

Development of a Prognostic Model and Risk Factor Analysis for Spontaneous Intracerebral Hemorrhage Patients Based on Clinical and Laboratory Indicators

Xiangning He

Qingdao University

Yulun Wang

Qingdao University

Qian Chen

Taian City Central Hospital

Haochen Yang

Qingdao University

Zhenyu Geng

Qingdao University

Jiahao Liu

Qingdao University

Xunlu Yin

Qingdao University

Yawei Rong

Taian City Central Hospital

Shizhong Zhang

Taian City Central Hospital

GuoJun Wang

Taian City Central Hospital

Fangfang Zhao

taszxuyzff@sina.com

Taian City Central Hospital

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Abstract

Background

This study identified independent risk factors for poor 30 day prognosis in spontaneous intracerebral hemorrhage (ICH) and developed a nomogram.

Methods

This retrospective study included 376 patients admitted for spontaneous intracerebral hemorrhage (ICH) between December 2014 and August 2025, who were randomly divided into a training set and a validation set in a 7:3 ratio. A poor prognosis was defined as an mRS score of ≥ 3 at 30 days. Risk factors were identified using LASSO and multivariate logistic regression, and a nomogram model was constructed. Model performance was assessed using ROC curves, calibration curves and decision curve analysis (DCA).

Results

In the training set, 174 patients (66.2%) had a poor prognosis. Independent predictors included: GCS score on admission GCS (3–8) (OR = 21.882, 95% CI = 8.394–67.786), GCS (9–12) (OR = 9.477, 95% CI = 3.883–25.656), EHE (OR = 2.629, 95% CI = 1.144–6.349), HTN (OR = 2.866, 95% CI = 1.214–7.076), IVH (OR = 3.346, 95% CI = 1.573–7.467), V1 (OR = 1.039, 95% CI = 1.012–1.07), APTT (OR = 1.219, 95% CI = 1.101–1.365), and TyG (OR = 1.676, 95% CI = 1.07–2.717). The nomogram demonstrated an AUC of 0.885 (95% CI: 0.844–0.927) in the training set and 0.742 (95% CI: 0.647–0.837) in the validation set. Calibration curves and decision curve analysis confirmed good calibration and clinical net benefit.

Conclusion

This nomogram reliably predicts 30 day poor outcomes, aiding early risk stratification and personalized care.

1. Introduction

Spontaneous intracerebral hemorrhage (ICH) refers to intracranial bleeding caused by the rupture of large or small arteries, veins or capillaries within the skull in the absence of trauma; it has become a serious global public health issue, with a 30-day mortality rate as high as 40–50 percent[1–3]. Despite early intervention with medical treatments such as dehydration to reduce intracranial pressure, sedation and analgesia, as well as surgical procedures including minimally invasive burr hole drainage and ventriculostomy, the prognosis remains poor[4, 5]. We found that the original ICH Score, ICH-FUNCTIONAL OUTCOME SCORE (ICH-FOS), Essen-ICH Score, max-ICH Score, and FUNC Score lack the

inclusion of blood test parameters upon admission. However, it remains unclear whether common clinical blood parameters in patients with early-stage intracerebral hemorrhage upon admission can reflect their prognosis. Although many prognostic factors have been well established for years, several controversial ones have recently been reported by investigators [6]. Consequently, this study aims to identify key prognostic risk factors through a retrospective analysis. A prognostic model will be developed and subjected to internal validation to assess its validity, thereby providing a reference for determining patient prognosis and facilitating early intervention.

2. Materials and methods

2.1 Study population

We selected patients with intracerebral hemorrhage who were admitted to the Department of Neurosurgery at Taian Central Hospital, Affiliated to Qingdao University, between December 2014 and August 2025. A total of 706 patients with ICH were included in the initial cohort. The patients included in this study all received standard clinical treatment, including but not limited to conservative management and various surgical interventions. Following the application of eligibility criteria, a total of 376 cases were included in the final analysis and divided into a training set and a validation set in a 7:3 ratio, comprising 263 cases in the training set and 113 cases in the validation set (Fig. 1).

Inclusion criteria were as follows

(1) Time from onset to admission < 24 hours; (2) Meet the ICH diagnostic criteria and is confirmed by imaging examinations at our hospital [7]; (3) Age $\geq$ 18 years; (4) The interval from symptom onset to the completion of laboratory and imaging examinations was less than 24 hours. All patients were required to have two imaging examinations obtained within 48 hours after onset, with an interval of more than 24 hours between the two scans; (5) Complete medical records, including necessary laboratory test results and imaging findings;

Exclusion criteria included

(1) Patients with hemorrhagic transformation of ischemic stroke, hemorrhage from a brain tumor, recurrent cerebral hemorrhage, secondary cerebral hemorrhage (e.g. following traumatic brain injury), vasculitis or other medical conditions; (2) Patients with severe coagulopathy or dysfunction/failure of other vital organs (e.g. NYHA Class IV heart failure, uremia, liver failure); (3) Patients with missing required laboratory data or fewer than two imaging examinations within 48 hours after symptom onset. All patients were treated in accordance with the 2022 Guidelines for the Management of Patients with Spontaneous Intracerebral Hemorrhage during their hospital stay [7].

This study has been approved by the institutional review board of Taian Central Hospital (approval number, 2026-01-K515).

2.2 Patient grouping

The Glasgow Coma Scale (GCS) score was obtained within 30 minutes of admission[8]. The 30-day modified Rankin Scale (mRS) score was retrospectively collected by reviewing patients' inpatient medical records[9]. For patients with a hospital stay of less than 28 days or those who died during hospitalization, the mRS score was directly determined via telephone follow-up or discharge evaluation. To ensure the objectivity and consistency of the assessments, each patient was independently evaluated by two senior neurosurgeons. Any scoring discrepancy was resolved through discussion to reach a consensus, and the unified data results were recorded. Patients with an mRS score of ≥ 3 were defined as having a poor prognosis, whereas those with an mRS score of ≤ 2 were defined as having a favorable prognosis. A repeat CT scan was performed within 48 hours of admission. All CT images were jointly evaluated by two senior neurosurgeons, and the unified data results were recorded.

2.3 Data collection

2.3.1 Baseline data

Baseline data were collected on admission, including Age, Gender, Onset time, Admission GCS score (GCS), History of hypertension (HTN), History of diabetes (DM), History of hyperlipidemia (HLP), History of cerebral infarction (CI), systolic blood pressure on admission (SBP), diastolic blood pressure on admission (DBP). The mRS score at 30 days after onset was recorded as the prognostic outcome.

2.3.2 Laboratory tests

Venous blood samples were taken within 24 hours of admission to measure neutrophil count (Neut), neutrophil percentage (Neut%), lymphocyte count (Lym), lymphocyte percentage (Lym%), red blood cells (RBC), hemoglobin (Hb), hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), red blood cell distribution width (RDW), Eosinophil count (Eos), Eosinophil percentage (Eos%), Basophil count (Baso), Basophil percentage (Baso%), Platelets (PLT), Platelet distribution width (PDW), Platelet hematocrit (PCT), Prothrombin activity (PTA), Prothrombin time ratio (PTR), International Normalised Ratio (INR), Activated partial thromboplastin time (APTT), Fibrinogen (FIB), Fibrinogen clotting time (FCT), Thrombin time (TT), Total protein (TP), Albumin (Alb), Globulin (Glb), albumin-to-globulin ratio (A/G), prealbumin (PA), aspartate transaminase (AST), alanine transaminase (ALT), AST/ALT ratio (AST/ALT), γ -glutamyltransferase (GGT), total bilirubin (TBil), direct bilirubin (DBil), direct bilirubin/ Total Bilirubin (DBil/TBil), urea (Ur), creatinine (Cr), uric acid (UA), superoxide dismutase (SOD), Sialic acid (SA), Lactate dehydrogenase (LDH), Creatine kinase (CK), Creatine kinase isoenzyme (CK-MB), α -Hydroxybutyrate dehydrogenase (HBDH), total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), lipoprotein (a) (Lp(a)), lipoprotein(a)/high-density lipoprotein cholesterol (Lp(a)/HDL-C), homocysteine (Hcy), lymphocyte-to-creatinine ratio% (LCR%), neutrophil-to-lymphocyte ratio (NLR), platelet-to- albumin ratio (PAR), Triglyceride-Glucose index (TyG).

2.3.3 Comparison of imaging data

Immediately upon admission, a plain cranial CT scan was performed to determine whether there was any bleeding, the location of the hemorrhage, whether the hematoma had ruptured into the ventricles (IVH), and whether there was any midline shift. The volume of the hematoma was calculated using the Saito formula ($V = ABC/2$, where A is the longest diameter of the hematoma in the axial plane, B is the longest diameter perpendicular to A in the same plane, and C is the number of visible hemorrhagic layers multiplied by the thickness of each layer) [10]. A head CT scan was repeated within 48 hours. If the hematoma volume increases by > 33% or the absolute volume change compared with the initial scan is > 6 mL, this is defined as early hematoma expansion (EHE) [10]. Measure and calculate the admission hematoma volume (V1), as well as early hematoma expansion (EHE).

2.3.4 Statistical analysis

The statistical analysis in this study was conducted using R software (version 4.5.3)[11]. The 376 study participants were randomly divided into a training group (263 participants) and a validation group (113 participants) in a 7:3 ratio. In the training group, continuous data were tested for normality: continuous data that followed a normal distribution were expressed as mean $\pm$ standard deviation ($\bar{x} \pm s$), and comparisons between groups were performed using an independent samples t-test; continuous data that did not follow a normal distribution were expressed as the interquartile range M (P25, P75), and comparisons between groups were performed using the Mann-Whitney U test. Categorical data are expressed as percentages (%), and comparisons between groups were performed using the chi-square test. In the training set, feature selection for 69 variables was conducted using the least absolute shrinkage and selection operator (LASSO), and 12 features with non-zero coefficients corresponding to one standard error of the optimal λ value were initially identified. After assessing multicollinearity by the variance inflation factor (VIF), these 12 candidates were entered into a multivariable logistic regression model using stepwise backward elimination to identify independent prognostic factors. Seven variables were ultimately retained, and a predictive nomogram was constructed based on these predictors.

The discriminative ability of the nomogram was evaluated using receiver operating characteristic (ROC) curves and the area under the curve (AUC) in both the training and validation sets. Internal validation was performed via 1,000 bootstrap resamples to calculate the C-index. Model calibration was assessed using the Hosmer-Lemeshow goodness-of-fit test and calibration plots with 1,000 bootstrap resamples; the Brier score, calibration slope, and calibration intercept were also computed. Clinical net benefit was assessed using decision curve analysis (DCA). All performance evaluations were applied in both the training and validation sets.

3. Results

3.1 Baseline characteristics

Of the 263 patients included in the training set for this study, 89 (33.8%) were in the favorable prognosis group ($mRS \leq 2$) and 174 (66.2%) were in the poor prognosis group ($mRS \geq 3$). There were statistically significant differences between the two groups in terms of GCS scores on admission, V1, EHE, IVH,

Lym%, MCH, PTA, APTT, FIB, FCT, A/G, PA, ALT, DBil/TBil, SOD, SA, LDH, CK/MB, NLR, and TyG ($p < 0.05$). There were no statistically significant differences between the two groups in terms of other factors ($p > 0.05$) (Table 1).

Table 1
Baseline characteristics of the favorable prognosis group and the poor prognosis group

Variables	Total(n = 263)	mRS ≤ 2(n = 89)	mRS ≥ 3(n = 174)	p-value
Age(n), Median (Q1, Q3)	63 (55, 70)	64 (55, 72)	62 (55, 70)	0.486
Gender, (n)				0.788
female	99 (38)	32 (36)	67 (39)	
male	164 (62)	57 (64)	107 (61)	
Admission GCS score, (n)				< 0.001
13–15	124 (47)	74 (83)	50 (29)	
9–12	49 (19)	9 (10)	40 (23)	
3–8	90 (34)	6 (7)	84 (48)	
History of hypertension (n)				0.429
No	59 (22)	23 (26)	36 (21)	
Yes	204 (78)	66 (74)	138 (79)	
History of diabetes (n)				0.093
No	235 (89)	84 (94)	151 (87)	
Yes	28 (11)	5 (6)	23 (13)	
History of hyperlipidemia (n)				0.553
No	260 (99)	89 (100)	171 (98)	
Yes	3 (1)	0 (0)	3 (2)	
History of cerebral infarction (n)				0.665
No	157 (60)	51 (57)	106 (61)	
Yes	106 (40)	38 (43)	68 (39)	
Admission SBP (mmHg), Median (Q1, Q3)	156 (137, 180)	156 (137, 180)	155.5 (137, 180)	0.881
Admission DBP (mmHg), Median (Q1, Q3)	88 (79, 103)	88 (78, 100)	88.5 (79.25, 103)	0.729
Onset time (n), Median (Q1, Q3)	3 (2, 5)	3 (2, 5)	3 (2, 5)	0.212

Variables	Total(n = 263)	mRS ≤ 2(n = 89)	mRS ≥ 3(n = 174)	p-value
V1(ml), Median (Q1, Q3)	9.06 (4.67, 21.59)	5.54 (2.8, 9.13)	12.73 (6.34, 28.97)	< 0.001
EHE, n (%)				0.025
NO	205 (78)	77 (87)	128 (74)	
Yes	58 (22)	12 (13)	46 (26)	
IVH, n (%)				0.036
NO	174 (66)	67 (75)	107 (61)	
Yes	89 (34)	22 (25)	67 (39)	
Neut(×10 ⁹ /L), Median (Q1, Q3)	6.95 (4.58, 9.37)	6.69 (4.45, 9)	7.48 (4.75, 9.69)	0.057
Neut% (%), Median (Q1, Q3)	79.2 (70.8, 85.55)	78 (70.3, 84.3)	79.8 (71.08, 86.85)	0.105
Lym(×10 ⁹ /L), Median (Q1, Q3)	1.05 (0.79, 1.48)	1.09 (0.86, 1.43)	1.02 (0.74, 1.48)	0.195
Lym% (%), Median (Q1, Q3)	12.6 (8.35, 19.3)	13.3 (10.3, 19.7)	12.05 (7.23, 19.05)	0.026
RBC(×10 ¹² /L), Mean ± SD	4.3 ± 0.66	4.37 ± 0.56	4.27 ± 0.7	0.228
Hb(g/L), Median (Q1, Q3)	119 (30.7, 165.5)	118 (31.1, 168)	119.5 (30.5, 163.5)	0.245
HCT(L/L), Median (Q1, Q3)	0.39 (0.36, 0.43)	0.39 (0.36, 0.43)	0.39 (0.36, 0.43)	0.767
MCV (fL), Median (Q1, Q3)	90 (87.25, 92.9)	90 (87.7, 93.6)	90.1 (87.1, 92.68)	0.592
MCH (pg), Median (Q1, Q3)	30.5 (29.5, 31.7)	30.9 (29.9, 32.1)	30.4 (29.3, 31.5)	0.022
RDW (%), Median (Q1, Q3)	41.6 (40, 44.35)	41.6 (39.5, 44)	41.75 (40.12, 44.68)	0.307
Eos(×10 ⁹ /L), Median (Q1, Q3)	0.04 (0, 0.12)	0.05 (0.01, 0.08)	0.03 (0, 0.13)	0.591
Eos% (%), Median (Q1, Q3)	0.5 (0.05, 1.5)	0.6 (0.1, 1.4)	0.4 (0, 1.5)	0.447
Baso(×10 ⁹ /L), Median (Q1, Q3)	0.02 (0.01, 0.03)	0.02 (0.01, 0.03)	0.02 (0.01, 0.03)	0.923
Baso% (%), Median (Q1, Q3)	0.2 (0.1, 0.3)	0.2 (0.1, 0.3)	0.2 (0.1, 0.3)	0.172
PLT((×10 ⁹ /L)), Median (Q1, Q3)	219 (183.5, 255)	222 (185, 259)	216 (183.25, 253.75)	0.717
PDW (fL), Median (Q1, Q3)	11.4 (10.4, 12.85)	11.2 (10.4, 12.6)	11.45 (10.5, 13)	0.186
PCT (%), Median (Q1, Q3)	0.22 (0.19, 0.26)	0.22 (0.19, 0.27)	0.22 (0.19, 0.26)	0.793

Variables	Total(n = 263)	mRS ≤ 2(n = 89)	mRS ≥ 3(n = 174)	p-value
PTA (%), Median (Q1, Q3)	89.1 (84.55, 94.5)	90.9 (87, 96)	88.9 (83.15, 94.5)	0.015
PTR, Median (Q1, Q3)	1.08 (1.03, 1.14)	1.07 (1.02, 1.13)	1.08 (1.04, 1.15)	0.05
INR, Median (Q1, Q3)	1.08 (1.03, 1.14)	1.07 (1.03, 1.13)	1.08 (1.04, 1.15)	0.092
APTT(s), Median (Q1, Q3)	26.2 (23.7, 29)	25.5 (23, 28.2)	26.7 (24.13, 30.12)	0.004
FIB(g/L), Median (Q1, Q3)	2.69 (2.34, 3.22)	2.65 (2.27, 2.98)	2.72 (2.39, 3.4)	0.012
FCT(s), Mean ± SD	9.03 ± 2.17	9.51 ± 1.69	8.79 ± 2.35	0.005
TT(s), Mean ± SD	16.14 ± 1.05	16.26 ± 0.93	16.08 ± 1.11	0.164
TP(g/L), Mean ± SD	64.99 ± 5.57	64.45 ± 4.78	65.27 ± 5.93	0.229
Alb(g/L), Median (Q1, Q3)	50 (40, 152.95)	50.2 (40.2, 116.6)	48.2 (39.5, 154.43)	0.545
Glb (g/L), Median (Q1, Q3)	21.7 (2.5, 25.4)	21.3 (2.46, 23.8)	21.85 (2.53, 26)	0.219
A/G, Median (Q1, Q3)	1.68 (1.5, 1.9)	1.74 (1.61, 2)	1.6 (1.39, 1.85)	< 0.001
PA (mg/L), Mean ± SD	219.18 ± 66.87	232.37 ± 61.65	212.44 ± 68.58	0.018
AST(U/L), Median (Q1, Q3)	20.1 (16.25, 25.7)	20 (16, 23.6)	21 (16.72, 27.08)	0.069
ALT(U/L), Median (Q1, Q3)	17.2 (12.6, 24.55)	16.2 (12.1, 21)	18 (13.05, 26)	0.025
AST/ALT, Median (Q1, Q3)	1.18 (0.88, 1.48)	1.21 (0.93, 1.58)	1.17 (0.87, 1.45)	0.28
GGT(U/L), Median (Q1, Q3)	21.6 (14.1, 34.5)	21 (14.2, 33.2)	22.05 (14.2, 34.75)	0.552
TBil (μmol/L), Median (Q1, Q3)	12.5 (8.35, 16.8)	13.1 (10.1, 16.3)	12.2 (7.8, 17.08)	0.141
DBil (μmol/L), Median (Q1, Q3)	5.3 (3.7, 7.3)	5.1 (3.8, 6.9)	5.4 (3.7, 7.4)	0.963
DBil/TBil, Median (Q1, Q3)	0.46 (0.38, 0.51)	0.41 (0.34, 0.48)	0.47 (0.4, 0.53)	< 0.001
Ur(mmol/L), Median (Q1, Q3)	4.7 (3.73, 6.09)	4.62 (3.82, 5.7)	4.8 (3.7, 6.32)	0.386
Cr (μmol/L), Median (Q1, Q3)	59 (49.5, 73)	59 (50, 70)	59 (49.25, 76)	0.67
UA (μmol/L), Median (Q1, Q3)	229 (174, 287)	235 (197, 281)	223 (153.25, 287)	0.269
SOD(U/mL), Median (Q1, Q3)	163 (139.5, 186)	172 (151, 194)	159.5 (132.25, 184)	0.004

Variables	Total(n = 263)	mRS ≤ 2(n = 89)	mRS ≥ 3(n = 174)	p-value
SA(g/L), Median (Q1, Q3)	58.9 (53.4, 70.05)	57 (52.3, 63)	60.3 (53.65, 73.25)	0.002
LDH(U/L), Median (Q1, Q3)	206 (177, 244)	201 (175, 221)	213.5 (178, 250)	0.017
CK(U/L), Median (Q1, Q3)	21 (11, 79.5)	24 (12, 91)	20 (11, 64.75)	0.222
CK/MB(U/L), Median (Q1, Q3)	5.92 (3.3, 16.3)	4.35 (3.07, 14)	9.39 (3.4, 18)	0.016
HBDH(U/L), Median (Q1, Q3)	159 (137, 185.5)	156 (138, 173)	161.5 (137, 193)	0.108
TC (mmol/L), Median (Q1, Q3)	4.26 (3.63, 5)	4.36 (3.79, 5.15)	4.18 (3.62, 4.94)	0.205
TG (mmol/L), Median (Q1, Q3)	0.91 (0.64, 1.45)	0.81 (0.59, 1.24)	0.94 (0.7, 1.53)	0.087
LDL-C(mmol/L), Median (Q1, Q3)	1.62 (0.5, 2.56)	1.62 (0.49, 2.6)	1.64 (0.55, 2.51)	0.772
HDL-C(mmol/L), Mean ± SD	1.34 ± 0.4	1.41 ± 0.4	1.31 ± 0.4	0.065
Lp(a)(mmol/L), Median (Q1, Q3)	151 (84.4, 295.55)	153.6 (80.3, 297.2)	150 (90.5, 291.75)	0.695
Lp(a)/HDL-C, Median (Q1, Q3)	122.88 (61.73, 225.99)	118.95 (54.19, 208.07)	124.86 (66.26, 228.47)	0.394
Hcy (μmol/L), Median (Q1, Q3)	12.5 (9.7, 17.6)	12.5 (10.6, 18)	12.55 (9.4, 16.98)	0.289
LCR% (%), Median (Q1, Q3)	1.72 (1.26, 2.56)	1.96 (1.4, 2.57)	1.65 (1.14, 2.54)	0.101
NLR, Median (Q1, Q3)	6.24 (3.71, 10.23)	5.84 (3.44, 8.03)	6.64 (3.88, 11.78)	0.033
PAR, Median (Q1, Q3)	4.09 (1.44, 5.52)	4.09 (1.76, 5.36)	4.18 (1.43, 5.58)	0.793
TyG, Median (Q1, Q3)	1.15 (0.69, 1.66)	0.99 (0.61, 1.44)	1.18 (0.78, 1.84)	0.031

3.2 Variable Selection via LASSO and Multivariable Logistic Regression

To identify the most pertinent predictors of poor prognosis following intracerebral hemorrhage, LASSO regression was integrated with multivariable logistic regression analysis. The LASSO algorithm was utilized to shrink coefficients and mitigate model overfitting during feature selection (Fig. 2A). Based on the "one standard error" criterion derived from cross-validation, 12 non-zero coefficient candidate predictors were initially extracted (GCS, EHE, HTN, DM, IVH, V1, SOD, A/G, CK-MB, APTT, NLR, and TyG) (Fig. 2B). Prior to model building, multicollinearity was assessed, revealing that all candidate variables had variance inflation factors (VIF) well below 1.2 (range: 1.015–1.183), indicating no significant collinearity (Supplementary Table 1). Subsequently, these 12 factors were entered into a multivariable logistic regression model using a stepwise backward elimination method (Supplementary Table 2).

Ultimately, seven variables were retained as independent predictors of an unfavorable outcome (Table 2). Among the indicators reflecting initial neurological injury, with an admission GCS score of 13–15 as the reference, GCS scores of 9–12 (OR = 9.477, 95% CI: 3.883–25.656) and 3–8 (OR = 21.882, 95% CI: 8.394–67.786) were both significantly associated with an increased risk of poor prognosis, and lower scores corresponded to a stepwise increase in the risk of unfavorable outcomes. Regarding imaging features, early hematoma expansion (EHE) (OR = 2.629, 95% CI: 1.144–6.349), intraventricular hemorrhage (IVH) (OR = 3.346, 95% CI: 1.573–7.467), and larger admission hematoma volume (V1) (OR = 1.039, 95% CI: 1.012–1.07) were independent risk factors for poor prognosis. Furthermore, a history of hypertension (HTN) (OR = 2.866, 95% CI: 1.214–7.076), prolonged activated partial thromboplastin time (APTT) at admission (OR = 1.219, 95% CI: 1.101–1.365), and an elevated triglyceride-glucose (TyG) index (OR = 1.676, 95% CI: 1.07–2.717) were also independently associated with unfavorable 30-day functional outcomes.

Table 2
Multivariate logistic regression analysis results for prognosis in ICH patients

variable	B	SE	Wald	OR	95%CI	P
(Intercept)	-8.15	1.717	22.536	0	0 ~ 0.007	< 0.001
GCS (9–12)	2.249	0.478	22.105	9.477	3.883 ~ 25.656	< 0.001
GCS (3–8)	3.086	0.527	34.305	21.882	8.394 ~ 67.786	< 0.001
EHE	0.967	0.434	4.951	2.629	1.144 ~ 6.349	0.026
HTN	1.053	0.447	5.549	2.866	1.214 ~ 7.076	0.018
IVH	1.208	0.396	9.323	3.346	1.573 ~ 7.467	0.002
V1	0.038	0.014	7.14	1.039	1.012 ~ 1.07	0.008
APTT	0.198	0.055	13.066	1.219	1.101 ~ 1.365	< 0.001
TyG	0.516	0.235	4.816	1.676	1.07 ~ 2.717	0.028
B, regression coefficient; SE, standard error; Wald, Wald test; OR, odds ratio						

3.3 Construction and Visualization of the Predictive Nomogram

To facilitate intuitive clinical application, a nomogram for predicting 30-day poor functional outcome (defined as an mRS score ≥ 3) was constructed based on the final multivariable logistic regression model. The nomogram incorporates the seven selected predictors (GCS, EHE, HTN, IVH, V1, APTT, and TyG). Clinicians can sum the individual scores assigned to a patient's specific characteristics to obtain a total score, which is then mapped to the probability axis at the bottom to rapidly estimate the patient's individual risk of experiencing an adverse outcome (Fig. 3). This tool streamlines the calculation process

and facilitates early identification of high-risk patients, thereby aiding in the formulation of personalized intervention strategies.

3.4 Model Performance Evaluation in the Training and Validation Sets

The discriminative ability and calibration of the nomogram were assessed using receiver operating characteristic (ROC) curves, bootstrap resampling, and calibration plots. In the training set, the area under the ROC curve (AUC) was 0.885 (95% CI: 0.844–0.927) (Fig. 4A). Internal validation via bootstrap resampling (1,000 repetitions) yielded a mean C-index of 0.859 (95% CI: 0.796–0.923), further corroborating the model's robust stability. Meanwhile, the validation set achieved an AUC of 0.742 (95% CI: 0.647–0.837), indicating acceptable discriminative capacity (Fig. 4B).

Calibration curves were plotted to evaluate the agreement between the predicted probabilities and observed outcomes. Both the training and validation set calibration curves closely aligned with the ideal diagonal line (Fig. 4C,4D). The Hosmer–Lemeshow goodness-of-fit test yielded non-significant P values for both sets (train: $\chi^2 = 12.205$, $P = 0.142$; test: $\chi^2 = 5.639$, $P = 0.688$), suggesting excellent calibration without significant deviation. Furthermore, the mean absolute errors (MAE) for the training and validation sets were 0.015 and 0.051, respectively, both falling within acceptable limits.

Further quantitative calibration assessment in the validation set revealed a calibration slope of 0.474, a calibration intercept of 0.021, and a Brier score of 0.213. While the calibration intercept and Brier score were within acceptable ranges, the slope deviated from the ideal value of 1.0, suggesting a certain degree of model overfitting. Additionally, using the Youden index to determine the optimal probability threshold in the training set yielded a cutoff value of 0.596. When applied to the validation set, this threshold produced a sensitivity of 0.736, a specificity of 0.538, a positive predictive value (PPV) of 0.842, and a negative predictive value (NPV) of 0.378.

3.5 Decision Curve Analysis (DCA)

To assess the clinical net benefit of the predictive model, decision curve analysis (DCA) was performed on both the training and validation sets (Fig. 4E, 4F). The results demonstrated that within a broad range of decision threshold probabilities (7% to 96%), the model effectively provided net benefit for predicting the risk of poor prognosis. This stable net-benefit performance confirms that the nomogram not only exhibits good calibration and discrimination but also holds practical value across a wide spectrum of clinical decision-making preferences.

4. Discussion

Due to the severe complications and poor prognosis associated with ICH, it places a significant burden on patients' families and society[12]. Its pathogenesis involves a variety of mechanisms, including mass effect from the hematoma, inflammatory cascades, neuronal ferroptosis, impaired collateral circulation, and abnormal increases in intracranial pressure[13]. This study is based on a retrospective analysis of

clinical data from 376 patients with ICH. It aims to identify independent risk factors affecting the 30-day prognosis of patients with intracerebral hemorrhage and to construct a nomogram prediction model. The model exhibited good discriminative ability in the training set (AUC = 0.885) and acceptable discrimination in the internal validation set (AUC = 0.742). Calibration curves and the Hosmer-Lemeshow test indicated strong agreement between predicted and observed probabilities; decision curve analysis further confirmed that the nomogram provides a positive net clinical benefit across a broad range of threshold probabilities, thereby supporting its potential clinical utility for early risk stratification.

Among the final predictors, admission GCS score and hematoma volume are well-established prognostic factors for ICH, directly reflecting the severity of initial neurological injury and mass effect[14, 15]. Intraventricular hemorrhage and early hematoma expansion have also been consistently shown in prior studies to be closely associated with secondary brain injury and clinical neurological deterioration[13, 16, 17]. Consistent with previous studies, our analysis identified a history of hypertension as an independent risk factor for poor prognosis[18]. The underlying mechanisms may involve chronic vascular damage, endothelial dysfunction, and impaired cerebral autoregulation, which render hypertensive patients more susceptible to hematoma expansion and perihematomal edema[19]. Additionally, long-standing hypertension may promote arterial wall fragility and microaneurysm formation, thereby increasing the severity of initial hemorrhage[20]. The presence of hypertension in our cohort also closely interacted with other metabolic and coagulation factors, further compounding the adverse outcomes[21, 22]. Furthermore, prolonged APTT suggests a propensity for coagulopathy, which may exacerbate hematoma progression by interfering with hemostatic balance[23–25]. The TyG index, as a reliable surrogate marker of insulin resistance, has recently been reported to be significantly associated with poor outcomes in cerebrovascular diseases[26–29]. Collectively, these variables comprehensively encompass multiple pathophysiological dimensions, including neurological status, structural lesion characteristics, metabolic homeostasis, and coagulation function[30–32], which likely accounts for the comprehensive performance of our predictive model.

Despite the promising predictive performance demonstrated in this study, several limitations must be thoroughly considered before clinical application. First, this was a single-center, retrospective study, inevitably subject to selection bias, and the treatment protocols and patient demographics at a single center may not fully represent the realities of other regions or healthcare tiers[33–35]. Furthermore, our study spanned an 11-year period, during which the management of spontaneous intracerebral hemorrhage underwent considerable changes. Blood pressure management protocols, anticoagulation reversal strategies, minimally invasive surgical techniques, and the standardization of neurocritical care all evolved over time, leading to variations in both patient characteristics and clinical outcomes[36]. Our manuscript did not systematically examine this temporal heterogeneity[37]; we acknowledge that such changes may affect the model's stability across different eras. Second, and most critically, the model lacks external validation. Although internal validation via random splitting and bootstrap resampling ensured a certain degree of model stability, the validation set was derived from the same source population[38, 39]. Therefore, this still results in poor generalizability of the model for external validation[40, 41]. The relatively low AUC (0.742) in the internal validation set, coupled with a calibration

slope (0.474) that deviates significantly from the ideal value of 1.0 strongly suggests a degree of overfitting, despite our adoption of relatively stringent statistical strategies during variable selection. Third, while our sample size was within an acceptable range, it remains relatively limited, which may lead to overly optimistic estimates of regression coefficients[42, 43]. Fourth, this study lacks several covariates with potential predictive value. First, clinical data such as dynamic blood pressure trends, timing and surgical procedures of intervention, and whether life-sustaining treatment was withdrawn are unavailable[44–46]. Moreover, refined imaging indicators including the spot sign, hematoma morphological irregularity, and perihematoma edema characteristics were not included[47, 48]. The absence of these data stems from the fact that this retrospective study did not dynamically collect or systematically archive the relevant information, which also limits the potential for further improvement in the predictive performance of our model. Fifth, this study used 30-day functional outcomes as the endpoint, whereas current ICH clinical trials and prognostic research increasingly favor 90-day follow-up to more comprehensively capture patients' neurological recovery trajectories and long-term dependency status[49, 50]. During our follow-up, we observed that post-discharge rehabilitation training substantially improved patients' functional performance[51–53]. Sixth, the proportion of poor outcomes in this cohort was as high as 66.2%, significantly exceeding the incidence of ICH reported in community-based populations, likely reflecting inherent bias toward more severely ill patients admitted to our center. Therefore, calibration may require appropriate adjustment when applying this model to lower-risk populations. As a result, before applying this model, it is still necessary to compare it with currently commonly used scoring models. The fact that this study did not make such a comparison is also a limitation.

Furthermore, the absence of external validation constitutes a major limitation of the present study. In future investigations, we will prioritize performing external validation based on a multicenter prospective cohort. Meanwhile, we aim to incorporate more comprehensive radiomic features and dynamic therapeutic parameters, and extend the follow-up duration to evaluate patients' long-term functional recovery and all-cause mortality[54, 55]. In addition, we intend to integrate machine learning algorithms or radiomic biomarkers to further break the predictive ceiling of conventional regression models, delivering a more accurate and individualized decision-support tool for clinicians.

Conclusion

In this study, we developed and internally validated a novel nomogram incorporating seven readily available clinical and laboratory parameters, admission GCS score, EHE, HTN, IVH, initial hematoma volume (V1), APTT, and TyG, to predict 30-day poor functional outcome ($mRS \geq 3$) in patients with spontaneous intracerebral hemorrhage. The model demonstrated good discriminative ability in the training set ($AUC = 0.885$) and acceptable performance in the validation set ($AUC = 0.742$), with a bootstrap-corrected C-index of 0.859 supporting its internal stability. Calibration was satisfactory in both cohorts, as indicated by non-significant Hosmer–Lemeshow test results and low mean absolute errors. Decision curve analysis further confirmed that the nomogram provides positive net clinical benefit

across a wide range of threshold probabilities (7%–96%), which underscores its potential value in supporting individualized risk stratification and clinical decision making.

Nevertheless, several limitations should be acknowledged. The calibration slope in the validation set (0.474) deviated from the ideal value, suggesting a certain degree of model overfitting, which warrants cautious interpretation. The moderate validation AUC (0.742) also indicates that further optimization and external validation in larger, multicenter cohorts are needed before widespread clinical adoption. In addition, the retrospective design of this study and the lack of head-to-head comparison with established ICH prognostic scores limit the generalizability of our findings. Future work should focus on prospective data collection, temporal or geographic external validation, and integration of this nomogram with existing tools to better define its incremental value in routine practice.

Declarations

Ethics approval

The study was conducted in accordance with the guidelines of the Declaration of Helsinki, and approved by the institutional review board of Taian Central Hospital (approval number, 2026-01-K515). The institutional review board waived the requirement for informed consent because of the retrospective nature of the study and all data were anonymized to protect patient confidentiality.

Consent for publication

Not applicable. No identifiable individual patient data are included in this manuscript.

Availability of data and materials

The original contributions presented in the study are included in the article and supplementary material, and further inquiries can be directed to the corresponding author(s).

Competing interests

The authors declare no potential conflicts of interest.

Finding

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CRedit authorship contribution statement

Software, Writing - original draft preparation and Writing - review and editing: Xiangning He, Yulun Wang, and Qian Chen; Data collection and Investigation: Haochen Yang, Zhenyu Geng, Jiahao Liu, Xunlu Yin, Yawei Rong, Shizhong Zhang; Writing - review and editing, Supervision and Project administration: Fangfang Zhao and Guojun Wang. All authors revised and approved the final manuscript.

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Figures

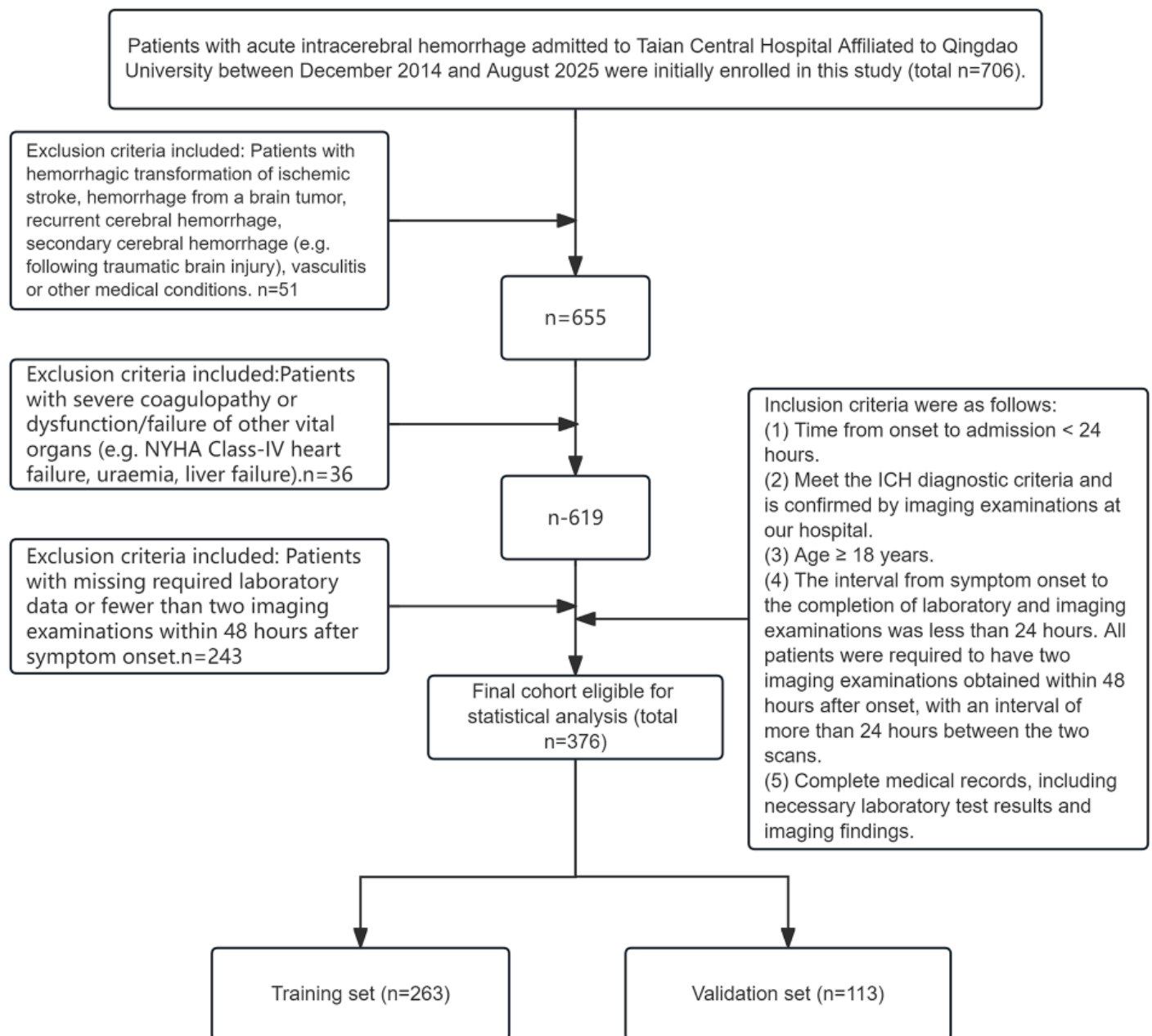


Figure 1

Patient Enrolment Flowchart. This flowchart illustrates the screening process for patients with acute intracerebral hemorrhage admitted to Qingdao University Affiliated Taian Central Hospital between December 2014 and August 2025. Following screening according to the exclusion criteria, a total of 376 patients with ICH were ultimately included; based on the predefined criteria, 263 patients were assigned to the training set and 113 to the validation set.

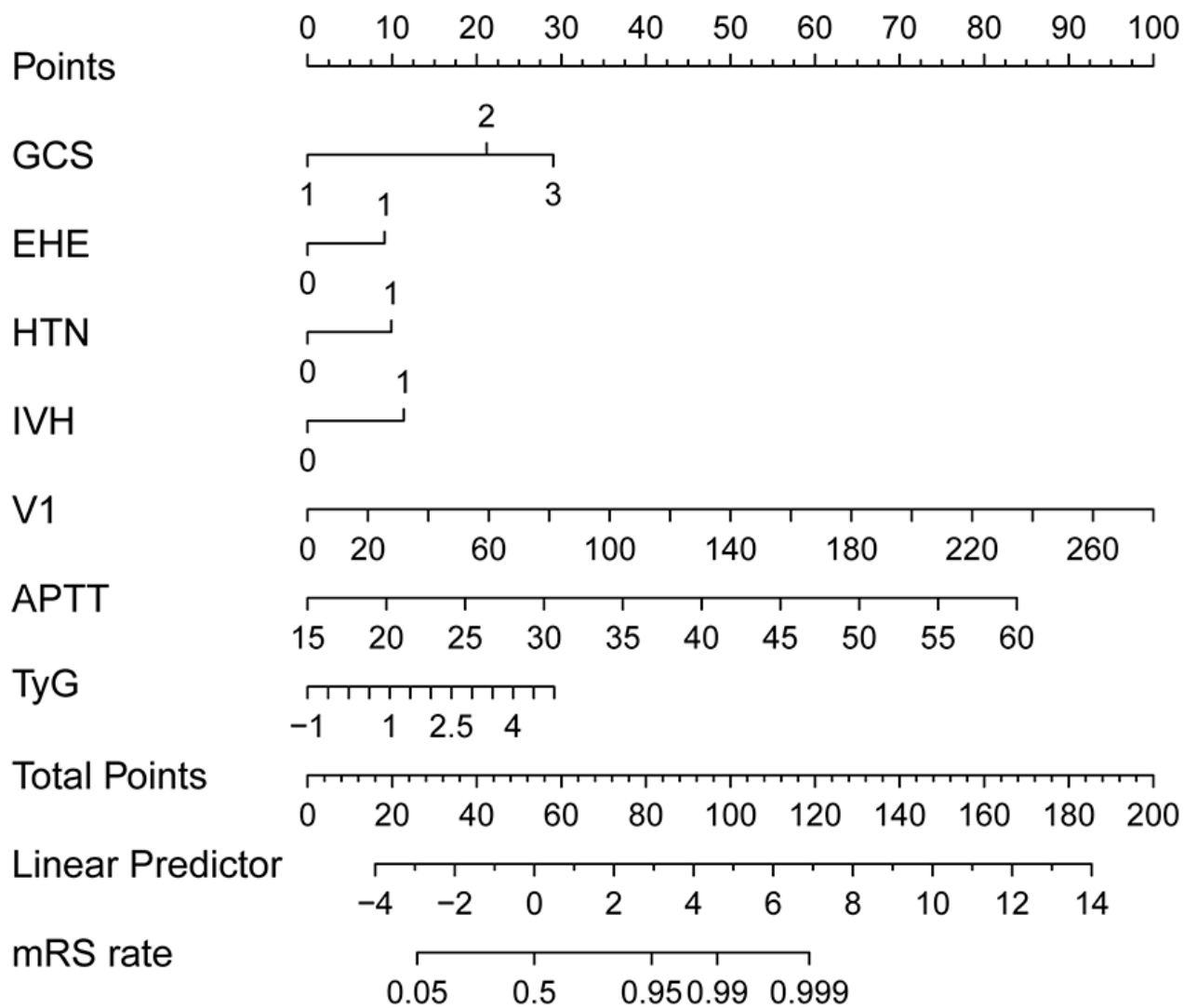


Figure 3

Risk nomogram model for prognosis in ICH patients.

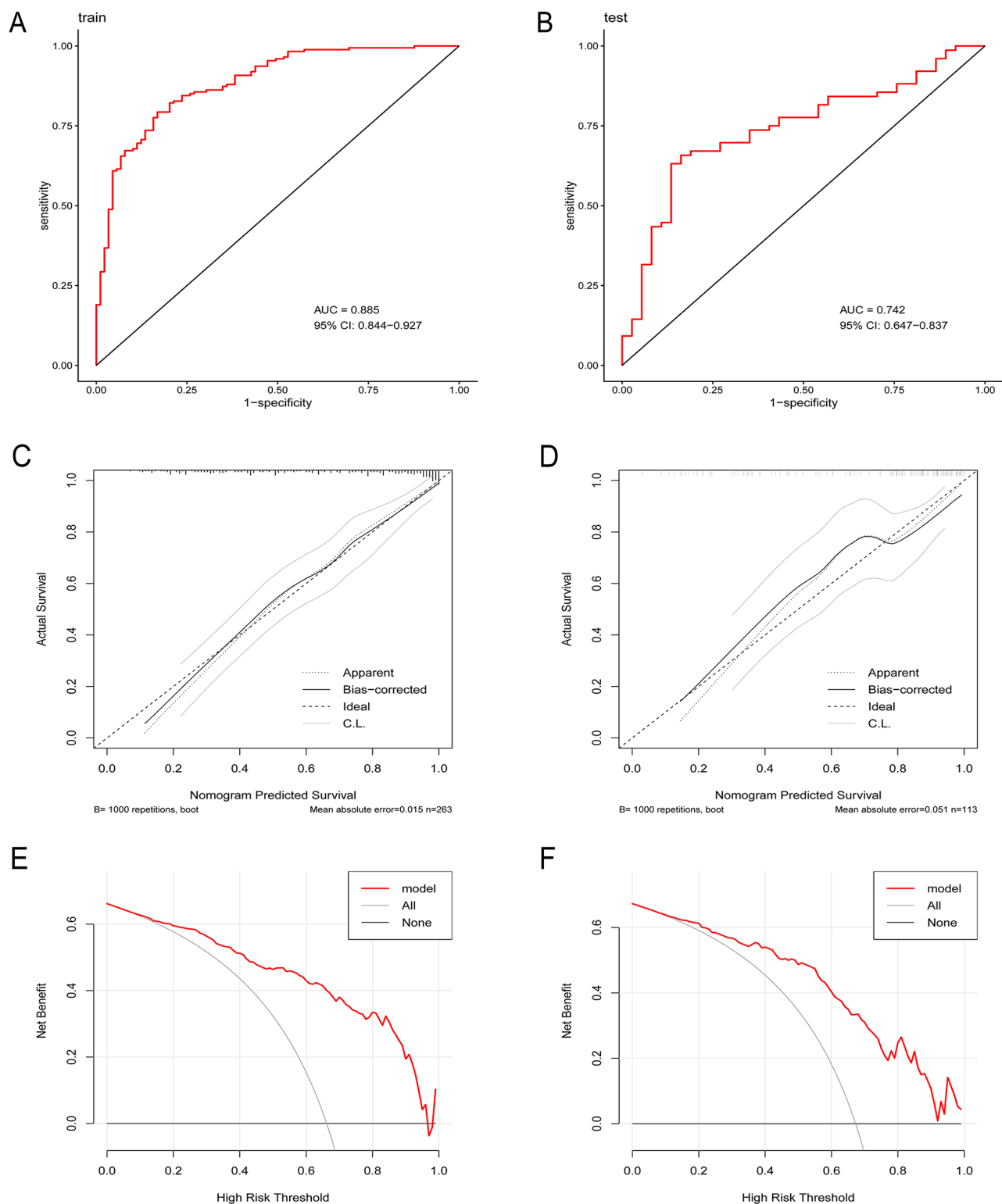


Figure 4

(A, B) ROC curves in the training and validation cohorts, respectively; (C, D) Calibration curves in the training and validation cohorts, respectively. The dashed ideal line indicates perfect prediction. The dotted apparent line indicates the original calibration performance based on the observed dataset, whereas the solid bias-corrected line corrected by 1000 bootstrap replicates estimates the optimism-corrected calibration for external cohorts and reflects generalized calibration performance. Light gray

lines represent the 95% confidence limits of the corrected curve; (E, F) DCA curves in the training and validation cohorts, respectively.

Supplementary Files

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