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RESEARCH

A nomogram based on clinical multivariate factors predicts delayed cure after microvascular decompression for hemifacial spasm

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Abstract

Background The course of disease after microvascular decompression (MVD) in patients with hemifacial spasm (HFS) is variable. The purpose of this study was to develop and validate a nomogram to predict the probability of delayed cure after microvascular decompression in patients with hemifacial spasms based on clinical multivariate factors.

Methods A retrospective data collection was performed on 290 patients with HFS undergoing MVD at our center from January 2017 to January 2022. The patients were randomly assigned to the training cohort ($n = 232$) and validation cohort ($n = 58$) at a ratio of 8:2. Retrospective analysis was performed of information on clinical, radiological, and intraoperative findings and clinical outcomes. Univariate and multivariate analyses were performed in the training cohort, and a nomogram was constructed using a stepwise logistic regression approach. The receiver operating characteristic (ROC) was calculated to evaluate the reliability of the nomogram model. Decision curve analysis (DCA) was used to assess the clinical application value of the nomogram model.

Results In the training cohorts, 73 patients (73/232) had a delayed cure. In the validation cohorts, 18 patients (18/58) had a delayed cure. We developed a novel nomogram model to predict the risk of delayed cure after MVD in HFS patients based on the presence of vertebral artery compression, venous compression, absence of LSR, degree of facial nerve indentation, degree of neurovascular compression, and internal auditory canal vascular looThe area under the curve (AUC) of the nomogram model was 0.9483 in the training cohort and 0.9382 in the validation cohort. The calibration curve showed good correspondence between the predicted and actual probabilities in the training and validation groups. The decision curve showed that the nomogram model had good performance in clinical applications.

Conclusions We developed and validated a preoperative and intraoperative multivariate factors nomogram to predict the possibility of delayed cure after MVD in HFS patients, which may help clinicians in the comprehensive management of HFS.

Keywords Hemifacial spasm · Delayed cure · Nomogram · Prediction model

Background

Hemifacial spasm (HFS) is characterized by involuntary spasms, tonic contractions, and synchronous movements of the muscles innervated by the facial nerve [1, 2]. The theory of neurovascular compression (NVC) has been widely accepted, making microvascular decompression (MVD) one of the most effective and reliable methods of treating HFS, with a cure rate of around 90% [3]. Interestingly, the postoperative course of MVD in HSF patients was variable, even when treated with the same surgical procedure. Delayed cure is not uncommon, as previous studies have reported that approximately 3.6 to 50.3% of HFS patients experience delayed cure after MVD [4–6]. Previous studies have found that many factors affect delayed cure, including disease progression, severity of HFS symptoms, resolution of abnormal muscle response (AMR) during surgery, and surgical methods [4–6]. However, most current studies are single-factor studies that isolate the relationship between different factors rather than integrating them. This shortcoming has

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been addressed by the development of nomograms, which can integrate various important factors to model and predict patient prognosis. At present, nomograms have been used in the diagnosis and prognostic evaluation of tumor diseases, but we have not found a nomogram to predict the delayed cure of HFS after MVD.

Therefore, the purpose of this study was to explore the possible risk factors for delayed cure after MVD in patients with HFS. To construct and validate a nomogram model to predict the risk of delayed cure after MVD in HFS patients. This study should further assist clinicians in differentiating patients at high risk for delayed cure.

Methods

Patients and study design

Data from patients with HFS who underwent MVD at Zhongnan Hospital of Wuhan University from January 2017 to January 2022 were retrospectively analyzed. The Ethics Committee of Zhongnan Hospital, Wuhan University, approved this study. Informed consent was waived due to the retrospective character of this study. Inclusion criteria were (1) age ≥18 years and (2) primary HFS patients treated with MVD for the first time. Exclusion criteria were (1) postoperative delayed facial paralysis and (2) secondary HFS. All patients underwent preoperative magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) to exclude the possibility of secondary HFS.

Clinical data collection

Clinical data were collected through the medical record, including sex, age, body mass index (BMI), duration of HFS symptoms, sides of HFS, history of hypertension, history of diabetes, history of hyperlipidemia, history of botulinum toxin injection, preoperative tinnitus, and preoperative hearing loss. The degree of NVC was assessed by preoperative MRI, and based on previous studies, NVC was graded as follows: Grade I indicates the absence of vessels close to the nerve or the presence of vessels close to the nerve but not in direct contact with the nerve. Grade II indicates that there is vascular compression of the nerve, but not enough to distort the normal course of the nerve. Grade III indicates that the vessel distorts the normal course of the nerve.

Surgery

All patients were operated in the lateral decubitus position under electrophysiological monitoring. A rectosigmoid approach was used and a straight incision of approximately 5 cm was made within the retroarticular hairline. A 1.5 cm diameter bone hole was drilled into the skull at the junction of the sigmoid and transverse sinuses. The bone around the bone hole was removed with bone forceps and retained to form a 3cm×3cm bone window. After collecting the lateral spreading reaction (LSR) waveform, the dura mater was incised in an arc and the cerebrospinal fluid was slowly released. Under the microscope, the arachnoids around the facial and auditory nerves were carefully classified and the facial nerve root outlet zone (REZ) was exposed. The entire facial nerve was then carefully examined from the root of the brainstem to the internal auditory canal. All blood vessels associated with the facial nerve were removed, and an appropriately sized Teflon sponge was placed between them. The LSR was again monitored, and after saline irrigation, the dura mater was sutured. The previously retained bone fragments were covered with a gelatin sponge after placement in situ, and the skin was sutured in layers. The whole procedure was videotaped.

Intraoperative findings

Intraoperative findings, including compression vessels and the degree of depression in the REZ, were confirmed by surgical video and operative notes. The indentation caused by vascular compression of the facial nerve root entry zone was evaluated in the surgical field and classified into three grades: Grade I, no indentation or mild indentation; Grade II, moderate indentation; and Grade III, severe indentation and discoloration [7–9].

Intraoperative electrophysiological monitoring

All patients underwent intraoperative electrophysiological monitoring with LSR and brainstem auditory-evoked potential (BAEP). Intraoperative electrophysiological monitoring was performed by experienced technicians using an electromyogram (EMG) instrument. The recording electrode (needle electrode) was placed on the affected side of the orbicularis oculi muscle and the ipsilateral mentalis muscle, and the stimulating electrode was placed on the mandibular branch of the facial nerve. The stimulation intensity was 300–500mA, the sampling time was 30ms, and the monitoring parameters included LSR latency and amplitude. Multiple examinations were performed to detect the elimination of the LSR.

Outcomes

All patients were followed up for at least 1 year after MVD, including outpatient and telephone follow-uAccording to the duration and spasticity of the spasm after MVD, the cure group was divided into an immediate cure group and a delayed cure grouImmediate cure was defined as the complete

disappearance of spasticity and achievement of a spasticity-free state on the second day after surgery. Delayed cure was defined as incomplete resolution of spasticity on the second day after surgery, including improvement or no significant improvement in spasticity symptoms.

Construction of the nomogram

Baseline characteristics of the training cohort were compared according to the presence or absence of delayed healing after MVD. First, univariate analysis was used to compare all variables between the immediate cure and delayed cure groups, and univariate logistic regression was used to identify risk factors for delayed cure in the training cohort. Variables that were statistically significant in univariate logistic regression were then analyzed using stepwise multiple logistic regression. The nomogram model was constructed using variables that were statistically significant in the multivariate analysis. Scores were calculated for each variable based on the regression coefficient values.

Validation of the nomogram

Validation of the nomogram model was performed in the training and validation groups. First, the area under the curve (AUC) of the receiver operating characteristic (ROC) was used to assess the discriminative power of the nomogram in the training grouCalibration curves were evaluated graphically by smoothing scatter plots of predicted and actual probabilities. Decision curve analysis (DCA) was used to determine the net clinical benefit of the model. ROC, calibration curve, and DCA analyses were then repeated in the validation group.

Statistical analysis

Normal distribution was tested by the Shapiro–Wilk test. Continuous variables with a normal distribution were expressed as mean and standard deviation (SD). Categorical variables were expressed as frequencies (percentages), and the χ^2 test was used to compare between groups. The comparison of continuous variables between the two groups was performed using the *t* test or the Kruskal-Walli test. All statistical analyses were performed using the R software (version 3.5.1, R foundation; Vienna, Austria) and SPSS 25.0 (IBM, Armonk, NY, USA). Statistical significance was set at a bilateral *p* value of less than 0.05.

Results

Baseline data

A total of 290 patients with HFS who met the study criteria were randomly divided into a training group ($n = 232$) and a validation group ($n = 58$) in a ratio of 8:2. In the training and validation cohorts, there were 73 (73/232) and 18 (18/58) patients with delayed cure after surgery, respectively. Between the training and validation cohorts, there were no significant differences in baseline characteristics (Table 1).

Univariate and multivariate analyses

Univariate logistic regression analysis was performed between the immediate cure and delayed cure groups in the training cohort to identify risk factors for delayed cure (Table 2). Significant differences were found in the following variables: duration of symptoms, vertebral artery compression, venous compression, LSR, indentation grade, compression severity, loop characteristics, and compression site. The statistically significant variables in the univariate analysis described above were then included in the multivariate logistic regression analysis. Multivariate logistic regression analysis showed that vertebral artery compression, venous compression, LSR, indentation grade, compression severity, and loop characteristics were independently associated with the occurrence of delayed cure.

Nomogram development and validation

A nomogram was developed from the training cohort data based on univariate and multivariate logistic regression analysis (Fig. 1). The AUC of the training cohort was 0.9483 (Fig. 2a). The AUC of the validation cohort was 0.9382 (Fig. 2b). The nomogram had a good predictive performance in both the training and validation sets. The nomogram had a good predictive performance in both the training cohorts and validation cohorts. The calibration plot of the nomogram shows the training cohorts (Fig. 3a) and the validation cohorts (Fig. 3b). DCA (Fig. 4a and Fig. 4b) showed that our nomogram model provided a better net benefit in predicting delayed cures. This indicates that the nomogram has good performance for clinical application.

Discussion

Neurovascular compression is the main cause of HFS, as confirmed by some studies [8, 10, 11]. However, the anatomical and hemodynamic changes caused by NVC alone

Table 1 The comparison of baseline characteristics of the patients in training and validation cohorts

Variables	Feature	Training cohort (*n*=232)	Validation cohort (*n*=58)	*p* value
Sex	Male	77	22	0.523
	Female	155	36	
Age (years)	Mean ± SD	52.73±10.238	53.36±10.726	0.679
Side	Left	117	29	0.93
	Right	115	29	
BMI	Mean ± SD	23.725±3.41	23.299±3.627	0.402
Symptom duration (month)	Mean ± SD	140.14±125.402	139.24±140.863	0.451
Hypertensive	Yes	53	14	0.822
	No	179	44	
Diabetes	Yes	22	4	0.543
	No	210	54	
Hyperlipidemia	Yes	24	5	0.702
	No	208	53	
Botulinum toxin	Yes	43	6	0.14
	No	189	52	
Tinnitus	Yes	25	3	0.199
	No	207	55	
Hearing loss	Yes	19	5	0.908
	No	213	53	
Preoperative facial paralysis	Yes	30	9	0.597
	No	202	49	
Vertebral artery compression	Yes	86	18	0.403
	No	146	40	
Vein compression	Yes	24	3	0.228
	No	208	55	
Offending artery	Single	120	37	0.78
	Multiple	112	21	
Abnormal muscle response	Unchanged	44	10	0.773
	Disappeared	188	48	
Cohen degree	I	158	38	0.211
	II	19	9	
	III	55	11	
Compression severity score	I	154	39	0.976
	II	35	9	
	III	43	10	
Loop characteristics	Yes	72	17	0.766
	No	160	41	
Compression site	Single	192	49	0.754
	Multiple	40	9	
Delayed cure	Delayed cure	73	18	0.965
	Immediate cure	159	40	

are not sufficient to explain the symptoms associated with NVC. Some other related factors such as demyelination, central sensitization, changes in brain structure (white matter/gray matter), and ion channels cannot be ignored [10, 12]. MVD has become the first-choice treatment for HFS due to its high cure rate. However, there are still 3.6–50.3% of MVD patients with HFS who are not cured immediately after surgery [4–6]. Although some patients experienced a period after surgery, these spasms gradually reduced and disappeared. However, delayed cure can cause psychological distress for patients. The cause of delayed cure is unknown. Some studies have found that delayed repair of the facial nerve, gradual stabilization, and plasticization of the facial nerve nucleus, which can cause symptoms, may be related

Table 2 Comparisons of baseline characteristics based on the presence or absence of a delayed cure, and univariate and multivariate logistic regression analyses of delayed cure in the training cohort

Variables	Feature	Training cohort		Univariate analysis		Multivariable analysis	
		Immediate cure (n =159)	Delayed cure (n =73)	OR (95% CI)	p value	OR (95% CI)	p value
Sex	Male	106 (66.7%)	48 (65.8%)	1.04 (0.58–1.87)	0.891		
	Female	53 (33.3%)	25 (34.2%)				
Age (years)	Mean ± SD	52.8 ± 9.9	52.4 ± 11.0	1.00 (0.97–1.02)	0.782		
Side	Left	79 (49.7%)	38 (52.1%)	1.10 (0.63–1.91)	0.738		
	Right	80 (50.3%)	35 (47.9%)				
BNI	Mean ± SD	23.9 ± 3.3	23.3 ± 3.6	1.05 (0.97–1.14)	0.222		
Symptom duration (month)	Mean ± SD	151.1 ± 140.7	110.6 ± 106.5	1.00 (1.00–1.00)	0.032*	1.00 (0.99–1.00)	0.106
Hypertensive	Yes	33 (20.8%)	19 (26%)	1.34 (0.70–2.57)	0.372		
	No	126 (79.2%)	54 (74%)				
Diabetes	Yes	14 (8.8%)	7 (9.6%)	1.10 (0.42–2.85)	0.847		
	No	145 (91.2%)	66 (90.4%)				
Hyperlipidemia	Yes	15 (9.4%)	9 (12.3%)	1.35 (0.56–3.25)	0.503		
	No	144 (90.6%)	64 (87.7%)				
Botulinum_toxin	Yes	30 (18.9%)	12 (16.4%)	0.85 (0.41–1.76)	0.656		
	No	129 (81.1%)	61 (83.6%)				
Tinnitus	Yes	15 (9.4%)	10 (13.7%)	1.52 (0.65–3.58)	0.333		
	No	144 (90.6%)	63 (86.3%)				
Hearing loss	Yes	12 (7.5%)	6 (8.2%)	1.10 (0.39–3.05)	0.859		
	No	147 (92.5%)	67 (91.8%)				
Preoperative facial paralysis	Yes	20 (12.6%)	10 (13.7%)	1.10 (0.49–2.49)	0.813		
	No	139 (87.4%)	63 (86.3%)				
Vertebral artery compression	Yes	46 (28.9%)	40 (54.8%)	2.98 (1.68–5.29)	<.001#	2.53 (1.15–5.57)	0.021*
	No	113 (71.1%)	33 (45.2%)				
Vein compression	Yes	6 (3.8%)	18 (24.7%)	8.35 (3.15–22.10)	<.001#	3.98 (1.13–14.01)	0.032*
	No	153 (96.2%)	55 (75.3%)				
Offending artery	Single	70 (44%)	42 (57.5%)	0.59 (0.34–1.03)	0.063		
	Multiple	89 (56%)	31 (42.5%)				
Abnormal muscle response	Unchanged	16 (10%)	28 (38.4%)	5.60 (2.78–11.27)	<.001#	6.77 (2.15–21.34)	0.001*
	Disappeared	143 (90%)	45 (61.6%)				
Cohen degree	I	127 (79.9%)	31 (42.5%)	9.16 (4.58–18.33)	<.001#	12.87 (4.47–37.07)	<.001#
	II	15 (9.4%)	4 (5.5%)				
	III	17 (10.7%)	38 (52.1%)				
Compression severity score	I	115 (72.3%)	39 (53.4%)	4.51 (2.21–9.18)	<.001#	5.86 (1.42–24.10)	0.014*
	II	27 (17%)	8 (11%)				
	III	17 (10.7%)	26 (35.6%)				
Loop characteristics	Yes	34 (21.4%)	38 (52.1%)	3.88 (2.14–7.01)	<.001#	6.95 (2.16–22.35)	0.001*
	No	125 (78.6%)	35 (47.9%)				
Compression site	Single	139 (87.4%)	53 (72.6%)	2.62 (1.31–5.26)	0.007*	0.86 (0.22–3.31)	0.832
	Multiple	20 (12.6%)	20 (27.4%)				

to delayed cure [13–15]. Further studies will be necessary in the future to investigate the specific mechanisms of this delay in healing. In this study, we developed a practical and simple nomogram for individual patients with HFS to predict the likelihood of postoperative MVD delayed cure. In the training and validation cohorts, the nomogram showed good discrimination and good clinical value. This nomogram, based on vertebral artery compression, venous compression, LSR, indentation grade, compression severity, and loop characteristics, provides a friendly and convenient tool

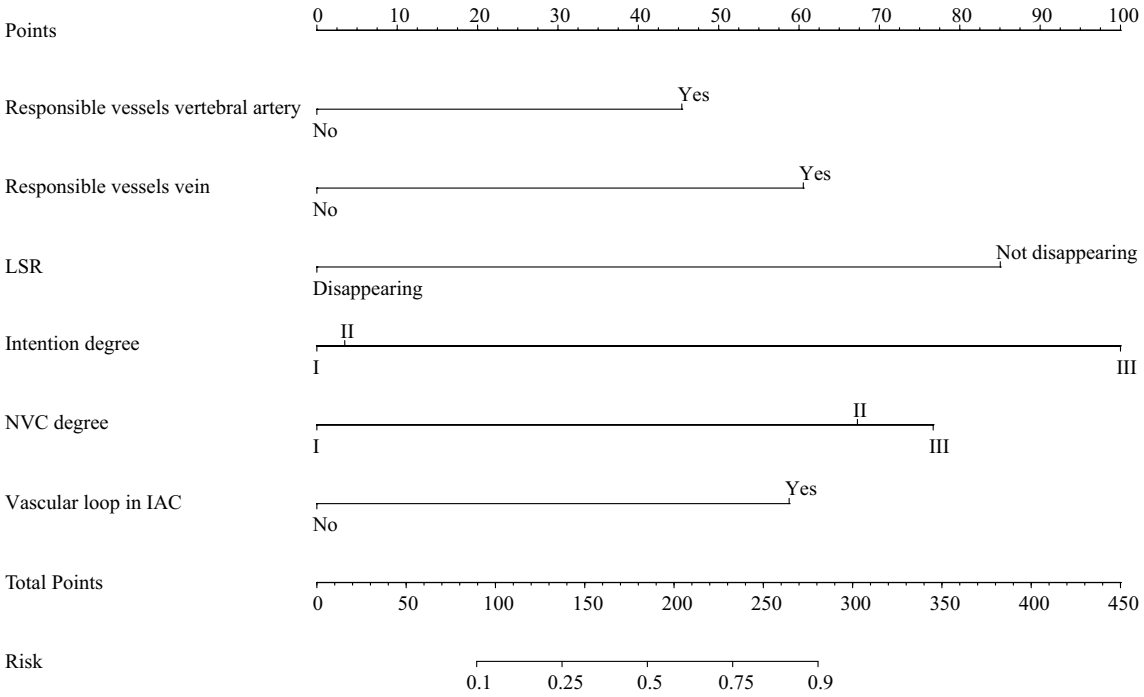

Fig. 1 Nomogram for predicting the probability of delayed cure after microvascular decompression in hemifacial spasm patients

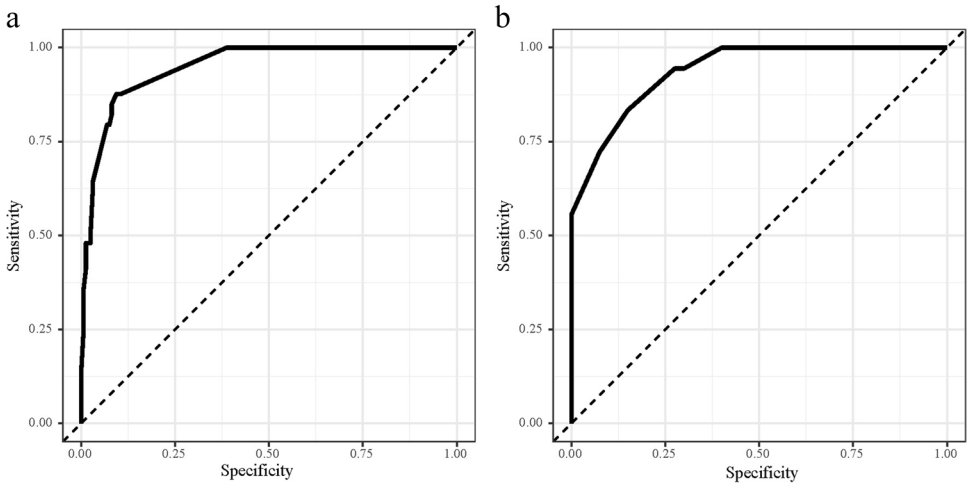

Fig. 2 Receiver operating characteristic validation of the nomogram. **a** From the training set and **b** from the validation set.

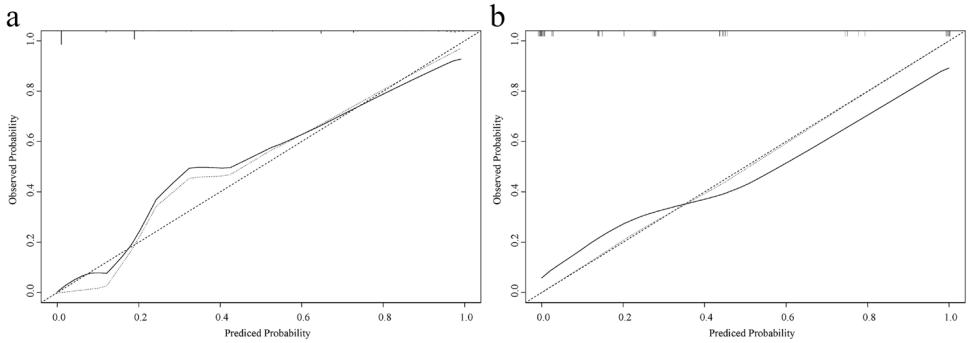

Fig. 3 Calibration curves for the nomogram in training cohort (**a**) and validation cohort (**b**)

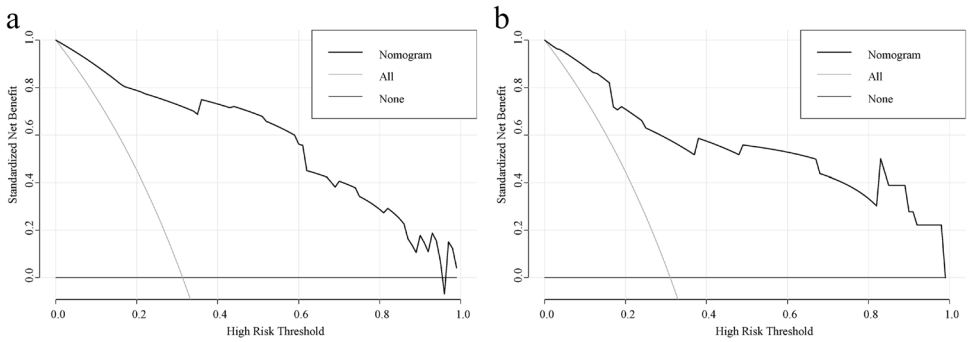

Fig. 4 Decision curve analysis for the nomogram. **a** From the training set and **b** from the validation set

for clinical practice. To the best of our knowledge, our nomogram was the first based on the clinical characteristics of HFS patients to predict the postoperative delayed cure MVD model. Risk factors are included in a column chart with others similar to the results of the prediction model. Due to the vertebral artery has characteristic of large diameter and low plasticity, making mobile and facial nerve decompression is difficult. Some studies have found that facial nerve indentation in HFS with vertebral artery compression is more significant than that in HFS without vertebral artery compression, and the incidence of postoperative complications is higher [16]. It has also been shown that the compression force of the offending vessel compressing the facial nerve is significantly associated with the severity of symptoms and the duration of delayed cure [17]. This is similar to the results of our study. The pressure of the vertebral artery is higher than that of other common culprit vessels, such as the posterior inferior cerebellar artery and anterior inferior cerebellar artery, which may be one of the reasons for the higher incidence of delayed cure of HFS with vertebral artery compression. Studies have reported that the vein in patients with HFS compression, pure venous compression is rare, more often compression vein with artery compression [18]. Venous compression has also been shown to be associated with worse outcomes in trigeminal neuralgia and hemifacial spasm, even with complete decompression [19]. This is by the results of our case-cohort study. Venous compression will cause difficulties with MVD, and the facial nerve will likely be injured when dealing with these veins. HFS patients with venous compression are more likely to have delayed cures after surgery. LSR is the HFS physical examination treatment process of abnormal performance, which can pass over the nerve-muscle EMG monitoring to record LSR, identify the responsible blood vessels, and determine whether intraoperative facial nerve decompression is a fully effective index [11]. Some studies have shown that intraoperative regression of LSR after MVD is an important predictor of a good outcome of MVD in HFS [7, 20]. The results of our study showed that intraoperative regression of LSR was an independent factor predicting delayed cure after MVD.

Some studies have reported that delayed cure is associated with the degree of facial nerve injury. The longer the course of the symptoms, the greater the degree of demyelination of the facial nerve fibers, the more severe the symptoms, and the longer the recovery time of the nerve fibers after decompression [21]. In our study, we evaluated indention grade and compression severity, two variables that reflect the degree of NVC. Compression severity was used to assess the degree of NVC based on preoperative MRI, and indention grade was used to assess the degree of NVC based on intraoperative observation of the color of facial nerve compression. Both were associated with delayed cure in univariate and multivariate analyses. This further confirms that the more severe the facial nerve compression and the more severe the injury, the higher the incidence of delayed cure after MVD. Delayed cure is related to the degree of injury caused by compression of the pathogenic vessels in the facial nerve. In addition, studies have shown that patients with a long duration of disease are more likely to have delayed cure compared to those with a short duration of disease [22].

However, in our study, disease duration was statistically significant in univariate analysis, but was not independently associated with delayed cure in multivariate analysis. However, not all patients with long disease duration and severe symptoms show delayed cure, which may be related to individual differences in facial nerve self-healing in each patient. It is generally believed that HFS is caused by compression of the facial nerve in the REZ area, but a growing number of studies have found that distal compression of the facial nerve may also cause HFS [23]. Some studies have found that contact between the vascular loop in the internal auditory canal and the facial nerve is associated with hemifacial spasms [24]. It has also been found that proximal neurovascular compression of the facial nerve in the REZ is associated with early healing compared to distal neurovascular compression [25]. In our study, we used preoperative MRI to determine whether there was a vascular loop compressing the facial nerve in the internal auditory canal on the affected side. The results of the study showed that patients with a vascular ring compressing the facial nerve in the internal auditory canal were more likely to have delayed cure. Studies have reported botulinum toxin injection treatment in patients with HFS alleviated the postoperative period significantly

longer than in patients who were not botulinum toxin injection treatment, and a preoperative number of botulinum toxin injection treatment, and the significant relationship between postoperative HFS alleviation cycle [13]. We did not find an association between preoperative botulinum toxin injection treatment and delayed cure after surgery in our study, and the different results may be because preoperative botulinum toxin injection treatment was not performed in the same center in our cohort and there may be technical differences. It has also been found that a positive response to preoperative anticonvulsant therapy is significantly associated with a delayed cure of HFS after MVD [5]. Few patients in our study cohort were receiving anticonvulsant therapy, so we did not include this variable. Studies have found that patients with HFS MVD have early cure and prognosis related to preoperative facial paralysis and late postoperative facial paralysis [26–28]. Preoperative facial paralysis was not found to be associated with the healing pattern after MVD in HFS patients in our study. Since our study aimed to construct a nomogram based on preoperative and intraoperative relevant factors, we did not include delayed cure after surgery as a relevant factor.

This study has several notable strengths. Our study shows that the nomogram has good discrimination and predictability, and these factors can be easily obtained before and during surgery. This study has some limitations, firstly, it is a single-center retrospective study and only applies to HFS patients undergoing MVD. Secondly, although we used an internal validation cohort to validate the performance of the nomogram, the sample size was still relatively small. To verify the applicability of the prediction model, a large sample of external data will be necessary in the future.

Conclusion

To sum up, the column chart model by compression vertebral artery, venous pressure, LSR, intension degree, compression severity, and loop characteristics of MVD in patients with HFS postoperative delayed healing has good predictive ability. Delayed healing can cause distress and psychological stress to HFS patients. The nomogram model is simple and feasible and can be used in clinical practice to identify early high-risk patients with delayed cures. However, more validation studies in multiple external, independent patient populations are needed in the future.

Abbreviations *MVD*: Microvascular decompression; *HFS*: Hemifacial spasm; *ROC*: Receiver operating characteristic; *DCA*: Decision curve analysis; *AUC*: Area under the curve; *NVC*: Neurovascular compression; *MRI*: Magnetic resonance imaging; *MRA*: Magnetic resonance angiography; *REZ*: Root entry zone; *LSR*: Lateral spreading reaction; *BAEP*: Brainstem auditory-evoked potential; *EMG*: Electromyogram; *SD*: Standard deviation

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Authors' contributions Conceptualization: Keyu Chen, Nanxiang Xiong. Data curation: Keyu Chen, Lei Shen, Jingyi Yang, Jingwei Zhao, Ji Wu, Yuankun Cai, Songshan Chai, Dongyuan Xu. Funding acquisition: Project administration: Pucha Jiang, Nanxiang Xiong. Supervision: Nanxiang Xiong. Validation: Nanxiang Xiong. Writing—original draft: Keyu Chen. Writing—review and editing: Keyu Chen.

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

Declarations

Ethics approval and consent to participate The study protocol was also approved by the Research and Ethics Committee of Zhongnan Hospital of Wuhan University. All procedures involving human participants in this study were performed by institutional and national Research Council ethical standards.

Consent for publication The authors declare no competing interests.

Competing interests The authors declare no competing interests.

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