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Research Article

Keywords: Aneurysmal subarachnoid hemorrhage, WFNS grade IV-V, Fisher grade III-IV, Prognosis, Modified Rankin Scale

Posted Date: June 22nd, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-9920848/v1>

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Additional Declarations: No competing interests reported.

Short-term Survival Outcomes of Poor-Grade Aneurysmal Subarachnoid Hemorrhage with Fisher Grade III-IV: A Single-Centre Retrospective Study

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Abstract

Background: Poor-grade aneurysmal subarachnoid hemorrhage (PGASAH) remains a critical focus and clinical challenge, characterized by an extremely poor prognosis influenced by multiple intertwined factors. This study aimed to comprehensively analyze independent risk factors for PGASAH, identify key effect modifiers, and develop and validate survival prediction models at different time points for short-term prognosis.

Methods: A retrospective study included 500 patients diagnosed with PGASAH. Comprehensive analyses were conducted on baseline clinical characteristics, complication patterns, and prognostic factors. To explore the nonlinear relationship between age and outcomes, Kaplan-Meier survival curves, restricted cubic spline regression, and Cox proportional hazards models were utilized. Subgroup analyses and interaction tests were performed to detect potential effect modifiers. Based on identified independent prognostic factors, Nomogram models predicting 1-month and 6-month survival probabilities were developed. The performance of these models was rigorously assessed using 500-time Bootstrap resampling for internal validation and external validation.

Results: The patient cohort had a mean age of 57.01 ± 11.13 years, with a slight predominance of females (55.4%). Postoperative pneumonia developed in a substantial proportion of patients, reaching an incidence rate of 82.6%. Multivariate Cox regression modeling revealed several independent prognostic factors: age (HR 1.02), maximum aneurysm diameter (HR 1.04), WFNS grade V (HR 1.86), cerebral vasospasm (HR 4.31), brain herniation (HR 2.18), and hydrocephalus (HR 0.28). Among these, cerebral vasospasm emerged as the most significant risk factor, whereas hydrocephalus appeared to confer a protective effect. The influence of age on prognosis showed a nonlinear threshold pattern, with 72 years identified as the critical cutoff; individuals aged 72 years or older experienced a doubling of mortality risk (HR=2.01). Notably, brain herniation ($P=0.013$) and delayed cerebral infarction ($P=0.032$) acted as significant effect modifiers, with age stratification showing greater prognostic relevance in patients lacking these complications. The prognostic value of aneurysm size was modified by sex, hypertension, and decompressive craniectomy status, while the predictive value of Fisher grade was highly dependent on treatment modality. The 1-month survival prediction model exhibited robust discriminative performance (AUC=0.741, Brier score 17.3%) with reproducible validation outcomes across both internal and external datasets; however, the 6-month model performed poorly (AUC=0.513), approaching random chance.

Conclusions: The prognostic evaluation of PGASAH integrates demographic, clinical, and complication-related factors for comprehensive risk assessment. This study establishes a 72-year age threshold and identifies brain herniation and delayed cerebral infarction as critical modifiers of risk stratification. A validated 1-month survival prediction nomogram demonstrated robust clinical utility for decision-making in acute management phases. However, extending predictive accuracy to long-term outcomes necessitates incorporating post-acute variables like functional

recovery metrics and neurocognitive assessments.

Key words: Aneurysmal subarachnoid hemorrhage, WFNS grade IV-V, Fisher grade III-IV, Prognosis, Modified Rankin Scale

Subarachnoid hemorrhage (SAH) constitutes roughly 2 – 7% of all stroke cases, with approximately 80% of these instances attributed to ruptured intracranial aneurysms, known as aneurysmal SAH (aSAH)[1-3]. Although relatively uncommon, aSAH is associated with significant mortality and morbidity rates. An estimated 25% of patients succumb before reaching medical care, and among those presenting with a Glasgow Coma Scale (GCS) score of 3 – 5, the 30-day mortality reaches as high as 55.7%[4, 5]. Moreover, less than half of the survivors achieve independent functional recovery, resulting in a considerable strain on both families and the broader society[6]. Patients admitted with aSAH classified under World Federation of Neurological Surgeons (WFNS) grades IV – V are categorized as having poor-grade aSAH (PGASAH)[7]. This subgroup faces particularly severe early brain injury (EBI), complex secondary pathophysiological complications, difficult clinical decision-making, and extremely unfavorable outcomes, making them a critical and challenging focus in clinical practice.[8, 9].

The prognosis of PGASAH patients is determined by multiple intertwined factors. Initial hemorrhage severity, assessed by modified Fisher grade and WFNS grade, plays a pivotal role in predicting outcomes [10, 11]. Significant bleeding can trigger acute intracranial hypertension, leading to a sharp reduction in cerebral perfusion pressure and resulting in global cerebral ischemia and early brain injury[12]. On this basis, cerebral vasospasm and delayed cerebral ischemia (DCI) act as secondary injuries, further aggravating brain damage through mechanisms such as microcirculatory dysfunction, cortical spreading depolarization, and neurovascular uncoupling[13, 14]. Additionally, acute hydrocephalus and refractory intracranial hypertension may necessitate decompressive craniectomy (DC), which itself carries a risk of brain herniation [9, 15]. Numerous studies have highlighted the close association between prognosis and factors such as patient age, aneurysm size, WFNS grade, cerebral vasospasm, brain herniation, and hydrocephalus [10, 16-20]. However, the interactions and effect modifications among these factors remain poorly elucidated.

Age has long been recognized as a critical prognostic factor in PGASAH, with historical assumptions indicating a straightforward linear association between increasing age and poorer clinical outcomes [21, 22]. However, recent findings challenge this perspective, suggesting that the impact of age may instead follow a nonlinear pattern, potentially involving threshold effects [23]. Furthermore, the extent to which treatment approaches influence the prognostic relevance of aneurysm size and Fisher grade remains insufficiently explored [10]. Current predictive models largely emphasize short-term outcomes, while long-term survival estimates are often affected by post-acute factors such as the intensity of rehabilitation, management of chronic conditions, and availability of social support[10, 24-26]. Therefore, conducting an in-depth analysis of independent risk factors for PGASAH, identifying key effect modifiers, and developing and validating survival prediction models at various time points for short-term prognosis are crucial steps toward enabling personalized precision risk assessment and enhancing clinical decision-making.

Given these clinical imperatives, we conducted a retrospective analysis of 500 PGASAH patients to systematically evaluate baseline demographics, complication patterns, and prognostic

determinants. This investigation specifically focused on elucidating nonlinear age-related effects while identifying effect modifiers through rigorous statistical modeling. Building on these findings, we developed 1- and 6-month survival prediction nomograms incorporating key clinical variables, which demonstrate robust discriminative capacity and calibration accuracy. These validated tools offer practical frameworks for precision stratification and tailored therapeutic interventions in PGASAH management.

Methods

Study Subjects

The purpose of this study was to explore the potential clinical risk factors and prognosis of PGASAH. The study protocol received approval from the General Hospital of Ningxia Medical University, as sanctioned the Ethics Committee of Ningxia Hui Autonomous Region, which it is university teaching hospitals. The study cohort comprised 500 eligible patients with PGASAH, and the data collection period extended from January 2018 to April 2025. The clinical data are detailed in Table 1.

The following are the inclusion criteria: all patients diagnosed with PGASAH; diagnosis confirmed through computed tomographic angiography (CTA) and / or digital subtraction angiography (DSA) within 24 hours after the onset of the attack; treatment in the neurosurgery intensive care unit after surgery; Patients families signed the informed consent form, indicating agreement to participate in the follow-up; and complete clinical data and a follow-up period exceeding one month, excluding cases where death occurred within one month.

The exclusion criteria are as follows: patients with preoperative cerebral herniation; SAH caused by hypertensive cerebral hemorrhage, arteriovenous malformation, etc.; PGASAH cases with Fisher I to II grades; and presence of primary cerebral infarction or other acute fatal diseases, such as pulmonary embolism, acute myocardial infarction, or systemic organ failure.

The relative definitions: Cerebral vasospasm (CVS) is defined as the degree of stenosis in the parent artery lumen is more than 50% during preoperative CTA or DSA examination. The cerebral herniation is mainly characterized by tentorial herniation and cerebellar tonsillar herniation, with the main clinical feature being elevated intracranial pressure, dilated pupil, consciousness disorder, and abnormalities of vital sign.

Analysis of outcomes

Follow-up evaluations were carried out through telephone communication, outpatient appointments or family visits. In this study, the prognostic indicator was the modified Rankin score (mRS) after 1 month of follow-up. A score of 0 ~ 5 is alive, while a score of 6 was considered indicative of die.

Statistical analysis

Statistical analysis and image presentation were conducted using R (version 4.0.0). Mean $\pm$ SD was used to represent continuous figures, while frequency (percentage) represented categorical figures. The chi-square test, t-test, and Wilcoxon rank sum test were utilized to assess the differences in clinical characteristics between the two groups. Survival analysis of patients was conducted using Kaplan Meier and univariate, and multivariate Cox regression analyses. The forest distribution map provided a more visible and intuitive representation of the factors affecting mortality. A statistically significant threshold was set at a p-value of < 0.05 .

Results

The study initially included 526 cases; 15 of them died of conservative treatment, and 5 cases

lost their visit. and 6 cases had preoperative cerebral herniation, resulting in 500 cases meeting the criteria.

Table 1 Baseline characteristics of the study participants.

Variables		Total (n = 500)	dead		Statistic (χ^2, t, Z)	P
			no, n = 353 (%)	yes, n = 147(%)		
Age, y		57.01 $\pm$ 11.13	56.22 $\pm$ 11.25	58.91 $\pm$ 10.62	-2.48	0.014
Age (60 ys)		312 (62.40)	228 (64.59)	84 (57.14)	2.45	0.117
Sex (male)		223 (44.60)	152 (43.06)	71 (48.30)	1.15	0.283
Smoking		147 (29.40)	102 (28.90)	45 (30.61)	0.15	0.701
Alcohol abuse		68 (13.60)	46 (13.03)	22 (14.97)	0.33	0.565
Hyperlipidemia		84 (16.83)	61 (17.33)	23 (15.65)	0.21	0.647
Diabetes		31 (6.20)	25 (7.08)	6 (4.08)	1.61	0.205
Hypertension		392 (78.40)	274 (77.62)	118 (80.27)	0.43	0.512
Timing (day), median [IQR]		2.00 (1.00, 3.00)	2.00 (1.00, 3.00)	1.00 (1.00, 2.50)	-1.49	0.137
Size (mm)		5.93 $\pm$ 3.61	5.64 $\pm$ 3.20	6.62 $\pm$ 4.37	-2.79	0.005
Neck width (mm)		3.97 $\pm$ 2.07	3.86 $\pm$ 2.12	4.22 $\pm$ 1.94	-1.74	0.082
Location of aneurysm	anterior	447 (89.40)	312 (88.39)	135 (91.84)	1.31	0.253
	posterior	53 (10.60)	41 (11.61)	12 (8.16)		
Fisher grade	III	128 (25.60)	101 (28.61)	27 (18.37)	5.72	0.017
	IV	372 (74.40)	252 (71.39)	120 (81.63)		
WFNS grade	IV	314 (62.80)	248 (70.25)	66 (44.90)	28.56	<0.001
	V	186 (37.20)	105 (29.75)	81 (55.10)		
Treatment method	coiling	232 (46.40)	165 (46.74)	67 (45.58)	0.06	0.812
	clipping	268 (53.60)	188 (53.26)	80 (54.42)		
Vasospasm		427 (85.40)	286 (81.02)	141 (95.92)	18.48	<0.001
LD		98 (19.60)	62 (17.56)	36 (24.49)	3.16	0.075
DC		159 (31.80)	101 (28.61)	58 (39.46)	5.63	0.018
Pneumonia		413 (82.60)	296 (83.85)	117 (79.59)	1.31	0.252
Intracranial infection		132 (26.40)	93 (26.35)	39 (26.53)	<0.01	0.966
Cerebral herniation		69 (13.80)	26 (7.37)	43 (29.25)	41.79	<0.001
DCI		291 (58.20)	210 (59.49)	81 (55.10)	0.89	0.365
Epilepsy		12 (2.40)	9 (2.55)	3 (2.04)	<0.01	0.986
Hydrocephalus		84 (16.80)	70 (19.83)	14 (9.52)	7.89	0.005

Abbreviation: *IQR*, interquartile range; *LD*: continuous lumbar drainage; *DC*: decompressive craniectomy; *DCI*: delayed cerebral infarction.

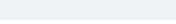
Baseline characteristics

Clinical data showed that female patients (277 cases, 55.4%) outnumbered male patients (223 cases, 44.6%). The age ranged from 18 to 87 years, with a mean age of 57.01 $\pm$ 11.13 years. Elderly patients (≥ 60 years) accounted for 62.4% of the cohort. Among the 500 cases, 392 patients (78.4%) had a history of hypertension, and 31 (6.2%) had diabetes mellitus. According to WFNS grading, 314 cases (62.8%) were classified as WFNS IV grade and 186 cases (37.2%) as WFNS V grade. Treatment methods included surgical clipping in 268 cases (53.6%) and endovascular embolization in 232 cases (46.4%). Of the ruptured aneurysms, 447 (89.4%) were

located in the anterior circulation and 53 (10.6%) in the posterior circulation. The median time to treatment was 2.00 days (IQR: 1.00, 3.00). Postoperative complications included pneumonia (413 cases, 82.6%), intracranial infection (132 cases, 26.4%), delayed cerebral ischemia (291 cases, 58.2%), cerebral herniation (69 cases, 13.8%), symptomatic epilepsy (12 cases, 2.4%), and hydrocephalus (84 cases, 16.8%). (table 1)

Patients in the death group had a significantly higher mean age ($P=0.014$) and larger maximum aneurysm diameter ($P=0.005$). Regarding disease severity, the death group showed a higher proportion of Fisher grade IV ($P=0.017$) and WFNS grade V ($P<0.001$). In terms of complications, the death group exhibited significantly increased incidences of cerebral vasospasm ($P<0.001$), brain herniation ($P<0.001$), and delayed cerebral infarction. Notably, the incidence of hydrocephalus was paradoxically lower in the death group ($P=0.005$), which may be attributable to timely external ventricular drainage intervention. (table 1)

Table 2 Univariate and multivariate Cox regression analyses showing the HR of death in 500 patients with poor-grade aSAH.

Group	HR (95% CI)	● un ● mul	Univariate analysis		Multivariate analysis	
			HR(95% CI)	p value	HR(95% CI)	p value
Age, y			1.02(1.00,1.03)	0.029	1.02(1.00,1.03)	0.018
Age ≥ 60ys			0.78(0.56,1.09)	0.142		
Sex (male)			0.84(0.61,1.16)	0.284		
Smoking			1.05(0.74,1.50)	0.768		
Alcohol abuse			1.15(0.73,1.80)	0.557		
Hyperlipidemia			0.90(0.58,1.41)	0.650		
Diabetes			0.54(0.24,1.22)	0.139		
Hypertension			1.09(0.73,1.64)	0.673		
Timing 24h to 48h			0.76(0.51,1.14)	0.186		
Timing > 48h			0.77(0.52,1.14)	0.186		
Size (mm)			1.05(1.01,1.09)	0.006	1.04(1.00,1.08)	0.043
Neck width (mm)			1.06(0.99,1.13)	0.104		
Location of aneurysm (posterior)			0.69(0.38,1.24)	0.213		
Fisher grade (IV)			1.53(1.01,2.32)	0.047	1.02(0.65,1.60)	0.919
WFNS grade (V)			2.41(1.74,3.33)	<0.001	1.86(1.25,2.77)	0.002
Treatment (clipping)			1.03(0.74,1.42)	0.880		
Vasospasm			4.47(1.97,10.13)	<0.001	4.31(1.88,9.86)	0.001
LD			1.22(0.83,1.78)	0.311		
DC			1.45(1.04,2.02)	0.029	1.22(0.86,1.74)	0.271
Pneumonia			0.73(0.49,1.10)	0.132		
Intracranial infection			0.91(0.63,1.31)	0.612		
Cerebral herniation			3.59(2.59,4.96)	<0.001	2.18(1.42,3.34)	<0.001
DCI			0.92(0.66,1.28)	0.632		
Epilepsy			0.86(0.28,2.71)	0.801		
Hydrocephalus			0.44(0.26,0.77)	0.004	0.28(0.16,0.49)	<0.001

Low Risk High Risk

Abbreviation: HR, Hazard Ratio; IQR, interquartile range; LD: continuous lumbar drainage; DC: decompressive craniectomy; DCI: delayed cerebral infarction.

Predictors of survival outcomes

Univariate Cox regression analysis revealed that Fisher grade IV ($P=0.047$) and DC ($P=0.029$) were significantly associated with mortality risk. However, after adjustment in the multivariate analysis, neither factor remained statistically significant, suggesting that they may be influenced by confounding variables rather than serving as independent predictors of death. Multivariate Cox regression analysis identified the following independent predictors of mortality: age (HR 1.02, 95%CI: 1.00 – 1.03, $P=0.018$), maximum aneurysm diameter (HR 1.04, 95%CI: 1.00 – 1.08, $P=0.043$), WFNS grade V (HR 1.86, 95%CI: 1.25 – 2.77, $P=0.002$), cerebral vasospasm (HR 4.31, 95%CI: 1.88 – 9.86, $P=0.001$), brain herniation (HR 2.18, 95%CI: 1.42 – 3.34, $P<0.001$), and hydrocephalus (HR 0.28, 95%CI: 0.16 – 0.49, $P<0.001$). Notably, cerebral vasospasm emerged as the strongest risk factor, with a more than 4-fold increase in mortality risk; conversely, hydrocephalus demonstrated a protective effect, which may be attributable to timely external ventricular drainage intervention. (table 2)

Restricted cubic spline (RCS) regression analysis revealed a nonlinear relationship between age and mortality risk, characterized by a complex "rise-fall-rise" pattern: in the <50 years group, HR was <1 and gradually increased, indicating relatively low risk with age-related escalation; a first minor risk peak emerged in the 50 – 55.9 years group ($HR>1$); HR declined to a nadir (~ 0.85) in the 55.9 – 72.4 years group—this phenomenon likely reflects reference point selection, patient selection bias, or more aggressive treatment strategies in this age group rather than a true biological protective effect of age; HR increased sharply beyond 72.4 years, forming a second risk peak with substantial magnitude. Although the overall trend supports a positive correlation between advancing age and mortality risk, RCS analysis uncovered nonlinear features that linear models fail to capture, with 72.4 years identified as a critical threshold for abrupt risk escalation, holding significant clinical decision-making value (Fig.1 A). The relationship between aneurysm size and mortality risk was more straightforward: using 5.005 mm as the reference point, HR remained consistently above baseline and rose steadily with diameters >5 mm, consistent with Cox regression findings (4% increase in mortality risk per 1 mm increment, HR 1.04) (Fig.1 B). This monotonic ascending pattern aligns with the clinical understanding that "larger aneurysms portend worse outcomes after rupture," providing a clear quantitative basis for risk stratification.

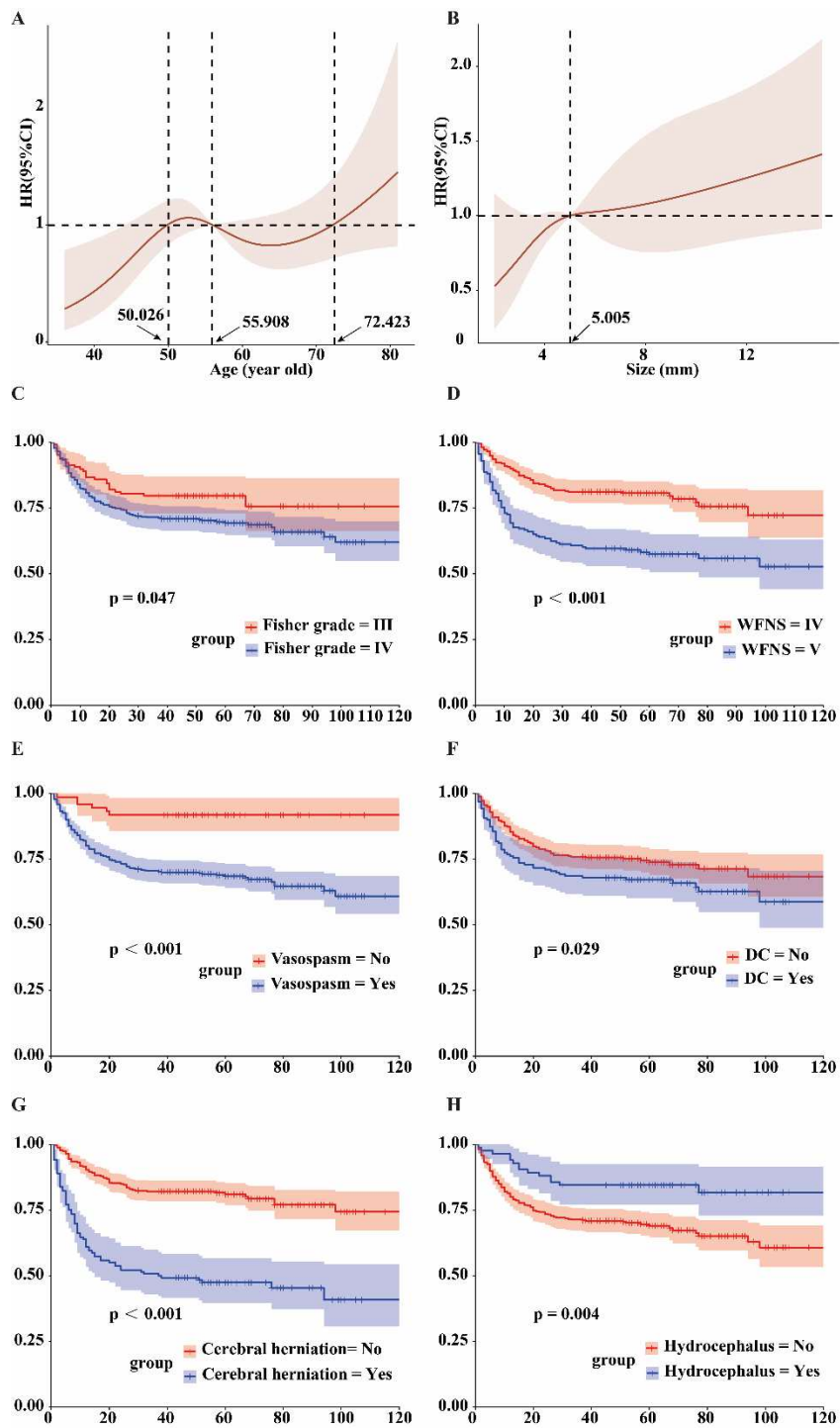
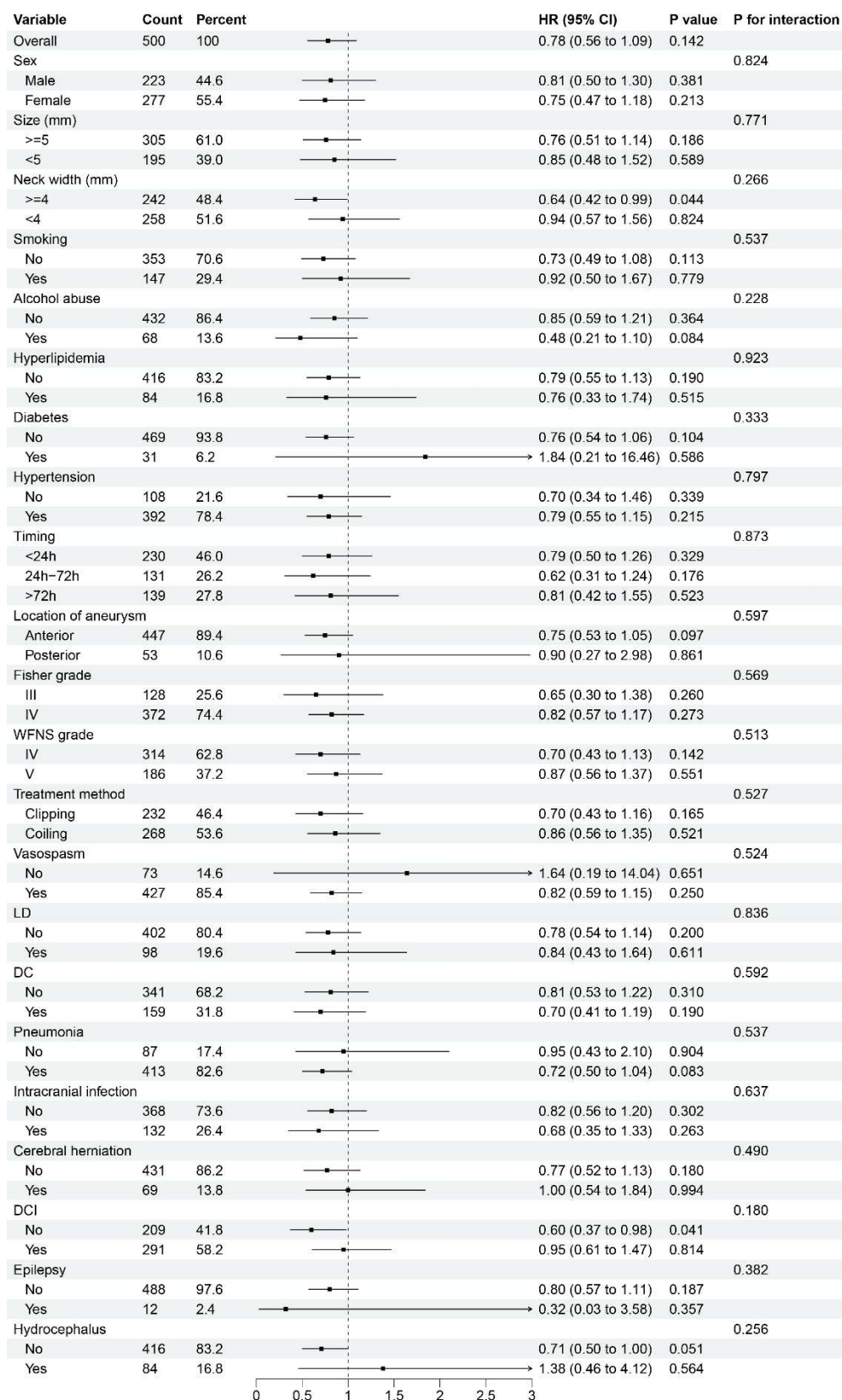


Fig.1 Survival analysis of prognostic factors in PGASAH with Fisher Grade III-IV: restricted cubic spline curves and Kaplan-Meier survival curves.

Table 3 Cox regression subgroup analysis stratified by multiple factors, using age (<60 vs. ≥ 60 years) as the exposure variable.



Abbreviation: HR, Hazard Ratio; IQR, interquartile range; LD: continuous lumbar drainage; DC: decompressive craniectomy; DCI: delayed cerebral infarction.

Survival Outcomes in different subgroups

Subgroup analyses of this study revealed complex interaction patterns influencing survival in poor-grade aSAH patients: Age showed no statistically significant main effect (HR=0.78, P=0.142), although a protective trend was observed, with no significant effect modifiers identified (all interaction P>0.05) (table 3). The prognostic value of aneurysm size was significantly modified by sex, hypertension, and DC status, with statistically significant interaction effects (all P=0.03 – 0.034), rendering aneurysm size a significant independent prognostic factor specifically in female, non-hypertensive, and non-DC patients (table S1). The predictive value of Fisher grade was highly dependent on treatment modality, demonstrating important prognostic stratification significance for clipping patients but limited value for coiling patients (table S2). WFNS grade showed relatively stable effects across subgroups, yet exhibited more prominent prognostic discrimination in patients without complications such as pneumonia (table S4). These findings suggest that clinical decision-making for poor-grade aSAH requires comprehensive consideration of multiple factors including patient demographics, comorbidities, and treatment modalities to achieve individualized precision risk assessment and prognostication.

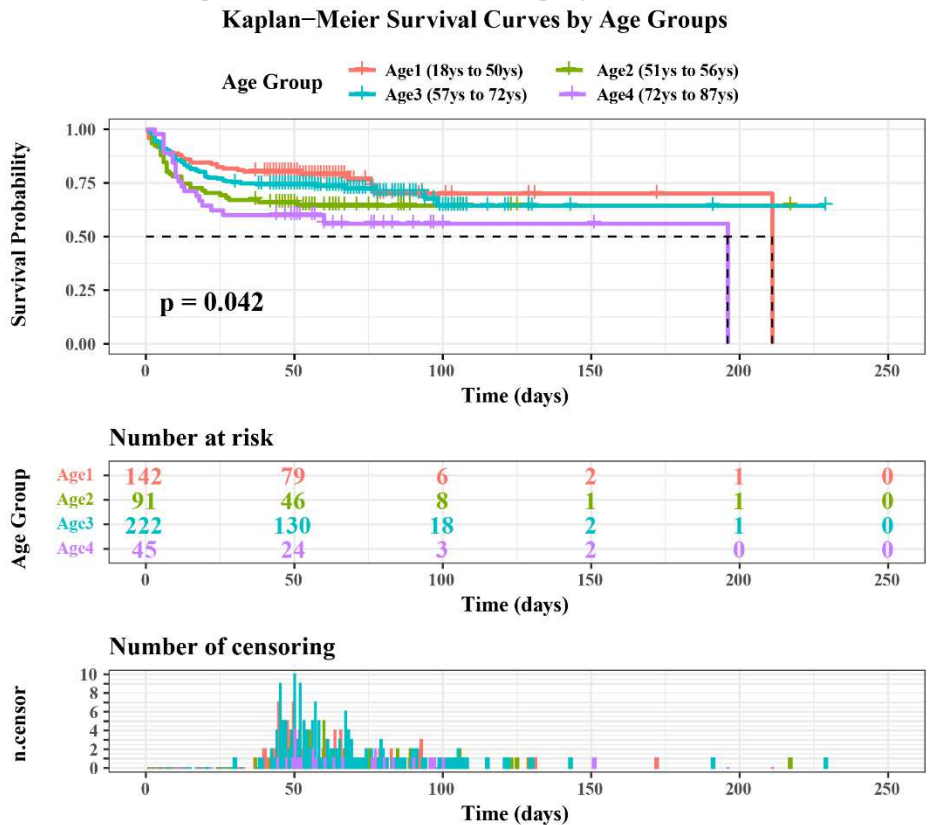


Fig.2 Kaplan-Meier survival curves stratified by age groups, illustrating the survival probability over time for patients in different age groups, accompanied by the number-at-risk tables at each time point and the censoring data distribution plot.

Survival Outcomes in different age groups

Patients were stratified into four age groups: Age1 (18 – 50 years, n=142), Age2 (51 – 56 years, n=91), Age3 (57 – 72 years, n=222), and Age4 (72 – 87 years, n=45). Kaplan-Meier survival curve analysis was performed to compare survival outcomes across age groups in poor-grade aSAH patients (Fig.2). The results demonstrated that age exerted a nonlinear threshold effect on prognosis, with 72 years identified as a critical cutoff point—patients beyond this age

exhibited significantly worse survival, whereas differences among groups aged <72 years were relatively modest. Subgroup analyses using age groups were performed with age1 (18 – 50 years) as the reference group to evaluate hazard ratios (HR) for mortality across different age strata. The results demonstrated that only Age4 (≥ 72 years) showed a statistically significant increase in mortality risk (HR 2.01, 95%CI: 1.15 – 3.51), consistent with findings from Kaplan-Meier survival curves and restricted cubic spline regression analyses, further confirming 72 years as a critical prognostic threshold for poor-grade aSAH patients.

Two significant effect modifiers were identified: cerebral herniation (P=0.013) and delayed cerebral infarction (DCI, P=0.032) (table S4). As a strong adverse prognostic factor, cerebral herniation may lead to uniformly poor outcomes across all age groups, thereby attenuating the discriminative value of age stratification; in patients without cerebral herniation, age emerged as a more important prognostic factor, with Age4 group showing significantly elevated mortality risk (HR 3.02, 95%CI: 1.55 – 5.89). Similarly, the presence of DCI resulted in poor prognosis across all age groups, diluting the age effect; conversely, in patients without DCI, age stratification demonstrated reater prognostic value, with Age4 group showing nearly a 5-fold increase in mortality risk (HR 4.75, 95%CI: 2.05 – 11.02).

Additionally, Fisher grade showed a marginally significant effect modification (P=0.055) (table S4). In patients with Fisher grade III, Age2 (51 – 56 years) demonstrated an abnormally high mortality risk (HR 4.91, 95%CI: 1.77 – 13.58), possibly attributable to the small sample size in this subgroup (n=16) or potential selection bias; whereas in patients with Fisher grade IV, the age effect was masked by the severity of hemorrhage, with no significant differences observed among age groups (table S4).

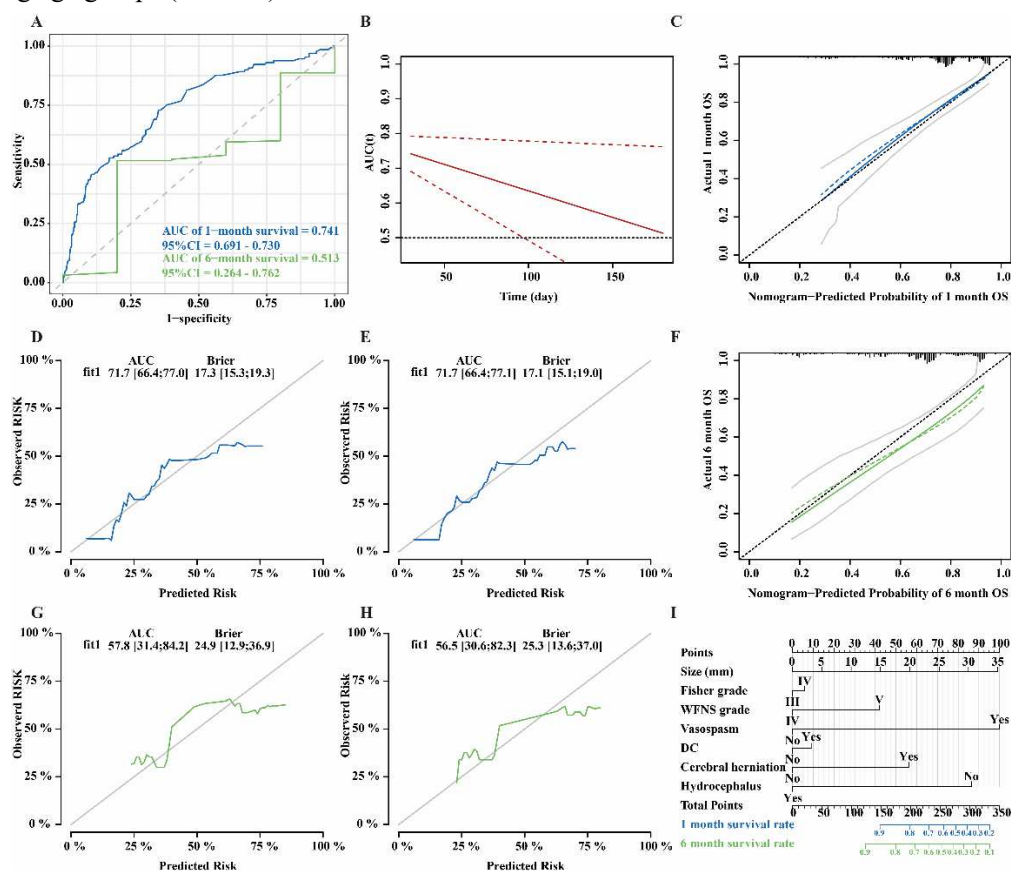


Fig.3 A comprehensive analysis of nomogram prognostic model construction and validation,

including ROC curve (A), time-dependent AUC curve (B), calibration curve for 1-month survival (C), AUC and Brier score for training and validation sets (D, E), calibration curve for 6-month survival (F), AUC and Brier score for training and validation sets (G, H), and the nomogram (I).

Analysis of survival prediction model

This study developed a survival prediction model for patients with PGASAH based on seven clinical variables: aneurysm size, Fisher grade, WFNS grade, cerebral vasospasm, DC, cerebral herniation, and hydrocephalus.

The 1-month survival prediction model demonstrated good performance. The AUC was 0.741 (95%CI: 0.691-0.730), exceeding the acceptable threshold of 0.70, indicating satisfactory discriminative ability (Fig.3 A). Bootstrap internal validation revealed good calibration with consistent predicted and observed probabilities (Fig.3 B,C). The internal training set showed an AUC of 71.7% with a Brier score of 17.3%, while the external validation set achieved an AUC of 71.7% with a Brier score of 17.1% (Fig.3 D,E). The highly consistent results between internal and external validation indicate excellent generalizability.

In contrast, the 6-month survival prediction model performed poorly. The AUC was only 0.513 (95%CI: 0.264-0.762) (Fig.3 A), approaching random chance (0.5), with wide confidence intervals suggesting limited stability. Calibration curves revealed a systematic tendency to overestimate survival probabilities, with limited improvement after correction (Fig.3 F). External validation yielded an AUC of 56.5% and a Brier score of 25.3% (Fig.3 G,H), comparable to the internal training set results, indicating consistently poor performance and insufficient reliability.

In conclusion, the nomogram tool based on these seven predictors is more suitable for short-term (1-month) survival prediction, while its long-term (6-month) predictive accuracy is limited and requires cautious clinical interpretation (Fig.3 I).

Discussion

The study cohort comprised 500 PGASAH patients exhibiting notably elevated postoperative complication rates, including pneumonia, DCI, intracranial infection, brain herniation, and hydrocephalus. These findings highlight the critical clinical status, therapeutic challenges, and management complexity inherent to PGASAH patients. The substantial complication burden not only poses immediate life-threatening risks but also functions as a critical mediator of unfavorable outcomes. Multivariate Cox regression modeling delineated six independent prognostic indicators: patient age, maximal aneurysm diameter, WFNS grade V classification, cerebral vasospasm, brain herniation, and absence of hydrocephalus. As corroborated by existing literature [11, 27-29], these variables collectively form the essential parameter set for survival prediction in PGASAH patients.

Determination of Independent Prognostic Factors

Among the various risk factors associated with aSAH, cerebral vasospasm stands out as the most significant, increasing the risk of mortality by more than fourfold. The pathophysiological basis of this complication lies in the release of vasoactive agents, such as oxyhemoglobin, during the degradation of subarachnoid blood clots. These substances promote prolonged vasoconstriction by scavenging nitric oxide and upregulating endothelin-1, ultimately contributing to the development of DCI[30, 31]. Studies further indicate that DCI is not solely the result of macrovascular spasm but rather a complex process involving multiple mechanisms, including microcirculatory dysfunction, cortical spreading depolarization, and neurovascular uncoupling. As

a critical determinant of post-aSAH outcomes, DCI remains the leading cause of mortality and long-term disability in affected patients.[14, 32].

Hydrocephalus exhibited a paradoxical protective effect (HR: 0.28) with significant clinical relevance. This phenomenon appears attributable to two key factors: Postoperative hydrocephalus was the exclusive patient population in this study. Early detection and cerebrospinal fluid drainage management during hospitalization disrupted the escalating intracranial pressure cycle, creating crucial recovery windows for primary brain injuries[18, 33]. Additionally, survivor bias might affect outcomes, as only patients with sufficient physiological reserves to endure secondary ventriculoperitoneal shunting and long-term follow-up were included post-discharge. These findings highlight both the therapeutic value of timely CSF diversion and methodological considerations in postoperative hydrocephalus research. The findings underscore the critical role of prompt diagnosis and proactive management of hydrocephalus, while calling attention to the necessity of rigorously assessing the impact of therapeutic interventions on risk factors and accounting for potential survivor bias in observational studies.

The survival risk factors in patients with PGASAH form a complex and interconnected pathophysiological network. The severity of the initial hemorrhage, as indicated by the Fisher and WFNS grades, serves as the primary prognostic determinant; extensive bleeding leads to a rapid increase in intracranial pressure, a sharp decline in cerebral perfusion pressure, and consequently triggers global cerebral ischemia and early brain injury[34]. Superimposed on this primary insult, cerebral vasospasm and DCI act as secondary contributors, further compromising neurological function through microcirculatory dysfunction and uncoupling of neurovascular regulation[31]. Additionally, acute hydrocephalus and refractory intracranial hypertension may necessitate DC, which, although life-saving, carries an inherent risk of brain herniation. Importantly, while DC is an effective intervention for reducing mortality, it also reflects the severity of illness and independently predicts poor clinical outcomes[15, 35, 36].

Moreover, while pneumonia itself did not act as an independent life-threatening risk factor in this study, its high incidence rate of 82.6% highlights the significant clinical susceptibility of PGASAH patients[37]. The development of pneumonia is mainly attributed to the loss of airway protection caused by coma and an increased risk of aspiration, which has been strongly linked to higher overall mortality and serves as a crucial mediator influencing patient outcomes[18]. The timing and approach of treatment critically affect survival: ultra-early intervention (within 24 hours) can notably enhance long-term prognosis, whereas delayed treatment not only raises the likelihood of rebleeding but also opens a window for DCI, thereby creating a harmful cycle of secondary complications[37, 38]. However, this study failed to replicate such findings.

Nonlinear Characteristics and Threshold Effects of Age

Among the PGASAH patients analyzed in this study, the age2 cohort (51 – 56 years) exhibited a higher mortality risk than the age3 group (57 – 72 years). Pathophysiologically, age operates as a continuous variable, with younger individuals demonstrating increased susceptibility to vasospasm. Supporting this, a meta-analysis of 23 studies involving 8,843 patients showed that for each additional year of age, the risk of radiographic vasospasm (RVS) decreased [39]. The age2 group is characterized by greater vascular reactivity, which elevates RVS risk [21]. Additionally, this group experiences less cerebral atrophy, reduced brain compliance, and limited intracranial compensatory capacity, leading to more pronounced acute intracranial pressure increases[21]. Regarding clinical selection bias, clinicians often favor aggressive interventions for

younger patients (18 – 55 years) with PGASAH, even in cases of extreme clinical severity. In contrast, some of the most critically ill individuals in the oldest age group may instead be shifted to palliative care, effectively filtering the older cohort into a subgroup with comparatively better prognosis. From a statistical perspective, the age2 group featured a limited sample size ($n = 16$), which could have magnified the influence of random error. Altogether, this paradoxical observation likely stems from a combination of increased vasospasm susceptibility, diminished intracranial compensatory capacity, clinical selection bias, and the distorting impact of small sample sizes.

Subgroup analysis demonstrated that age exhibits a nonlinear relationship with prognosis in PGASAH patients. A critical cutoff value of 72 years was identified—those above this age showed a significantly elevated mortality risk ($HR=2.01$), whereas younger patients displayed only minimal differences in outcomes. These findings contradict the previously assumed linear association of "older age, worse prognosis" and instead suggest the presence of an age threshold effect in PGASAH[17, 27, 40]. Consistent with recent reports, patients aged over 65 generally experience poorer outcomes, and mortality rates are markedly higher among those above 70 within the first year post-event [2, 4, 25, 41]. Pathophysiologically, 72 years emerges as a pivotal threshold in cerebral reserve and systemic compensatory capacity decline. At this threshold age, patients demonstrate marked deterioration in three critical domains: tolerance to initial hemorrhagic insult, capacity for secondary brain injury repair, and resilience against complications. This study's clinical significance centers on two key contributions: defining 72 years as the critical age boundary for PGASAH prognostic stratification, and elucidating the modifying roles of brain herniation and delayed cerebral ischemia (DCI) in age-related outcomes, thereby informing precision medicine approaches.

Identification of Effect Modifiers and Clinical Stratification

This study identified two significant effect modifiers—brain herniation and DCI—revealing complex interaction patterns among prognostic factors. In patients without brain herniation or DCI, age stratification value was prominent, with mortality risk in elderly groups increasing 3-fold and nearly 5-fold, respectively; whereas in patients with these severe complications, prognosis was extremely poor across all age groups, and the age effect was "diluted." This finding has dual clinical significance: first, for patients without brain herniation and DCI, age should serve as an important consideration for individualized treatment decision-making, with elderly patients requiring more intensive monitoring and more aggressive complication prevention; second, for patients who have already developed severe complications, emergency intervention is required regardless of age, and age should not be a limiting factor for aggressive treatment. Additionally, the prognostic value of aneurysm size is modified by sex, hypertension, and DC status, while the predictive value of Fisher grade is highly dependent on treatment modality. These interaction effects collectively indicate that PGASAH risk assessment must abandon single-factor thinking and construct a multidimensional evaluation system integrating patient demographic characteristics, comorbidities, and treatment modalities.

Construction, Validation, and Clinical Application of Prediction Models

Based on independent prognostic factors, this study constructed nomogram prediction models for 1-month and 6-month survival. The 1-month model demonstrated excellent performance: AUC reached 0.741, with 500-fold Bootstrap internal validation and external validation both showing good discriminative ability and calibration, Brier scores controlled around 17%, and stable

generalization capability. This performance is comparable to previous aSAH prediction models and meets the basic requirements for clinical auxiliary decision-making tools[42].

However, the 6-month model performed poorly, with an AUC of only 0.513—near random chance—and a systematic tendency to overestimate survival probability. Previous literature has reported rising mortality at 6 months[11], a finding that contradicts our results. This discrepancy stems from multiple factors. First, 6-month outcomes are influenced by post-acute variables such as rehabilitation intensity, adherence to secondary prevention, family and social support, and fatal comorbidities in other organs—none of which were included in the model. Second, PGASAH patients have high early mortality, and long-term survivors exhibit significant selection bias—patients who can survive to 6 months inherently possess better baseline conditions and recovery potential[24]. Third, the sample size was relatively insufficient for long-term event prediction, resulting in poor model stability and wide confidence intervals. The limitations of the 6-month model highlight a common challenge in predictive model development: whereas short-term prediction can rely on acute-phase clinical variables, long-term prediction must integrate complex factors encompassing functional recovery, complication management, and chronic disease care.

Study Limitations

The single-center retrospective design may introduce selection bias, affecting result generalizability; the 6-month prediction model performed poorly, and long-term prognosis prediction tools require further development; detailed recording of specific timing and intensity of therapeutic interventions was lacking, potentially omitting important prognostic modifier information.

Expand sample size to improve statistical power for long-term events; incorporate functional outcome indicators (such as mRS score trajectory) as intermediate variables; develop dynamic prediction models that continuously update prognosis probabilities during the treatment course; integrate imaging biomarkers and molecular biological indicators to enhance the biological basis of prediction; conduct prospective multicenter studies to validate prediction models; and conduct randomized controlled trials to optimize individualized treatment strategies for PGASAH.

Conclusion

In summary, prognostic assessment of PGASAH requires comprehensive consideration of demographic characteristics, disease severity, and complication status. The short-term prediction model constructed in this study can provide reliable support for clinical decision-making; however, long-term prognosis prediction still requires optimization through the integration of more functional recovery factors. The identification of the 72-year age threshold and the effect-modifying roles of brain herniation and DCI facilitates more precise risk stratification and individualized treatment. Future research efforts should focus on multicenter collaboration and the development of dynamic prediction models to further advance the precision medicine level for PGASAH.

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Table S1 Cox regression subgroup analysis stratified by multiple factors, using aneurysmal size (< 5 mm vs. ≥ 5 mm) as the exposure variable.

Variable	Count	Percent		HR (95% CI)	P value	P for interaction
Overall	500	100		1.29 (0.93 to 1.79)	0.122	
Sex						0.034
Male	223	44.6		0.88 (0.55 to 1.41)	0.600	
Female	277	55.4		1.86 (1.17 to 2.96)	0.009	
Age group						0.714
<60 ys	188	37.6		1.40 (0.85 to 2.31)	0.183	
≥60 ys	312	62.4		1.24 (0.81 to 1.90)	0.330	
Neck width (mm)						0.357
≥4	242	48.4		1.26 (0.76 to 2.08)	0.373	
<4	258	51.6		0.83 (0.44 to 1.56)	0.560	
Smoking						0.125
No	353	70.6		1.57 (1.05 to 2.33)	0.026	
Yes	147	29.4		0.85 (0.47 to 1.56)	0.605	
Alcohol abuse						0.202
No	432	86.4		1.43 (1.00 to 2.04)	0.047	
Yes	68	13.6		0.75 (0.31 to 1.80)	0.524	
Hyperlipidemia						0.375
No	416	83.2		1.23 (0.86 to 1.75)	0.252	
Yes	84	16.8		1.82 (0.80 to 4.16)	0.155	
Diabetes						0.276
No	469	93.8		1.38 (0.99 to 1.92)	0.057	
Yes	31	6.2		0.41 (0.07 to 2.46)	0.330	
Hypertension						0.030
No	108	21.6		2.78 (1.32 to 5.82)	0.007	
Yes	392	78.4		1.07 (0.75 to 1.54)	0.713	
Timing						0.301
<24h	230	46.0		1.03 (0.65 to 1.61)	0.913	
24h–72h	131	26.2		1.66 (0.83 to 3.35)	0.154	
>72h	139	27.8		1.44 (0.76 to 2.76)	0.264	
Location of aneurysm						0.266
Anterior	447	89.4		1.24 (0.89 to 1.74)	0.207	
Posterior	53	10.6		2.46 (0.65 to 9.38)	0.186	
Fisher grade						0.635
III	128	25.6		1.50 (0.70 to 3.19)	0.294	
IV	372	74.4		1.23 (0.86 to 1.76)	0.266	
WFNS grade						0.199
IV	314	62.8		1.47 (0.91 to 2.39)	0.120	
V	186	37.2		0.98 (0.63 to 1.52)	0.930	
Treatment method						0.381
Clipping	232	46.4		1.08 (0.66 to 1.74)	0.767	
Coiling	268	53.6		1.49 (0.95 to 2.33)	0.082	
Vasospasm						0.152
No	73	14.6		0.30 (0.03 to 2.53)	0.267	
Yes	427	85.4		1.34 (0.96 to 1.87)	0.083	
LD						0.486
No	402	80.4		1.36 (0.93 to 1.97)	0.110	
Yes	98	19.6		0.96 (0.48 to 1.90)	0.896	
DC						0.030
No	341	68.2		1.71 (1.12 to 2.60)	0.012	
Yes	159	31.8		0.77 (0.46 to 1.32)	0.345	
Pneumonia						0.770
No	87	17.4		1.39 (0.68 to 2.85)	0.369	
Yes	413	82.6		1.30 (0.90 to 1.87)	0.165	
Intracranial infection						0.387
No	368	73.6		1.41 (0.97 to 2.07)	0.075	
Yes	132	26.4		1.00 (0.53 to 1.89)	0.994	
Cerebral herniation						0.340
No	431	86.2		1.30 (0.88 to 1.91)	0.180	
Yes	69	13.8		0.98 (0.53 to 1.79)	0.938	
DCI						0.785
No	209	41.8		1.35 (0.83 to 2.19)	0.231	
Yes	291	58.2		1.27 (0.82 to 1.97)	0.283	
Epilepsy						0.492
No	488	97.6		1.31 (0.95 to 1.82)	0.104	
Yes	12	2.4		0.53 (0.05 to 5.86)	0.605	
Hydrocephalus						0.989
No	416	83.2		1.31 (0.93 to 1.85)	0.116	
Yes	84	16.8		1.33 (0.46 to 3.85)	0.597	

Abbreviation: HR, Hazard Ratio; IQR, interquartile range; LD: continuous lumbar drainage; DC:

decompressive craniectomy; DCI: delayed cerebral infarction.

Table S2 Cox regression subgroup analysis stratified by multiple factors, using fisher grade as the exposure variable.

Variable	Count	Percent		HR (95% CI)	P value	P for interaction
Overall	500	100		1.53 (1.01 to 2.32)	0.047	
Sex						0.428
Male	223	44.6		1.82 (0.95 to 3.47)	0.069	
Female	277	55.4		1.32 (0.76 to 2.29)	0.326	
Age group						0.569
<60	188	37.6		1.33 (0.72 to 2.45)	0.361	
≥60	312	62.4		1.72 (0.97 to 3.06)	0.064	
Size (mm)						0.347
≥5	305	61.0		1.30 (0.78 to 2.14)	0.312	
<5	195	39.0		2.04 (0.95 to 4.35)	0.067	
Neck width (mm)						0.407
≥4	242	48.4		1.32 (0.77 to 2.25)	0.312	
<4	258	51.6		1.86 (0.94 to 3.66)	0.074	
Smoking						0.720
No	353	70.6		1.47 (0.90 to 2.41)	0.120	
Yes	147	29.4		1.67 (0.75 to 3.76)	0.212	
Alcohol abuse						0.777
No	432	86.4		1.50 (0.97 to 2.33)	0.072	
Yes	68	13.6		1.84 (0.43 to 7.88)	0.413	
Hyperlipidemia						0.070
No	416	83.2		1.87 (1.15 to 3.05)	0.012	
Yes	84	16.8		0.61 (0.25 to 1.48)	0.274	
Diabetes						0.732
No	469	93.8		1.58 (1.03 to 2.42)	0.035	
Yes	31	6.2		0.81 (0.09 to 7.30)	0.848	
Hypertension						0.669
No	108	21.6		1.87 (0.71 to 4.91)	0.203	
Yes	392	78.4		1.45 (0.91 to 2.31)	0.115	
Timing						0.341
<24h	230	46.0		1.66 (0.89 to 3.08)	0.108	
24h–72h	131	26.2		0.89 (0.41 to 1.93)	0.771	
>72h	139	27.8		2.20 (0.92 to 5.28)	0.077	
Location of aneurysm						0.618
Anterior	447	89.4		1.47 (0.95 to 2.28)	0.081	
Posterior	53	10.6		2.10 (0.45 to 9.80)	0.344	
WFNS grade						0.975
IV	314	62.8		1.15 (0.68 to 1.95)	0.603	
V	186	37.2		1.16 (0.53 to 2.51)	0.714	
Treatment method						0.017
Clipping	232	46.4		4.06 (1.48 to 11.18)	0.007	
Coiling	268	53.6		1.04 (0.64 to 1.70)	0.865	
Vasospasm						0.523
No	73	14.6		0.83 (0.17 to 4.12)	0.821	
Yes	427	85.4		1.38 (0.89 to 2.14)	0.156	
LD						0.980
No	402	80.4		1.52 (0.96 to 2.43)	0.077	
Yes	98	19.6		1.49 (0.58 to 3.86)	0.410	
DC						0.193
No	341	68.2		1.24 (0.78 to 1.99)	0.363	
Yes	159	31.8		2.74 (0.86 to 8.78)	0.090	
Pneumonia						0.716
No	87	17.4		1.40 (0.65 to 3.02)	0.386	
Yes	413	82.6		1.72 (1.03 to 2.88)	0.039	
Intracranial infection						0.997
No	368	73.6		1.56 (0.97 to 2.49)	0.065	
Yes	132	26.4		1.53 (0.60 to 3.93)	0.375	
Cerebral herniation						0.339
No	431	86.2		1.40 (0.87 to 2.24)	0.167	
Yes	69	13.8		0.87 (0.34 to 2.21)	0.765	
DCI						0.225
No	209	41.8		2.02 (1.08 to 3.78)	0.029	
Yes	291	58.2		1.22 (0.70 to 2.14)	0.485	
Epilepsy						0.586
No	488	97.6		1.56 (1.02 to 2.38)	0.042	
Yes	12	2.4		0.87 (0.08 to 9.58)	0.906	
Hydrocephalus						0.880
No	416	83.2		1.69 (1.10 to 2.61)	0.016	
Yes	84	16.8		1.44 (0.19 to 11.02)	0.725	

0 0.5 1 1.5 2 2.5 3

Abbreviation: HR, Hazard Ratio; IQR, interquartile range; LD: continuous lumbar drainage; DC:

decompressive craniectomy; DCI: delayed cerebral infarction.

Table S3 Cox regression subgroup analysis stratified by multiple factors, using WFNS grade as the exposure variable.

Variable	Count	Percent		HR (95% CI)	P value	P for interaction
Overall	500	100		1.53 (1.01 to 2.32)	0.047	
Sex						0.428
Male	223	44.6		1.82 (0.95 to 3.47)	0.069	
Female	277	55.4		1.32 (0.76 to 2.29)	0.326	
Age group						0.569
<60	188	37.6		1.33 (0.72 to 2.45)	0.361	
≥60	312	62.4		1.72 (0.97 to 3.06)	0.064	
Size (mm)						0.347
≥5	305	61.0		1.30 (0.78 to 2.14)	0.312	
<5	195	39.0		2.04 (0.95 to 4.35)	0.067	
Neck width (mm)						0.407
≥4	242	48.4		1.32 (0.77 to 2.25)	0.312	
<4	258	51.6		1.86 (0.94 to 3.66)	0.074	
Smoking						0.720
No	353	70.6		1.47 (0.90 to 2.41)	0.120	
Yes	147	29.4		1.67 (0.75 to 3.76)	0.212	
Alcohol abuse						0.777
No	432	86.4		1.50 (0.97 to 2.33)	0.072	
Yes	68	13.6		1.84 (0.43 to 7.88)	0.413	
Hyperlipidemia						0.070
No	416	83.2		1.87 (1.15 to 3.05)	0.012	
Yes	84	16.8		0.61 (0.25 to 1.48)	0.274	
Diabetes						0.732
No	469	93.8		1.58 (1.03 to 2.42)	0.035	
Yes	31	6.2		0.81 (0.09 to 7.30)	0.848	
Hypertension						0.669
No	108	21.6		1.87 (0.71 to 4.91)	0.203	
Yes	392	78.4		1.45 (0.91 to 2.31)	0.115	
Timing						0.341
<24h	230	46.0		1.66 (0.89 to 3.08)	0.108	
24h–72h	131	26.2		0.89 (0.41 to 1.93)	0.771	
>72h	139	27.8		2.20 (0.92 to 5.28)	0.077	
Location of aneurysm						0.618
Anterior	447	89.4		1.47 (0.95 to 2.28)	0.081	
Posterior	53	10.6		2.10 (0.45 to 9.80)	0.344	
WFNS grade						0.975
IV	314	62.8		1.15 (0.68 to 1.95)	0.603	
V	186	37.2		1.16 (0.53 to 2.51)	0.714	
Treatment method						0.017
Clipping	232	46.4		4.06 (1.48 to 11.18)	0.007	
Coiling	268	53.6		1.04 (0.64 to 1.70)	0.865	
Vasospasm						0.523
No	73	14.6		0.83 (0.17 to 4.12)	0.821	
Yes	427	85.4		1.38 (0.89 to 2.14)	0.156	
LD						0.980
No	402	80.4		1.52 (0.96 to 2.43)	0.077	
Yes	98	19.6		1.49 (0.58 to 3.86)	0.410	
DC						0.193
No	341	68.2		1.24 (0.78 to 1.99)	0.363	
Yes	159	31.8		2.74 (0.86 to 8.78)	0.090	
Pneumonia						0.716
No	87	17.4		1.40 (0.65 to 3.02)	0.386	
Yes	413	82.6		1.72 (1.03 to 2.88)	0.039	
Intracranial infection						0.997
No	368	73.6		1.56 (0.97 to 2.49)	0.065	
Yes	132	26.4		1.53 (0.60 to 3.93)	0.375	
Cerebral herniation						0.339
No	431	86.2		1.40 (0.87 to 2.24)	0.167	
Yes	69	13.8		0.87 (0.34 to 2.21)	0.765	
DCI						0.225
No	209	41.8		2.02 (1.08 to 3.78)	0.029	
Yes	291	58.2		1.22 (0.70 to 2.14)	0.485	
Epilepsy						0.586
No	488	97.6		1.56 (1.02 to 2.38)	0.042	
Yes	12	2.4		0.87 (0.08 to 9.58)	0.906	
Hydrocephalus						0.880
No	416	83.2		1.69 (1.10 to 2.61)	0.016	
Yes	84	16.8		1.44 (0.19 to 11.02)	0.725	

Abbreviation: HR, Hazard Ratio; IQR, interquartile range; LD: continuous lumbar drainage; DC:

decompressive craniectomy; DCI: delayed cerebral infarction.

Table S4 Cox regression subgroup analysis stratified by multiple factors, using age groups: Age1 (18–50 years), Age2 (51–56 years), Age3 (57–72 years), and Age4 (72–87 years) as the exposure variable.

Subgroup	Age1		Age2		Age3		Age4		HR .95 CI (Age2 vs. Age1)	HR .95 CI (Age3 vs. Age1)	HR .95 CI (Age4 vs. Age1)	Group (Age2 vs. Age1) Group (Age3 vs. Age1) Group (Age4 vs. Age1)	P.value.for.interaction
	Count	total	Count	total	Count	total	Count	total					
All	33	142	32	91	62	222	20	45	1.61 (0.99 ~ 2.63)	1.20 (0.79 ~ 1.83)	2.01 (1.15 ~ 3.51)		
Sex													
Male	23	95	20	47	22	89	6	12	1.91 (1.04 ~ 3.48)	1.31 (0.73 ~ 2.35)	2.04 (0.82 ~ 5.10)		0.910
Female	10	47	12	44	40	153	14	33	1.39 (0.60 ~ 3.24)	1.28 (0.64 ~ 2.56)	2.22 (0.98 ~ 5.03)		
Smoking													
No	18	80	23	64	45	171	16	38	1.82 (0.98 ~ 3.38)	1.21 (0.70 ~ 2.09)	2.08 (1.06 ~ 4.09)		0.844
Yes	15	62	9	27	17	51	4	7	1.33 (0.58 ~ 3.05)	1.34 (0.67 ~ 2.68)	2.03 (0.64 ~ 6.39)		
Alcohol abuse													
No	29	121	26	74	52	197	18	40	1.57 (0.92 ~ 2.67)	1.10 (0.70 ~ 1.73)	1.96 (1.09 ~ 3.53)		0.678
Yes	4	21	6	17	10	25	2	5	2.00 (0.56 ~ 7.11)	2.18 (0.68 ~ 6.94)	2.65 (0.48 ~ 14.58)		
Hyperlipidemia													
No	29	118	27	71	49	185	19	42	1.66 (0.98 ~ 2.80)	1.06 (0.67 ~ 1.67)	1.91 (1.07 ~ 3.41)		0.339
Yes	4	24	5	20	13	37	1	3	1.58 (0.42 ~ 5.92)	2.47 (0.80 ~ 7.58)	2.95 (0.33 ~ 26.70)		
Diabetes													
No	33	139	29	84	60	208	19	38	1.56 (0.95 ~ 2.57)	1.22 (0.80 ~ 1.86)	2.36 (1.34 ~ 4.15)		0.305
Yes	0	3	3	7	2	14	1	7	1151942854.67 (0 ~ Inf)	350155606.17 (0 ~ Inf)	1.00 (0.00 ~ Inf)		
Hypertension													
No	9	38	4	21	10	38	6	11	0.78 (0.24 ~ 2.55)	1.06 (0.43 ~ 2.61)	2.89 (0.96 ~ 7.58)		0.338
Yes	24	104	28	70	52	184	14	34	1.91 (1.10 ~ 3.29)	1.25 (0.77 ~ 2.02)	1.84 (0.95 ~ 3.56)		
Size (mm)													
≥5	18	81	21	62	23	112	9	19	1.57 (0.84 ~ 2.95)	0.87 (0.47 ~ 1.62)	2.21 (0.99 ~ 4.93)		0.425
<5	15	61	11	29	39	110	11	26	1.85 (0.85 ~ 4.04)	1.54 (0.85 ~ 2.80)	1.88 (0.86 ~ 4.11)		
Neck width (mm)													
<4	17	80	19	53	20	107	7	18	1.84 (0.95 ~ 3.54)	0.87 (0.46 ~ 1.66)	1.94 (0.80 ~ 4.68)		0.336
≥4	16	62	13	38	42	115	13	27	1.37 (0.66 ~ 2.85)	1.43 (0.80 ~ 2.54)	1.90 (0.91 ~ 3.98)		
Timing													
<24h	20	71	16	47	31	96	9	16	1.27 (0.66 ~ 2.45)	1.10 (0.63 ~ 1.93)	2.23 (1.01 ~ 4.91)		0.879
24h~72h	6	37	7	17	15	63	6	14	3.01 (1.01 ~ 8.96)	1.43 (0.56 ~ 3.71)	2.12 (0.64 ~ 7.02)		
>72h	7	34	9	27	16	63	5	15	1.80 (0.67 ~ 4.85)	1.32 (0.54 ~ 3.22)	1.74 (0.55 ~ 5.49)		
Location of aneurysm													
Anterior	29	126	31	88	59	197	16	36	1.63 (0.98 ~ 2.70)	1.29 (0.83 ~ 2.02)	2.09 (1.13 ~ 3.85)		0.625
Posterior	4	16	1	3	3	25	4	9	1.47 (0.16 ~ 13.18)	0.50 (0.11 ~ 2.25)	1.36 (0.30 ~ 6.10)		
Fisher grade													
III	7	49	8	16	10	55	2	8	4.91 (1.77 ~ 13.58)	1.25 (0.48 ~ 3.30)	1.83 (0.38 ~ 8.80)		0.055
IV	26	93	24	75	52	167	18	37	1.15 (0.66 ~ 2.00)	1.11 (0.70 ~ 1.78)	1.83 (1.00 ~ 3.34)		
WFNS													
IV	14	94	17	53	26	141	9	26	2.35 (1.15 ~ 4.78)	1.22 (0.64 ~ 2.34)	2.21 (0.95 ~ 5.15)		0.313
V	19	48	15	38	36	81	11	19	1.03 (0.52 ~ 2.05)	1.16 (0.66 ~ 2.04)	1.66 (0.78 ~ 3.53)		
Treatment method													
Clipping	18	75	16	52	31	95	2	10	1.28 (0.65 ~ 2.52)	1.31 (0.73 ~ 2.34)	0.73 (0.17 ~ 3.15)		0.115
Coiling	15	67	16	39	31	127	18	35	2.28 (1.12 ~ 4.64)	1.16 (0.62 ~ 2.15)	2.70 (1.35 ~ 5.38)		
Vasospasm													
No	0	25	2	11	4	34	0	3	650541352.29 (0 ~ Inf)	39812995.10 (0 ~ Inf)	1.00 (0.00 ~ Inf)		0.129
Yes	33	117	30	80	58	188	20	42	1.40 (0.85 ~ 2.29)	1.08 (0.70 ~ 1.65)	1.75 (1.00 ~ 3.05)		
Drainage													
No	22	115	25	77	47	169	17	41	1.82 (1.03 ~ 3.23)	1.49 (0.90 ~ 2.48)	2.33 (1.24 ~ 4.39)		0.320
Yes	11	27	7	14	15	53	3	4	1.55 (0.59 ~ 4.03)	0.65 (0.30 ~ 1.42)	1.53 (0.41 ~ 5.70)		
DC													
No	16	88	18	51	37	163	18	39	2.12 (1.08 ~ 4.15)	1.24 (0.69 ~ 2.22)	2.83 (1.44 ~ 5.56)		0.167
Yes	17	54	14	40	25	59	2	6	1.28 (0.62 ~ 2.64)	1.52 (0.81 ~ 2.86)	0.79 (0.17 ~ 3.64)		
Pneumonia													
No	8	33	6	17	14	34	2	3	1.37 (0.47 ~ 4.02)	1.68 (0.70 ~ 4.00)	1.66 (0.31 ~ 8.88)		0.608
Yes	25	109	26	74	48	188	18	42	1.70 (0.98 ~ 2.95)	1.12 (0.69 ~ 1.82)	2.11 (1.15 ~ 3.88)		
Intracranial infection													
No	24	95	23	61	45	173	16	39	1.59 (0.90 ~ 2.82)	0.99 (0.60 ~ 1.63)	1.54 (0.81 ~ 2.91)		0.190
Yes	9	47	9	30	17	48	4	6	1.67 (0.66 ~ 4.22)	1.95 (0.87 ~ 4.41)	5.80 (1.76 ~ 19.04)		
Cerebral herniation													
No	19	124	26	80	43	190	16	37	2.38 (1.32 ~ 4.31)	1.48 (0.86 ~ 2.54)	3.02 (1.55 ~ 5.89)		0.013
Yes	14	18	6	11	19	32	4	8	0.56 (0.21 ~ 1.48)	0.67 (0.33 ~ 1.36)	0.52 (0.17 ~ 1.61)		
DCI													
No	11	68	15	37	29	87	11	17	2.93 (1.34 ~ 6.40)	2.23 (1.11 ~ 4.46)	4.75 (2.05 ~ 11.02)		0.032
Yes	22	74	17	54	33	135	9	28	1.06 (0.58 ~ 1.99)	0.77 (0.45 ~ 1.32)	1.05 (0.48 ~ 2.27)		
Epilepsy													
No	32	137	30	87	62	219	20	45	1.59 (0.96 ~ 2.61)	1.22 (0.79 ~ 1.87)	2.01 (1.15 ~ 3.52)		0.397
Yes	1	5	2	4	0	3	0	0	2.38 (0.21 ~ 26.32)	0.00 (0.00 ~ Inf)			
Hydrocephalus													
No	29	127	31	76	58	184	15	29	1.98 (1.19 ~ 3.28)	1.39 (0.89 ~ 2.17)	2.44 (1.31 ~ 4.57)		0.161
Yes	4	15	1	15	4	38	5	16	0.24 (0.03 ~ 2.14)	0.39 (0.10 ~ 1.57)	1.28 (0.34 ~ 4.76)		

Abbreviation: HR, Hazard Ratio; IQR, interquartile range; LD: continuous lumbar drainage; DC: decompressive craniectomy; DCI: delayed cerebral infarction.