Archives of Rehabilitation Research and Clinical Translation (2022) 4, 100213

ACRM
AMERICAN CONGRESS OF REHABILITATION MEDICINE

Archives of Rehabilitation Research and Clinical Translation

Archives of Rehabilitation Research and Clinical Translation 2022;4:100213

Available online at www.sciencedirect.com

Original Research

Development and Validation of a Nomogram to Predict Hemiplegic Shoulder Pain in Patients With Stroke: A Retrospective Cohort Study

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KEYWORDS
Hemiplegia;
Nomograms;
Rehabilitation;
Risk factors;
Shoulder Pain;
Stroke

Abstract *Objective:* The development and validation of a nomogram for the individualized prediction of hemiplegic shoulder pain (HSP) during the inpatient rehabilitation of patients with stroke.
Design: Retrospective cohort study.
Setting: The rehabilitation department at a tertiary hospital.
Participants: A total of 376 patients (N=376) with stroke admitted to inpatient rehabilitation from January 2018 to April 2021 were included in this study.
Interventions: Not applicable.
Main Outcome Measures: The outcome measure was shoulder pain on the patients' hemiplegic side occurring at rest or with movement during hospitalization.
Results: Among the 376 patients with stroke, 113 (30.05%) developed HSP. Five independent predictors were included in the nomogram: subluxation, Brunnstrom stage, hand edema, spasticity, and sensory disturbance. The nomogram was a good predictor, with a C-index of 0.85 (95% confidence interval, 0.81-0.89) and corrected C-index of 0.84. The Homer-Lemeshow test (χ^2=13.854, *P*=.086) and calibration plot suggested good calibration ability of the nomogram. The optimal cutoff value for the predicted probability of HSP was 0.30 (sensitivity, 0.73; specificity, 0.83). Moreover, the decision curve analysis revealed that the nomogram would add net clinical benefits if the threshold possibility of HSP risk was from 5%-88%.
Conclusions: Our nomogram could accurately predict HSP, which may help clinicians accurately quantify the HSP risk in individuals and implement early interventions.

List of abbreviations: CI, confidence interval; DCA, decision curve analysis; HSP, hemiplegic shoulder pain; OR, odds ratio.
Disclosures: none
Cite this article as: Arch Rehabil Res Clin Transl. 2022;4:100213
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https://doi.org/10.1016/j.arrct.2022.100213

Hemiplegic shoulder pain (HSP) is a common disabling complication that often occurs between 2 and 3 months post stroke.[1] Previous studies have reported incidences of HSP ranging from 9%-73%. HSP in patients with stroke may result in negative effects, such as interference with the rehabilitation process, decreased quality of life, poor functional recovery of the upper limbs, and prolonged hospital stay.[1-3] The complex and multifactorial nature of HSP frequently complicates treatment.[4] Therefore, early and accurate identification of patients at a high risk of HSP is essential.

HSP is associated with several factors. Many clinical features have been reported as predictors, including shoulder subluxation, spasticity, sensory disturbance, stroke type, and restricted range of motion.[5-7] A recent systematic review revealed that decreased motor activity of the upper extremities, diabetes, and shoulder pain history were significant predictors of HSP.[8] Nonetheless, the precise mechanism of HSP remains unclear, and its predictors emerge from various sources, posing a major challenge in the identification of high-risk groups for HSP. Previous studies have developed multivariate models to identify independent predictors of HSP and consequently screen potentially high-risk populations.[2,3,9,10] However, obtaining individualized HSP risk probabilities is difficult using these models because they generally lack comprehensive evaluations of their performance parameters, such as discrimination, calibration, and clinical usefulness. The clinical use of these models is limited. Therefore, a simple and practical predictive model that integrates HSP-associated predictors is required.

A nomogram is a graphical display tool of a multivariable model used to visualize the relative contributions of each predictor to the outcome event.[11] A complex mathematical model can be converted into a continuous scoring system for the quantification of the individualized risk probability of specific diseases or events.[12] It has more advantages than traditional scoring systems, such as user-friendly graphical interfaces, increased accuracy, and easily comprehensible features.[13] In recent years, nomograms have been used in patients with stroke at the convalescence stage, including the risk evaluation of complications and the prognosis prediction after stroke. Wang et al[14] developed a nomogram for predicting dysphagia recovery in patients with stroke used to assist the decision making of enteral nutrition to achieve individualized swallowing rehabilitation. Zhang et al[15] also created a nomogram for the quantification of risks of unfavorable outcomes in patients with stroke undergoing mechanical thrombectomies, allowing clinicians to accurately assess the unfavorable outcome risks using easily accessible clinical parameters. Therefore, this study aimed to develop and internally validate a nomogram for the prediction of HSP in patients with stroke during inpatient rehabilitation. This nomogram is expected to help clinicians accurately predict the risks of HSP and implement early interventions.

Methods

Study design and participants

This retrospective cohort study was conducted in compliance with the Transparent Reporting of a Multivariate Prediction Model for Individual Prognosis or Diagnosis.[16] The medical records of 376 patients with stroke admitted to the rehabilitation department of our hospital from January 2018 to April 2021 were reviewed. The inclusion criteria were as follows: (1) first occurrence of stroke confirmed by magnetic resonance imaging examination or brain computed tomography scan and 1-sided paralysis; (2) older than 18 years; (3) stable medical condition for at least 48 hours post stroke; and (4) regular rehabilitation therapy on the upper limbs before the occurrence of HSP. The exclusion criteria were as follows: (1) previous brain injury or other neurologic disorders, such as brain tumors, Parkinson disease, or Alzheimer disease; (2) history of shoulder pain on the affected side before rehabilitation treatment; (3) other pathologies compatible with the clinical characteristics of HSP, such as inflammatory arthritis, muscular dystrophy, or peripheral neuropathy; (4) severe cognitive impairment; (5) severe organic disease or psychiatric impairment; and (6) missing data.

Data collection and variable definition

A self-designed Excel sheet was used to record detailed clinical characteristics. The typical predictors for HSP including age, sex, disease course on admission, length of stay, stroke type (ischemic or hemorrhagic), affected side (left or right), hypertension, diabetes mellitus, arm strength (manual muscle testing), Brunnstrom stage, subluxation, spasticity, sensory disturbance, and hand edema were collected at admission. Hypertension was diagnosed based on the 2010 Chinese guidelines to manage hypertension.[17] Diabetes mellitus was diagnosed using the 1999 World Health Organization's Diabetes Mellitus diagnosis and classification criteria.[18] The upper limb motor function was assessed using the Brunnstrom stage (upper limb score). Shoulder subluxation of the affected side was assessed by palpating the space between the acromion and humeral head while the hemiplegic arm hung freely. Subluxation was defined as present when the gap between the acromion and humerus exceeded half a finger.[19] Spasticity was measured at the shoulder flexor muscles and defined as a score on the Modified Ashworth Scale $\geq$1.[9] Sensory disturbance was defined as an abnormal superficial sensation or proprioceptive sensation at the affected side and diagnosed when the sensation was reported as increased or decreased.[5] The superficial sensation was assessed by evaluating factors such as light touch, cold perception, and sensation of sharpness of the affected side, using the contralateral side as a reference. Proprioception was assessed using joint position sense at the thumb of both hands. The presence of hand edema was

determined by visual inspection. Shoulder pain on the hemiplegic side occurring at rest or with movement during hospitalization was the measured outcome.[20] The clinical data were extracted into an Excel spreadsheet by an independent researcher uninvolved in the patients' treatments. Subsequently, patients' identifying information was removed and saved in a new data sheet, thereby blinding the data. Two researchers independently reviewed the new data set, coded all the relevant variables, and crosschecked the data. This study was approved by the ethics hospital's committee (ethics no.: KL901296, date: May 12, 2021), which authorized the collection of clinical data. The requirement for informed consent was waived by the ethics committee because of the retrospective nature of the study.

Sample size

We calculated the sample size using the method previously proposed by Peduzzi et al,[21] using events per variable of 10 as the optimal value. At least 90 patients with HSP events were required for the evaluation of 9 candidate predictors. An HSP prevalence at 35%[7] required a minimum sample size of 257.

Statistical analysis

The statistical analyses was performed using SPSS 22.0[a] and R 4.0.5.[b] The data are presented as the median (interquartile range) for nonnormally distributed continuous variables, the mean and standard deviation for normally distributed continuous variables and as frequencies (percentages) for categorical variables. Mann-Whitney *U* tests or independent *t* tests were used to analyze the continuous variables, and Fisher exact tests or χ^2 tests were used to analyze the categorical variables between groups. To determine the independent HSP predictors, the variables obtained from the univariate analysis ($P<.05$) were subsequently subjected to multivariable logistic regression analysis. The resulting variables from the multivariate analysis with $P<.1$ were applied in the construction of the nomogram using the R software with the "rms" package. To reduce the overfitting bias, an internal validation procedure was performed using 1000 bootstrap resamples. Furthermore, a web application facilitating the use of nomograms was developed using the "DynNom" package in the R software. Statistical significance was set at $P<.05$ (2-sided).

The C-index was calculated to evaluate the discriminatory power of the nomogram and ranged from 0.5-1. Higher C-indices indicate models with high discrimination abilities. Therefore, a value of 0.5 represented no discrimination, 0.7-0.8 represented acceptable discrimination, 0.8-0.9 represented excellent discrimination, and >0.9 represented outstanding discrimination.[22] A calibration curve and the Hosmer-Lemeshow test were performed to evaluate the nomogram's calibration ability. The calibration curve reflects the consistency between the predicted and actual

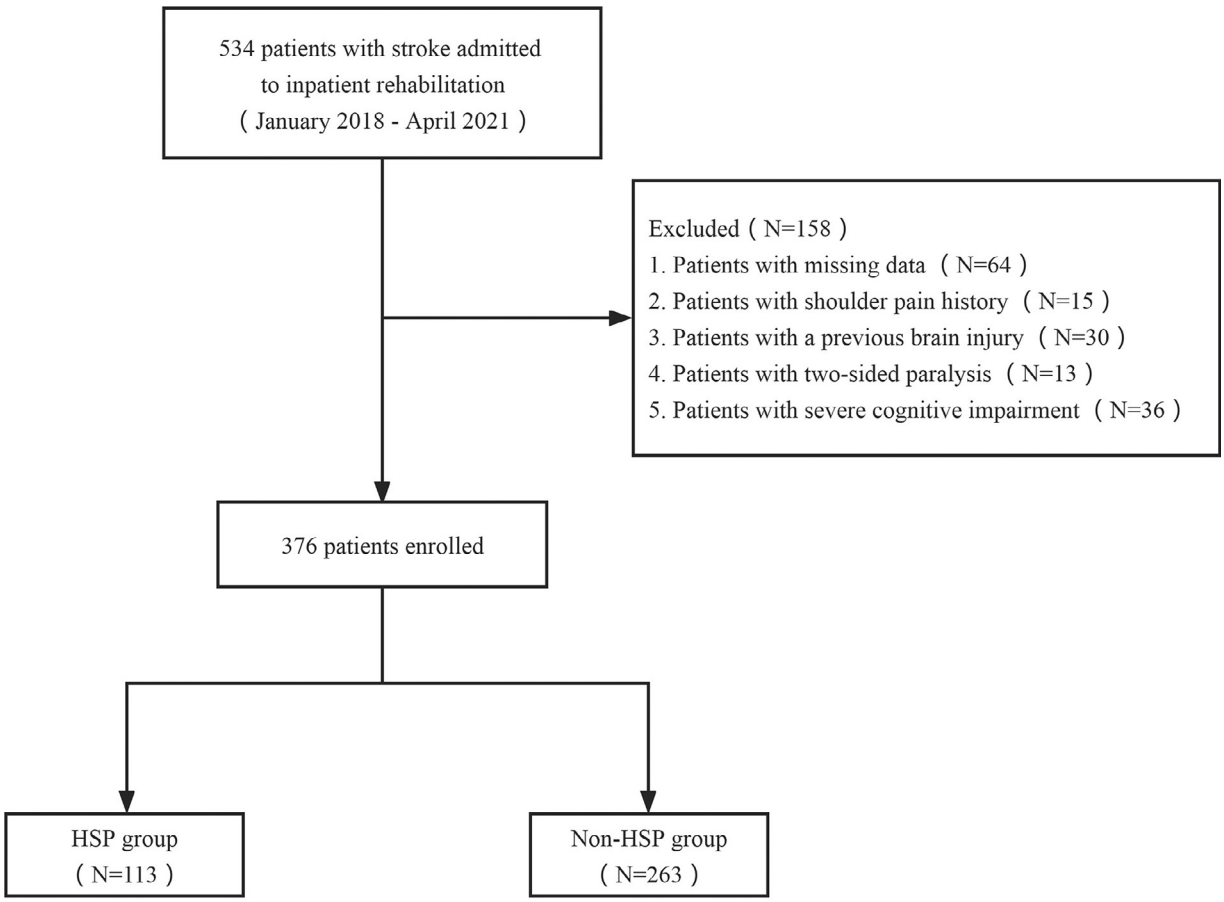

Fig 1 Enrollment flow chart of the patients.

Table 1 Patient characteristics and univariate analysis for predictors of HSP

Characteristic	Non-HSP (n=263)	HSP (n=113)	*P* Value	Statistical Magnitude
Age (y), median (IQR)	68 (57-74)	64 (54-74)	.030*	z=−2.169
Sex, female, n (%)	94 (35.74)	42 (37.17)	.792	χ^2=0.700
Disease course on admission (d), median (IQR)	19 (13-41)	22 (14-44.5)	.260	z=−1.125
Length of stay (d), median (IQR)	51 (10,162)	59 (7-177)	.570	z=−3.276
Stroke type, n (%)				
Ischemic	203 (77.19)	60 (53.10)	<.001*	χ^2=21.818
Hemorrhagic	60 (22.81)	53 (46.90)		
Affected side, left, n (%)	117 (44.49)	59 (52.21)	.169	χ^2=1.895
Hypertension, n (%)	193 (73.38)	82 (72.57)	.870	χ^2=0.027
Diabetes mellitus, n (%)	102 (38.78)	22 (19.47)	<.001*	χ^2=13.340
Arm strength, median (IQR)	3 (2-4)	2 (1-2)	<.001*	z=−6.315
Spasticity, n (%)	79 (30.0)	55 (44.2)	.001*	χ^2=11.966
Brunnstrom stage, median (IQR)	4 (2-5)	2 (2-3)	<.001*	z=−6.564
Sensory disturbance, n (%)	105 (39.92)	78 (69.03)	<.001*	χ^2=26.796
Subluxation, n (%)	21 (7.98)	67 (59.29)	<.001*	χ^2=116.066
Hand edema, n (%)	16 (6.08)	18 (15.93)	0.002*	χ^2=9.315

Abbreviation: IQR, interquartile range
* *P*<.05.

outcomes. A well-calibrated nomogram matches the observed and predicted risks. A Hosmer-Lemeshow test with *P*>.05 was used to indicate the goodness of fit of the nomogram model. The Youden index (Youden index=sensitivity +specificity−1) was used to determine the optimum cutoff value of the predicted probability of HSP and to identify the corresponding sensitivity and specificity.[23]

Decision curve analysis (DCA) was performed to identify the clinical utility of the nomogram by measuring the net benefits at distinct probability thresholds. DCA plotted the net benefit of the nomogram model and compared it with 2 default strategies: "treat all" or "treat none." The "treat-all" strategy is grounded on the supposition that all the patients will be treated, irrespective of their estimated risks. In contrast, the "treat-none" strategy is grounded on the supposition that no patients will be treated because all of them have low risks.[24] When the net benefit curve of a nomogram is higher than that of the "treat all" or "treat none" within a range of the reasonable risk thresholds, the nomogram model is considered clinically useful.

Results

Patient characteristics

This study included 376 patients with stroke, of whom 113 (30.05%) presented with HSP. The enrollment flow chart is summarized in fig 1. The characteristics of the non-HSP and HSP groups are also depicted in table 1. In total, 240 men and 136 women were enrolled in this study. The age of the participants ranged from 27-90 years, with a median age of 67 years. Approximately 46.81% and 53.19% of patients had left and right hemiplegia, respectively, and 69.95% and 30.05% had ischemic and hemorrhagic strokes, respectively.

Univariate and multivariate HSP risk factors

Univariate analysis was performed to compare the candidate predictors between non-HSP and HSP groups. The results showed that 9 variables were statistically significant (*P*<.05): age, type of stroke, diabetes mellitus, arm strength, spasticity, Brunnstrom stage, sensory disturbance, subluxation, and hand edema (see table 1). These predictors were subsequently subjected to multivariate logistic regression analysis, which showed that subluxation (odds ratio [OR], 10.137; 95% confidence interval [CI], 5.289-19.428; *P*<.001), Brunnstrom stage (OR, 0.671; 95% CI. 0.454-0.992; *P*=.046), hand edema (OR, 2.316; 95% CI, 0.922-5.818; *P*=.074), spasticity (OR, 2.065; 95% CI, 1.163-3.668; *P*=.013), and sensory disturbance (OR, 1.982; 95% CI, 1.109-3.543; *P*=.021) were independent predictors of HSP. The results of multivariate analysis are presented in table 2. Subsequently, a nomogram was constructed using the 5 independent predictors (fig 2A), and a web application of the nomogram was then developed to facilitate its use by clinicians and patients. A web version of the nomogram (see fig 2B) is available at https://dynomogram.shinyapps.io/DynNomapp/. The predicted HSP risk probability can be easily calculated by inputting the predictor results and reading the graphical output generated by the web application.

Table 2 Multivariate logistic regression analysis for predictors of HSP

Variables/Intercept	β	*P* Value	OR	95% CI
Age	−0.005	.659	0.995	0.971-1.019
Stroke type	0.423	.202	1.527	0.797-2.926
Diabetes mellitus	−0.432	.202	0.649	0.334-1.261
Arm strength	0.221	.307	1.247	0.817-1.905
Spasticity	0.725	.013*	2.065	1.163-3.668
Brunnstrom stage	−0.398	.046*	0.671	0.454-0.992
Sensory disturbance	0.684	.021*	1.982	1.109-3.543
Subluxation	2.316	<.001*	10.137	5.289-19.428
Hand edema	0.840	.074*	2.316	0.922-5.818
Intercept	−1.233	.229	0.291	

Abbreviation: β, regression coefficient.
* *P*<.1.

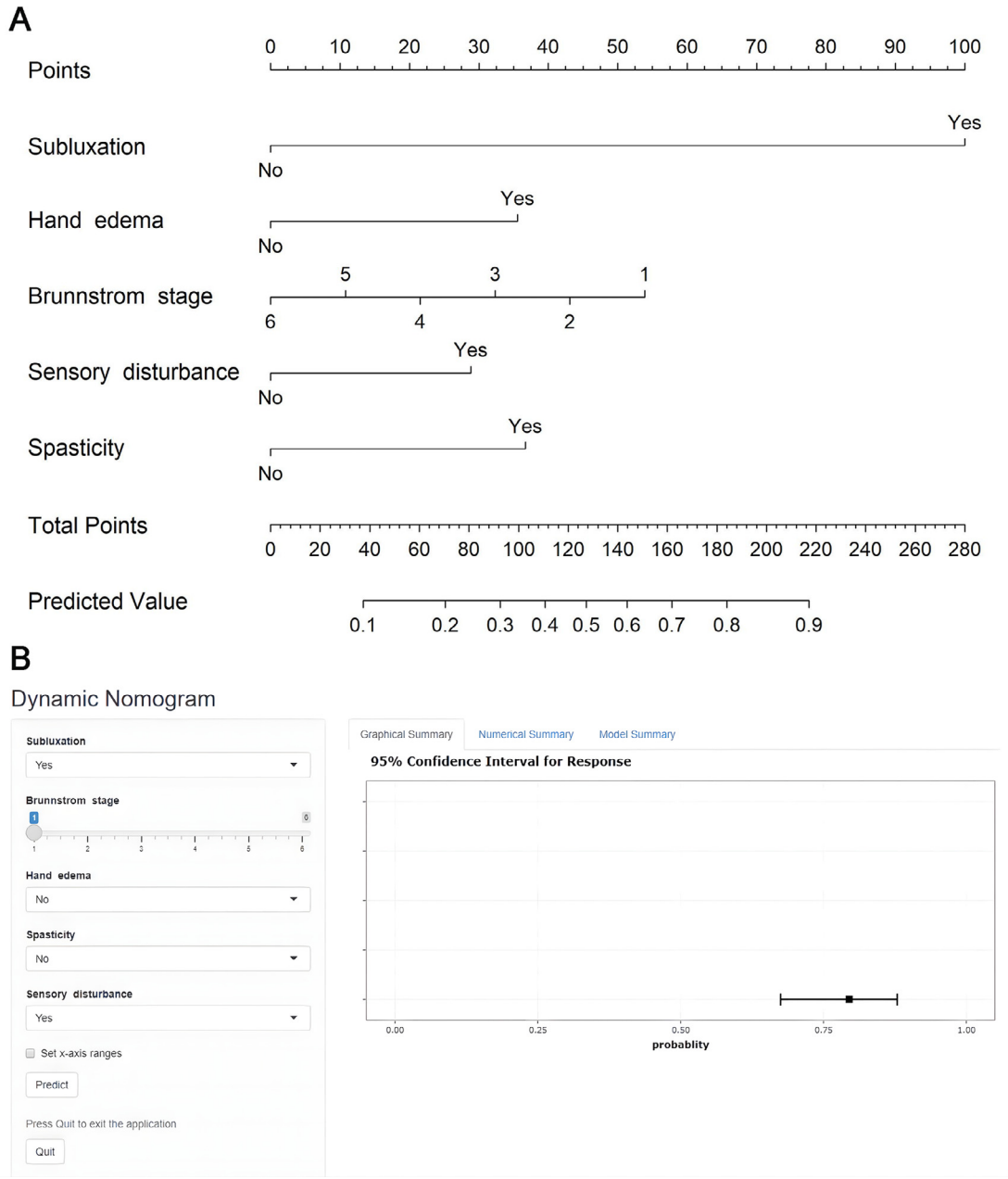

Fig 2 Nomogram for predicting the HSP. **(A)** Nomogram predicting the probability of HSP using subluxation, Brunnstrom stage, hand edema, spasticity, and sensory disturbance as independent predictive factors. Points are assigned for each factor, for which each value is assigned a score by plotting an upward line toward the points line and plotting the sum of 5 scores on the total point line. Subsequently, a vertical line is drawn till the predicted value line from the total points line to obtain the risk probability of HSP. For example, a patient with subluxation (100 points), Brunnstrom stage 1 (47 points), and sensory disturbance (31 points) but no hand edema (0 points) and spasticity (0 points) had a total score of 178 points. The risk probability of HSP would thus be approximately 79.5% (95% CI, 57.9%-91.6%). **(B)** The example of a screen from the web application developed from the prediction model reported in this study.

Nomogram development and validation

Internal validation was performed using the bootstrap technique with 1000 resamples. The nomogram demonstrated excellent discrimination ability for assessing HSP risk, with an unadjusted C-index of 0.85 (95% CI, 0.81-0.89) and a bootstrap-corrected C-index of 0.84. The Hosmer-Lemeshow test also indicated a good fit of the prediction nomogram (χ^2=13.854, *P*=.086). The calibration plot suggested good calibration ability of the nomogram (fig 3), demonstrating good agreement between the forecasted probabilities and actual observations. The optimal cutoff

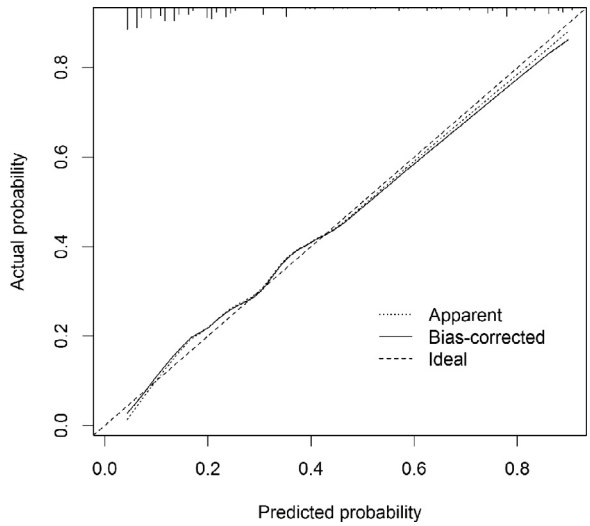

Fig 3 Nomogram calibration curve for predicting HSP. The x-axis designates the nomogram forecasted probability of HSP, whereas the y-axis designates the actual probability of HSP. The reference line is illustrated as 45°, indicating perfect calibration.

value was 0.30, and the corresponding sensitivity and specificity were 0.73 and 0.83, respectively.

Clinical utility

Figure 4 presents the DCA curve used to assess the clinical utility of the nomogram, demonstrating a threshold probability range of 5%-88%. This result revealed that the threshold probability range in the application of this nomogram might be more beneficial than the "treat-all" or "treat-none" strategy. For example, based on a 50% HSP probability, the nomogram added a net benefit of 11.9% compared with the "treat-all" or "treat-none" strategy.

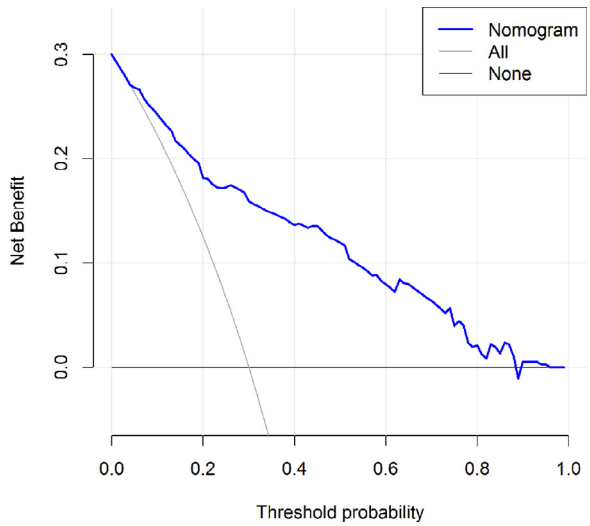

Fig 4 Decision curve for the nomogram. The x-axis illustrates the probability threshold, and the y-axis illustrates the net benefit.

Discussion

In this study, a nomogram was developed and internally validated for the risk prediction of HSP development by combining the following independent predictors: subluxation, Brunnstrom stage, hand edema, spasticity, and sensory disturbance. These 5 predictors are readily available through routine clinical practice. The nomogram exhibited promising performance in terms of discrimination, calibration, and clinical utility. With this nomogram, the HSP risk in patients with stroke can be accurately quantified, consequently assisting clinicians in the implementation of individualized treatments.

The results of the current study showed that the HSP incidence was 30.05%, consistent with a recent systematic review that reported an incidence ranging from 22%-47%.[25] The 5 variables included in the nomogram reportedly correlate with HSP in previous studies.[5-7,26] Poor upper limb motor function was generally considered an independent predictor for HSP.[6-7,26] Our results showed that the Brunnstrom stage, a variable used to reflect the recovery stage of movement functions in patients with hemiparesis, was significantly associated with HSP, a finding consistent with the results of a previous study by Pong et al.[20] Because of the lack of major protective mechanism of the shoulder girdle muscles, patients with severe motor impairment of the arm had increased susceptibility to periarticular soft tissue and nerve injuries, especially during inappropriate stretching or passive arm exercises.[27,28] Thus, overstretching of the hemiplegic upper extremity should be avoided to prevent HSP development, particularly in patients with severe paralysis. Moreover, shoulder subluxation significantly increased the risk of HSP, with an OR of 10.630 (95% CI, 5.581-20.246). The regression coefficient of subluxation was the highest in the multivariate analysis, indicating a strong relationship between subluxation and HSP. A high incidence of shoulder subluxation has been reported in patients with stroke and shoulder pain.[7,26,29] Subluxation may not result in shoulder pain at the early stages but can cause pain if it persists into chronic and spastic phases.[10] Long-standing shoulder subluxation could lead to soft tissue overstretching and repeated microtrauma of joint structures, especially in the supraspinatus and biceps muscles, which are the major causes of shoulder pain post stroke.[29,30] Therefore, prevention and treatment of shoulder subluxation should be sufficiently considered. The relationship between spasticity and HSP remains controversial. Some studies have reported a significant role of spasticity in HSP development,[5,20] whereas others have revealed contrasting results.[7,9,29] In this study, a positive relationship was found between the spasticity and HSP. Increased muscle tone around the shoulder has been generally reported to cause acromion impingement, humeral head displacement, and traction on muscle attachment points, thereby leading to HSP development.[31] Additionally, the treatments aiming to decrease spasticity of the shoulder girdle muscles have been proven effective in improving HSP.[32,33] In this study, 48.7% of patients presented with sensory disturbances of the upper limb, a finding consistent with that of a previous observational study.[7] Sensory disturbances, including superficial sensation and proprioception, have been reportedly associated with HSP.[3,6,34,35]

Isaksson et al[6] argued that light touch loss further increases the vulnerability of hemiplegic shoulders to soft tissue injury. Niessen et al[35] found that proprioceptive deficits can result in shoulder instability, an indirect cause of HSP. However, a few studies did not reach similar conclusions.[6,9] The conflicting results emerging from these studies may be attributed to the different measurement methods applied and the vague definitions of sensory disturbance.[9] Although hand edema is a common problem in patients with stroke, with an incidence rate reportedly ranging from 30%-40%,[36,37] studies on the association between hand edema and HSP are limited. To our knowledge, only 1 study has reported the moderate association of hand edema with HSP.[24] Patients with stroke with shoulder pain and upper limb swelling are frequently diagnosed with shoulder-hand syndrome, a common cause of HSP.[38,39] The present study demonstrated hand edema as an independent predictor for HSP, suggesting that edema plays a role in HSP development. However, hand edema, especially in the initial stages, is often overlooked. Thus, these findings have clinical implications. Early recognition and intervention for hand edema by clinicians is essential to prevent HSP development.

In the current study, the proposed nomogram showed good discrimination with unadjusted and corrected C-indices of 0.85 and 0.84, respectively. The Hosmer-Lemeshow test results and calibration plot supported good consistency between the forecasted and actual probabilities, indicating a good degree of calibration of the nomogram. However, in addition to discrimination and calibration, clinical utility is an important indicator for assessing the prediction models. DCA is a helpful decision-making tool frequently used to measure clinical utility by calculating the net benefits of predictive models. The net benefit is determined by obtaining the sum of all true positives and subtracting the sum of all false positives while considering the odds of the chosen risk probability threshold.[40] The model with higher net benefit for specified probabilities is deemed as having higher clinical utility. In our study, when the predicted probability range was 5%-88%, the application of this nomogram could increase the benefits as opposed to the "treat-none" or "treat-all" strategy. Thus, the nomogram developed in this study has good clinical utility.

Our study provides further evidence of the key role of several clinical variables thought to increase the risk of HSP. This may be helpful for a better understanding of HSP pathogenesis. The identified HSP predictors are noninvasive and readily available at hospital admission. Additionally, the nomogram is a simple and practical tool for clinicians to rapidly predict the risk of developing HSP, resulting in the implementation of early and individualized interventions. Further studies from other medical centers are required to verify the scope of clinical applications of the nomogram model.

Study limitations

This study has several limitations. First, the retrospective design, which may lead to potential biases, may have weakened the implications of the statistical analyses. Second, the external validation of the nomogram was not conducted in this study, and further studies are required to validate the performance of the nomogram externally using a multicenter cohort. Finally, some risk factors, such as depression, hemineglect, and the shoulder's range of motion, were not included in the analysis. These factors act as potential predictors of HSP and may affect the results of the nomogram.

Conclusions

We developed a simple and practical nomogram for forecasting the risk of HSP in patients with stroke during inpatient rehabilitation. This nomogram used 5 easily ascertainable clinical characteristics: subluxation, Brunnstrom stage, hand edema, spasticity, and sensory disturbance. It exhibited satisfactory prediction performance and good clinical utility, potentially assisting clinicians in accurately predicting the patient's risks of HSP and the implementation of early interventions. Further external validation of this nomogram is required in future studies.

Suppliers

a. SPSS 22.0; IBM, Armonk, NY.
b. R 4.0.5; R Foundation, Vienna, Austria.

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Acknowledgments

We thank Huaide Qiu and Ying Shen for their technical assistance.

References

1. Korkmaz N, Yaşar E, Demir Y, et al. Sonographic predictors in patients with hemiplegic shoulder pain: a cross-sectional study. J Stroke Cerebrovasc Dis 2020;29:105170.
2. Adey-Wakeling Z, Arima H, Crotty M, et al. Incidence and associations of hemiplegic shoulder pain poststroke: prospective population-based study. Arch Phys Med Rehabil 2015;96:241-7.
3. Lindgren I, Jönsson AC, Norrving B, et al. Shoulder pain after stroke: a prospective population-based study. Stroke 2007;38:343-8.
4. Koog Y, Jin S, Yoon K, et al. Interventions for hemiplegic shoulder pain: systematic review of randomised controlled trials. Disabil Rehabil 2010;32:282-91.
5. Roosink M, Renzenbrink GJ, Buitenweg JR, et al. Persistent shoulder pain in the first 6 months after stroke: results of a prospective cohort study. Arch Phys Med Rehabil 2011;92:1139-45.
6. Isaksson M, Johansson L, Olofsson I, et al. Shoulder pain and concomitant hand oedema among stroke patients with pronounced arm paresis. Eur J Physiother 2013;15:208-14.
7. Blennerhassett JM, Gyngell K, Crean R. Reduced active control and passive range at the shoulder increase risk of shoulder pain during inpatient rehabilitation post-stroke: an observational study. J Physiother 2010;56:195-9.

8. Holmes RJ, McManus KJ, Koulouglioti C, et al. Risk factors for poststroke shoulder pain: a systematic review and meta-analysis. J Stroke Cerebrovasc Dis 2020;29:104787.
9. Kim YH, Jung SJ, Yang EJ, et al. Clinical and sonographic risk factors for hemiplegic shoulder pain: a longitudinal observational study. J Rehabil Med 2014;46:81–7.
10. Karaahmet OZ, Eksioglu E, Gurcay E, et al. Hemiplegic shoulder pain: associated factors and rehabilitation outcomes of hemiplegic patients with and without shoulder pain. Top Stroke Rehabil 2014;21:237–45.
11. Ye L, Hu C, Wang C, et al. Nomogram for predicting the overall survival and cancer-specific survival of patients with extremity liposarcoma: a population-based study. BMC Cancer 2020;20:889.
12. Park SY. Nomogram: an analogue tool to deliver digital knowledge. J Thorac Cardiovasc Surg 2018;155:1793.
13. Wei L, Champman S, Li X, et al. Beliefs about medicines and non-adherence in patients with stroke, diabetes mellitus and rheumatoid arthritis: a cross-sectional study in China. BMJ Open 2017;7:e017293.
14. Wang Z, Shi Y, Zhang L, et al. Nomogram for predicting swallowing recovery in patients after dysphagic stroke. JPEN J Parenter Enteral Nutr 2022;46:433–42.
15. Zhang XG, Wang JH, Yang WH, et al. Nomogram to predict 3-month unfavorable outcome after thrombectomy for stroke. BMC Neurol 2022;22:111.
16. Collins GS, Reitsma JB, Altman DG, et al. Transparent Reporting of a Multivariable Prediction Model for Individual Prognosis or Diagnosis (TRIPOD): the TRIPOD Statement. Eur Urol 2015;67:1142–51.
17. Liu LS. Writing Group of 2010 Chinese Guidelines for the Management of Hypertension. [2010 Chinese guidelines for the management of hypertension] [Chinese]. Zhonghua Xin Xue Guan Bing Za Zhi 2011;39:579–615.
18. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabet Med 1998;15:539–53.
19. Paci M, Nannetti L, Rinaldi LA. Glenohumeral subluxation in hemiplegia: an overview. J Rehabil Res Dev 2005;42:557–68.
20. Pong YP, Wang LY, Huang YC, et al. Sonography and physical findings in stroke patients with hemiplegic shoulders: a longitudinal study. J Rehabil Med 2012;44:553–7.
21. Peduzzi P, Concato J, Kemper E, et al. A simulation study of the number of events per variable in logistic regression analysis. J Clin Epidemiol 1996;49:1373–9.
22. Hosmer DW, Lemeshow S. Assessing the fit of the model. Applied logistic regression. p 160, 2nd Ed John Wiley and Sons; 2000. p. 160–4.
23. Rücker G, Schumacher M. Summary ROC curve based on a weighted Youden index for selecting an optimal cutpoint in meta-analysis of diagnostic accuracy. Stat Med 2010;29:3069–78.
24. Zhang B, Liu Q, Zhang X, et al. Clinical utility of a nomogram for predicting 30-days poor outcome in hospitalized patients with COVID-19: multicenter external validation and decision curve analysis. Front Med 2020;7:590460.
25. Anwer S, Alghadir A. Incidence, prevalence, and risk factors of hemiplegic shoulder pain: a systematic review. Int J Environ Res Public Health 2020;17:4962.
26. Nadler M, Pauls M, Cluckie G, et al. Shoulder pain after recent stroke (SPARS): hemiplegic shoulder pain incidence within 72 hours post-stroke and 8-10 week follow-up (NCT 02574000). Physiotherapy 2020;107:142–9.
27. Basmajian JV, Bazant FJ. Factors preventing downward dislocation of the adducted shoulder joint. An electromyographic and morphological study. J Bone Joint Surg Am 1959;41-A:1182–6.
28. Estape R, Ferel D, Barth R. Brachial plexus lesions in hemiplegics. Acta Neurol Belg 1979;79:444–9.
29. Aras MD, Gokkaya NK, Comert D, et al. Shoulder pain in hemiplegia: results from a national rehabilitation hospital in Turkey. Am J Phys Med Rehabil 2004;83:713–9.
30. Chae J, Mascarenhas D, Yu DT, et al. Poststroke shoulder pain: its relationship to motor impairment, activity limitation, and quality of life. Arch Phys Med Rehabil 2007;88:298–301.
31. Coskun Benlidayi I, Basaran S. Hemiplegic shoulder pain: a common clinical consequence of stroke. Pract Neurol 2014;14:88–91.
32. Yelnik AP, Colle FM, Bonan IV, et al. Treatment of shoulder pain in spastic hemiplegia by reducing spasticity of the subscapular muscle: a randomised, double blind, placebo controlled study of botulinum toxin A. J Neurol Neurosurg Psychiatry 2007;78:845–8.
33. Marco E, Duarte E, Vila J, et al. Is botulinum toxin type A effective in the treatment of spastic shoulder pain in patients after stroke? A double-blind randomized clinical trial. J Rehabil Med 2007;39:440–7.
34. Gamble GE, Barberan E, Laasch HU, et al. Poststroke shoulder pain: a prospective study of the association and risk factors in 152 patients from a consecutive cohort of 205 patients presenting with stroke. Eur J Pain 2002;6:467–74.
35. Niessen MH, Veeger DH, Meskers CG, et al. Relationship among shoulder proprioception, kinematics, and pain after stroke. Arch Phys Med Rehabil 2009;90:1557–64.
36. Boomkamp-Koppen HG, Visser-Meily JM, Post MW, et al. Post-stroke hand swelling and oedema: prevalence and relationship with impairment and disability. Clin Rehabil 2005;19:552–9.
37. Leibovitz A, Baumoehl Y, Roginsky Y, et al. Edema of the paretic hand in elderly post-stroke nursing patients. Arch Gerontol Geriatr 2007;44:37–42.
38. Barlak A, Unsal S, Kaya K, et al. Poststroke shoulder pain in Turkish stroke patients: relationship with clinical factors and functional outcomes. Int J Rehabil Res 2009;32:309–15.
39. Lo SF, Chen SY, Lin HC, et al. Arthrographic and clinical findings in patients with hemiplegic shoulder pain. Arch Phys Med Rehabil 2003;84:1786–91.
40. Fitzgerald M, Saville BR, Lewis RJ. Decision curve analysis. JAMA 2015;313:409–10.