma enlargement. We conducted statistical research and analysis on these factors, and identified clinical factors that met the hypothesis as risk factors for hematoma enlargement, thus establishing a predictive model.

This study aimed to investigate the relationship between hematoma expansion and both clinical and imaging markers in individuals with spontaneous ICH. Risk factors associated with hematoma expansion were identified, and follow-up evaluations were conducted for those receiving conservative management to assess the predictive value of these factors. Furthermore, predictive scores for hematoma expansion were developed based on these risk factors, providing a framework for clinical risk assessment and supporting the development of individualized treatment strategies.

Method

Patient Selection

A retrospective analysis was conducted on individuals diagnosed with primary ICH through head CT imaging at the Department of Neurosurgery, Wuxi People's Hospital, between December 2023 and August 2024. Written informed consent was obtained from all participants or their legal guardians. The study received approval from the Ethics Committee of Wuxi People's Hospital, affiliated with Nanjing Medical University (approval number: KY21088).

Inclusion Criteria

Were as follows: (1) Diagnosis of acute ICH confirmed by an initial head CT scan, with symptom onset occurring within 8 hours. (2) Initiation of conservative treatment within 24 hours of hospital admission.

Exclusion Criteria

Were as follows: (1) Individuals with cerebral hemorrhage secondary to intracranial aneurysms, vascular malformations, moyamoya disease, intracranial space-occupying lesions (eg, brain tumors or strokes), congenital hematological disorders, traumatic injuries, vascular amyloidosis, or hemorrhage resulting from cerebral infarction. (2) Presence of a large hematoma on the initial head CT scan requiring urgent surgical intervention. (3) A prior history of stroke.

CT Imaging Analysis

All individuals underwent an initial head CT scan at the time of admission, followed by a re-evaluation after 24 hours.[8] Raw image data in DICOM format were imported into the 3D Slicer image analysis system, which automatically identified and marked pixels corresponding to the hematoma. The CT threshold range was manually set between 50 and 100 Hounsfield units (HU), and the three-dimensional structure and volume of the hematoma were rendered using the "MakeModel" function. Hematoma volume was calculated based on CT images, and hematoma expansion was defined as an increase in volume of at least 33% or 12.5 mL upon follow-up imaging compared to the initial scan.[9] Based on this criterion, individuals were categorized into the hematoma expansion group (n = 70) and the non-expansion group (n = 154).

CT imaging characteristics were assessed according to the following definitions: (1) Irregular density: An irregular hematoma shape with heterogeneous density was observed within its internal region and margins.[10] (2) Black hole sign: A circular, oval, or rod-like area of relatively low density within a high-density region, with low-density areas not adjacent to surrounding tissue and exhibiting well-defined borders. The CT attenuation difference between high- and low-density areas was at least 28 HU.[11] (3) Swirl sign: A low-density or isodense area within a high-density hematoma, appearing in a circular, linear, or irregular shape.[12] (4) Lobulated hematoma: A hematoma displaying scattered or irregular projections at its margins on non-contrast CT imaging.[13] (5) Blend Sign: A hematoma containing two distinct areas of differing density, with a CT attenuation difference of at least 18 HU. The boundary between these regions was clearly defined and visually distinguishable, with the relatively low-density area not completely enclosed by the high-density region.[14]

CT imaging findings were independently evaluated by radiologists and neurosurgeons at Wuxi People's Hospital. In cases of discrepancies, final determinations were reached through discussion and consensus.

Data Collection

Data were collected on age, gender, coagulation function, Glasgow Coma Scale (GCS) score, systolic blood pressure at admission (The inclusion criteria is the diagnostic criteria for hypertension, which is greater than 140mmHg), initial hematoma volume, and head CT imaging characteristics (Figure 1). Coagulation dysfunction was defined as a thromboelastographic reaction time (R) > 10 minutes or a maximum amplitude (MA) < 54 millimeters.

Data Analysis

Statistical analyses were conducted using SPSS 22.0. Continuous variables were expressed as mean ± standard deviation (SD) or median ± interquartile range (IQR), while categorical variables were reported as counts with percentages (0–100%). The normality of data distribution was assessed using the Shapiro–Wilk test. Normally distributed variables were presented as mean (±SD). Comparisons between continuous variables were performed using the Mann–Whitney *U*-test, whereas binary categorical variables were analyzed using Pearson's chi-square test.

Survival curves were generated using the Kaplan-Meier method, and survival differences between groups were assessed with the Log rank test. Univariate and multivariate logistic regression analyses were conducted to determine risk factors associated with hematoma expansion. Variables with a $p < 0.05$ in univariate analysis were included in the multivariate logistic regression model to establish predictive factors. The predictive performance of the model was evaluated using receiver operating characteristic (ROC) curve analysis, with the area under the curve (AUC) calculated to assess discrimination ability. A p value of < 0.05 was considered statistically significant.

Results

Clinical Data

A total of 226 individuals were included in the study, comprising 148 males and 78 females. The median initial hematoma volume was 19.9 mL (range: 10–29 mL). CT image analysis showed irregular density at the hematoma margin in 71 individuals, the presence of a black hole sign in 56, a vortex sign in 51, a lobulated hematoma in 53, and a blend hematoma in 58 patients.

Key Factors Associated with Hematoma Expansion

Age, sex, and initial hematoma volume did not show statistically significant differences between the hematoma expansion and non-expansion groups ($p > 0.05$). However, the enlarged hematoma group had a higher proportion of individuals with a history of diabetes mellitus and coagulation abnormalities compared to the non-expansion group. The hematoma expansion group exhibited a higher proportion of individuals with lower GCS scores, elevated systolic blood pressure at admission, and specific head CT imaging signs, such as irregular density at the hematoma margin, black hole sign, vortex sign, lobulated hematoma, and blend hematoma ($p < 0.05$,see Table 1).

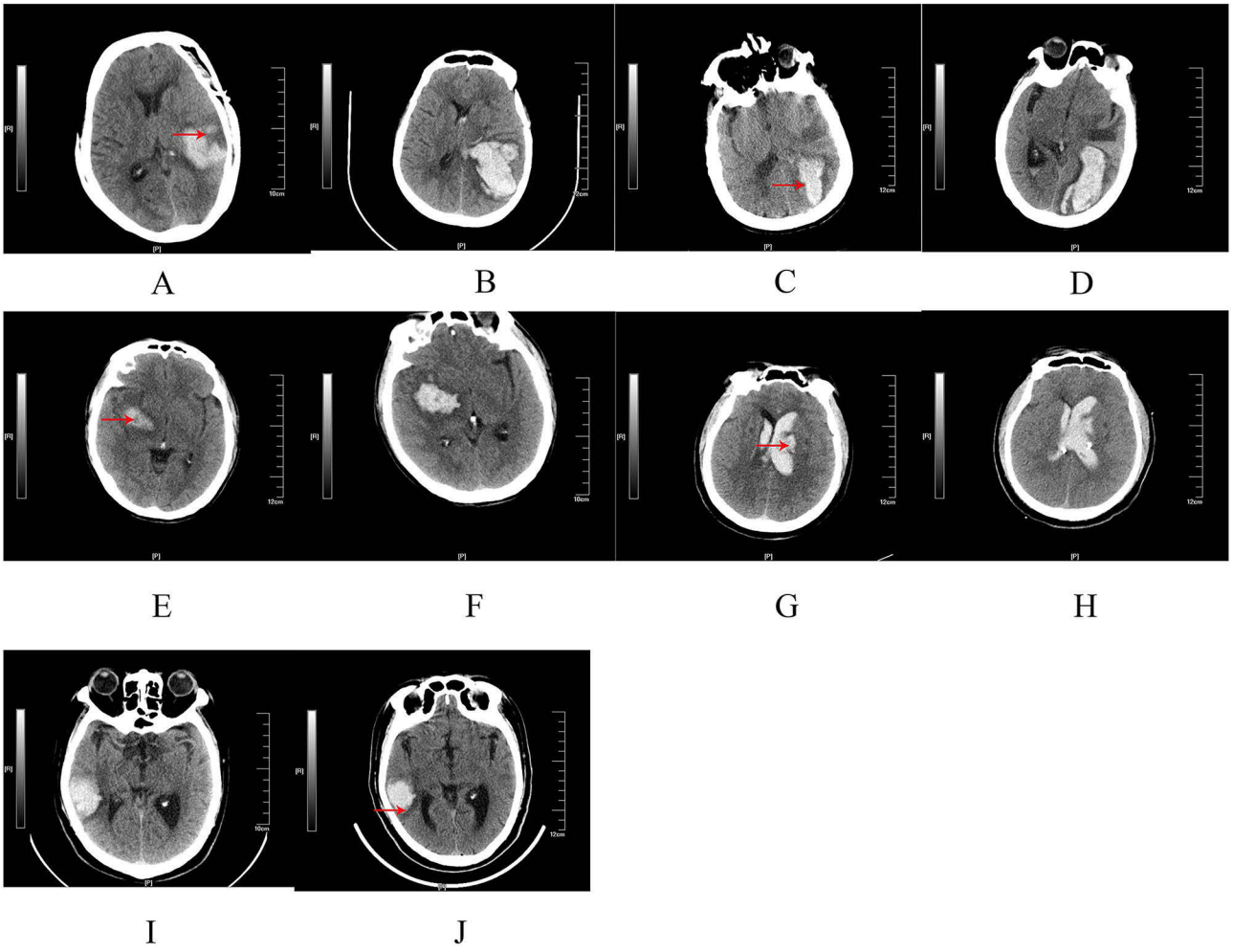

Figure 1 Representative CT axial images taken on admission and at 24-hour follow-up. (**A**) The first CT scan of a patient with left temporal parietal lobe hemorrhage showed irregular density at the edge of the hematoma (as indicated by the arrow). (**B**) The follow-up CT scan showed a significant enlargement of the hematoma compared to that on admission. (**C**) The first CT scan of a patient with left temporal occipital lobe hemorrhage showed black hole sign (as indicated by the arrow). (**D**) The follow-up CT scan showed significant enlargement of hematoma. (**E**) The first CT scan of a patient with bleeding in the right basal ganglia area showed vortex sign (indicated by the arrow). (**F**) The follow-up CT scan showed a significant enlargement of the hematoma. (**G**) The first CT scan of a patient with left basal ganglia rupture into bilateral ventricles showed lobed sign (indicated by the arrow). (**H**) The follow-up CT scan showed hematoma enlargement. (**I**) The first CT scan of a patient with right temporal lobe hemorrhage showed mixed sign (indicated by the arrow). (**J**) The follow-up CT scan showed hematoma enlargement.

We conducted multicollinearity analysis on these statistically significant risk factors, and the results showed that there was no multicollinearity among these risk factors (see Table 2).

Consequently, a history of diabetes mellitus, lower GCS scores at admission, elevated systolic blood pressure, coagulation abnormalities, and specific CT imaging signs were identified as significant risk factors for hematoma expansion in individuals with ICH.

Prediction of Hematoma Enlargement

The predictive ability of each risk factor for hematoma expansion was analyzed. While each factor demonstrated high specificity, sensitivity was generally low, with some factors exhibiting sensitivity levels below 50% (see Table 3). After analyzing the results of the single factor and multiple factor unconditional logistic regression mentioned above, we identified independent risk factors such as abnormal coagulation function, GCS score, admission blood pressure, irregular edge mixed density sign, black hole sign, vortex sign, lobulation sign, and confounding sign in CT imaging. Evaluating hematoma enlargement in patients through a single independent risk factor may be limited. We assign values to each independent related risk factor and calculate the comprehensive score for each patient, which serves as the basis for predicting hematoma enlargement. We multiply the beta coefficient of each

Table 1 Patient Characteristics by Hematoma Status

Variable	Hematoma		P-Value
	Yes (n=71)	No (n=155)	
Age (years)	62.3±16.6	65.1±13.7	0.607
Sex			0.103
Male (%)	45 (63.4)	81 (52.3)	
Female (%)	26 (36.6)	74 (47.7)	
Diabetes (%)	41 (57.7)	44 (28.4)	<0.001
Median hematoma volume (cm^3)	20.2 (14.5–23.4)	18.7 (13.1–23.9)	0.732
Systolic blood pressure on admission, mm Hg	192.6±21.3	167.2±19.5	0.006
Median GCS at admission	7 (5–8)	10 (8–12)	<0.001
Abnormal coagulation function (%)	39 (55)	11 (7.1)	<0.001
Irregular density sign (%)	38 (53.5)	33 (21.3)	<0.001
Black hole sign (%)	31 (43.7)	25 (16.1)	<0.001
Vortex sign (%)	29 (40.8)	22 (14.2)	<0.001
Lobed sign (%)	25 (35.2)	28 (18.1)	0.003
Mixed sign (%)	28 (39.4)	30 (19.3)	0.001

Notes: Values are expressed as numbers (%) or as median (range).

Table 2 Multicollinearity Analysis on Statistically Significant Risk Factors

Model	Unstandardized B	Coefficients Std.Error	Standardized Coefficients Beta	t	Sig.	Collinearity Tolerance	Statistics VIF
Systolic blood pressure on admission	0.000	0.001	0.053	0.337	0.739	0.677	1.478
GCS at admission	0.000	0.009	−0.008	−0.041	0.967	0.479	2.089
Abnormal coagulation function	0.001	0.001	0.145	0.999	0.328	0.799	1.252
Irregular density sign	−0.007	0.008	−0.180	−0.849	0.404	0.372	2.686
Black hole sign	0.001	0.000	0.518	3.010	0.006	0.567	1.764
Vortex sign	0.037	0.015	0.512	2.488	0.020	0.396	2.528
Lobed sign	0.000	0.000	0.215	1.124	0.272	0.459	2.180
Mixed sign	0.000	0.000	0.176	0.911	0.371	0.447	2.236
Diabetes	−0.023	0.009	−0.441	−2.604	0.016	0.585	1.709

Table 3 Sensitivity, Specificity, and Predictive Values of Qualitative Predictors for Intracranial Hematoma Enlargement

	Sensibility	Specificity	Negative Predictive value	Positive Predictive Value
Systolic blood pressure on admission	58.1%	83.2%	71.6%	62.9%
GCS at admission	47.1%	90.9%	79.1%	70.2%
Abnormal coagulation function	52.9%	91.6%	81.0%	74%
Irregular density sign	54.3%	78.6%	79.1%	53.5%
Black hole sign	44.3%	83.8%	76.8%	55.4%
Vortex sign	41.4%	85.7%	76.3%	56.9%
Lobed sign	35.7%	81.8%	73.7%	47.1%
Mixed sign	40.0%	80.5%	74.7%	48.3%
Diabetes	45.2%	72.3%	75.6%	69.4%

Table 4 Multivariate Logistic Regression Analysis of Factors Affecting the Expansion of Hematoma in Patients with Spontaneous Cerebral Hemorrhage

Variable	β	SE	Wald X^2	Odds Ratio	95% Confidence Interval	P	Scores
Diabetes, number	2.174	0.532	16.718	8.797	3.102–24.946	<0.01	22
Systolic blood pressure on admission, mmhg	1.424	0.515	7.654	4.154	1.515–11.392	0.006	14
GCS at admission, score	−1.394	0.465	8.981	0.248	0.100–0.617	0.003	14
Abnormal coagulation function, number	2.380	0.558	18.212	10.809	3.622–32.253	<0.01	24
Irregular density sign (CT), number	1.391	0.499	7.752	4.017	1.509–10.692	0.005	14
Black hole sign (CT), number	1.597	0.512	9.750	4.939	1.812–13.462	0.002	16
Vortex sign (CT), number	1.227	0.509	5.810	3.410	1.258–9.245	0.016	12
Lobed sign (CT), number	1.098	0.474	5.357	2.998	1.183–7.597	0.021	11
Mixed sign (CT), number	1.197	0.481	6.198	3.311	1.290–8.495	0.013	12

risk factor by 10 and round it to the nearest integer to obtain the score for that factor. The sum of the scores for each risk factor constitutes the score for each patient's clinical prediction model (see Table 4).

The ROC curve analysis demonstrated that the model had robust predictive performance, with an AUC of 0.771 (95% CI, 0.628–0.915, P = 0.002). The model achieved a maximum Youden index of 0.489, with an optimal cutoff score of 38 points, yielding a sensitivity of 54.5% and a specificity of 94.4% (see Figure 2).

This prediction model shows the best predictive value when the cutoff value is 38 points. Among 226 patients with cerebral hemorrhage, there were a total of 91 patients with a score greater than 38. Among these 91 patients, 52

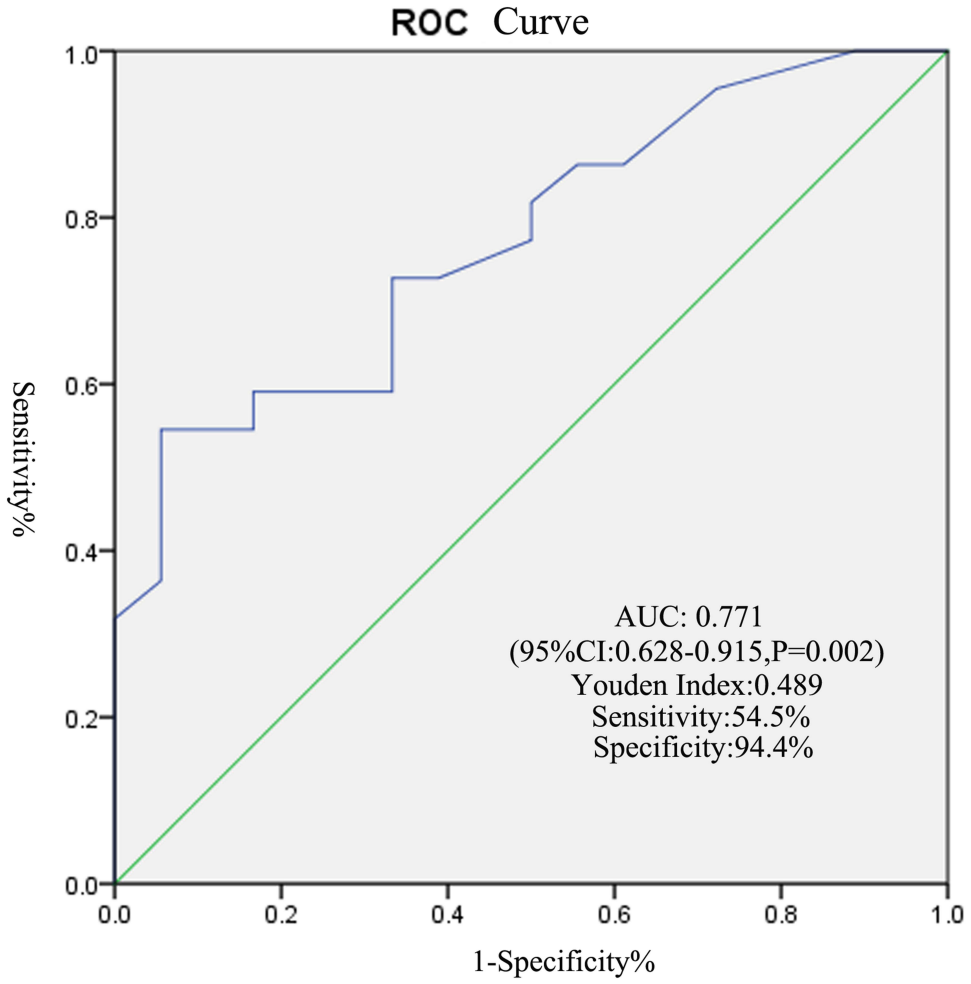

Figure 2 Receiver operating characteristic curve of the risk prediction model for hematoma enlargement after spontaneous intracerebral hemorrhage.

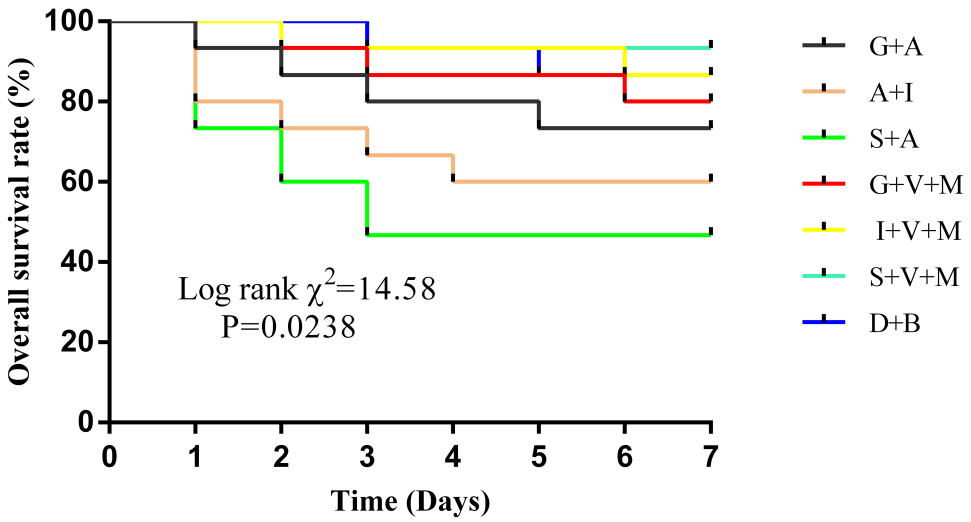

Figure 3 Survival curves of the different patient groups (G - GCS at admission, A - abnormal coagulation function, I–irregular density sign, S - systolic blood pressure on admission, V–vortex sign, M - mixed sign, D - diabetes, B - black hole sign).

experienced hematoma enlargement, with a probability of 57%. Therefore, this model has good predictive value for hematoma enlargement.

We subsequently selected 50 patients with spontaneous cerebral hemorrhage who received conservative treatment for hematoma enlargement prediction. Among them, 33 patients experienced hematoma enlargement, and 21 patients had a comprehensive risk factor score greater than 38 points. The success rate of prediction was 64%, reflecting the good predictive value of the model.

Relationship Between Risk Factors and Mortality

Since the optimal cutoff value for the model score was 38, the corresponding risk factors were selected to assess their association with mortality. Survival curves were constructed to determine which combinations of risk factors were linked to the highest mortality rates. According to literature research reports,[6] the time window for hematoma enlargement after cerebral hemorrhage is generally 3–7 days. Therefore, we set the follow-up time for hematoma enlargement to 7 days. Mortality was assessed over a 7-day follow-up period in individuals with enlarged cerebral hematomas who received conservative treatment.

As illustrated in Figure 3, individuals presenting with coagulopathy in conjunction with elevated systolic blood pressure at admission were associated with the most unfavorable prognosis, with a mortality rate of 53.3%. In contrast, individuals with a history of diabetes and the black hole sign on CT imaging demonstrated the most favorable prognosis, with a mortality rate of 6.6%.

Discussion

Several clinical factors have been associated with hematoma expansion in individuals with spontaneous ICH.[15] Notably, initial hematoma volume, systolic blood pressure at admission, diabetes, low GCS scores, abnormal liver function, abnormal renal function, and a history of alcohol abuse have been identified as risk factors for hematoma expansion in the early stages of ICH.[16]

Furthermore, the anatomical location of the hemorrhage within the brain may serve as an independent risk factor for hematoma enlargement. Hematomas located in proximity to the center of the basal ganglia have been associated with a high risk of expansion, with reported rates reaching 36.7% in this region.[17] The basal ganglia is a common site for ICH, primarily due to the presence of the lenticulostriate arteries, which traverse the internal capsule.[18] Alterations in blood flow dynamics at the bifurcations of the lenticulostriate arteries may lead to impaired perfusion.

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Furthermore, the proximity of the basal ganglia to the ventricles, along with limited structural support from surrounding tissues, reduces the self-repair mechanisms and pressure resistance of cerebral vessels. Consequently, hematomas in this region exhibit a higher propensity for significant enlargement.[19]

Individual risk factors demonstrated limited predictive value for hematoma expansion, and the relationship between initial hematoma volume and subsequent expansion remains unclear. Although multiple studies have identified initial hematoma volume as a risk factor for expansion, a study conducted in China reported no such association.[20,21] This discrepancy may be attributed to variations in the definition of hematoma expansion, as most studies classify expansion as an increase in hematoma volume of ≥ 8 cm³ or ≥ 1.2-fold.[22] In the present study, hematoma enlargement was defined as an increase in volume of $\geq 33\%$ or ≥ 12.5 mL. Based on this criterion, initial hematoma volume did not emerge as a significant risk factor for subsequent expansion.

A history of diabetes mellitus and elevated systolic blood pressure at admission were identified as risk factors for hematoma expansion, consistent with findings from previous studies.[23] Evidence suggests that elevated blood glucose levels in the early stages of ICH may indirectly enhance sodium absorption, increase blood volume, and increase the response of vascular smooth muscle to sympathetic stimulation, leading to vasoconstriction and increased blood pressure.[24] Additionally, insulin resistance associated with diabetes mellitus has been shown to increase compensatory insulin secretion, facilitating the accumulation of toxic metabolites within blood vessels. This process compromises vascular integrity and promotes hematoma expansion.[25]

Furthermore, vascular endothelial injury in individuals with ICH triggers the release of various inflammatory cytokines. In the presence of persistent hypertension, the capacity for cerebrovascular tissue repair is diminished, increasing susceptibility to rebleeding.[26]

The level of consciousness is associated with multiple factors, including the size and location of the hematoma.[27] A reduced level of consciousness often indicates severe brain injury; however, there has been limited research on the relationship between hematoma expansion and level of consciousness.[28] In the present study, hematoma enlargement was associated with lower GCS scores at admission, a finding that requires further validation in studies with larger sample sizes.

Extensive research has demonstrated that the use of antiplatelet and anticoagulant medications adversely affects platelet function and coagulation factors, particularly factor VII, leading to vascular endothelial damage, vessel rupture, and subsequent bleeding.[29] A significant association was observed between hematoma enlargement and abnormal coagulation. Previous studies have similarly reported a higher likelihood of hematoma expansion in individuals with coagulation abnormalities compared to those with normal coagulation profiles.[30] A reduction in platelet count or thrombin levels compromises platelet aggregation and inhibits the conversion of fibrinogen to fibrin, thereby facilitating hematoma expansion.

The correlation between head CT imaging features and hematoma enlargement has been extensively documented. Yang et al reported that the presence of specific imaging characteristics, including irregular blend density at the hematoma margins, the black hole sign, the vortex sign, lobulated hematoma, and blend hematoma, demonstrated improved predictive accuracy for hematoma expansion.[31] Similarly, Sato et al evaluated CT images of individuals with hematoma enlargement who underwent follow-up imaging within three hours of the initial scan. Their findings indicated the presence of intermediate- to low-density regions within or surrounding high-density lesions.[32] The authors suggested that high density hematomas developed after the initial hemorrhage, signifying active bleeding sites emerging over varying time intervals. This imaging feature may therefore serve as a predictive marker for hematoma expansion.

Several limitations of the present study should be acknowledged. The analysis did not account for hematoma location or individuals' medical histories, which may have influenced hematoma expansion. Additionally, as a single-center retrospective study, further validation through investigations with a larger sample size is necessary. Despite these limitations, a multifactorial predictive model has the potential to improve risk assessment for hematoma expansion in clinical settings and contribute to enhanced prognostic outcomes in individuals with spontaneous ICH.

Conclusion

A predictive model for hematoma expansion following ICH was developed by integrating multiple risk factors. Coagulopathy, GCS score, systolic blood pressure at admission, CT imaging characteristics, and diabetes mellitus were associated with hematoma expansion. An optimal cutoff score of 38 points was determined for predicting this

outcome. We found that the sensitivity of the ROC curve was 54.5%, which may affect its practicality in clinical diagnosis. This may be due to the retrospective single center study, which has certain limitations in the study of risk factors for spontaneous intracerebral hemorrhage hematoma enlargement. In this prediction model, we may need to add more dimensional case data to explore the risk factors for hematoma enlargement, and further expand the sample size for statistical research. Although the sensitivity of the curve is less than 70%, its specificity is higher than 70%, which can effectively exclude non cases and has certain value in clinical treatment.

Among individuals meeting this threshold, the highest mortality was observed in those with coagulopathy and elevated systolic blood pressure at admission when hematoma expansion occurred. These findings provide a novel approach for predicting hematoma expansion in individuals with ICH, facilitating early risk stratification and clinical decision-making.

Abbreviations

ICH, Intracerebral hemorrhage; ROC, Receiver operating characteristic curve; AUC, Area under the curve; CT, Computer tomography; GCS, Glasgow coma scale; DICOM, Digital imaging and communications in medicine; INR, International normalized ratio; MA, Maximum amplitude; CI, Confidence interval; SD, Standard deviation; IQR, Interquartile range.

Data Sharing Statement

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

Ethics Approval and Consent to Participate

This study was conducted with approval from the Ethics Committee of Wuxi People's Hospital of Nanjing Medical University (KY-21088). This study was conducted in accordance with the declaration of Helsinki. Written informed consent was obtained from all participants.

Consent for Publication

All participants signed a document of informed consent.

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Disclosure

The authors declare that they have no competing interests in this work.

References

1. Woo D, Comeau ME, Venema SU, et al. Risk Factors Associated With Mortality and Neurologic Disability After Intracerebral Hemorrhage in a Racially and Ethnically Diverse Cohort. *JAMA Network Open*. 2022;5(3):e221103.
2. Wang X, Arima H, Al-Shahi Salman R, et al. Clinical prediction algorithm (BRAIN) to determine risk of hematoma growth in acute intracerebral hemorrhage. *Stroke*. 2015;46(2):376–381. doi:10.1161/STROKEAHA.114.006910
3. Cao L, Liu M, Wang M, et al. 3D slicer-based calculation of hematoma irregularity index for predicting hematoma expansion in intracerebral hemorrhage. *BMC Neurol*. 2022;22(1):452. doi:10.1186/s12883-022-02983-w
4. Yan Y, Ren H, Luo B, et al. Clinical characteristics of spontaneous intracranial basal ganglia hemorrhage and risk factors for hematoma expansion in the plateaus of China. *Front Neurol*. 2023;14:1183125. doi:10.3389/fneur.2023.1183125
5. Lim JK, Hwang HS, Cho BM, et al. Multivariate analysis of risk factors of hematoma expansion in spontaneous intracerebral hemorrhage. *Surg Neurol*. 2008;69(1):40–45. doi:10.1016/j.surneu.2007.07.025
6. Ji N, Lu JJ, Zhao YL, et al. Imaging and clinical prognostic indicators for early hematoma enlargement after spontaneous intracerebral hemorrhage. *Neurol Res*. 2009;31(4):362–366. doi:10.1179/174313209X444035

7. Kayahara T, Kikkawa Y, Komine H, et al. Predictors of subacute hematoma expansion requiring surgical evacuation after initial conservative treatment in patients with acute subdural hematoma. *Acta Neurochir*. 2020;162(2):357–363. doi:10.1007/s00701-019-04187-7
8. Yabuuchi H, Kamitani T, Sagiyama K, et al. Clinical application of radiation dose reduction for head and neck CT. *Eur J Radiol*. 2018;107:209–215. doi:10.1016/j.ejrad.2018.08.021
9. Brott T, Thalinger K, Hertzberg V. Hypertension as a risk factor for spontaneous intracerebral hemorrhage. *Stroke*. 1986;17(6):1078–1083. doi:10.1161/01.STR.17.6.1078
10. Barras CD, Tress BM, Christensen S, et al. Density and shape as CT predictors of intracerebral hemorrhage growth. *Stroke*. 2009;40(4):1325–1331. doi:10.1161/STROKEAHA.108.536888
11. Zhang X, Li T, Liu F, et al. Comparative Analysis of Droplet-Based Ultra-High-Throughput Single-Cell RNA-Seq Systems. *Mol Cell*. 2019;73 (1):130–42e5. doi:10.1016/j.molcel.2018.10.020
12. Xiong X, Li Q, Yang WS, et al. Comparison of Swirl Sign and Black Hole Sign in Predicting Early Hematoma Growth in Patients with Spontaneous Intracerebral Hemorrhage. *Med Sci Monit*. 2018;24:567–573. doi:10.12659/MSM.906708
13. B SP, Kemmling A, Schwake M, et al. Triage of 5 Noncontrast Computed Tomography Markers and Spot Sign for Outcome Prediction After Intracerebral Hemorrhage. *Stroke*. 2018;49(10):2317–2322. doi:10.1161/STROKEAHA.118.021625
14. Sporns PB, Schwake M, Kemmling A, et al. Comparison of Spot Sign, Blend Sign and Black Hole Sign for Outcome Prediction in Patients with Intracerebral Hemorrhage. *J Stroke*. 2017;19(3):333–339. doi:10.5853/jos.2016.02061
15. Hu X, Fang Y, Li H, et al. Protocol for seizure prophylaxis following intracerebral hemorrhage study (SPICH): a randomized, double-blind, placebo-controlled trial of short-term sodium valproate prophylaxis in patients with acute spontaneous supratentorial intracerebral hemorrhage. *Int J Stroke*. 2014;9(6):814–817. doi:10.1111/ijs.12187
16. Lu JJ, Ji N, Zhao YL, et al. Neuroimaging and clinical predictors of hematoma enlargement in spontaneous intracerebral hemorrhage. *Zhonghua Yi Xue Za Zhi*. 2007;87(7):438–441.
17. Cai B, Peng L, Wang ZB, et al. Association between Serum Lipid and Hematoma Expansion after Spontaneous Intracerebral Hemorrhage in Chinese Patients. *J Stroke Cerebrovasc Dis*. 2020;29(6):104793. doi:10.1016/j.jstrokecerebrovasdis.2020.104793
18. Tsai HH, Lee BC, Chen YF, et al. Cerebral Venous Reflux and Dilated Basal Ganglia Perivascular Space in Hypertensive Intracerebral Hemorrhage. *J Stroke*. 2022;24(3):363–371. doi:10.5853/jos.2022.01004
19. Rzeplinski R, Slugocki M, Tarka S, et al. Mechanism of Spontaneous Intracerebral Hemorrhage Formation: an Anatomical Specimens-Based Study. *Stroke*. 2022;53(11):3474–3480. doi:10.1161/STROKEAHA.122.040143
20. Eilertsen H, Menon CS, Law ZK, et al. Haemostatic therapies for stroke due to acute, spontaneous intracerebral haemorrhage. *Cochrane Database Syst Rev*. 2023;10(10):CD005951. doi:10.1002/14651858.CD005951.pub5
21. Santalucia P. Intracerebral hemorrhage: medical treatment. *Neurol Sci*. 2008;29(2):S271–3. doi:10.1007/s10072-008-0961-y
22. Cao D, Li Q, Fu P, et al. Early Hematoma Enlargement in Primary Intracerebral Hemorrhage. *Curr Drug Targets*. 2017;18(12):1345–1348. doi:10.2174/1389450118666170427151011
23. Pinto A, Tuttolomondo A, Di Raimondo D, et al. Cerebrovascular risk factors and clinical classification of strokes. *Semin Vasc Med*. 2004;4 (3):287–303. doi:10.1055/s-2004-861497
24. Qureshi AI, Huang W, Lobanova I, et al. Effect of Moderate and Severe Persistent Hyperglycemia on Outcomes in Patients With Intracerebral Hemorrhage. *Stroke*. 2022;53(4):1226–1234. doi:10.1161/STROKEAHA.121.034928
25. Dobrynina LA, Gnedovskaya EV, Shabalina AA, et al. Biomarkers and mechanisms of early vascular damage. *Zh Nevrol Psikhiatr Im S S Korsakova*. 2018;118(122):23–32. doi:10.17116/jnevro201811812223
26. Keep RF, Andjelkovic V, Xiang J, et al. Brain endothelial cell junctions after cerebral hemorrhage: changes, mechanisms and therapeutic targets. *J Cereb Blood Flow Metab*. 2018;38(8):1255–1275. doi:10.1177/0271678X18774666
27. Liu M, Wang Z, Meng X, et al. Predictive Nomogram for Unfavorable Outcome of Spontaneous Intracerebral Hemorrhage. *World Neurosurg*. 2022;164:e1111–e22. doi:10.1016/j.wneu.2022.05.111
28. Li F, Chen A, Li Z, et al. Machine learning-based prediction of cerebral hemorrhage in patients with hemodialysis: a multicenter, retrospective study. *Front Neurol*. 2023;14:1139096. doi:10.3389/fneur.2023.1139096
29. Sang Y, Roest M, De Laat B, et al. Interplay between platelets and coagulation. *Blood Rev*. 2021;46:100733. doi:10.1016/j.blre.2020.100733
30. Sprugel MI, Kuramatsu JB, Gerner ST, et al. Age-dependent clinical outcomes in primary versus oral anticoagulation-related intracerebral hemorrhage. *Int J Stroke*. 2021;16(1):83–92. doi:10.1177/1747493019895662
31. Li R, Yang M. A comparative study of the blend sign and the black hole sign on CT as a predictor of hematoma expansion in spontaneous intracerebral hemorrhage. *Biosci Trends*. 2017;11(6):682–687. doi:10.5582/bst.2017.01283
32. Sato S, Delcourt C, Heeley E, et al. Significance of Cerebral Small-Vessel Disease in Acute Intracerebral Hemorrhage. *Stroke*. 2016;47(3):701–707. doi:10.1161/STROKEAHA.115.012147