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Development and internal validation of a Nomogram-based risk prediction model for post-stroke cognitive impairment

Chunyan Chen[a,1], Xiangbo Wu[a,1], Tao Han[a,1], Bin Zhang[b,c], Xiaodong Lin[a], Guiqing Cheng[a], Xinyu Liu[a], Yixing Lu[a], Hangkun Zhao[a], Hua Yuan[a,*], Xiaolong Sun[a,*]

[a] Department of Rehabilitation Medicine, Xijing Hospital, Air Force Medical University (Fourth Military Medical University), Xi'an 710032, China
[b] Department of Wound Repair and Rehabilitation Medicine, Center of Bone Metabolism and Repair, Laboratory for Prevention and Rehabilitation of Training Injuries, State Key Laboratory of Trauma, Burns and Combined Injury, Trauma Center, Research Institute of Surgery, Daping Hospital, Army Medical University, Chongqing 400042, China
[c] Rehabilitation Center, Key Specialty of Neck and Low Back Pain Rehabilitation, Xingcheng Special Duty Sanatorium, Xingcheng 125100, China

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ABSTRACT

Background: Post-stroke cognitive impairment (PSCI) severely affects patients' quality of life and survival, and no curative treatment is currently available. Therefore, early detection and prevention are crucial.
Objective: To develop and internally validate a nomogram for predicting the risk of PSCI within 6 months after stroke.
Study design: Bidirectional observational cohort study.
Methods: Demographic, clinical, and cognitive data were collected from patients with stroke treated at the First Affiliated Hospital of Air Force Medical University between January 2019 and January 2024, within 40 days of stroke onset. Six-month cognitive outcomes were obtained from medical records for the retrospective component (January 2019 to August 2023), while patients in the prospective component (August 2023 to January 2024) were followed up for 6 months. Patients were categorized into PSCI and non-PSCI groups based on the 6-month cognitive assessment. The least absolute shrinkage and selection operator regression and multivariable logistic regression were used to identify independent predictors and construct the prediction model. Model accuracy was evaluated and validated internally using the bootstrap method.
Results: Of 286 patients with stroke assessed, 107 presented with PSCI at the 6-month follow-up. The key predictors identified were baseline Mini-Mental State Examination score, time from lesion to rehabilitation facility, lesion location, and hypoalbuminemia. The risk prediction model using these factors showed strong performance, with an apparent area under the curve (AUC) of 0.940 (95% confidence interval [CI], 0.913–0.967), indicating good discrimination. The mean apparent AUC across 1000 bootstrap training samples was 0.947 (95% CI: 0.918–0.970). After optimism correction, the internally validated AUC was 0.937 (mean optimism = 0.003), indicating minimal overfitting.
Conclusion: The developed nomogram-based model predicted the risk of PSCI. It may aid in counseling patients with stroke and their families, and help clinicians identify high-risk patients for early intervention to improve outcomes.

Abbreviations: ADL, activities of daily living; AUC, area under the curve; CHD, coronary heart disease; ChiCTR, Chinese Clinical Trial Registry; CI, confidence interval; DCA, decision curve analysis; DVT, deep vein thrombosis; EPV, events-per-variable; LASSO, Least Absolute Shrinkage and Selection Operator; MBI, Modified Barthel Index; MCI, mild cognitive impairment; MMSE, Mini-Mental State Examination; MoCA, Montreal Cognitive Assessment; NIHSS, National Institutes of Health Stroke Scale; PSCI, post-stroke cognitive impairment; ROC, receiver operating characteristic; TLRF, time from lesion to rehabilitation facility

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* Correspondence to: Department of Rehabilitation Medicine, Xijing Hospital, Air Force Medical University (Fourth Military Medical University), No. 15, Changle West Road, Xi'an, Shaanxi 710032, China.
E-mail addresses: yuanhua@fmmu.edu.cn (H. Yuan), xlsun@fmmu.edu.cn (X. Sun).

[1] Chunyan Chen, Xiangbo Wu and Tao Han contributed equally to this manuscript

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1. Introduction

Stroke is the second-leading cause of mortality worldwide. Its high incidence and substantial disability rates severely impair patients' quality of life and pose major safety risks.[1] Consequently, stroke has emerged as a critical global public health issue. Post-stroke cognitive impairment (PSCI) encompasses various manifestations that emerge within the critical 6-month window after stroke, fulfilling the rigorous diagnostic criteria for cognitive decline.[2] PSCI is among the most prevalent complications associated with stroke. The reported prevalence of PSCI ranges from 20% to 75% at 3–6 months after stroke.[3,4] Clinically, PSCI is primarily characterized by cognitive deficits across multiple domains, including orientation, attention, comprehension, executive function, and language.[2] PSCI often develops insidiously and gradually progresses, leading to underrecognition by patients and their families. Its incidence is approximately 3.5–5.6 times higher than that in cognitively healthy individuals, representing a significant public health burden and leading to a substantial loss of social resources.[5] The mortality rate of patients with PSCI is significantly elevated compared with that of patients with stroke with normal cognitive function.[6] Furthermore, PSCI substantially increases the disability rate and degree of life dependence among patients, rendering them more susceptible to anxiety and depression. Consequently, this diminishes their quality of life and activities of daily living (ADL) performance, thereby burdening their families and society. The presence of PSCI can significantly affect various aspects of stroke treatment, including motor function and ADL, and nearly doubles the risk of adverse outcomes.[1]

A definitive diagnosis of PSCI is typically made 3–6 months after stroke, as cognitive functions may initially improve within the first few months owing to rehabilitation and neuroplasticity, with diminished improvement observed beyond the 6-month mark.[7] PSCI can be effectively managed if identified early. Empirical evidence suggests that early identification, diagnosis, prevention, and intervention for PSCI significantly improve the prognosis of stroke survivors and mitigate associated social and economic burdens.[8] Nevertheless, there is a notable deficiency in clinical tools specifically designed for the early diagnosis of PSCI in patients with stroke. The current body of evidence substantiates that hypertension, diabetes, smoking, and other vascular predictors are significantly associated with an elevated risk of PSCI.[9] The recently developed $SIGNAL_2$ risk score, which incorporates both clinical and neuroimaging variables, has demonstrated efficacy in accurately identifying patients at risk of developing PSCI within 3–6 months and 12–18 months post-stroke.[10] Similarly, the CHANGE score, which also integrates clinical and imaging data, has proven effective in assessing the risk of PSCI in patients with ischemic stroke within an 18-month period following the stroke.[11] However, the aforementioned scoring systems necessitate that radiologists precisely extract imaging data, a task that proves challenging to implement in community hospitals. The time-consuming, complex, and expensive nature of these predictive models further complicates their adoption in clinical settings. Consequently, clinically reliable and readily accessible data need to be identified urgently to facilitate the efficient and convenient identification of high-risk patients with PSCI. The objective of this study was to construct a nomogram to predict the risk of PSCI within 6 months in patients with stroke. This tool is intended to assist clinicians in the early and convenient identification of PSCI, thereby facilitating timely initiation of cognitive rehabilitation and pharmacological interventions.

2. Methods

2.1. Study design and patients

This was a bidirectional observational cohort study comprising both retrospective and prospective components. Data from patients with stroke admitted to the Department of Rehabilitation Medicine at the First Affiliated Hospital of Air Force Medical University between January 2019 and January 2024 were analyzed. For the retrospective component (January 2019 to August 11, 2023), comprehensive data on predictive variables, including demographic information, clinical characteristics, and assessments, were collected within 40 days of stroke onset from electronic medical records.[12] Six-month cognitive outcomes were also obtained from medical records for this component. For the prospective component (from August 11, 2023 onward), patients were enrolled and followed up for 6 months. Based on the Mini-Mental State Examination (MMSE) scores recorded at the 6-month assessment, patients were dichotomized into the PSCI and non-PSCI groups.[13] Subsequently, the predictors associated with PSCI were analyzed, and a predictive model for the identification of PSCI was developed. The inclusion criteria for the study were as follows: (1) age $\geq$ 18 years and right-handedness (Edinburgh Handedness Inventory); (2) confirmed stroke diagnosis based on cranial computed tomography or magnetic resonance imaging; and (3) capability to complete the cognitive and functional scale assessments, defined as having stable vital signs and no severe disturbance of consciousness (Glasgow Coma Scale score > 8), no severe aphasia that would preclude reliable responses to MMSE items, no severe visual or hearing impairment that would interfere with test administration, and sufficient physical capacity to sit for the duration of the assessment.[14,15] The exclusion criteria were (1) a prior diagnosis of cognitive impairments such as dementia or Alzheimer's disease; (2) incomplete clinical data; and (3) concurrent mental illnesses, including depression, anxiety, or mania.

2.2. Sample size determination

For logistic regression, the events-per-variable (EPV) rule recommends at least 10–20 outcome events per predictor to avoid overfitting.[16] A conservative EPV ratio of 20:1 was used. We included up to four candidate predictors requiring a minimum of 80 PSCI events. With a conservative estimate of 30% for the PSCI incidence, the required sample size was 267.[17] The target sample size was increased to 281 patients to allow for an estimated 5% missing data rate. To manage the risk of overfitting with multiple candidate predictors, we employed Least Absolute Shrinkage and Selection Operator (LASSO) regression with 10-fold cross-validation for variable selection, followed by refitting of the selected variables in a multivariable logistic model.

2.3. Baseline clinical characteristics collection

The baseline clinical characteristics of the patients were collected within the first 24 h of admission. The first segment encompassed demographic characteristics including age, sex, educational attainment, smoking history, alcohol use, and blood type. Smoking history was defined as the consumption of 10 or more cigarettes per day for at least 6 months.[5] Alcohol use was defined as consuming at least 50 g of alcohol per week for at least 6 months.[5] Blood type was categorized as A, B, O, and AB.[18]

The second segment pertained to admission status, encompassing the time from lesion to rehabilitation facility (TLRF), National Institutes of Health Stroke Scale (NIHSS), baseline MMSE, and Modified Barthel Index (MBI). TLRF was defined as the number of days from stroke onset to admission to the rehabilitation facility.[12] The NIHSS was employed to evaluate the severity of stroke, with a maximum score of 42 points. A score of 0 denotes normal neurological function, while higher NIHSS scores correspond to more severe neurological impairment.[19] The MMSE is the most widely used cognitive assessment tool in stroke populations and has been extensively applied in the assessment of PSCI. It demonstrates high sensitivity and specificity for screening PSCI in community and primary healthcare settings.[20] This scale evaluated and quantified five cognitive domains: memory, orientation, attention and calculation,

recall, and language, with a maximum score of 30 points. Lower scores indicate diminished cognitive function. The MBI was used to assess patients' activities of daily living, with higher scores indicating greater independence.[19]

The third section pertained to disease conditions, including stroke type, location, side, and history. Stroke was classified as ischemic or hemorrhagic. To facilitate analysis, we categorized lesions as cortical or subcortical.[21] Lesion location was further classified by hemisphere: left-side, right-side, or bilateral.

We also considered comorbidities associated with stroke, including hypertension, diabetes, dyslipidemia, coronary heart disease (CHD), hypoalbuminemia, and deep vein thrombosis (DVT). Hypertension was defined as having a systolic blood pressure of ≥ 140 mmHg or a diastolic blood pressure of ≥ 90 mmHg after repeated measurements, or a documented history of antihypertensive medication use.[3] Diabetes was characterized by a fasting blood glucose level of ≥ 7.0 mmol/L, a postprandial (2 h) blood glucose level of ≥ 11.1 mmol/L, an HbA1c level of ≥ 6.5%, or the presence of typical symptoms accompanied by a random blood glucose level of ≥ 11.1 mmol/L.[3] Dyslipidemia was defined by a fasting venous blood test that met at least one of the following criteria: total cholesterol ≥ 5.2 mmol/L, low-density lipoprotein cholesterol ≥ 2.6 mmol/L, triglycerides ≥ 1.70 mmol/L, or current treatment with lipid-lowering medications.[5] CHD was identified based on a self-reported history of myocardial infarction or angina pectoris.[22] Serum albumin was measured at admission using fasting venous blood samples collected within 24 h of hospital admission. Hypoalbuminemia was defined as a serum albumin level < 35 g/L.[23,24] DVT was diagnosed by compression ultrasonography, with non-compressibility of the venous segment as the primary diagnostic criterion.[25,26]

2.4. Outcome measures

Cognitive function was assessed using the MMSE. The Chinese version of the MMSE was administered at two time points: at baseline (within 40 days post-stroke, as part of the predictor variables) and at the 6-month follow-up (as the outcome variable). To ensure consistency, the same version of the MMSE was used for both assessments. All examinations were performed by trained rehabilitation therapists with at least 2 years of clinical experience in cognitive assessment. Prior to the study, all assessors completed a standardized training program that included a didactic session on MMSE administration and scoring, and supervised practice with at least 10 pilot patients. Assessments were performed in a quiet, well-lit room with the patients seated comfortably, with each session lasting approximately 10–15 min. PSCI was defined according to education-adjusted MMSE cutoffs: ≤ 17 for illiterate individuals, ≤ 20 for those with primary education, and ≤ 24 for those with secondary education or above.[1,5,27] The primary outcome was the diagnosis of PSCI 6 months post-stroke.[4] Patients were categorized into the PSCI and non-PSCI groups based on the presence or absence of PSCI at that time point, respectively.

Data were collected by trained rehabilitation physicians and therapists using a standardized case registration form. This study was approved by the Ethics Committee of the First Affiliated Hospital of the Air Force Medical University (approval number KY20232243-C-1) and adhered to both Chinese legal requirements and the principles outlined in the Declaration of Helsinki (2024 revision). The study was registered with the Chinese Clinical Trial Registry (ChiCTR) under the identifier ChiCTR2400081107. For the retrospective component, the requirement for informed consent was waived by the Ethics Committee. For the prospective component, written informed consent was obtained from all patients or their legally authorized representatives.

2.5. Statistical analysis

Statistical analyses were conducted using SPSS version 24.0 and R version 4.3.1. Continuous variables are presented as means ± standard deviations for normally distributed data or as medians and interquartile ranges (IQRs) for non-normally distributed data. Categorical variables are expressed as counts and percentages. The Mann–Whitney U test or two-sample t-test was used to compare continuous variables between the two groups. Categorical variables were compared between the two groups using the chi-square test. LASSO regression was applied to identify predictors associated with PSCI. Multiple logistic regression analysis was performed to identify independent predictors associated with PSCI, leading to the development of a PSCI risk prediction model. A nomogram was generated to represent the model visually. The predictive performance of the nomogram was assessed by several methods. The ability of the model to differentiate between PSCI and non-PSCI cases was evaluated using a receiver operating characteristic (ROC) curve. The calibration of the model was assessed using calibration curves, the Hosmer-Lemeshow test, and Brier score. The Brier score assesses both calibration and discrimination, quantifying the mean squared error between observed and predicted outcomes, with values ranging from 0.0 (indicating perfect prediction) to 0.25 (indicating poor prediction) at a 50% event occurrence rate.[12] Decision curve analysis (DCA) was employed to assess the clinical efficacy, benefits, and applicability of the model.[28] Internal validation was performed using 1000 bootstrap resamples, with the entire modeling process (including LASSO selection and multivariable logistic regression) repeated in each sample. The optimism-corrected area under the curve (AUC) was calculated by subtracting the mean optimism from the apparent AUC.[29] Pairwise comparisons of the AUCs between the models were performed using the DeLong test. Differences were considered statistically significant at $P < 0.05$.

3. Results

3.1. Clinical characteristics of patients

The study flowchart is presented in Fig. 1. This study included 377 patients with stroke, 65 of whom were excluded. Specifically, 11 patients had a history of cognitive impairment, 48 had incomplete data, and 6 had a history of mental illness. Additionally, 26 patients did not complete follow-up: 3 died and 23 were lost to follow-up. To assess the potential selection bias owing to loss to follow-up, we compared the baseline characteristics of the 286 patients included in the analysis with those of the 26 patients who were lost to follow-up (Supplemental Table 1). Most baseline variables were comparable between the two groups, except for the side of the lesion, which showed a significantly higher proportion of right-sided lesions in the lost-to-follow-up group (73.1% vs. 49.0%, $P = 0.013$). No other significant differences in age, sex, education, smoking, alcohol use, blood type, TLRF, NIHSS, baseline MMSE, MBI, stroke type, infarct distribution, prior stroke, or comorbidities were observed between the groups (all $P > 0.05$). A total of 286 patients with stroke were evaluated and categorized into the PSCI and non-PSCI groups based on the presence or absence of PSCI. At 6 months post-stroke, 107 patients exhibited PSCI. Table 1 provides a detailed comparison of baseline characteristics between the PSCI and non-PSCI groups. Significant differences in age ($P = 0.042$), years of education ($P = 0.007$), TLRF ($P < 0.001$), NIHSS ($P < 0.001$), baseline MMSE ($P < 0.001$), MBI ($P < 0.001$), lesion location ($P < 0.001$), CHD ($P = 0.031$), and hypoalbuminemia ($P < 0.001$) were observed between the two groups.

3.2. Predictive nomogram development

To identify the predictors associated with PSCI, we employed LASSO regression to screen 20 variables. As illustrated in Fig. 2, following a 10-fold cross-validation procedure, four factors were identified as being

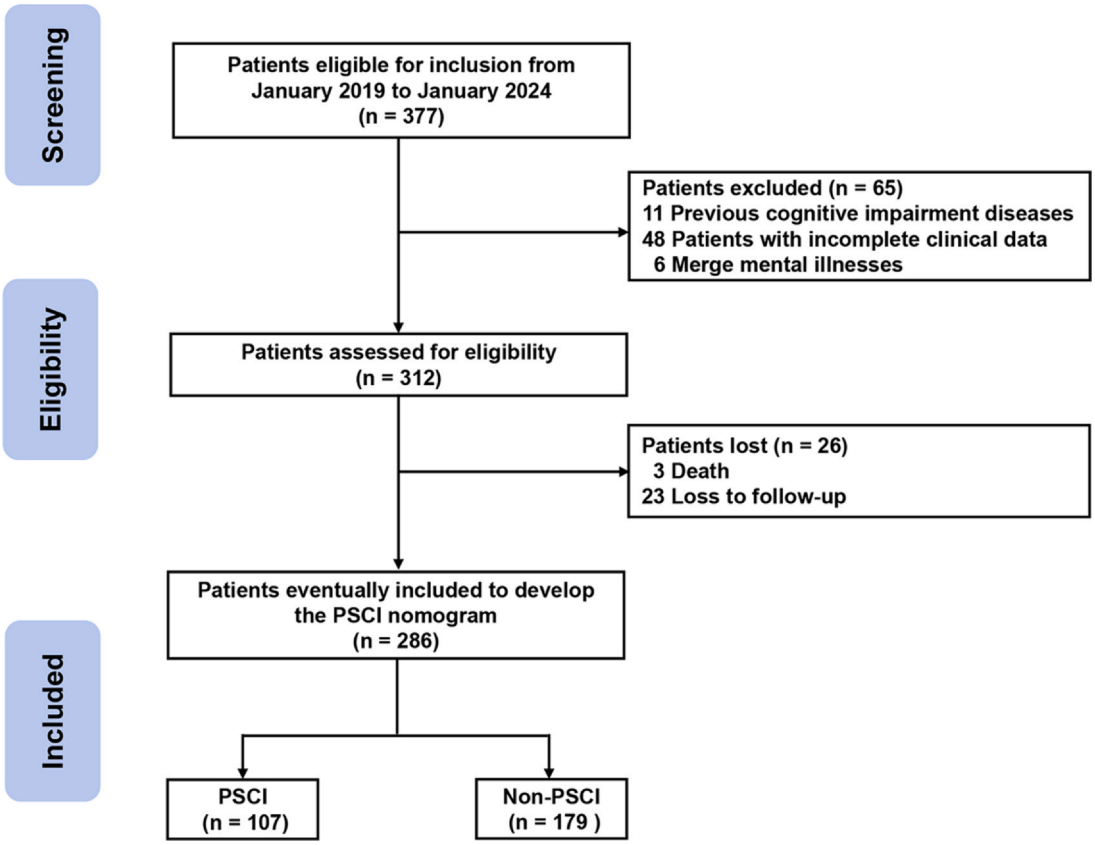


Fig. 1. Flowchart of selection process.

significantly associated with PSCI when the regularization parameter (λ) was minimized: baseline MMSE, TLRF, lesion location, and hypoalbuminemia. These variables were subsequently incorporated into a multivariate logistic regression analysis. Multivariate logistic regression analysis identified baseline MMSE (odds ratio [OR] = 0.787, 95% confidence interval [CI]: 0.742–0.835, $P < 0.001$), TLRF (OR = 1.115, 95% CI: 1.067–1.165, $P < 0.001$), location of lesion (left side, OR = 4.241, 95% CI: 1.826–9.852, $P = 0.001$), and hypoalbuminemia (OR = 4.515, 95% CI: 1.705–11.958, $P = 0.002$) as independent predictors of 6-month PSCI status in patients with stroke (Table 2).

A nomogram was developed based on the results of the multivariate logistic regression to predict the risk of PSCI at 6 months, incorporating four identified variables: baseline MMSE, TLRF, lesion location, and hypoalbuminemia (Fig. 3). The full logistic regression equation, including the model intercept and all β coefficients, is provided in the Supplemental Material. To analyze the data, we first drew a vertical line on the axis and calculated the score for each variable. These preliminary scores were summed to obtain the total score. This total score corresponded to the total score axis, whereas the predicted probability of PSCI aligned with the lower axis. To facilitate the convenient conversion of the total score to the predicted PSCI probability, we provided a numerical lookup table corresponding to the nomogram (Supplemental Figure 1). The associated risk probability was directly obtained from the table for each total score derived from the nomogram, thereby enhancing the clinical practicality of the prediction tool.

3.3. Performance of the nomogram and internal validation

The nomogram demonstrated strong discrimination, as evidenced by an apparent AUC of 0.940 (95% CI: 0.913–0.967) (Fig. 4A). To facilitate the clinical application of the nomogram, we determined the optimal cutoff value for the predicted PSCI probability using the Youden index. Based on the ROC curve of the full model, the optimal cutoff was 0.52, at which point the model achieved a sensitivity of 83.2% and a specificity of 92.7%. The calibration curve of the prediction model was plotted and evaluated using the Hosmer-Lemeshow test ($P = 0.337$), indicating that the model exhibited a good goodness of fit (Fig. 4B). The Brier score of 0.091 corroborated the superior calibration of the prediction model. The DCA demonstrated that the threshold probability of the prediction model ranged from 0 to 0.92, with a relative net benefit between 7% and 38%, indicating the model's substantial clinical applicability and value (Fig. 4C). Furthermore, as assessed through 1000 bootstrap resamples, the mean apparent AUC was 0.947 (95% CI: 0.918–0.970) (Fig. 4D). After optimism correction, the mean optimism was 0.003, yielding an optimism-corrected AUC of 0.937, which confirmed the robustness of the model with minimal overfitting.

To further evaluate the incremental predictive value of the four identified predictors, we compared the full model (incorporating baseline MMSE, TLRF, lesion location, and hypoalbuminemia) with two reduced models: the MMSE-only model (using baseline MMSE alone) and the MMSE-excluded model (using TLRF, lesion location, and hypoalbuminemia without baseline MMSE) (Fig. 5). The full model achieved an AUC of 0.940 (95% CI: 0.913–0.967), significantly outperforming the MMSE-only model (AUC = 0.880, 95% CI: 0.834–0.927; $P < 0.001$) and the MMSE-excluded model (AUC = 0.808, 95% CI: 0.757–0.858; $P < 0.001$). Thus, incorporating both cognitive and clinical predictors yielded superior risk stratification for 6-month PSCI.

4. Discussion

A low baseline MMSE score, delayed TLRF, left-sided cerebral injury, and hypoalbuminemia were identified as independent predictors of PSCI development. Our model may provide a practical framework for early risk stratification that may inform clinical decision-making. However, prospective studies are needed to determine whether model-guided risk stratification translates into improved patient outcomes.

Table 1
Patient demographic and baseline characteristics.

Variable	All patients (N = 286)	Non-PSCI (N = 179)	PSCI (N = 107)	χ^2/Z*	*P*
Demographic characteristics					
Age, years, median (P_{25}, P_{75})	59.0(51.0, 68.0)	59.0(46.0, 68.0)	60.0(54.0, 69.0)	- 2.036	0.042 (Mann–Whitney U)
Sex (male, %)	207(72.4)	133(74.3)	74(69.2)	0.886	0.347 (χ^2)
Education, years, median (P_{25}, P_{75})	12.0(9.0, 14.0)	9.0(6.0, 12.0)	9.0(9.0, 14.0)	- 2.685	0.007 (Mann–Whitney U)
Smoking, n (%)	193(67.5)	119(66.5)	74(69.2)	0.219	0.640 (χ^2)
Alcohol use, n (%)	78(27.3)	54(30.2)	24(22.4)	2.021	0.155 (χ^2)
Blood type					
A	57(19.9)	33(18.4)	24(22.4)	5.274	0.153 (χ^2)
B	85(29.7)	58(32.4)	27(25.2)		
AB	71(24.8)	38(21.2)	33(30.8)		
O	73(25.5)	50(27.9)	23(21.5)		
Status on admission					
TLRF, days, median (P_{25}, P_{75})	20.0(12.0, 28.0)	17.0(10.0, 24.0)	24.0(16.0, 34.0)	- 5.099	< 0.001 (Mann–Whitney U)
NIHSS, median (P_{25}, P_{75})	10.0(5.0, 15.0)	8.0(4.0, 12.0)	14.0(8.0, 22.0)	- 5.674	< 0.001 (Mann–Whitney U)
Baseline MMSE, median (P_{25}, P_{75})	17.0(10.0, 26.0)	25.0(18.0, 26.0)	7.0(1.0, 14.0)	- 10.789	< 0.001 (Mann–Whitney U)
MBI, median (P_{25}, P_{75})	33.0(12.0, 54.0)	38.0(25.0, 61.0)	10.0(0, 39.0)	- 7.148	< 0.001 (Mann–Whitney U)
Disease conditions					
Stroke subtype					
Hemorrhagic	100(35.0)	60(33.5)	40(37.4)	0.440	0.507 (χ^2)
Ischemic	186(65.0)	119(66.5)	67(62.6)		
Distribution of lesions					
Cortical	61(21.3)	32(17.9)	29(27.1)	3.414	0.181 (χ^2)
Subcortical	169(59.1)	110(61.5)	59(55.1)		
Both	56(19.6)	37(20.7)	19(17.8)		
Location of lesion					
Left	123(43.0)	56(31.3)	67(62.6)	38.519	< 0.001 (χ^2)
Right	140(49.0)	113(63.1)	27(25.2)		
Bilateral	23(8.0)	10(5.6)	13(12.1)		
Prior stroke, n (%)	37(12.9)	18(10.1)	19(17.8)	3.526	0.060 (χ^2)
Comorbidity					
Hypertension, n (%)	185(64.7)	113(63.1)	72(67.3)	0.508	0.476 (χ^2)
Diabetes, n (%)	61(21.3)	41(22.9)	20(18.7)	0.709	0.400 (χ^2)
Dyslipidemia, n (%)	67(23.4)	40(22.3)	27(25.2)	0.311	0.577 (χ^2)
CHD, n (%)	49(17.1)	24(13.4)	25(23.4)	4.676	0.031 (χ^2)
Hypoalbuminemia, n (%)	68(23.8)	28(15.6)	40(37.4)	17.466	< 0.001 (χ^2)
DVT, n (%)	41(14.3)	21(11.7)	20(18.7)	2.641	0.104 (χ^2)

* Z values correspond to the Mann–Whitney *U* test for non-normally distributed continuous variables, whereas χ^2 values correspond to the chi-square test for categorical variables.

Abbreviations: PSCI, post-stroke cognitive impairment; IQR, interquartile range; TLRF, time from lesion to rehabilitation facility; NIHSS, National Institute of Health Stroke Scale; MMSE, Mini-Mental State Examination; MBI, Modified Barthel Index; CHD, coronary heart disease; DVT, deep vein thrombosis.

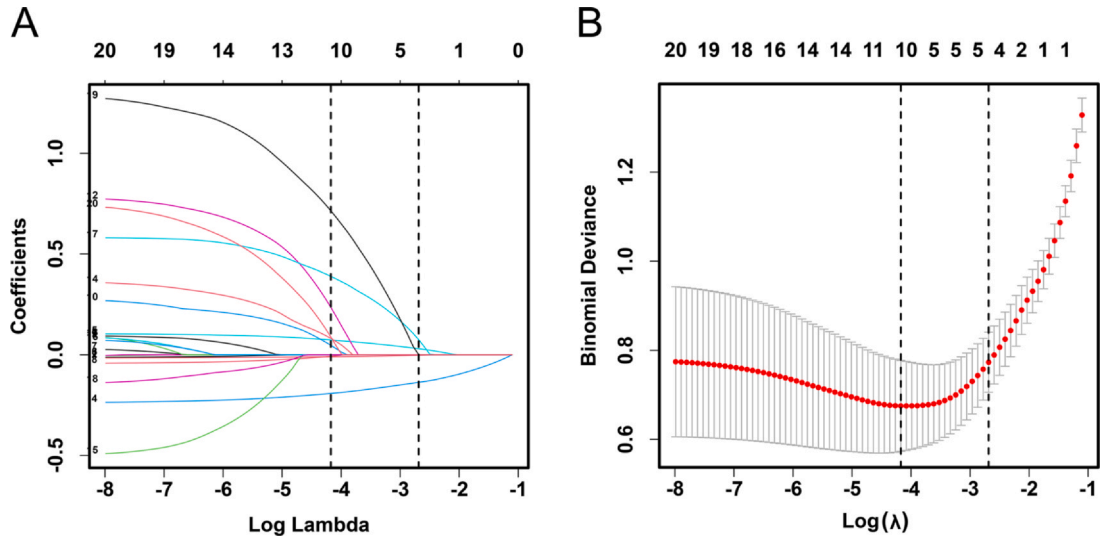


Fig. 2. Feature selection using the LASSO regression model. (A) Log lambda value of the 20 features in the LASSO model. A coefficient profile plot was produced against the log lambda sequence. (B) Parameter selection in the LASSO model was performed using 10-fold cross-validation via minimum criterion. Abbreviation: LASSO, Least Absolute Shrinkage and Selection Operator.

Commonly utilized screening instruments for PSCI include the MMSE and Montreal Cognitive Assessment (MoCA). We utilized the MMSE rather than the MoCA as the predictor and outcome variables in our investigation because not all participants in our study underwent MoCA. The MMSE is the most extensively utilized cognitive screening instrument, both nationally and globally, demonstrating high

Table 2
Multivariate logistic regression analysis of predictors for patients developing PSCI 6 month after stroke.

Variable	*b*	Waldχ^2	*P*	Odds ratio (95% CI)
Baseline MMSE	−0.239	63.7	< 0.001	0.787(0.742–0.835)
TLRF, days	0.109	23.774	< 0.001	1.115(1.067–1.165)
Location of lesion				
Right	—	—	—	—
Left	1.445	11.287	0.001	4.241(1.826–9.852)
Bilateral	1.129	3.098	0.078	3.093(0.880–10.873)
Hypoalbuminemia				
No	—	—	—	—
Yes	1.507	9.200	0.002	4.515(1.705–11.958)

Reference groups: lesion, Right; hypoalbuminemia, No.
Abbreviations: PSCI, post-stroke cognitive impairment; CI, confidence interval; MMSE, mini mental status exam; TLRF, time from lesion to rehabilitation facility.

sensitivity and specificity for assessing memory and language cognitive domains. However, it exhibits a relatively low sensitivity in detecting mild cognitive impairment (MCI). By contrast, the MoCA shows superior sensitivity and specificity in identifying MCI compared with the MMSE.[30] Although the MoCA was initially developed to detect MCI rather than PSCI, it does not encompass the evaluation of certain common cognitive impairments associated with PSCI.[31] Nonetheless, some studies have reported that baseline MoCA scores also effectively identify PSCI. Dong et al. identified baseline MoCA score as an independent risk factor for PSCI occurring 6 months after an acute ischemic stroke. They subsequently developed a predictive model based on baseline MoCA, demonstrating significant diagnostic value.[30] Additionally, another study has indicated that MoCA and MMSE exhibit comparable efficacy in detecting PSCI within 2–3 weeks post-stroke.[32] Our study identified that baseline MMSE possesses predictive value as a screening tool for PSCI. However, the diagnostic classification of PSCI relies exclusively on MMSE thresholds, which have inherent limitations. Although MMSE is effective for assessing the memory and language domains, it demonstrates reduced sensitivity for detecting executive dysfunction and processing speed impairments, which are characteristic of the cognitive profile of vascular cognitive impairment.[33]

During the early post-stroke period, the brain can restore cognitive function through neuroplasticity via the functional reorganization of neural cells, a process that is likely attributable to the plasticity of the nervous system. Numerous studies have corroborated that early cognitive rehabilitation training exerts a beneficial effect on cognitive recovery in patients with PSCI.[34] The inherent plasticity of the central nervous system underpins the theoretical framework for functional training in cognitive rehabilitation, facilitating the brain's ability to reconstruct neural networks and functions through the release of various neurotransmitters. Numerous studies conducted worldwide have substantiated the efficacy of early cognitive training in cognitive rehabilitation, with its therapeutic benefits being widely acknowledged by clinical practitioners.[35] Our research further identified delayed time to rehabilitation admission as an independent predictor for the onset of PSCI. In the absence of proactive treatment for vascular cognitive dysfunction, vascular dementia may progress, which can result in irreversible prognostic deterioration. Consequently, early intervention is imperative for significantly enhancing patient outcomes.

This study identified a higher propensity for PSCI following the presence of left-hemisphere lesions. Consistent with previous research, a significant correlation was observed between left-sided strokes and PSCI incidence, particularly when key brain regions such as the left frontotemporal lobe, left basal ganglia, left internal capsule, and left thalamus were affected.[5,36] Supporting these findings, a prospective cohort study conducted in Brazil, which included 103 patients with

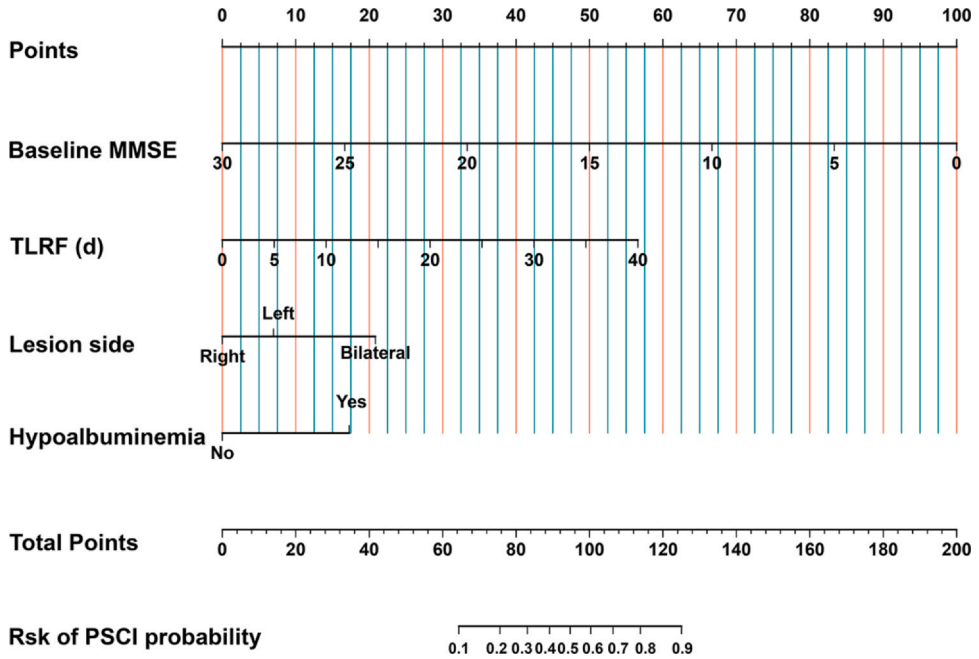


Fig. 3. Nomogram for patients with PSCI. Each variable corresponds to its own score, and the sum of these scores corresponds to the probability of PSCI occurring on the probability axis.
Abbreviations: PSCI, post-stroke cognitive impairment; MMSE, Mini-Mental State Examination; TLRF, time from lesion to rehabilitation facility.

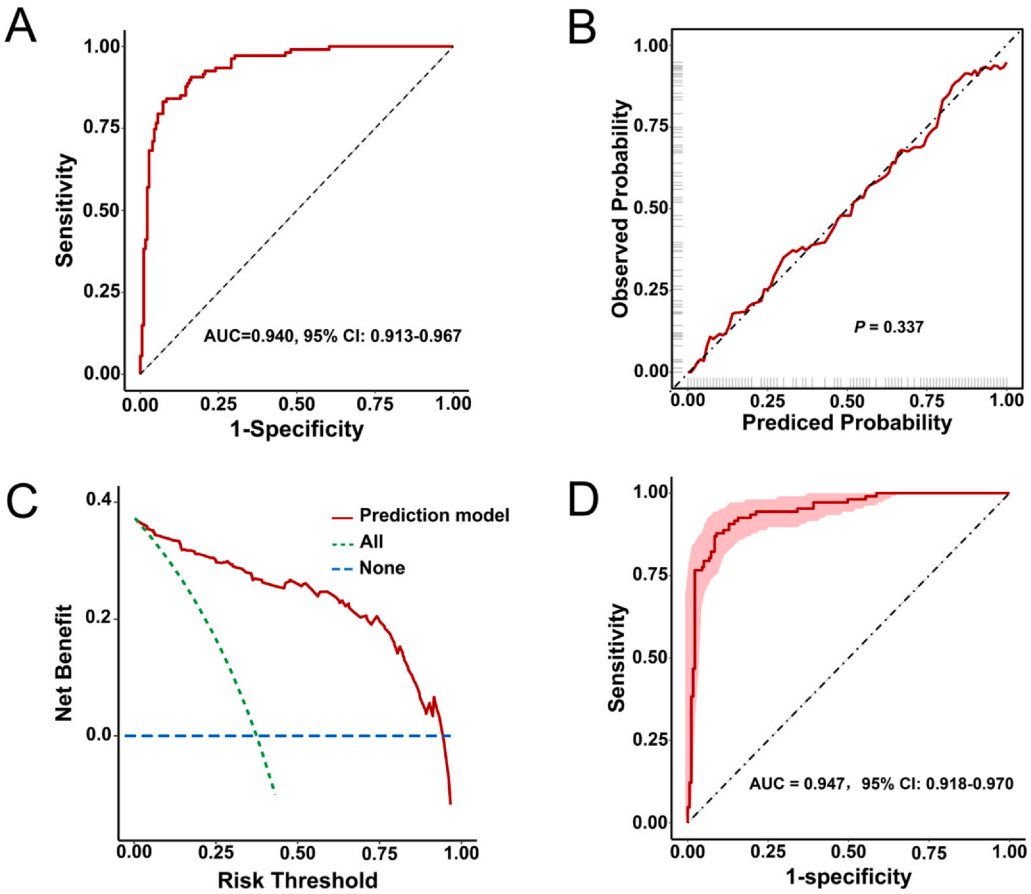


Fig. 4. Performance of the nomogram and internal validation. (A) ROC curve for PSCI nomogram. (B) Calibration curve for PSCI nomogram. (C) DCA for PSCI nomogram. (D) Bootstrap apparent performance of the PSCI nomogram. The ROC curve shows the mean apparent AUC across 1000 bootstrap training samples (mean AUC = 0.947). The optimism-corrected AUC was 0.937, as described in the Results section.
Abbreviations: ROC, receiver operating characteristic; AUC, area under the curve; DCA, decision curve analysis.

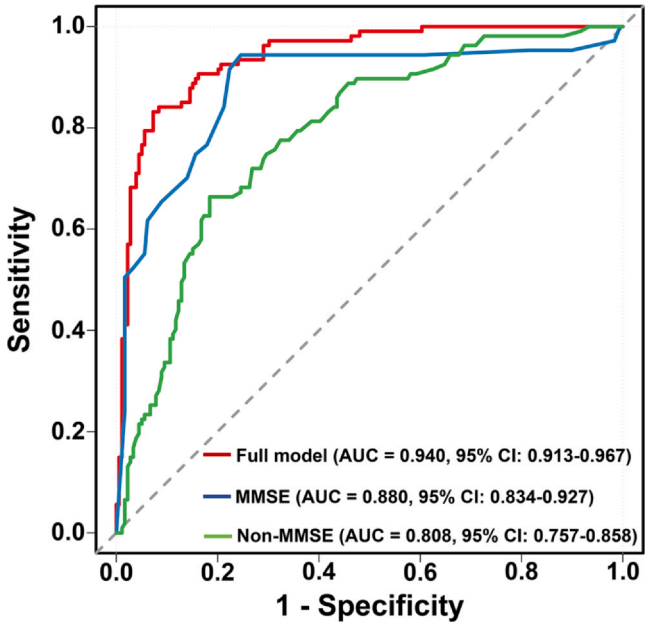


Fig. 5. Comparison of ROC curves among the full model, MMSE-only model, and MMSE-excluded model for predicting PSCI at 6 months. The full model showed significantly higher AUC than the MMSE-only model ($P < 0.001$) and MMSE-excluded model ($P < 0.001$). P values were calculated using the DeLong test.
Abbreviations: ROC, receiver operating characteristic; PSCI, post-stroke cognitive impairment; AUC, area under the curve; MMSE, Mini-Mental State Examination.

subacute stroke, demonstrated a stronger association between left-hemisphere strokes and PSCI compared with right hemisphere strokes.[37] Baccaro et al. conducted a 2-year longitudinal study involving 100 stroke survivors, revealing that left-hemisphere stroke independently predicted PSCI, particularly characterized by diminished computational abilities.[38] Furthermore, a separate investigation encompassing 5888 patients with ischemic stroke demonstrated a significant positive correlation between PSCI and left-hemisphere stroke. Specifically, cognitive decline in patients with left-hemisphere stroke progressed 1.6 times more rapidly than in those with right hemisphere stroke.[39] Given the fact that all patients in this cohort were right-handed, the left-hemisphere constituted the dominant side. This hemisphere encompasses four principal language zones: the motor speech area, auditory language center, writing center, and visual language center.[40] Left-hemisphere lesions were associated with a higher risk of cognitive dysfunction in this right-handed cohort. However, we acknowledge that our left/right/bilateral classification is a crude simplification. Future studies incorporating refined strategic lesion location scoring systems are warranted to enhance predictive precision. The comparison of included versus lost-to-follow-up patients showed comparable baseline characteristics, except for a higher prevalence of right-sided lesions in the latter group; however, the small proportion of patients with incomplete follow-up (8.3%) suggests a limited impact on the overall results.

In this study, hypoalbuminemia was identified as an independent predictor of PSCI. Serum albumin plays a crucial role as an inflammatory regulator and a free radical scavenger in the body. The physiological functions of serum albumin encompass the maintenance of normal osmotic pressure in the blood, as well as the binding and transport of various proteins, fatty acids, steroids, drugs, and toxins.[41] Recent studies have elucidated the significant role of albumin within the central nervous system, including the regulation of astrocyte and microglial activities.[41] Additionally, albumin promotes the repair of ischemic brain tissue through its antioxidant properties.[42] Furthermore, it binds to amyloid-beta protein, thereby mitigating neuronal loss and

synaptic alterations, and reducing microtubule-associated protein aggregation and tau formation.[43] Recent clinical studies, albeit with small sample sizes, have indicated that serum albumin replacement therapy may delay the progression of Alzheimer's disease, demonstrating significant improvements in memory and language functions in patients after 6 months of treatment.[44] Thus, hypoalbuminemia independently predicted PSCI, and albumin may exert a protective effect on compromised brain tissue.

Early identification of high-risk groups and implementation of preventive interventions can significantly reduce PSCI incidence. This reduction is crucial for enhancing the patients' quality of life, extending survival time, and alleviating the overall disease burden. Such measures facilitate the development of tailored rehabilitation and discharge plans, thereby increasing the likelihood of patients returning to work and improving their quality of life. Current guidelines advocate cognitive screening in patients with stroke.[45] Additionally, scholars have developed risk-scoring models to enable early PSCI identification. The $SIGNAL_2$ risk-scoring model, which integrates clinical and neuroimaging variables, including age, education, acute cortical infarcts, white matter hyperintensity, chronic lacunes, global cortical atrophy, and intracranial large vessel stenosis, was utilized to predict PSCI at 3–6 months. The model demonstrated a risk score with an AUC of 0.829.[10] The CHANGE risk-scoring model, which integrates variables including chronic lacunes, hyperintensities, age, non-lacunar cortical infarcts, global atrophy, and education, and exhibits an AUC of 0.820, has demonstrated superior reliability and convenience compared with the $SIGNAL_2$ model for predicting the risk of PSCI in patients with subacute and chronic ischemic stroke.[11] Ding et al. have formulated a prognostic model for PSCI with a predictive window of 6–12 months. The model incorporated variables such as age, years of education, periventricular high-signal classification, diabetes, and number of acute non-lacunar infarctions. The AUC for this model was 0.884.[46] However, these models require uniform neuroimaging data. The use of magnetic resonance angiography or computed tomography angiography poses significant challenges in certain clinical stroke centers. Additionally, the variability in quantitative methodologies for intricate radiological variables such as brain atrophy and white matter hyperintensities further constrains their practical utility. By contrast, our nomogram operated independently of neuroimaging data and demonstrated an AUC of 0.940, thereby enhancing its applicability in both severe stroke wards and community hospital settings. To enable a fair methodological comparison, we also evaluated an MMSE-excluded version of our model (AUC = 0.808), which was comparable to existing models such as $SIGNAL_2$ (0.829) and CHANGE (0.820). A head-to-head comparison with a prospective external validation cohort is necessary to determine whether our model offers superior predictive performance and practical advantages.

In the present study, we developed a nomogram to predict PSCI. A nomogram was constructed from a thorough synthesis of predictors, enabling intuitive and convenient prediction of clinical outcomes.[47] Increasing evidence indicates that nomograms outperform traditional risk scores. In contrast to risk groups, nomograms visualize and provide personalized estimates of specific outcomes for individual patients, helping make medical decisions tailored to individual disease characteristics.[48] This study primarily developed a predictive model utilizing a nomogram, grounded on the identified independent predictors. Healthcare professionals can evaluate the risk of PSCI in patients with stroke by considering specific predictors, thereby enabling early intervention strategies to prevent or mitigate PSCI incidence. This study underscores the importance of early screening and prompt and comprehensive intervention in the management of PSCI. A nomogram based on these predictors can aid clinicians in predicting patients' cognitive outcomes, thereby facilitating informed treatment decisions and customizing personalized treatment plans. High-risk patients require rigorous clinical monitoring and preventive interventions through pharmacological, rehabilitative, or combined approaches. Despite the poor prognosis associated with PSCI, the predictive indicators incorporated into this nomogram, including demographic data, medical history, auxiliary examinations, and clinical evaluations, are readily accessible in routine clinical practice. Early intervention, guided by predictive analytics and targeting modifiable predictors in high-risk patients with PSCI, can effectively prevent the onset of PSCI. Screening individuals at a high risk for PSCI during the early stages of stroke and enhancing comprehensive cognitive assessment scales to accurately gauge the severity of cognitive impairment facilitate timely intervention and thereby offers earlier benefits to patients. Our nomogram provides a practical screening tool for identifying patients at high risk for PSCI, although positive screening results should be followed by comprehensive neuropsychological assessment for definitive diagnosis and tailored rehabilitation planning, as recommended by international guidelines. This is particularly crucial at the 6-month follow-up, as milder cognitive impairments or specific domain deficits may not be fully captured by screening tests alone.

5. Limitations

This study has some limitations. First, the single-center design may have introduced selection and recall biases, particularly as certain patients were excluded during follow-up. Second, because we exclusively analyzed right-handed patients (to control for hemispheric lateralization effects), our findings may not be generalizable to left-handed individuals (approximately 10% of the population). Moreover, the predictive role of left-sided lesions may not be applicable to non-right-handed patients because of the variability in language dominance. Third, the 6-month observation period limited our ability to assess long-term cognitive outcomes. Additionally, a single assessment at 6 months without intermediate time points (e.g., 1 or 3 months) precludes differentiation between transient and persistent PSCI. Fourth, the use of MMSE for both baseline prediction and 6-month outcome assessment may introduce construct overlap, potentially contributing to the high apparent AUC. Nonetheless, the minimal optimism (0.003) indicates limited overfitting, and external validation with alternative cognitive instruments is needed to confirm generalizability. Fifth, although the minimal gap between the apparent AUC (0.940) and optimism-corrected AUC (0.937) with a mean optimism of only 0.003 suggests limited overfitting, the potential risk of overfitting cannot be entirely excluded given the number of candidate predictors relative to the sample size. External validation in independent, multicenter cohorts is therefore essential to confirm the model's generalizability. Sixth, comparison of included versus lost-to-follow-up patients revealed a significantly higher proportion of right-sided lesions in the latter group, which may have introduced selection bias. However, the small proportion of patients lost to follow-up (8.3%) suggests a limited impact on the overall results. Other potential biases include the use of a single cognitive screening tool, and the absence of pre-stroke cognitive assessments. Finally, although internal validation showed good performance, external validation is required to confirm generalizability.

6. Conclusion

As PSCI incidence is high, we developed a nomogram-based risk prediction model to identify PSCI. The proposed model proved applicable in clinical settings and could facilitate consultation with patients with stroke and their families. Although this nomogram demonstrated good internal validity, further prospective studies with external validation are needed to confirm its generalizability and clinical utility before its widespread implementation.

CRediT authorship contribution statement

Chunyan Chen: Writing – original draft, Methodology, and investigation. **Xiangbo Wu:** Writing – original draft and Formal analysis.

Tao Han: Methodology, Formal analysis, and investigation. **Bin Zhang:** Methodology and investigation. **Xiaodong Lin:** Conceptualization. **Guiqing Cheng:** Formal analysis. **Xinyu Liu:** Supervision. **Yixing Lu:** Investigation. **Hangkun Zhao:** Investigation. **Hua Yuan:** Writing – review & editing, and funding acquisition. **Xiaolong Sun:** Writing – review & editing, and funding acquisition. All the authors have read and approved the final version of this manuscript.

Ethical approval

This study was approved by the Ethics Committee of the First Affiliated Hospital of the Air Force Medical University (KY20232243-C-1) and adhered to both Chinese legal requirements and the principles outlined in the Declaration of Helsinki (2024 revision). The study was registered with the Chinese Clinical Trial Registry (ChiCTR) under the identifier ChiCTR2400081107. For the retrospective component, the requirement for informed consent was waived by the Ethics Committee. For the prospective component, written informed consent was obtained from all patients or their legally authorized representatives.

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Data availability

The data supporting the findings of this study can be obtained from the corresponding author upon reasonable request.

Declaration of Competing Interest

The authors declare that they have no conflicts of interest.

Declaration of Generative AI and AI-assisted technologies in the writing process

DeepSeek-V3.2 was used solely for language polishing and formatting, with no involvement in data processing or conclusion generation. All content was reviewed by the authors, who assume full responsibility for this study.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.hcr.2026.100095.

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