Predicting Discharge Outcomes from In-Hospital Characteristics after Cerebral Arterial Aneurysm Rupture: A Single Institutional Experience from Ukraine

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■ INTRODUCTION: **Most existing predictive models developed to assess outcomes after aneurysmal subarachnoid hemorrhage (aSAH) use a dichotomized approach. However, this strategy does not reflect the full clinical spectrum of patient status after aSAH and leads to underestimation of differences in rehabilitation needs and associated economic costs. Based on the Hospital Assessment Scale (HAS), we developed and validated an ordinal predictive model to predict in-hospital outcomes after aSAH.**

■ METHODS: **We conducted a single-center retrospective cohort study in Ukraine including 489 patients with aSAH. The outcome was categorized into four HAS scores. Candidate predictors included demographic and medical history variables, clinical and radiological characteristics, including cerebral arterial aneurysm (CAA) features, treatment strategy, and complications. Missing data were handled using multiple imputation. An ordinal logistic regression model with proportional odds assumption and penalized maximum likelihood estimation was applied. Internal validation was performed using bootstrap resampling (B = 200).**

■ RESULTS: **Independent predictors of worse HAS outcome included aneurysm re-rupture, larger aneurysm size, limb paresis at admission, conservative treatment, cerebral vasospasm, and hospital-acquired pneumonia. The final ordinal predictive model was constructed using 9 variables: sex, age groups, presence of CAA re-rupture and limb paresis at admission, size of the CAA, mWFNS grade at admission, treatment strategy, and the occurrence of cerebral vasospasm and hospital-acquired pneumonia. The model demonstrated good discrimination and calibration after internal bootstrap validation (C-index ≈ 0.82).**

■ CONCLUSIONS: **We developed and internally validated an ordinal predictive model to estimate HAS outcomes after aSAH. The proposed model integrates baseline clinical characteristics and dynamic changes occurring throughout hospitalization.**

INTRODUCTION

Aneurysmal subarachnoid hemorrhage (aSAH) is an acute life-threatening condition associated with a high risk of early mortality and long-term persistent disability among survivors.[1,2] Overall mortality after aSAH reaches 25%–30% within the first 24 hours after cerebral arterial aneurysm (CAA) rupture, increases to 40%–45% during the first week and rises further to 50%–60% within the first month.[3]

Advances in neuroimaging and surgical technologies have improved patient survival and reduced the severity of disease-related consequences. However, these advances have increased

Key words
- Aneurysmal subarachnoid hemorrhage
- Ordinal predictive model
- Outcome

Abbreviations and Acronyms
ACA: Anterior cerebral artery
aSAH: Aneurysmal subarachnoid hemorrhage
aSAH-HAScore: Aneurysmal subarachnoid hemorrhage hospital assessment score
BA: Basilar artery
CAA: Cerebral arterial aneurysm
CV: Cerebral vasospasm
GOS: Glasgow Outcome Scale
HAP: Hospital-acquired pneumonia
HAS: Hospital assessment scale
ICA: Internal carotid artery
IQR: Interquartile range
MCA: Middle cerebral artery
mRS: Modified rankin scale
mWFNS: Modified world federation of neurosurgical societies scale
VA: Vertebral artery

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resource utilization and imposed a substantial financial burden on healthcare systems.[4] In most published studies, including those focused on predictive model development, outcomes after aSAH are assessed using dichotomized categories such as "favorable" versus "unfavorable" or "good" versus "poor". This approach does not adequately reflect the full clinical spectrum of patient status after aSAH. In addition, commonly used outcome scales in existing predictive models such as the modified Rankin Scale (mRS) and Glasgow Outcome Scale (GOS) were developed to assess overall disability and functional recovery of patients during long-term follow-up (3 months or longer) rather than in-hospital outcomes at the time of discharge. Although these scales are widely used in clinical research, they do not directly reflect rehabilitation requirements at hospital discharge. Patients with similar mRS or GOS scores may have substantially different needs for post-discharge support and rehabilitation services. It leads to underestimation of differences in rehabilitation needs and fails to account for variability in rehabilitation-related economic costs according to the achieved treatment outcome. In contrast, the Hospital Assessment Scale (HAS) was specifically designed for in-hospital outcome assessment after aSAH and categorizes patients according to clinically relevant rehabilitation needs, including no rehabilitation, outpatient rehabilitation and inpatient rehabilitation. This structure may provide more practical information for discharge planning and rehabilitation resource allocation.[5-7]

Clinical predictive models aim to estimate the probability of achieving a specific clinical outcome within a defined time interval. They estimate individual patient risk to inform patients, their relatives and healthcare professionals about prognosis and to support informed decisions regarding patient management.[8] Despite numerous studies on outcome predictors after aSAH, no validated and widely accepted model is currently available for routine clinical prognosis of the in-hospital course.[6,7]

We developed and validated a clinical ordinal predictive model to predict outcomes according to the HAS after aSAH.[5] This model is referred to as the aneurysmal subarachnoid hemorrhage hospital assessment score (aSAH-HAScore).

MATERIALS AND METHODS

Study Design and Data Source

We conducted a single-center retrospective cohort study in Ukraine that included 489 medical records. Patients with acute subarachnoid hemorrhage caused by a ruptured CAA confirmed by CT angiography were eligible. Only individuals older than 18 years were included. Patients with traumatic subarachnoid hemorrhage or other causes of intracranial hemorrhage were excluded.

Selection of Predictive Variables

We analyzed a broad range of potential predictors reflecting baseline characteristics, clinical presentation and treatment strategy. Demographic and medical history variables included age, sex, arterial hypertension, and ischemic heart disease. Age was divided into 4 groups: 18–44 years, 45–60 years, 61–75 years, and ≥76 years. Time from CAA rupture to hospital admission was recorded and stratified into early admission (≤3 days), delayed admission (4–14 days), and late admission (≥15 days). Length of hospital stay was also included. Radiological and anatomical features included hemorrhage distribution, CAA location, type, and size. Hemorrhage distribution was classified as aSAH with or without a parenchymal component. CAA location was categorized according to vascular territory as anterior cerebral artery (ACA), internal carotid artery (ICA), middle cerebral artery (MCA), basilar artery (BA), and vertebral artery (VA). Morphological types of CAA included saccular, fusiform, multilobular, and bilobular aneurysms. The number of CAAs was recorded as single or multiple. In cases of multiple CAAs, only the ruptured CAA was considered for analysis of location, size, and type. The presence of aneurysm re-rupture was also recorded. Clinical characteristics at admission included severity according to the modified World Federation of Neurosurgical Societies scale (mWFNS), presence of seizures, headache, and limb paresis. Treatment strategy was classified as microsurgical or conservative. Microsurgical treatment included clipping or trapping of the CAA. In surgical patients, mWFNS was assessed at admission and on the day of surgery. Change in clinical status was calculated as ΔmWFNS = mWFNS at admission minus mWFNS on the day of surgery. A negative ΔmWFNS indicated clinical deterioration before surgery, a zero value indicated a stable condition and a positive value indicated clinical improvement. For statistical analysis, patients were categorized into 4 groups according to treatment strategy: surgical patients with clinical deterioration, surgical patients with stable condition, surgical patients with clinical improvement, and conservatively treated patients. Complications included cerebral vasospasm (CV), hospital-acquired pneumonia (HAP), and conditions requiring tracheostomy.

Outcome Measure

Treatment outcome was assessed using the HAS. HAS includes 4 categories: no neurological deficit, neurological deficit not requiring assistance, neurological deficit requiring assistance, and death. This approach allows evaluation of clinical outcome and stratification according to rehabilitation needs. Patients with HAS 0 do not require rehabilitation, patients with HAS 1 require outpatient rehabilitation and patients with HAS 2 require inpatient rehabilitation.[5]

Development of the Predictive Model

Descriptive statistics were performed for all potential predictors at the first stage of the study. Descriptive analysis was conducted without data imputation to preserve the original structure of the dataset. Categorical and ordinal variables were presented as absolute and relative frequencies. Continuous variables were not normally distributed according to the Shapiro-Wilk test ($P < 0.001$). Therefore, they were summarized using median and interquartile range (IQR). Univariate ordinal logistic regression was performed to identify variables associated with HAS outcome. Variables that were statistically significant or clinically relevant were included in the multivariable analysis. Variables that remained statistically significant and clinically justified were included in the final predictive model. The final model was constructed using ordinal logistic regression based on the proportional odds assumption. The proportional odds assumption was explored using cumulative link models implemented in the ordinal package prior to model fitting. Penalized maximum

likelihood estimation was applied with a penalty of 0.5 to reduce overfitting and stabilize regression coefficients. To facilitate clinical application, the final ordinal logistic regression model was converted into a point-based scoring system using the nomogram function from the rms package. The assigned points were proportional to the regression coefficients of the final model, with predictors having a greater impact on outcome receiving a higher number of points. The total score was calculated by summing the points assigned to all predictors and was used to estimate the probability of each HAS outcome category. All analyses were performed in R version 4.5.1 (2025–06–13) using RStudio version 2025.09.1 (Posit Software, USA). The rms package was used for model development.

Complete data were available for sex, age, medical history of arterial hypertension and/or ischemic heart disease, aneurysm re-rupture, treatment strategy and HAS outcome. For variables with missing values, multiple imputation by chained equations was performed under the assumption of missing at random. Before imputation, missing value markers were standardized and data cleaning was performed. Variable types were appropriately specified. Predictive mean matching was used for continuous variables. Logistic regression was used for binary variables. Multinomial logistic regression was applied to nominal variables. Ordinal logistic regression was used for ordinal variables. Five imputed datasets were generated with 20 iterations. All subsequent analyses were performed in R using the MICE package.

RESULTS

Baseline Characteristics and Treatment Outcomes

A total of 56 patients were excluded from further analysis due to missing outcome data following transfer to other healthcare facilities. An additional 5 patients were excluded because outcome data were completely unavailable. Three more patients were excluded because the performed interventions did not meet the inclusion criteria: decompressive craniectomy was performed in 2 patients and ventriculostomy in 1 patient. The final derivation cohort consisted of 425 patients. Missing data among the included patients were limited. Data were unavailable for 96 patients regarding the size of the ruptured CAA and for 8 patients regarding the morphological type of the ruptured CAA. Missing data were recorded for 5 patients regarding presence of headache and limb paresis at admission; for 3 patients regarding CV; for 2 patients regarding time from CAA rupture to hospital admission, mWFNS grade at admission, seizures at admission, HAP, and conditions requiring tracheostomy; and for 1 patient regarding hemorrhage distribution, location of the ruptured CAA, and number of CAAs.

Women slightly predominated in the study population (54%). The largest proportion of patients belonged to the 45–60 year age group (49%). Cardiovascular comorbidity, including arterial hypertension and/or ischemic heart disease, was present in 67% of patients. Most patients (72%) were admitted within the first 3 days after CAA rupture. The median length of hospital stay was approximately three weeks. Approximately one third of patients had aSAH with a parenchymal component. The most frequent location of ruptured CAAs was the anterior cerebral artery (48%). Aneurysm re-rupture was documented in 17% of cases. Most CAAs were saccular. The median aneurysm size was 7 mm. Multiple CAAs were present in fewer than one fifth of patients. According to mWFNS grading at admission, most patients had grade I (45%), whereas approximately one fifth were admitted in severe condition (grades IV-V). Headache was the predominant clinical symptom at presentation. Microsurgical treatment was performed in the majority of cases. Among surgical patients, the majority were in a stable condition before surgery (59%). Conservative management was applied in approximately one quarter of patients. CV was recorded in half of the cohort. HAP developed in one quarter of patients, whereas tracheostomy was required in 17% of cases. Assessment of treatment outcomes using the HAS demonstrated that nearly half of the patients were discharged without neurological deficit (44%). Approximately one quarter had a severe neurological deficit. Mortality was observed in nearly one fifth of patients (Table 1).

Selection and Design of Predictors

Univariate analysis was performed to identify factors associated with treatment outcomes after aSAH. Variables that demonstrated a statistically significant association with the outcome included time from CAA rupture to hospital admission, hemorrhage distribution, aneurysm re-rupture, size of the ruptured aneurysm, mWFNS grade at admission, presence of seizures, headache, and limb paresis at admission, treatment strategy, as well as the occurrence of CV, HAP, and conditions requiring tracheostomy. These variables were entered into the multivariable analysis to determine independent predictors. Based on these predictors, a predictive model was developed to estimate the probability of achieving each HAS outcome category. Although sex and age were not statistically significant in either univariate or multivariable analysis, they were included in the final model due to their clinical relevance and a priori importance. According to the multivariable analysis, independent predictors of treatment outcome after aSAH were aneurysm re-rupture, size of the ruptured aneurysm, presence of limb paresis at admission, treatment strategy, as well as the occurrence of CV and HAP. The mWFNS grade at admission demonstrated borderline statistical significance in the multivariable analysis ($P = 0.063$). This variable was also included in the final predictive model because of its well-established predictive value in patients with aSAH.[9]

Univariate and multivariable analyses (Table 2), as well as subsequent development of the predictive model, were performed using imputed data.

Development of the Ordinal Predictive Model

The final ordinal predictive model was constructed using 9 variables: sex, age groups, presence of aneurysm re-rupture, size of the ruptured aneurysm, mWFNS grade at admission, presence of limb paresis at admission, treatment strategy, and the occurrence of CV and HAP.

Assessment of the proportional odds assumption using cumulative link models suggested possible departures from the assumption for several predictors. However, attempts to fit a fully non-proportional odds model resulted in convergence difficulties and unstable parameter estimates, likely due to sparse data in some outcome categories. Therefore, the proportional odds

Table 1. Distribution of Baseline Characteristics in the Derivation Cohort. Data are Presented Without Imputation

Predictors	Derivation Cohort (n = 425)
Sex	
Female	231 (54%)
Male	194 (46%)
Age groups	
18—44 year	107 (25%)
45—60 year	207 (49%)
61—75 year	101 (24%)
76+ year	10 (2%)
Arterial hypertension and/or ischemic heart disease	
Yes	283 (67%)
No	142 (33%)
Time from CAA rupture to hospital admission (n = 423)	
Early admission (≤3 days)	303 (72%)
Delayed admission (4—14 days)	106 (25%)
Late admission (≥15 days)	14 (3%)
Length of hospital stay, Median (IQR)	20 (15)
Hemorrhage distribution (n = 424)	
aSAH with parenchymal component	136 (32%)
aSAH without parenchymal component	288 (68%)
Location of the ruptured CAA (n = 424)	
ACA	203 (48%)
MCA	104 (25%)
ICA	88 (21%)
BA	15 (4%)
VA	14 (3%)
Re-rupture of a CAA	
Yes	71 (17%)
No	354 (83%)
Size of the ruptured CAA (n = 329), Median (IQR)	7 (4)
Morphological type of the ruptured CAA (n = 417)	
Saccular	399 (96%)
Fusiform	7 (2%)
Multilobular	5 (1%)
Bilobular	6 (1%)
The number of CAAs (n = 424)	
Single CAA	352 (83%)
Multiple CAAs	72 (17%)
mWFNS grade at admission (n = 423)	
I	189 (45%)
II	76 (18%)
III	74 (18%)
IV	66 (16%)
V	18 (4%)
Seizures at admission (n = 423)	
Yes	36 (9%)
No	387 (92%)
Headache at admission (n = 420)	
Yes	355 (85%)
No	65 (16%)
Limb paresis at admission (n = 420)	
Yes	75 (18%)
No	345 (82%)
Treatment strategy	
Surgical patients with clinical deterioration	16 (4%)
Surgical patients with stable condition	249 (59%)
Surgical patients with clinical improvement	64 (15%)
Conservatively treated patients	96 (23%)
Cerebral vasospasm (n = 422)	
Yes	211 (50%)
No	211 (50%)
Hospital-acquired pneumonia (n = 423)	
Yes	106 (25%)
No	317 (75%)
Conditions requiring tracheostomy (n = 423)	
Yes	71 (17%)
No	352 (83%)
Treatment outcome according to the HAS	
HAS 0	187 (44%)
HAS 1	38 (9%)
HAS 2	105 (25%)
HAS 3	95 (22%)

Continues

model was retained as a stable and interpretable modelling approach.

Internal validation of the developed predictive model was performed using bootstrap resampling (B = 200) with the validate procedure. The optimism-corrected discrimination index Dxy was 0.639. This corresponds to an approximate C-index of 0.82. These results confirm the good discriminative ability of the model to distinguish between patients with different scores of functional

Table 2. Univariate and Multivariable Analysis Using Ordinal Logistic Regression to Identify Potential Predictive Factors

	Univariate Analysis		Multivariable Analysis	
Predictors	**Or (95% CI)**	***P* Value**	**Or (95% CI)**	***P* Value**
Sex	1.08 (−0.28 to 0.425)	0.687	0.987 (−0.45148 to 0.4245)	0.953
Age groups	1.07 (−0.161 to 0.305)	0.544	0.775 (−0.55641 to 0.044)	0.095
Arterial hypertension and/or ischemic heart disease	0.976 (−0.4 to 0.352)	0.898		
Time from CAA rupture to hospital admission	0.719 (−0.673 to 0.00479)	0.055	0.846 (−0.58557 to 0.2434)	0.429
Length of hospital stay	0.994 (−0.0161 to 0.00324)	0.221		
Hemorrhage distribution	2.66 (0.603 to 1.35)	*<0.001*	1.053 (−0.44854 to 0.5457)	0.838
Location of the ruptured CAA				
ACA — reference				
MCA	1.18 (−0.266 to 0.591)	0.456		
ICA	1.01 (−0.451 to 0.466)	0.969		
BA	1.12 (−0.924 to 1.127)	0.828		
VA	1.38 (−0.629 to 1.261)	0.504		
Re-rupture of a CAA	4.16 (0.938—1.92)	*<0.001*	2.778 (0.42249—1.6301)	*<0.001*
Size of the ruptured CAA	1.07 (0.0247—0.105)	*0.002*	1.05 (0.00208—0.0967)	*0.042*
Morphological type of the ruptured CAA				
Saccular — reference				
Fusiform	1.051 (−1.658 to 1.667)	0.952		
Multilobular	0.402 (−2.879 to 0.67)	0.287		
Bilobular	1.796 (−0.936 to 2.107)	0.437		
The number of CAAs	0.872 (−0.596 to 0.316)	0.556		
mWFNS grade at admission	1.86 (0.476—0.773)	*<0.001*	1.241 (−0.01242 to 0.4442)	0.063
Seizures at admission	2.5 (0.282—1.56)	*0.005*	1.274 (−0.52564 to 1.0214)	0.538
Headache at admission	0.313 (−1.64 to (−0.69))	*<0.001*	0.742 (−0.96113 to 0.3662)	0.377
Limb paresis at admission	3.63 (0.85—1.74)	*<0.001*	2.318 (0.23648—1.4557)	*0.007*
Treatment strategy				
Surgical patients with stable condition — reference				
Surgical patients with clinical deterioration	2.8 (0.1265—1.94)	*0.025*	1.304 (−0.85945 to 1.3795)	0.639
Surgical patients with clinical improvement	1.74 (0.0664—1.04)	*0.026*	1.092 (−0.52878 to 0.6988)	0.778
Conservatively treated patients	2.77 (0.5606—1.48)	*<0.001*	2.232 (0.21642—1.3917)	*0.007*
Cerebral vasospasm	2.25 (0.457—1.17)	*<0.001*	2.097 (0.30878—1.1779)	*<0.001*
Hospital-acquired pneumonia	50.5 (3.33—4.56)	*<0.001*	31.084 (2.77309—4.1432)	*<0.001*
Conditions requiring tracheostomy	9.66 (1.77—2.79)	*<0.001*	1.854 (−0.08576 to 1.327)	0.086

The values in italics indicate statistically significant results.

outcome according to HAS. The explanatory performance of the model remained high after correction for optimism. The optimism-corrected R^2 was 0.53 indicating that the model explains more than half of the variability in treatment outcome. The calibration slope after correction was 0.95 which is close to the ideal value of 1 and indicates only a moderate degree of overfitting (Figure 1). These findings demonstrate good agreement between predicted and observed outcomes in the study cohort. The level of optimism was low for all key performance measures with values of approximately 0.03 for Dxy and 0.03 for R^2. These results

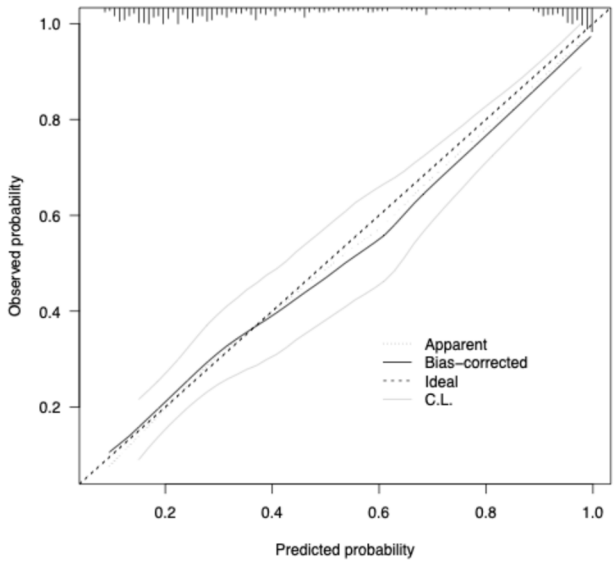

Figure 1. Calibration curve (bootstrap-corrected).

further confirm the stability and reproducibility of the model during internal validation.

To facilitate individualized risk estimation, a nomogram based on the final ordinal logistic regression model was developed (Figure 2). The nomogram provides a graphical representation of the model and allows estimation of the probability of each HAS outcome category for an individual patient.

For practical application, the ordinal predictive model is presented in tabular form. The table shows the contribution of each predictor to the scoring system and allows calculation of the total score and the corresponding probabilities for each HAS category (Table 3).

This provides an individual risk index with corresponding probabilities for each HAS outcome category. For example, a 47-year-old woman with a 5 mm ruptured CAA, mWFNS grade III at admission, without limb paresis at admission, re-rupture, CV or HAP, who have stable condition before surgery and underwent microsurgical treatment, would have a total predictive score of $6 + 0 + 7 + 18 + 0 + 0 + 0 + 0 + 0 = 31$. Points according to the size of the ruptured CAA are presented in Figure 3. According to the predictive model and Figure 4, this score corresponds to the following outcome probabilities: HAS 0—71%, HAS 1—10%, HAS 2—17%, and HAS 3—2%.

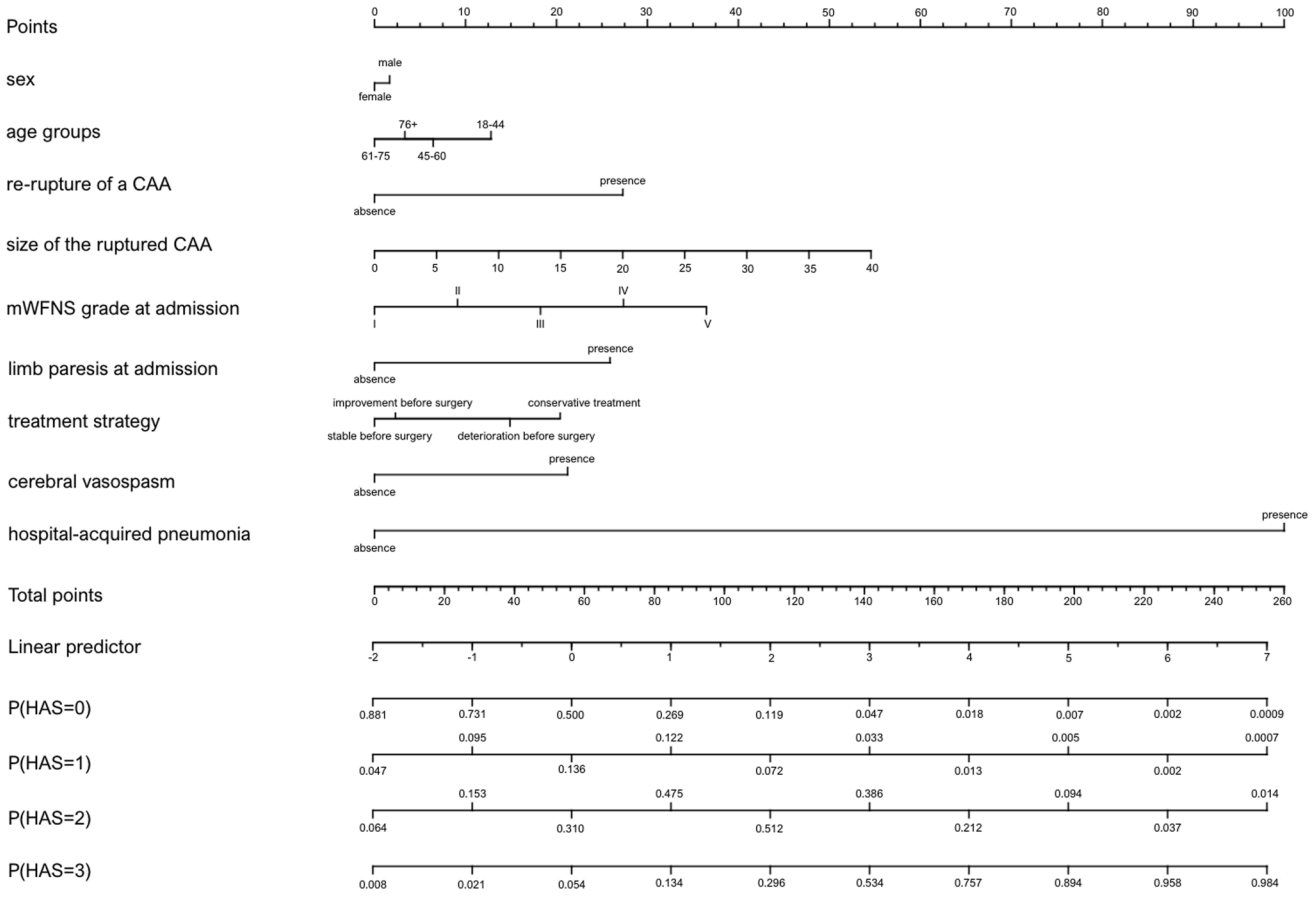

Figure 2. Nomogram for prediction of HAS outcomes after rupture of CAAs.

Table 3. Predictors Included in the Ordinal Predictive Model for Estimating the Risk of Achieving Treatment Outcomes According to HAS

Ordinal predictive Model	Points
Sex	
Female	0
Male	2
Age groups	
18–44 year	13
45–60 year	6
61–75 year	0
76+	3
Re-rupture of a CAA	
Absence	0
Presence	27
Size of the ruptured CAA, mm	1.3643 × CAA size (mm)
mWFNS grade at admission	
I	0
II	9
III	18
IV	27
V	36
Limb paresis at admission	
Absence	0
Presence	26
Treatment strategy	
Surgical patients with clinical deterioration	15
Surgical patients with stable condition	0
Surgical patients with clinical improvement	2
Conservatively treated patients	20
Cerebral vasospasm	
Absence	0
Presence	21
Hospital-acquired pneumonia	
Absence	0
Presence	100

Note: to calculate the total predictive score for an individual patient, the points assigned to each predictor are summed.

DISCUSSION

In 2013 Jaja BNR et al. published the first systematic review of clinical predictive models in aSAH. Of 69 potentially relevant studies only 11 met the inclusion criteria. The review reported that the most frequently included predictors were age, severity at admission assessed by WFNS or Hunt–Hess scale, hemorrhage volume on CT according to Fisher or modified Fisher scale, and size of the ruptured aneurysm. In most studies where age was analyzed as a predictor, it was simplified by dichotomization.[6] In our study, age was stratified into 4 categories and included in the predictive model due to its clinical relevance, despite the absence of a statistically significant association with treatment outcome. A distinct distribution of points was observed across age groups. The youngest group (18–44 years) received the highest number of points (n = 13). The groups aged 45–60, 61–75, and 76–90 years received 6, 0, and 3 points, respectively. The 45–60-year group comprised the largest proportion of patients, the 18–44 and 61–75 year groups were comparable in size, and the 76- to 90-year group was the smallest. Several explanations are possible. Firstly, the score reflects the independent contribution of each age category after adjustment for other variables included in the model. It does not represent the overall risk associated with age alone. If age correlates with other predictors, such as mWFNS severity or development of complications, its independent contribution may shift. Secondly, given that prehospital mortality after aSAH is estimated at 22%–26%,[10] a selection effect should be considered. Younger patients with severe presentation are more likely to reach hospital care, whereas older patients are less likely to be admitted to hospital. These findings are consistent with the results reported by Ikawa F et al., who demonstrated that the association between age and unfavorable outcome after aSAH is nonlinear and depends on baseline clinical severity. In patients with WFNS grades I-III, the risk of unfavorable outcome remained relatively stable up to 50 years of age, increased gradually between 50 and 70 years, and rose sharply after 70 years. In patients with WFNS grades IV-V, a high risk of unfavorable outcome was already present in younger age groups and increased progressively with age.[11] Our findings support this logic, indicating that the effect of age is not universal and interacts with baseline severity and in-hospital events.

Another distinguishing feature of our model is that most studies included in the review by Jaja BNR et al. relied primarily on admission data. In contrast, our study incorporated information collected throughout the entire hospitalization period. This approach allowed inclusion of dynamic changes that significantly influenced outcome. For instance, postoperative pneumonia occurs in 13%–43% of patients undergoing surgical treatment for aSAH and is associated with higher mortality, prolonged hospital stay and increased healthcare costs.[12,13] In our study, HAP demonstrated the strongest association with a worse HAS outcome in univariate and multivariable analyses, and in the final predictive model. Similarly, CV is a key pathophysiological determinant of poor prognosis after aSAH. Mortality attributable to CV is approximately 20%. Angiographic vasospasm is detected in about 70% of patients with aSAH, but only 30% develop neurological deficits.[14] The review by Jaja BNR et al. noted that when CV was included in predictive models, it was typically recorded only at admission. In our study, CV was evaluated throughout hospitalization, as it predominantly occurs between 3 and 21 days after aneurysm rupture.[15]

It should also be considered that the Fisher scale does not account for a parenchymal component, which may substantially increase hemorrhage volume. Therefore, hemorrhage distribution

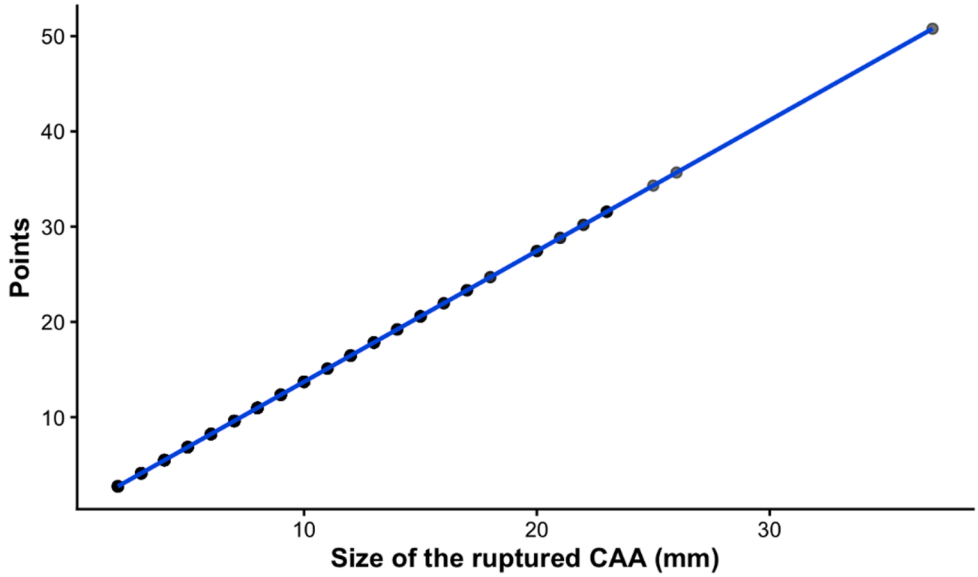

Figure 3. Linear relationship between the size of the ruptured CAA and the assigned score in the predictive model.

in our study was categorized as aSAH with or without a parenchymal component. In many published models, hemorrhage volume and intracerebral hematoma were significant predictors. In this analysis, this variable did not remain statistically significant in multivariable analysis and was not included in the final predictive model.

In studies included in the review by Jaja BNR et al., outcomes were predicted using dichotomized endpoints. These included mortality or unfavorable outcomes defined as severe disability, vegetative state or death at 3–12 months after aSAH. Dichotomization simplifies analysis but does not reflect the full clinical spectrum of outcome after aSAH. Neither the mRS nor the GOS was developed specifically for aSAH. In contrast, HAS was proposed for outcome assessment after aSAH.[5] For this reason, we developed an ordinal predictive model using HAS as the outcome measure.

In 2023, Parekh A et al. published an updated systematic review that expanded upon the earlier work by Jaja BNR et al. Most studies

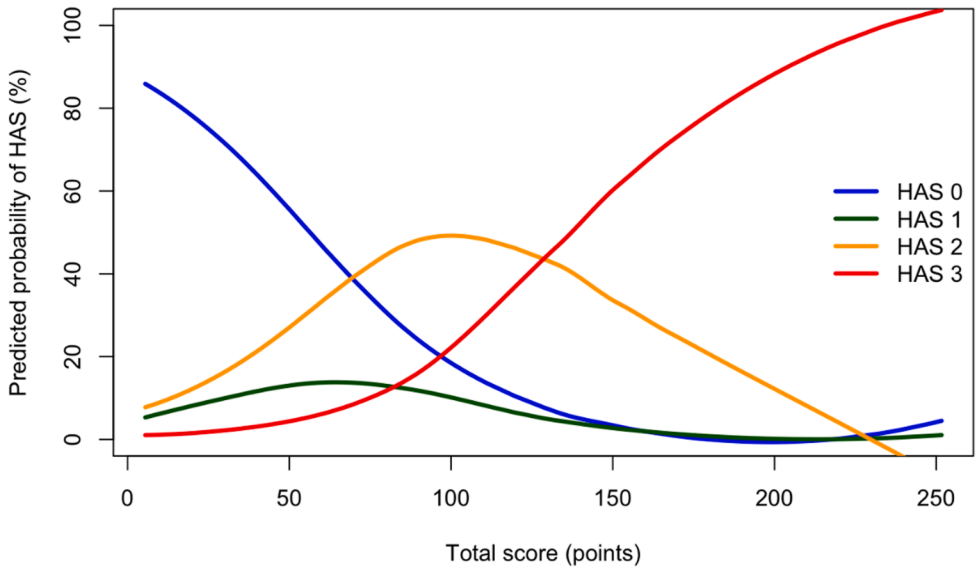

Figure 4. Predicted probabilities of HAS score according to total score in the ordinal predictive model.

continued to use mRS or GOS, which maintain the limitations related to sensitivity to clinical heterogeneity. Parekh A et al. emphasized the limited accuracy of models based solely on admission variables. They suggested that dynamic models integrating in-hospital data may have greater clinical utility.[7] In this context, our model is a dynamic predictive approach that integrates key in-hospital events and reflects the clinical course of patients.

Another limitation of existing predictive models after aSAH is the lack of standardization regarding outcome assessment. Studies using mRS or GOS evaluate outcomes at markedly different time points, including hospital discharge, 3 months, 6 months or 12 months. As a result, direct comparison of predictive models is challenging because they often estimate different clinical endpoints. In addition, dichotomized outcome assessment introduces further heterogeneity. There is no universal consensus regarding which mRS or GOS scores should be considered favorable or unfavorable. For example, Katsuki M et al.[16] defined a favorable outcome as mRS 0–3, whereas Maragkos GA et al.[17] classified mRS 0–2 as favorable and mRS 3–6 as unfavorable. Similar inconsistencies exist for GOS. Hong JY et al.[18] and Zhang P et al.[19] considered GOS scores 1–3 as poor outcomes, while Zafar SF et al.[20] distinguished 3 outcome categories: poor (GOS 1–2), intermediate (GOS 3), and good (GOS 4–5). Such variability complicates comparisons across studies, limits the reproducibility of prognostic models, and may affect clinical interpretation of results.

Furthermore, mRS has been criticized for placing substantial emphasis on physical mobility while providing less detailed assessment of cognitive, emotional, and social functioning, domains that are highly relevant for patients recovering from aSAH. In addition, detailed assessment using multi-level disability scales often requires specific training and may be time-consuming in routine clinical practice. In contrast, HAS provides a standardized, non-dichotomized approach specifically designed for in-hospital assessment after aSAH. The scale is simple to use and directly reflects rehabilitation requirements at discharge.[5]

Most previous models assessed severity only at admission and therefore did not capture changes in neurological status before intervention. However, preoperative clinical dynamics may influence timing and method of treatment, and also affect functional outcome. Therefore, in our study, mWFNS was assessed at admission and on the day of surgery in surgical patients, who were subsequently categorized into 3 groups based on deterioration, stability, or improvement before intervention. Also we added a separate group of conservatively treated patients. In the final predictive model, patients who received conservative treatment had the highest number of points and, consequently, the highest probability of a worse HAS outcome. The second highest number of points was assigned to surgical patients whose clinical condition deteriorated before surgery, which is consistent with clinical expectations. In contrast, surgical patients who remained clinically stable before intervention received zero points. This group demonstrated the best predicted outcome according to HAS, even compared with patients who showed preoperative clinical improvement. In our opinion, this finding may be explained by delayed surgery in patients who showed preoperative improvement, as surgeons may have waited for further clinical stabilization.

After the updated review by Parekh A et al., several new prognostic models were proposed. In 2023, Wan X et al. developed a model for patients with aSAH requiring mechanical ventilation. Their model focused on survival prediction in a critically ill subgroup.[21] In the same year, Li R et al. proposed the TAPS score based on early brain injury markers to predict unfavorable 90-day outcome. The endpoint was dichotomized as mRS 0–2 versus mRS≥3, which limits sensitivity to clinical heterogeneity.[22] In 2024, Wang et al. developed a logistic model to predict 30-day mortality in aSAH. In addition to clinical variables, they included albumin-corrected anion gap as a marker of metabolic and inflammatory disturbance.[23]

In this context, our predictive model demonstrates the advantages of a dynamic approach to outcome prognosis after aSAH. Unlike most previous models that rely exclusively on admission variables and dichotomized endpoints, our model integrates key in-hospital variables reflecting the entire hospitalization period and provides a four-score outcome assessment. This approach may offer greater clinical utility for optimizing management of patients with aSAH.

Limitations

This study has several limitations that should be considered when interpreting the results. First, the study was conducted at a single center and had a retrospective design. Therefore, selection bias and information bias cannot be completely excluded. Patient inclusion depended on the availability and completeness of historical medical records, and unmeasured confounding factors may have influenced both treatment decisions and outcomes. In addition, the database spans more than 2nulldecades and reflects clinical outcomes achieved by different neurosurgeons over time. Changes in surgical techniques and perioperative management during this period may have introduced additional heterogeneity into the dataset.

Second, although complete data were available for several key variables, missing values were present for some predictors. To reduce potential bias and preserve statistical power, multiple imputation by chained equations was used. However, the validity of this approach depends on the assumption that data were missing at random, which cannot be fully verified in retrospective datasets.

Third, although the proposed model incorporates dynamic variables collected during hospitalization, the temporal structure of some predictors was simplified. Complications such as CV, HAP, and conditions requiring tracheostomy were recorded as binary variables (present or absent), and the timing of their onset was not included in the analysis. As a result, the model may not fully capture the temporal relationship between these complications and treatment outcomes.

Fourth, the proportional odds assumption demonstrated possible deviations for several predictors, and a fully non-proportional odds model could not be reliably estimated because of convergence difficulties. Nevertheless, the proportional odds model demonstrated good discrimination and calibration during internal validation and was therefore retained as the final modelling approach.

Finally, although internal validation demonstrated satisfactory model performance, external validation was not performed.

Therefore, validation in independent multicenter cohorts is required before the model can be recommended for widespread clinical implementation.

CONCLUSIONS

A clinical ordinal predictive model was developed and internally validated to assess treatment outcomes in patients with aSAH using the HAS. The proposed model integrates baseline clinical characteristics and dynamic changes occurring throughout hospitalization, allowing a more comprehensive representation of the patient's clinical course after aSAH.

CRediT AUTHORSHIP CONTRIBUTION STATEMENT

Yuliia Solodovnikova: Writing — review & editing, Supervision, Project administration, Methodology, Conceptualization. **Anastasiia Revurko:** Writing — original draft, Methodology, Formal analysis, Data curation, Conceptualization. **Anatoliy Son:** Writing — review & editing, Supervision, Project administration, Conceptualization.

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