• Clinical Research •

Nomogram model for predicting oculomotor nerve palsy in patients with intracranial aneurysm

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Received: 2021-01-31 Accepted: 2022-04-14

Abstract

● **AIM:** To explore the risk factors of oculomotor nerve palsy (ONP) in patients with intracranial aneurysm (IA) and develop a nomogram model for predicting ONP of IA patients.

● **METHODS:** A total of 329 IA patients were included. Logistic regression analysis was applied to identify independent factors, which were then integrated into the nomogram model. The performance of the nomogram model was evaluated by calibration curve, receiver operating curve (ROC), and decision curve analysis.

● **RESULTS:** Univariate and multivariate logistic regression analysis indicated posterior communicating artery (PCoA) aneurysm [hazard ratio (HR)=17.13, *P*<0.001] and aneurysm diameter (HR=1.31, *P*<0.001) were independent risk factors of ONP in IA patients. Based on the results of logistic regression analysis, a nomogram model for predicting the ONP in IA patients was constructed. The calibration curve indicated the nomogram had a good agreement between the predictions and observations. The nomogram showed a high predictive accuracy and discriminative ability with an area under the curve (AUC) of 0.863. The decision curve analysis showed that the nomogram was powerful in the clinical decision. PCoA aneurysm (HR=3.38, *P*=0.015) was identified to be the only independent risk factor for ONP severity.

● **CONCLUSION:** PCoA aneurysm and aneurysm diameter are independent risk factors of ONP in IA patients. The nomogram established is performed reliably and accurately for predicting ONP. PCoA aneurysm is the only independent risk factor for ONP severity.

● **KEYWORDS:** intracranial aneurysm; oculomotor nerve palsy; Logistic regression analysis; posterior communicating artery; nomogram

DOI:10.18240/ijo.2022.08.14

Citation: Cui YY, Wang B, Jiang B, Zhao SH. Nomogram model for predicting oculomotor nerve palsy in patients with intracranial aneurysm. *Int J Ophthalmol* 2022;15(8):1316-1321

INTRODUCTION

Intracranial aneurysm (IA) is a common cerebrovascular condition with a rupture rate of 13%-25%[1-3]. Because of the following subarachnoid hemorrhage (SAH) and its complications, the mortality rate of ruptured IA is 27%-45%[4]. Regrettably, the early diagnostic rate for IA is relatively low due to the lack of specificity of the presenting symptoms. Choosing the optimal treatment time and measures for unruptured IA is controversial. Some scholars held that unruptured IA with a diameter less than 5 mm could be treated conservatively[5]. However, it has been reported that the rupture rate of small- and medium-sized aneurysms in the Japanese was significantly higher than that in Europeans and Americans[6]. Although the rupture rate of a small aneurysm has been reported to be low, it can cause life-threatening SAH once ruptured[7]. To the best of our knowledge, the clinical treatment principles for small aneurysms have not been established. A considerable number of IA patients are in the stage of clinical follow-up due to differences in medical conditions and clinician's treatment principles[8].

Oculomotor nerve palsy (ONP) is a common neuro-ophthalmologic condition with manifestations of ptosis, exotropia, eye movement restrictions, dilated pupil, loss of

pupillary light reflex, and diplopia. The etiology of ONP is complex, such as diabetes mellitus, IA, tumors, painful ophthalmoplegia, pituitary lesions, cavernous sinus lesions, central nervous system infections, and SAH[9]. Recent reports indicated that 29.8% of ONP was caused by IA, a potentially life-threatening condition with a high morbidity and mortality rate[10]. Thus this study aimed to explore the predictive factors of ONP in patients with IA. Moreover, we also established a nomogram for individualized prediction of ONP prognosis with IA.

SUBJECTS AND METHODS

Ethical Approval The study was approved by the Clinical Research Ethics Committee of Shanghai Changhai Hospital and Peking University International Hospital. Informed consent was waived due to the retrospective nature of the study.

Subjects and Clinical Data Consecutive patients diagnosed with IA from January 2012 to December 2019 were retrospectively identified by searching the electronic medical system. We confirmed the diagnosis of IA by imaging scans (computed tomography, magnetic resonance imaging) or cerebral angiography (Figure 1). The exclusion criteria were as follows: 1) presence of other intracranial lesions or neurological conditions; 2) concomitant eye diseases other than ONP, such as strabismus, glaucoma, fundus ophthalmopathy,and optic neuropathy; 3) ONP caused by other conditions, such as cerebrovascular infarction, hemorrhagic disease, diabetes, intracranial inflammation, trauma or congenital ONP. Also we retrieved clinical characteristics which included patient age, sex, concomitance with ONP or not, ONP degree, number of aneurysms, aneurysm diameter and location, the time span from symptom onset to treatment, presence of SAH or not, history of hypertension and cerebral infarction. Complete ONP was defined as complete ptosis of the upper eyelid, paralysis of the external ocular muscle, dilated pupil, and disappearance of direct or interfacial light reflection. By contrast, incomplete ptosis or partial vision, inner vision, impaired vision or incomplete pupil dilation and reduced light reflex constituted partial ONP. IA diameter was calibrated by the distance from the midpoint of the aneurysm neck plane to the furthest point of the aneurysm in digital subtraction angiography images.

Statistical Analysis Variables with normal and skewed distribution were presented as mean±standard deviation (SD) and median (range), respectively. Student *t*-test and the Mann-Whitney *U* test were used to compare two groups, as appropriate. Chi-square test was carried out for dichotomous data, and non-parametric test was employed for hierarchical data. Univariate and multivariate analyses of influencing factors were performed by logistic regression analysis.

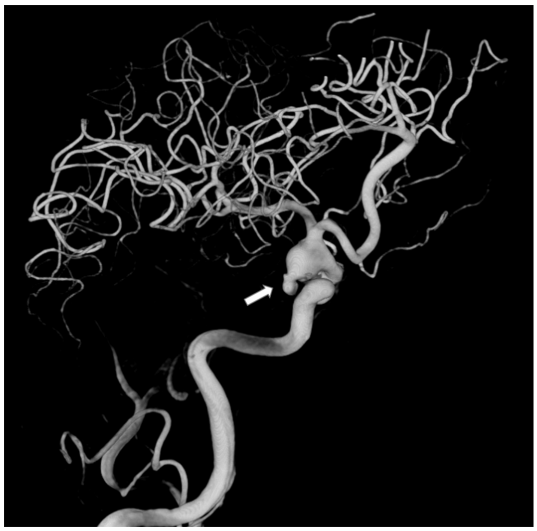

Figure 1 Imaging data of one posterior communicating artery aneurysm patient Digital substraction angiography (DSA) shows a posterior communicating artery aneurysm (white arrow).

Variables were significantly related to ONP, and the degree of ONP in the univariate analysis (P<0.05) was subsequently selected for multivariate Logistic regression analysis. A two-sided P value less than 0.05 was considered to be statistically significant.

To further facilitate individualized prediction of ONP, a nomogram were established using the rms package in R, version 3.5.1 based on the results of multivariate Logistic regression analysis. In order to verify the prediction ability of the nomogram, a calibration curve was performed to evaluate the calibration ability of nomogram according to the consistency between the nomogram prediction and observed real outcomes. Subsequently, the area under the curve (AUC) was calculated with the receiver-operating characteristic curve (ROC). In order to evaluate the clinical utility of the nomogram, decision curve analysis (DCA) was performed by rmda package of R to analyze the net benefit of the nomogram we established.

RESULTS

Clinical Features The clinical data of 329 IA patients collected (124 males and 205 females, 81 with ONP and 248 without ONP) were presented in Table 1. There were significant differences with regard to gender, location of the aneurysm, diameter of aneurysm and cerebral infarction between the ONP group and non-ONP group (all P<0.05).

Predictors for Oculomotor Nerve Palsy The results of the univariate and multivariate analysis were shown in Table 2. By univariate analysis, female gender, PCoA aneurysm and aneurysm diameter were identified to be predictive factors of ONP. Nonetheless, only PCoA aneurysm [hazard ratio (HR)=17.13, 95% confidence interval (CI): 7.93-37.01, P<0.001] and aneurysm diameter (≥15 mm; HR=1.31, 95%CI:

Table 1 Baseline clinicopathologic characteristics of patients with intracranial aneurysm

Features	non-ONP (*n*=248)	ONP (*n*=81)	*P*
Sex (female/male)	144/104	61/20	0.005
Age (≥60y/<60y)	102/146	36/45	0.600
Location (PCoA aneurysm/other sites)	45/203	50/31	<0.001
Mean diameter (mm)	5.869	14.219	<0.001
SAH (yes/no)	53/195	16/65	0.756
Number (multiple/single)	40/208	16/85	0.451
Interval time (<7d/7-30d/≥30d)	52/83/113	18/31/32	0.619
Hypertension (yes/no)	121/127	40/41	0.926
Cerebral infarction (yes/no)	36/212	4/77	0.022

ONP: Oculomotor nerve palsy; PCoA: Posterior communicating artery; SAH: Subarachnoid hemorrhage.

Table 2 Independent factors predicting oculomotor paralysis in patients with intracranial aneurysm

Features	Univariate analysis			Multivariate analysis		
	HR	95%CI	*P*	HR	95%CI	*P*
Sex (female *vs* male)	0.45	0.26-0.80	0.006	0.65	0.32-1.31	0.225
Age (≥60y *vs* <60y)	0.15	0.69-1.90	0.600	NA	NA	NA
Location (PCoA aneurysm *vs* other sites)	7.28	4.19-12.64	<0.001	17.13	7.93-37.01	<0.001
Diameter (≥15 mm *vs* <15 mm)	1.24	1.17-1.32	<0.001	1.31	1.21-1.41	<0.001
SAH (yes *vs* no)	1.10	0.59-2.06	0.756	NA	NA	NA
Number (multiple *vs* single)	1.28	0.67-2.44	0.452	NA	NA	NA
Interval time						
7-30d *vs* <7d	1.08	0.55-2.12	0.826	NA	NA	NA
≥30d *vs* <7d	0.82	0.42-1.59	0.544	NA	NA	NA
Hypertension (yes *vs* no)	0.98	0.59-1.61	0.926	NA	NA	NA
Cerebral infarction (yes *vs* no)	3.27	1.13-9.49	0.029	2.917	0.798-10.660	0.106

PCoA: Posterior communicating artery; SAH: Subarachnoid hemorrhage; HR: Hazard ratio; 95%CI: 95% confidence intervals; NA: Not available.

1.21-1.41, *P*<0.001) were independent risk factors of ONP by multivariate analysis.

Nomogram for Oculomotor Nerve Palsy and Performance

We established the nomogram for predicting the prognosis of ONP according to the independent risk factor identified by the multivariate logistic regression analysis. A score of 0-100 was assigned to each variable, which was transformed to the coefficients of the independent risk factor. The sum of corresponding scores of different variables was added to reach the total score, which corresponded to the risk axis that denoted the risk of individual ONP. The greater the influence of the variables, the higher the nomogram score. The nomogram we established suggested that aneurysm diameter had the greatest impact on ONP, followed by location (Figure 2). In a representative case, a 56-year-old man with PCoA aneurysm (21 points) that measured 25 mm (50 points), leading to a sum score of 71 points that could be converted to >90% probability of ONP.

The calibration curve was conducted to evaluate the performance of the nomogram in ONP, which indicated that the nomogram possessed favourable calibration and discriminative ability supported by a fair uniformity between prediction and observation of the nomogram (Figure 3). The ROC curve showed the high diagnostic utility of the nomogram (Figure 4), as indicated by an AUC of 0.863. Furthermore the clinical practicality of nomogram was established by the DCA which showed satisfactory net benefits among most of the threshold probabilities in both groups and was superior in predicting ONP than conventional predictive methods. All these data supported that the nomogram we established provided constructive guidance for clinical decision-making (Figure 5).

Independent Factors for Oculomotor Nerve Palsy Severity

We further categorized ONP patients into the complete and partial ONP group based on the degree of ONP (35 complete ONP and 46 partial ONP). Patients with partial ONP tended to have PCoA more than complete ONP (*P*=0.01). The results of univariate and multivariate analysis of ONP severity were shown in Table 3. The results indicated that PCoA aneurysm (HR=3.38, 95%CI: 1.27-8.98, *P*=0.015) was an independent risk factor of ONP severity.

Table 3 Independent prognostic factors predicting severity in patients with both intracranial aneurysm and oculomotor paralysis

Features	Univariate analysis			Multivariate analysis		
	HR	95%CI	*P*	HR	95%CI	*P*
Sex (female *vs* male)	0.91	0.33-2.51	0.852	NA	NA	NA
Age (≥60y *vs* <60y)	1.10	0.45-2.65	0.841	NA	NA	NA
Location (PCoA aneurysm *vs* other sites)	3.38	1.27-8.98	0.015	3.38	1.27-8.98	0.015
Diameter (≥15 mm *vs* <15 mm)	0.97	0.93-1.02	0.238	NA	NA	NA
SAH (yes *vs* no)	1.41	0.47-4.22	0.542	NA	NA	NA
Number (multiple *vs* single)	0.53	0.17-1.70	0.286	NA	NA	NA
Interval time						
7-30d *vs* <7d	0.94	0.29-2.97	0.913	NA	NA	NA
≥30d *vs* <7d	0.52	0.16-1.70	0.282	NA	NA	NA
Hypertension (yes *vs* no)	0.63	0.26-1.53	0.307	NA	NA	NA
Cerebral infarction (yes *vs* no)	4.22	0.42-42.42	0.222	NA	NA	NA

PCoA: Posterior communicating artery; SAH: Subarachnoid hemorrhage; HR: Hazard ratio; 95%CI: 95% confidence intervals; NA: Not available.

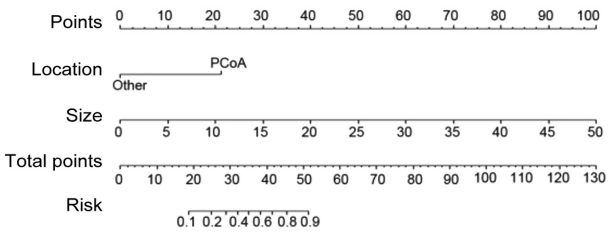

Figure 2 Nomogram for predicting ONP in patients with intracranial aneurysm PCoA: Posterior communicating artery; ONP: Oculomotor nerve palsy.

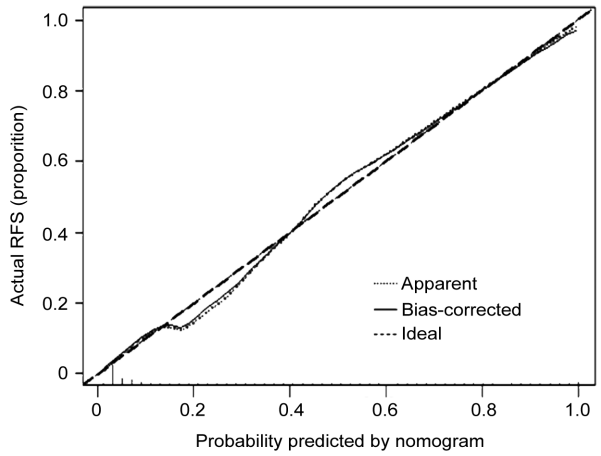

Figure 3 The calibration curve for predicting ONP in patients with intracranial aneurysm in the training cohort RFS: Recurrence-free survival; ONP: Oculomotor nerve palsy.

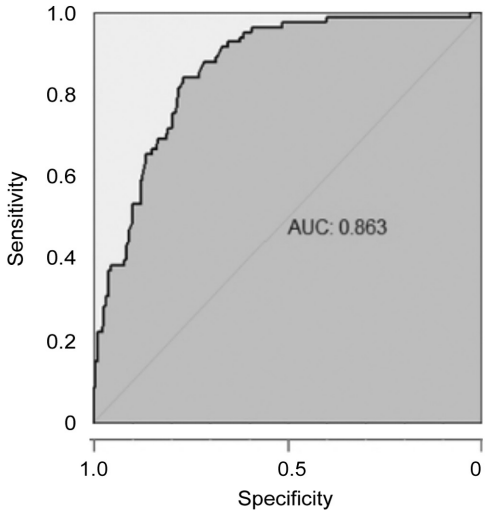

Figure 4 The ROC curve for evaluating the diagnostic performance of the nomogram AUC: Area under the curve; ROC: Receiver-operating characteristic curve.

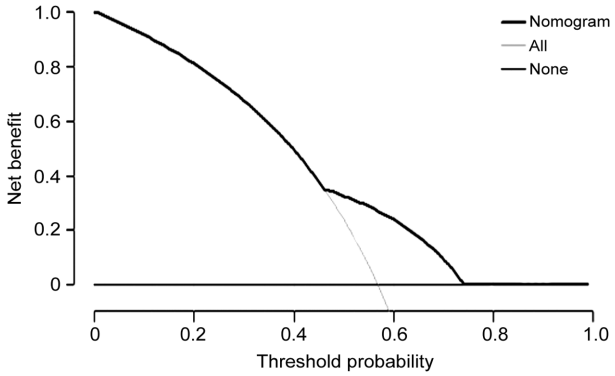

Figure 5 Decision curve analysis (DCA) for nomogram.

DISCUSSION

IA is a common cerebrovascular condition that is predominantly caused by injury of vascular intima associated with wall shear stress[11-13]. When ruptured, they could easily generate SAH that may cause high morbidity and mortality.

The oculomotor nerve is the third cranial nerve that emerges from the interpeduncular fossa in the midbrain. It travels between the posterior cerebral artery and the superior cerebellar artery that parallels to the PCoA before finally enters the cavernous sinus. Any IA adjacent to the oculomotor nerve may predispose to the development of ONP. This study revealed the correlation between IA and ONP. More importantly, we established an individualized prediction model for assessing

the risk of ONP in patients with IA, which may have important clinical significance for guiding clinicians to make appropriate medical decisions and reduce the risk of disability or mortality.

The present study showed that PCoA aneurysm was an independent risk factor for ONP and its severity. Of the total 329 IA patients, 81 were having ONP, which is consistent with previous studies reporting that IA, especially PCoA aneurysms is a common cause of ONP. For example, it has been noted that approximately 7%-23% of patients with PCoA aneurysms will develop ONP[14-16]. The intimate relationship between PCoA aneurysm and ONP may be closely related to the anatomy and structure of the oculomotor nerve. First, the oculomotor nerve travels on the lower lateral side of the PCoA and is closely surrounded by the arachnoid membrane, thus the puffed aneurysm could directly compresses the adjacent oculomotor nerve. The persistent throbbing of the aneurysm causes chronic damages to the oculomotor nerve with nerve venous congestion and oedema. Furthermore, ruptured aneurysms can directly stimulate the oculomotor nerve or produce a progressive effect[17-19]. ONP is an important and clinically useful indicator of PCoA aneurysm on the verge of rupture[14,20]. SAH occurs in about 50% of PCoA aneurysm patients with ONP, of which only 15% presented with ONP prior to aneurysm rupture. It is reported that an unruptured PCoA aneurysm has similar morphologic and hemodynamic characteristics with a ruptured PCoA aneurysm in ONP patients[21]. The degree of ONP in the early period is a useful predictor of ONP recovery in both ruptured and unruptured aneurysms[22]. The outcome for complete ONP is generally worse than partial ONP[21]. This study alerted clinicians to pay more attention to ONP once they have encountered IA patients, especially those with PCoA aneurysms. IA patients with ONP (especially complete ONP) should be given more vigilance and timely treatment to avoid the life-threatening rupture of aneurysms.

Currently, the relationships between aneurysm diameter and ONP occurrence have not been thoroughly established. Chalouhi *et al*[23] found that the diameters of PCoA aneurysms with ONP were larger than those without ONP. Previous reports[24-25] indicated that aneurysms with a diameter greater than 4 or 7 mm were inclined to generate ONP. In line with these reports, our study showed that IA diameter was an independent predictive factor for ONP. It was reported that 54% of ruptured aneurysms ranged from 5 to 10 mm in diameter[2]. Aneurysms larger than 7 mm in diameter tended to rupture[26], the risk of which can be reduced by 59% if active treatment is initiated[2]. Other risk factors for aneurysm rupture include location, genetics, smoking and others[27]. Clinicians can better assess the risk based on the aneurysms' diameter and thus take more active therapeutic measures.

Nomogram provides individualized risk assessment based on clinical variables. This study has established a practical and internally validated nomogram for predicting the prognosis of ONP with IA. The nomogram showed that PCoA aneurysm and IA diameters were independent risk factors for ONP. The calibration curve indicated that the nomogram possessed favourable calibration and discriminative ability, which was further validated by the high AUC. In addition, the DCA proved that the nomogram was beneficial to the clinical decision by demonstrating better net benefit for predicting the prognosis of ONP. The nomogram we established has demonstrated adequate discriminative and diagnostic value to predict the occurrence of ONP individually.

This study suffers several limitations. First, the small sample size and the retrospective nature of the design would limit study robustness. Prospective studies with larger sample size and longer follow-up is required in the future. Second, all the patients included were Chinese that limited the study's extrapolation result to patients with other ethnicities. A heterogeneous patient population that represented different genetic backgrounds in multi-centers would further validate our study results. Third, additional risk factors, such as tumor orientation and tumor morphology, may also influence study results.

In conclusion, early recognition and evaluation of ONP is important to rule out potential PCoA aneurysms. Especially, more attention should be paid to PCoA aneurysm and aneurysm diameter. The nomogram we established for individualized prediction of the prognosis of ONP with IA has a good predictive accuracy and clinical diagnostic value, which provided important clinical evidence for clinicians in the diagnosis and selection of therapeutic schedule. Ultimately it contributed to reducing the incidence rate of ONP and rupture rate of IA and improving the quality and survival of life.

ACKNOWLEDGEMENTS

The authors thank Hong Liu and Ji-Fang Qu for their support and help in writing the manuscript.

Authors' contributions: Conception and design: Zhao SH; Provision of study materials or patients: Cui YY and Wang B; Collection and assembly of data: Cui YY and Jiang B; Data analysis and interpretation: Cui YY, Jiang B, and Wang B; Manuscript writing: All authors; Final approval of manuscript: All authors.

Foundation: Supported by National Natural Science Foundation of China (No.81970822).

Conflicts of Interest: Cui YY, None; **Wang B,** None; **Jiang B,** None; **Zhao SH,** None.

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