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Constructing a nomogram for short-term prognosis in postoperative patients with aneurysmal subarachnoid hemorrhage: a two-center retrospective study

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Aneurysmal subarachnoid hemorrhage (aSAH) is a life-threatening condition with high morbidity and mortality. Early prediction of prognosis remains challenging. This study aimed to develop a nomogram incorporating clinical and inflammatory biomarkers to predict short-term outcomes in postoperative aSAH patients. Thus optimizing the intervention strategy and improve patient quality of life. Logistic regression analysis was used to determine the single predictor of aneurysmal subarachnoid hemorrhage. Based on these independent predictors, a nomogram was created in R studio. The results showed that the aneurysmal site (3.35[95% CI, 1.05–10.66], *P* = 0.041), affected side (3.77[95% CI, 1.17–12.11], *P* = 0.026), hydrocephalus (0.03[95% CI, 0.01–0.12], *P* < 0.001), Hunt-Hess grade (4.13[95% CI, 1.17–14.49], *P* = 0.027), GCS score (4.08[95% CI, 1.02–16.25], *P* = 0.046), hypertension history (0.18[95% CI, 0.06–0.55], *P* = 0.003), WBC (3.49[95% CI, 1.06–11.56], *P* = 0.04), MLR (0.33[95% CI, 0.12–0.92], *P* = 0.035) were independent predictors. The nomogram demonstrated superior predictive accuracy compared to existing models, with lower calibration errors (training group: 0.018; validation group: 0.052) and high AUC values (0.95 and 0.901, respectively). Given the class imbalance (84.3% of patients had favorable outcomes), sensitivity analyses were performed to verify the consistency and reliability of the findings. The nomogram constructed based on aneurysm location, affected side, presence of hydrocephalus, Hunt-Hess grade, GCS score, hypertension, WBC and MLR can enables clinicians to identify high-risk aSAH patients early, facilitating targeted interventions such as anti-inflammatory therapy or hydrocephalus management. This tool may improve resource allocation and reduce disability rates in critical care settings. Thus optimizing the intervention strategy and improve patient quality of life.

Keywords Aneurysmal subarachnoid hemorrhage, Short-term prognosis, Inflammatory factor, Nomogram

Abbreviations

SAH	Subarachnoid hemorrhage
aSAH	Aneurysmal subarachnoid hemorrhage
GCS	Glasgow Coma Score
WBC	White blood cell count
LY	Lymphocyte count
MO	Monocyte count
PLT	Blood platelet count
NB	Neutrophil count

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TBil	Total bilirubin
DBil	Direct bilirubin
ALT	Alanine transaminase
AST	Aspartate aminotransferase
Cre	Creatinine
Urea	Urea
HDL-C	High density lipoprotein
LDL-C	Low density lipoprotein
TC	Total cholesterol
TP	Total protein
Alb	Albumin
PCT	Procalcitonin
TG	Triglyceride
FBG	Fasting blood glucose
HCT	Hematocrit
K	Potassium
Na	Sodium
Cl	Chlorine
Ca	Total calcium
PT	Prothrombin time
APTT	Activated partial thrombin time
D-dimer	D-dimer
FIB	Fibrinogen
NLR	Neutrophil count/lymphocyte count
dNLR	Neutral Granulocyte count/(White blood cell count - Lymphocyte count)
MLR	Monocyte count/lymphocyte count
NMLR	(Monocyte count + neutrophil count)/lymphocyte count
SIRI	Neutrophil count × monocyte count/lymphocyte count
SII	Platelet count × neutrophil count/lymphocyte count
PLR	Blood plate count/lymphocyte count
AISI	Neutrophil count × platelet count × monocyte count/lymphocyte count
NHR	Neutrophil count ratio/high-density fat egg White
MHR	Monocyte count/high density lipoprotein
LHR	Lymphocyte count/high density lipoprotein
PHR	Platelet count/high density lipoprotein
TyG	Triglyceride-glucose index
TyG-BMI	Glucose triglyceride /BMI index

Subarachnoid hemorrhage (SAH) is an acute cerebrovascular disease that seriously damages the central nervous system. It has pathophysiological effects on multiple organs of the body, resulting in high mortality and morbidity[1]–[2]. Hypertensive arteriosclerosis is the most common cause, followed by ruptured brain aneurysms. Current surgical methods include aneurysmal clipping and intravascular therapy. However, there are many complications during treatment, such as increased intracranial pressure, seizure, hyponatremia, delayed cerebral ischemia, etc., which can be life-threatening in severe cases[3–6]. Although the diagnosis and treatment of aSAH patients and the careful management of the intensive care unit (ICU) have improved significantly in recent years, the prognosis of ASAH patients is still the most concerned issue[7]–[8]. According to existing studies, patients with aSAH after surgery have a poor prognosis. Therefore, predicting prognostic outcomes is of great value in the treatment selection and evaluation of aSAH patients.

Current prognostic tools for aSAH primarily rely on clinical scales (e.g., Hunt-Hess, WFNS), yet emerging evidence highlights systemic inflammation as a key driver of secondary brain injury. Such as leukocytes, neutrophils, hypersensitive C-reactive protein, fibrinogen, etc., can be used as strong predictors for predicting the prognosis of patients with aSAH. These factors increase significantly after the occurrence of aSAH and are closely related to the severity of the disease and poor prognosis[9–13]. However, current changes in a single indicator may not provide clinicians with strong and sufficient clinical evidence to diagnose the disease. Predictive models are derivative statistical tools that help predict prognostic outcomes based on aggregated assessments of physical, laboratory, and radiological tests. High-quality predictive models can guide bedside decisions and patient consultations, ensure that resources are appropriately allocated to reduce healthcare costs, and improve the design and analysis of clinical trials. The establishment of a production model may be beneficial for the management of aSAH patients. Nomograms, as visual predictive tools, integrate multiple variables to estimate individualized risks and have been increasingly adopted in neurocritical care[14]. For example, Dongzhou Zhuang et al. have developed a dynamic nomogram for predicting poor prognosis after aSAH[15]. Xiao Jin et al. used serological indicators to construct a nomogram model of postoperative pneumonia after[16]. Xin Feng et al. developed and validated a novel nomogram for predicting aneurysm rupture in patients with multiple intracranial aneurysms[17].

Therefore, this study aimed to collect clinical data, including baseline data, blood and inflammatory factor levels, of postoperative patients with aSAH through a two-center retrospective analysis and construct a nomogram model to predict the short-term prognosis of postoperative patients. Through this model, doctors can more accurately evaluate the recovery of patients and provide a scientific basis for clinical decision-making.

Materials and methods

Patient section

This is a two-center retrospective cohort study from China. The study included patients diagnosed with aSAH at two large medical centers in China between January 2020 and January 2024. Inclusion criteria are as follows: (1) Patients diagnosed aSAH by CTA, MRA or DSA; (2)Age > 18 years old; (3)After surgical treatment; The exclusion criteria are as follows: (1)Age below 18 years; (2)The admission blood drawing index is not perfect; (3)Recent use of hormones and other drugs; (4)Combined immune system and blood system diseases; (5)Excluding non-aneurysmal diseases, such as cerebral arteriovenous malformation, moyamoya disease, craniocerebral trauma, etc.; (6)No surgical treatment was performed. In this study, patients meeting the criteria in Huizhou Central People's Hospital and Gaozhou People's Hospital were summarized, and the patients were randomly divided into training groups and verification groups at a ratio of 7:3. Data access was restricted to authorized researchers, and the study complied with China's Personal Information Protection Law (PIPL). The study has been approved by the Committee of Huizhou Central People's Hospital, and due to the retrospective nature of the study, the Huizhou Central People's Hospital Ethics Review Committee waived the need of obtaining informed consent. It complies with the Declaration of Helsinki and the Chinese Measures for Ethical Review of Biomedical Research Involving People.

Data collection and definition

This study collected baseline clinical data, blood routine, biochemistry, coagulation function, liver function, procalcitonin, and other test results. Baseline clinical data included gender, age, site, affected side, history of hypertension, diabetes, alcohol abuse, smoking, hydrocephalus, cerebral infarction, surgical method, Hunt-Hess grade, Fisher grade, Glasgow Coma Score(GCS) score, WFNS grade, etc. Laboratory parameters included hematological indices (e.g., white blood cell count, platelet count), biochemical markers (e.g., creatinine, albumin), and inflammatory ratios (NLR, MLR), etc. We compared the baseline data of the training and validation groups to verify the comparability of the data between the two groups.

Then, We calculated the inflammatory biomarker ratio using the following equation: NLR = neutrophil count/lymphocyte count, dNLR = neutrophil count/(white blood cell count - lymphocyte count), MLR = monocyte count/lymphocyte count, NMLR= (monocyte count + neutrophil count)/lymphocyte count, PLR = Platelet count/lymphocyte count, NHR = neutrophil count ratio/high-density lipoprotein, MHR = monocyte count/high-density lipoprotein, LHR = lymphocyte count/high-density lipoprotein, PHR = Platelet count/high-density lipoprotein, Systemic Inflammatory Response Index (SIRI) = neutrophil count × monocyte Count/lymphocyte count, systemic immunoinflammatory index (SII) = platelet count × neutrophil count/lymphocyte count, Systemic inflammatory index (AISI) = neutrophil count × platelet count × monocyte count/lymphocyte count, triglyceride-glucose index (TyG) = In{(fasting triglyceride × fasting blood glucose) /2}, glucose triglyceride /BMI index (TYG-BMI) = TyG/BMI.

Outcome

In this study, the Glasgow Outcome Scale (GOS) score was assessed 6 months post-discharge to evaluate short-term prognosis. Follow-up evaluations can through outpatient visits or telephone interviews. A GOS score > 3 indicates a good prognosis, while a GOS score ≤ 3 indicates a poor prognosis.

Statistical analysis

In this study, we first generate a table of random numbers using a simple random method with the PROC PLAN procedure statement of SAS 9.4 statistical software: 01,02,03,… According to this allocation method, 600 was allocated to the two groups according to a random number table, in which the training set was 420, and the verification set was 180. Patients were numbered from 01 to 600 successively according to the medical record number and finally assigned to the corresponding group according to the group planned by the number. Then, SPSS21.0 (SPSS Inc., Chicago, IL) was used to compare the baseline data between the training group and the validation group. Secondly, the data of the training group was divided into good and bad prognoses, and the clinical data and test results were compared between the groups. The Shapiro normality test was used to determine the normality of continuous data. If the data fit the normal distribution, the data were represented by mean ± standard deviation. Independent univariate ANOVA was used to compare the groups. If the normal distribution did not conform, the data were expressed by the median (25% quantile, 75% quantile), and the Kruskal-Wallis test was used to compare groups. Frequency (percentage) was used to describe categorical data, and the Chi-square or Fisher's exact test was used to compare groups. The difference was considered statistically significant when the bilateral p-value was less than 0.05.

Logistic regression analysis was performed on all clinical data in the training group, and the mean was used as the dividing point for binary classification processing. More significant than the mean was defined as 1, and less than the mean was defined as 2. Then, univariate logistic regression analysis was performed. Factors with $p < 0.1$ in univariate analysis were included in multivariate logistic regression analysis to evaluate independent predictors of prognosis. For variable selection criteria, variables with p-value < 0.1 in univariate analysis were included in the multivariate logistic regression analysis, screened by stepwise regression (backward method), and finally retained independent predictors with $p < 0.05$ to construct the nomogram. For example, although age was significant in the univariate analysis ($p = 0.001$), it was excluded in the multivariate analysis due of collinearity with other variables.

Using R Studio (v4.2.2), a nomogram was constructed based on independent predictors. Model performance was evaluated via ROC curves (AUC), calibration plots (mean error), and decision curve analysis (DCA). Decision Curve Analysis (DCA) analyzed the model's clinical benefit. All tests are bidirectional, and a P-value of less than 0.05 is considered significant.

To evaluate the robustness of the model, we conducted sensitivity analyses using two approaches: (1) applying random undersampling to balance the outcome classes, followed by re-running the entire logistic regression modeling process; and (2) evaluating alternative machine learning algorithms (Random Forest and Gradient Boosting) for comparative analysis. The consistency of predictors and model performance metrics (ROC, PR, calibration) was assessed.

Result

Prognostic relationship between clinical factors and aSAH

In this study, a total of 692 patients diagnosed with aSAH were collected from two large medical centers. A total of 92 patients were excluded due to the above factors, and 600 patients were finally included in a retrospective study, as shown in Fig. 1. There were 420 patients in the training set and 180 patients in the validation set. Patients with incomplete blood index at admission (92 cases) were excluded, and the proportion of missing data was 13.3% (92/692). All the analyses were based on the complete data, and no imputation method was used. Due to the low proportion of deletions and the random deletions, the impact on the results was limited.

The baseline data of the two groups were compared between the two groups. In the training set, there were 174 males (41.43%), median age 56 years [IQR:49,64], 145 patients (34.52%) with responsible aneurysms located in the internal carotid artery segment, and 202 patients (48.10%) with left-side aneurysms. There were 339 cases (80.71%) of aneurysm embolism, 305 cases (72.62%) of Hunt-Hess grade 1–2 and 261 cases (62.14%) of Fisher grade 1–2. There were 366 cases (87.14%) with a GCS score of 9–15, 319 cases (75.95%) with WFNS grade 1–3, 123 cases (29.29%) with hydrocephalus. Among them, the patients with hypertension, diabetes, smoking, alcoholism and cerebral infarction were 210 cases (50.00%), 27 cases (6.43%), 81 cases (19.29%), 19 cases (4.52%) and 27 cases (6.43%), respectively. There was no statistical difference in baseline data between the two groups ($p > 0.05$), indicating that the data were comparable (Table 1).

We then compared the data from the training group. The results showed that hydrocephalus, surgical method, Hunt-Hess scale, Fisher scale, GCS score, WFNS scale, history of hypertension, history of cerebral infarction, age, BMI, D-dimer, FBG, TP, Alb, The differences among PCT, ALT, AST, Urea, Cl, Ca, MLR, SIRI and AISI were statistically significant. (Table 2).

We included all clinical factors in the training group into univariate logistic regression analysis, and the results showed that hydrocephalus, surgical method, Hunt-Hess rating, Fisher rating, GCS score, WFNS classification, history of hypertension, history of cerebral infarction, age, BMI, D-dimer, FBG, TP, Alb, PCT, ALT, AST, Urea, Cl, Ca, MLR, SIRI, AISI were all predictors of short-term prognosis. We then included the factors with $p < 0.1$ in the single factor into the multivariate binary logistic regression analysis. The results showed that the aneurysmal site (3.35[95% CI, 1.05–10.66], $P = 0.041$), affected side (3.77[95% CI, 1.17–12.11], $P = 0.026$), hydrocephalus (0.03[95% CI, 0.01–0.12], $P < 0.001$), Hunt-Hess grade (4.13[95% CI, 1.17–14.49], $P = 0.027$), GCS score (4.08[95% CI, 1.02–16.25], $P = 0.046$), hypertension history (0.18[95% CI, 0.06–0.55], $P = 0.003$), WBC

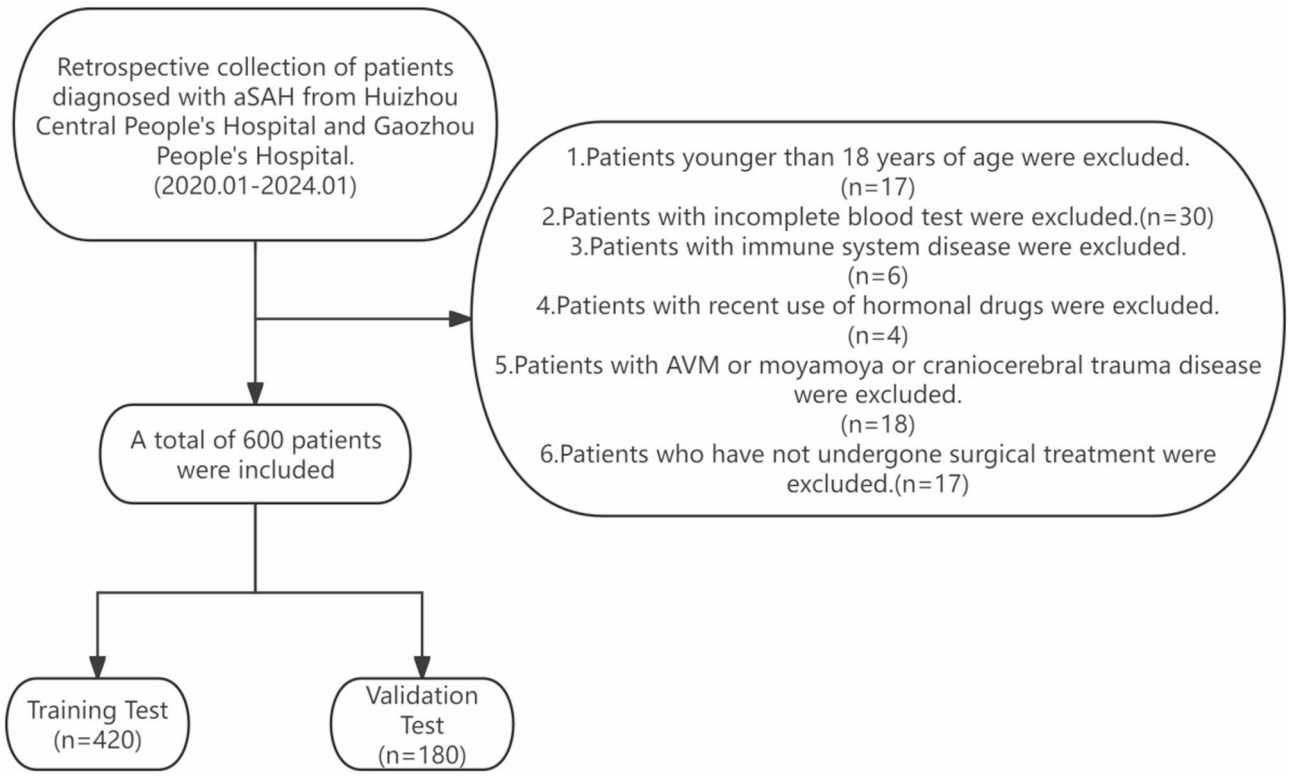

Fig. 1. Flow chart of this study.

Variable	Training test ($n=420$)	Validation test ($n=180$)	statistic	p
Sex			1.411	0.235
Male	174 (41.43%)	84 (46.67%)		
Female	246 (58.57%)	94 (53.33%)		
Age	56 (49,64)	55 (47,66)	0.126	0.900
Arterial responsibility			0.059	0.808
ICA	145 (34.52%)	64 (35.56%)		
Other	275 (65.48%)	116 (64.44%)		
Side			0.183	0.669
Left	202 (48.1%)	90 (50.0%)		
Right	218 (51.9%)	90 (50.0%)		
Hydrocephalus			0.031	0.860
Yes	123 (29.29%)	54 (30.0%)		
No	297 (70.71%)	126 (70.0%)		
Method			0.074	0.785
Aneurysm embolism	339 (80.71%)	147 (81.67%)		
Aneurysm clipping	81 (19.29%)	33 (18.33%)		
Hunt-Hess			0.860	0.354
1–2 Grade	305 (72.62%)	124 (68.89%)		
3–5 Grade	115 (27.38%)	56 (31.11%)		
Fisher			1.581	0.209
1–2 Grade	261 (62.14%)	102 (56.67%)		
3–4 Grade	159 (37.86%)	78 (43.33%)		
GCS			1.119	0.290
9–15 Points	366 (87.14%)	151 (83.89%)		
3–8 Points	54 (12.86%)	29 (16.11%)		
WFNS			0.062	0.803
1–3 Grade	319 (75.95%)	135 (75%)		
4–5 Grade	101 (24.05%)	45 (25%)		
Hypertension			0.389	0.533
Yes	210 (50%)	95 (52.78%)		
No	210 (50%)	85 (47.22%)		
Diabetes			0.021	0.884
Yes	27 (6.43%)	11 (6.11%)		
No	393 (93.57%)	169 (93.89%)		
Smoke			0.949	0.330
Yes	81 (19.29%)	41 (22.78%)		
No	339 (80.71%)	139 (77.22%)		
Alcohol addiction			2.567	0.109
Yes	19 (4.52%)	14 (7.78%)		

Continued

Variable	Training test ($n=420$)	Validation test ($n=180$)	statistic	p
No	401 (95.48%)	166 (92.22%)		
Cerebral Infarction			0.903	0.342
Yes	27 (6.43%)	8 (4.44%)		
No	393 (93.57%)	172 (95.56%)		

Table 1. Baseline data table for comparison of training test and validation test.

(3.49[95% CI, 1.06–11.56], $P=0.04$), MLR (0.33[95% CI, 0.12–0.92], $P=0.035$) were independent predictors (Table 3).

Nomogram construction and evaluation

Based on multivariate analysis, a nomogram incorporating aneurysm location, affected side, hydrocephalus, Hunt-Hess grade, GCS score, hypertension, WBC, and MLR was developed using R Studio (Fig. 2).

We used R studio software to verify the predictive efficacy of patient nomograms. The ROC curve of the training group (FIG. 3A) suggested that the C index was 0.95 and the confidence interval was (0.929–0.972), while the ROC curve of the verification group (FIG. 3B) suggested that the C index was 0.901 and the confidence interval was (0.849–0.953), showing good prediction efficiency. The calibration curve of the training group (FIG. 4A) showed that the predicted value of the model was consistent with the observed value, with an average error of 0.018; the verification group (FIG. 4B) showed that the expected value of the model was consistent with the observed value, with an average error of 0.052. The DCA curve of the training group (FIG. 5A) suggested that the threshold value was between 0.01 and 0.98, and the DCA curve of the verification group (FIG. 5B) indicated that the threshold value was between 0.04 and 0.91, and the clinical benefit was good.

It is not difficult to see that the prognostic nomogram we constructed showed good predictive efficacy in both the training and validation groups, with minor errors, and could benefit most clinical patients.

Sensitivity analysis

The favorable prognosis group ($n=354$) and the poor prognosis group ($n=66$) showed a significant imbalance (5.4:1). To rigorously evaluate the potential impact of this imbalance on our findings, we implemented a comprehensive sensitivity analysis approach:

First, we performed random undersampling of the majority class to create a balanced dataset (1:1 ratio). When we reapplied logistic regression to this balanced cohort, the model consistently identified the same key predictors: MLR, WBC, hydrocephalus, Hunt-Hess grade, and GCS score. Importantly, both discrimination metrics (AUC-ROC:=0.967; AUC-PR:=0.968) and calibration performance remained strong (Table 4; Fig. 7), demonstrating the robustness of our original findings.

Furthermore, we conducted internal validation using random forest and gradient boosting machine models, both of which are more robust to class imbalance. Variable importance rankings derived from these models were highly consistent with the logistic regression results. Notably, GCS score and hydrocephalus consistently ranked as the top two predictors across all models (Fig. 6; Data 6 A; Data 6B).

These findings support the stability of the identified predictors and reinforce the clinical relevance of our nomogram under varied modeling strategies (Fig. 7).

Discussion

In an in-depth study and review of the published literature on the prognosis of patients with aSAH, we can find some standard features and factors. Specifically, men of older age, combined hypertension, a history of smoking, alcohol abuse, and poor scores on admission were more likely to have a poor prognosis. In addition, those patients with underlying diseases such as coronary heart disease, liver and kidney disease, and cerebral infarction are also more likely to face the risk of adverse prognosis. Therefore, we have considered including these factors in the analysis before conducting a multi-factor regression analysis, intending to minimize the impact of these potential confounders on the results.

After careful analysis and research, we came up with some significant findings. The results showed that aneurysmal location, affected side, presence or absence of hydrocephalus, Hunt-Hess grade, GCS score, hypertension, WBC, and MLR were all independent predictors of short-term poor prognosis in patients after aSAH. Based on these findings, we built a nomogram prediction model that can help doctors and researchers predict patient outcomes more accurately, providing strong support for clinical decision-making.

Previous studies have shown that patients with high Hunt-Hess grades and low GCS scores at the onset of disease tend to have poor predictive outcomes and high disability and mortality rates. The Hunt-Hess scale is a crucial indicator for assessing the degree of neurological damage and level of consciousness in patients with aSAH. It is divided into I-V scales. To a large extent, this rating directly indicates the severity of the disease. For patients with Hunt-Hess grades I to III, early surgical intervention is usually recommended because these patients have less blood loss and are relatively mild. Most patients can obtain a good prognosis with the active intervention of emergency surgery. However, for patients with Hunt-Hess grade IV-V intracranial aneurysms, the clinical prognosis is generally poor, whether surgical or conservative treatment is selected. Most of these

Variable	Good prognosis group($n=354$)	Poor prognosis group($n=66$)	p
Sex			0.47
Male	144 (40.68%)	30 (45.45%)	
Female	210 (59.32%)	36 (54.55%)	
Arterial responsibility			0.056
ICA	129 (36.44%)	16 (24.24%)	
Other	225 (63.56%)	50 (75.76%)	
Side			0.07
Left	177 (50.00%)	25 (37.88%)	
Right	177 (50.00%)	41 (62.12%)	
Hydrocephalus			< 0.001
Yes	63 (17.80%)	60 (90.91%)	
No	291 (82.20%)	6 (9.09%)	
Method			< 0.001
Aneurysm embolism	300 (84.75%)	39 (59.09%)	
Aneurysm clipping	54 (15.25%)	27 (40.91%)	
Hunt-Hess			< 0.001
1–2 Grade	289 (81.64%)	16 (24.24%)	
3–5 Grade	65 (18.36%)	50 (75.76%)	
Fisher			< 0.001
1–2 Grade	245 (69.21%)	16 (24.24%)	
3–4 Grade	109 (30.79%)	50 (75.76%)	
GCS			< 0.001
9–15 Points	332 (93.79%)	34 (51.52%)	
3–8 Points	22 (6.21%)	32 (48.48%)	
WFNS			< 0.001
1–3 Grade	297 (83.90%)	22 (33.33%)	
4–5 Grade	57 (16.10%)	44 (66.67%)	
Hypertension			0.007
Yes	167 (47.18%)	43 (65.15%)	
No	187 (52.82%)	23 (34.85%)	
Diabetes			0.492
Yes	21 (5.93%)	6 (9.09%)	
No	333 (94.07%)	60 (90.91%)	
Smoke			0.205
Yes	72 (20.34%)	9 (13.64%)	
No	282 (79.66%)	57 (86.36%)	
Alcohol addiction			1
Yes	16 (4.52%)	3 (4.55%)	
No	338 (95.48%)	63 (95.45%)	
Cerebral Infarction			0.004
Yes	17 (4.80%)	10 (15.15%)	
No	337 (95.20%)	56 (84.85%)	
Age			0.001
>55	165 (46.61%)	46 (69.70%)	
≤55	189 (53.39%)	20 (30.30%)	
BMI			0.011
>23	178 (50.28%)	22 (33.33%)	
≤23	176 (49.72%)	44 (66.67%)	
HCT			0.179
>0.38	198 (55.93%)	31 (46.97%)	
≤0.38	156 (44.07%)	35 (53.03%)	
WBC			0.093
>13.4	143 (40.4%)	34 (51.52%)	
≤13.4	211 (59.6%)	32 (48.48%)	
NB			0.183
>11.35	151 (42.66%)	34 (51.52%)	
Continued			

Variable	Good prognosis group($n=354$)	Poor prognosis group($n=66$)	p
≤11.35	203 (57.34%)	32 (48.48%)	
LY			0.092
>1.33	135 (38.14%)	18 (27.27%)	
≤1.33	219 (61.86%)	48 (72.73%)	
MO			0.195
>0.69	131 (37.01%)	30 (45.45%)	
≤0.69	223 (62.99%)	36 (54.55%)	
PLT			0.288
>240	170 (48.02%)	27 (40.91%)	
≤240	184 (51.98%)	39 (59.09%)	
PT			0.787
>13.1	164 (47.26%)	30 (45.45%)	
≤13.1	183 (52.74%)	36 (54.55%)	
APTT			0.452
>33.5	170 (48.99%)	29 (43.94%)	
≤33.5	177 (51.01%)	37 (56.06%)	
D-dimer			< 0.001
>3290	71 (21.01%)	31 (50.00%)	
≤3290	267 (78.99%)	31 (50.00%)	
FIB			0.435
>3.4	145 (41.79%)	31 (46.97%)	
≤3.4	202 (58.21%)	35 (53.03%)	
FBG			< 0.001
>146.6	123 (34.75%)	42 (63.64%)	
≤146.6	231 (65.25%)	24 (36.36%)	
TP			0.02
>71	200 (56.50%)	27 (40.91%)	
≤71	154 (43.50%)	39 (59.09%)	
Alb			0.011
>41.5	206 (58.52%)	27 (41.54%)	
≤41.5	146 (41.48%)	38 (58.46%)	
PCT			< 0.001
>0.57	16 (6.06%)	19 (33.33%)	
≤0.57	248 (93.94%)	38 (66.67%)	
TBil			0.743
>12.4	114 (37.75%)	24 (40.00%)	
≤12.4	188 (62.25%)	36 (60.00%)	
DBil			0.136
>4.1	110 (36.42%)	28 (46.67%)	
≤4.1	192 (63.58%)	32 (53.33%)	
ALT			0.019
>21	100 (28.99%)	28 (43.75%)	
≤21	245 (71.01%)	36 (56.25%)	
AST			< 0.001
>26	86 (24.93%)	34 (53.12%)	
≤26	259 (75.07%)	30 (46.88%)	
TC			0.056
>4.94	133 (42.63%)	18 (29.51%)	
≤4.94	179 (57.37%)	43 (70.49%)	
TG			0.91
>120.68	100 (32.05%)	20 (32.79%)	
≤120.68	212 (67.95%)	41 (67.21%)	
HDL-c			0.157
>51.6	138 (44.23%)	21 (34.43%)	
≤51.6	174 (55.77%)	40 (65.57%)	
LDL-c			0.139
Continued			

Variable	Good prognosis group($n=354$)	Poor prognosis group($n=66$)	p
>112.4	155 (49.68%)	24 (39.34%)	
≤112.4	157 (50.32%)	37 (60.66%)	
Urea			0.005
>4.7	142 (40.34%)	39 (59.09%)	
≤4.7	210 (59.66%)	27 (40.91%)	
Cre			0.11
>71.2	129 (36.54%)	31 (46.97%)	
≤71.2	224 (63.46%)	35 (53.03%)	
Na			0.577
>139.5	179 (50.71%)	31 (46.97%)	
≤139.5	174 (49.29%)	35 (53.03%)	
K			0.805
>3.6	176 (49.86%)	34 (51.52%)	
≤3.6	177 (50.14%)	32 (48.48%)	
Cl			0.026
>103	172 (48.73%)	42 (63.64%)	
≤103	181 (51.27%)	24 (36.36%)	
Ca			<0.001
>2.21	185 (52.56%)	18 (27.27%)	
≤2.21	167 (47.44%)	48 (72.73%)	
NLR			0.238
>11.5	144 (40.68%)	32 (48.48%)	
≤11.5	210 (59.32%)	34 (51.52%)	
dNLR			0.791
>0.945	160 (45.20%)	31 (46.97%)	
≤0.945	194 (54.80%)	35 (53.03%)	
MLR			<0.001
>0.61	125 (35.31%)	40 (60.61%)	
≤0.61	229 (64.69%)	26 (39.39%)	
NMLR			0.234
>12.14	149 (42.09%)	33 (50.00%)	
≤12.14	205 (57.91%)	33 (50.00%)	
SIRI			<0.001
>7.65	104 (29.38%)	34 (51.52%)	
≤7.65	250 (70.62%)	32 (48.48%)	
SII			0.935
>2655.7	139 (39.60%)	25 (39.06%)	
≤2655.7	212 (60.40%)	39 (60.94%)	
PLR			0.78
>231.5	149 (42.09%)	29 (43.94%)	
≤231.5	205 (57.91%)	37 (56.06%)	
AISI			0.006
>1733.2	105 (30.17%)	31 (47.69%)	
≤1733.2	243 (69.83%)	34 (52.31%)	
NHR			0.203
>0.243	126 (40.38%)	30 (49.18%)	
≤0.243	186 (59.62%)	31 (50.82%)	
MHR			0.554
>0.0154	110 (35.37%)	24 (39.34%)	
≤0.0154	201 (64.63%)	37 (60.66%)	
LHR			0.601
>0.0298	108 (34.62%)	19 (31.15%)	
≤0.0298	204 (65.38%)	42 (68.85%)	
PHR			0.962
>5.147	134 (42.95%)	26 (42.62%)	
≤5.147	178 (57.05%)	35 (57.38%)	
Continued			

Variable	Good prognosis group($n=354$)	Poor prognosis group($n=66$)	p
TyG			0.299
>8.832	136 (43.59%)	31 (50.82%)	
≤8.832	176 (56.41%)	30 (49.18%)	
TyG-BMI			0.589
>203.47	155 (49.68%)	28 (45.90%)	
≤203.47	157 (50.32%)	33 (54.10%)	

Table 2. Comparison of clinical factors between the good prognosis group and the poor prognosis group.

patients will receive conservative treatment first and then consider further surgical intervention when the condition is stabilized. Numerous studies have shown that the higher the Hunt-Hess rating in aSAH patients, the higher the risk of poor prognosis[18–20]. Similarly, the GCS has been recognized as a clinical assessment tool worldwide and is of great significance for predicting patient outcome[21–23]. Although age was associated with prognosis in univariate analyses, its effect was overwritten in multivariate models by measures such as Hunt-Hess grading, GCS score, and MLR, suggesting that age may indirectly influence prognosis by influencing neurofunctional status or inflammatory responses.In addition, high blood pressure is also an independent risk factor. Long-term hypertension will lead to arterial wall damage and increase the risk of aneurysm rupture. Secondly, the vascular regulation function of hypertensive patients is impaired, which affects the recovery after bleeding[24]. Hypertensive patients also may co-exist with other cardiovascular and cerebrovascular diseases, such as coronary heart disease and heart failure, which further increases the risk of poor prognosis.

Furthermore, it is not difficult to find that the specific location and affected side of the aneurysm also have an important impact on the prognosis of patients[25]. For example, the rupture risk and treatment difficulty of internal carotid aneurysms, middle cerebral aneurysms, anterior communicating aneurysms, posterior communicating aneurysms and basal aneurysms are different, and the symptoms produced by different parts of the damage are different, and these differences are directly related to the recovery degree and survival rate of patients. Compared with most people, the left side of the brain is the dominant hemisphere, with extremely important functions, and has a huge regulatory role in the language, action, understanding and cognitive functions of patients, while bleeding on the right side may lead to emotional indifference, memory disorders and other manifestations. However, the specific prognosis still needs to be combined with factors such as blood loss, aneurysm size, shape, patient age and basic health status.

Hydrocephalus is a common and severe complication of aSAH, and about 20–30% of people with aSAH will develop acute hydrocephalus due to impaired circulation or abnormal absorption of cerebrospinal fluid. Its mechanism may be because the blood clot prevents the average circulation of cerebrospinal fluid, or the blood affects the arachnoid particles, leading to the decrease of cerebrospinal fluid reabsorption or the excessive secretion of cerebrospinal fluid and other reasons, which can cause brain tissue displacement and injury by increasing intracranial pressure. Acute hydrocephalus will not only aggravate the early neurological damage of aSAH patients, further aggravate the clinical condition, but also damage the neurological function of patients in the recovery period after aSAH surgery[26]. Han-Yu Huang et al. established a column graph model for clinical outcomes of patients with severe subarachnoid hemorrhage and proposed that systemic inflammatory response and complications unrelated to surgery after SAH, especially hydrocephalus, delayed cerebral ischemia and pneumonia, may be important risk factors leading to poor prognosis in patients with severe SAH[27]. At the same time, Nicolai Maldaner et al. established hemorrhage, age, treatment, clinical status, and hydrocephalus (HATCH) scores and verified that HATCH scores were strong independent predictors of functional prognosis. Incorporating it into daily practice may benefit patient care in aSAH[28].

In addition, a large number of studies have verified that hydrocephalus is a predictor of poor prognosis and is closely related to inflammation[6,29,30]. In patients with aSAH complicated with hydrocephalus, due to the damaged cerebrospinal fluid circulation pathway, the secretion or absorption of cerebrospinal fluid is unbalanced, which leads to the accumulation of inflammatory cells in the subarachnoid space and further exacerbates the inflammatory response. Various studies have proved that early systemic inflammatory changes are the prognosis factors affecting aSAH[31]. Inflammation runs through the whole process of aSAH injury mechanism[32]. When the aneurysm ruptures, the blood deposited in the subarachnoid space stimulates brain tissue and activates immune regulatory cells in the central nervous system, and a large number of inflammatory cells enter the subarachnoid space, rapidly causing inflammation[33]- [34]. The inflammatory response can activate the immune system to release inflammatory cells and inflammatory mediators, such as cytokines, adhesion molecules and chemokines, leading to the occurrence and development of cerebrovascular spasm, exacerbating the occurrence of brain injury and affecting the prognosis of patients[35–38]. For example, WBC studies have shown that aSAH can trigger brain injury and delayed cerebral ischemia by stimulating systemic cellular response, which will lead to an increase in the number of white blood cells[39]- [40], while the growth and rupture of brain aneurysms are promoted by leukocyte infiltration and inflammatory response in the aneurysm wall[41]. Moreover, the thinning of the aneurysm wall is related to leukocytosis[42]. Similarly, higher white blood cell counts are associated with hematoma growth and early neurological deterioration[43]. Therefore, there is a significant relationship between high and low white blood cell count and the prognosis of patients.

Combined with previous studies, we found that MLR can predict the course of many diseases, such as diabetes, depression, heart disease, etc[44–51]. In our study, we found that MLR is closely related to the short-term prognosis of patients after aSAH, which has not been found in the past. We found that high levels of

Variable	OR[95%CI]	*p*	OR[95%CI]	*p*
Sex		0.47		
Male	1			
Female	0.82[95% CI, 0.48,1.40]			
Arterial responsibility		0.058		0.041
ICA	1		1	
Other	1.79[95% CI, 0.98,3.27]		3.35[95% CI, 1.05,10.66]	
Side		0.072		0.026
Left	1		1	
Right	1.64[95% CI, 0.96,2.81]		3.77[95% CI, 1.17,12.11]	
Hydrocephalus		< 0.001		< 0.001
Yes	1		1	
No	0.02[95% CI, 0.01,0.05]		0.03[95% CI, 0.01,0.12]	
Method		< 0.001		
Aneurysm embolism	1			
Aneurysm clipping	3.85[95% CI, 2.18,6.80]			
Hunt-Hess		< 0.001		0.027
1–2 Grade	1		1	
3–5 Grade	13.89[95% CI, 7.44,25.93]		4.13[95% CI, 1.17,14.49]	
Fisher		< 0.001		
1–2 Grad	1			
3–4 Grade	7.02[95% CI, 3.83,12.88]			
GCS		< 0.001		0.046
9–15 Points	1		1	
3–8 Points	14.2[95% CI, 7.43,27.14]		4.08[95% CI, 1.02,16.25]	
WFNS		< 0.001		
1–3 Grade	1			
4–5 Grade	10.42[95% CI, 5.81,18.71]			
Hypertension		0.008		0.003
Yes	1		1	
No	0.48[95% CI, 0.28,0.83]		0.18[95% CI, 0.06,0.55]	
Diabetes		0.341		
Yes	1			
No	0.63[95% CI, 0.24,1.63]			
Smoke		0.209		
Yes	1			
No	1.62[95% CI, 0.76,3.42]			
Alcohol addiction		0.993		
Yes	1			
No	0.99[95% CI, 0.28,3.51]			
Cerebral Infarction		0.003		
Yes	1			
No	0.28[95% CI, 0.12,0.65]			
Age		0.001		
> 55	1		1	
≤ 55	0.38[95% CI, 0.22,0.67]		0.44[95% CI, 0.15,1.32]	0.142
BMI		0.012		
> 23	1			
≤ 23	2.02[95% CI, 1.16,3.51]			
HCT		0.181		
>0.38	1			
≤ 0.38	1.43[95% CI, 0.85,2.43]			
WBC		0.095		0.04
>13.4	1		1	
≤ 13.4	0.64[95% CI, 0.38,1.08]		3.49[95% CI, 1.06,11.56]	
NB		0.185		
> 11.35	1			
Continued				

Variable	OR[95%CI]	*p*	OR[95%CI]	*p*
≤ 11.35	0.70[95% CI, 0.41,1.19]			
LY		0.095		
> 1.33	1			
≤ 1.33	1.64[95% CI, 0.92,2.94]			
MO		0.196		
> 0.69	1			
≤ 0.69	0.70[95% CI, 0.41,1.20]			
PLT		0.289		
> 240	1			
≤ 240	1.33[95% CI, 0.78,2.27]			
PT		0.787		
> 13.1	1			
≤ 13.1	1.08[95% CI, 0.63,1.82]			
APTT		0.452		
>33.5	1			
≤ 33.5	1.23[95% CI, 0.72,2.08]			
D-dimer		< 0.001		
>3290	1			
≤ 3290	0.27[95% CI, 0.15,0.47]			
FIB		0.436		
> 3.4	1			
≤ 3.4	0.81[95% CI, 0.48,1.37]			
FBG		< 0.001		
> 146.6	1			
≤ 146.6	0.30[95% CI, 0.18,0.53]			
TP		0.021		0.103
> 71	1		1	
≤ 71	1.88[95% CI, 1.10,3.20]		2.35[95% CI, 0.84,6.55]	
Alb		0.012		
> 41.5	1			
≤ 41.5	1.99[95% CI, 1.16,3.40]			
PCT		< 0.001		0.084
> 0.57	1		1	
≤ 0.57	0.13[95% CI, 0.06,0.27]		0.31[95% CI, 0.08,1.17]	
TBil		0.743		
> 12.4	1			
≤ 12.4	0.91[95% CI, 0.52,1.60]			
DBil		0.137		
>4.1	1			
≤ 4.1	0.65[95% CI, 0.37,1.14]			
ALT		0.021		
>21	1			
≤ 21	0.52[95% CI, 0.30,0.91]			
AST		< 0.001		0.142
> 26	1		1	
≤ 26	0.29[95% CI, 0.17,0.51]		0.44[95% CI, 0.15,1.32]	
TC		0.058		
> 4.94	1			
≤ 4.94	1.77[95% CI, 0.98,3.22]			
TG		0.91		
>120.68	1			
≤ 120.68	0.97[95% CI, 0.54,1.74]			
HDL-c		0.159		
>51.6	1			
≤ 51.6	1.51[95% CI, 0.85,2.68]			
LDL-c		0.141		
Continued				

Variable	OR[95%CI]	*p*	OR[95%CI]	*p*
> 112.4	1			
≤ 112.4	1.52[95% CI, 0.87,2.66]			
Urea		0.005		
> 4.7	1			
≤ 4.7	0.47[95% CI, 0.27,0.8]			
Cre		0.111		
> 71.2	1			
≤ 71.2	0.65[95% CI, 0.38,1.10]			
Na		0.577		
> 139.5	1			
≤ 139.5	1.16[95% CI, 0.69,1.97]			
K		0.805		
> 3.6	1			
≤ 3.6	0.94[95% CI, 0.55,1.58]			
Cl		0.028		
> 103	1			
≤ 103	0.54[95% CI, 0.32,0.93]			
Ca		< 0.001		
> 2.21	1			
≤ 2.21	2.95[95% CI, 1.65,5.28]			
NLR		0.239		
> 11.5	1			
≤ 11.5	0.73[95% CI, 0.43,1.23]			
dNLR		0.791		
>0.945	1			
≤ 0.945	0.93[95% CI, 0.55,1.58]			
MLR		< 0.001		0.035
> 0.61	1		1	
≤ 0.61	0.35[95% CI, 0.21,0.61]		0.33[95% CI, 0.12,0.92]	
NMLR		0.235		
>12.14	1			
≤ 12.14	0.73[95% CI, 0.43,1.23]			
SIRI		0.001		
> 7.65	1			
≤ 7.65	0.39[95% CI, 0.23,0.67]			
SII		0.935		
> 2655.7	1			
≤ 2655.7	1.02[95% CI, 0.59,1.77]			
PLR		0.78		
> 231.5	1			
≤ 231.5	0.93[95% CI, 0.55,1.58]			
AISI		0.007		
> 1733.2	1			
≤ 1733.2	0.47[95% CI, 0.28,0.81]			
NHR		0.204		
> 0.243	1			
≤ 0.243	0.70[95% CI, 0.40,1.21]			
MHR		0.555		
>0.0154	1			
≤ 0.0154	0.70[95% CI, 0.40,1.21]			
LHR		0.601		
>0.0298	1			
≤ 0.0298	1.17[95% CI, 0.65,2.11]			
PHR		0.962		
>5.147	1			
≤ 5.147	1.01[95% CI, 0.58,1.76]			
Continued				

Variable	OR[95%CI]	*p*	OR[95%CI]	*p*
TyG		0.3		
> 8.832	1			
≤ 8.832	0.75[95% CI, 0.43,1.30]			
TyG-BMI		0.59		
> 203.47	1			
≤ 203.47	1.16[95% CI, 0.67,2.02]			

Table 3. Single and multivariate binary logistic regression analysis results.

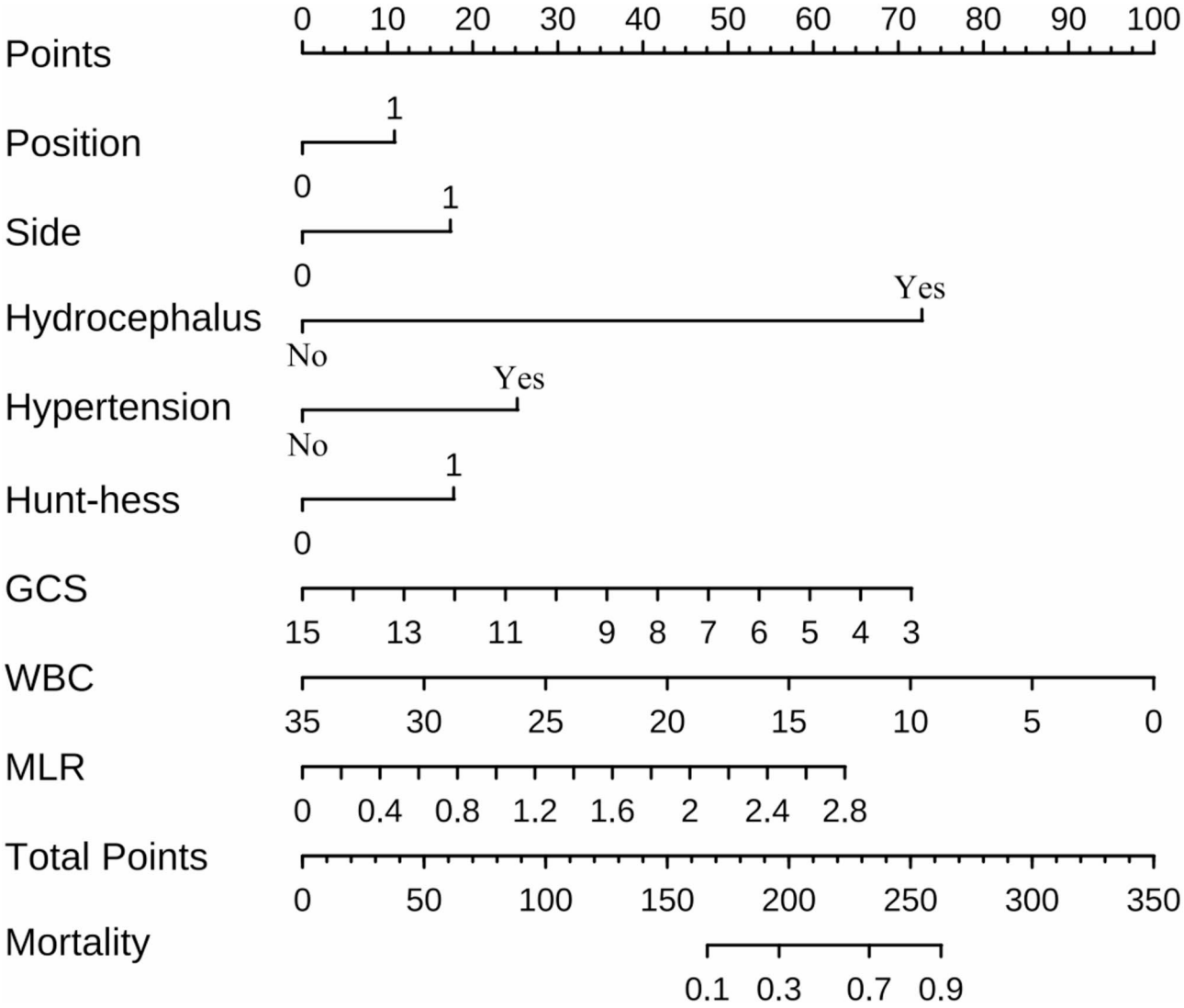

Fig. 2. The nomogram.

MLR may increase the risk of a poor prognosis six months after surgery. From previous studies, we can see that monocytes, which originate from bone marrow and circulate in the bloodstream[52], are critical components of the innate immune system and play a vital role in the initiation, regulation and resolution of inflammation through cytokine production and antigen presentation[53]. aSAH can also trigger early monocyte activation[54]. In a study by Walsh et al., absolute monocyte counts were independently associated with 30-day mortality in 240 adult patients with cerebral hemorrhage (ICH)[55]. In a previous study by Mackey et al., elevated monocyte count was also an independent risk factor for 30-day death from disease[56]. In addition, a previous study calculated the lymphocyte to monocyte neutrophil (L/MN) gene list darr ratio in patients with aneurysms. It demonstrated that lower values were significantly associated with mortality during hospitalization. Therefore, it can also be concluded that monocyte and neutrophil activity increases after aSAH while lymphocyte response decreases[57].

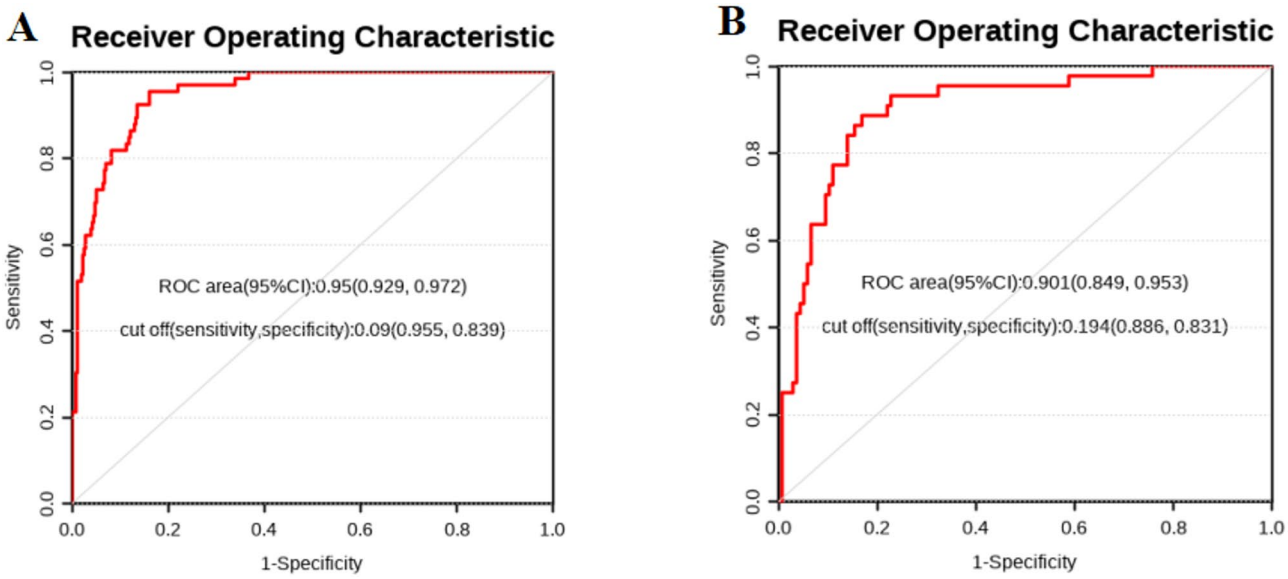

Fig. 3. R0C curve.

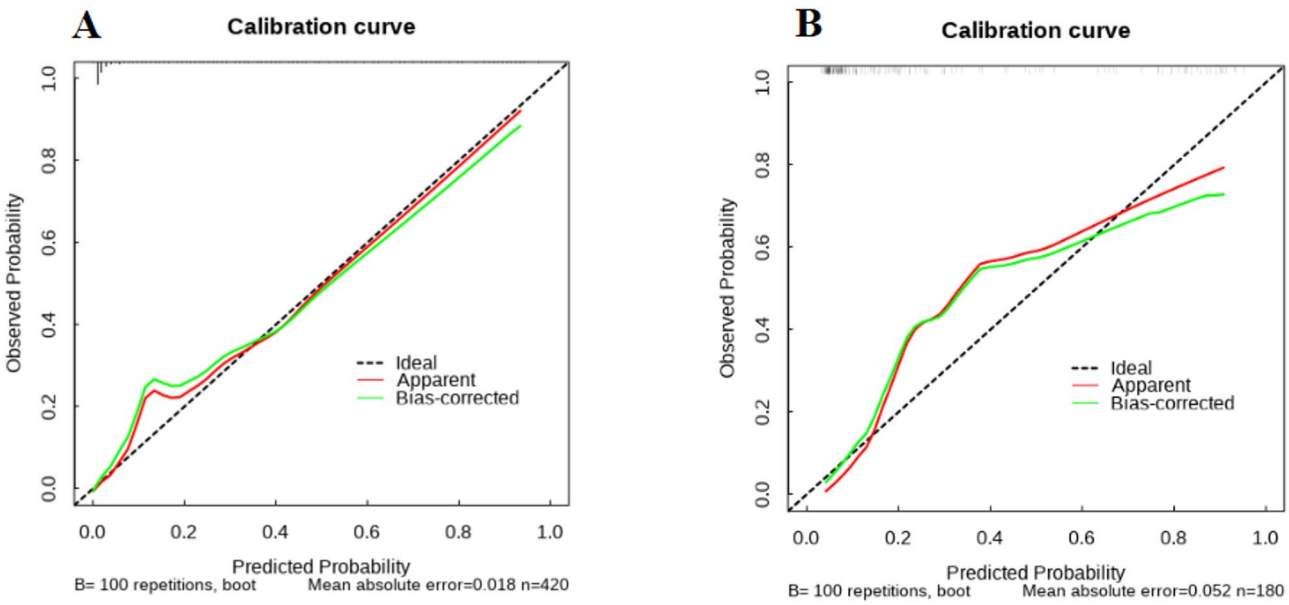

Fig. 4. Calibration curve.

Therefore, the greater the MLR ratio, the worse the prognosis of patients, which is consistent with the results of our study. It is important to note that the process of early brain injury, vasospasm, and delayed cerebral deficiency is actually complex and multifactorial. These processes may involve other mechanisms and pathways that interact with inflammatory pathways and monocytes[58]. This needs to be further studied.

Since the prevention and treatment of aSAH are of great clinical significance, it is necessary to evaluate the prognosis of patients as early as possible. Therefore, the development of the nomogram of adverse prognostic factors of aSAH has become a hot topic for many scholars in recent years. However, at present, there is no nomographic map for poor prognosis of patients with aSAH based on aneurysm location, affected side, hydrocephalus, Hunt-Hess grade, GCS score, hypertension, WBC and MLR. Meanwhile, the nomographic map in this study has good predictive performance. The construction of a nomogram based on the blood test results of patients admitted to the hospital can achieve clinical benefits for a large proportion of patients. Compared to the HATCH score (AUC = 0.82)[28] and the FRESH score (AUC = 0.87)[59], our nomogram achieved higher discriminative ability (AUC = 0.95). For another example, compared with the dynamic nomogram (AUC = 0.86) developed by Zhuang et al. (2023)[15], the AUC of the training set and validation set of this model is 0.95 and 0.90, respectively, showing a higher degree of differentiation. Unlike previous models, we incorporated inflammatory

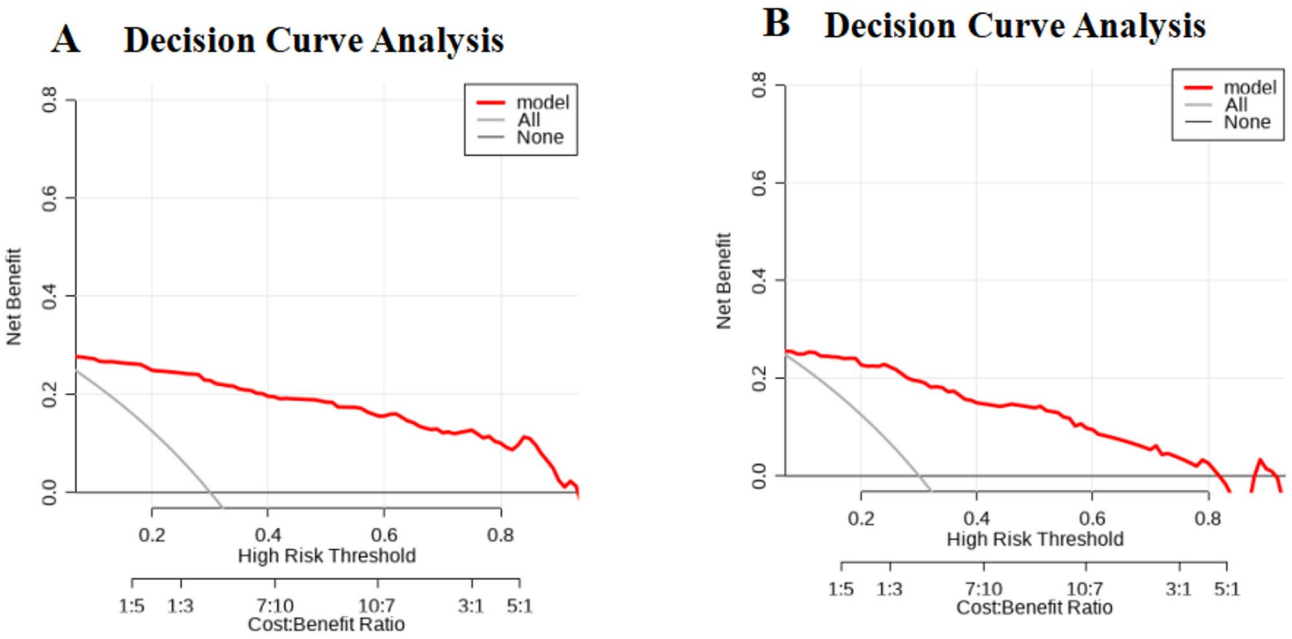

Fig. 5. DCA curve.

Metric/ Predictors	Original Model (95% CI)	Undersampled Model (95% CI)
AUC-ROC	0.950 (0.929–0.972)	0.967 (0.931–0.993)
AUC-PR	0.791 (0.685–0.876)	0.968 (0.935–0.993)
Mean Absolute Error	0.018	0.017
Key Predictors	Position, Side, Hydrocephalus, Hypertension, Hunthess, GCS, WBC, MLR	MLR, WBC, Hydrocephalus, Hunthess, GCS

Table 4. Comparison between original model and undersampled model.

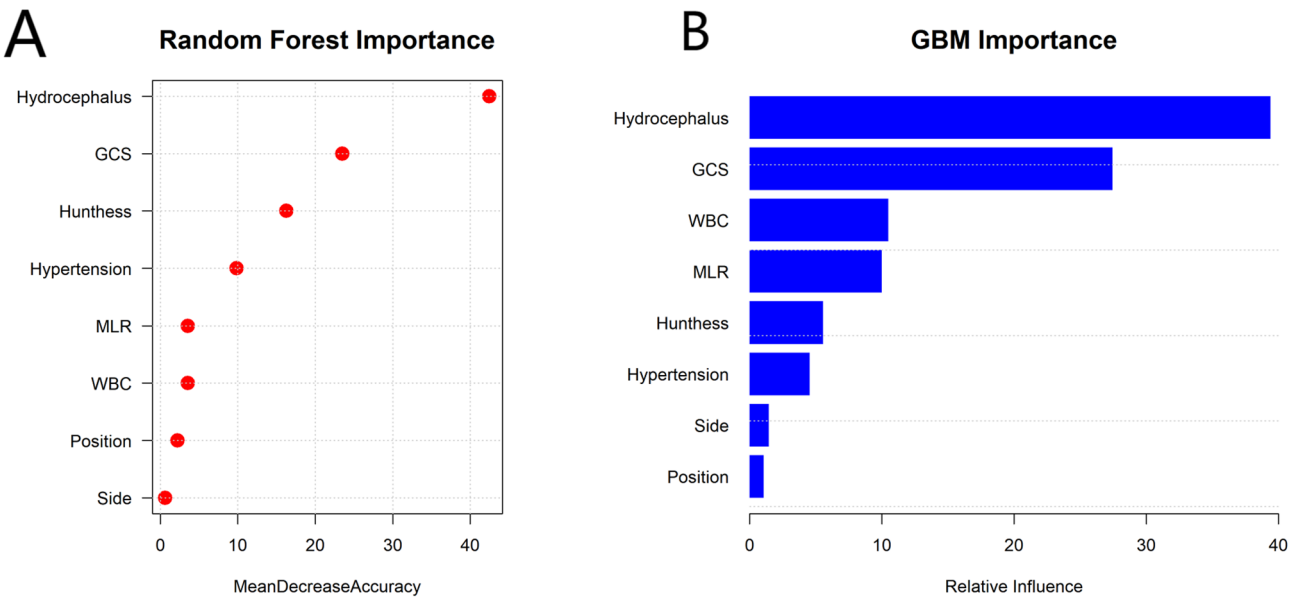

Fig. 6. Machine learning model results. Data 6 A Random Forest Feature Importance: Hydrocephalus (41.6%). GCS (23%). Hunthess (15.9%). Hypertension (9.7%). WBC (3.5%). MLR (3.5%). Position (2.2%). Side (0.6%). Data 6 B Gradient Boosting Feature Importance: Hydrocephalus (39.4%). GCS (27.4%). WBC (10.5%). MLR (10%). Hunthess (5.6%). Hypertension (4.6%). Side (1.5%). Position (1.1%).

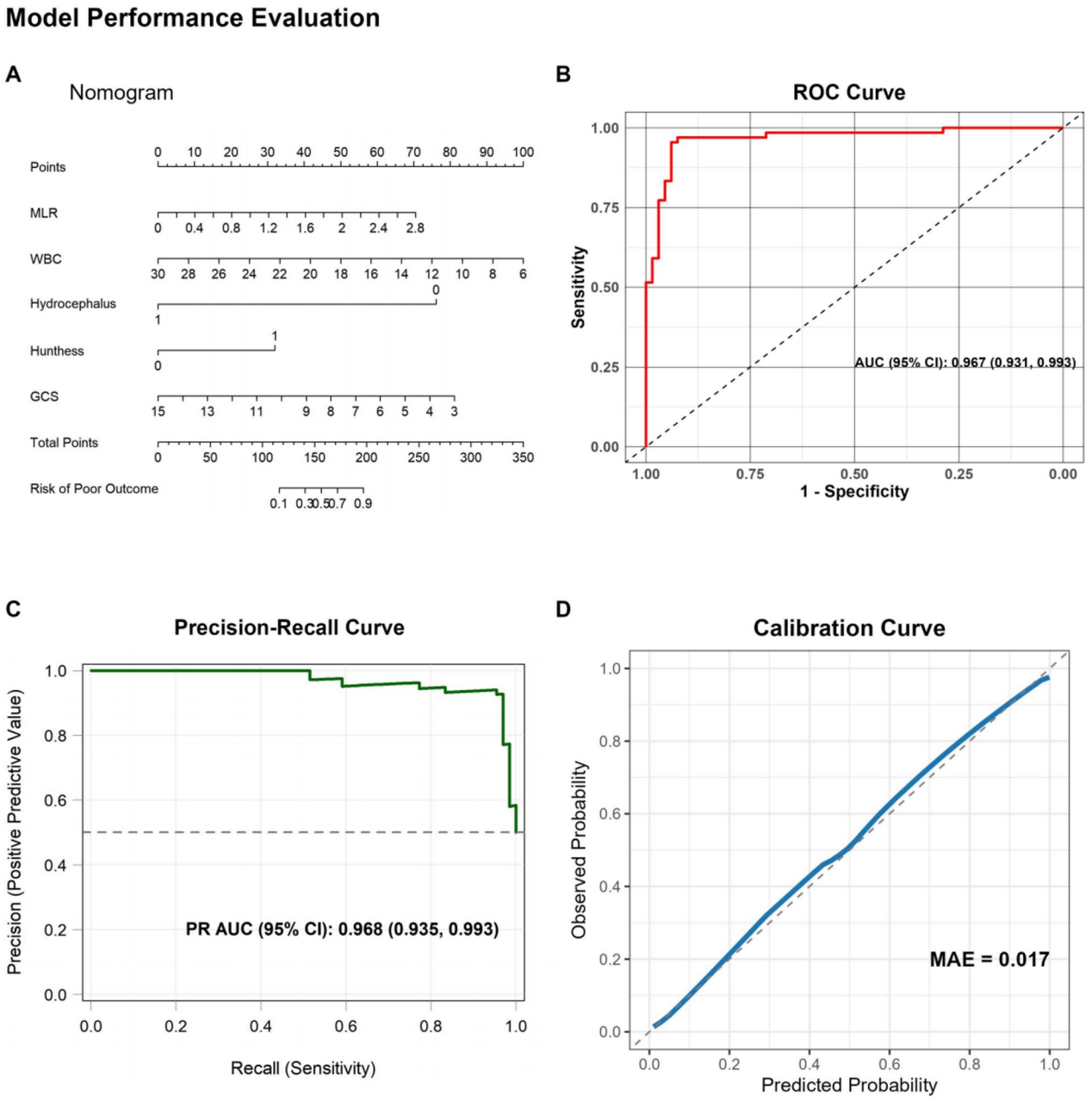

Fig. 7. Downsampled Model performance evaluation.

biomarkers (e.g., MLR) and hydrocephalus status, which may enhance predictive accuracy for short-term outcomes. Beside, decision curve analysis (DCA) shows that the model provides a net benefit for clinical decisions when the threshold probability is in the range of 0.01 to 0.98. For example, if the physician thinks the probability of a poor patient prognosis exceeds 20% (threshold = 0.2), using this model can significantly reduce unnecessary interventions while avoiding missing high-risk patients. This implies that clinicians can confidently use the model to stratify high-risk patients for early interventions, even when varying the acceptable risk thresholds for treatment.

In this study, the model was constructed based on the data from two medical centers in China, and the internal validation set was conducted (AUC = 0.901), indicating that the model has good generalization ability. However, because the sample source is limited in southern China, the external validation in more geographical and ethnic populations is needed in the future to further confirm the universality of the model. Although individual factors (e.g. -Hunt-Hess grade, GCS score) have been reported as predictors of SAH outcome, for the first time integrated aneurysm location, affected side, hydrocephalus, hypertension, WBC, and MLR into one nomogram model. This multi-dimensional integration enables a more comprehensive assessment of short-term patient outcomes, providing an intuitive quantification tool for the clinic.

This study also has some advantages: (1) As a two-center retrospective study from China, the nomogram was verified by an external validation set, the model was reliable, and the application was simple and convenient; (2) The purpose of this study is to collect the relevant data of patient admission, so as to achieve the very early prediction and strive to benefit more patients; (3) However, this study also has some disadvantages: (1) patients have a certain rate of lost follow-up; (2) The center and data volume can be further expanded; (3) As a retrospective study, selection bias may be information bias (such as some confounders not documented). Imbalance in outcome categories is also the main limitation. Although sensitivity analyses supported the robustness of the main findings, future prospective, multi-center studies with a more balanced sample distribution are recommended. Despite the bias reduction through a multicenter design and strict exclusion criteria, future prospective studies are needed to verify the robustness of the model.

Conclusion

The nomogram constructed based on aneurysm location, affected side, presence of hydrocephalus, Hunt-Hess grade, GCS score, hypertension, WBC and MLR can help clinicians to identify high-risk aSAH patients early, facilitating targeted interventions such as anti-inflammatory therapy or hydrocephalus management. This tool may improve resource allocation and reduce disability rates in critical care settings. Thus optimizing the intervention strategy and improve patient quality of life.

Data availability

The data used to support the findings of this study are available from the corresponding author upon request.

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Author contributions
HHL and GZ designed this study. YCW, XYL and MJL are responsible for writing articles, conducting statistical analysis, reviewing articles, and creating images.WCL are responsible for collecting data and conducting statistical analysis.All authors reviewed the manuscript. All authors read and approved the final manuscript.

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Declarations

Consent for publication
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Competing interests
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Ethical approval
Data access was restricted to authorized researchers, and the study complied with China's Personal Information Protection Law (PIPL). The study has been approved by the Committee of Huizhou Central People's Hospital, and due to the retrospective nature of the study, the Huizhou Central People's Hospital Ethics Review Committee waived the need of obtaining informed consent. It complies with the Declaration of Helsinki and the Chinese Measures for Ethical Review of Biomedical Research Involving People.

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