Stroke

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Cerebellar Stroke Score and Grading Scale for the Prediction of Mortality and Outcomes in Ischemic Cerebellar Stroke

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BACKGROUND: Several individual predictors for outcomes in patients with cerebellar stroke (CS) have been previously identified. There is, however, no established clinical score for CS. Therefore, the aim of this study was to develop simple and accurate grading scales for patients with CS in an effort to better estimate mortality and outcomes.

METHODS: This multicentric retrospective study included 531 patients with ischemic CS presenting to 5 different academic neurosurgical and neurological departments throughout Germany between 2008 and 2021. Logistic regression analysis was performed to determine independent predictors related to 30-day mortality and unfavorable outcome (modified Rankin Scale score of 4–6). By weighing each parameter via calculation of regression coefficients, an ischemic CS-score and CS-grading scale (CS-GS) were developed and internally validated.

RESULTS: Independent predictors for 30-day mortality were aged ≥70 years (odds ratio, 5.2), Glasgow Coma Scale score 3 to 4 at admission (odds ratio, 2.6), stroke volume ≥25 cm³ (odds ratio, 2.7), and involvement of the brain stem (odds ratio, 3.9). When integrating each parameter into the CS-score, age≥70 years and brain stem stroke were assigned 2 points, Glasgow Coma Scale score 3 to 4, and stroke volume≥25 cm³ 1 point resulting in a score ranging from 0 to 6. CS-score of 0, 1, 2, 3, 4, 5, and 6 points resulted in 30-day mortality of 1%, 6%, 6%, 17%, 21%, 55%, and 67%, respectively. Independent predictors for 30-day unfavorable outcomes consisted of all components of the CS-score with an additional variable focused on comorbidities (CS-GS). Except for Glasgow Coma Scale score 3 to 4 at admission, which was assigned 3 points, all other parameters were assigned 1 point resulting in an overall score ranging from 0 to 7. CS-GS of 0, 1, 2, 3, 4, 5, 6, and 7 points resulted in 30-day unfavorable outcome of 1%, 17%, 33%, 40%, 50%, 80%, 77%, and 100%, respectively. Both 30-day mortality and unfavorable outcomes increased with increasing CS-score and CS-GS (*P*<0.001).

CONCLUSIONS: The CS-score and CS-GS are simple and accurate grading scales for the prediction of 30-day mortality and unfavorable outcome in patients with CS. While the score systems proposed here may not directly impact treatment decisions, it may help discuss mortality and outcome with patients and caregivers.

GRAPHIC ABSTRACT: A graphic abstract is available for this article.

Key Words: cerebellum ■ ischemic stroke ■ mortality ■ outcome ■ predictor ■ prognosis

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Nonstandard Abbreviations and Acronyms

CS cerebellar strokeGCS Glasgow Coma Scale

GS grading scale
OR odds ratio

erebellar stroke (CS) accounts for ≈3% of all ischemic strokes with an annual CS incidence of 20 000/ years within the United States.¹ The rate of functional independence after CS ranges from 35% to 82% while the mortality ranges from 9% to 39% depending on the amount of territory involved/extent of the infarct.²-5 Previously, there have been predictors identified that portend unfavorable outcome(s) in CS including increased age, involvement of the brainstem, bilateral infarcts, and poor clinical status on admission.¹-5

Of note, recent American Heart and Stroke Association guidelines suggest that guidelines suggest surgical management of patients with CS that display evidence of mass effect may be good candidates for surgical intervention. Despite this, there is no current scale for cerebellar infarcts with regard to risk stratification to enable appropriate communication with patients/care givers that would ultimately influence clinical decisions and treatment strategies.⁶

Accordingly, we sought to develop a CS score for the prediction of CS-associated mortality and a CS-grading scale (CS-GS) for the prediction of clinical outcomes after primary cerebellar stroke.

METHODS

Standard Protocol Approvals and Patient Consent

This study was approved by each of the participating institutional review boards; the overall study was managed/approved by the University Medical Center Rostock institutional review board (Registration No. A2020-0266). Due to its retrospective and blinded nature, formal consent of patients included within the final analyses was not deemed necessary. The content of the article was followed by the STROBE guideline (The Strengthening the Reporting of Observational Studiies in Epidemiology; Supplemental Material). The data/datasets used and analyzed in the current study are available upon request (S.-Y.W).

Study Design

Patients with an ischemic CS presenting to 5 academic neurosurgical and neurological departments (University Medical Center Rostock, University Hospital Jena, University Hospital Heidelberg, University Hospital Göttingen, University Hospital Frankfurt) throughout Germany between the years 2008 and 2021 were eligible to be enrolled within this study (Figure S1). Patients were excluded if there were no radiological

data (magnetic resonance image scan), electronical, or insufficient clinical data. All variables potentially affecting the outcome model were extracted from the patient databases and are as follows: age, sex, pertinent medical history (ie, hypertension, diabetes, coronary artery disease, atrial fibrillation, chronic obstructive pulmonary disease/asthma, remote history of stroke, anticoagulation or antiplatelet use), admission status as reflected by Glasgow Coma Scale (GCS), and the primary treatment modality (surgical versus medical). Similar to previous studies, the GCS score was calculated based on data obtained at the time of transfer to either the intensive care/ stroke unit, hospital ward, or operating room.7 Surgery was defined as either suboccipital craniotomy and necrosectomy or suboccipital craniectomy; the placement of an external ventricular drain in isolation was defined as an invasive modality used in the course of an otherwise conservative treatment plan. The volume of the cerebellar infarct was calculated using the initial head magnetic resonance image scan via a region-of-interest measurement using Brainlab software (Brainlab AG, München, Germany); these scans were also used to assess brainstem involvement. Primary end points assessed were 30-day mortality and 30-day functional outcome as defined by the modified Rankin Scale; unfavorable outcomes were defined as a modified Rankin Scale score between 4 and 6. The information was derived by either electronic database from the study investigators or in case of insufficient data, patients were contacted by telephone or seen in an outpatient clinic.

In the univariate analyses, mean±SD values of continuous parameters (ie, age, GCS, infarct volume) were analyzed using the Mann-Whitney *U* test. Dichotomous parameters were analyzed using χ^2 statistics. Both age and volume of stroke when examined as continuous parameters were proven to be potential predictors; the subsequent development of outcome models in which these critical variables were dichotomized into categorical variables with a cutoff value of 70 years and 25 cm³, respectively were therefore pursued. Further, GCS was divided into 3 different categories: GCS scores 3 to 4, 5 to 12, and 13 to 15, which is indeed consistent with previous literature.⁷ All potential predictors from the univariate analysis were then included in a multivariate logistic regression analysis with stepwise backward elimination of variables that were deemed to not be contributing to the resultant model(s). Odds ratios (OR) with CIs (CI 95%) were calculated for independent predictors.

To develop a CS score addressing the risk of 30-day mortality and a CS-grading scale (CS-GS) score centered on the risk of 30-day unfavorable outcome(s), regression coefficients were scaled and rounded to obtain the weighting of independent predictors. The predictive accuracy of the resultant scoring system was analyzed via the generation of receiver operating characteristic curves. Further, internal validation was performed using the same set of patients via a contingency table analysis.

All tests were 2-sided and $P \le 0.05$ were deemed to be statistically significant in the univariate and multivariate analysis. All statistical analyses were performed using IBM SPSS Statistics (version 24, IBM Corp, Armonk, NY).

RESULTS

Of the 569 patients identified with an ischemic CS, 531 patients (93.3%) were ultimately deemed eligible for

inclusion in the final analyses while 38 patients (6.7%) were ultimately excluded due to missing clinical/radiological data and loss of follow-up (Figure S1). Regarding demographics, the mean age of the cohort was 68.7 ± 14.6 years (range, 18-95) and 301 patients (56.7%) were women. At the time of admission, the mean GCS was 13.1 ± 3.5 (range, 3-15), and mean stroke volume was 17.1 ± 23.2 cm³ (range, 0.1-88.8). In 127 cases (23.9%), patients underwent surgical treatment while 404 patients (76.1%) underwent conservative treatment (ie, medical management). The 30-day mortality within the cohort was 9.8% (52 patients), and 28.1% (149 patients) had 30-day unfavorable outcome(s). Details are reported in Table 1.

In the univariate analysis, age≥70 years, atrial fibrillation, GCS at admission, stroke volume ≥25 cm³, brain stem involvement, thrombolysis, endovascular thrombectomy, and treatment modality were significant potential predictors for 30-day mortality. Among those aged ≥70 years (OR, 5.2 [95% CI, 2.4–11.1]), GCS score 3 to 4 at admission (OR, 2.6 [95% CI, 1.1–5.8]),

stroke volume \geq 25 cm³ (OR, 2.7 [95% CI, 1.3–5.4]), and brain stem involvement (OR, 3.9 [95% CI, 2.1–7.5]) were independent predictors in the multivariate analysis (Table 2).

Regarding the functional outcome, the same variables were significant potential predictors within the univariate analysis (ie, age≥70 years, atrial fibrillation, GCS at admission, stroke volume ≥25 cm³, brain stem involvement, thrombolysis, endovascular thrombectomy, and treatment modality). Further, hypertension, diabetes, and atrial fibrillation were identified as comorbidities potentially affecting outcomes. In the subsequent multivariate analysis, age ≥70 years (OR, 2.3 [95% CI, 1.4-3.6]), GCS score 3 to 4 and GCS score 5 to 12 at admission (OR, 7.6 [95% CI, 3.1-16.4] and OR, 2.5 [95% CI, 1.2-5.2], respectively), stroke volume ≥25 cm³ (OR, 2.3 [95% CI, 1.4-3.7]), brain stem involvement (OR, 2.6 [95% CI, 1.6-4.3]), atrial fibrillation (OR, 1.7 [95% Cl, 1.0-2.7]), and diabetes (OR, 2.3 [95% Cl, 1.4-3.7]) were independent predictors for 30-day unfavorable outcome(s) (Table 2).

Table 1. Univariate Analysis of Predictors of 30-Day Mortality and Unfavorable Outcome in Ischemic Cerebellar Stroke Cohort

Variables	n (%)	30-d mortality, n (%)	P value	30-d unfavorable outcome, n (%)	P value
Age ≥70 y	287 (54.0)	41 (14.3)	<0.001	98 (34.1)	<0.001
Sex					
Male	230 (43.3)	27 (11.7)	0.466	70 (30.4)	0.287
Female	301 (56.7)	25 (8.3)		79 (26.2)	
Medical history					
Hypertension	420 (79.1)	43 (10.2)	0.502	127 (30.2)	0.030
Diabetes	133 (25.0)	12 (9.0)	0.730	53 (39.8)	<0.001
Coronary disease	94 (17.7)	13 (13.8)	0.147	26 (27.7)	0.924
Atrial fibrillation	129 (24.3)	20 (15.5)	0.012	50 (38.8)	0.002
COPD/asthma	54 (10.2)	6 (11.1)	0.731	16 (29.6)	0.787
Remote stroke	100 (18.8)	9 (9)	0.767	31 (31)	0.468
Anticoagulation	89 8 (16.8)	9 (10.1)	0.911	26 (29.2)	0.791
Antiplatelet	169 (31.8)	15 (8.9)	0.627	46 (27.2)	0.768
Admission status				1	'
GCS score 13-15	444 (83.6)	32 (7.2)	<0.001	95 (21.4)	<0.001
GCS score 5–12	39 (7.3)	6 (15.4)	0.222	18 (46.2)	0.009
GCS score 3-4	48 (9.1)	14 (29.2)	<0.001	36 (75)	<0.001
Radiological parameter					
Stroke volume ≥25 cm³	150 (28.2)	24 (16)	0.003	67 (43.7)	<0.001
Stroke associated with brain stem	126 (23.7)	25 (19.8)	<0.001	55 (43.7)	<0.001
Medical and interventional treatment			'		'
Thrombolysis	81 (15.3)	13 (16.0)	0.040	31 (38.3)	0.026
Endovascular thrombectomy	42 (7.9)	9 (21.4)	0.008	23 (54.8)	<0.001
Further treatment					
Surgery	127 (23.9)	20 (15.7)	0.010	59 (46.5)	<0.001
Conservative	404 (76.1)	32 (7.9)		90 (22.3)	

COPD indicates chronic obstructive pulmonary disease; and GCS, Glasgow Coma Scale.

Table 2. Multivariate Analysis of Independent Predictors of 30-Day Mortality and Unfavorable Outcome(s)

Variables	P value	OR (CI 95%)			
30-d mortality					
Age ≥70 y	<0.001	5.2 (2.4-11.1)			
Atrial fibrillation	0.130	1.7 (0.9-3.2)			
GCS score 13-15	0.519	0.7 (0.3-2.0)			
GCS score 3-4	0.026	2.6 (1.1-5.8)			
Stroke volume ≥25 cm³	0.006	2.7 (1.3-5.4)			
Brain stem stroke	<0.001	3.9 (2.1-7.5)			
Thrombolysis	0.722	1.2 (0.5-2.7)			
Endovascular thrombectomy	0.361	1.5 (0.6-3.9)			
Surgery	0.680	0.8 (0.3-2.5)			
30-d unfavorable outcome					
Age ≥70 y	<0.001	2.3 (1.4-3.6)			
Atrial fibrillation	0.038	1.7 (1.0-2.7)			
Diabetes	<0.001	2.3 (1.4-3.7)			
Hypertension	0.740	0.9 (0.5-1.7)			
GCS score 5-12	0.013	2.5 (1.2-5.2)			
GCS score 3-4	<0.001	7.6 (3.1–16.4)			
Stroke volume ≥25 cm³	0.001	2.3 (1.4-3.7)			
Brain stem stroke	<0.001	2.6 (1.6-4.3)			
Thrombolysis	0.950	1.0 (0.5-1.7)			
Endovascular thrombectomy	0.216	1.6 (0.7-3.6)			
Surgery	0.937	1.0 (0.5-2.3)			

GCS indicates Glasgow Coma Scale; and OR, odds ratio.

CS- and CS-GS Scores

The CS score was developed by integrating the 4 parameters shown to be independent predictors of 30-day mortality within the logistic regression analysis. Each parameter was assigned a weighing that was directly proportional to its influence outcome; as a result, age≥70 years and stroke involving the brainstem were the parameters most strongly associated with the mortality and therefore each variable was assigned 2 points. Other parameters (ie, GCS score 3-4 and stroke volume ≥25 cm³) were each assigned 1 point. The total CS score was calculated via the sum of each point and ranged from 0 to 6 points (Table 3). Prediction accuracy was estimated by calculating the area under the receiver operating characteristic (area under curve 0.767; Figure S2). In addition, internal validation was performed on the CS scores predictive power; a good correlation between CS score and 30 days mortality was demonstrated (P<0.001; Nagelkerke r²=0.199). Patients with a 0-point CS score had 1%, 1 point: 6%, 2 points: 6%, 3 points: 17%, 4 points: 21%, 5 points: 55%, and 6 points: 67% risk for 30-day mortality, respectively (Figure 1).

Similarly, the CS-GS score was developed by including the 5 independent predictors of 30-day unfavorable outcome(s). Of note, GCS score 3 to 4 was the parameter

Table 3. CS Score for 30-Day Mortality

Components	CS score points			
Age				
≥70 y	2			
<70 y	0			
GCS				
≤4	1			
>4	0			
Stroke volume, cm³				
≥25	1			
<25	0			
Stroke in brainstem				
Yes	2			
No	0			
Total CS score	0–6			

CS indicates cerebellar stroke; and GCS, Glasgow Coma Scale.

most strongly associated with the outcome resulting in an OR of 7.5, which proved to be ≈3× higher than the other parameters; therefore, GCS score 3 to 4 was assigned to 3 points. All other parameters (age≥70 years, comorbidities—atrial fibrillation or diabetes, GCS score 5 to 12, stroke involving the brainstem) were each assigned 1 point. The total CS-GS score was obtained by calculating the sum of points and ranged from 0 to 7 (Table 4). The receiver operating characteristic analysis revealed an area under curve value of 0.758 (Figure S3) confirming prediction accuracy. As per the above, the internal validation of the CS-GS score demonstrated good predictive power with a good correlation between CS-GS and 30 days of unfavorable outcome(s) (P<0.001; Nagelkerke

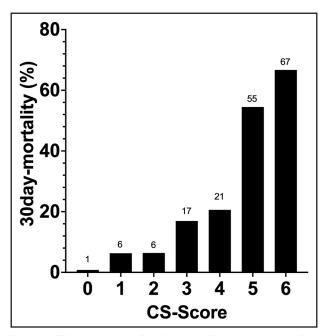


Figure 1. Thirty-day mortality risk according to the cerebellar stroke (CS) score.

Table 4. CS-GS for 30-Day Unfavorable Outcome(s)

Components	CS-GS score points
Age	00 000 0000 points
≥70 y	1
<70 y	0
GCS	
3–4	3
5–12	1
13–15	0
Comorbidities (diabetes or atrial fibrillation)	,
Yes	1
No	0
Stroke volume, cm³	·
≥25	1
<25	0
Stroke in brainstem	
Yes	1
No	0
Total CS-GS score	0-7

CS-GS indicates cerebellar stroke-grading scale; and GCS, Glasgow Coma Scale.

 r^2 =0.247). Patients with a CS-GS score of 0 point had a 1%, 1 point: 17%, 2 points: 33%, 3 points: 40%, 4 points: 50%, 5 points: 80, 6 points: 77%, and 7 points: 100% risk unfavorable outcome(s) at 30 days follow-up, respectively (Figure 2).

DISCUSSION

This multicenter retrospective study focused on CS represents the largest sample size to date and is the first study to have developed a risk stratification system for such infarcts. The CS and CS-GS were developed for prediction of 30-day mortality and functional outcome. As demonstrated above internal validation revealed good predictive power and good correlation suggesting that such grading scales may ultimately be employed as tools in the management of stroke patients (eg, shared decision models, etc).

The CS-score consists of 4 components: age, GCS at admission, volume of stroke, and brainstem involvement. Unsurprisingly, one of the critical parameters identified and incorporated into both scales was the patient's age. While age itself is not an illness, correlations between age and an increase in white matter lesions have been informed in a litany of previous studies. Grips et al¹⁰ evaluated the relevance of age-related white matter changes in patients with primary CS and found that the presence and degree of subcortical white matter damage was a strong predictor of early recovery. The underlying pathophysiological mechanism is assumed to be a synergistic lesion with diffuse destruction of the neuronal network. By

the loss of cerebellar tissue, the disrupted neuronal preexisting network is not compensated by the cerebellum, on which, presumably, the recovery of each patient depends on.¹⁰ In general, it is well known that an isolated cerebellar infarction has good functional outcome(s) as compared with infarctions in other vascular territories.^{10–12} However, it seems that the potential of favorable outcome relies on the presence or absence of multilocular neurological lesions in case of the focal cerebellar lesion (eq. infarct).

Besides advanced age, cerebrovascular risk factors like hypertension, diabetes, smoking, and cholesterol level are associated with poor cranial perfusion and decreased vessel density, which is reflected by more white matter hyperintensities.¹³ Given this, such chronic processes may be one of the possible explanations for the delayed recovery of such patients with CS. Such a contention is supported by our clinical data with the significant influence of comorbidities (eg, diabetes, atrial fibrillation) having been identified and ultimately factored into our novel scales.

It is not surprising that the volume of stroke and the involvement of the brain stem have a negative association with the outcome.14 Recently, Wang et al15 reported the rate of infarct-edema growth as a promising biomarker for the necessity of surgical intervention. In our study, we performed the volumetric analysis with the initial magnetic resonance image scan; however, no serial image analysis was performed. The reason for this was that the comparison between magnetic resonance image scan and follow-up computed tomography scans is difficult due to the limitation of edema/infarct differentiation. Further, the purpose of this study was to estimate the prognosis of patients in the initial stadium of the disease. Previous studies reported the baseline CS volume between 22 and 33 mL and involving 40% of one cerebellar hemisphere or multiple vascular territories as

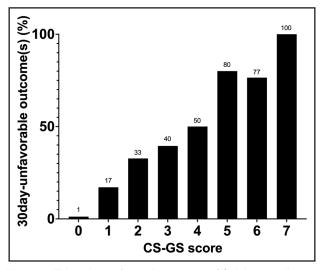


Figure 2. Thirty-day unfavorable outcome(s) risk according to the cerebellar stroke-grading scale (CS-GS) score.

risk factors for the development of malignant cerebellar edema. 15,16 Accordingly, our cutoff value of CS with ≈ 25 mL seems to be an adequate value to integrate it as a threshold for the risk stratification given predefined range is between 22 and 33 mL.

One of the limitations of our study is the absence of the National Institutes of Health Stroke Scale at admission/discharge since the majority of the patients presented to neurosurgical departments were clinically evaluated by GCS. That being said it is prudent to note that the National Institutes of Health Stroke Scale and modified National Institutes of Health Stroke Scale may underperform a clinical evaluation tool for patients with perturbations in the posterior circulation (eg, CS); accordingly, several studies have been using the GCS as for outcome analyses in such clinical scenarios. 4,17-20 Similar to the widely used intracerebral hemorrhage score introduced by Hemphill et al,7 the goal of this study was to establish a scoring system that is as simple as possible, and we therefore integrated the easy-to-acquire GCS as a proxy for clinical status. In line with such thinking, it is important to note that GCS remains critical for decisionmaking with regard to surgical decompressive treatment.²¹ Another limitation of our study might be the time frame of the clinical course and the follow-up analysis. There was inconsistent data concerning the time from stroke onset to the initial image and treatment wherefore we could not include those parameters in our analysis. For the follow-up analysis, we analyzed the outcome at 30 days follow-up similar to the prognosis score by the group of Hemphill et al7; however, the long-term outcome might be an interesting topic for future studies.

Common to all prognostic scales, the CS- and CS-GS score may have an application in providing a rough framework to evaluate the potential efficacy of treatments, provide information to patients/family, and avoid futile invasive treatment in cases that are expected to have poor outcomes/survivability. As always, scoring systems provide a general approximation of potential outcomes that must be weighed against the complexities of the presenting individual and not lead to self-fulfilling prophecy. Additionally and directly relevant to the work presented, an external validation of the CS- and CS-GS score on an independent data set is warranted in the future to reevaluate the predictive power and accuracy before integration into clinical practice.

While the CS- and CS-GS scores developed herein may not directly impact the standardized method of patient assessment, it may help to discuss and allow improved clinical care and shared decision-making with patients and care givers.

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Supplemental Material

Figures S1-S3 STROBE checklist

REFERENCES

- Edlow JA, Newman-Toker DE, Savitz SI. Diagnosis and initial management of cerebellar infarction. *Lancet Neurol.* 2008;7:951-964. doi: 10.1016/S1474-4422(08)70216-3
- Tohgi H, Takahashi S, Chiba K, Hirata Y. Cerebellar infarction Clinical and neuroimaging analysis in 293 patients The Tohoku Cerebellar Infarction Study Group. Stroke. 1993;24:1697–1701. doi: 10.1161/01.str.24.11.1697
- Calic Z, Cappelen-Smith C, Cuganesan R, Anderson CS, Welgampola M, Cordato DJ. Frequency, aetiology, and outcome of small cerebellar infarction. *Cerebrovasc Dis Extra*. 2017;7:173–180. doi: 10.1159/000481459
- Jüttler E, Schweickert S, Ringleb PA, Huttner HB, Köhrmann M, Aschoff A. Long-term outcome after surgical treatment for space-occupying cerebellar infarction: experience in 56 patients. Stroke. 2009;40:3060–3066. doi: 10.1161/STROKEAHA.109.550913
- Lindeskog D, Lilja-Cyron A, Kelsen J, Juhler M. Long-term functional outcome after decompressive suboccipital craniectomy for space-occupying cerebellar infarction. *Clin Neurol Neurosurg.* 2019;176:47–52. doi: 10.1016/j.clineuro.2018.11.023
- Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, Biller J, Brown M, Demaerschalk BM, Hoh B, et al. Guidelines for the early management of patients with acute ischemic stroke: 2019

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- update to the 2018 guidelines for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke. Stroke. 2019;50:e344-e418. doi: 10.1161/STR.00000000000000211
- 7. Hemphill JC, Bonovich DC, Besmertis L, Manley GT, Johnston SC. The ICH score: a simple, reliable grading scale for intracerebral hemorrhage. Stroke. 2001;32:891-897. doi: 10.1161/01.str.32.4.891
- 8. Taylor WD, MacFall JR, Provenzale JM, Payne ME, McQuoid DR, Steffens DC, Krishnan KRR. Serial MR imaging of volumes of hyperintense white matter lesions in elderly patients: correlation with vascular risk factors. AJR Am J Roentgenol. 2003;181:571-576. doi: 10.2214/ajr.181.2.1810571
- Taylor WD, Bae JN, MacFall JR, Payne ME, Provenzale JM, Steffens DC, Krishnan KRR. Widespread effects of hyperintense lesions on cerebral white matter structure. AJR Am J Roentgenol. 2007;188:1695-1704. doi: 10.2214/AJR.06.1163
- 10. Grips E, Ο, Bäzner Η, Sedlaczek . DaffertshoferM, HennericiM. Supratentorial age-related white matter changes predict outcome in cerebellar stroke. Stroke. 2005;36:1988-1993. doi: 10.1161/01.STR.0000177869.02361.dc
- 11. Kelly PJ, Stein J, Shafqat S, Eskey C, Doherty D, Chang Y, Kurina A, Furie KL. Functional recovery after rehabilitation for cerebellar stroke. Stroke. 2001;32:530-534. doi: 10.1161/01.str.32.2.530
- 12. Jauss M, Krieger D, Hornig C, Schramm J, Busse O. Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. J Neurol. 1999;246:257-264. doi: 10.1007/s004150050344
- 13. Williamson W, Lewandowski AJ, Forkert ND, Griffanti L, Okell TW, Betts J, Boardman H, Siepmann T, McKean D, Huckstep O, et al. Association of cardiovascular risk factors with MRI indices of cerebrovascular structure and function and white matter hyperintensities in young adults. JAMA. 2018;320:665-673. doi: 10.1001/jama.2018.11498

- 14. Kumral E, Bayulkem G, Akyol A, Yunten N, Sirin H, Sagduyu A. Mesencephalic and associated posterior circulation infarcts. Stroke. 2002;33:2224-2231. doi: 10.1161/01.str.0000027438.93029.87
- 15. Wang Y, Binkley MM, Qiao M, Pardon A, Keyrouz S, Dhar R, Ford AL. Rate of infarct-edema growth on CT predicts need for surgical intervention and clinical outcome in patients with cerebellar infarction. Neurocrit Care. 2022;36:1011-1021. doi: 10.1007/s12028-021-01414-x
- 16. Fabritius MP, Thierfelder KM, Meinel FG, Othman AE, Dorn F, Sabel BO, Scheffler P, Ertl-Wagner B, Sommer WH, Kunz WG. Early imaging prediction of malignant cerebellar edema development in acute ischemic stroke. Stroke, 2017:48:2597-2600, doi: 10.1161/STROKEAHA.117.018237
- 17. Nickel A, Cheng B, Pinnschmidt H, Arpa E, Ganos C, Gerloff C, Thomalla G. Clinical outcome of isolated cerebellar stroke-a prospective observational study. Front Neurol. 2018;9:580. doi: 10.3389/fneur.2018.00580
- 18. Martin-Schild S, Albright KC, Tanksley J, Pandav V, Jones EB, Grotta JC, Savitz SI. Zero on the NIHSS does not equal the absence of stroke. Ann Emerg Med. 2011;57:42-45. doi: 10.1016/j.annemergmed.2010.06.564
- 19. Villalobos-Díaz R, Ortiz-Llamas LA, Rodríguez-Hernández LA, Flores-VázquezJG,Calva-GonzálezM,Sangrador-DeitosMV,Mondragón-SotoMG, Uribe-Pacheco R, Villanueva Castro E, Barrera-Tello MA. Characteristics and long-term outcome of cerebellar strokes in a single health care facility in Mexico. Cureus. 2022;14:e28993. doi: 10.7759/cureus.28993
- 20. Briley DP, Coull BM, Goodnight SH. Neurological disease associated with antiphospholipid antibodies. Ann Neurol. 1989;25:221-227. doi: 10.1002/ana.410250303
- 21. Wijdicks EFM, Sheth KN, Carter BS, Greer DM, Kasner SE, Kimberly WT, Schwab S, Smith EE, Tamargo RJ, Wintermark M; American Heart Association Stroke Council. Recommendations for the management of cerebral and cerebellar infarction with swelling: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2014;45:1222-1238. doi: 10.1161/01.str.0000441965.15164.d6