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Quantitative electroencephalography predicts delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage: a retrospective study

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ABSTRACT

Purpose: Delayed cerebral ischemia (DCI) is a common complication that occurs in aneurysmal subarachnoid hemorrhage (aSAH). This complication can lead to clinical deterioration and poor prognosis. The aim of this study is to explore the risk factors for DCI in aSAH patients in neurological ICU, develop a nomogram including quantitative electroencephalography (qEEG) parameters, and evaluate its performance.

Methods: We retrospectively analyzed and processed Severe aneurysmal subarachnoid hemorrhage (SaSAH) patients from June 2022 to May 2024 who underwent bedside qEEG monitoring and analyzed the qEEG indices, brain CT, and clinical data of these patients. Logistic multivariate regression analysis was employed to identify the independent risk factors of DCI. A clinical prediction model in the form of a nomogram for DCI was developed using the R programming language and subsequently evaluated for its performance and quality.

Results: A total of 145 patients with SaSAH were included in the analysis, comprising 101 patients in the training set and 44 patients in the validation set. 77 patients (53.10 %) developed DCI. Multivariate regression analysis revealed that GCS, modified Fisher grade, hypothermia, alpha/delta ratio (ADR) and PAV grade were independent risk factors for DCI. The nomogram exhibited excellent discriminative performance in both the training set (AUC = 0.84) and the validation set (AUC = 0.80).

Conclusion: Quantitative EEG can predict DCI following SaSAH, the resulting nomogram demonstrated substantial predictive value and may help target therapies to patients at highest risk of secondary brain injury. It needs to be further confirmed in the future by multi-center large sample studies.

1. Introduction

Severe aneurysmal subarachnoid hemorrhage (SaSAH) is an acute cerebrovascular contititon that significantly impacts the central nervous system [1,2]. Despite the rapid advancements in therapeutic tools, its mortality and disability rates remain significantly elevated. Complications following SaSAH have a substantial impact on the diagnosis and prognosis of patients and pose considerable challenges in perioperative management of neurocritical care. Delayed cerebral ischemia (DCI) is a common complication after the onset of aSAH, occurring predominantly within 14 days after aSAH [3]. DCI can lead to irreversible brain damage and regarded as a significant contributor to an important cause of disability and death in aSAH. Thus, early prediction and prevention are essential. However, the pathogenesis and pathophysiological process of DCI are complex, and there may be a lag in conventional assessment methods, thus exploring early and objective diagnostic tools to assist early prediction has become a research hotspot.

Electroencephalography (EEG) provides real-time insights about cortical activity. When neuronal function is compromised due to inadequate cortical perfusion, various EEG changes manifest, such as a depression of fast activity or a slowing of rhythms [4]. The EEG might be suitable to detect DCI at an early stage and monitor effects of therapeutic interventions, but interpreting these EEG data poses challenges. Quantitative EEG (qEEG) offers a potential solution to overcome these challenges as it allows more efficient post hoc analysis, immediate detection of events such as ischemia or epilepsy, and can provide a simplified

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overview of EEG for non-neurophysiologists.

Common quantitative EEG features, including amplitude-integrated EEG(aEEG), relative alpha variability(RAV), alpha/delta ratio (ADR) and spectral entropy(SE), have demonstrated utility in assessing the functional state of the brain and brain metabolism, and the extent of brain damage [5]. However, whether qEEG parameters are associated with DCI and functional prognosis following aSAH remains unclear. In this study, we analyzed the relationship between ~~the~~ clinical features, qEEG parameters, and DCI in aSAH patients, ~~so as~~ to facilitate the early identification of DCI risk factors and timely intervention measures to improve the quality of life and clinical prognosis of patients.

2. Materials and methods

2.1. Patients

Patients who received treatment in the neuro-intensive care unit (NICU) of Henan Provincial People's Hospital due to aSAH were eligible for this retrospective observational study. The research was conducted in accordance with the Declaration of Helsinki and received ethical approval from the Research Ethics Committee of Henan Provincial People's Hospital (2022, No. 6).

The inclusion criteria were as follows: (1) age $>$ 18 years; (2) aSAH was confirmed by computed tomography angiography (CTA) or digital subtraction angiography (DSA); (3) Embolization or surgical clipping was performed within 48 h after admission; (4) The diagnostic criteria for aSAH are in accordance with the Expert Consensus in the Management of Severe Aneurysmal Subarachnoid Hemorrhage (2015) and other current clinical guidelines [1], which stipulate a Hunt-Hess grade $\geq$ III in aSAH patients.

The exclusion criteria were as follows: (1) patients with significant non-neurological diseases that severely affect prognosis, such as severe heart failure or liver disease; (2) patients who did not receive EEG monitoring for various reasons (e.g., agitated patients or absence of the local skull following surgery); (3) patients who are pregnant or lactating; (4) EEG recordings during sedation maintained at greater than 2 mg/h of midazolam or an infusion dose of propofol exceeding 1.2 mg/kg/h; (5) incomplete clinical data.

The participating patients were followed up three months after the onset of the disease, and 145 patients with aSAH were ultimately included. The flowchart illustrating the inclusion and exclusion criteria is presented in Fig. 1.

2.2. Clinical data

An electronic database was utilized to collect baseline characteristics and clinical information. These baseline characteristics comprised demographic information (age, gender), medical history (smoking, hypertension, diabetes, coronary heart disease, and stroke history), and medical records (location of aneurysms, surgical procedures, temperature management, etc.). Serum biomarkers included white blood cell count, the percentage of neutrophils, platelet count, D-dimer, and plasma fibrinogen. Admission CT scans assessed subarachnoid hemorrhage severity using the modified Fisher scale and were classified as Modified Fisher grade I–IV. Hunt-Hess grade was employed to evaluate the neurological dysfunction of patients at admission, classified as Hunt-Hess grade III–V. Additionally, patients' clinical status was assessed using the Glasgow Coma Scale (GCS) and the Acute Physiology and Chronic Health Evaluation II scale (APACHE II) score. Treatment was not modified based on the results of the qEEG monitoring.

2.3. EEG recordings

The patients were monitored using a digital video EEG monitor (Nicolet EEG). qEEG monitoring was started immediately after securing the aneurysm by coiling or clipping. Each patient was monitored at least once for a minimum of 3 h. Eight electrodes were used, placed according to the international 10–20 system: FP1, FP2, C3, C4, O1, O2, T3, and T4. Additionally, one ground electrode (Fz) and one reference electrode (Cz) were placed. The impedance of each electrode was maintained below 5 kΩ, the sampling rate was 1,000 Hz, and the filtering range was 0.5–70 Hz. Data were recorded through the longitudinal connection of C3–O1

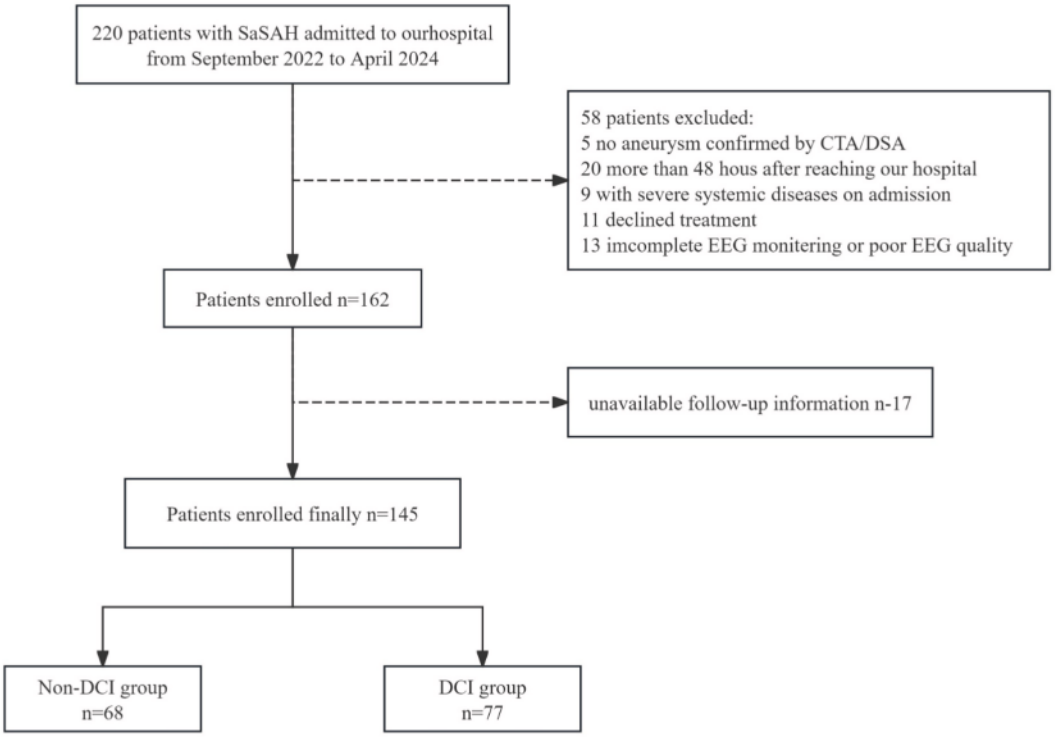

Fig. 1. Flow chart of study patients.

and C4–O2. The selected continuous EEG data, taken from the first EEG monitoring data after admission without motion interference and with a length of about 30 min, were analyzed quantitatively with the study room (5.95.0) software. aEEG, alpha/delta ratio (ADR), percentage of α variability (PAV) grade, and spectral entropy (SE) were acquired through raw EEG.

aEEG was a qEEG parameter based on raw EEG amplitude. The maximum amplitude of EEG within 15 s represented the upper bound of aEEG, and the minimum amplitude represented the lower bound of aEEG. ADR was calculated as the power ratio from the alpha (8–13 Hz) and delta (0.5–4 Hz) frequency bands. The relative alpha data were calculated every 2 min automatically. The frequency band for relative alpha was the proportion of 6 to 14 Hz power in 1 to 20 Hz total power activity. These trends were collected and two experienced physicians who have passed the neurophysiological electroencephalogram technology examination and obtained the qualification certificates will evaluate the EEG images of the same patient. They are unaware of the clinical situation. Excursions exceeding 15 % of the median values occurring once per hour were recorded as excellent (grade IV); excursions of 10 % at least every 4 h were recorded as good (grade III); small excursions during the whole monitoring process were recorded as fair (grade II); and excursions less than 2 % of the median values were recorded as poor (grade I). SE was the EEG power spectrum obtained by Fourier transformation based on frequency domain analysis. SE values were calculated every 10 s and presented in the form of a line graph.

2.4. Outcome assessment

The main outcomes included DCI during hospitalization and a poor prognosis. According to previous guidelines, DCI was defined as clinical signs of deterioration resulting from newly diagnosed focal nerve function injury. Patients exhibiting functional changes such as hemiparesis, aphasia, apraxia, hemianopia, neglect, or a decrease by at least 2 points on the Glasgow Coma Scale (GCS) for at least 1 h, and/or the presence of a new ischemic or infarct focus in the affected area observed on computed tomography (CT) or magnetic resonance imaging (MRI), were considered to have DCI.

Three months after the onset of the disease, the patients were followed up. The modified Rankin Scale (mRS) was utilized to evaluate their neurological recovery. Based on the mRS scores, the patients were categorized into two groups: those with a good prognosis (mRS 0–2) and those with a poor prognosis (mRS 3–6). Trained graduate students conducted outpatient clinic visits and telephone interviews to assess the mRS grades of the patients.

2.5. Statistical analysis

SPSS Statistics 27.0 and R version 4.2.1 were utilized for all statistical analyses. Chi-squared tests were performed for categorical variables, while two-tailed t-tests were used for normally distributed continuous variables, which were expressed as mean ± standard deviation (SD). For non-normally distributed continuous variables, the Mann-Whitney *U* test was employed, and these data were described by the median (inter quartile range). Missing data was dealt with by means of the multiple imputation method. Independent variables were determined based on statistically significant differences in univariate analysis, and independent risk factors associated with DCI were identified through multifactorial logistic regression analysis. The R language analysis software was used to construct a column-line graphical model for predicting DCI in patients with SaSAH upon admission, based on the included potential predictors.

To validate the diagnostic accuracy of the prediction model, the prognostic predictive value of the column-line diagram was assessed by plotting the receiver operating characteristic (ROC) curve. The Hosmer-Lemeshow goodness-of-fit test was selected to evaluate the model's accuracy, and the calibration of the model was further evaluated through the plotting of calibration curves after 1000 repetitions of Bootstrap sampling. Calibration curves were also plotted to assess the consistency and stability between predicted and actual outcomes. Additionally, clinical decision curve analysis was conducted to evaluate the clinical applicability of the model.

3. Results

3.1. Clinical characteristics of patients in the training and validation set

A total of 145 patients with SaSAH were included in the analysis, comprising 101 patients in the training set and 44 patients in the validation set. In the training cohort, 52 patients (51.49 %) developed DCI, while in the validation cohort, 25 patients (56.82 %) experienced DCI. As indicated in Table 1, there were no significant differences in patient characteristics between the training and validation sets ($p > 0.05$).

Table 1
Baseline and clinical characteristics of patients in the training set and validation set.

Variables	Training set (n = 101)	Validation set (n = 44)	$t/\chi^2/Z$	*p* Value
Age,years	57.25 ± 12.17	58.25 ± 12.69	0.451	0.653
Male	47(46.53)	16(36.66)	1.292	0.256
Hypertension	45(44.55)	27(61.36)	3.464	0.063
Diabetes	34(33.66)	22(50.00)	3.451	0.063
Coronary heart disease	49(48.51)	16(36.36)	0.010	0.920
Smoking	35(34.65)	19(43.18)	0.954	0.329
GCS	7(6, 10)	7(6, 10)	−0.400	0.690
APACHE II	19.82 ± 7.12	21.39 ± 6.49	1.253	0.214
Modified fisher grade			2.520	0.472
I	2(1.98)	3(6.82)		
II	28(27.72)	10(22.73)		
III	36(35.64)	17(38.64)		
IV	35(34.65)	14(31.82)		
Hunt-Hess grade			2.803	0.246
III	32(31.68)	8(18.18)		
IV	35(34.65)	18(40.91)		
V	34(33.66)	18(40.91)		
Aneurysmal location (Anterior circulation)	58(57.43)	27(61.36)	0.196	0.658
Aneurysmal treatment			0.276	0.599
Endovascular coil	53(52.48)	21(47.73)		
Surgical clip	48(47.52)	23(52.27)		
Hypothermia	42(41.58)	17(38.64)	0.113	0.740
mRS ≤ 2	50(49.50)	18(40.91)	0.909	0.340
Hematology index				
WBC	10.00(8.82, 13.09)	10.55(9.38, 14.26)	−1.453	0.148
Neutrophil	87.33 ± 5.99	87.26 ± 5.76	−0.060	0.949
PLT	226.20 ± 57.74	223.45 ± 65.56	0.257	0.801
D-Dimer	4.19(1.60, 6.60)	3.15(1.60, 5.86)	−1.284	0.199
Plasma fibrinogen	3.78(2.82, 5.30)	4.06(2.90, 6.27)	−0.523	0.600
qEEG parameters				
aEEG max	15.66 ± 5.88	15.22 ± 5.67	−0.425	0.676
aEEG min	8.02 ± 2.37	7.30 ± 2.45	−1.667	0.098
ADR	0.16(0.13, 0.19)	0.17(0.13, 0.20)	−1.163	0.248
PAV grade			5.616	0.132
I	34(33.66)	10(22.73)		
II	34(33.66)	18(40.91)		
III	23(22.77)	16(36.36)		
IV	9(8.91)	1(2.27)		
SE	41.22 ± 9.84	39.20 ± 9.09	−1.167	0.249

3.2. Baseline characteristics of the patients in the two groups in the training set

The Descriptive Analysis revealed significant differences between the two groups in several variables, including smoking (p = 0.037), GCS (p = 0.008), Modified Fisher grade (p = 0.019), Hunt-Hess grade (p = 0.037), hypothermia (p = 0.023), WBC (p = 0.022), aEEG min (p = 0.023), ADR (p < 0.001), and PAV grade (p = 0.004). These findings are summarized in Table 2.

3.3. Identifying the independent risk factors for DCI in SaSAH patients

All potential risk factors identified in the univariate regression analysis with p-values less than 0.05 were included in the multivariate regression model. The multivariate logistic regression analysis revealed that the following variables were independent risk factors for DCI in patients with SaSAH: GCS (p = 0.014, OR = 1.217), Modified Fisher grade II/I (p = 0.012, OR = 1.542), Modified Fisher grade III/I (p = 0.022, OR = 2.485), Modified Fisher grade IV/I (p = 0.026, OR = 1.764), hypothermia (p = 0.039, OR = 0.847), ADR (p = 0.004, OR = 0.206), PAV grade I/IV (p = 0.004, OR = 1.809), PAV grade II/IV (p = 0.001, OR = 1.256), and PAV grade III/IV (p = 0.011, OR = 1.708). These findings are summarized in Table 3.

Table 2
Baseline and clinical characteristics of patients in the training set.

Variables	Non-DCI group(n = 49)	DCI group(n = 52)	$t/\chi^2/Z$	*p* Value
Age,years	59.20 ± 10.86	55.40 ± 13.12	1.583	0.117
Male	22(44.90)	25(48.08)	0.101	0.749
Hypertension	18(36.73)	27(51.92)	2.356	0.125
Diabetes	13(26.53)	21(40.38)	2.168	0.141
Coronary heart disease	20(40.82)	29(55.77)	2.258	0.133
Smoking	12(24.49)	23(44.23)	4.342	0.037
GCS	9(6, 14)	7(5, 9)	−2.665	0.008
APACHE II	19.41 ± 8.05	20.21 ± 6.17	−0.562	0.574
Modified fisher grade			10.002	0.019
I	1(2.04)	1(1.92)		
II	20(40.82)	8(15.38)		
III	17(34.69)	19(36.54)		
IV	11(22.45)	24(46.15)		
Hunt-Hess grade			6.617	0.037
III	21(42.86)	11(21.54)		
IV	12(24.49)	23(44.23)		
V	16(32.65)	18(34.62)		
Aneurysmal location (Anterior circulation)	26(53.06)	32(61.54)	0.742	0.389
Aneurysmal treatment			3.530	0.060
Endovascular coil	21(42.86)	32(61.54)		
Surgical clip	28(57.14)	20(38.46)		
Hypothermia	26(53.06)	16(30.77)	5.167	0.023
mRS ≤ 2	29(59.18)	21(40.38)	3.567	0.059
Hematology index				
WBC	10.18 ± 2.88	11.51 ± 2.87	−2.326	0.022
Neutrophil	87.75 ± 6.36	86.93 ± 5.65	0.692	0.493
PLT	227.59 ± 53.17	224.88 ± 62.24	0.234	0.815
D-Dimer	4.25(1.78, 5.86)	4.18(1.59, 7.82)	−0.932	0.354
Plasma fibrinogen	3.50(2.82, 4.96)	4.12(2.80, 6.33)	−1.091	0.274
qEEG parameters				
aEEG max	16.56 ± 6.20	14.81 ± 5.47	1.512	0.135
aEEG min	8.45 ± 2.84	7.38 ± 1.57	2.331	0.023
ADR	0.19 (0.15, 0.21)	0.15 (0.12, 0.18)	−3.654	<0.001
PAV grade			13.423	0.004
I	10(20.41)	24(46.15)		
II	16(32.65)	18(34.62)		
III	15(30.61)	8(15.38)		
IV	8(16.32)	1(1.92)		
SE	42.33 ± 9.74	40.17 ± 9.92	1.101	0.274

3.4. The predictive nomogram development

Based on the results from the multivariate logistic model, a nomogram was developed to predict the risk of DCI in patients with SaSAH. This nomogram includes five variables and is depicted in Fig. 2. To use the nomogram, a vertical line is drawn up from each variable's value to the corresponding "Point" axis to determine its score. The scores for all variables are then summed to obtain a total score, which is located on the "Total Points" axis. By drawing a vertical line from the total score down to the bottom axis, the predicted risk of DCI can be determined.

3.5. The performance of the nomogram

The calibration curve for the nomogram predicting the probability of DCI in patients with SaSAH demonstrated good agreement between the predicted and observed probabilities for both the training and validation sets, as shown in Fig. 3. The Hosmer–Lemeshow H test confirmed that the model fit well, with non-statistical significance in both the training set (p = 0.5196) and validation set (p = 0.3383).

The area under the curve (AUC) for the nomogram was 0.84 (95 % CI: 0.76–0.91) in the training set (Fig. 4A) and was validated internally in the validation set with an AUC of 0.80 (95 % CI: 0.66–0.93) (Fig. 4B). These results demonstrate that the nomogram has excellent

Table 3
Univariate and multivariate logistic regression analyses of risk factors for DCI in the training set.

Variables	Univariate		Multivariate	
	OR(95 % CI)	*p* Value	OR(95 % CI)	*p* Value
Smoking	0.538(0.243, 1.191)	0.126		
GCS	1.179(1.020, 1.364)	0.026	1.217(1.040, 1.423)	0.014
Modified fisher grade				
I	Reference		Reference	
II	1.099(1.019, 1.186)	0.015	1.542(1.368, 1.725)	0.012
III	1.631(1.056, 2.518)	0.027	2.485(1.140, 5.419)	0.022
IV	1.969(1.012, 3.839)	0.046	1.764(1.071, 2.906)	0.026
Hunt-Hess grade				
III	Reference			
IV	0.975(0.946, 1.004)	0.085		
V	1.986(1.019, 3.870)	0.044		
Hypothermia	0.798(0.650, 0.981)	0.032	0.847(0.724, 0.991)	0.039
WBC	1.735(0.285, 10.547)	0.550		
aEEG min	0.933(0.866, 1.004)	0.065		
ADR	0.862(0.780, 0.953)	0.004	0.206(0.071, 0.597)	0.004
PAV grade				
I	2.180(1.495, 3.178)	<0.001	1.809(1.212, 2.698)	0.004
II	1.199(1.034, 1.390)	0.016	1.256(1.107, 1.424)	0.001
III	1.673(1.094, 2.558)	0.018	1.708(1.130, 2.582)	0.011
IV	Reference		Reference	

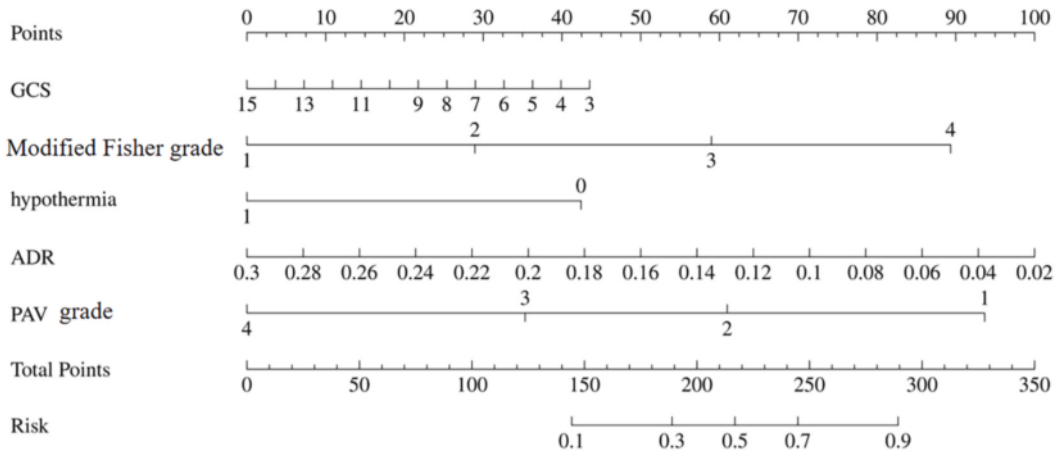

Fig. 2. A nomogram for predicting the occurrence of delayed cerebral ischemia (DCI) in patients after severe aneurysmal subarachnoid hemorrhage (SaSAH) based on 5 predictors.

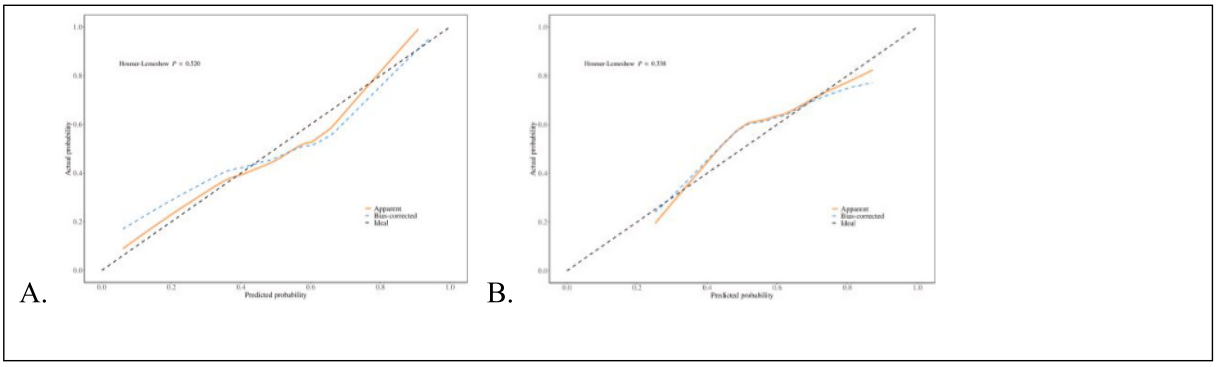

Fig. 3. Calibration curves of the nomogram in the training set and the validation set. (A) Calibration curves in the training set. (B) Calibration curves in the validation set. The y-axis represents the observed rate of CVS, and the x-axis represents the nomogram-predicted probability of CVS. The dotted lines represented by the nomogram are closer to the diagonal gray lines representing a better prediction.

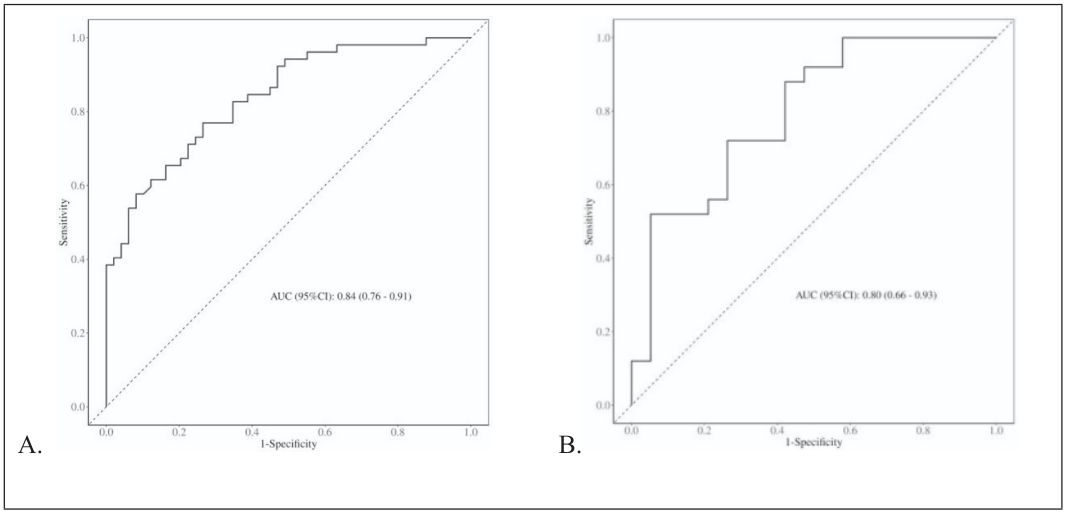

Fig. 4. The receiver operating characteristic (ROC) curve of the nomogram in the training set and the validation set. (A) ROC in the training set. (B) ROC in the validation set.

discriminatory performance.

Furthermore, the decision curve analysis (DCA) shows that when the threshold probability ranges from approximately 0.10 to 0.92, the nomogram yields a higher net benefit in predicting the risk of DCI, as illustrated in Fig. 5.

4. Discussion

DCI can cause irreversible brain damage, which is considered to be an important cause of disability and death in SaSAH [6]. The mechanisms underlying DCI are complex. As research progresses, some

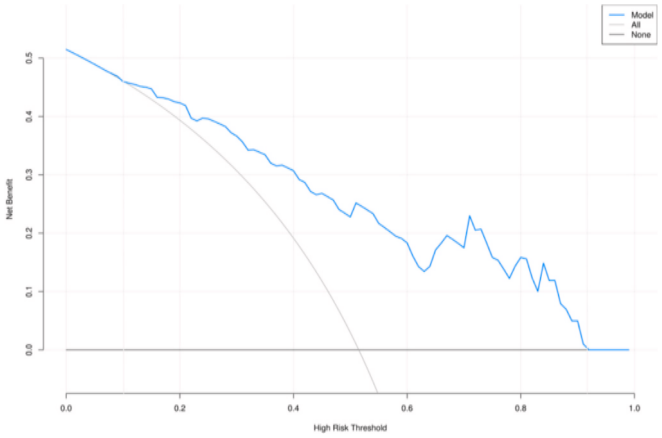

Fig. 5. Decision curve analysis of nomogram model.

scholars have suggested that, given the known factors involved in its pathophysiological processes, it is essential to shift treatment strategies from existing models to preventive approaches [7]. However, identifying DCI before clinical symptoms manifest remains challenging, especially in cases where patients exhibit atypical clinical manifestations or are unable to undergo a thorough neurological exam due to factors like invasive mechanical ventilation or delayed consciousness recovery [8]. Taking into account factors like invasive mechanical ventilation and frequently administered medications in ICU, patients may be unable to undergo a thorough neurological examination [9]. Consequently, biomarkers have garnered significant attention in recent years as a potential tool for predicting DCI in aSAH patients. Current prognostic models for DCI prediction primarily incorporate age, hematological biomarkers, basic neurological assessments, and radiographic findings as key predictors [10].

The findings of this study revealed that among 145 patients with SaSAH who underwent qEEG monitoring, 77 developed DCI, resulting in an incidence rate of 53.11 %. Our research leverages early EEG characteristics to predict the occurrence of DCI in SaSAH patients. After accounting for confounding variables through multivariate Logistic regression analysis, hypothermia, Fisher grade, GCS score, ADR, and PAV grade emerged as significant predictors of DCI. Furthermore, the nomogram prediction model incorporating qEEG data demonstrated a favorable predictive performance for the occurrence of DCI. We excluded pregnant and lactating individuals to control for potential confounders. While this restriction may limit the external validity of the findings, the homogeneity of core outcome measures and the robustness of data analyses suggest that the conclusions remain valid without substantial bias.

EEG can achieve functional monitoring of neurovascular coupling and assess the functional status of both cortical and subcortical brain regions, as well as the degree of neuronal damage [11]. In our pilot study, we found that patients who developed DCI exhibited poor aEEG, lower ADR, and a poor PAV grade. Research has demonstrated that continuous spasms in arterioles, microthrombosis, and disturbances in cerebral blood flow autoregulation following SAH can impact local blood flow and metabolism, subsequently affecting neuronal electrical activity [12]. EEG changes are closely tied to cerebral blood flow and metabolism. During ischemia, the ratio of slow waves (δ, θ) to fast waves (α, β) in the EEG decreases, leading to alterations in quantitative EEG metrics such as amplitude-integrated electroencephalogram, relative frequency band energy, relative α variation, and spectral patterns [13,14]. Established criteria for predicting DCI rely on modifications in continuous EEG spectral features, including a decline in the alpha-to-delta power ratio, variations in relative alpha power, epileptiform discharges, rhythmic and periodic ictal-interictal continuum patterns, and isolated alpha suppression [15,16].

We conducted an investigation into previously reported qEEG features, evaluating their additional predictive value. Our findings identified ADR, alpha power, and alpha variability as the most promising indicators [17]. M. L. Rot et al. reported that eight DCI patients exhibited a decrease of over 38 % in either ADR or alpha variability [18]. A prospective study on severe SAH patients (Hunt-Hess score 4–5) demonstrated that continuous EEG monitoring accurately predicts the occurrence of DCI in 78 % of cases [19]. EEG features such as new or worsening ADR trends, relative alpha variability (RAV) trends, focal slowing, epileptiform discharges, or rhythmic and periodic patterns exhibited excellent sensitivity and high specificity across different baseline risk levels (low risk: 91 % sensitivity, 83 % specificity; high risk: 95 % sensitivity, 77 % specificity). Similarly, our study found that patients with DCI had significantly poorer PAV grades and lower ADR compared to those without DCI, aligning with previous findings and further confirming the predictive capability of early qEEG features in DCI after SAH.

Brain tissue ischemia often leads to abnormal EEG activity, characterized by a decrease in PAV grading and an elevated percentage of slow-wave frequency bands. This indicates that quantitative EEG can predict early neurological recovery by reflecting nerve cell electrical activity. A PAV grade of I or II suggests inadequate cerebral perfusion and metabolism, severe brain injury, and a heightened risk of poor prognosis and death. Gollwitzer's research highlighted that a significant reduction in alpha power is indicative of a greater likelihood of poor functional outcomes [20]. In our study, the proportion of patients with an mRS score of 2 or lower in the DCI group was lower than in the Non-DCI group, although this difference was not statistically significant. This could be attributed to the small sample size or the influence of other clinical factors.

Recent advancements in technology and increased accessibility have made qEEG a viable option for assessing a wider range of diseases. EEG offers a continuous assessment of cerebral function with predictable responses. Several studies have linked qEEG features to cognitive functions, emotions, and stress levels in various diseases. Jakob et al. [21] reviewed existing research on quantitative and qualitative EEG measurements after stroke, concluding that EEG is valuable for predicting functional outcomes, mortality, the development of post-stroke cognitive decline, and epilepsy. A secondary analysis of the CAPTAIN-RTMS trial data explored the significance of qEEG indices as indicators of recovery in TBI patients, revealing significant increases in the delta alpha ratio (DAR) and delta theta/alpha beta ratio (DTABR), indicating changes in neurophysiological dynamics post-treatment [22]. In a similar study, Schleiger et al. found that an elevated DAR was negatively correlated with functional and cognitive outcomes after stroke [23]. Rhee et al. associated lower DAR values with reduced mental workloads and cognitive stress, while a decreased theta beta ratio (TBR) suggested increased attention [24].

The *meta*-analysis by Spitzmiller RE et al. verified that aEEG is highly valuable in selecting early treatment for hypoxic-ischemic encephalopathy (HIE) and predicting long-term neurological prognosis in children [25]. As EEG monitoring technology evolves, the application of quantitative EEG in adult research has gradually gained traction both domestically and internationally. Studies indicate that aEEG can assess brain function and prognosis in patients with cerebral infarction, HIE, and coma during cardiopulmonary resuscitation [26]. The findings of this study reveal that the DCI group exhibited a lower aEEG amplitude. This may be attributed to various pathophysiological changes resulting in altered neuronal metabolism and electrical activity. Increased brain slow wave activity and decreased aEEG amplitude are caused by disordered cell excitability.

qEEG can, to a certain extent, mimic the functionality of SPECT and PET functional imaging by quantitatively reflecting cerebral blood flow (CBF) coupled with the cerebral metabolic rate for oxygen [27,28]. Through performing EEG recordings on patients with acute ischemic stroke in the emergency room, the researchers found that there was a

correlation between local CBF and local alpha power ($\rho = 0.43$, $p < 0.01$) [29]. Hsin Yi Chen et al. suggested that combining spectral continuous EEG measures associated with DCI, such as the alpha-delta ratio, relative alpha variability, and total power, could enhance DCI prediction when used in conjunction with transcranial Doppler (TCD) [30]. However, in a study involving patients with acute severe brainstem hemorrhage, qEEG was found to be effective in informing the clinical prognosis regarding 90-day mortality ($p = 0.022$), whereas TCD was not ($p > 0.05$) [31]. Future research should further explore multimodal monitoring, which includes a variety of monitoring methods, to effectively monitor brain metabolism and cerebral blood flow and predict early brain injury.

Previous studies have investigated the predictive grading of radiological, clinical, and composite scores [32–35]. Consistent with these studies, our findings indicate that smoking, Hunt-Hess grade, and white blood cell count are associated with an increased risk of DCI. However, when these clinical variables were included in the multiple regression model, they did not emerge as independent predictors. This may be attributed to limitations in sample size, which necessitates further research in the future.

We acknowledge some limitations of this study. Firstly, it was a single-center observational study that lacked external validation, and the sample size could be expanded in future research. Secondly, we did not include a comprehensive selection of clinical indicators (such as Neuron-specific enolase), which may have underestimated the contribution of qEEG parameters. Thirdly, this study was a cross-sectional study without qEEG monitoring at multiple time points. In future studies, considering multiple time points and continuous qEEG monitoring would help capture changes before and after the disease. Additionally, further research is needed to investigate specific interventions aimed at reducing early brain injury.

5. Conclusion

The results of this pilot study show that EEG monitoring, using the quantitative features ADR and PAV grade, along with clinical data, is a promising tool for early detection of DCI in aSAH patients. Our results need additional validation in a larger patient cohort admitted after aSAH.

Author contributions

Bingsha Han, Mengyuan Xu and Pengzhao Zhang conceptualized and designed the study, drafted the initial manuscript, and reviewed and revised the manuscript. Yang Liu and Jiaqi Zhang collected the data and carried out the initial analyses. Bingsha Han and Guang Feng supervised the findings of this work and and critically reviewed the manuscript for important intellectual content.

CRediT authorship contribution statement

Mengyuan Xu: Writing – review & editing, Writing – original draft, Conceptualization. **Pengzhao Zhang:** Validation, Supervision, Data curation. **Yang Liu:** Software, Project administration, Methodology, Data curation. **Jiaqi Zhang:** Project administration, Investigation, Data curation. **Guang Feng:** Supervision, Project administration, Conceptualization. **Bingsha Han:** Writing – review & editing, Supervision, Project administration, Conceptualization.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

The datasets generated and analyzed during the current study areavailable from the corresponding author on reasonable request.

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