Aging Clinical and Experimental Research (2025) 37:145
https://doi.org/10.1007/s40520-025-03060-1

RESEARCH

Establishment and validation of early prediction model for post-stroke dysphagia

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Received: 25 February 2025 / Accepted: 26 April 2025

Abstract

Background Stroke is a leading cause of death and disability worldwide, with dysphagia being a common complication that worsens patient outcomes.

Methods Data from 200 stroke patients (development cohort) and 50 stroke patients (validation cohort) were analyzed to develop a nomogram for predicting post-stroke dysphagia (PSD). Risk factors were identified through univariate analysis, Least Absolute Shrinkage and Selection Operator (LASSO) regression, and multivariate logistic regression.

Results Univariate analysis revealed substantial differences in age, body mass index (BMI), diabetes, atrial fibrillation, National Institute of Health Stroke Scale (NIHSS) score, Activities of Daily Living (ADL) score, lesion site, stroke type, and several laboratory indicators across the groups. Further analysis of individual NIHSS items showed significant differences in consciousness level, best gaze, facial palsy, motor arm, motor leg, dysarthria, etc. LASSO regression identified three predictors: ADL score, motor leg, and dysarthria. Multivariable logistic regression revealed that ADL score [0.96 (0.94–0.97)], motor leg [5.70 (2.14–15.22)], and dysarthria [5.26 (2.00–13.82)] were independent risk factors for PSD. The prediction model's AUC was 0.915 (0.874–0.955), with a sensitivity of 0.920 (0.867–0.973), specificity of 0.800 (0.722–0.878), positive predictive value (PPV) of 0.821 (0.750–0.892), negative predictive value (NPV) of 0.909 (0.849–0.969), and F1 score of 0.859. External validation yielded an AUC of 0.995 (0.984–1.000).

Conclusions and Implications ADL score, motor leg, and dysarthria are independent predictors of PSD. The prediction model based on these factors shows high accuracy, sensitivity, balance, consistency, and clinical applicability. This nomogram can support decision-making for ultra-early rehabilitation care, ultimately improving patient prognosis.

Keywords Stroke · Dysphagia · Swallowing function · Prediction model

Introduction

Stroke is one of the leading causes of disability and death worldwide [1], and post-stroke dysphagia (PSD) is a major complication following acute stroke [2]. Previous studies have demonstrated that the prevalence of PSD varies widely depending on the methods of assessment, with prevalence rates ranging from 37 to 45% in screening studies, 51–55% in clinical assessment, and 64–78% in instrumental assessment [3]. PSD can lead to serious consequences such as malnutrition, dehydration, and aspiration pneumonia [4–7], and significantly prolongs the length of hospitalization and increases readmission rates [3, 8]. A prospective study including more than 820,000 stroke patients reported a markedly increased in-hospital mortality risk in PSD patients (OR = 7.3, 95% CI 6.23–8.61) [9]. Ultra-early rehabilitation care has been shown to have a positive effect on neurological recovery and prevention of related complications in stroke patients [10, 11]. Studies have indicated that initiation of ultra-early rehabilitation within 24 h of acute stroke onset is safe and effective [12]. Therefore, early prediction of PSD

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Published online: 11 May 2025

is crucial for timely intervention and reducing the risk of complications.

Although a variety of screening tools, such as the Kamada Water Swallow Test (KWST) [13], the Volume-Viscosity Swallow Test (V-VST) [14, 15], the Functional Oral Intake Scale (FOIS) [16], and the Video Fluoroscopic Swallowing Study (VFSS) [17], have been widely used, their dissemination in the acute phase has been limited by high cost and operational complexity. Nomograms, however, have been extensively used to predict the risks or prognosis of various diseases. They integrate multiple clinical variables into a quantifiable risk score, providing clinicians with a more intuitive and personalized risk assessment tool [18]. This aids in clinical decision-making and enhanced management of at-risk populations in advance [19]. Previous studies identified several risk factors including the NIHSS score [20–23]. The NIHSS score is widely used to assess the severity of stroke and predict outcomes after stroke [24, 25]. However, the NIHSS score is cumbersome to apply in the prehospital setting because it consists of more than 10 neurological examination items [26]. In addition, the occurrence of PSD is the result of multifactorial interactions, making it difficult for a single indicator to fully predict its occurrence. Current research on early prediction models for PSD is still limited and suffers from insufficient external validation [27] and focus on a single stroke subtype [28, 29]. Therefore, this study aims to identify independent predictive factors based on clinical data, combine various indicators, and develop and validate an early prediction model for PSD, with the goal of improving prediction accuracy and supporting decision-making for ultra-early rehabilitation care, ultimately reducing complication risks and improving patient outcomes.

Methods

Study population

This retrospective study included patients diagnosed with PSD at a Grade A tertiary hospital in Jiaxing between January 2023 and June 2024, forming the development cohort. Additionally, 100 stroke patients without dysphagia were randomly selected during the same period as the control group. To validate the model externally, stroke patients diagnosed at the same hospital between July 2024 and December 2024 were included as an independent validation cohort.

The sample size was determined using the events per variable (EPV) principle, which recommends at least 10 outcome events per predictor variable to ensure model stability and prevent overfitting. Given the inclusion of at least 10 independent variables, a minimum of 100 PSD patients was required. A nested case–control design with a 1:1 ratio was adopted, enrolling 100 PSD patients as cases and 100 non-PSD stroke patients as controls, ensuring statistical power and model robustness.

Inclusion Criteria: Age ≥ 18 years; stroke diagnosis confirmed by CT/MRI; complete clinical and laboratory data; underwent swallowing screening and assessment upon admission. Exclusion Criteria: Large infarction or hemorrhagic stroke with consciousness disturbance or mental disorders; complete aphasia; pharyngeal diseases (e.g., thyroid disorders, ulcers, infections); conditions with bleeding tendencies; severe cardiac, hepatic, or renal diseases; inability to cooperate with exams or treatment. Patients were classified into PSD and non-PSD groups based on swallowing dysfunction.

Data collection

The retrospective data of all patients included: (1) Demographic data: age, gender, body mass index (BMI), blood pressure, heart rate, smoking status, drinking status, and previous medical history (hypertension, diabetes, coronary heart disease, atrial fibrillation, liver disease, kidney disease, cancer, and stroke). The BMI was calculated as weight (kg) divided by height (m) squared. (2) Disease-related data: NIHSS score, Activities of Daily Living (ADL) score, lesion site, stroke type, and Aetiological Trial of Org 10,172 in acute stroke treatment (TOAST) classification. Additionally, all NIHSS items were categorized into two groups: 0 points (No) and ≥ 1 points (Yes). The "level of consciousness" group included response to questions and ability to follow instructions, and points were given for the "motor function of arm" and "motor function of leg" groups, either left or right. NIHSS and ADL scores were assessed by two experienced neurologists at the time of admission. 3) Laboratory data: complete blood count [white blood cells (WBC), neutrophil percentage, lymphocyte percentage, mean platelet volume, C-reactive protein (CRP), hemoglobin], homocysteine (Hcy), coagulation function, liver and kidney function, and other indicators.

Swallowing function assessment

Patients who met the eligibility criteria were screened for dysphagia using the KWST. The KWST is graded from Level 1 to Level 5 (from being able to swallow water smoothly in one attempt without coughing or aspiration to being unable to complete the test, with water spilling from the corner of the mouth or immediate aspiration). Swallowing function was further assessed using the V-VST and FOIS. A positive result on the V-VST was characterized by coughing, hoarseness, wet crackles, delayed swallowing, or residue. The FOIS ranges from Level 1 to Level 7 (from complete inability to take oral food to no restrictions on oral

intake). Severe impairment of oral intake was defined as a single consistency or worse oral diet (FOIS score < 5) [16]. Previous studies have shown that these patients exhibit significantly inadequate energy, protein, and total fluid intake [30, 31], making enteral feeding more suitable.

Statistical analysis

Quantitative data with normal distribution were presented as mean ± SD, and group differences were assessed using the independent two-sample t-test. Data with skewed distribution were expressed as median (Q1, Q3), and group comparisons were made using the Wilcoxon rank-sum test. Categorical data were presented as frequencies (n) and percentages (%), and differences between groups were analyzed using the chi-square or Fisher's exact test. Missing values were handled using Random Forest Imputation, a machine learning-based technique within the Multiple Imputation framework. Unlike traditional methods, such as mean or regression imputation, Random Forest Imputation can automatically address various types of missing data and capture nonlinear relationships among features [32]. Given the complexity and potential nonlinearities in the dataset, this method demonstrated strong adaptability and predictive accuracy while minimizing biases associated with simpler techniques, thereby enhancing data integrity and model performance. Variables with a univariate P-value < 0.05 were further analyzed using the Least Absolute Shrinkage and Selection Operator (LASSO) regression, and selected predictors were included in a binary logistic regression model for multivariate analysis. Independent predictors of PSD were used to construct a prediction model, which was visualized using a nomogram. The model's performance was evaluated using the Receiver Operating Characteristic (ROC) curve and the area under the curve (AUC). Sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and F1 score were calculated. Calibration was assessed using the calibration curve and Hosmer–Lemeshow test, while clinical applicability was determined through Decision Curve Analysis (DCA). Internal validation was performed by bootstrap resampling (1000 iterations), and external validation was conducted using data from 50 hospitalized stroke patients from a different time period. All analyses were performed using R version 4.3.3, with statistical significance set at $P < 0.05$.

Results

Clinical baseline characteristics of stroke patients with and without dysphagia

This study included 200 stroke patients diagnosed at a Grade A tertiary hospital in Jiaxing from January 2023 to June 2024, forming the development cohort. Of these, 100 patients were diagnosed with post-stroke dysphagia (PSD) and 100 without dysphagia as the control group. Baseline characteristics are detailed in Table 1. The mean age of the cohort was 70.33 ± 12.94 years, with 67.5% male. The median NIHSS score was 5 (IQR 2–11), and median ADL score was 45.0 (IQR 15.0–72.5). In the PSD group, the mean age was 73.50 ± 12.50 years, with 64% male, and median NIHSS score was 10 (IQR 5–17). In the NPSD group, the mean age was 73.50 ± 12.50 years, with 55% male, and median NIHSS score was 2 (IQR 0–4).

Compared to the NPSD group, the PSD group had significantly higher values or levels of age, BMI, NIHSS score, white blood cell count, neutrophil percentage, CRP, INR, D-dimer, AST, and total bilirubin, while the ADL score and levels of lymphocyte percentage, hemoglobin, albumin, and albumin-to-globulin ratio were lower ($P < 0.05$). In addition, PSD patients had a higher incidence of diabetes, atrial fibrillation, and hemorrhagic stroke, with lesions occurring more frequently in the anterior or both circulations. The TOAST classification of PSD patients consisted primarily of large-artery atherosclerosis and cardioembolism (Table 1). Among the NIHSS items, the PSD group had markedly more people with level of consciousness, best gaze, facial palsy, motor arm, motor leg, best language, dysarthria, and extinction/inattention scores ≥ 1 (Table 2). Relevant dysphagia assessment indicators were shown in Table S1.

Independent predictors of PSD

Variables that showed significant differences between groups in the univariate analysis (Tables 1 and 2, excluding NIHSS) were included in the LASSO regression for dimensionality reduction. The model with a Lambda value of 0.08831311, which yielded minimal error and a relatively simple structure, was selected (Figs. S1 & S2). This model included three variables: ADL score, motor leg, and dysarthria. Subsequently, multivariate logistic regression revealed that ADL score (OR = 0.96, 95% CI 0.94–0.97), motor leg (OR = 5.70, 95% CI 2.14–15.22), and dysarthria (OR = 5.26, 95% CI 2.00–13.82) were independent risk factors for dysphagia in stroke patients ($P < 0.05$) (Table 3). The collinearity test indicated that the variance inflation factors (VIFs) were less than 5 for all variables (Table S2).

Establishing a new PSD prediction model

Based on the results of the multivariate analysis, the logistic regression equation was derived as follows, where I indicates that I is 1 if dysarthria or motor leg impairments is present (score ≥ 1), and 0 otherwise.

Table 1 Baseline characteristics of the NPSD and PSD groups

Variables	Total (n = 200)	Patients		$\chi 2$/Z/T	*P* value
		NPSD group (n = 100)	PSD group (n = 100)		
Age, years	70.33 ± 12.94	67.15 ± 12.65	73.50 ± 12.50	– 3.571	< 0.001
Gender (Male), n (%)	135 (67.50)	71 (71.00)	64 (64.00)	1.117	0.291
BMI, kg/m2	23.82 ± 3.51	24.30 ± 3.31	23.17 ± 3.68	2.098	0.037
SBP, mmHg	155.81 ± 22.54	154.28 ± 21.13	157.34 ± 23.87	– 0.960	0.338
DBP, mmHg	84.61 ± 13.95	85.62 ± 13.53	83.61 ± 14.36	1.019	0.310
Heart Rate, beats per minute	80 (72, 92)	78 (71, 90)	82 (73, 92)	– 0.830	0.407
Smoking, n (%)				2.741	0.098
No	152 (76.00)	71 (71.00)	81 (81.00)		
Yes	48 (24.00)	29 (29.00)	19 (19.00)		
Drinking, n (%)				1.778	0.182
No	167 (83.50)	80 (80.00)	87 (87.00)		
Yes	33 (16.50)	20 (20.00)	13 (13.00)		
Hypertension, n (%)					
No	67 (33.50)	39 (39.00)	28 (28.00)	2.716	0.099
Yes	133 (66.50)	61 (61.00)	72 (72.00)		
Diabetes, n (%)				5.643	**0.018**
No	145 (72.50)	80 (80.00)	65 (65.00)		
Yes	55 (27.50)	20 (20.00)	35 (35.00)		
Coronary heart disease, n (%)				0.000	1.000
No	195 (97.50)	98 (98.00)	97 (97.00)		
Yes	5 (2.50)	2 (2.00)	3 (3.00)		
Atrial fibrillation, n (%)				5.531	**0.019**
No	175 (87.50)	93 (93.00)	82 (82.00)		
Yes	25 (12.50)	7 (7.00)	18 (18.00)		
Liver disease, n (%)				0.000	1.000
No	198 (99.00)	99 (99.00)	99 (99.00)		
Yes	2 (1.00)	1 (1.00)	1 (1.00)		
Kidney disease, n (%)				0.130	0.718
No	192 (96.00)	97 (97.00)	95 (95.00)		
Yes	8 (4.00)	3 (3.00)	5 (5.00)		
Malignancy, n (%)				0.172	0.678
No	194 (97.00)	98 (98.00)	96 (96.00)		
Yes	6 (3.00)	2 (2.00)	4 (4.00)		
Previous stroke history, n (%)				2.823	0.093
No	154 (77.00)	82 (82.00)	72 (72.00)		
Yes	46 (23.00)	18 (18.00)	28 (28.00)		
NIHSS	5 (2, 11)	2 (0, 4)	10 (5, 17)	– 8.835	< 0.001
ADL	45.0 (15.0, 72.5)	70 (45, 90)	20 (10, 45)	– 9.061	< 0.001
Lesion site, n (%)				6.308	0.043
Anterior circulation	133 (66.50)	65 (65.00)	68 (68.00)		
Posterior circulation	56 (28.00)	33 (33.00)	23 (23.00)		
Anterior and posterior circulation	11 (5.50)	2 (2.00)	9 (9.00)		
Stroke type, n (%)				4.188	0.041
Hemorrhagic	9 (4.50)	1 (1.00)	8 (8.00)		
Ischemic	191 (95.50)	99 (99.00)	92 (92.00)		
TOAST classification				28.444	< 0.001
Large-artery atherosclerosis	108 (54.00)	44 (44.00)	64 (64.00)		
Small-vessel occlusion	44 (22.00)	36 (36.00)	8 (8.00)		

Table 1 (continued)

Variables	Total (n = 200)	Patients		χ2/Z/T	*P* value
		NPSD group (n = 100)	PSD group (n = 100)		
Cardioembolism	31 (15.50)	9 (9.00)	22 (22.00)		
Other/unknown	17 (8.50)	11 (11.00)	6 (6.00)		
WBC, × 10^9/L	7.12 (5.96, 9.01)	6.74 (5.84, 7.96)	7.69 (6.23, 9.99)	– 3.109	0.002
Neutrophil percentage, %	71.03 ± 12.18	67.05 ± 11.93	75.01 ± 11.13	– 4.877	< 0.001
Lymphocyte percentage, %	19.15 (12.38, 25.23)	22.40 (16.03, 26.95)	15.55 (9.65, 22.32)	– 4.599	< 0.001
Mean platelet volume	10.35 (9.60, 11.20)	10.25 (9.40, 11.22)	10.45 (9.70, 11.12)	– 1.302	0.193
CRP	6.26 (2.14, 19.88)	3.37 (1.34, 8.69)	10.25 (3.34, 35.67)	– 4.754	< 0.001
Hb	128.78 ± 18.41	133.03 ± 17.06	124.54 ± 18.82	3.342	< 0.001
Hcy	14.29 (10.84, 18.81)	13.43 (10.41, 18.17)	15.02 (11.14, 19.43)	– 1.732	0.083
APTT	31.50 (26.20, 36.00)	32.50 (26.90, 35.20)	31.00 (25.42, 37.02)	– 0.666	0.505
PT	12.72 ± 1.41	12.55 ± 1.28	12.89 ± 1.52	– 1.669	0.097
INR	1.02 (0.97, 1.09)	1.01 (0.96, 1.06)	1.03 (0.98, 1.11)	– 2.238	0.025
D-dimer	0.55 (0.27, 1.41)	0.42 (0.24, 0.75)	1.02 (0.35, 2.14)	– 4.523	< 0.001
ALT	15.00 (11.00, 23.25)	14.00 (10.75, 21.25)	16.50 (11.00, 25.00)	– 1.316	0.188
AST	19.00 (15.00, 27.25)	17.00 (14.00, 22.00)	23.50 (17.75, 32.00)	– 4.875	< 0.001
Alkaline phosphatase	73.00 (60.00, 92.25)	73.00 (62.00, 92.00)	73.00 (60.00, 96.00)	– 0.098	0.922
GGT	26.00 (18.75, 42.25)	24.00 (18.00, 37.75)	27.50 (20.00, 43.75)	– 1.755	0.079
TB	16.30 (11.30, 24.75)	14.50 (10.15, 22.05)	18.40 (13.35, 25.85)	– 2.871	0.004
TP	63.20 (59.58, 68.03)	63.20 (59.55, 68.82)	63.10 (59.58, 66.85)	– 0.821	0.412
Albumin	36.87 ± 4.78	37.71 ± 4.16	36.02 ± 5.22	2.522	0.012
Globulin	26.50 (24.25, 29.02)	26.10 (24.05, 28.15)	26.70 (24.37, 29.85)	– 1.140	0.254
A/G Ratio	1.40 ± 0.25	1.45 ± 0.23	1.35 ± 0.26	2.816	0.005
Uric acid	319.03 ± 123.21	332.91 ± 95.53	305.02 ± 145.12	1.599	0.112
Cr	74.44 (64.66, 86.39)	75.39 (65.51, 88.30)	73.98 (62.58, 85.80)	– 0.464	0.643

Values are mean (SD), median (IQR) or number (%) as appropriate

PSD Post-stroke dysphagia, *NPSD* Stroke patients without dysphagia, *BMI* Body Mass Index, *SBP* Systolic blood pressure, *DBP* Diastolic blood pressure, *NIHSS* National Institutes of Health Stroke Scale (scores range from 0 to 42, with higher scores indicating severe stroke), *ADL* Activities of Daily Living, *TOAST* Aetiological Trial of Org 10,172 in acute stroke treatment (TOAST) classification, *WBC* White blood cell, *CRP* C-reactive protein, *Hb* haemoglobin, *Hcy* Homocysteine, *APTT* Activated partial thromboplastin time, *PT* Prothrombin time, *INR* International normalized ratio, *ALT* Alanine aminotransferase, *AST* Aspartate aminotransferase, *GGT* Gamma-glutamyl transferase, *TB* Total bilirubin, *TP* Total protein, *A/G Ratio* Albumin/Globulin Ratio, *Cr* Creatinine

$$\begin{aligned}\log it(P) &= -0.443084 - 0.042669 \times \mathrm{ADL} \\ &+ 1.741108 \times I(\text{Motor leg} = \text{Yes}) \\ &+ 1.660656 \times I(\text{Dysarthria} = \text{Yes})\end{aligned}$$

The prediction model for PSD is presented in the form of a nomogram (Fig. 1). The AUC of the model was 0.915 (95% CI 0.874–0.955), indicating a high predictive accuracy (Figure S3). Additionally, the model demonstrates a sensitivity of 0.920 (0.867–0.973), specificity of 0.800 (0.722–0.878), PPV of 0.821 (0.750–0.892), NPV of 0.909 (0.849–0.969), and F1 score of 0.859 (Table S3).

Evaluating the clinical utility of PSD prediction model

The DCA of the PSD prediction model showed that when the threshold probability was greater than 0, the model curve was above the two extreme lines. This indicates that timely clinical intervention when the model predicts a risk of PSD for patients can be beneficial, demonstrating good clinical value (Fig. S4).

Internal validation of the models

The Bootstrap method was used to repeatedly resample the model 1000 times for internal validation. The AUC value was 0.915 (95% CI 0.874–0.955), indicating stable model performance (Fig. 2). The calibration curve showed that the observed curve closely approximated the ideal curve. The Hosmer–Lemeshow test yielded a

Table 2 NIHSS items of the NPSD and PSD groups at baseline

Variables	Total (n = 200)	Patients		$\chi 2$/Z/T	P value
		NPSD group (n = 100)	PSD group (n = 100)		
Level of consciousness, n (%)				23.104	< 0.001
0	141 (70.50)	86 (86.00)	55 (55.00)		
≥ 1	59 (29.50)	14 (14.00)	45 (45.00)		
Best Gaze, n (%)				14.324	< 0.001
0	174 (87.00)	96 (96.00)	78 (78.00)		
≥ 1	26 (13.00)	4 (4.00)	22 (22.00)		
Visual fields, n (%)				0.255	0.614
0	196 (98.00)	97 (97.00)	99 (99.00)		
≥ 1	4 (2.00)	3 (3.00)	1 (1.00)		
Facial Palsy, n (%)				10.895	< 0.001
0	83 (41.50)	53 (53.00)	30 (30.00)		
≥ 1	117 (58.50)	47 (47.00)	70 (70.00)		
Motor arm, n (%)				60.598	< 0.001
0	73 (36.50)	63 (63.00)	10 (10.00)		
≥ 1	127 (63.50)	37 (37.00)	90 (90.00)		
Motor leg, n (%)				68.056	< 0.001
0	72 (36.00)	64 (64.00)	8 (8.00)		
≥ 1	128 (64.00)	36 (36.00)	92 (92.00)		
Ataxia, n (%)				0.000	1.000
0	198 (99.00)	99 (99.00)	99 (99.00)		
≥ 1	2 (1.00)	1 (1.00)	1 (1.00)		
Sensory, n (%)				1.684	0.194
0	190 (95.00)	93 (93.00)	97 (97.00)		
≥ 1	10 (5.00)	7 (7.00)	3 (3.00)		
Best language, n (%)				22.733	< 0.001
0	151 (75.50)	90 (90.00)	61 (61.00)		
≥ 1	49 (24.50)	10 (10.00)	39 (39.00)		
Dysarthria, n (%)				57.551	< 0.001
0	69 (34.50)	60 (60.00)	9 (9.00)		
≥ 1	131 (65.50)	40 (40.00)	91 (91.00)		
Extinction/inattention, n (%)				20.994	< 0.001
0	181 (90.50)	100 (100.00)	81 (81.00)		
≥ 1	19 (9.50)	0 (0.00)	19 (19.00)		

Values are mean (SD), median (IQR) or number (%) as appropriate

PSD Post-stroke dysphagia, *NPSD* Stroke patients without dysphagia, *NIHSS* National Institutes of Health Stroke Scale (scores range from 0 to 42, with higher scores indicating severe stroke)

P-value of 0.192, suggesting good consistency between the predicted values from the nomogram and the actual occurrence of dysphagia in stroke patients (Fig. S5).

External validation of the model

External validation of the model was performed in 50 hospitalized stroke patients at another time period. The median ADL score of the validation cohort was 35.0 (IQR 15.0–82.5). The median NIHSS score of the validation cohort was 5 (IQR 2–13), with 30 patients having a motor

Table 3 Multivariate logistic regression analysis results of independent predictors

Variables	Beta	S.E	Z	P	OR (95%CI)
ADL	– 0.04	0.01	– 4.98	< 0.001	0.96 (0.94–0.97)
Motor leg					
0					1.00 (Reference)
≥ 1	1.74	0.50	3.48	< 0.001	5.70 (2.14–15.22)
Dysarthria					
0					1.00 (Reference)
≥ 1	1.66	0.49	3.37	< 0.001	5.26 (2.00–13.82)

ADL Activities of Daily Living, *OR* Odds Ratio, *CI* Confidence Interval

leg score ≥ 1 and 35 patients having a dysarthria score ≥ 1 (Table S3). The AUC for the model was 0.995 (95% CI 0.984–1.000) (Fig. S6), reflecting the excellent accuracy of the nomogram.

Discussion

Stroke is prevalent worldwide, with an increasing incidence rate [33, 34]. Dysphagia occurs in nearly half of acute stroke patients [35] and is significantly associated with poor prognosis and increased mortality [9, 36]. The pathophysiology of PSD involves both central and peripheral sensory and motor system impairments, including reduced pharyngeal sensation leading to delayed or absent swallowing reflexes [37], impaired coordination of sensory input and motor feedback [38, 39], disruption of central nervous network functional connectivity [40], and compensatory neuroplasticity remodeling in the contralateral hemisphere [41, 42]. These changes collectively affect the recovery of swallowing function. This study, based on data from hospitalized stroke patients, employed univariate analysis, LASSO regression, and binary logistic regression. Among 53 candidate predictive factors, ADL score, motor leg, and dysarthria were identified as independent predictors of PSD.

The ADL assessment is used to measure an individual's ability to independently perform basic daily activities, including grooming/personal hygiene, dressing, toileting, mobility/walking, and eating [43]. It is commonly used in the nursing assessment of elderly and functionally impaired individuals. Tools like the Barthel Index have demonstrated high reliability in stroke patients [44, 45]. By assessing ten basic activities, ADL quantifies an individual's functional independence, with a maximum score of 100. The higher the score, the greater the independence, providing important guidance for clinical care and rehabilitation. Lee et al. found that ADL score was a predictive factor for the removal of nasogastric tubes in PSD patients [46]. In addition, robust ADL capacity has been recognized as a potential marker of healthy aging [47]. A decline in ADL is a critical sign of pre-frailty, and frailty in the elderly can negatively impact swallowing function [48]. A meta-analysis revealed a significant association between ADL decline and dysphagia in individuals aged 65 and older, further supporting the role of ADL scores as a protective factor for swallowing function [49].

The NIHSS score is a key indicator of stroke severity. Previous studies have found that the NIHSS score is more effective in predicting dysphagia than screening tools based on admission [22]. Besides, the NIHSS score may be more sensitive than the 3-sip water test [23]. In a study by Mattavelli et al., patients with PSD at discharge had an NIHSS score of 10 (6.5, 17.5) [28], which is similar to our findings. Another study also identified identified the NIHSS score as one of the predictors of PSD prognosis within one month

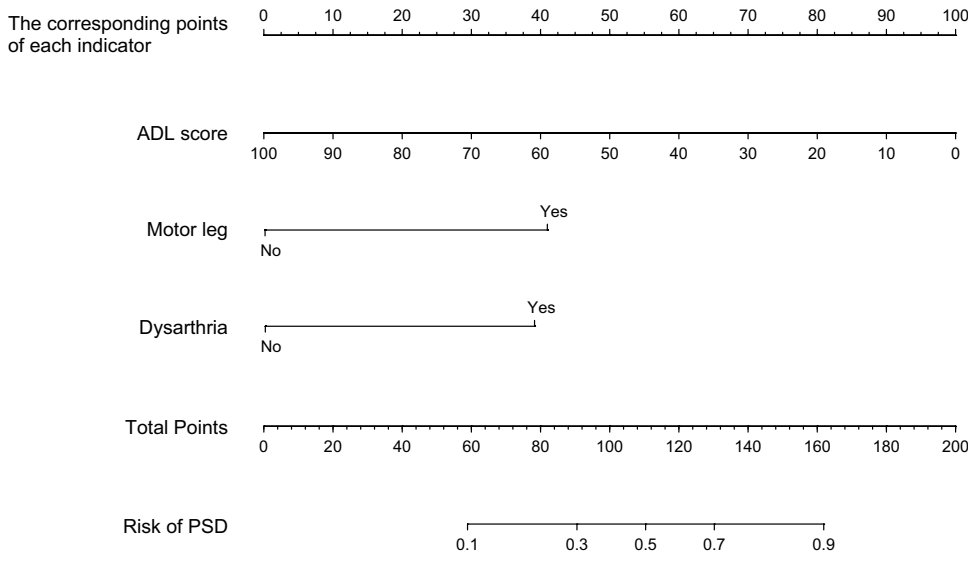

Fig. 1 The nomogram for the prediction model for PSD. Points are assigned based on the values of ADL, dysarthria, and motor leg by finding the corresponding values on the scales and projecting a vertical line to the "Points" scale. These points are then summed, and the total score is marked on the "Total Points" scale. A vertical line is projected from the "Total Points" scale to the "Risk of PSD" scale to determine the PSD risk

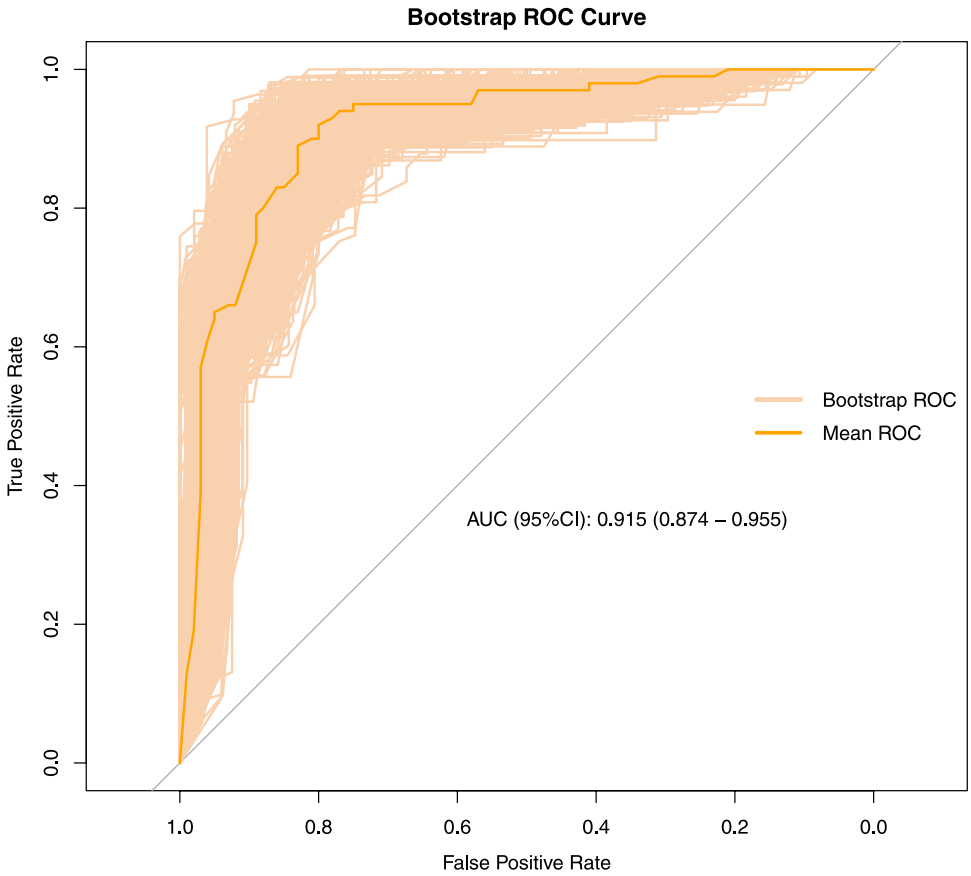

Fig. 2 ROC curve of Bootstrap internal validation for the PSD prediction model

after stroke onset [16]. Notably, most studies have focused on the total NIHSS score, with few exploring the role of individual NIHSS items in predicting PSD. Our study found that incorporating two individual items from the NIHSS score,"motor leg"and"dysarthria", into the predictive model not only simplified the model but also made it more applicable in clinical practice. Stroke typically leads to damage in multiple functional areas, including the cerebral cortex, corticospinal tract, and basal ganglia, which are involved in both limb motor control [50, 51] and swallowing coordination [52]. Neuroimaging studies have shown that different motor pathways are involved in upper and lower limb motor functions [53–55], with subcortical and spinal contributions having a greater impact on lower limb function [56]. One study indicated that lesions in the subcortical region are more likely to cause swallowing difficulties than those in the cortical regions [57], but the exact mechanisms remain to be further investigated. Therefore, lower limb motor impairment not only reflects the extent of local neural damage but also suggests the limitation of the overall neural network function, potentially leading to dysregulation of swallowing function. Additionally, hemiparesis is an important factor influencing the development of complications such as malnutrition in stroke patients [58]. Nishio M et al. evaluated 113 patients with speech difficulties and found a high correlation between swallowing function and all levels of speech intelligibility. The prevalence of concomitant dysphagia in patients with dysarthria was quite high irrespective of the primary etiology and time of onset, and its incidence and severity varied according to the type of dysarthria [59]. Additionally, it has been shown that dysarthria is strongly associated with an increased risk of PSD in both ischemic and hemorrhagic stroke patients [60, 61]. In children, oral and pharyngeal dysfunctions are often accompanied by more pronounced symptoms of dysarthria [62]. Moreover, a retrospective analysis explored the shared neural mechanisms between speech motor disorders and the risk of swallowing dysfunction in left hemisphere stroke patients. The results showed that this co-occurrence may reflect the damage to closely related neural structures, especially when acute stroke involves Broca's area [63].

In addition, we found that the albumin levels in patients with PSD were significantly lower. This finding is consistent with previous literature. Low albumin levels are commonly regarded as a marker of malnutrition [64] and are strongly associated with poor outcomes and mortality [65]. Zhang et al. an association between dysphagia and reduced albumin levels in stroke patients [20]. Therefore,

nutritional assessment and management are crucial for stroke patients, especially in the acute phase, as malnutrition after stroke may worsen over time [58]. We also observed a remarkable difference in age between the two groups. As age increases, muscle atrophy [66] and decreased connective tissue elasticity [67] lead to loss of strength and range of motion. A meta-analysis also identified age as a predictive factor for persistent dysphagia [68]. Furthermore, patients with hemorrhagic stroke were more likely to develop PSD, despite the small sample size in the study. This is consistent with the findings of a meta-analysis, which showed that the risk of PSD in hemorrhagic stroke patients was 1.52 times higher than in ischemic stroke patients [35]. Moreover, it is well-established that inflammatory markers such as CRP and white blood cell count increase during the acute phase of stroke and may persist for approximately three months [69], which are associated with stroke severity and poor functional prognosis [70, 71]. Microaspiration is a common source of inflammation in stroke patients receiving enteral feeding, and the degree of aspiration correlates with the severity of dysphagia [72]. This also explains the association between elevated inflammation levels and PSD.

We developed a nomogram prediction model for PSD based on ADL score, motor leg, and dysarthria, achieving high sensitivity (92.0%) and specificity (80.0%). Both internal and external validation confirmed that the model demonstrates good discriminatory power and calibration. The model requires only simple scoring and clinical symptom assessment, with results available within 24 h of admission, facilitating the early identification of stroke severity and providing timely alerts and support for clinical decision-making and early rehabilitation care. The PSD prediction model developed in this study had an AUC of 0.915 (95% CI 0.874–0.955), which is higher than previous models [28, 29], and included external validation, which improves the model's reliability and generalization ability, and provides a better performance compared to another model [27].

This study has several limitations. First, as a retrospective study, it may be subject to selection bias. Second, although the external validation results demonstrate good performance of the model, this study is a single-center study, and its applicability needs further validation in patients from different regions and ethnic backgrounds to ensure the model's generalizability and applicability. Third, this study focused mainly on the prediction of the baseline model and failed to assess the dynamic risk changes over time. Therefore, future studies should consider evaluating time series data to explore the predictive performance of the model at different time points. Finally, the relatively small sample size of this study may affect the robustness of the results. Future researches with multi-center and large sample sizes should be conducted to further validate the results of this study and explore more potential early predictors of PSD.

Conclusion

This study developed and validated a novel early prediction model for PSD based on ADL score, motor leg, and dysarthria. The model shows high accuracy, sensitivity, balance and consistency, providing a tool for early screening of high-risk patients and implementation of ultra-early rehabilitation care. Future studies should expand the sample size and validate the model's generalizability across multicenter and diverse populations.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s40520-025-03060-1.

Author contributions C.L.: collecting and processing data, statistical analysis of results; Y.Z.: statistical analysis of results, writing papers; Y.S.: collecting and processing data, revising papers; Y.W.: revising papers; S.Q.: supervision, reviewing writing. All authors reviewed the manuscript.

Funding This project was sponsored by the Jiaxing Science and Technology Program (2024 AD30069).

Data availability No datasets were generated or analysed during the current study.

Declarations

Conflict of interest The authors declare no competing interests.

Ethics statement The study was approved by the Ethics Committee of the Second Affiliated Hospital of Jiaxing University and complied with the Helsinki Declaration. Written informed consent for participation was not required for this study in accordance with the national legislation and the institutional requirements.

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