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ORIGINAL RESEARCH

Influencing Factors (History of Alcohol Consumption) and Construction of a Nomogram Prediction Model for In-Hospital Gastrointestinal Bleeding Secondary to Acute Cerebral Hemorrhage in a Certain Hospital

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Objective: To investigate the factors influencing acute cerebral hemorrhage (ACH) secondary to nosocomial gastrointestinal hemorrhage (GIH) and construct a nomogram prediction model.

Methods: A total of 500 ACH patients admitted to our hospital from August 2022 to August 2024 were retrospectively analyzed and divided into a modeling group (350 cases) and a validation group (150 cases) in a 7:3 ratio. Patients in the modeling group were further divided into the GIH and non-GIH groups. Clinical data were collected, and multivariate logistic regression was used to analyze risk factors. A nomogram model was constructed using R software. The predictive performance was evaluated using the ROC curve, calibration curve, and decision curve analysis (DCA).

Results: Among 500 patients, 78 (15.6%) developed GIH. In the modeling group (350 cases), 56 (16.0%) had GIH. There were significant differences in age, history of coronary heart disease, history of alcohol consumption, NIHSS score, systolic blood pressure, and hemorrhage volume between groups ($P<0.05$). Logistic regression analysis identified these factors as independent risk factors for secondary GIH ($P<0.05$). The Area Under Curve(AUC) was 0.798 in the modeling group and 0.978 in the validation group, with calibration curves showing good agreement between predicted and observed values (Hosmer-Lemeshow(H-L) test: modeling group, $\chi^2=7.156$, $P=0.732$; validation group, $\chi^2=7.015$, $P=0.703$). DCA indicated a high clinical application value when the probability ranged from 0.06 to 0.95.

Conclusion: Age, history of coronary heart disease, history of alcohol consumption, NIHSS score, systolic blood pressure, and hemorrhage volume are key risk factors for secondary GIH in ACH patients. The nomogram model constructed based on these factors demonstrates good predictive performance and clinical application value. It can help clinicians prevent early onset and reduce the risk of bleeding in patients.

Keywords: acute cerebral haemorrhage, gastrointestinal haemorrhage, influencing factors, nomogram

Introduction

Acute intracerebral hemorrhage, a severe type of stroke, is primarily caused by the rupture of cerebral arteries and predominantly affects elderly individuals, with high incidence and mortality rates, seriously impacting quality of life.[1,2] Acute intracerebral hemorrhage is often accompanied by hematoma formation, triggering inflammatory responses and other secondary brain injuries, ultimately resulting in neurological deficits.[3] The disease progresses rapidly, with sudden vascular ruptures potentially induced by intense emotional fluctuations. Clinical treatment mainly involves surgical intervention to alleviate clinical symptoms. However, the extent of bodily damage caused by the disease is significant, making postoperative complications such as gastrointestinal bleeding common.[4,5] Studies have found that approximately 30% of patients with cerebral hemorrhage develop gastrointestinal bleeding, which prolongs recovery time, affects quality

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of life, and contributes to postoperative mortality.[6,7] Therefore, identifying the factors influencing in-hospital secondary gastrointestinal bleeding in patients with acute intracerebral hemorrhage is crucial for improving patient prognosis. Nomograms can integrate and analyze the selected risk factors to individually predict the risk value of a specific event, quantify the risk of the event, and enable clinicians to formulate targeted interventions based on these risk factors, thereby effectively reducing the risk of gastrointestinal bleeding.[8,9] There are currently few studies reporting on the use of nomograms to investigate in-hospital secondary gastrointestinal bleeding following acute intracerebral hemorrhage. Therefore, this study aims to identify the influencing factors of in-hospital gastrointestinal bleeding secondary to acute cerebral hemorrhage and to construct a nomogram prediction model to assist clinicians in early intervention.

Materials and Methods

General Data

A retrospective study was conducted on 500 patients with acute intracerebral hemorrhage admitted to Ganzhou People's Hospital from August 2022 to August 2024. Using PASS 15 software ($\alpha = 0.05$, power = 70%, d = 0.60), the required total sample size was calculated to be 445 cases. Considering a 15% dropout rate, a total of 500 patients were included in this study. According to the simple random sampling method, patients were randomly divided into a modeling group (350 cases) and a validation group (150 cases) in a 7:3 ratio. The modeling group was further divided into a gastrointestinal bleeding group and a non-gastrointestinal bleeding group based on whether in-hospital secondary gastrointestinal bleeding occurred. The case collection flowchart is shown in Figure 1. **Inclusion criteria** (1) Meeting the criteria for acute intracerebral hemorrhage;[10] (2) Diagnosed by CT or MRI; (3) Onset time < 7 days; (4) Complete clinical data available; (5) Patients signed informed consent. **Exclusion criteria** (1) Patients with major organ failure; (2) Patients with malignant tumors; (3) Patients with cerebral infarction or brain tumors; (4) Patients with hematological disorders; (5) Patients with severe infections; (6) Patients with psychiatric disorders or those unable to cooperate with treatment. This study was approved by the hospital ethics committee.See follow Figure 1.

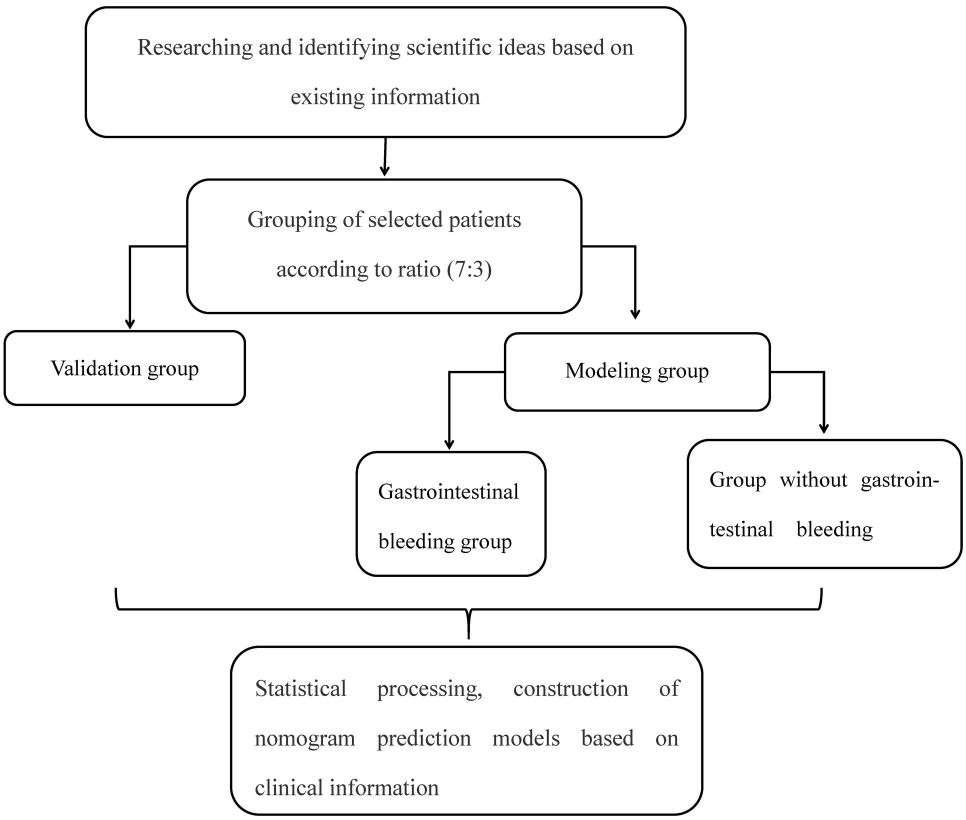

Figure 1 Case flow collection diagram.

Criteria for Secondary In-Hospital Gastrointestinal Bleeding[11]

During hospitalization, patients with acute intracerebral hemorrhage presenting with coffee-like gastric contents extracted via gastric tube or vomited, visible hematemesis or melena, and positive occult blood tests were diagnosed with secondary in-hospital gastrointestinal bleeding. Diagnoses excluded bleeding caused by primary diseases or trauma of the esophagus, intestine, or anus, or false positives due to factors such as iron supplement ingestion. Patients were grouped based on the presence of gastrointestinal bleeding during hospitalization.

Clinical Data

Clinical data (All personnel involved in data collection received professional training, and quality control measures were implemented to minimize bias) were collected from patient examinations and electronic medical records, including age, gender, body mass index (BMI), hypertension, diabetes, hyperlipidemia, atrial fibrillation, coronary artery disease history, stroke history, smoking history (continuous or cumulative smoking for over six months), alcohol consumption history (average 50g per drinking session, duration ≥1 year), antiplatelet medication history, bleeding site, NIHSS score upon admission, systolic and diastolic blood pressure upon admission, and hemorrhage volume. Laboratory data included total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), hemoglobin, platelet count, blood glucose, creatinine, time from onset to admission, and length of hospital stay.

Observation Indicators

(1) Comparison of clinical data between the modeling and validation groups; (2) Comparison of clinical data between the gastrointestinal bleeding and non-gastrointestinal bleeding groups; (3) Analysis of factors influencing secondary in-hospital gastrointestinal bleeding in acute intracerebral hemorrhage; (4) Development of a nomogram model for secondary in-hospital gastrointestinal bleeding in acute intracerebral hemorrhage; (5) Nomogram model for the modeling group; (6) Nomogram model for the validation group; (7) DCA curve of the nomogram model.

Statistical Analysis

SPSS 25.0 was used for data analysis. Count data were analyzed using the χ^2-test and expressed as cases (%). Normally distributed measurement data were analyzed using the *t*-test and expressed as mean (Eqn).The cutoff values for the parameters were determined based on the mean and were classified into binary variables accordingly.Multivariate logistic regression analysis was employed to identify risk factors for secondary in-hospital gastrointestinal bleeding in acute intracerebral hemorrhage. The R software was used to construct the nomogram model. The ROC curve was used to assess the discriminative ability of the nomogram model, while the decision curve analysis (DCA) evaluated the clinical utility of the model. A P-value < 0.05 was considered statistically significant.

Results

Comparison of Clinical Data Between the Modeling Group and the Validation Group

There were no significant differences in clinical data between the modeling group and the validation group (P > 0.05). See Table 1.

Comparison of Clinical Data Between the Gastrointestinal Bleeding Group and the Non-Gastrointestinal Bleeding Group

Out of the 500 patients, 78 experienced gastrointestinal bleeding, with an incidence rate of 15.60%. In the modeling group (350 patients), 56 experienced gastrointestinal bleeding, with an incidence rate of 16.00%. Significant differences were observed between the two groups in age, history of coronary artery disease, history of alcohol consumption, NIHSS score upon admission, systolic blood pressure upon admission, and hemorrhage volume (P < 0.05). No significant differences were found in other clinical data (P > 0.05). See Table 2.

Table 1 Comparison of Clinical Data Between the Modelling Group and the Validation Group

Factor	Modelling Group (n=350)	Validation Group (n=150)	t/χ^2	*P*
Age (years)			0.535	0.465
<60	206 (58.86)	83 (55.33)		
≥60	144 (41.14)	67 (44.67)		
Genders			0.193	0.660
Man	215 (61.43)	89 (59.33)		
Woman	135 (38.57)	61 (40.67)		
BMI (kg/m^2)			0.019	0.891
<24	196 (56.00)	83 (55.33)		
≥24	154 (44.00)	67 (44.67)		
Hypertension			0.408	0.523
Yes	248 (70.86)	102 (68.00)		
No	102 (29.14)	48 (32.00)		
Diabetes			0.548	0.459
Yes	45 (12.86)	23 (15.33)		
No	305 (87.14)	127 (84.67)		
Hypertriglyceridemia			0.076	0.782
Yes	39 (11.14)	18 (12.00)		
No	311 (88.86)	132 (88.00)		
Atrial fibrillation			0.264	0.607
Yes	7 (2.00)	2 (1.33)		
No	343 (98.00)	148 (98.67)		
History of coronary heart disease			0.091	0.763
Yes	26 (7.43)	10 (6.67)		
No	324 (92.57)	140 (93.33)		
History of stroke			0.734	0.392
Yes	48 (13.71)	25 (16.67)		
No	302 (86.29)	125 (83.33)		
Smoking history			0.300	0.584
Yes	168 (48.00)	68 (45.33)		
No	182 (52.00)	82 (54.67)		
Drinking history			0.006	0.937
Yes	202 (57.71)	86 (57.33)		
No	148 (42.29)	64 (42.67)		
History of antiplatelet drug use			0.416	0.519
Yes	42 (12.00)	15 (10.00)		
No	308 (88.00)	135 (90.00)		
Haemorrhage site			0.579	0.447
Behind the scenes	56 (16.00)	20 (13.33)		
Above the curtain	294 (84.00)	130 (86.67)		
NIHSS score on admission (points)			0.321	0.571
<14	187 (53.43)	76 (50.67)		
≥14	163 (46.57)	74 (49.33)		
Systolic blood pressure on admission (mmHg)			0.262	0.609
<155	200 (57.14)	82 (54.67)		
≥155	150 (42.86)	68 (45.33)		
Diastolic blood pressure on admission (mmHg)			0.202	0.653
<94	165 (47.14)	74 (49.33)		
≥94	185 (52.86)	76 (50.67)		

(*Continued*)

Table 1 (Continued).

Factor	Modelling Group (n=350)	Validation Group (n=150)	t/χ^2	*P*
Haemorrhage volume (mL)			0.185	0.667
<30	166 (47.43)	68 (45.33)		
≥30	184 (52.57)	82 (54.67)		
TC (mmol/L)	4.80±1.32	4.78±1.26	0.104	0.917
TG (mmol/L)	1.38±0.32	1.37±0.29	0.649	0.517
LDL-C (mmol/L)	3.10±0.30	3.10±0.28	0.452	0.651
HDL-C (mmol/L)	1.22±0.27	1.20±0.23	1.010	0.313
Haemoglobin (g/L)	149.78±14.79	149.18±14.25	0.311	0.756
Blood platelet count ($\times10^9$/L)	226.39±38.19	226.10±38.08	0.181	0.856
Blood glucose (mmol/L)	5.46±0.91	5.43±0.87	1.664	0.097
Creatinine (mmol/L)	62.95±6.44	62.91±6.37	0.373	0.709
Time from onset to admission (d)	2.04±0.39	2.05±0.40	1.774	0.077
Length of hospitalisation (d)	15.95±2.47	15.91±2.43	0.970	0.333

Table 2 Comparison of Clinical Data Between the Gastrointestinal Haemorrhage Group and Non Gastrointestinal Haemorrhage Group

Factor	Gastrointestinal Haemorrhage Group (n=56)	Non Gastrointestinal Haemorrhage Group (n=294)	t/χ^2	*P*
Age (years)			14.745	<0.001
<60	20 (35.71)	186 (63.27)		
≥60	36 (64.29)	108 (36.73)		
Genders			0.032	0.857
Man	35 (62.50)	180 (61.22)		
Woman	21 (37.50)	114 (38.78)		
BMI (kg/m^2)			0.481	0.488
<24	29 (51.79)	167 (56.80)		
≥24	27 (48.21)	127 (43.20)		
Hypertension			2.914	0.088
Yes	45 (80.36)	203 (69.05)		
No	11 (19.64)	91 (30.95)		
Diabetes			0.616	0.433
Yes	9 (16.07)	36 (12.24)		
No	47 (83.93)	258 (87.76)		
Hypertriglyceridemia			0.124	0.725
Yes	7 (12.50)	32 (10.88)		
No	49 (87.50)	262 (89.12)		
Atrial fibrillation			0.016	0.901
Yes	1 (1.79)	6 (2.04)		
No	55 (98.21)	288 (97.96)		
History of coronary heart disease			14.463	<0.001
Yes	11 (19.64)	15 (5.10)		
No	45 (80.36)	279 (94.90)		
History of stroke			0.967	0.325
Yes	10 (17.86)	38 (12.93)		
No	46 (82.14)	256 (87.07)		

(*Continued*)

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Table 2 (Continued).

Factor	Gastrointestinal Haemorrhage Group (n=56)	Non Gastrointestinal Haemorrhage Group (n=294)	t/χ^2	*P*
Smoking history			0.829	0.363
Yes	30 (53.57)	138 (46.94)		
no	26 (46.43)	156 (53.06)		
Drinking history			16.302	<0.001
Yes	46 (82.14)	156 (53.06)		
No	10 (17.86)	138 (46.94)		
History of antiplatelet drug use			1.046	0.306
Yes	9 (16.07)	33 (11.22)		
No	47 (83.93)	261 (88.78)		
Haemorrhage site			0.171	0.679
Behind the scenes	10 (17.86)	46 (15.65)		
Above the curtain	46 (82.14)	248 (84.35)		
NIHSS score on admission (points)			19.019	<0.001
<14	15 (26.79)	172 (58.50)		
≥14	41 (73.21)	122 (41.50)		
Systolic blood pressure on admission (mmHg)			17.014	<0.001
<155	18 (32.14)	182 (61.90)		
≥155	38 (67.86)	112 (38.10)		
Diastolic blood pressure on admission (mmHg)			0.986	0.321
<94	23 (41.07)	142 (48.30)		
≥94	33 (58.93)	152 (51.70)		
Haemorrhage volume (mL)			15.677	<0.001
<30	13 (23.21)	153 (52.04)		
≥30	43 (76.79)	141 (47.96)		
TC (mmol/L)	4.82±1.31	4.80±1.32	0.104	0.917
TG (mmol/L)	1.41±0.30	1.38±0.32	0.649	0.517
LDL-C (mmol/L)	3.12±0.32	3.10±0.30	0.452	0.651
HDL-C (mmol/L)	1.24±0.28	1.22±0.27	1.010	0.313
Haemoglobin (g/L)	150.34±15.34	149.67±14.68	0.311	0.756
Blood platelet count ($\times 10^9$/L)	227.24±36.25	226.23±38.56	0.181	0.856
Blood glucose (mmol/L)	5.64±0.89	5.42±0.91	1.664	0.097
Creatinine (mmol/L)	63.24±6.52	62.89±6.42	0.373	0.709
Time from onset to admission (d)	2.12±0.42	2.02±0.38	1.774	0.077
Length of hospitalisation (d)	16.24±2.34	15.89±2.50	0.970	0.333

Analysis of Factors Influencing Secondary In-Hospital Gastrointestinal Bleeding in Acute Intracerebral Hemorrhage

Whether secondary gastrointestinal bleeding occurred during hospitalization was used as the dependent variable (yes = 1, no = 0). Age, history of coronary artery disease, history of alcohol consumption, NIHSS score upon admission, systolic blood pressure upon admission, and hemorrhage volume were used as independent variables. Variable assignments are shown in Table 3. Multivariate logistic regression analysis revealed that age, history of coronary artery disease, history of alcohol consumption, NIHSS score upon admission, systolic blood pressure upon admission, and hemorrhage volume were risk factors for secondary in-hospital gastrointestinal bleeding in acute intracerebral hemorrhage ($P < 0.05$). See Table 4.

Table 3 Assignment Methods of Argument Variables

variable	Assignment Method
Age	<60 years old=0, ≥60 years old=1
History of coronary heart disease	no=0, yes=1
Drinking history	no=0, yes=1
NIHSS score on admission	≥14 potins=1, <14 potins=0
Systolic blood pressure on admission	≥155 mmHg=1, <155 mmHg=0
Haemorrhage	≥30 mL=1, <30 mL=0

Table 4 Analysis of Factors Influencing Acute Cerebral Haemorrhage Secondary to Nosocomial Gastrointestinal Haemorrhage

variable	βvalue	SEvariable	Waldχ^2variable	*P*variable	ORvariable	95% CI
Age	1.287	0.321	16.069	<0.001	3.621	1.930~6.791
History of coronary heart disease	0.927	0.372	6.221	0.013	2.527	1.220~5.236
Drinking history	1.528	0.350	19.088	<0.001	4.607	2.322~9.142
NIHSS score on admission	1.392	0.345	16.268	<0.001	4.021	2.045~7.907
Systolic blood pressure on admission	0.106	0.039	7.330	0.007	1.111	1030~1.200
Haemorrhage	0.700	0.312	5.028	0.025	2.013	1.092~3.710
Constant	−16.387	3.283	24.911	<0.001	<0.001	–

Development of a Nomogram Model for Secondary In-Hospital Gastrointestinal Bleeding in Acute Intracerebral Hemorrhage

In this model, the most influential factor was history of alcohol consumption, followed by systolic blood pressure at admission, history of coronary heart disease, age, NIHSS score at admission, and hemorrhage volume. For example, a patient aged ≥60 years (40.0 points) with a history of alcohol consumption (100.0 points), an NIHSS score upon admission ≥14 (393.5 points), systolic blood pressure upon admission ≥155 mmHg (78.5 points), and hemorrhage volume ≥30 mL (31.5 points) would have a total score of 289.5 points.A vertical line was drawn at the total score position, indicating an approximate 78% probability of developing in-hospital gastrointestinal bleeding secondary to acute cerebral hemorrhage.See Figure 2.

Nomogram Model for the Modeling Group

The AUC of the modeling group was 0.798 (95% CI: 0.732–0.865), the calibration curve slope was close to 1, and the H-L test result was $\chi^2 = 7.156$, P = 0.732. See Figures 3A and B.

Nomogram Model for the Validation Group

The AUC of the validation group was 0.978 (95% CI: 0.955–0.999), the calibration curve slope was close to 1, and the H-L test result was $\chi^2 = 7.015$, P = 0.703. See Figures 4A and B.

DCA Curve of the Nomogram Model

The DCA curve showed that when the probability was between 0.06 and 0.95, the clinical utility of the model in assessing secondary in-hospital gastrointestinal bleeding in acute intracerebral hemorrhage was high. See Figure 5.

Discussion

Acute intracerebral hemorrhage (ICH) is a non-traumatic hemorrhage in the brain parenchyma primarily caused by vascular rupture. The pathogenesis of this condition is complex and is often associated with factors such as hypertension.

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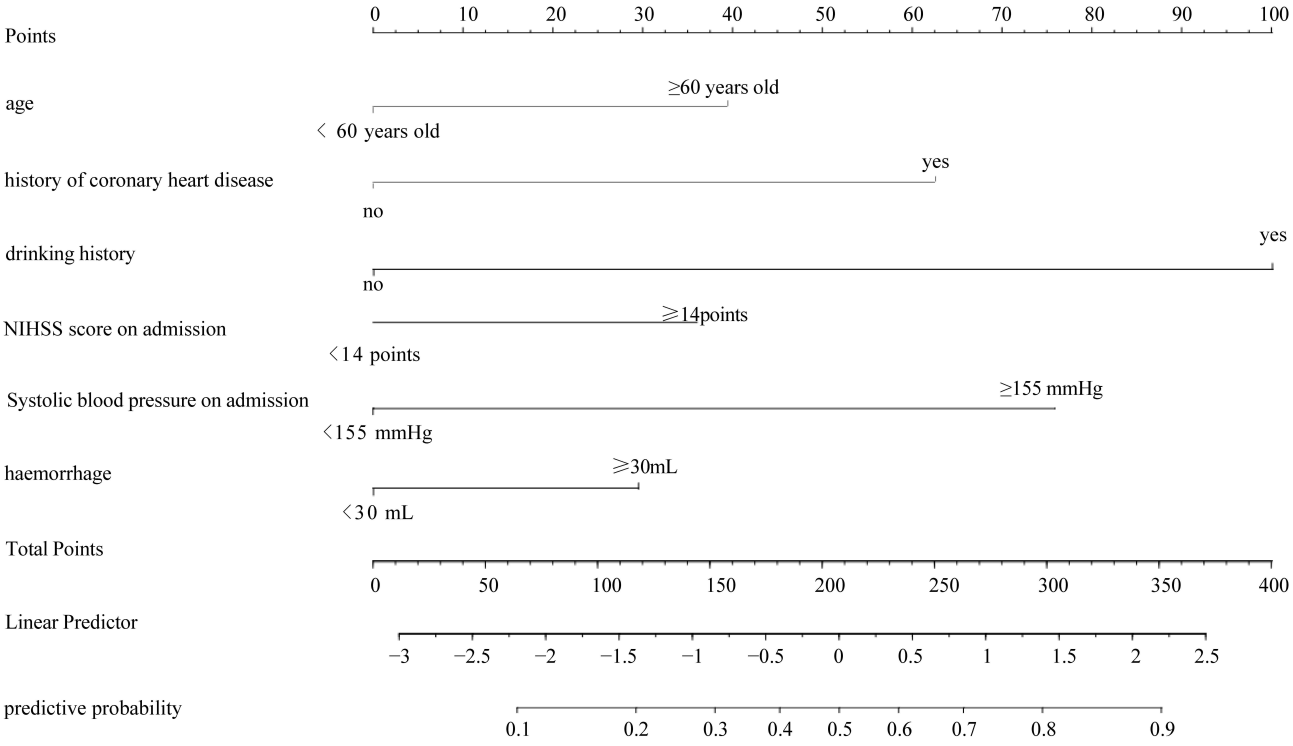

Figure 2 Development of a nomogram model of acute cerebral haemorrhage secondary to nosocomial gastrointestinal haemorrhage.

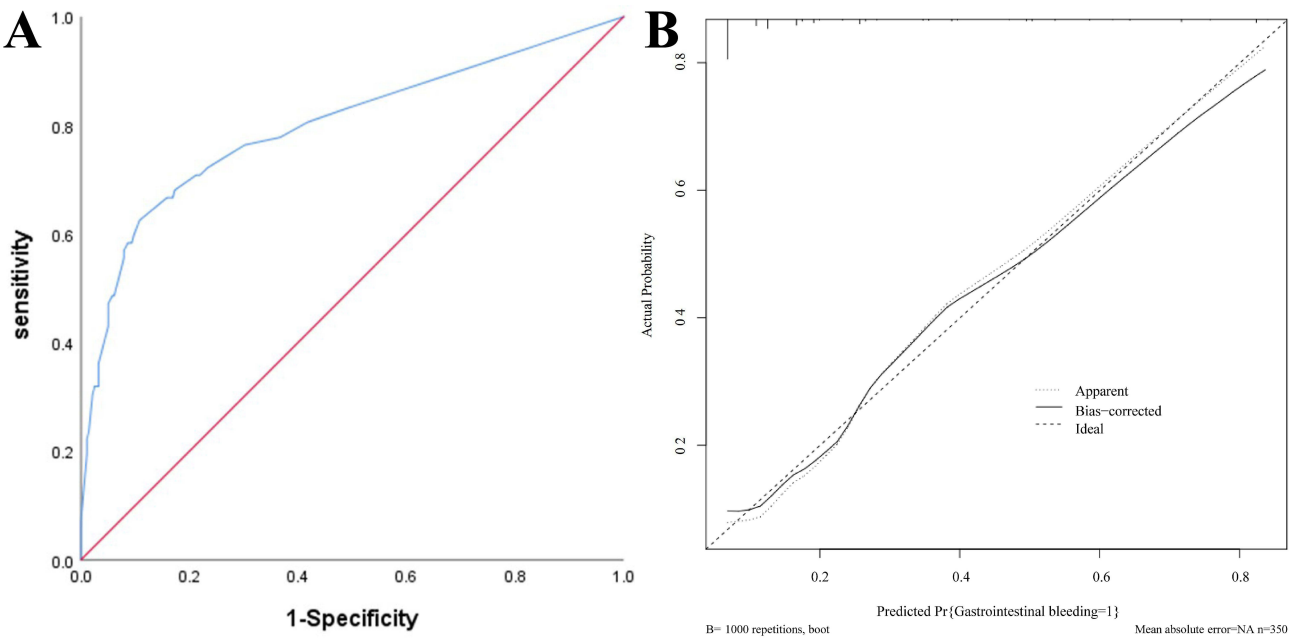

Figure 3 Model a line chart model for the modeling group. (**A**) ROC curve for modelling group (**B**) Calibration curve for Modelling group.

With the increasing prevalence of hypertension, the incidence of acute ICH is also rising.[12] Hematomas caused by cerebral hemorrhage can damage brain tissue, and inflammatory factors produced during the process can further impair neurological function. The bleeding area can also lead to ischemic injury to brain tissue, causing neurological deficits and even organ damage, which severely affects the patient's quality of life.[13,14] Gastrointestinal (GI) bleeding caused by

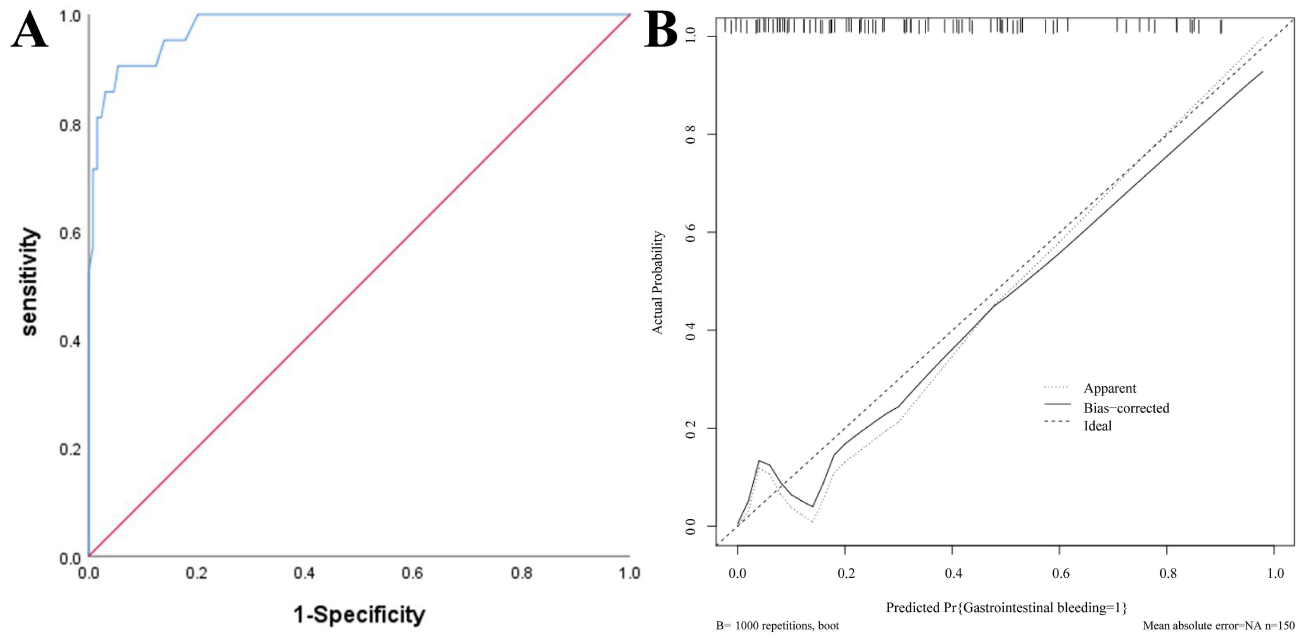

Figure 4 Model a line chart model for the validation group. (**A**) ROC curve for the validation group; (**B**) Calibration curve for the validation group.

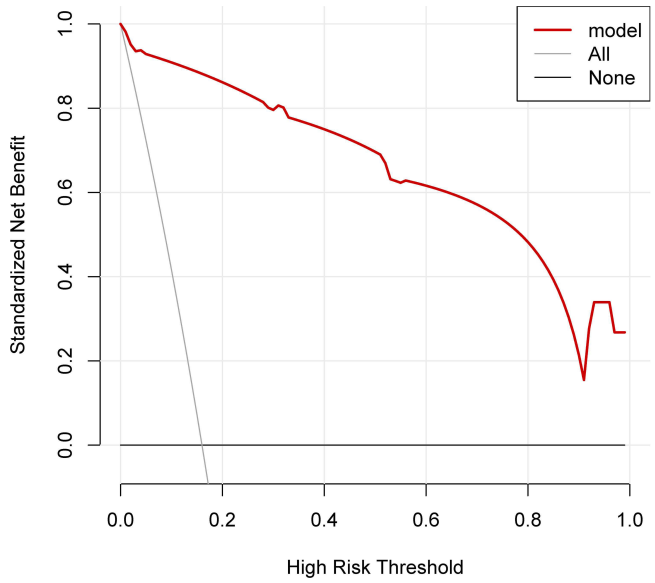

Figure 5 DCA curve for the nomogram.

gastric mucosal damage is a common complication of acute ICH. It impacts patients' digestive function and overall condition. Mild cases may only show positive occult blood tests, while severe cases can involve stress responses or even shock.[15] In this study, 78 out of 500 patients experienced GI bleeding, with an incidence rate of 15.60%. In the modeling group of 350 patients, 56 experienced GI bleeding, accounting for an incidence rate of 16.00%, indicating a relatively high occurrence.Therefore, identifying the influencing factors for gastrointestinal bleeding in these patients can facilitate early intervention, ultimately reducing the risk of bleeding.

This study identified six factors related to GI bleeding in patients: age, history of coronary artery disease, history of alcohol consumption, NIHSS score upon admission, systolic blood pressure upon admission, and hemorrhage volume. The reasons for these findings are analyzed as follows: (1)Age: Older patients are at a higher risk of GI bleeding due to

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the decline in physical function with age. Fluctuations in their condition can lead to complications in other systems. Additionally, elderly patients often have multiple comorbidities, which increase the risk of GI bleeding. Older individuals also tend to have higher blood viscosity and stiffer blood vessels compared to younger people, further elevating their risk of GI bleeding[16,17]. Therefore, in clinical practice, special attention should be given to older patients, with timely monitoring of their physical condition to reduce the risk of gastrointestinal bleeding.(2)History of Coronary Artery Disease: Consistent with previous studies[18,19], coronary artery disease was identified as a risk factor for GI bleeding. This is likely due to the long-term use of antiplatelet or anticoagulant medications in these patients, which increases the risk of GI bleeding. Therefore, in clinical practice, it is important to use medications appropriately in such patients to reduce the risk of bleeding. (3) History of Alcohol Consumption: Long-term alcohol consumption damages the gastric mucosa and stimulates histamine release from the mucosa. Histamine induces smooth muscle spasms and increases capillary permeability, leading to edema and erosion of the gastric mucosa, and potentially causing GI bleeding. Chronic alcohol consumption can result in pathological changes in the gastric mucosa, increasing the risk of GI bleeding.[20] Therefore, in clinical practice, patients should be advised to abstain from alcohol and be informed of its harmful effects to reduce the risk of bleeding.(4) NIHSS Score Upon Admission: Secondary GI bleeding is associated with the severity of the patient's condition. The NIHSS score is used to assess the degree of neurological deficits in acute stroke patients. A higher NIHSS score indicates a greater risk of GI bleeding.[21] (5) Systolic Blood Pressure Upon Admission: Elevated blood pressure at the time of onset also increases the risk of GI bleeding. Blood pressure levels are positively correlated with the severity of the patient's condition. Severe cerebral hemorrhage exacerbates GI bleeding risk, as high blood pressure is a marker of systemic stress and increases the likelihood of stress-induced GI bleeding.[22] Therefore, it is necessary in clinical practice to closely monitor patients' blood pressure. For those with sustained hypertension, appropriate antihypertensive medication can be administered to lower blood pressure and reduce the risk of bleeding. (6) Hemorrhage Volume: Larger hemorrhage volumes are a risk factor for GI bleeding. Increased brain hemorrhage volume elevates intracranial pressure, leading to a reactive rise in systemic blood pressure, which further increases the risk of GI bleeding.[23] Therefore, for these high-risk patients, optimizing antihypertensive regimens, standardizing the use of anticoagulant and antiplatelet medications, strengthening nutritional support, reducing alcohol consumption, and adopting gastric mucosal protective strategies can enhance the clinical guidance value of this study.

A nomogram is a predictive model presented in a graphical format. It assigns a score to each predictive variable based on its data, and then calculates the probability of a specific event occurring according to the total score.The nomogram model developed in this study yielded AUC values of 0.798 for the modeling group and 0.978 for the validation group, indicating high discrimination. The calibration curve slopes were close to 1, suggesting good predictive consistency. The DCA curve showed that when probabilities ranged between 0.06 and 0.95, the model had high clinical utility for assessing secondary in-hospital GI bleeding in acute ICH. This model can assist clinicians in implementing targeted interventions based on identified risk factors, thereby effectively improving patient outcomes. However, this study has several limitations. The sample size is relatively small, and as a retrospective study, there may be selection bias in the sample selection process. Moreover, the study did not explore the impact of genetic factors—especially adverse reactions and hypersensitivity—across different populations and subgroups. In future research, the sample size will be expanded, and prospective multicenter studies will be conducted to further optimize and validate the findings.

In summary, age, history of coronary artery disease, history of alcohol consumption, NIHSS score upon admission, systolic blood pressure upon admission, and hemorrhage volume are influencing factors for secondary in-hospital GI bleeding in acute ICH. The nomogram model based on these factors demonstrates good predictive performance for secondary GI bleeding. It can assist clinicians in early prevention and reduce the risk of bleeding in patients.

Data Sharing Statement

The original contributions presented in the study are included in the article.

Ethics and Consent Statement

The study was in accordance with GanZhou People`s Hospital ethics review board (No.GZSRMYY2024070012) and with the 1964 Helsinki Declaration. Written informed consent to participate in this study was provided by the participants.

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Disclosure

Authors declared no conflict of interest.

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