

Clinical-Imaging Nomogram for Estimating Hematoma Expansion After Spontaneous Intracerebral Hemorrhage

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Research Article

Keywords: Spontaneous intracerebral hemorrhage, Hematoma expansion, Risk model, Nomogram, Computed tomography

Posted Date: October 14th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11180043/v1>

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Additional Declarations: No competing interests reported.

Abstract

Early enlargement of an intracerebral hematoma can worsen neurological status, yet it is difficult to judge at first assessment which patients will enlarge. We examined whether routine admission findings could be combined into a practical prediction tool. The records of 279 adults with spontaneous intracerebral hemorrhage (SICH) treated from January 2019 through October 2025 were reviewed; all had baseline and follow-up computed tomography. Expansion was defined as hematoma growth > 6 mL or at least 33%. Variables showing univariable associations were considered in stepwise logistic regression, and the retained factors were incorporated into a nomogram. The expanded definition was met by 89 patients (31.9%). The Glasgow Coma Scale score, admission systolic blood pressure, initial hematoma volume, heterogeneous hematoma density, the satellite sign, and ionized calcium were all included in the final model. With a 95% confidence interval of 0.763–0.885, its area under the receiver operating characteristic curve was 0.821. Potential net benefit was examined using decision-curve analysis. For early risk assessment, the nomogram provides a condensed method of combining standard clinical and CT data. Confirmation of its calibration and performance in separate cohorts is still needed.

1 Introduction

According to population estimates used in earlier research, the incidence of spontaneous intracerebral hemorrhage (SICH) is approximately 20–30 incidents per 100,000 people, and SICH is thought to be responsible for 30% of strokes [1, 2]. 30-day mortality frequently exceeds 40%, indicating that early mortality is still significant [3]. Additionally, the initial hematoma is not static; detectable growth occurs in about one-third of individuals [4]. In INTERACT1, every additional 1 mL of hematoma growth was associated with about a 5% increase in the adjusted odds of death or dependency [5]. Identifying patients whose hematoma is likely to enlarge is therefore relevant to surveillance and treatment planning.

Several indicators are available at the time of presentation. Blood pressure and neurological status have repeatedly been linked to later hematoma growth, while non-contrast CT may show features such as heterogeneous density that suggest an unstable lesion [6–9]. A multivariable approach can bring these bedside and imaging findings together rather than relying on a single marker.

Accordingly, we reviewed a single-center SICH cohort and tested routinely collected clinical, laboratory, and CT variables. Factors retained after multivariable analysis were assembled into a nomogram intended to estimate the probability of hematoma expansion.

2 Methods

2.1 Study design and participants

Medical records were reviewed for adults with SIH managed in the Department of Emergency Medicine, Beijing Jingmei Group General Hospital, from January 2019 to October 2025. This was a single-center retrospective observational study. Eligible patients were aged 18 years or older, fulfilled the 2022 American Heart Association/American Stroke Association diagnostic criteria for SIH, had both an initial and a follow-up CT after admission, and had the clinical and imaging information required for analysis. We excluded hemorrhage secondary to trauma, vascular malformation, aneurysmal subarachnoid hemorrhage, hemorrhagic transformation of ischemic stroke, or tumor; severe systemic disease expected to interfere materially with assessment or follow-up; records with missing essential data; and pregnancy or breastfeeding. The Ethics Committee of Beijing Jingmei Group General Hospital approved the study, which was conducted in accordance with the Declaration of Helsinki and subsequent amendments. Written informed consent was obtained from participants or their legally authorized representatives.

2.2 Data collection and assessment of hematoma expansion

We abstracted demographic characteristics, relevant medical and exposure histories, admission findings, and laboratory tests from the electronic medical record. Recorded variables included age, sex, diabetes, previous stroke, coronary heart disease, smoking, alcohol use, admission Glasgow Coma Scale (GCS) score and blood pressure, platelet count, hemoglobin, blood glucose, neutrophil-to-lymphocyte ratio, lactate dehydrogenase, and ionized calcium. CT review provided baseline hematoma volume, hematoma location, intraventricular hemorrhage, density heterogeneity, and the satellite sign (Fig. 1). Smoking history was recorded when a patient had smoked at least one cigarette per day for 1 year or longer at any time; lesser or no exposure was classified as non-smoking. Regular alcohol consumption for at least 1 year was recorded as a positive alcohol history. Previous stroke included ischemic and hemorrhagic events. On CT, the satellite sign referred to one or more small hemorrhagic foci adjacent to the main clot, whereas density heterogeneity denoted visibly non-uniform attenuation within the hematoma.

Expansion was determined by comparing the initial and follow-up CT examinations. A case was classified as expanded when hematoma volume increased by more than 6 mL or by at least 33%. Volumetry was performed in 3D Slicer. After image import, an initial segmentation was generated with thresholding or region-growing tools and then corrected manually before volume calculation. Three trained physicians independently reviewed hematoma volume, expansion status, density heterogeneity, and the satellite sign.

2.3 Statistical analysis

Analyses were conducted in IBM SPSS Statistics 22.0 and R 4.1.0. Continuous data were summarized as mean (SD) when approximately normally distributed and as median (IQR) otherwise. The expansion and non-expansion groups were compared with an independent-samples t test or Mann-Whitney U test for continuous variables and with Pearson chi-square testing for categorical variables; Fisher's exact test

was used when expected cell counts were small. Variables with $P < 0.05$ in the univariable comparisons were considered for stepwise logistic regression. Effect estimates from the fitted model are reported as odds ratios (ORs) with 95% confidence intervals (CIs). All tests were two-sided, with $P < 0.05$ taken as statistically significant.

Predictors retained by logistic regression were translated into a point-based nomogram with the rms package in R. Discrimination was quantified by receiver operating characteristic (ROC) analysis using pROC. The rmda package was used for decision-curve analysis (DCA), comparing the model's net benefit with treat-all and treat-none reference strategies over threshold probabilities. Because calibration could not be verified from the available analysis output, no calibration statistic or plot is presented as a validated performance result.

3 Results

3.1 Patient characteristics and univariate analysis

Of 279 eligible patients, 89 (31.9%) met the definition of hematoma expansion and 190 (68.1%) did not. Mean age differed little between groups (65.2 vs 64.3 years). Men comprised 56 of 89 patients (62.9%) in the expansion group and 116 of 190 (61.1%) in the non-expansion group.

Table 1 summarizes the univariable comparisons. Nine variables met the prespecified $P < 0.05$ screening criterion: smoking history, admission systolic blood pressure (SBP), antiplatelet use, admission GCS score, baseline hematoma volume, hematoma density heterogeneity, the satellite sign, platelet count, and ionized calcium. Their respective P values were 0.046, 0.036, 0.044, 0.013, 0.014, 0.016, < 0.001 , 0.042, and 0.024. No other measured variable crossed this threshold.

Table 1
Baseline variables according to hematoma-expansion status

Variable	Hematoma expansion (n = 89)	No expansion (n = 190)	P value
Age, years, mean (SD)	65.2 (11.8)	64.3 (12.2)	0.556
Male sex, n (%)	56 (62.9)	116 (61.1)	0.765
BMI, kg/m ² , mean (SD)	25.2 (4.2)	24.1 (4.8)	0.052
Diabetes history, n (%)	17 (19.1)	38 (20.0)	0.860
Previous stroke, n (%)	13 (14.6)	23 (12.1)	0.561
Coronary heart disease, n (%)	10 (11.2)	26 (13.7)	0.570
Smoking history, n (%)	37 (41.6)	56 (29.5)	0.046
Alcohol-use history, n (%)	16 (18.0)	32 (16.8)	0.815
SBP at admission, mmHg, mean (SD)	185.6 (26.5)	178.6 (24.3)	0.036
DBP at admission, mmHg, mean (SD)	100.5 (14.3)	98.5 (16.7)	0.303
Antiplatelet use, n (%)	19 (21.3)	23 (12.1)	0.044
Anticoagulant use, n (%)	7 (7.9)	14 (7.4)	0.883
GCS score at admission, median (IQR)	12 (9–14)	9 (6–12)	0.013
Initial hematoma volume, mL, mean (SD)	25.3 (18.2)	19.8 (15.1)	0.014
Heterogeneous hematoma density, n (%)	22 (24.7)	25 (13.2)	0.016
Intraventricular hemorrhage, n (%)	14 (15.7)	32 (16.8)	0.816
Supratentorial location, n (%)	72 (80.9)	151 (79.5)	0.782
Satellite sign, n (%)	20 (22.5)	13 (6.8)	< 0.001
Blood glucose at admission, mmol/L, mean (SD)	10.1 (3.6)	9.5 (3.4)	0.187
NLR, mean (SD)	4.6 (3.7)	4.3 (3.5)	0.522
Platelet count, ×10 ⁹ /L, mean (SD)	160.5 (59.2)	176.2 (61.8)	0.042
Hemoglobin, g/dL, mean (SD)	12.2 (3.1)	12.9 (3.5)	0.091
Ionized calcium, mmol/L, median (IQR)	1.11 (1.05–1.19)	1.19 (1.12–1.24)	0.024
LDH, U/L, mean (SD)	257.6 (109.3)	263.4 (105.7)	0.675

Data are shown as mean (SD), median (IQR), or n (%). BMI, body mass index; DBP, diastolic blood pressure; GCS, Glasgow Coma Scale; IVH, intraventricular hemorrhage; LDH, lactate dehydrogenase; NLR, neutrophil-to-lymphocyte ratio; SBP, systolic blood pressure.

3.2 Multivariable analysis

All nine screened variables entered the stepwise logistic model. Six remained in the final equation: admission SBP (OR 1.85, 95% CI 1.32–2.47), admission GCS score (OR 2.51, 95% CI 1.83–3.34), initial hematoma volume (OR 2.48, 95% CI 1.79–3.55), hematoma density heterogeneity (OR 2.33, 95% CI 1.62–3.07), the satellite sign (OR 6.02, 95% CI 4.53–7.62), and ionized calcium (OR 0.42, 95% CI 0.31–0.62) (Fig. 2). Smoking history, antiplatelet use, and platelet count were removed during model selection.

3.3 Development and assessment of the nomogram

These six retained variables were used to build the point-based nomogram shown in Fig. 3.

The nomogram showed an area under the ROC curve of 0.821 (95% CI 0.763–0.885) in the study cohort (Fig. 4).

Decision-curve analysis compared the fitted model with treat-all and treat-none policies (Fig. 5). The available aggregate output did not permit verification of an exact threshold interval in which the model was superior; accordingly, no numerical threshold range is reported.

4 Discussion

Using data that is typically accessible shortly after presentation, the research produced a six-variable model. Three predictors were obtained from the admission CT scan (hematoma volume, density heterogeneity, and the satellite sign), two from the bedside assessment (SBP and GCS), and one from laboratory tests (ionized calcium). In this group, a good distinction between individuals with and without subsequent expansion is indicated by an AUC of 0.821. The result is promising, but as calibration and external validation have not yet been established, it should be considered evidence of model development rather than proof of therapeutic efficacy.

4.1 Interpretation of the retained predictors

The final model retained admission SBP (OR 1.85, 95% CI 1.32–2.47). This is in line with studies on vascular damage and cerebral blood flow modulation in hypertension [11–13], as well as reports that link increased pressure to active bleeding or subsequent hematoma expansion [10]. Although SBP is a desirable predictor due to its instantaneous availability, these observational data do not demonstrate that expansion was caused by blood pressure. In line with previous findings linking admission awareness level to expansion risk, GCS was also included in the model (OR 2.51, 95% CI 1.83–3.34) [14–16]. The expansion group's median GCS in this cohort was 12 (IQR 9–14), while the non-expansion

group's was 9 (IQR 6–12). The unit-specific interpretation of this OR should be used with caution since the original model output did not record the scaling or coding applied to the continuous GCS term.

The model benefited greatly from CT findings. The resulting equation retained the initial hematoma volume (OR 2.48, 95% CI 1.79–3.55) and heterogeneous density (OR 2.33, 95% CI 1.62–3.07), which is consistent with research linking larger or structurally heterogeneous hematomas to subsequent expansion [18–24]. The largest point estimate (OR 6.02, 95% CI 4.53–7.62) was found for the satellite sign. Although the pooled diagnostic performance shown by Yang et al. was moderate (sensitivity 0.50 and specificity 0.71) [28], prior findings and meta-analyses also support a relationship between this sign and enlargement [25–29]. The satellite sign is best understood as a component of a combined assessment than as a stand-alone rule, which is a clinically significant limitation.

In the fitted model, ionized calcium provided information in the opposite direction (OR 0.42, 95% CI 0.31–0.62), with lower quantities linked to higher expansion risk. In hypertensive intracerebral hemorrhage, similar correlations have been shown [30, 31]. The molecular background provided by calcium signaling experiments [32] does not immediately explain the clinical connection seen here. Therefore, rather than being proof of a causal pathway, we view ionized calcium in this dataset as a predictive correlation.

4.2 Potential clinical application of the nomogram

The nomogram's primary practical benefit is that it integrates data from the first examination, standard blood tests, and non-contrast CT without the need for a specific imaging biomarker. The decision curve indicated net benefit over the reference policies across a portion of the threshold range depicted in Fig. 5, and the AUC was 0.821 (95% CI 0.763–0.885). These are only internal estimations. The calibration and performance of the score should be evaluated in a separate cohort before it is used to direct monitoring intensity, repeat imaging, or other management.

4.3 Study limitations

The strength of inference is limited by a few points. The results may have been impacted by case selection and local practice because the cohort was constructed retrospectively and originated from a single center. There were only 89 expansion events available, and when the number of events is small, stepwise selection can yield optimistic coefficients. Over the course of the trial, there were some variations in CT acquisition and picture interpretation. Furthermore, no external cohort was employed, and calibration could not be confirmed using the analysis output supplied for this revision. Lastly, before the nomogram is used elsewhere, each continuous predictor's coding and scale should be specified in the original model record. These problems would be resolved by a prospective multicenter research using standardized imaging and predetermined validation.

5 Conclusion

Patients with and without hematoma expansion were fairly successfully differentiated in this group using a six-variable nomogram that combined admission SBP and GCS with initial hematoma volume, CT

density heterogeneity, the satellite sign, and ionized calcium. Rather than being a ready-to-use clinical rule, the model is a candidate risk-estimation tool; independent validation and verified calibration are the next steps.

Declarations

Author Contribution

J.W. conceived and designed the study, collected and curated the clinical data, performed the statistical analysis, and drafted the manuscript. S.Z. contributed to data collection, imaging assessment, data verification, and preparation of the figures and tables. B.Y. supervised the study, contributed to the study design and interpretation of the results, and critically revised the manuscript for important intellectual content. All authors reviewed and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Data Availability

The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request. The data are not publicly available because they contain information that could compromise patient privacy and confidentiality.

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Figures

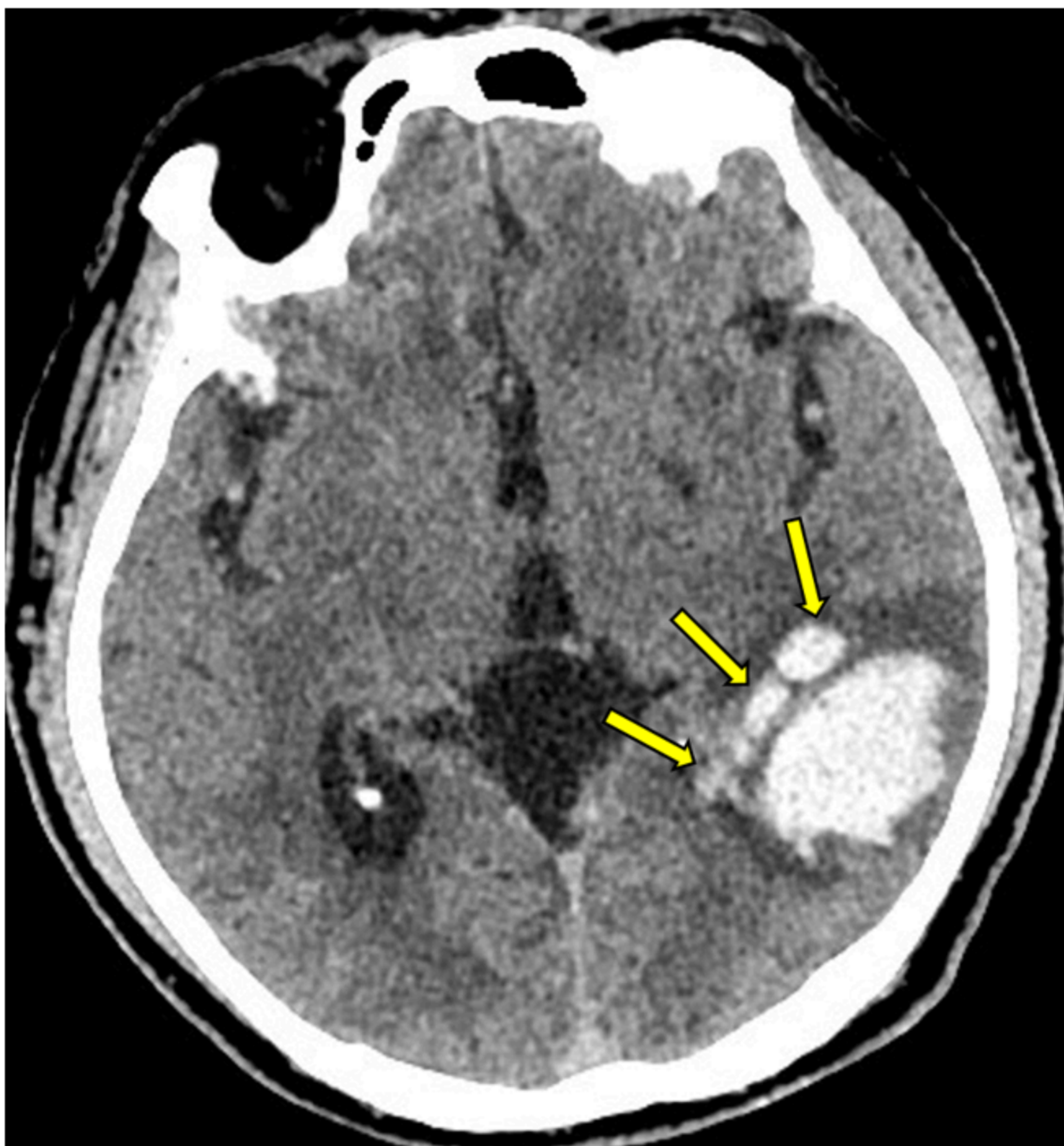


Figure 1

Representative non-contrast CT showing a satellite sign; arrows mark small hemorrhagic foci immediately adjacent to the principal hematoma.

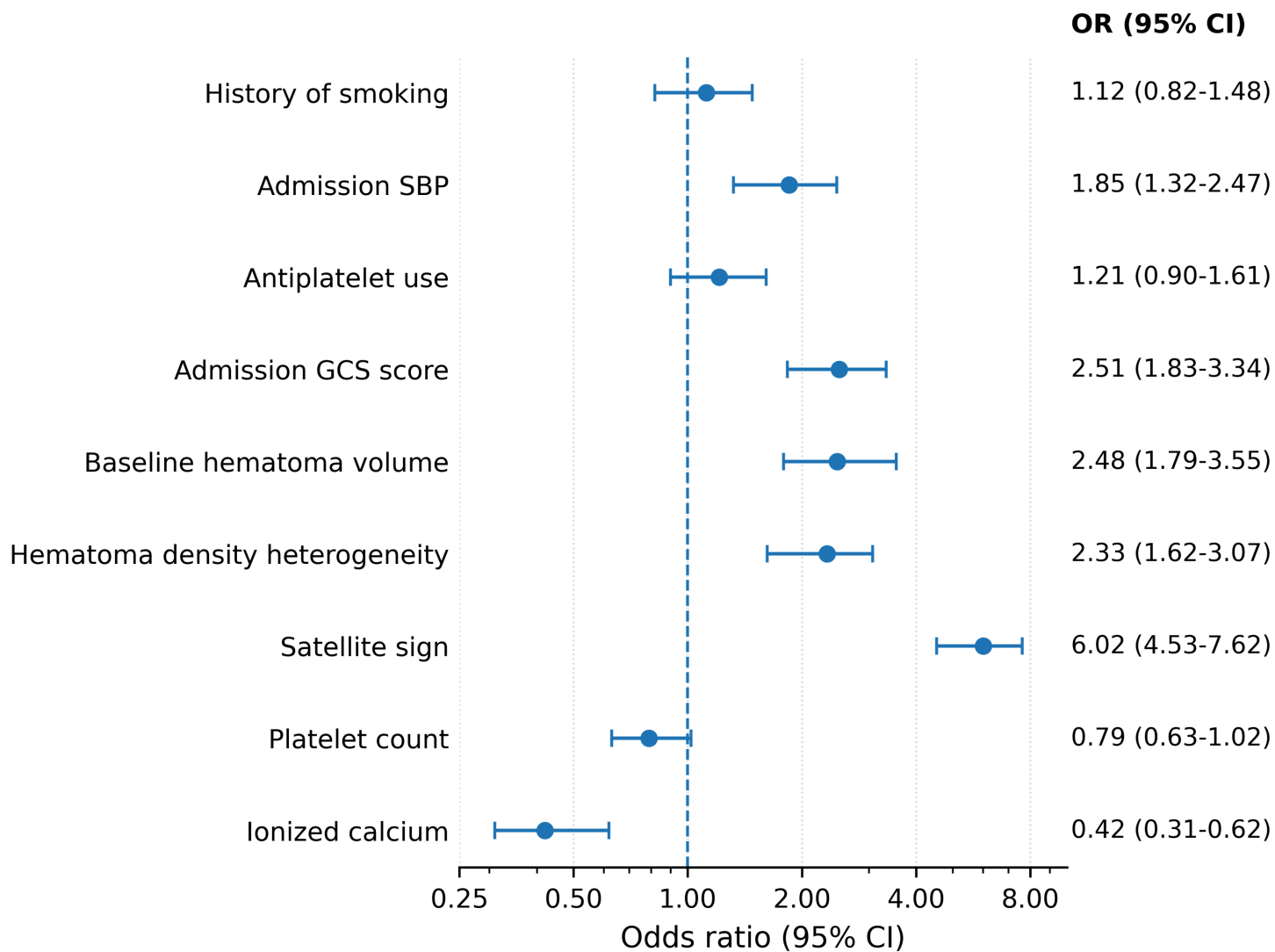


Figure 2

Effect estimates from the multivariable logistic model. Markers indicate ORs, horizontal bars show 95% CIs, and the dashed vertical reference is OR = 1. SBP, systolic blood pressure; GCS, Glasgow Coma Scale.

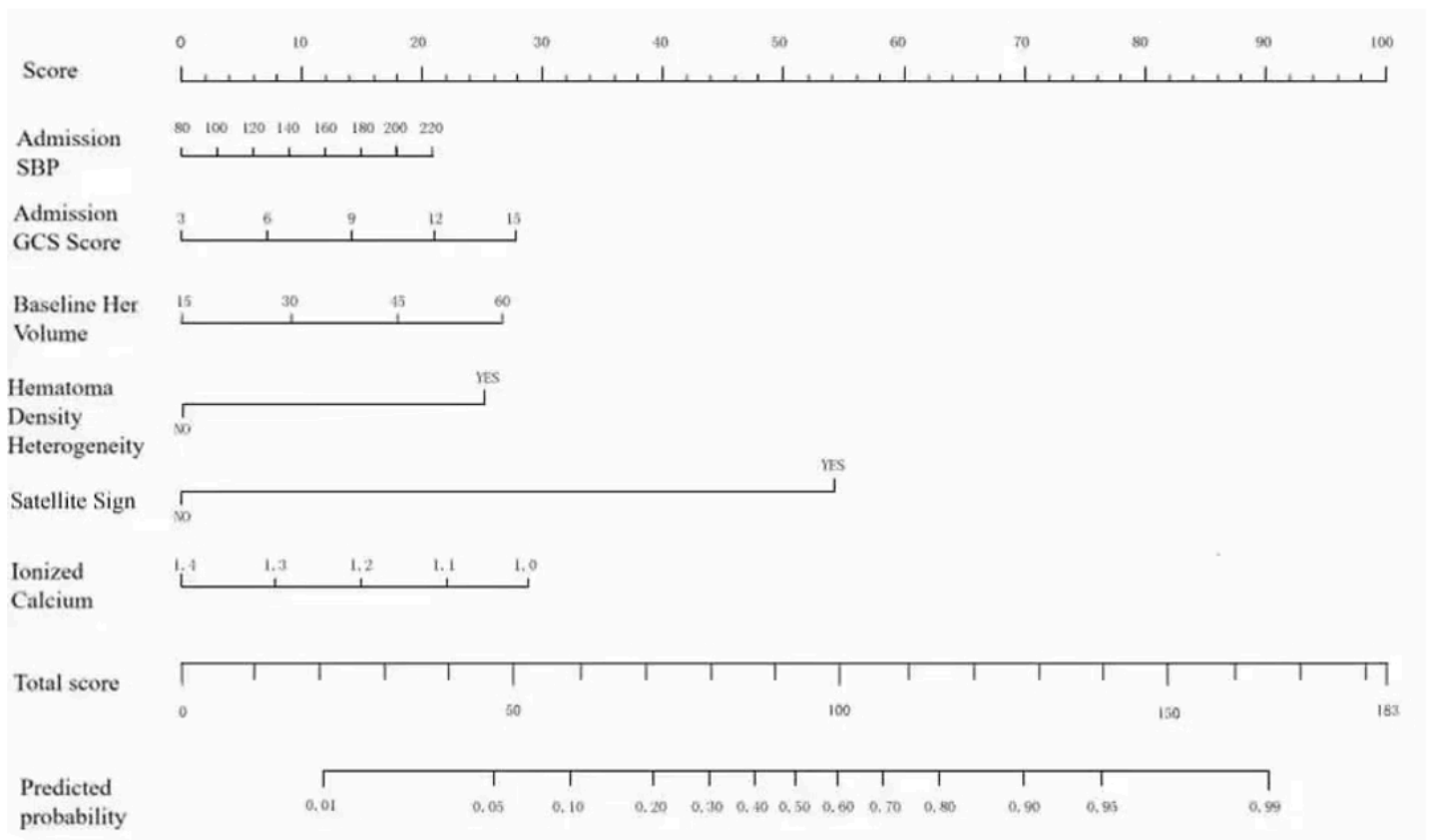


Figure 3

Point-based nomogram derived from the final six-variable model. Each predictor value is converted to points; the total corresponds to an estimated probability of hematoma expansion. SBP, systolic blood pressure; GCS, Glasgow Coma Scale.

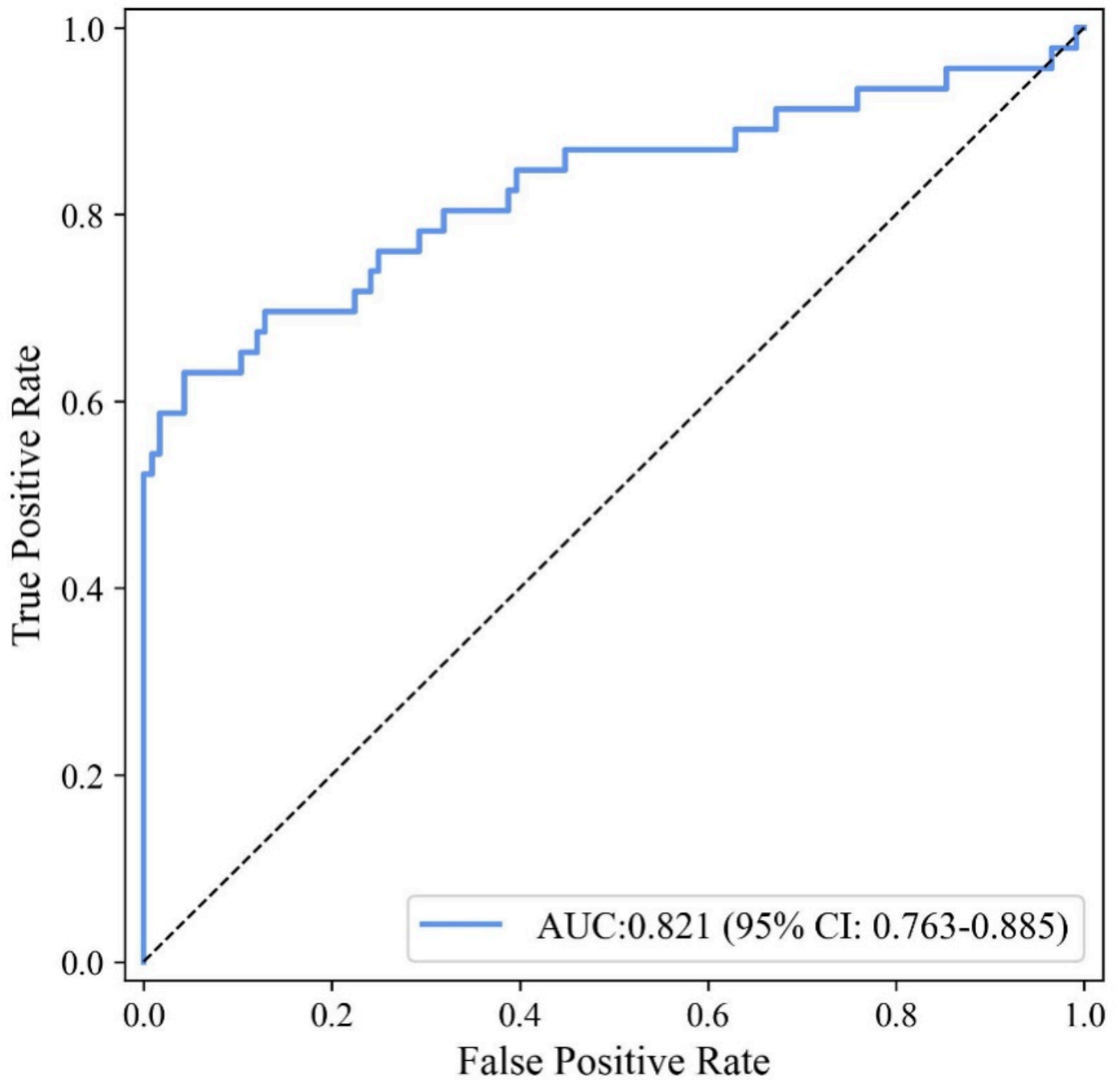


Figure 4

ROC curve for the nomogram (AUC 0.821, 95% CI 0.763-0.885). The diagonal line represents no discrimination.

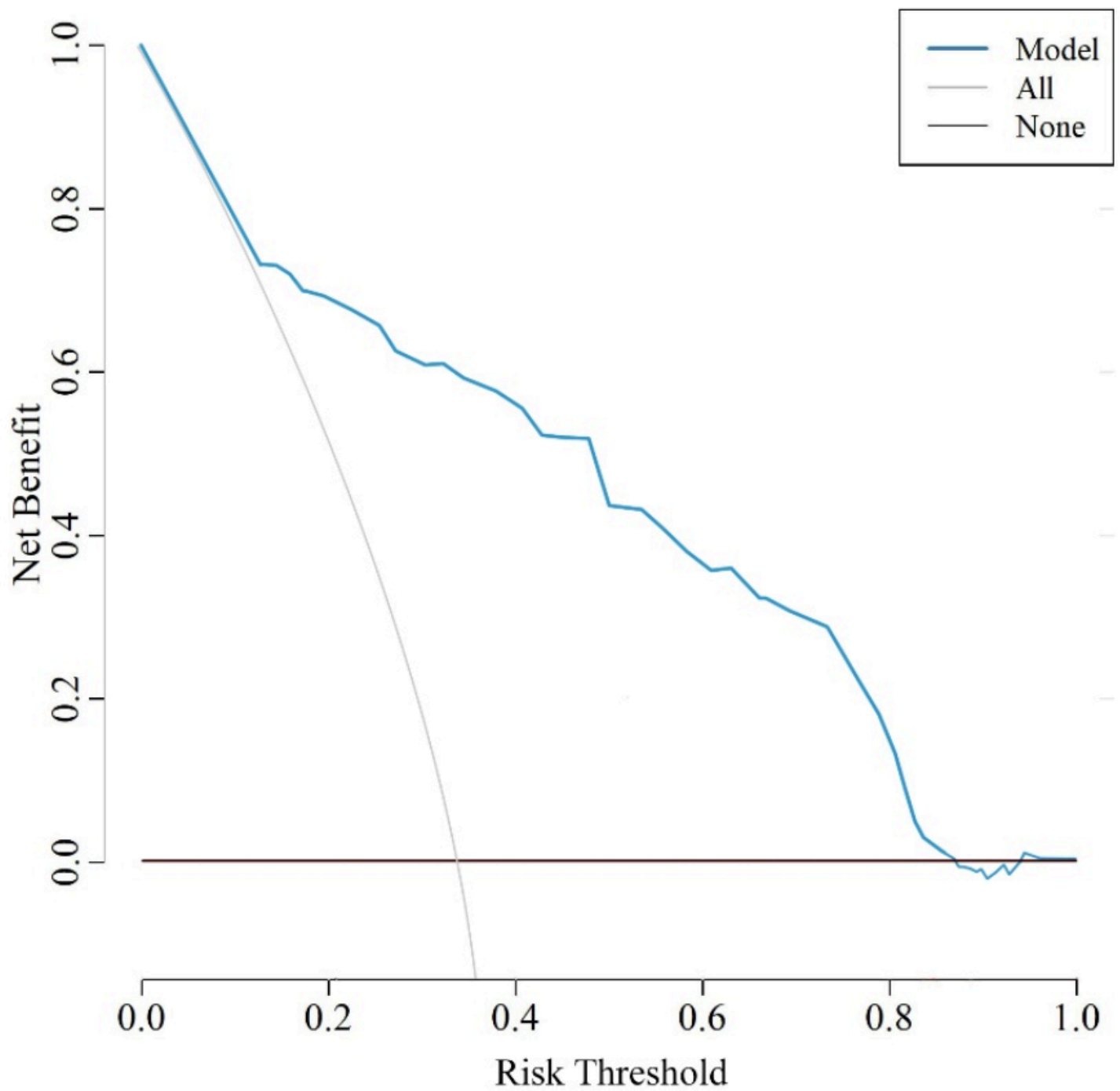


Figure 5

Decision-curve analysis showing net benefit for the nomogram and the treat-all and treat-none reference policies.