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Risk Score for Neurological Complications After Endovascular Treatment of Unruptured Intracranial Aneurysms

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Background and Purpose—Procedure-related neurological complications are common after endovascular treatment of unruptured intracranial aneurysms. We aimed to develop a score to quantify individual patient risk.

Methods—We retrospectively analyzed consecutive patients who underwent endovascular treatment for unruptured intracranial aneurysms between January 2012 and September 2015. After excluding those who lost to follow-up and those with fusiform unruptured intracranial aneurysms, included patients were randomly divided into a derivation group (60%) and a validation group (40%). A neurological complication was defined as any transient or permanent increase in the modified Rankin Scale score after aneurysm embolization. A risk score for neurological complications was derived from multivariable logistic regression analyses in the derivation group and validated in the validation group.

Results—Overall, 1060 patients were included (636 in the derivation group and 424 in the validation group). The incidence of neurological complications was 5.5% (95% confidence interval, 3.8%–7.4%). A 3-point risk score (S-C-C) was derived to predict neurological complications (size [≥10 mm=1], core areas [yes=1], and cerebral ischemic comorbidity [yes=1]). The incidence of neurological complications varied from 2.2% in 0-point patients to 25.0% in 3-point patients. The score demonstrated significant discrimination (*C*-statistic, 0.714; 95% confidence interval, 0.624–0.804) and calibration (McFadden R^2, 0.102) in the derivation group. Excellent prediction, discrimination, and calibration properties were reproduced in the validation group.

Conclusions—One in 20 patients will develop neurological complications after endovascular treatment of unruptured intracranial aneurysms. The S-C-C score may be useful for predicting these adverse outcomes based on variables in daily practice. **(*Stroke*. 2016;47:971-978. DOI: 10.1161/STROKEAHA.115.012097.)**

Key Words: complications ■ endovascular procedures ■ intracranial aneurysm

The prevalence of unruptured intracranial aneurysms (UIAs) is estimated at 1% to 7%.[1,2] Acute UIA rupture is associated with 30% to 67% mortality and 15% to 30% morbidity.[3–5] Endovascular coiling and surgical clipping are the primary treatments for UIAs.[1,2] The proportion of UIAs treated endovascularly has increased substantially in recent decades because detachable coils were introduced and the International Subarachnoid Aneurysm Trial (ISAT) results were published.[6] However, endovascular treatment of UIAs has the potential to cause life-threatening neurological complications. In the Analysis of Treatment by Endovascular Approach of Nonruptured Aneurysms (ATENA) series, the incidence of neurological complications, including transient or permanent neurological deficits or death, was 5.4%.[7] Several studies have proposed scales to evaluate the risk of neurological complications for UIA management, such as the Massachusetts General Hospital grade and the UIA treatment score.[8–11] These scales are easy to apply but are primarily derived from surgical treatment of UIAs or indirectly from published data. Furthermore, definitions of neurological complications in these scales do not include transient neurological deficits, such as transient ischemic attack, or epilepsy, which may influence decision-making on UIA management. Clinical parameters, including age, aneurysm size, location, history of ischemic stroke, and stent- or balloon-assisted coiling, have been shown to be associated with neurological complications after UIA management.[8–13] However, currently no reliable scoring system is available to predict the risk of neurological

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complications after endovascular treatment of UIAs that can be used for risk stratification in clinical practice.

Therefore, we developed a score for predicting the risk of neurological complications, including transient or permanent neurological deficits or death, after endovascular treatment of UIAs, and internally validated its predictive properties.

Methods

Study Design and Ethics

We conducted a retrospective observational study approved by the Beijing Tiantan Hospital review committee. All participants provided informed consent.

Data Source

Between January 2012 and September 2015, patients with UIAs who underwent endovascular treatment were prospectively entered into an SPSS database (version 19.0, SPSS, Chicago, IL). The database included information on patient-, aneurysm-, and treatment–specific characteristics, such as patient sex, age, smoking status, alcohol use, previous subarachnoid hemorrhage (SAH) history, family history of SAH, concomitant diseases, aneurysm size, location, morphological features, treatment modalities, instant angiographic outcomes, medications, periprocedural complications, preoperative neurological status (modified Rankin Scale [mRS]), and follow-up results. Modalities and indications for treatment were based on individual patient and UIA characteristics through interdisciplinary decision making by a neurovascular team, offering endovascular embolization as the primary treatment. The results of the International Study of Unruptured Intracranial Aneurysms (ISUIA) and Unruptured Cerebral Aneurysm Study (UCAS) of Japan served as frameworks for management decisions.[14–16] Patient preferences were also considered. Clinical follow-up was supplemented with telephone interviews scheduled at 1 and 6 months after endovascular treatment and annually thereafter. Neurological status was measured with mRS at follow-up assessments. At least 1 digital subtraction angiography examination was performed during follow-up to evaluate imaging outcomes.

Study Population

This study included data from consecutive patients with UIAs who underwent endovascular treatment. Exclusion criteria were (1) fusiform, traumatic, or mycotic UIAs, (2) unrepaired SAH underlying structural lesions or intracranial hemorrhage for unknown reasons, (3) follow-up duration <1 month, and (4) malignant brain tumors or UIAs associated with arteriovenous malformations, arteriovenous fistulas, and moyamoya disease.

Variable Definition

Neurological complications were defined as changes in neurological status (increase in mRS score) after endovascular treatment, including transient or permanent neurological deficits or death. Transient neurological deficits resolved within 1 month. Permanent neurological deficits and death were evaluated at 1 month. Transient or permanent neurological deficits were defined as an mRS score of 2 to 5. An mRS score of 6 indicated death. When the preoperative mRS score was >1, neurological deficits were defined by any increase in mRS score.

Core areas were defined as perforator-rich vessels (A1 segment of anterior cerebral artery, M1 segment of middle cerebral artery, P1 segment of posterior cerebral artery, internal carotid artery bifurcation, and basilar artery) and important cerebrovascular branches (posterior inferior cerebellar artery and anterior choroidal artery) supplying the brain stem and basal ganglia region.[17]

The following covariates were potential predictors or confounders:

1. Patient-specific: age (y), sex (men/women), hypertension (yes/no), smoking status (current or previous smoker; yes/no), alcohol use (current or previous intake >5 drinks per day; yes/no), hyperlipidemia (yes/no), diabetes mellitus (yes/no), cerebral ischemic comorbidities (transient ischemic attack, cerebral infarction, and cerebral vascular stenosis; yes/no), heart comorbidities (coronary heart disease, arrhythmias, heart valve disease, and heart dysfunction; yes/no), previous SAH history (yes/no), family history of SAH (yes/no).
2. Aneurysm-specific: size (mm), wide neck (neck >4 mm or dome-to-neck ratio <1.5; yes/no),[18] location (anterior cerebral artery and anterior communicating artery/middle cerebral artery/internal carotid artery/posterior circulation; posterior communicating artery aneurysms were counted among internal carotid artery aneurysms), multiplicity (yes/no), irregular shape or with a daughter sac (yes/no), location in core areas (yes/no).
3. Procedure-specific: endovascular treatment modality (coiling/stent-assisted coiling/balloon-assisted coiling/other), Raymond scale (RS) score (RS1/RS2/RS3; RS1 indicates complete occlusion; RS2, residual neck; RS3, residual aneurysm).[19]

We calculated the risk of neurological complications per procedure rather than per individual aneurysm or patient. In cases of multiple aneurysms treated in a single session, the features of the aneurysms considered responsible for neurological complications were included in the analysis.

Statistics

Data were analyzed with SPSS 19.0 for Windows. Patient characteristics were described with frequencies (percentages) for categorical variables and mean±SD or median (interquartile range) for continuous variables. Categorical variables were compared using Fisher exact test or the Pearson χ^2 test. Continuous variables were compared between groups using Mann–Whitney *U* test or Student *t* test.

The risk of neurological complications was estimated as the proportion of patients who had transient neurological deficits (mRS score, 2–5), such as transient ischemic attack and seizure within 1 month, and those who had permanent neurological deficits (mRS score, 2–5) or death (mRS score, 6) at 1 month. When preoperative mRS score was >1, any increase in mRS was considered as a neurological complication. Any such modification or death within 1 month after endovascular treatment was considered procedure related. Intraprocedural rupture (n=2), cerebral infarction (n=11), coil or stent migration (n=2), and puncture-site hematoma/bleeding/pseudoaneurysm (n=9) that had no subsequent change in mRS score (eg, headache or dizziness after embolization) were not included for analysis.

We followed current guidance for developing and reporting risk score models.[20] Crude and adjusted odds ratios (ORs) were calculated using univariate and multivariate logistic regression models, respectively. A 2-tailed *P* value <0.05 was considered statistically significant.

Missing data (hypertension [0.5%], age [0.6%], aneurysm size [0.5%], smoking status [0.7%], and alcohol use [0.4%]) were handled with multiple imputation. A complete-case analysis showed similar results to those from the analysis of the imputed data set.

To develop and validate the risk score, patient records were randomly divided into derivation and validation data sets (Figure 1). We selected 60% of the data as the derivation data set (derivation group) using computerized random sampling; the remaining 40% constituted the validation data set (validation group).

Risk Score Derivation

The risk score was developed from the derivation group. We first identified potential predictors of the risk of neurological complications with univariate analysis. Variables found to be significant ($P \leq 0.1$) were then included in a backward stepwise multivariable logistic regression model. We defined an OR≥1.20 as meaningful for predicting neurological complications in the final adjusted multivariable analysis. Variables reaching this OR were attributed points based on the relative ORs. The final risk

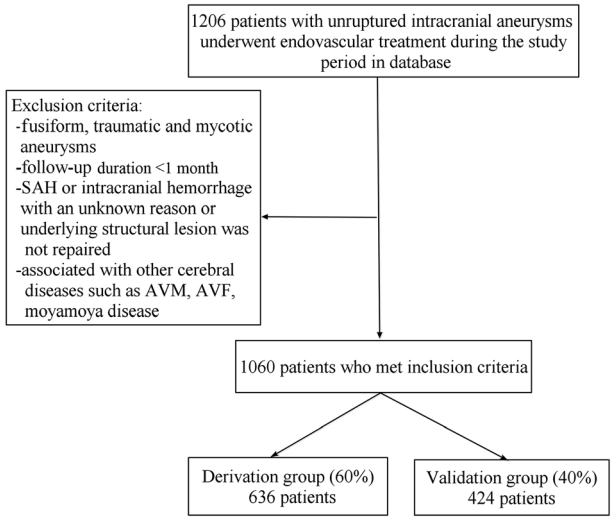

Figure 1. Study flowchart. AVF indicates arteriovenous fistula; AVM, arteriovenous malformation; and SAH, subarachnoid hemorrhage.

score was defined as the sum of all points. Neurological complication risk was stratified by risk score. The risk score's discrimination was assessed by the *C*-statistic to calculate sensitivity and specificity for prediction at each cut-off point. The *C*-statistic represents the areas under the receiver operating characteristic curve; a *C*-statistic of 0.5 indicates no ability and a *C*-statistic of 1.0 indicates perfect ability to discriminate between patients with or without neurological complications. We assessed the risk score's calibration with the Hosmer–Lemeshow statistic using the McFadden R^2, Cox and Snell R^2, and Nagelkerke R^2 as goodness-of-fit measures.

Internal Validation

We evaluated the risk score's accuracy and reliability (prediction, discrimination, and calibration) using validation group data. The validation data set was used to reproduce the observed properties for predicting neurological complication risk.

Results

Study Population

There were 1206 consecutive patients with UIAs who underwent endovascular treatment. Eight (0.7%) were lost to follow-up at 1 month. After exclusion (Figure 1), 1060 patients were analyzed (636 in the derivation group and 424 in the validation group).

Baseline Characteristics

Baseline characteristics of the study population and the whole population are shown in Table 1 and Table I in the online-only Data Supplement, respectively. Individuals with neurological complications had higher rates of cerebral ischemic comorbidities ($P<0.001$), irregular shape or with a daughter sac ($P=0.002$), larger aneurysms ($P<0.001$), and UIAs located in core areas ($P<0.001$).

Incidence of Neurological Complications

Incidences of neurological complications were 5.5% (95% confidence interval, 3.8%–7.4%) in the derivation group, 5.9% (95% confidence interval, 3.8%–8.3%) in the validation group, and 5.7% (95% confidence interval, 4.2%–7.1%) overall.

Table 1. Baseline Characteristics of the Derivation and Validation Groups

Characteristics	Derivation	Validation
n	636	424
Age, y		
Mean±SD	54.8±10.8	54.5±11.3
Median (IQR)	56 (48–62)	55 (48–62)
Women, n (%)	428 (67.3)	290 (68.4)
Smoking, n (%)	138 (21.7)	77 (18.2)
Alcohol use, n (%)	145 (22.8)	84 (19.8)
Hypertension, n (%)	278 (43.7)	182 (42.9)
Hyperlipidemia, n (%)	61 (9.6)	44 (10.4)
Diabetes mellitus, n (%)	68 (10.7)	35 (8.3)
Cerebral ischemic comorbidities, n (%)	77 (12.1)	58 (13.7)
Heart comorbidities, n (%)	40 (6.3)	26 (6.1)
Previous SAH, n (%)	12 (1.9)	8 (1.9)
Family history of SAH, n (%)	17 (2.7)	10 (2.4)
Size, mm		
Mean±SD	7.2±6.1	7.1±5.3
Median (IQR)	5.0 (3.5–8.4)	5.0 (3.9–8.0)
Wide neck, n (%)	515 (81.0)	366 (86.3)
Multiplicity, n (%)	183 (28.8)	96 (22.6)
Locations, n (%)		
ACA, AcomA	60 (9.4)	38 (9.0)
MCA	27 (4.2)	17 (4.0)
ICA	462 (72.6)	306 (72.2)
Posterior circulation (VA, BA, and PCA)	87 (13.7)	63 (14.9)
Core area, n (%)	66 (10.4)	48 (11.3)
Irregular shape or with a daughter sac, n (%)	64 (10.1)	50 (11.8)
Modality of treatment, n (%)		
Coiling only	150 (23.6)	78 (18.4)
Stent-assisted coiling	449 (70.6)	316 (74.5)
Balloon-assisted coiling and others	37 (5.8)	30 (7.1)
RS, n (%)		
RS1	460 (72.3)	291 (68.6)
RS2	153 (24.1)	106 (25.0)
RS3	23 (2.6)	27 (6.4)

Data are shown as n (%) unless otherwise specified. ACA indicates anterior cerebral artery; AcomA, anterior communicating artery; BA, basal artery; ICA, internal carotid artery; IQR, interquartile range; MCA, middle cerebral artery; PCA, posterior cerebral artery; RS, Raymond scale; SAH, subarachnoid hemorrhage; and VA, vertebral artery.

Risk Score Derivation

The association between clinical variables and neurological complications after endovascular treatment of UIAs on univariate analysis is shown in Table 2. The following were

Table 2. Predictors of Neurological Complications: Univariate Analysis

Characteristics	Derivation			Validation		
	NC, %	OR (95% CI)	*P* Value	NC, %	OR (95% CI)	*P* Value
Age, y						
<60	6.0	1.00	…	6.6	1.00	…
≥60	4.5	0.745 (0.351–1.580)	0.443	4.4	0.661 (0.258–1.695)	0.389
Sex						
Men	5.8	1.00	…	6.7	1.00	…
Women	5.4	0.928 (0.452–1.903)	0.837	5.5	0.811 (0.349–1.885)	0.627
Smoking						
No	3.5	1.00	…	5.5	1.00	…
Yes	5.7	1.230 (0.563–2.689)	0.604	7.8	1.459 (0.563–3.784)	0.437
Alcohol use						
No	5.1	1.00	…	5.3	1.00	…
Yes	6.9	1.381 (0.647–2.946)	0.404	8.3	1.626 (0.656–4.031)	0.294
Hypertension						
No	5.3	1.00	…	5.0	1.00	…
Yes	5.7	1.082 (0.546–2.145)	0.821	7.1	1.474 (0.656–3.312)	0.347
Hyperlipidemia						
No	5.9	1.00	…	6.1	1.00	…
Yes	1.6	0.265 (0.036–1.972)	0.195	4.5	0.739 (0.168–3.246)	0.689
Diabetes mellitus						
No	5.5	1.00	…	5.6	1.00	…
Yes	5.9	1.083 (0.370–3.166)	0.885	8.8	1.619 (0.459–5.711)	0.454
Cerebral ischemic comorbidities						
No	4.7	1.00	…	4.6	1.00	…
Yes	11.7	2.713 (1.220–6.032)	0.014	13.8	3.285 (1.347–8.007)	0.009
Heart comorbidities						
No	5.2	1.00	…	5.5	1.00	…
Yes	10.0	2.025 (0.678–6.050)	0.206	11.5	2.229 (0.621–7.999)	0.219
Previous SAH						
No	1.7	1.00	…	1.8	1.00	…
Yes	5.7	3.582 (0.754–17.013)	0.109	4.0	1.806 (0.220–14.844)	0.583
Family history of SAH						
No	2.7	1.00	…	2.3	1.00	…
Yes	2.9	1.075 (0.138–8.350)	0.945	4.0	2.333 (0.276–19.742)	0.437
Size, mm						
<10	3.9	1.00	…	4.3	1.00	…
≥10	11.7	3.239 (1.608–6.523)	0.001	13.2	3.354 (1.444–7.788)	0.005
Wide neck						
No	3.3	1.00	…	3.4	1.00	…
Yes	6.0	1.873 (0.649–5.411)	0.246	6.3	1.878 (0.431–8.184)	0.402
Multiplicity						
No	5.5	1.00	…	5.8	1.00	…
Yes	5.5	0.990 (0.465–2.104)	0.978	6.3	1.084 (0.420–2.796)	0.867

(*Continued*)

Table 2. Continued

Characteristics	Derivation			Validation		
	NC, %	OR (95% CI)	*P* Value	NC, %	OR (95% CI)	*P* Value
Locations						
ICA	4.5	1.00	…	4.9	1.00	…
Others	8.0	1.837 (0.912–3.700)	0.088	8.5	1.796 (0.783–4.120)	0.167
Core area						
No	4.6	1.00	…	4.8	1.00	…
Yes	13.6	3.304 (1.476–7.394)	0.004	14.6	3.396 (1.339–8.614)	0.010
Irregular shape						
No	4.7	1.00	…	5.1	1.00	…
Yes	12.5	2.884 (1.250–6.650)	0.013	12.0	2.548 (0.966–6.720)	0.059
Modalities of treatment						
Stent-assisted coiling	4.5	1.00	…	5.1	1.00	…
Others	8.0	1.855 (0.928–3.708)	0.080	8.3	1.705 (0.730–3.979)	0.218
RS						
RS1	5.0	1.00	…	5.8	1.00	…
RS2+RS3	6.8	1.390 (0.676–2.858)	0.370	6.0	1.032 (0.434–2.454)	0.944

CI indicates confidence interval; ICA, internal carotid artery; NC, neurological complication; OR, odds ratio; RS, Raymond scale; and SAH, subarachnoid hemorrhage.

significant ($P<0.1$) on univariate analysis and subsequently included in the multivariable analysis: aneurysm size, cerebral ischemic comorbidities, location, core areas, irregular shape or with a daughter sac, and treatment modalities. A multivariable logistic regression model with backward stepwise variable selection showed that the following were significantly associated with postoperative neurological complications and reached the previously defined OR of 1.20: size, core areas, and cerebral ischemic comorbidities.

The following variables were entered into the final adjusted multivariable logistic regression model (Table 3): cerebral ischemic comorbidities, size, and core areas. All ORs were ≈3; 1 point was assigned to each independent predictor. Thus, the 3-point S-C-C score was derived (Table II in the online-only Data Supplement).

Internal Validation

Demographic and clinical characteristics were comparable between the validation and derivation groups (Table 1). All postprocedural neurological complication predictors in the derivation group were significantly associated with neurological complications in the validation group in univariate and multivariate analyses (Tables 2 and 3). Discrimination and calibration measures of the derivation and validation groups are shown in Table 4. The cut-off point–specific sensitivity and specificity for the S-C-C score in the derivation and validation groups and overall are shown in Tables III, IV, and V in the online-only Data Supplement. The proportion of neurological complications varied from 2.2% in patients with a score of 0% to 25% in patients with a score of 3 in the derivation group, from 2.2% to 33.3% in the validation group, and from 2.2% to 28.6% overall (Figure 2).

Interactions

Plausible interactions were tested and did not improve the predictive ability of the final model. Hence, plausible interactions were not included.

Discussion

Main Findings

This study's main findings were as follows: (1) procedure-related neurological complications after endovascular treatment of UIAs occurred more frequently with larger aneurysms (≥10 mm), UIAs located in core areas, and with cerebral ischemic comorbidity; and (2) a simple score composed of these variables could predict the risk of neurological complications after endovascular treatment of UIAs.

The S-C-C score is purposefully based on variables commonly examined in daily practice. The predictive performance of the S-C-C score is solid, as evidenced by its discriminative ability and calibration. This score is useful in 2 different contexts as follows: (1) clinical identification of high-risk individuals in whom preventive interventions could be more successful; and (2) aiding clinicians, patients, and families in decision making when faced with UIA treatment.

Previous Studies

The incidence of neurological complications was 5.5%, similar to that observed previously, although comparisons between studies are difficult because of different patient populations, criteria for neurological complications, and follow-up periods.[8,12–14]

Previous studies have explored risk factors associated with ischemic and hemorrhagic events after endovascular treatment

Table 3. Predictors of Neurological Complications: Final Adjusted Multivariable Analysis

	Derivation Group			Validation Group		
Characteristics	OR	95% CI	*P* Value	OR	95% CI	*P* Value
Size, mm						
<10	1.00	…	…	1.00	…	…
≥10	3.230	1.574–6.628	0.001	3.413	1.436–8.112	0.005
Core area						
No	1.00	…	…	1.00	…	…
Yes	2.865	1.243–6.608	0.014	2.954	1.113–7.841	0.030
Cerebral ischemic comorbidities						
No	1.00	…	…	1.00	…	…
Yes	2.848	1.241–6.536	0.014	2.881	1.135–7.312	0.026

CI indicates confidence interval; and OR, odds ratio.

of UIAs.[21] However, some events may be asymptomatic.[22] Here, aneurysm size was a prominent predictor of neurological complications, in agreement with previous reports.[21,23] Larger aneurysms increase the risk of thromboembolic events, which may lead to neurological deficits.[21] In addition, larger aneurysms typically exhibit larger mass and unexpected tissue formation, which may cause neurological deficits, such as visual field defects or sudden blindness after endovascular treatment of paraclinoid UIAs.[24] Risk resulting from cerebral ischemic comorbidities was an important predictor in this study, which is in agreement with previous observations.[12,22] Jang et al[12] found that patients with histories of ischemic stroke experienced neurological deficits from high rates of thromboembolic events after endovascular treatment. A history of ischemic stroke may be correlated with vessel injuries in the surgical field and microembolisms.[21,22] Core areas contain perforators and important small vessels supplying the thalamus, basal ganglia region, and brain stem.[17,25] Most perforators cannot be displayed on digital subtraction angiography but sacrificing such vessels may result in neurological deficits.[17,25] Furthermore, endovascular treatment of UIAs involving the anterior choroidal and posterior inferior cerebellar arteries remains challenging with high rates of neurological complications.[26,27]

Location, treatment modality, and irregular shape or daughter sac presence predicted neurological complications on univariate analysis. However, these predictors were excluded from the final model. In contrast to results from previous studies,[21,28] stent-assisted coiling protected against neurological complications possibly because of the statistical consideration of dichotomous variables for treatment modality; the rate of stent-assisted coiling was >70%, higher for other modalities including parent artery occlusion and balloon-assisted coiling. However, patients with parent artery occlusion had higher incidences of neurological complications in agreement with previous studies.[29]

Wide neck, older age, multiplicity, diabetes mellitus, heart comorbidities, hyperlipidemia, hypertension, and smoking have been reported to increase the risk of ischemic or hemorrhagic events,[12,21,22,30,31] which may be indirectly related to neurological complications. However, in this study, these risk factors were nonsignificant.

A tool identifying patients at high risk of neurological complications after UIA management would be beneficial. Khanna et al[8] proposed a grading system based on aneurysm size, location, and patient age. This system is simple to apply but was derived from surgical treatment of UIAs with a small sample. Ogilvy et al[9] proposed a 5-point grading system including patient age, aneurysm size, location, Hunt and Hess grade, and Fisher scale. This is a comprehensive grading system but was derived from surgical treatment of intracranial aneurysms. Etminan et al[10] developed a UIA treatment score model on UIA management; the merit of this score is that it includes many different factors such as age, life expectancy,

Table 4. Evaluation of Discrimination and Calibration Abilities of the Risk Score

	Derivation Group	Validation Group	All Patients
Discrimination			
AUCROC (95% CI)	0.714 (0.624–0.804)	0.721 (0.618–0.824)	0.717 (0.649–0.785)
P value	<0.001	<0.001	<0.001
Calibration, goodness of fit			
Cox and Snell R^2	0.043	0.059	0.040
Nagelkerke R^2	0.124	0.163	0.113
McFadden R^2	0.103	0.136	0.093
P value (χ^2)	0.552 (2.100)	0.236 (4.246)	0.268 (3.943)

AUCROC indicates area under the receiver operating curve; and CI, confidence interval.

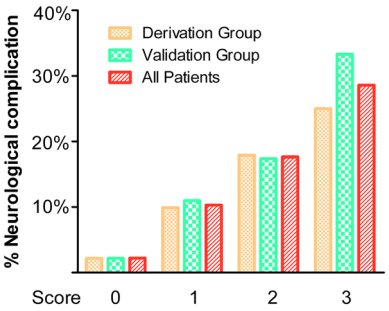

Figure 2. Incidences of neurological complications by clinical score in the derivation group, the validation group, and for all patients.

aneurysm size, location, morphological features, and treatment-related risks. However, the score was derived from a multidisciplinary consensus on contemporary practice of UIA management using the Delphi method, and indirectly from published data. Morgan et al[11] produced a complication-effectiveness model. Their model was derived by dividing the neurological complication risk by the 10-year cumulative freedom from retreatment or rupture proportion in a prospective cohort study with a large sample but it was derived from patients with UIAs who underwent craniotomies. However, our S-C-C score was derived from patients with UIAs who underwent endovascular treatment only. Furthermore, we included transient neurological deficits for analysis and the score was internally validated.

Strengths and Limitations

This is the first study to develop a simple scoring system to predict neurological complications, including transient or permanent neurological deficits or death, after endovascular treatment of UIAs based on routine clinical data. To avoid selection bias, we randomly divided patients and included into derivation and validation groups. However, we also acknowledge several limitations. First, our study is limited by its retrospective nature; data were prospectively recorded but retrospectively examined. Second, the clinical follow-up at 1 month was performed primarily by phone (90.8%), and transient neurological complications may have been underreported. Third, some confounders may remain. Fourth, we defined permanent neurological deficits and death at 1 month postoperatively. However, delayed neurological deficits may have occurred.[32] Furthermore, we included all neurological deficits and deaths within 1 month, although some may not have been procedure-related. Fifth, changes in cognitive status were not evaluated, although they were in the ISUIA study.[14] Similarly, health-related quality of life could have been evaluated with the SF-36 questionnaire. Finally, although our population is a perfect representation of our center's daily practice, we could not determine whether this population was representative of the entirety of patients with UIAs. Thus, the S-C-C score requires external validation using a large series in other centers or countries.

Conclusions

The incidence of neurological complications after endovascular treatment of UIAs was 5.5%. The S-C-C score may be able to estimate the risk of neurological complications after further validation to establish its generalizability.

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Disclosures

None.

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