Predictors of neurological deterioration during admission for patients with cerebellar strokes

Thanyalak Amornpojnimman (athanyal@medicine.psu.ac.th)
Prince of Songkla University https://orcid.org/0000-0002-8673-7040
Pornchai Sathirapanya
Prince of Songkla University
Utcharee Intusoma
Prince of Songkla University
Nuttha Sanghan
Prince of Songkla University
Anukoon Kaewborisutsakul
Prince of Songkla University

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Abstract

Background: Despite less common, cerebellar stroke frequently results in unfavorable outcomes, especially after deterioration. Therefore, this study was aimed to identify the significant predictors of neurological deterioration during admission (NDDA) in ischemic and hemorrhagic cerebellar strokes.

Materials and methods: We retrospectively reviewed all medical records of the patients diagnosed with ischemic and hemorrhagic cerebellar strokes during 2002-2018 in Songklanagarind hospital. Comparison of patients' demographic data, initial clinical presentations, neuroradiological results, timing and signs of NDDA, and outcomes between cerebellar strokes were descriptively analyzed. Logistic regression model was applied for determining the significant predictors of NDDA from initial clinical presentations.

Results: 74 of 100 patients were eligible. They comprised of 42 (57%) cerebellar ischemia (CI) and 32 (43%) cerebellar hemorrhage (CH). Elevated diastolic blood pressure (DBP) and the neuro-radiological evidences suggesting increased posterior cranial fossa pressure were significantly prevalent in neurological deterioration patients. NDDA was found in 31 patients (42%) without significant difference between CI and CH. 42 (56.8%) patients had poor neurological outcomes. The independent predictors for NDDA were DBP B 120 mmHg (adjusted odds ratio [adj. OR] 15.39, 95% CI 1.58-149.59; p = 0.004), time from onset to arrival (adj. OR 0.98, 95% CI 0.97-1.00; p = 0.044), and hemispheric cerebellar signs (adj. OR 0.22, 95% CI 0.06-0.75; p = 0.012).

Conclusions: CH was not an independent predictor of NDDA in overall cerebellar strokes. Only high DBP predicted NDDA, whereas time to arrival and hemispheric signs showed protective impact.

Background

Although cerebellar stroke is less common (1–3% of all strokes), it frequently results in high morbidity and mortality rates (25–100%).[1, 2] Because of the limited space of posterior cranial fossa where cerebellum locates and the closed contact of cerebellum, particularly cerebellar vermis, with brain stem, ischemic cerebellar edema or cerebellar hematoma poses high risk of tonsillar herniation as well as direct brain stem compression. These are key mechanisms of secondary neurological deterioration during admission (NDDA) from cerebellar strokes. Generally, NDDA occurs a few days after the onset of cerebellar infarction (CI), while it commonly takes a few hours after cerebellar hemorrhage (CH).[3] Therefore, close observation for early detection of neurological deterioration, and timely initiation of neurosurgical interventions are necessary for favorable outcomes after a cerebellar stroke.

Previous series reported about natural course and outcomes of cerebellar strokes. The predictors of poor outcome in cerebellar strokes from several studies included high blood pressure, high blood glucose level, hydrocephalus and obliterated fourth ventricle from radiologic findings. There have been limited studies reporting the prognosticators of NDDA that required neurosurgical interventions in initially non-surgically indicated patients.[1–10] Thence, this study was aimed to determine the prognostic factors of surgically-

indicated NDDA for both CI and CH from the patient's demographic and presentation characteristics. Our findings might help identify patients at risk who need intensive monitoring or early intervention.

Materials And Methods

Objectives

Our primary objective was to identify predictors of secondary neurological deterioration after admission. The secondary objective was to know neurological outcomes of patients admitted with cerebellar strokes at discharge and at 90 days after onset.

Study Designs And Setting

This retrospective cohort study was conducted in Songklanagarind Hospital, an 800-bed tertiary and medical teaching university hospital in southern Thailand. The medical records between 1 January 2002 and 31 December 2018 were reviewed.

Eligible Criteria

Medical records of admitted patients aged ≥ 18 years diagnosed with cerebellar strokes during the study period were reviewed. We excluded the patients who had Glasgow coma score (GCS) ≤ 3 or underwent emergency neurosurgical interventions at the time of presentations.

Operational Definition Of Main Variables

Cerebellar strokes

The diagnosis of cerebellar strokes must be confirmed by neuroimaging either brain CT or brain MRI interpreted by a certified radiologist.

Secondary neurological deterioration during admission (NDDA)

Secondary neurological deterioration during admission was defined by the emergence of any new pyramidal tract signs, new brainstem signs, new cerebellar signs or lowering of GCS ≥ 1 point from the baseline assessment.

Neurological outcome

The neurological outcomes evaluated by modified Rankin score (mRs) on the day of hospital discharge as well as 90 days after stroke onset. Stroke outcomes evaluated by mRs were classified into 2 groups: favorable outcome (mRs 0–2) and unfavorable outcome (mRs 3–6).

Data Collection

We collected data from the electronic medical record including the patients' demographic data, presenting symptoms and signs, results of routine blood analysis, brain imaging reports, date of NDDA occurrence, neurosurgical interventions performed for treating NDDA.

Statistical analysis

Comparison of patient demographic data, initial clinical characteristics, the presence of NDDA and outcome between CI and CH were descriptively analyzed. The discrete data were analyzed by Chi-square test. The continuous data were analyzed by independent t-test and Mann Whitney U test. The significant variables with $p < 0.2$ in univariate analysis were entered to multivariate logistic regression model. The variables were considered as independent predictors if they meet the statistical significance with $p < 0.05$ in multivariate logistic regression analysis.

Results

Demographic and presenting characteristics

One hundred cerebellar stroke patients were initially included. After exclusion of twenty-six patients (11 cases with GCS $\leq$ 3, and 17 cases with surgical interventions performed at the presentation), 74 initially non-surgically indicated patients admitted for supportive treatments and clinical observation were eligible for final analysis. (Fig. 1)

There were fifty-two (70%) male and 22 female patients. Among them, 42 patients were CI and 32 patients were CH. No significant difference in median (IQR) age was found between the two groups of patients. (Table 1) The overall median (IQR) time from stroke onset to hospital arrival was 8 (2.12, 27.94) hours with significantly shorter time in neurological deterioration patients. Thirteen patients (5 CH and 8 CI) were initially misdiagnosed as peripheral vertigo on their presentations to the emergency department. On evaluation at the presentation, we found predominantly high diastolic blood pressure (DBP) in neurological deterioration group. Significant difference in the presence of cerebellar signs (midline and hemispheric structure signs) was shown between the two groups. (Table 1) Baseline CT scans and MRI brain were done in 70 (95%) and 4 (5%) cases, respectively. Repeated CT scan (12 cases) or MRI brain (17 cases) was done to confirm CI in initially indefinite CT scan brain reports. The neuro-radiological evidences suggesting increased posterior cranial fossa pressure included obstructive hydrocephalus in 7 (9.5%), distortion of the fourth ventricle in 6 (8%), and tonsillar herniation in 3 (4%) cases. Obstructive hydrocephalus and distortion of the fourth ventricle were significantly shown on brain images of neurological deterioration cases. (Table 1)

Table 1
Comparison of patient presentation characteristics between neurological deterioration and no neurological deterioration group during admission in mixed-type cerebellar strokes

Variables	Neurological deterioration n (%), N = 31	No neurological deterioration n (%), N = 43	*p*-value
Male	24 (77.4)	28 (65.1)	0.376
Age, (years) median (IQR)	67 (60.5,74.5)	69 (60,79)	0.288
Time from onset to hospital arrival (hours) median (IQR)	3 (1,9)	15 (5.5,48)	0.005#
Misdiagnosis as peripheral vestibular disorders at presentation	8 (25.8)	5 (11.6)	0.203
Risk factors	19 (61.3)	25 (58.1)	0.974
Hypertension	11 (35.5)	12 (27.9)	0.660
Smoking	6 (19.4)	11 (25.6)	0.728
Diabetes mellitus	8 (25.8)	11 (25.6)	1
Dyslipidemia	3 (9.7)	5 (11.6)	1
Anticoagulant used	6 (19.4)	3 (7)	0.153
History of CAD			
Clinical presentation	20 (64.5)	31 (72.1)	0.660
Nausea or vomiting	17 (54.8)	33 (76.7)	0.083
Vertigo or dizziness	17 (54.8)	19 (44.2)	0.504
Headache	10 (32.3)	18 (41.9)	0.550
Gait ataxia	9 (29)	10 (23.3)	0.771
Dysarthria	8 (25.8)	7 (16.3)	0.476
Limb ataxia	4 (12.9)	1 (2.3)	0.154
Alteration of consciousness	0 (0)	2 (4.7)	0.506
Diplopia	1 (3.2)	1 (2.3)	1
Tinnitus			

Variables	Neurological deterioration n (%), N = 31	No neurological deterioration n (%), N = 43	*p*-value
Initial clinical signs;	17 (54.8)	13 (30.2)	0.059
SBP $\geq$ 180 mmHg	10 (32.3)	1 (2.3)	< 0.001$^{\delta}$
DBP $\geq$ 120 mmHg	27 (87.1)	42 (97.7)	0.069
Glasgow coma scale (GCS)	1 (3.2)	1 (2.3)	0.031$^{\varphi}$
GCS $\geq$ 13	3 (9.7)	0 (0)	0.025$^{\varphi}$
GCS 9–12	13 (41.9)	30 (69.8)	
GCS 4–8	16 (51.6)	34 (79.1)	
Cerebellar signs			
Midline structure signs			
Hemispheric signs			
Initial laboratory finding	21 (67.7)	19 (44.2)	0.077
WBC $\geq$10,000 cells/mm^3	2 (6.9)	3 (11.1)	0.664
INR $\geq$ 1.5	12 (38.7)	9 (20.9)	0.158
Blood sugar $\geq$ 140 mg/dL			

Variables	Neurological deterioration	No neurological deterioration	*p*-value
	n (%), N = 31	n (%), N = 43	
Type of cerebellar stroke	14 (45.2)	28 (65.1)	0.141
Ischemic stroke	7 (22.6)	0 (0)	0.001$^{\delta}$
Initial neuro-imaging results showing increased posterior fossa pressure	6 (19.4)	0 (0)	0.004$^{\delta}$
Obstructive hydrocephalus	3 (9.7)	0 (0)	0.069
4th ventricular distortion	9 (29)	15 (34.9)	0.780
Tonsillar herniation	20 (64.5)	0 (0)	< 0.001$^{\varphi}$
Vermis involvement	8 (25.8)	0 (0)	< 0.001$^{\delta}$
Deterioration during admission (n = 31)	6 (19.4)	0 (0)	< 0.001$^{\varphi}$
Signs	6 (19.4)	0 (0)	< 0.001$^{\varphi}$
GCS dropped ≥ 1	16 (51.6)	29 (67.4)	
New cerebellar sign	5 (31.2)	14 (32.6)	
New pyramidal sign	6 (37.5)	32 (74.4)	
New brainstem sign	5 (31.2)	11 (25.6)	
Treatment	3 (9.7)		
Surgical treatment (n = 16)	28 (90.3)		
Type of surgery	7 (22.6)		
Ventriculostomy	24 (77.4)		
Suboccipital craniectomy			
Both			
Outcome			
mRS at discharge			
0–2 (non-dependency)			
3–6 (dependency)			
mRS at 90 days after onset			
0–2 (non-dependency)			
3–6 (dependency)			

Neurological Deterioration During Admission And Predictors

NDDA developed in 31 (42%) patients, in whom decreasing of GCS ≥ 1 point was the most common alarming neurological sign, and was common in CH patients also. The median (IQR) time from onset to NDDA was significantly shorter in CH (22 (5, 48) vs. 57 (27.5, 106); p = 0.031). (Table 2) At the time of NDDA, neurosurgical interventions were done in 16 (51.6%) cases: 6 (37.5%) with decompression craniectomy, 5 (31.25%) with ventriculostomy, and combination of both in 5 (31.25%) patients. The rest of the patients who were neurosurgically indicated did not consent for the interventions.

Table 2
Comparison of patient presentation characteristics between hemorrhagic and ischemic cerebellar stroke

Variables	Hemorrhage n (%), N = 32	Ischemia n (%), N = 42	*p*-value
Male	25 (78.1)	27 (64.3)	0.301
Age, (years) median (IQR)	67.5 (60.2,79.3)	67 (60.25,75.8)	0.658
Time from onset to hospital arrival (hours) median (IQR)	5.5 (1,13.2)	13.5 (3,48)	0.035#
Misdiagnosis as peripheral vestibular disorders at presentation	5 (15.6)	8 (19)	0.940
Risk factors	17 (53.1)	27 (64.3)	0.466
Hypertension	7 (21.9)	16 (38.1)	0.215
Smoking	4 (12.5)	13 (31)	0.112
Diabetes mellitus	6 (18.8)	13 (31)	0.357
Dyslipidemia	5 (15.6)	3 (7.1)	0.280
Anticoagulant used	4 (12.5)	5 (11.9)	1
History of CAD			
Clinical presentation	22 (68.8)	29 (69)	1
Nausea or vomiting	19 (59.4)	31 (73.8)	0.288
Vertigo or dizziness	23 (71.9)	13 (31)	0.001φ
Headache	8 (25)	20 (47.6)	0.081
Gait ataxia	9 (28.1)	10 (23.8)	0.879
Dysarthria	3 (9.4)	12 (28.6)	0.081
Limb ataxia	3 (9.4)	2 (4.8)	0.647
Alteration of consciousness	1 (3.1)	1 (2.4)	1
Diplopia	1 (3.1)	1 (2.4)	1
Tinnitus			

Variables	Hemorrhage n (%), N = 32	Ischemia n (%), N = 42	*p*-value
Initial clinical signs;	17 (53.1)	13 (31)	0.092
SBP $\geq$ 180 mmHg	8 (25)	3 (7.1)	0.048$^{\delta}$
DBP $\geq$ 120 mmHg	29 (90.6)	40 (95.2)	0.039$^{\delta}$
Glasgow coma scale (GCS)	0 (0)	2 (4.8)	0.319
GCS $\geq$ 13	3 (9.4)	0 (0)	0.660
GCS 9–12	16 (50)	27 (64.3)	
GCS 4–8	23 (71.9)	27 (64.3)	
Cerebellar signs			
Midline structure signs			
Hemispheric signs			
Initial laboratory finding	22 (68.8)	18 (42.9)	0.048$^{\varphi}$
WBC $\geq$10,000 cells/mm^3	4 (13.3)	1 (3.8)	0.358
INR $\geq$ 1.5	12 (37.5)	9 (21.4)	0.208
Blood sugar $\geq$ 140 mg/dL			

Variables	Hemorrhage n (%), N = 32	Ischemia n (%), N = 42	*p*-value
Initial neuro-imaging results showing increased posterior fossa pressure	6 (18.8)	1 (2.4)	0.038φ
Obstructive hydrocephalus	6 (18.8)	0 (0)	0.005φ
4th ventricular distortion	2 (6.2)	1 (2.4)	0.575
Tonsillar herniation	9 (28.1)	15 (35.7)	0.660
Vermis involvement	17 (53.1)	14 (33.3)	0.141
Deterioration during admission	22 (5,48)	57 (27.5,106)	0.031#
Neurological deterioration	13 (40.6)	7 (16.7)	0.042φ
Time from onset to deterioration (hours): median (IQR)	2 (6.2)	6 (14.3)	0.453
Signs	4 (12.5)	2 (4.8)	0.393
GCS dropped ≥ 1	4 (12.5)	2 (4.8)	0.393
New cerebellar sign	8 (25)	8 (19)	0.740
New pyramidal sign	4 (50)	1 (12.5)	0.283
New brainstem sign	3 (37.5)	3 (37.5)	0.040φ
Treatment	1 (12.5)	4 (50)	0.266
Surgical treatment (n = 16)	9 (28)	23 (55)	
Type of surgery	23 (72)	19 (45)	
Ventriculostomy	14 (43.8)	25 (59.5)	
Suboccipital craniectomy	18 (56.2)	17 (40.5)	
Both			
Outcome			
mRS at discharge			
0–2 (non-dependency)			
3–6 (dependency)			
mRS at 90 days after onset			
0–2 (non-dependency)			
3–6 (dependency)			

Subsequently, the significant independent predictor for NDDA was DBP $\geq$ 120 mmHg (adjusted OR [adj. OR] 15.39, 95% CI 1.58-149.59; p = 0.004). Notably, time from onset to hospital arrival (adj. OR 0.98, 95%

CI 0.97-1; p = 0.044), and hemispheric cerebellar signs (adj. OR 0.22, 95% CI 0.06–0.75; p = 0.012) were significant protective factors of NDDA. (Table 3)

Table 3
Factors associated with neurological deterioration during admission in mixed-type cerebellar strokes

Variables	Univariate analysis		Multivariate analysis	
	Crude OR (95% CI)	p-value*	Adjusted OR (95% CI)	p-value
Hemorrhagic stroke (ref = ischemic)	2.27 (0.88–5.83)	0.141	1.43 (0.4–5.17)	0.586
Previous history of CAD (ref = none)	3.2 (0.73–13.96)	0.153	4.04 (0.76–21.51)	0.139
Time from onset to hospital arrival (hours)	0.98 (0.96-1)	0.005	0.98 (0.97-1)	0.044**
Main presenting symptoms before arrival				
Dizziness/vertigo (ref = none)	2.72 (1-7.39)	0.083	0.99 (0.25–3.92)	0.986
Alteration of consciousness (ref = none)		0.154		
Clinical profiles at hospital arrival				
SBP≥180 mmHg (ref<180 mmHg)	2.8 (1.07–7.33)	0.059	0.68 (0.18–2.56)	0.564
DBP≥120 mmHg (ref<120 mmHg)	20 (2.4-166.77)	< 0.001	15.39 (1.58-149.59)	0.004**
GCS < 13 (ref≥13)		0.154		
Midline structure signs (ref = none)	0.28 (0.1–0.78)	0.031	0.22 (0.06–0.75)	0.012**
Hemispheric cerebellar signs (ref = none)		0.025		
WBC≥10,000 cells/mm^3 (ref<10,000)	2.65 (1.01–6.96)	0.077	3.06 (0.94–10.02)	0.148
BS≥140 mg/dL (ref<140 mg/dL)	2.39 (0.85–6.69)	0.158	1.54 (0.39–5.97)	0.538
Neuro-imaging finding		0.001		
Hydrocephalus (ref = none)		0.069		
Tonsillar herniation (ref = none)		0.004		
4th ventricular distortion (ref = none)				

Outcomes Of Cerebellar Strokes

The mean (± SD) hospital stay was 17 ± 29.8 days. The overall outcomes were 42 (56.8%) patients had unfavorable neurological outcomes, in which 8 (10.8%) patients were deceased on the discharge. Thirty-five (47.3%) patients had unfavorable outcomes at 90 days after onset. CI had better outcome evaluated by mRS at the discharge date ($p = 0.04$), but no difference in the outcome at 90 days after onset. (Table 2)

In comparison of the number of cases with unfavorable outcomes between patients with and without NDDA, there were 28 (90%) and 24 (77%) cases in NDDA group comparing to 14 (33%) and 11 (26%) cases in no NDDA patients, at the discharge date and 90 days after onset, respectively. (Fig. 1) We also found that NDDA cerebellar stroke patients had significant proportion of unfavorable outcomes evaluated by mRs at hospital discharge ($p < 0.001$) and at 90 days after onset ($p < 0.001$). (Fig. 2)

Discussion

Our study found significantly higher proportion of headache as a presenting symptom and elevated DBP in CH patients. Moreover, white blood cells (WBC) $\geq$ 10,000 cells/mm^3, obstructive hydrocephalus, and distortion of the fourth ventricle on initial brain scans were significantly more prevalent in CH as well. (Table 2) We propose that an abrupt and rapid increment of posterior cranial fossa pressure from cerebellar hematoma plus its later expansion possibly causes the anatomical distortions. Furthermore, acute physiological reaction to the acutely elevated intracranial pressure (ICP) probably results in leukocytosis. A study by Furlan showed that leukocytosis on admission was associated with poor outcome too.[11]

Notably, we found no significant difference in NDDA between the two subtypes of cerebellar strokes. In addition, CH was not an independent risk factor of NDDA by logistic regression analysis in the current study. (Table 3) However, the time from onset to NDDA was significantly shorter in CH. (Table 2) This is explainable by more rapid rising of ICP in CH than in CI. Unlike a few previous studies,[6, 8] which included wider range of severe cerebellar stroke cases, the significant differences in characteristics between CH and CI were more obvious than ours. As we aimed to determine the predictors of NDDA in the patients initially without indication for neurosurgical interventions, we excluded all the cases with neurological deterioration at their first presentations. And, the limited number of cases enrolled in our study was likely to have fewer clinical parameters with statistically significant differences reported.

The shorter time from stroke onset to hospital arrival and hemispheric cerebellar signs at presentation were protective factors on multivariate analysis. (Table 3) We considered that the presence of cerebellar hemispheric signs was well realized by most physicians of having hemispheric cerebellar disorder, facilitating immediate neuro-imaging study and therapy. In contrast, in cases of cerebellar vermis stroke mostly are under evaluated, or missed as a peripheral vestibular disorder causing delayed diagnosis and also proper management. As found in our study, the presence of hemispheric cerebellar signs was a

significant protective factor for NDDA in our study. (Table 3) Positive hemispheric cerebellar signs corresponding with the presence of hemispheric cerebellar lesions seen on the imaging studies were 51/74 (70%) cases in our study. 34/43 of them (79%) acquired favorable outcome eventually. A study by Erik, et al. supported our finding as they reported that cerebellar vermis hemorrhage was associated with higher rates of neurological deterioration.[6] Direct compression of the hematoma against brainstem was attributed.

Our study revealed that DBP $\geq$ 120 mmHg was an independent predictor of NDDA (adj. OR 15.39, 95% CI 1.58-149.59; p = 0.004). (Table 3) Elevation of blood pressure has been considered as a response to elevation of ICP at the stroke onset, however, it probably leads to neurological deterioration because of increased risk of massive cerebral edema and hematoma expansion as well.[12–17]

Hyperglycemia (Blood sugar $\geq$ 140 mg/dL) was not an independent predictor of NDDA in the current study. (Table 3) Actually, we found that the median (IQR) Blood sugar (BS) level in our cases (117 (100, 133.7) mg/dL) was lower than some previous studies (> 150 mg/dL).[7, 8, 18] Therefore, lower BS levels would contribute to better cerebellar stroke outcomes in our study. To our knowledge, hyperglycemia worsens the overall stroke outcomes, because high blood sugar level has been known to exert adverse effects on the structures of cerebral vascular endothelial cells, and to induce acute oxidative stress along with vascular endothelial inflammation.[19, 20]

Koh[1] concluded that hydrocephalus, brain stem deformity and basal cistern compression were associated with NDDA in cerebellar infarction. St. Louis[6] also reported that patients with a cerebellar vermis hematoma and acute hydrocephalus were at high risk for NDDA. Furthermore, Ho[8] reported that obliteration of basal cistern on the initial CT brain scans was associated with NDDA in cerebellar hemorrhages. Based on our available results, we found no neuro-imaging abnormality as a predictor of NDDA by multivariate analysis. (Table 3) Since most of the brain images done in our study were CT scans, demonstration of such mentioned imaging abnormalities in association with NDDA is possibly obscured.

Thirty-one (42%) of all cerebellar stroke patients developed NDDA, and 28 of the 31 (90%) patients acquired unfavorable neurological outcomes at hospital discharge. When compare with the overall cerebellar stroke outcomes, only 37.8% had favorable outcomes at discharge (2.4 folds higher in NDDA cases). Some previous studies reported a slightly higher percentage of unfavorable final outcomes (50%).[1, 6, 8] With the available information and based on our current findings, a worse neurological outcome is undoubtedly higher in NDDA cerebellar stroke patients.

Under limitation of accessibility of MRI brain, particularly under emergency service setting in our center and the similar others, we speculated that some clinical presentation characteristics could be practically useful to predict the occurrence of NDDA among the initially non-surgical-indicated patients. We hope that our findings could facilitate appropriate monitoring and timely starting of necessary neurosurgical interventions aiming at favorable cerebellar stroke outcomes.

The limitations of the current study are retrospective design and single-center study with limited sample size. Further prospective and multi-center studies, which include more study samples with variability of cerebellar stroke severity would be useful in providing an appropriate management decision on initiation of early neurosurgical interventions for cerebellar stroke patients.

Conclusions

Cerebellar hemorrhage was not an independent predictor for NDDA for all cerebellar strokes in this study. Some initial clinical presentations of cerebellar stroke, regardless of types of cerebellar stroke, are potentially applicable to predict NDDA along with favorable short and long-term outcomes.

Abbreviations

NDDA: Neurological deterioration during admission; CI: Cerebellar infarction; CH: Cerebellar hemorrhage; GCS: Glasgow coma score; mRs: Modified Rankin score; IQR: Interquartile range; DBP: Diastolic blood pressure; WBC: White blood cells; ICP: Intracranial pressure; BS: Blood sugar

Declarations

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Author contributions

Amornpojnimman, Sathirapanya and Intusoma designed the study, analyzed data, contributed to the interpretation of data and wrote the manuscript. Sanghan and Kaewborisutsakul contributed to the interpretation of the data and to the writing the manuscript. All authors contributed to the final version of the manuscript.

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Availability of data and materials

The data of this study are available from the corresponding author upon reasonable request.

Ethics approval

The study protocol was reviewed and approved by the Ethics Committee of the Faculty of Medicine, Prince of Songkla University (registration No. 62-307-14-1). All identifiable personal information of the study subjects was completely anonymized. We strictly followed the regulation statements and guidelines documented in the 1964 Declaration of Helsinki, its later amendments, and the current best practice guidelines for performing the research. Patient consents to access their clinical data for this research were obtained through the approval of the aforementioned Ethics Committee, which acted on behalf of all study patients.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Author details

[1] Division of Neurology, Department of Internal Medicine, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla, Thailand. [2]Division of Neurology, Department of Pediatrics, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla, Thailand. [3]Division of Neuroradiology, Department of Radiology, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla, Thailand. [4]Division of Neurological surgery, Department of Surgery, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla, Thailand.

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Figures

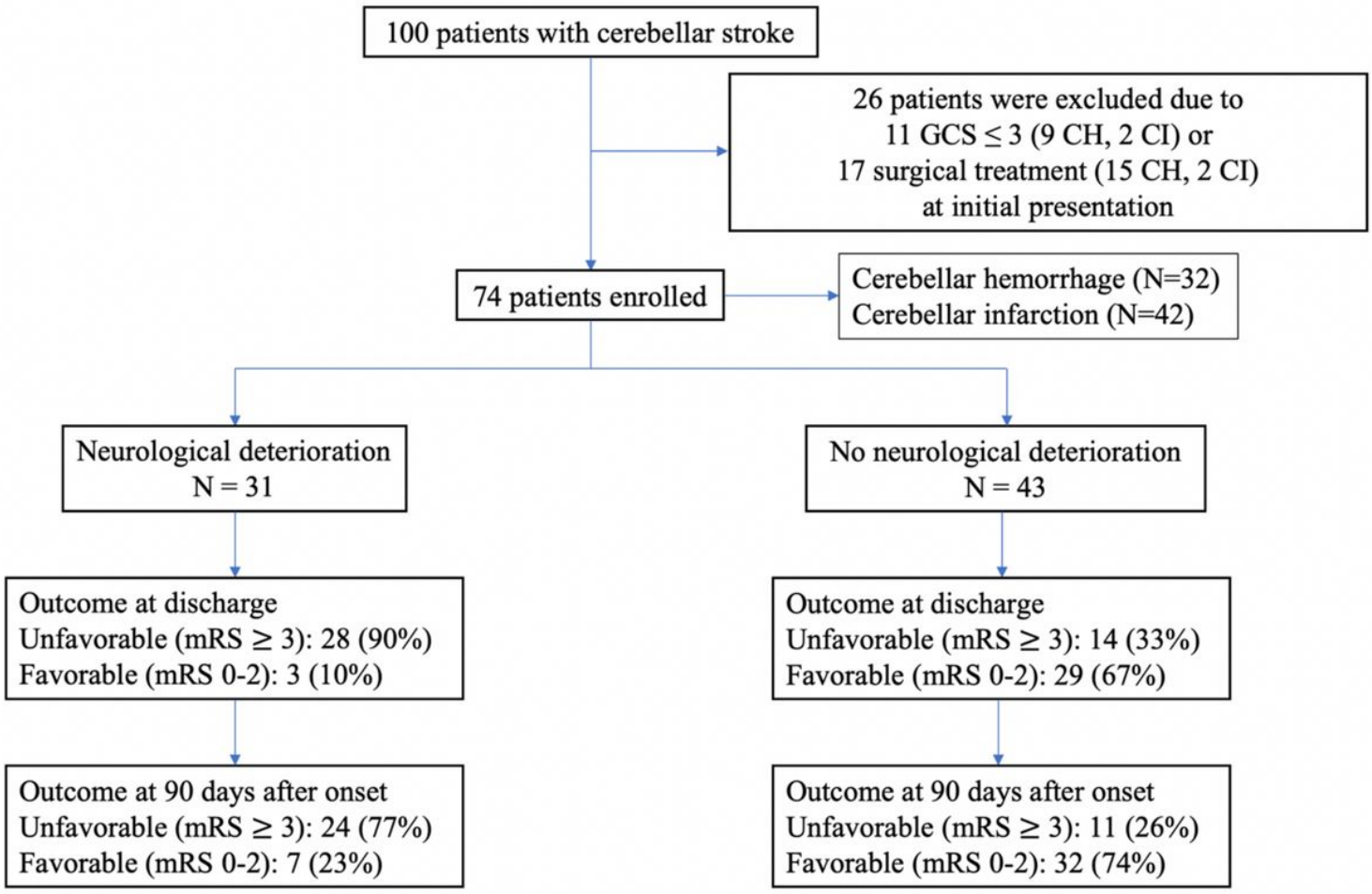

Figure 1

Study flow

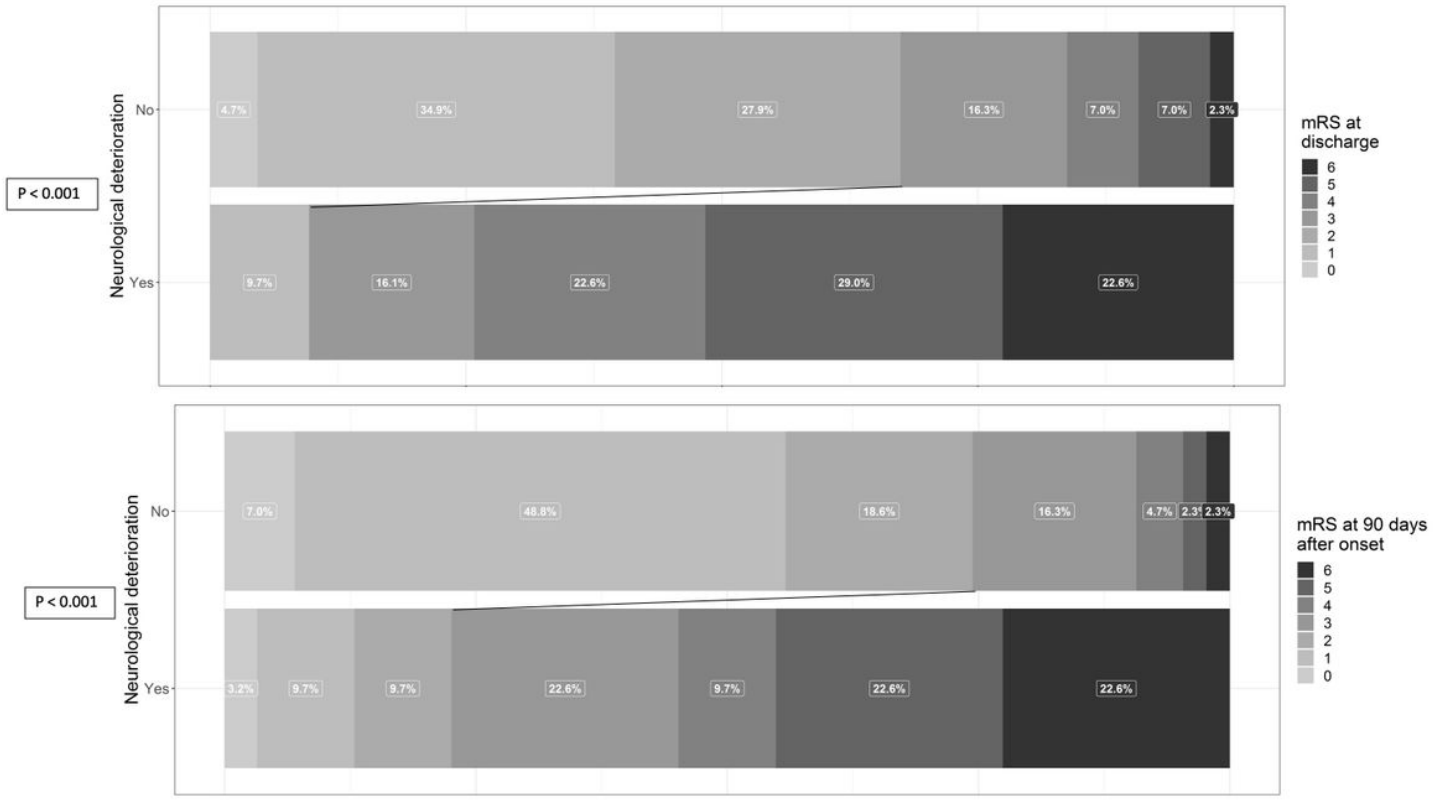

Figure 2

Comparison of modified Rankin scores (A) at discharge date and (B) 90 days after onset between cerebellar stroke patients with and without neurological deterioration