Prognostic nomogram for patients with minor stroke and transient ischaemic attack

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ABSTRACT

Background Ischaemic stroke and transient ischaemic attack (TIA) share a common cause. We aim to develop and validate a concise prognostic nomogram for patients with minor stroke and TIA.

Methods A total of 994 patients with minor stroke and TIA were included. They were split into a derivation (n=746) and validation (n=248) cohort. The modified Rankin Scale (mRS) scores 3 months after onset were used to assess the prognosis as unfavourable outcome (mRS≥2) or favourable outcome (mRS<2).

Result The final model included seven independent predictors: gender, age, baseline National Institute of Health Stroke Scale (NIHSS), hypertension, diabetes mellitus, white blood cell and serum uric acid. The Harrell's concordance index (C-index) of the nomogram for predicting the outcome was 0.775 (95% CI 0.735 to 0.814), which was confirmed by the validation cohort (C-index=0.787 (95% CI 0.722 to 0.853)). The calibration curve showed that the nomogram-based predictions were consistent with actual observation in both derivation cohort and validation cohort.

Conclusion The proposed nomogram showed favourable predictive accuracy for minor stroke and TIA. This has the potential to contribute to clinical decision-making.

INTRODUCTION

Stroke is the second leading cause of death in the world,[1] and the global burden of stroke is continuously increasing.[2] Among them, minor stroke and transient ischaemic attack (TIA) have been attracting more and more attention during recent years. As is well known, TIA and ischaemic stroke share a common cause. Both minor stroke and TIA are prevalent among cerebrovascular diseases. A registry study conducted in Switzerland included 1633 patients showed that minor stroke accounts for approximately 40.8% of ischaemic stroke.[3]

Patients with minor stroke and TIA have mild or even no symptoms when they are admitted to hospital; therefore, the expectations for a favourable outcome from themselves or their family are usually higher than others. Their most concerned question is 'May I/my family recover?' Though there is an improvement in evaluation and treatment in the past few years, a considerable percentage unfortunately about 15% of these patients end in a poor outcome.[4] Intensive monitoring and more intensive treatment are required for these patients in the early stages.[5 6] Therefore, early identification of the patients at high risk is critical.

Accumulating evidence suggests that several factors have been predictors of poor outcome in patients with minor stroke and TIA. These factors include the severity of stroke itself, age, diabetes mellitus, some haematological biomarkers, such as white blood cell (WBC), serum uric acid (SUA).[4 7 8] However, there is still no commonly used prognostic score—which synthesises prognostic predictors—that is created to concisely and accurately predict the functional outcome of patients with minor stroke and TIA.

In this study, we aimed to acquire and validate a prognostic score to predict the functional outcome of patients with minor stroke and TIA after 3 months of symptom onset. Moreover, for convenience of future clinical application, the prognostic scores are based on clinical data, which can be measured simply and inexpensively.

METHODS

Study subjects

This was a retrospective registry study. We analysed patients with minor stroke and TIA admitted within 48 hours of stroke symptom onset from January 2013 to January 2020 in Shenzhen Second People's Hospital consecutively and followed up for 3 months. Inclusion criteria include (1) patient age ≥18 years and (2) National Institute of Health Stroke Scale (NIHSS) ≤5 on hospital admission; ≤1 NIHSS score in single item scores such as vision, language, neglect and single limb; and no score in consciousness item. Exclusion criteria include (1) significant neurological deficits before onset (the premorbid modified Rankin Scale (pre-mRS>1), (2) neurological deficit after epileptic seizures, (3) other serious illness that would confound the clinical outcome and (4) lost to follow-up. A flow diagram of participants eligible is shown in figure 1. No thrombolytic agent other than recombinant human tissue-type plasminogen (rt-PA) was used for intravenous thrombolysis, and no patient underwent endovascular treatment. Seventy-five per cent of the study subjects were randomly assigned to the derivation cohort, and the remaining 25% to the validation cohort. The study was approved by the ethics committee of Shenzhen Second People's Hospital.

Variables of interest

Baseline demographic characteristics including age and sex, and vascular risk factors including history of hypertension, diabetes, coronary heart disease and atrial fibrillation were self-reported on

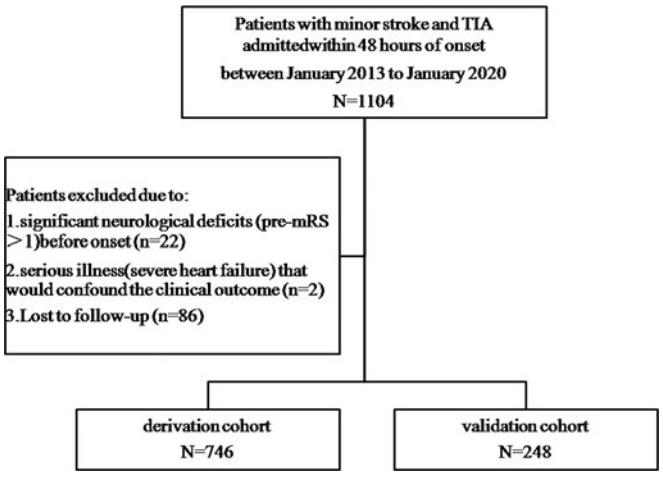

Figure 1 Flow diagram of participants eligible. Pre-mRS, premorbid modified Rankin Scale; TIA, transient ischaemic attack.

a questionnaire on admission and verified by a trained neurologist. Baseline NIHSS was assessed by trained neurologists. Blood samples were all collected from patients within 24 hours after admission. Laboratory data included fasting blood glucose (FBG), haemoglobin A1 c (HbA1c), WBC count, high-sensitivity C reactive protein (hs-CRP), low-density lipoprotein cholesterol, SUA value, etc. Baseline systolic blood pressure (SBP) and baseline diastolic blood pressure were measured on admission. Missing data were imputed using multiple imputation. All patients underwent cranial vascular imaging assessment, magnetic resonance angiography (US General Motors 1.5 T superconducting magnetic resonance apparatus) or CT angiography (Siemens dual-source 64-row spiral CT) or digital subtraction angiography (DSA) (AXIOM Artisd TA DSA machine from Siemens, Germany).

Outcome assessment

The mRS was used to assess the functional outcome 3 months after symptom onset by trained neurologists. The mRS was dichotomised to define favourable outcome (mRS<2) and unfavourable outcome (mRS≥2). We conducted telephone interview if the patient was unable to be present in the hospital for assessment.

Statistical analysis

Continuous variables are reported as mean±SD or medians (IQR), and categorical variables are reported as frequencies. Comparisons were performed on favourable outcome group and unfavourable outcome group according to baseline characteristics and functional outcome in the derivation cohort. Variables with the value of $p<0.1$ in the univariate analyses were subjected to multivariable analysis. The selection of the final prediction model was performed with a backward step-down selection process using Akaike's Information Criterion (AIC).[9] We used the variance inflation factor (VIF) for the multicollinearity diagnosis. A prognostic nomogram model was formulated based on the final prediction model. Discrimination of the nomogram was assessed by Harrell's concordance index (C-index),[10] which is equivalent to the area under the receiver operator characteristic curve. A larger C-index indicated more accurate prognostic stratification. Calibration was assessed by comparing the observed 3-month unfavourable outcome probability with the nomogram-predicted from the final model. Moreover, bootstrapping using 1000 resampling procedures was performed. The performance of the nomogram was internally and externally validated using the derivation cohort and validation cohort, respectively. Statistical analyses to identify prognostic factors were performed using SPSS 22.0 for Windows (Armonk, NY). The nomogram was analysed with the rms[11] package in R version 3.5.1 (http://www.r-project.org/). All statistical tests were two-tailed, and p values of less than 0.05 were considered to be statistically significant.

RESULTS

Overall, a total of 994 patients with minor stroke and TIA were included, of which 746 constituted the derivation cohort and 248 constituted the validation cohort. After a median follow-up time of 3 months, 147(19.7%) patients in derivation cohort and 56 (22.6%) patients in validation cohort developed unfavourable outcome (mRS≥2), respectively. The characteristics of the patients in the derivation cohort and the validation cohort are listed in table 1. In general, the baseline characteristics showed no significant differences between the derivation and validation cohort (p=0.061–0.942).

The unfavourable outcome rate in the derivation cohort was 19.7%. Table 2 shows the association between the possible predictors and 3-month outcome in the derivation cohort.

Table 1 Characteristics of patients in the derivation and validation cohort

Characteristics	Derivation cohort	Validation cohort	P value
Total (n)	746	248	
Female	229(30.7%)	71(28.6%)	0.539*
Age (years)	58.21±13.08	58.35±13.32	0.780§
Pre-mRS=1	74(9.9%)	25(10.1%)	0.942*
Intravenous thrombolysis (rt-PA)	86(11.5%)	32(12.9%)	0.562*
Baseline NIHSS	2(0, 3)	2(0, 4)	0.791‡
Diabetes mellitus	192(25.7%)	50(20.2%)	0.076*
Hypertension	433(58.0%)	142(57.3%)	0.828*
Coronary heart disease	63(8.4%)	25(10.1%)	0.432*
Atrial fibrillation	22(2.9%)	3(1.2%)	0.130†
Index events (TIA)	55(7.4%)	20(8.1%)	0.721*
Baseline SBP (mmHg)	149.05±24.62	148.17±23.58	0.512§
Baseline DBP (mmHg)	89.85±15.53	90.01±16.05	0.399§
FBG (mmol/L)	5.7(4.9, 7.5)	5.5(4.9, 7.2)	0.184‡
HBA1c (%)	6.0(5.6, 6.7)	5.8(5.6, 6.4)	0.243‡
hs-CRP (mg/dL)	1.6(0.6, 4.0)	1.6(0.6, 3.6)	0.652‡
WBC ($\times 10^9$/L)	7.2(6.0, 8.7)	7.0(5.9, 8.9)	0.366‡
Hcy (μmol/L)	13.0(10.2, 16.1)	13.1(10.1, 16.9)	0.347‡
SUA (μmol/L)	354.32±101.55	363.57±114.72	0.061§
LDL-C (mmol/L)	3.0(2.4, 3.5)	2.9(2.3, 3.6)	0.673‡
mRS≥2 at 3 months	147(19.7%)	56(22.6%)	0.330*

*Pearson's χ^2 test.
†Fisher test.
‡Mann-Whitney U test.
§t-Test.
Data are shown as numbers(%) or mean±SD) or medians (IQRs).
No significant differences in these variables between the derivation and validation cohort.
Baseline DBP, diastolic blood pressure at admission; baseline NIHSS, NIHSS at admission; baseline SBP, systolic blood pressure at admission; FBG, fasting blood glucose; HBA1c, haemoglobin A1c; Hcy, homocysteine; hs-CRP, high-sensitivity C reactive protein; LDL-C, low-density lipoprotein cholesterol; mRS, modified Rankin Scale; NIHSS, National Institute of Health Stroke Scale; pre-mRS, premorbid mRS; rt-PA, recombinant human tissue-type plasminogen; SUA, serum uric acid; TIA, transient ischaemic attack; WBC, white blood cell.

Table 2 Patient demographics and clinical characteristics in the derivation cohort

Characteristics	Favourable outcome (mRS<2)	Unfavourable outcome (mRS≥2)	P value
Total (n)	599	147	
Female	167(27.9%)	62(42.2%)	0.001*
Age (years)	57.48±12.86	61.16±13.61	0.002§
Pre-mRS=1	50(8.3%)	24(16.3%)	0.004*
Intravenous thrombolysis (rt-PA)	63(10.5%)	23(15.6%)	0.081*
Baseline NIHSS	2(0, 3)	3(2, 4)	<0.001‡
Diabetes mellitus	130(21.7%)	62(42.2%)	<0.001*
Hypertension	334(55.8%)	99(67.3%)	0.011*
Coronary heart disease	46(7.7%)	17(11.6%)	0.129*
Atrial fibrillation	17(2.8%)	5(3.4%)	0.718†
Index events (TIA)	52(8.7%)	3(2.0%)	0.006†
Baseline SBP (mmHg)	148.25±23.98	152.31±26.91	0.074§
Baseline DBP (mmHg)	89.98±15.68	89.32±14.93	0.644§
FBG (mmol/L)	5.5(4.9, 7.0)	6.4(5.3, 9.1)	<0.001‡
HBA1c (%)	5.9(5.6, 6.6)	6.1(5.6, 7.3)	0.112‡
hs-CRP (mg/dL)	1.5(0.6, 3.8)	6.1(5.6, 7.3)	<0.001‡
WBC ($\times 10^9$/L)	7.1(5.9, 8.6)	7.7(6.6, 8.9)	0.001‡
Hcy (μmol/L)	13.0(10.6, 16.3)	12.0(9.8, 15.6)	0.024‡
SUA (μmol/L)	361.72±102.09	324.15±93.74	<0.001§
LDL-C (mmol/L)	3.0(2.4, 3.5)	3.0(2.4, 3.5)	0.890‡

*Pearson's χ^2 test.
†Fisher test.
‡Mann-Whitney U test.
§t-Test.
Data are shown as numbers (%) or mean±SD or medians (IQRs).
Baseline DBP, diastolic blood pressure at admission; baseline SBP, systolic blood pressure at admission; FBG, fasting blood glucose; HBA1c, haemoglobin A1c; Hcy, homocysteine; hs-CRP, high-sensitivity C reactive protein; LDL-C, low-density lipoprotein cholesterol; mRS, modified Rankin Scale; NIHSS, National Institute of Health Stroke Scale; baseline NIHSS, NIHSS at admission; pre-mRS, premorbid mRS; rt-PA, recombinant human tissue-type plasminogen; SUA, serum uric acid; TIA, transient ischaemic attack; WBC, white blood cell.

Table 3 Predictors for unfavourable outcome after 3 months of onset in patients with minor stroke and TIA

	Univariate logistic regression			Multivariate logistic regression		
Variable	OR	95% CI	P value	OR	95% CI	P value
Gender (female)	1.887	1.300 to 2.740	<0.001	1.586	1.037 to 2.426	0.033
Age (years)	1.022	1.008 to 1.036	<0.001	1.018	1.002 to 1.035	0.026
Pre-mRS=1	2.142	1.268 to 3.620	0.004	–	–	–
Intravenous thrombolysis (rt-PA)	1.578	0.942 to 2.643	0.083	–	–	–
Baseline NIHSS	1.590	1.411 to 1.791	<0.001	1.563	1.377 to 1.775	<0.001
Diabetes mellitus	2.631	1.798 to 3.850	<0.001	2.397	1.575 to 3.646	<0.001
Hypertension	1.636	1.118 to 2.394	0.011	1.657	1.092 to 2.515	0.018
Index events (TIA)	0.219	0.067 to 0.712	0.012	–	–	–
Baseline SBP (mmHg)	1.007	0.999 to 1.014	0.074	–	–	–
FBG (mmol/L)	1.077	1.030 to 1.127	0.001	–	–	–
hs-CRP (mg/dL)	1.003	0.992 to 1.015	0.576	–	–	–
WBC ($\times 10^9$/L)	1.139	1.056 to 1.229	0.001	1.131	1.035 to 1.235	0.006
Hcy (μmol/L)	0.992	0.972 to 1.012	0.412	–	–	–
SUA (μmol/L)	0.996	0.994 to 0.998	<0.001	0.997	0.995 to 0.999	0.003

Baseline NIHSS, NIHSS at admission; FBG, fasting blood glucose; HBA1c, haemoglobin A1c; Hcy, homocysteine; hs-CRP, high-sensitivity C reactive protein; mRS, modified Rankin scale; pNIHSS, National Institute of Health Stroke Scale; pre-mRS, premorbid mRS; SUA, serum uric acid; TIA, transient ischaemic attack; WBC, white blood cell.

Univariate analysis showed that gender, age, pre-mRS, intravenous thrombolysis (rt-PA), baseline NIHSS, diabetes mellitus, hypertension, index events (TIA), baseline SBP, FBG, hs-CRP, WBC, Hcy and SUA were associated with unfavourable outcome at the value of $p<0.1$. All these variables were included in multivariate analysis.

The selection of the final prediction model was performed with a backward step-down selection process using AIC. As shown in table 3, seven variables—gender, age, baseline NIHSS, hypertension, diabetes mellitus, WBC and SUA—were included in the final model. The VIFs of them ranged from 1.0312 to 1.0685, indicating that there was no collinearity. The multivariable analyses demonstrate that they were independent related to the outcome. Patients with minor stroke and TIA can benefit from higher SUA levels (per 1 μmol/L) (OR=0.997, 95% CI 0.995 to 0.999). Female, older age (per year), higher baseline NIHSS, diabetes mellitus, hypertension and higher WBC (per 1×10^9/L) were positively correlated with unfavourable outcome (ORs and 95% CI for them were as follows: OR=1.586, 95% CI=1.037–2.426; OR=1.018, 95% CI=1.002–1.035; OR=1.563, 95% CI 1.377 to 1.775; OR=2.397, 95% CI 1.575 to 3.646; OR=1.657, 95% CI 1.092 to 2.515; OR=1.131, 95% CI 1.035 to 1.235, respectively). The regression coefficients for the variables and intercepts were as follows: female (0.461), age (0.018), higher baseline NIHSS (0.447), diabetes mellitus (0.874), hypertension (0.505), WBC (0.123), SUA (−0.003) and intercept (−4.109). According to multivariate analysis results, the final model included the seven most predictive variables: gender, age, baseline NIHSS, hypertension, diabetes mellitus, WBC and SUA. We acquire a nomogram to predict the probability of unfavourable outcome 3 months after symptom onset using the variables of the final model (figure 2). The nomogram allows the user to compute the probability of unfavourable outcome corresponding to a patient's combination of covariates. As an example, locate the patient's baseline NIHSS and draw a line straight upwards to the 'Points' axis to determine the score associated with that baseline NIHSS. Repeat the process for each variable, sum the scores achieved for each covariate and locat this sum on the 'Total Points' axis. Draw a line straight down to determine the likelihood of unfavourable outcome. The internal bootstrap validation showed that the C-index of the nomogram for patients' outcome was 0.775 (95% CI 0.735 to 0.814) (figure 3). In figure 4, the x-axis is the predicted probability of unfavourable outcome by the nomogram, the y-axis is the observed probability of unfavourable outcome, dotted line with slope 1 represents the ideal reference line and the calibration plot showed optimal agreement between the prediction by the nomogram and actual observation. The external validation was based on the validation cohort. The nomogram achieved a C-index of 0.787 (95% CI 0.722 to 0.853) (figure 5). A calibration curve also showed good consistency between predicted and observed probability of unfavourable outcome (figure 6). Thus, regardless internal or external validation, the establishednomogram performed well in predicting a 3-month prognosis in patients with minor stroke and TIA.

DISCUSSION

This study developed and validated a concise prognostic nomogram to predict the outcome of patients with minor stroke and TIA

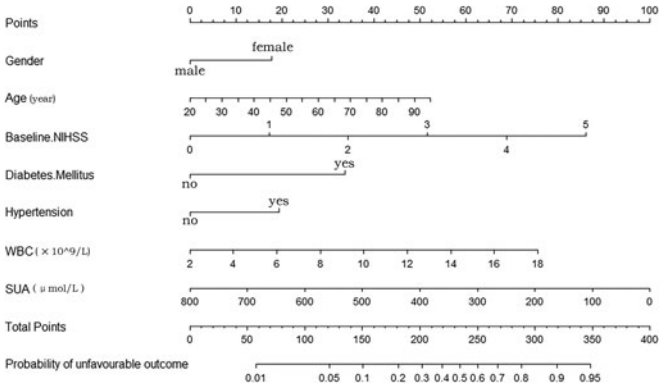

Figure 2 Nomogram, including gender, age, baseline National Institute of Health Stroke Scale (NIHSS), hypertension, diabetes mellitus, white blood cell (WBC) and serum uric acid (SUA), to predict 3-month unfavourable outcome rate in patients with minor stroke and TIA. The nomogram allows the user to obtain the probability of 3-month unfavourable outcome corresponding to a patient's combination of covariates. As an example, locate the patient's baseline NIHSS and draw a line straight upward to the 'Points' axis to determine the score associated with that Baseline NIHSS. Repeat the process for each variable, and sum the scores achieved for each covariate, and locate this sum on the 'Total Points' axis. Draw a line straight down to determine the likelihood of unfavourable outcome after 3 months of onset.

Baseline NIHSS, NIHSS at admission; TIA, transient ischaemic attack.

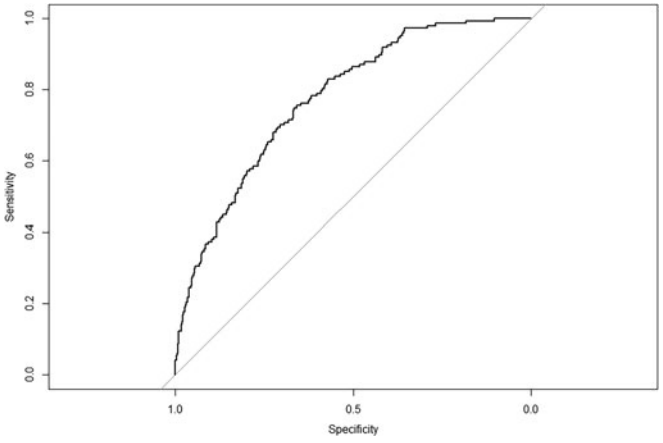

Figure 3 ROC of the nomogram in the derivation cohort. It showed good discrimination with a C-index of 0.775 (95% CI 0.735 to 0.814).

C-index, concordance index; ROC, receiver operating characteristic curve.

after 3 months of onset. The nomogram took seven variables into account: gender, age, baseline NIHSS, hypertension, diabetes mellitus, WBC and SUA. All of them are clinical data acquired in general clinical practice easily and inexpensively. The instruction of using the nomogram is as follows: locate the patient's gender and draw a line straight upward to the 'Points' axis to determine the score associated with the gender. Repeat the process for each variable, and sum the scores achieved for each covariate, and locate this sum on the 'Total Points' axis. Draw a line straight down to determine the likelihood of unfavourable outcome after 3 months of onset. For example, there is a 50 -year-old female patient with hypertension, without diabetes mellitus, baseline NIHSS=3, WBC=10.0×10^9/L, SUA=400 μmol/L. As is shown in figure 7, total points=17.5+21+19.5+0+52+38+50=198.

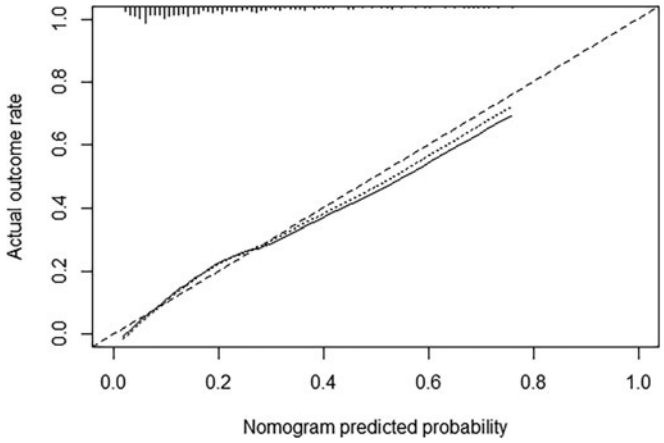

Figure 4 Calibration curve of the nomogram for predicting 3-month unfavourable outcome rate in the derivation cohort. Actual unfavourable outcome rate is plotted on the y-axis; nomogram-predicted probability of unfavourable outcome is plotted on the x-axis.

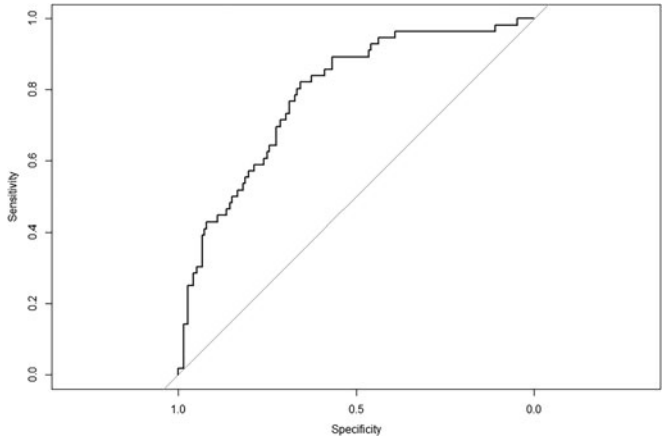

Figure 5 ROC of the nomogram in the validation cohort. It showed good discrimination with a C-index of 0.787 (95% CI 0.722 to 0.853).

C-index, concordance index; ROC, receiver operating characteristic curve.

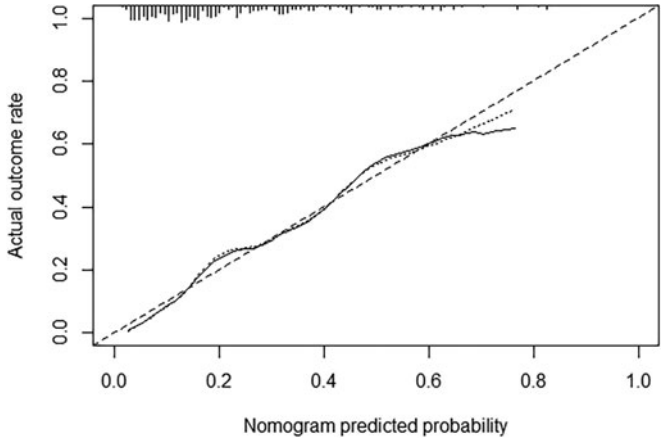

Figure 6 Calibration curve of the nomogram for predicting 3-month unfavourable outcome rate in the validation cohort. Actual unfavourable outcome rate is plotted on the y-axis; nomogram-predicted probability of unfavourable outcome is plotted on the x-axis.

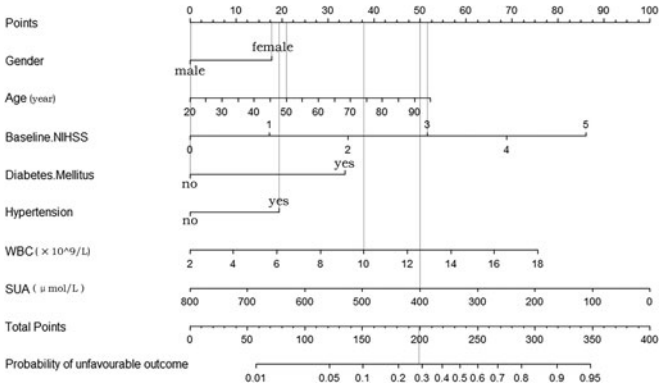

Figure 7 A 50 -year-old female patient with hypertension, without diabetes mellitus, baseline NIHSS=3, WBC=10.0×10^9/L, SUA=400 µmol/L. Total points=17.5+21+19.5+0+52+38+50=198.The predicted probability of unfavourable outcome after 3 months of onset is 28%.

Baseline NIHSS, NIHSS at admission; NIHSS, National Institute of Health Stroke Scale; SUA, serum uric acid; WBC, white blood cell.

The predicted probability of unfavourable outcome after 3 months of onset is 28%. Additionally, the nomogram showed excellent discriminative ability in both derivation cohort and validation cohort when validated internally and externally. As was shown in the result, patients with minor stroke and TIA can benefit from higher SUA levels. Female, older age, higher baseline NIHSS, diabetes mellitus, hypertension and higher WBC were positively correlated with unfavourable outcome.

Among the independent predictors, baseline NIHSS contributed the most to predicting unfavourable outcome. It has been widely accepted that baseline NIHSS was the most important prognostic factor for patients with ischaemic stroke. Baseline NIHSS, representing the extent of neurological deficits, is the most important indicator of the severity of patients with stroke.[12] It is well understood that patients with more severe conditions have a higher probability of poor long-term outcome. Previous studies had also certified such relationship.[4 13 14] In our study, we found that female patients are more likely to end up with a poor prognosis, which was consistent with the results of the Platelet-Oriented Inhibition in TIA and Minor Ischemic Stroke Trial study.[15] Hypertension and diabetes are two important risk factors for ischaemic stroke; in line with the previous studies, both of them were associated with prediction of poor outcomes in patients with minor stroke and TIA.[4 15] WBC was an important inflammatory factor. In the China Antihypertensive Trial in Acute Ischemic Stroke study, a total of 3891 patients with cerebral infarction were included in the analysis, and the results showed WBC was positively related to the poor prognosis of cerebral infarction.[7]SUA is another biomarker in the nomogram. It is a well-known neuroprotective antioxidant for its free radical scavenger activity.[16] A prospective study involving 881 patients with acute ischaemic stroke found that neurological impairment on admission and lesion volumes were inversely associated with SUA levels.[17] A systematic review and meta-analysis of 8131 subjects showed that SUA had neuroprotective effects in patients with acute ischaemic stroke, and higher SUA was a biomarker for excellent outcome.[8] The reasons why SUA has such neuroprotective effect may include the following two points: (1) SUA is a strong anti-oxidant substance with anti-lipid peroxidation, which can reduce oxidative stress in cerebral ischaemia and protect brain cells,[18] and (2) SUA can also remove excessive free radicals released from brain tissue ischaemia to allaeviate ischemia–reperfusion injury.[19]

It is worth noting that the efficacy of rt-PA intravenous thrombolytic therapy in patients with minor stroke and TIA remains controversial. One of the important reasons is that this kind of patients were often excluded in the previous large-scale thrombolytic clinical studies.[20] Recently, the potential of rtPA for Ischemic Strokes with Mild Symptoms study conducted in the USA from May 2014 to December 2016 demonstrated that there was no significant difference of the 90-day functional outcome between alteplase group and aspirin group.[21] According to our study, the result showed that patients with minor stroke and TIA were not benefited significantly from intravenous thrombolysis either.

Our study has several limitations. First, it was a retrospective study and was susceptible to the selection bias and recall bias. Some patients lost to follow-up, which may bias the results. Second, this study was based on data from single-centre patients with minor stroke and TIA. We look forward to receiving repeated results from other institutes. Third, to make the nomogram more convenient to be used in clinical practice, we had not included the biomarkers that are not widely used in clinical practice, such as soluble CD40 ligand,[22] lipoprotein-related phospholipase A2 activity[23] and anti-phosphatidylserine–prothrombin antibodies,[24] which have been proved to be associated with clinical outcomes in patients with minor stroke and TIA. If the aforementioned biomarkers were included, the sensitivity and specificity of the nomogram may be higher and the accuracy of the prediction will be better. However, including these biomarkers will make the progress of using our nomograms complicated, which we do not expect. Therefore, we only include the necessary factors concisely.

CONCLUSIONS

In conclusion, we developed and validated a nomogram predicting the outcome for patients with minor stroke and TIA after 3 months of onset. The proposed nomogram in our study provides statistically excellent discrimination. It offers a practical and feasible tool to predict outcomes. Not only could this nomogram help clinicians with clinical assessments and guiding treatment but also provide patients with counsel. To generalise the use of the nomogram, validation in other stroke units is expected.

Main messages

- First, patients with minor stroke and TIA can benefit from higher SUA levels. Female, older age, higher baseline NIHSS, diabetes mellitus, hypertension and higher WBC were positively correlated with unfavourable outcome.
- Second, we developed and validated a nomogram predicting the outcome for patients with minor stroke and transient ischaemic attack after 3 months of onset.
- At last, the nomogram offers a practical and feasible tool to predict outcomes. Not only could this nomogram help clinicians with clinical assessments and guiding treatment but also provide patients with counsel.

What is already known on the subject

- First, minor stroke and transient ischemic attack account for a considerable part of ischemic stroke.
- Second,a considerable percentage of patients with minor stroke or transient ischemic attack end in a poor outcome.

Current research questions

- First, our study was a retrospective study and was susceptible to the selection bias and recall bias. Some patients lost to follow-up, which may have an impact on the results. Second, this study was based on data from single-centre patients with minor stroke and transient ischaemic attack (TIA). We look forward to receiving repeated results from other institutes.
- Third, to make the nomogram more convenient to be used in clinical practice, we had not included the biomarkers that are not widely used in clinical practice, such as soluble CD40 ligand, lipoprotein-related phospholipase A2 activity and anti-phosphatidylserine-prothrombin Antibodies, which have been proved to be associated with clinical outcomes in patients with minor stroke and TIA. If the above biomarkers were included, the sensitivity and specificity of the nomogram may be higher and the accuracy of the prediction will be better.

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