Original Article

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Predictors of Neurological Deterioration during Admission for Patients with Cerebellar Strokes

Thanyalak Amornpojnimman, M.D.[1], Utcharee Intusoma, M.D.[2], Nuttha Sanghan, M.D.[3], Anukoon Kaewborisutsakul, M.D.[4], Pornchai Sathirapanya, M.D.[1]

[1]Division of Neurology, Department of Internal Medicine, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla 90110, Thailand.

[2]Division of Neurology, Department of Pediatrics, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla 90110, Thailand.

[3]Division of Neuroradiology, Department of Radiology, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla 90110, Thailand.

[4]Division of Neurological Surgery, Department of Surgery, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla 90110, Thailand.

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Abstract:

Objective: 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.

Material and Methods: We retrospectively reviewed all medical records of 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: From this, 74 of 100 patients were eligible. They comprised of 42 (57.0%) cerebellar ischemia (CI) and 32 (43.0%) cerebellar hemorrhage (CH). Elevated diastolic blood pressure (DBP) and neuro-radiological evidences suggesting increased posterior cranial fossa pressure were significantly prevalent in neurological deterioration patients. NDDA was

Contact: Pornchai Sathirapanya, M.D.
Division of Neurology, Department of Internal Medicine, Faculty of Medicine, Prince of Songkla University, Hat Yai, Songkhla 90110, Thailand.
E-mail: sporncha@medicine.psu.ac.th

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found in 31 patients (42.0%), without significant difference between CI and CH. 42 (56.8%), patients had poor neurological outcomes. The independent predictors for NDDA were DBP 120 mmHg (adjusted odds ratio [adj. OR] 15.39, 95% CI 1.58–149.59; p–value=0.004), time from onset to arrival (adj. OR 0.98, 95% CI 0.97–1.00; p–value=0.044), and hemispheric cerebellar signs at presentation (adj. OR 0.22, 95% CI 0.06–0.75; p–value=0.012).

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

Keywords: cerebellar strokes, during admission, neurological deterioration, predictors

Introduction

Although, cerebellar stroke is less common (1.0–3.0% of all strokes), it frequently results in high morbidity and mortality rates (25.0–100%).[1,2] Because of the limited space of posterior cranial fossa, where cerebellum locates and the close contact of cerebellum, particularly cerebellar vermis, with the brain stem, ischemic cerebellar edema or cerebellar hematoma poses a 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.

There were previous studies reporting the 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 immediate 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 initial presentation characteristics at the emergency department (ED). The findings of our study will be helpful in identify patients at risk, who need intensive monitoring or early neurosurgical intervention.

Material and Methods

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

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

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

Operational definition of main variables

The cerebellar strokes were diagnosed by the brain imaging studies, either computed tomography (CT) or magnetic resonance imaging (MRI), and verified by a certified neuroradiologist.

Midline cerebellar signs were defined by the presence of one, or more truncal ataxia, and a wide based gait.

Hemispheric cerebellar signs were defined by the presence of one, or more impaired finger to nose tests, impaired heel to knee to shin test, dysdiadochokinesia and nystagmus.

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.

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

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

Comparison of patient demographic data, initial clinical characteristics at the ED, the presence of NDDA and outcomes 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-value<0.200 in univariate analysis were entered to multivariate logistic regression model. The variables were considered as independent predictors if they meet the statistical significance with a p-value<0.050 in multivariate logistic regression analysis.

Results

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

There were fifty-two (70.0%) 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 presentation to the emergency department. On evaluation at presentation, we found predominantly high diastolic blood pressure (DBP) in the neurological deterioration group. Significant difference in the presence of cerebellar signs at presentation (midline and hemispheric structure signs) was shown between the two groups. (Table 1) No significant difference in white blood cell count was found between the two groups (Table 1); however, white blood cells ≥10,000 cells/mm^3 were more prevalent in CH patients. Baseline CT scans and MRI brain were conducted in 70 (95.0%) and 4 (5.0%) cases, respectively. Repeated CT scanning (12 cases) or MRI brain (17 cases) was perfomred to confirm CI in initially indefinite CT scan brain reports. The neuro-radiological evidences suggesting increased posterior cranial fossa pressure including obstructive hydrocephalus in 7 (9.5%), distortion of the fourth ventricle in 6 (8.0%), and tonsillar herniation in 3 (4.0%) cases. Obstructive hydrocephalus and distortion of the fourth ventricle were significantly shown on the brain images of neurological deterioration cases (Table 1).

The overall median (IQR) time from stroke onset to definite diagnosis was 9 (3.1, 34.2) hours. There were 12/42 (28.6%) patients with acute CI who was diagnosed within 4.5 hours. Notably, no patient received intravenous

thrombolysis, because of misdiagnosis at presentation as peripheral vertigo, or having contraindications for thrombolytic treatment; such as, high bleeding risk with prolongation of international normalized ratio (INR) and high blood pressure.

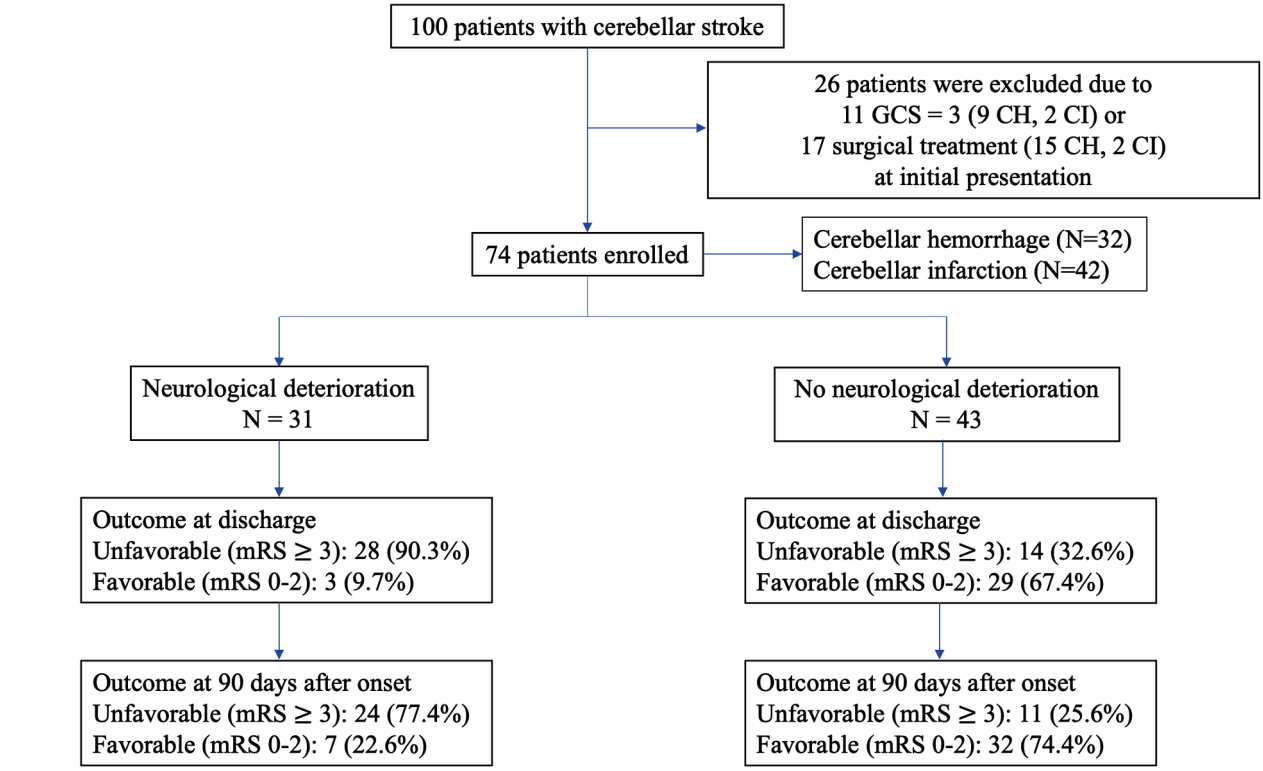

Figure 1 Study flow

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 Number (%), n=31	No neurological deterioration Number (%), n=43	p-value
Male	24 (77.4)	28 (65.1)	0.376
Age, (years) median (IQR)	67 (60.5, 74.5)	69 (60.0, 79.0)	0.288
Time from onset to hospital arrival (hours) median (IQR)	3 (1.0, 9.0)	15 (5.5, 48.0)	0.005[#]
Misdiagnosis as peripheral vestibular disorders at presentation	8 (25.8)	5 (11.6)	0.203
Risk factors			
Hypertension	19 (61.3)	25 (58.1)	0.974
Smoking	11 (35.5)	12 (27.9)	0.660
Diabetes mellitus	6 (19.4)	11 (25.6)	0.728
Dyslipidemia	8 (25.8)	11 (25.6)	1.000
Anticoagulant used	3 (9.7)	5 (11.6)	1.000
History of CAD	6 (19.4)	3 (7.0)	0.153
Clinical presentation			
Nausea or vomiting	20 (64.5)	31 (72.1)	0.660
Vertigo or dizziness	17 (54.8)	33 (76.7)	0.083
Headache	17 (54.8)	19 (44.2)	0.504
Gait ataxia	10 (32.3)	18 (41.9)	0.550
Dysarthria	9 (29.0)	10 (23.3)	0.771
Limb ataxia	8 (25.8)	7 (16.3)	0.476
Alteration of consciousness	4 (12.9)	1 (2.3)	0.154
Diplopia	0 (0.0)	2 (4.7)	0.506
Tinnitus	1 (3.2)	1 (2.3)	1.000
Initial clinical signs;			
SBP ≥180 mmHg	17 (54.8)	13 (30.2)	0.059
DBP ≥120 mmHg	10 (32.3)	1 (2.3)	<0.001[d]
Glasgow coma scale (GCS)			0.069
GCS 13	27 (87.1)	42 (97.7)	
GCS 9-12	1 (3.2)	1 (2.3)	
GCS 4-8	3 (9.7)	0 (0.0)	
Cerebellar signs at presentation			
Midline structure signs	13 (41.9)	30 (69.8)	0.031[f]
Hemispheric signs	16 (51.6)	34 (79.1)	0.025[f]
Initial laboratory finding			
WBC ≥10,000 cells/mm^3	21 (67.7)	19 (44.2)	0.077
INR ≥1.5	2 (6.9)	3 (11.1)	0.664
Blood sugar 140 mg/dL	12 (38.7)	9 (20.9)	0.158
Type of cerebellar stroke			
Ischemic stroke	14 (45.2)	28 (65.1)	0.141
Initial neuro-imaging results showing increased posterior fossa pressure			
Obstructive hydrocephalus	7 (22.6)	0 (0.0)	0.001[d]
4^{th} ventricular distortion	6 (19.4)	0 (0.0)	0.004[d]
Tonsillar herniation	3 (9.7)	0 (0.0)	0.069
Vermis involvement	9 (29.0)	15 (34.9)	0.780

Table 1 (continued)

Variables	Neurological deterioration Number (%), n=31	No neurological deterioration Number (%), n=43	p-value
Deterioration during admission (n=31)			
Signs			
GCS dropped >1	20 (64.5)		
New cerebellar sign	8 (25.8)		
New pyramidal sign	6 (19.4)		
New brainstem sign	6 (19.4)		
Treatment			
Surgical treatment (n=16)	16 (51.6)	0 (0.0)	<0.001[f]
Type of surgery			<0.001[f]
Ventriculostomy	5 (31.2)	0 (0.0)	
Suboccipital craniectomy	6 (37.5)	0 (0.0)	
Both	5 (31.2)	0 (0.0)	
Outcome			
mRS at discharge			<0.001[f]
0-2 (non-dependency)	3 (9.7)	29 (67.4)	
3-6 (dependency)	28 (90.3)	14 (32.6)	
mRS at 90 days after onset			<0.001[f]
0-2 (non-dependency)	7 (22.6)	32 (74.4)	
3-6 (dependency)	24 (77.4)	11 (25.6)	

CAD=coronary artery disease, SBP=systolic blood pressure, DBP=diastolic blood pressure, GCS=Glasgow coma score, Midline structure signs=one or more of truncal ataxia and wide based gait, Hemispheric signs=one or more of impaired finger to nose test, impaired heel to knee to shin test, dysdiadochokinesia and nystagmus WBC=white blood cell, INR=international normalized ratio, mRS=modified Rankin scale, #=Rank sum test p-value<0.050, f=Chi square test p-value<0.050, d=Fisher's exact test p-value<0.050, IQR=interquartile range

NDDA developed in 31 (42.0%) patients, in whom decreasing of GCS >1 point was the most common, alarming neurological sign: this finding was common in CH patients. The median (IQR) time from onset to NDDA was significantly shorter in CH (22 (5, 48) vs. 57 (27.5, 106); p-value=0.031) (Table 2). At the time of NDDA, neurosurgical interventions were performed in 16 (51.6%) cases: 6 (37.5%) either with decompression craniectomy, 5 (31.3%) with ventriculostomy, and a combination of both in 5 (31.3%) patients. The rest of the patients, who were indicated for neurosurgical treatment, did not consent for the interventions.

Subsequently, the significant independent predictor for NDDA was DBP 120 mmHg (adjusted OR [adj. OR] 15.39, 95% CI 1.58-149.59; p-value=0.004). Notably, time from onset to hospital arrival (adj. OR 0.98, 95% CI 0.97-1; p-value=0.044), and hemispheric cerebellar signs at presentation (adj. OR 0.22, 95% CI 0.06-0.75; p-value=0.012) were significant protective factors of NDDA (Table 3).

The mean (±standard deviation, S.D.) hospital stay was 17.0±29.8 days. The overall outcomes were 42 (56.8%) patients had unfavorable neurological outcomes, in which 8 (10.8%) patients were deceased at time of discharge. Thirty-five (47.3%) patients had unfavorable outcomes at 90 days after onset. CI had better outcome evaluated by mRS on the discharge date, but no difference in the outcome at 90 days after onset was found (Table 2).

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

Variables	Hemorrhage Number (%), n=32	Ischemia Number (%), 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 (1.0, 13.2)	13.5 (3.0, 48.0)	0.035[#]
Misdiagnosis as peripheral vestibular disorders at presentation	5 (15.6)	8 (19.0)	0.940
Risk factors			
Hypertension	17 (53.1)	27 (64.3)	0.466
Smoking	7 (21.9)	16 (38.1)	0.215
Diabetes mellitus	4 (12.5)	13 (31.0)	0.112
Dyslipidemia	6 (18.8)	13 (31.0)	0.357
Anticoagulant used	5 (15.6)	3 (7.1)	0.280
History of CAD	4 (12.5)	5 (11.9)	1.000
Clinical presentation			
Nausea or vomiting	22 (68.8)	29 (69.0)	1.000
Vertigo or dizziness	19 (59.4)	31 (73.8)	0.288
Headache	23 (71.9)	13 (31.0)	0.001[f]
Gait ataxia	8 (25.0)	20 (47.6)	0.081
Dysarthria	9 (28.1)	10 (23.8)	0.879
Limb ataxia	3 (9.4)	12 (28.6)	0.081
Alteration of consciousness	3 (9.4)	2 (4.8)	0.647
Diplopia	1 (3.1)	1 (2.4)	1.000
Tinnitus	1 (3.1)	1 (2.4)	1.000
Initial clinical signs;			
SBP ≥180 mmHg	17 (53.1)	13 (31.0)	0.092
DBP ≥120 mmHg	8 (25.0)	3 (7.1)	0.048[d]
Glasgow coma scale (GCS)			0.039[d]
GCS ≥13	29 (90.6)	40 (95.2)	
GCS 9–12	0 (0.0)	2 (4.8)	
GCS 4–8	3 (9.4)	0 (0.0)	
Cerebellar signs at presentation			
Midline structure signs	16 (50.0)	27 (64.3)	0.319
Hemispheric signs	23 (71.9)	27 (64.3)	0.660
Initial laboratory finding			
WBC ≥10,000 cells/mm^3	22 (68.8)	18 (42.9)	0.048[f]
INR ≥1.5	4 (13.3)	1 (3.8)	0.358
Blood sugar ≥140 mg/dL	12 (37.5)	9 (21.4)	0.208
Initial neuro-imaging results showing increased posterior fossa pressure			
Obstructive hydrocephalus	6 (18.8)	1 (2.4)	0.038[f]
4^{th} ventricular distortion	6 (18.8)	0 (0.0)	0.005[f]
Tonsillar herniation	2 (6.2)	1 (2.4)	0.575
Vermis involvement	9 (28.1)	15 (35.7)	0.660
Deterioration during admission			
Neurological deterioration	17 (53.1)	14 (33.3)	0.141
Time from onset to deterioration (hours): median (IQR)	22 (5.0, 48.0)	57 (27.5, 106.0)	0.031[#]
Signs			
GCS dropped >1	13 (40.6)	7 (16.7)	0.042[f]
New cerebellar sign	2 (6.2)	6 (14.3)	0.453
New pyramidal sign	4 (12.5)	2 (4.8)	0.393
New brainstem sign	4 (12.5)	2 (4.8)	0.393

Table 2 (continued)

Variables	Hemorrhage Number (%), n=32	Ischemia Number (%), n=42	p-value
Treatment			
Surgical treatment (n=16)	8 (25.0)	8 (19.0)	0.740
Type of surgery			0.283
Ventriculostomy	4 (50.0)	1 (12.5)	
Suboccipital craniectomy	3 (37.5)	3 (37.5)	
Both	1 (12.5)	4 (50.0)	
Outcome			
mRS at discharge			0.040[f]
0-2 (non-dependency)	9 (28.1)	23 (54.8)	
3-6 (dependency)	23 (71.9)	19 (45.2)	
mRS at 90 days after onset			0.266
0-2 (non-dependency)	14 (43.8)	25 (59.5)	
3-6 (dependency)	18 (56.2)	17 (40.5)	

CAD=coronary artery disease, SBP=systolic blood pressure, DBP=diastolic blood pressure, GCS=Glasgow coma score, Midline structure signs=one or more of truncal ataxia and wide based gait, Hemispheric signs=one or more of impaired finger to nose test, impaired heel to knee to shin test, dysdiadochokinesia and nystagmus WBC=white blood cell, INR=international normalized ratio, mRS=modified Rankin scale, #=Rank sum test p-value<0.050, f=Chi square test p-value<0.050, d=Fisher's exact test p-value<0.050, IQR=interquartile range

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 (0.40-5.17)	0.586
Previous history of CAD (ref=none)	3.20 (0.73-13.96)	0.153	4.04 (0.76-21.51)	0.139
Time from onset to hospital arrival (hours)	0.98 (0.96-1.00)	0.005	0.98 (0.97-1.00)	0.044**
Main presenting symptoms before arrival				
Dizziness/vertigo (ref=none)	2.72 (1.00-7.39)	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)	2.80 (1.07-7.33)	0.059	0.68 (0.18-2.56)	0.564
DBP ≥120 mmHg (ref<120 mmHg)	20.00 (2.40-166.77)	<0.001	15.39 (1.58-149.59)	0.004**
GCS<13 (ref≥13)		0.154		
Midline structure signs (ref=none)		0.031		
Hemispheric cerebellar signs (ref=none)	0.28 (0.10-0.78)	0.025	0.22 (0.06-0.75)	0.012**
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				
Hydrocephalus (ref=none)		0.001		
Tonsillar herniation (ref=none)		0.069		
4^{th} ventricular distortion (ref=none)		0.004		

CAD=coronary artery disease, SBP=systolic blood pressure, DBP=diastolic blood pressure, GCS=Glasgow coma score, Midline structure signs=one or more of truncal ataxia and wide based gait, Hemispheric signs=one or more of impaired finger to nose test, impaired heel to knee to shin test, dysdiadochokinesia and nystagmus, WBC=white blood cell, BS=blood sugar, mmHg=millimetres mercury, ref=reference, OR=odd ratio

*p-value<0.200, **p-value<0.050 adjusted with all variables shown in the table

In comparison of the number of cases with unfavorable outcomes between patients, with and without NDDA, at the discharge date and 90 days after onset, there were 28 (90.0%) and 24 (77.0%) cases in NDDA group compared to 14 (33.0%) and 11 (26.0%) cases in non-NDDA patients, respectively (Figure 1). We also found that NDDA cerebellar stroke patients had a significant proportion of unfavorable outcomes evaluated by mRs at hospital discharge (p-value<0.001) and at 90 days after onset (p-value<0.001) (Figure 2, 3).

Discussion

Our study revealed that DBP 120 mmHg was an independent predictor of NDDA (adj. OR 15.39, 95% CI 1.58-149.59; p-value=0.004) (Table 3). Elevation of blood pressure has been considered as a response to elevation of ICP at stroke onset; however, it probably leads to neurological deterioration because of increased risk of massive cerebral edema and hematoma expansion as well.[11-16]

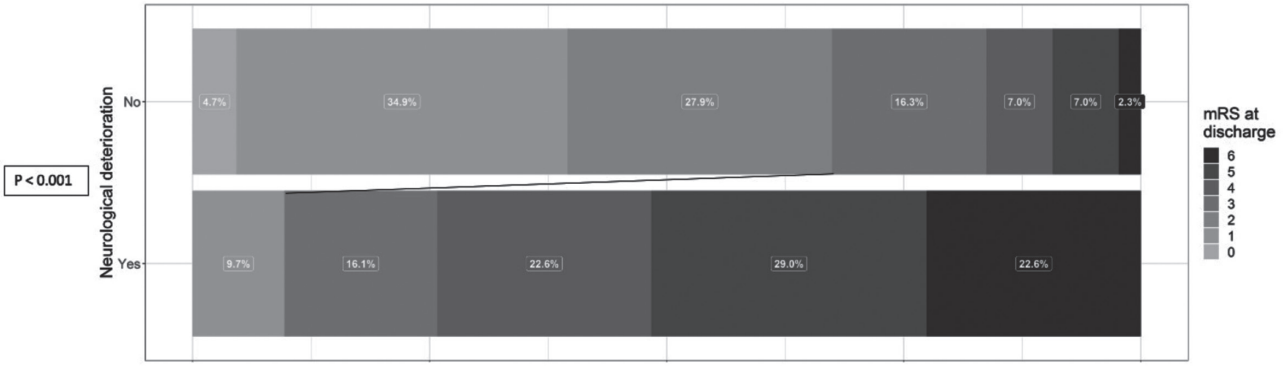

Figure 2 Comparison of modified Rankin scores at discharge date between cerebellar stroke patients with and without neurological deterioration

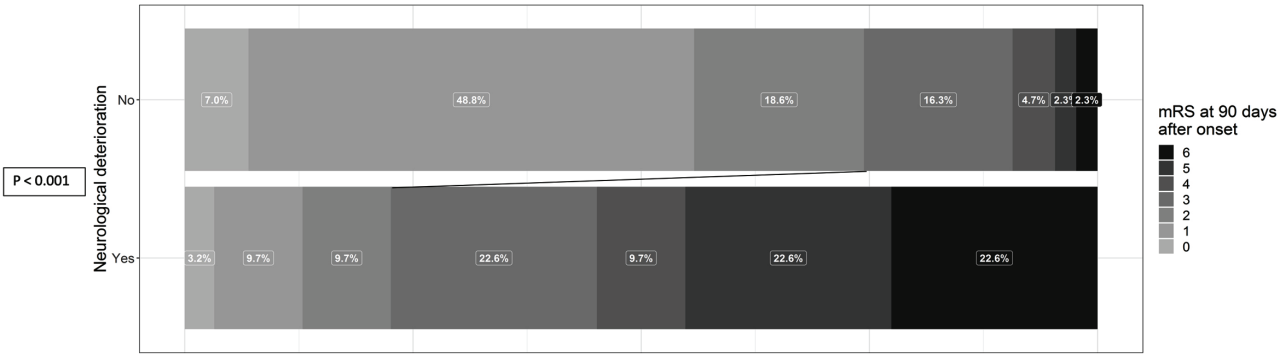

Figure 3 Comparison of modified Rankin scores at 90 days after onset between cerebellar stroke patients with and without neurological deterioration

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 as having a hemispheric cerebellar disorder: facilitating immediate neuro-imaging study and management. In contrast, in cases of cerebellar vermis stroke, they are mostly under evaluated, or missed as peripheral vestibular disorders causing delayed diagnosis and proper management. As found in our study, the presence of hemispheric cerebellar signs was a significant protective factor for NDDA (Table 3). Positive hemispheric cerebellar signs, corresponding with the presence of hemispheric cerebellar lesions seen on the imaging studies, were 50/74 (68.0%) cases in our study. From this, 34/43 of patients with positive hemispheric cerebellar signs (79.0%) acquired a favorable outcome; eventually. A study by Erik et al. supported our findings, in that cerebellar vermis hemorrhage was associated with higher rates of neurological deterioration due to early, direct compression of the hematoma against the brainstem.[6]

Our study found significantly higher proportions of headaches as a presenting symptom, and elevated DBP in CH patients. Moreover, white blood cells ≥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.[17]

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 a 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.

Hyperglycemia (Blood sugar 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 level possibly contributes 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 brain scans, demonstration of such mentioned imaging abnormalities in association with NDDA is possibly obscured.

Thirty-one (42.0%) of all cerebellar stroke patients developed NDDA, and 28 of them (90.0%) acquired unfavorable neurological outcomes at hospital discharge. When compare with the overall cerebellar stroke outcomes, only 37.8% had unfavorable outcomes at discharge (2.4 folds higher number in NDDA cases) (Table 1). Some previous studies reported a slightly higher percentage of unfavorable final outcomes (50.0%).[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 expect 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 will be useful in providing an appropriate management decision on initiation of early neurosurgical interventions for cerebellar stroke patients.

Conclusion

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.

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Conflict of interest

The authors report no conflicts of interest.

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