석 사 학 위 논 문

Predictors of Early Neurological Deterioration in Patients with Acute Ischemic Stroke of Posterior Circulation

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이 논문을 석사학위 논문으로 제출함

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1. Introduction

Advances in acute stroke treatment have been made over the past two decades. However, disability and mortality after stroke remains high. Neurological deterioration (ND) precedes the severe prognosis. Early ND is a common event that occurs as part of 15% to 40% of acute strokes (1-3). Therefore, it is extremely important to understand and find for the underlying pathogenic mechanisms and for predictors of ND. Presumed neurological and clinical mechanisms are thrombus propagation, brain edema, hemorrhagic transformation, epilepsy, and recurrent stroke, as well as systemic illnesses like infections or metabolic disturbances (4, 5).

Posterior circulation stroke account for about 20% of all strokes (6). Clinical manifestations and outcome of posterior circulation stroke are different from anterior circulation stroke because of the unique anatomy of the posterior circulation and central nerve system (7). Consequently, ND of posterior circulation stroke may be different from anterior circulation stroke. In contrast to anterior circulation stroke, there has been relatively little research into the outcome and predictors for ND specialized in patients with posterior circulation stroke (8-10).

We aimed to evaluate the predictive factors of ND and explore the outcome in patients with acute ischemic stroke (AIS) involving posterior circulation.

2. Materials and Methods

2.1. Study design:

Using a single-center registry, we retrospectively investigated 3,501 AIS patients who were admitted between May 2014 and December 2019.

We retrieved data on demographic and clinical characteristics, neuroimaging findings and acute treatment. Patients were eligible for inclusion if they could be clinically diagnosed with acute stroke by trained neurologists.

2.2. Patients:

Among the 3,501 patients enrolled in the Keimyung University Dongsan Hospital database between May 2014 and December 2019, we excluded 47 who initially had acute hemorrhagic stroke, 344 diagnosed with transient ischemic attack (TIA) or central vertigo without any confirmed acute lesion on diffusion-weight magnetic resonance imaging (MRI) (Figure 1). One patient for whom carbon monoxide poisoning was not excluded was also excluded.

Finally, 900 (25.7%) AIS patients with clinical and radiologic evidence on computed tomography (CT) or diffusion-weighted MRI were included. This study was approved by the institutional review board (IRB) of Dongsan Hospital (IRB number 2021-12-056).

2.3. Measurement:

Stroke onset was defined as the first time a neurologic symptom was noticed. Onset to arrival time was calculated as the time from the stroke onset a neurologic symptom noticed by a patient or a witness to the time when the patient was arrived at the emergency department of the hospital. The stroke onset time was replaced with the last observed normal time when it was unknown.

Previous medical history was documented if reported by the patient during the initial encounter, or if the information were available via record review.

We categorized stroke mechanisms according to a modified version of the Trial of Org 10712 in Acute Stroke Treatment (TOAST) classification system (11). Major stroke etiologies were divided into large artery disease, small vessel disease, cardioembolic, other determined etiology, undetermined two or more etiologies, undetermined negative.

The National Institutes of Health Stroke Scale (NIHSS) score was assessed initially at the arrival on emergency department, and then at least daily during hospitalization by stroke team physicians and nurses. When neurological worsening was observed, the patient was further assessed. Physicians and nurses who assessed the NIHSS and modified Rankin Scale (mRS) were all trained and certified in a standard manner.

The posterior circulation Acute Stroke Prognosis Early CT Score (pc-ASPECTS) was calculated by two readers. The pc-ASPECTS is a 10-point grading system to quantify ischemic damage in the posterior circulation territories, where 10 points represent no ischemic damage and zero points represent substantial damage of the posterior circulation (12). The pc-ASPECTS was calculated based on initial imaging studies, with

priority being given to the initial MRI performed in the emergency department, considering that pc-ASPECTS on diffusion-weighted MRI can be a powerful marker for predicting functional outcome and ND (13). If intra-arterial (IA) thrombectomy was performed, which may affect the degree of ischemia, the score was calculated based on initial CT. Also, we divided the pc-ASPECTS into groups of less than six points and more than six points.

The mRS score and death after discharge were detected during patients' routine clinic visits or through inquiries to patients or their caregivers at three months and one year after the index stroke.

2.4. Definition of neurological deterioration:

ND was defined as any new neurologic symptoms or signs or neurologic worsening that satisfies a change of ≥1 point in the motor or level of consciousness subitems of the NIHSS score or ≧ 2 points in other NIHSS subitems during hospitalization in neurology department within 3 weeks of symptom onset (14, 15).

We categorized the causes of ND as stroke recurrence, stroke progression, symptomatic hemorrhagic transformation, TIA, others, and unknown (16).

2.5. Statistical analysis:

Baseline characteristics of the study participants are presented as percentages for categorical variables and as means ± standard deviations (SDs) for continuous variables.

Comparisons of baseline characteristics between patients with and without ND were made using Pearson $\chi 2$ test for categorical variables, and Student t test for continuous variables, respectively. All variables with the P value < 0.2 on univariate analysis were entered into multivariable logistic regression analysis to the adjusted odds ratios (OR) and their 95% confidence intervals (CI) for factors associated with ND.

All analysis were performed using the statistical software R (version 3.3.2, R Foundation for Statistical Computing, Vienna, Austria [64-bit]) and PASW (version 18.0; SPSS, Chicago, IL).

P values < 0.05 were considered statistically significant.

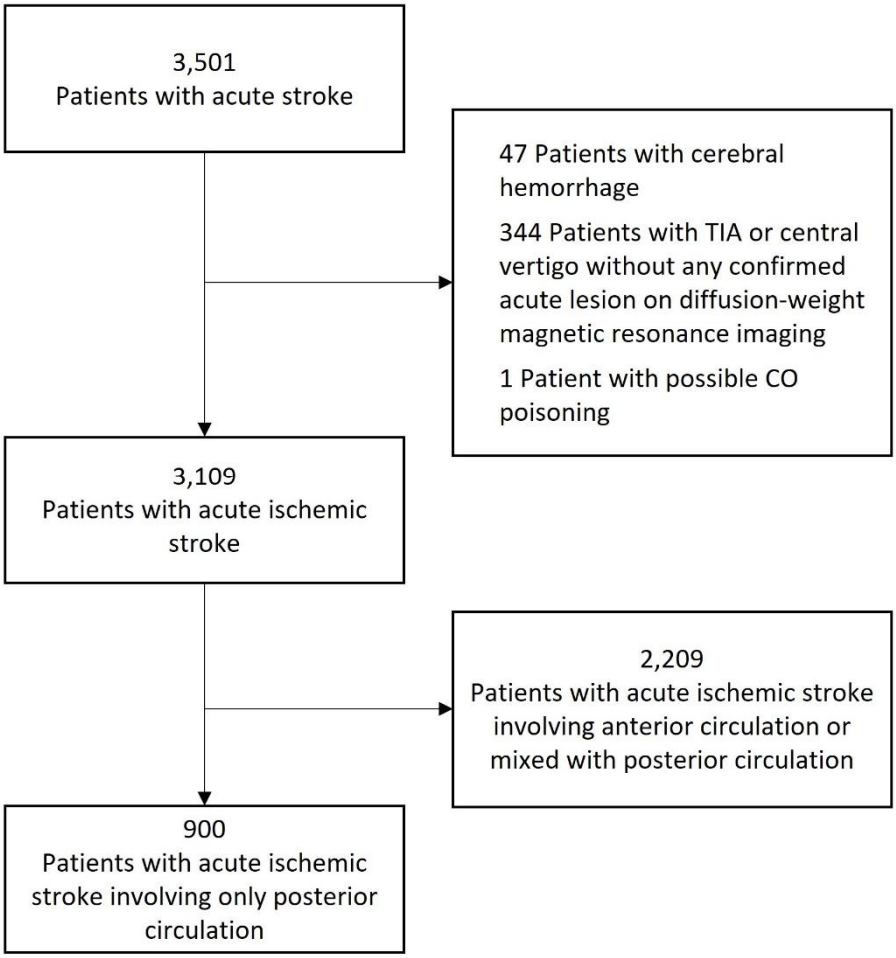

Figure 1. Patients inclusion flowchart. TIA: transient ischemic attack; CO: carbon monoxide

3. Results

3.1. Demographic and laboratory parameters:

Of the included 900 patients (mean age 67 years, 41% female) of AIS of posterior circulation, 133 (14.8%) experienced ND (Table 1). There were no significant differences in the gender distribution, body mass index, previous level of independence (pre-mRS), and TOAST classification between those patients who experienced ND and those who did not. However, there were significant differences in several items in the medial history. Patients with ND had significantly shorter time from onset to arrival at the hospital (mean time 22.9 hours vs 38.3 hours, $P < 0.001$). Significantly more patients had a previous history of stroke (27.1% vs 19.6%, $P < 0.05$) or use of anticoagulants (9.1% vs 4.5%, $P < 0.05$). However, there were no significant differences in the medical history for other items such as previous history of hypertension, diabetes mellitus, hyperlipidemia, coronary heart disease, atrial fibrillation, high risk of potential cardiac sources of embolism (PSCE), TIA, and use of antiplatelet agent. The most common etiology of stroke was large artery atherosclerosis, which accounted for 35%.

Patients with ND had significantly higher mean NIHSS score on admission (6.0 vs 3.3, $P < 0.001$) and lower pc-ASPECTS score (7.5 vs 8.2, $P < 0.001$). The percentage of patients with pc-ASPECTS score of six or more were significantly higher in the ND group (87.2%) compared to the no ND group (97.3%) ($P < 0.001$). Patients with ND had significantly higher fasting blood glucose level (134 vs 122, $P < 0.05$). We could not find any statistically significant difference in platelet

counts and serum C-reactive protein (CRP) levels between two groups. However, a trend toward lower platelet counts and higher CRP level and higher percentage of ND was observed. There were no significant differences in other laboratory parameters on admission, including serum hemoglobin, white blood cell (WBC) count, prothrombin time, D-dimer, blood urea nitrogen (BUN), creatinine, initial random glucose, total cholesterol, high-density lipoproteins (HDL), and low-density lipoproteins (LDL), total triglycerides (TG), hemoglobin A1c (HbA1c). Both initial systolic and diastolic blood pressure were higher in patients with ND than those without ND, but only systolic blood pressure showed statistical significance (158 vs 150, $P < 0.05$).

When comparing the presence of stenosis or occlusion of the representative arteries responsible for the posterior circulation, the posterior cerebral artery (PCA), the basilar artery (BA), and the vertebral artery (VA), only significant differences were observed between the patients with and without ND in the BA ($P < 0.001$) (Table 2). BA occlusion was observed in 16.5% of the patients with ND but only 4.4% of the patients without ND.

3.2. Comparison of functional outcome and symptom severity between patients with and without ND:

Mortality at 3 months was 20.6% in ND group and 1.6% in no ND group (Figure 2). Mortality at 1 year was 25.2% in ND group and 3.9% in no ND group. The proportion of patients with mRS 0 to 2, which is considered a good functional outcome, was 53.6% in the ND group at 1 year and 83.7% in the no ND group.

Comparing the NIHSS scores at admission and discharge, patients with a score of less than 5 at admission were 53.4% in the ND group and 78% in the no ND group (Figure 3). The number of cases where the score at discharge was 17 or higher indicating bed-ridden life was 21.8% in the ND group, but only 1.7% in the no ND group.

3.3. Etiology of ND:

Of etiologies of ND, stroke progression accounted for the largest proportion (81%) (Figure 4). Next, other causes including medical condition (8%), symptomatic hemorrhagic transformation (6%), and stroke recurrence (5%) followed. There were no causes of ND corresponding to unknown and TIA in our study.

3.4. Predictors of ND:

We conducted multivariate logistic regression analysis to test factors that were identified as potentially associated with ND (Table 3). Although it was not statistically significant, platelet count and CRP level were also included considering that their P values were each less than 0.1. Multivariate logistic regression analysis revealed that independent predictors of ND were onset to arrival time (OR 0.999, CI 0.9997-0.9999, $P < 0.05$), ischemic damage reflected in pc-ASPECTS (OR 0.415, CI 0.167-0.996, $P < 0.05$), initial systolic blood pressure (OR 1.009, CI 1.001-1.017, $P < 0.05$), and the presence of any stenosis (OR 1.920, CI 1.024-3.472, $P < 0.05$) or occlusion (OR 4.803, CI 1.726-13.781, $P < 0.05$) of BA.

Table 1A. Comparison of Demographic and Laboratory Parameters between Patients with and without ND

Variables	Total (N = 900)	ND (N = 133)	No ND (N = 767)	P value
Age, y, mean ± SD	67.3 ± 13	69.9 ± 12.8	66.9 ± 13	< 0.05*
Female, n (%)	369 (41.0)	59 (44.4)	310 (40.4)	NS
Time from onset to arrival, hr, mean ± SD	36 ± 44.8	22.9 ± 29.2	38.3 ± 46.6	< 0.001**
BMI, kg/㎡, mean ± SD	23.8 ± 3.2	23.5 ± 3.7	23.8 ± 3.1	NS
Risk factors and medical history, n (%)				
Hypertension	586 (65.2)	92 (69.2)	494 (64.5)	NS
Diabetes mellitus	340 (37.8)	52 (39.1)	288 (37.6)	NS
Hyperlipidemia	177 (19.7)	21 (15.8)	156 (20.4)	NS
Coronary heart disease	80 (9.4)	11 (8.7)	69 (9.5)	NS
Atrial fibrillation	118 (13.2)	22 (16.5)	96 (12.6)	NS
PSCE high	124 (13.8)	22 (16.5)	102 (13.3)	NS
Previous stroke	186 (20.7)	36 (27.1)	150 (19.6)	< 0.05*
Previous TIA	10 (1.1)	2 (1.5)	8 (1.0)	NS
Previous antiplatelet	275 (30.7)	39 (29.5)	236 (30.9)	NS
Previous anticoagulant	46 (5.1)	12 (9.1)	34 (4.5)	< 0.05*
TOAST, n (%)				NS
Large artery atherosclerosis	315 (35)	49 (36.8)	266 (34.5)	
Small vessel occlusion	274 (30.4)	42 (31.6)	232 (30.2)	
Cardioembolism	120 (13.3)	18 (13.5)	102 (13.3)	
Other determined etiology	15 (1.7)	2 (1.5)	13 (1.7)	
Undetermined two or more	35 (3.9)	8 (6)	27 (3.5)	

Table 1B. Comparison of Demographic and Laboratory Parameters between Patients with and without ND (continued)

Variables	Total (N = 900)	ND (N = 133)	No ND (N = 767)	P value
Undetermined negative	115 (12.8)	9 (6.8)	106 (13.8)	
Undetermined incomplete	25 (2.8)	5 (3.8)	20 (2.6)	
Initial NIHSS, median [IQR]	2 [1-5]	2 [1-4]	4 [1-6]	< 0.001**
<5, n (%)	669 (74.3)	71 (53.4)	598 (78)	
5-8, n (%)	154 (17.1)	37 (27.8)	117 (15.3)	
9-16, n (%)	41 (4.6)	13 (9.8)	28 (3.7)	
≥17, n (%)	36 (4)	12 (9)	24 (3.1)	
Previous mRS, mean ± SD	0.2 ± 0.8	0.3 ± 1.0	0.2 ± 0.8	NS
pc-ASPECTS, mean ± SD	8.1 ± 1.1	7.5 ± 1.5	8.2 ± 1.1	< 0.001**
pc-ASPECTS ≥6, n (%)	862 (95.8)	116 (87.2)	746 (97.3)	< 0.001**
Recanalization therapy, n (%)				< 0.001**
IV	38 (4.2)	13 (9.8)	25 (3.3)	
IA	27 (3)	10 (7.5)	17 (2.2)	
IV+IA	10 (1.1)	2 (1.5)	8 (1)	
Laboratory findings, mean ± SD				
Hemoglobin, g/dL	13.5 ± 1.7	13.4 ± 1.9	13.5 ± 1.7	NS
WBC, $\times 10^3/\mu L$	8.2 ± 3.1	8.4 ± 3.2	8.2 ± 3.1	NS
Platelet count, $\times 10^3/\mu L$	247 ± 81	236 ± 68	249 ± 83	< 0.1***
Prothrombin time, INR	1 ± 0.3	1 ± 0.2	1 ± 0.3	NS
D-dimer, μg/mL	1.1 ± 4.3	1.1 ± 3.3	1.1 ± 4.4	NS

Table 1C. Comparison of Demographic and Laboratory Parameters between Patients with and without ND (continued)

Variables	Total (N = 900)	ND (N = 133)	No ND (N = 767)	P value
BUN, mg/dL	18.0 ± 8.0	19 ± 9.0	18.0 ± 8.0	NS
Creatinine, mg/dL	1.0 ± 1.1	1.1 ± 1.4	1.0 ± 1.1	NS
Fasting blood glucose, mg/dL	124 ± 46	134 ± 56	122 ± 43	< 0.05*
Initial random glucose, mg/dL	153 ± 61	157 ± 61	152 ± 61	NS
Total cholesterol, mg/dL	173 ± 46	173 ± 46	173 ± 46	NS
HDL, mg/dL	45 ± 12	44 ± 12	45 ± 12	NS
LDL, mg/dL	111 ± 39	111 ± 43	111 ± 39	NS
TG, mg/dL	130 ± 74	129 ± 80	130 ± 72	NS
HbA1c, mg/dL	6.6 ± 1.5	6.5 ± 1.3	6.6 ± 1.5	NS
CRP, mg/L	1.1 ± 3.4	1.6 ± 3.8	1.0 ± 3.3	< 0.1***
Initial SBP, mmHg, mean ± SD	152 ± 26	158 ± 28	150 ± 26	< 0.05*
Initial DBP, mmHg, mean ± SD	86 ± 15	88 ± 16	86 ± 15	NS

BMI: body mass index; CRP: c-reactive protein; DBP: diastolic blood pressure; HbA1c: hemoglobin A1c; HDL: high-density lipoproteins; IA: intra-arterial thrombectomy; INR: international normalized ratio; IV: intra-venous alteplase; LDL: low-density lipoproteins; mRS: modified Rankin Scale; ND: neurological deterioration; NIHSS: National Institutes of Health Stroke Scale; NS: non-significant; pc-ASPECTS: posterior circulation Acute Stroke Prognosis Early CT Score; PSCE: potential cardiac sources of embolism; SBP: systolic blood pressure; SD: standard deviation; TG: total triglycerides; TIA: transient ischemic attack; TOAST: Trial of Org 10712 in Acute Stroke Treatment; WBC: white blood cell; *: $P < 0.05$, **: $P < 0.001$, ***: $P < 0.1$

Table 2. Comparison of Status of Vessel Stenosis between Patients with and without ND Based on Magnetic Resonance Angiography

Vessel	Total	ND	No ND	P value
PCA, n (%)				NS
No stenosis	793 (88.2%)	124 (93.2%)	669 (87.3%)	
Any stenosis	38 (4.2%)	3 (2.3%)	35 (4.6%)	
Occlusion	68 (7.6%)	6 (4.5%)	62 (8.1%)	
BA, n (%)				< 0.001*
No stenosis	756 (84%)	91 (68.4%)	665 (86.7%)	
Any stenosis	88 (9.8%)	20 (15%)	68 (8.9%)	
Occlusion	56 (6.2%)	22 (16.5%)	34 (4.4%)	
VA, n (%)				NS
No stenosis	757 (84.3%)	111 (83.5%)	646 (84.4%)	
Any stenosis	75 (8.4%)	11 (8.3%)	64 (8.4%)	
Occlusion	66 (7.3%)	11 (8.3%)	55 (7.2%)	

BA: basilar artery; ND: neurological deterioration; NS: non-significant; PCA: posterior cerebral artery; VA: vertebral artery; *: P < 0.001

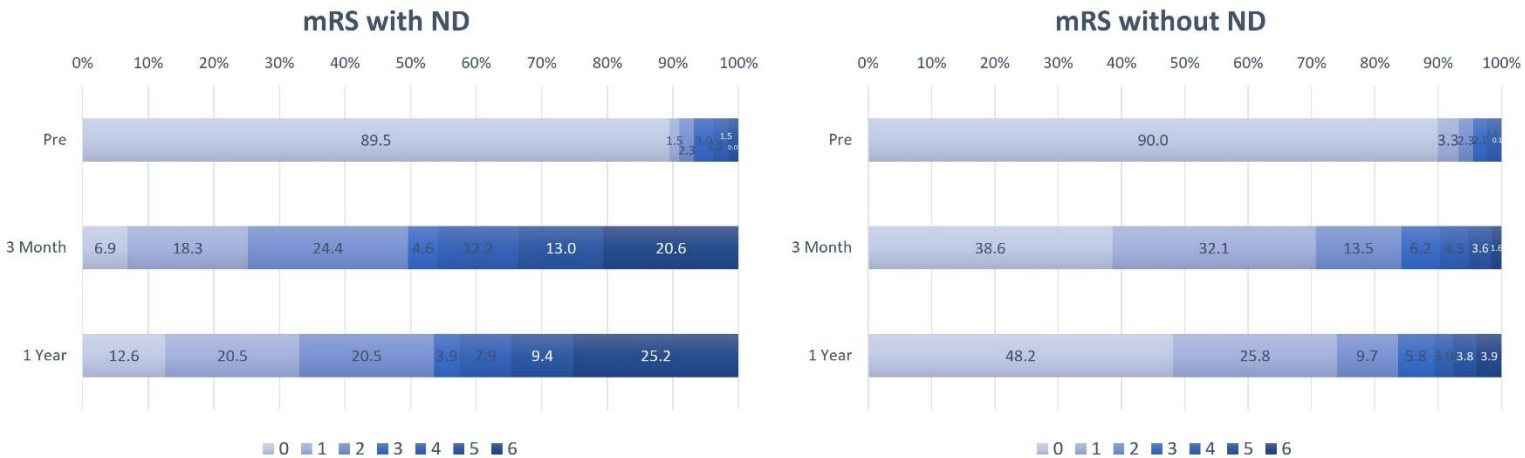

Figure 2. Functional outcomes before stroke an at 3 months and 1 year after stroke according to ND. mRS: modified Rankin Scale; ND: neurological deterioration

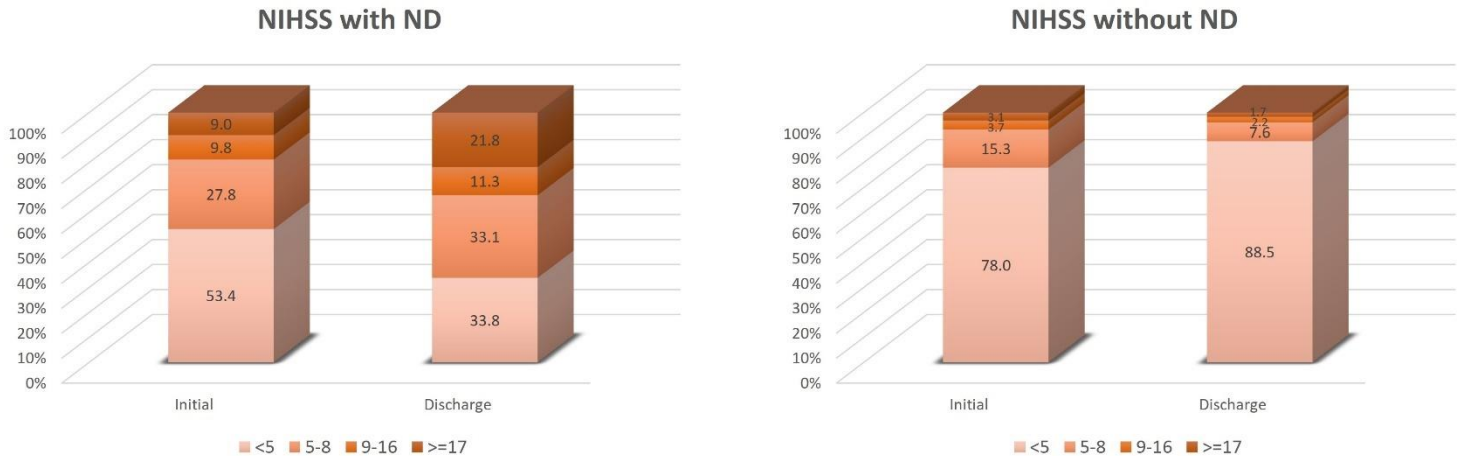

Figure 3. Comparison of NIHSS score at initial arrival and discharge according to ND. ND: neurological deterioration; NIHSS: National Institutes of Health Stroke Scale

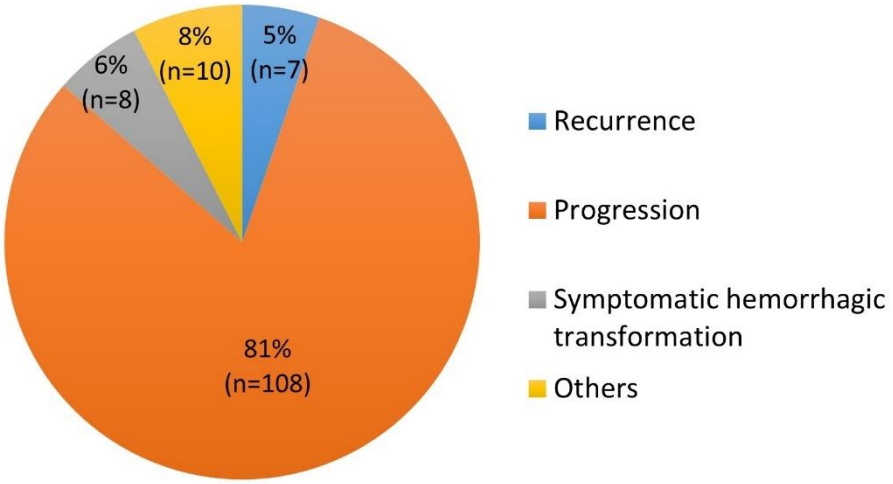

Figure 4. Etiology of ND. ND: neurological deterioration

Table 3A. Multivariate Logistic Regression Analysis for Possible Factors Associated with ND

	Univariable analysis	Multivariable analysis		
	P value	OR	95% CI	P value
Age	< 0.05	1.007	0.989-1.025	NS
Onset to arrival time	< 0.001	0.999	0.9997-0.9999	< 0.05*
Risk factors and medical history				
Previous stroke	< 0.05	1.180	0.710-1.917	NS
Previous anticoagulant	< 0.05	1.803	0.803-3.802	NS
Initial NIHSS	< 0.001	1.032	0.988-1.078	< 0.1**
pc-ASPECTS ≥6	< 0.001	0.415	0.167-0.996	< 0.05*
Basilar artery status	< 0.001			
No stenosis		1.000 (referent)		
Any stenosis		1.920	1.024-3.472	< 0.05*
Occlusion		4.803	1.726-13.781	< 0.05*
Recanalization therapy	< 0.001	0.796		
No		1.000 (referent)		
IV		2.206	0.928-5.015	NS
IA		0.376	0.088-1.454	NS
IV+IA		0.177	0.020-1.105	NS

Table 3B. Multivariate Logistic Regression Analysis for Possible Factors Associated with ND (continued)

	Univariable analysis	Multivariable analysis		
	P value	OR	95% CI	P value
Laboratory findings				
Platelet count	< 0.1	0.998	0.995-1.001	NS
Fasting blood glucose	< 0.05	1.003	0.998-1.007	NS
CRP	< 0.1	1.021	0.966-1.073	NS
Initial SBP	< 0.05	1.009	1.001-1.017	< 0.05*

CI: confidence interval; CRP: c-reactive protein; IA: intra-arterial thrombectomy; IV: intra-venous alteplase; ND: neurological deterioration; NIHSS: National Institutes of Health Stroke Scale; NS: non-significant; OR: odds ratio; pc-ASPECTS: posterior circulation Acute Stroke Prognosis Early CT Score; SBP: systolic blood pressure; *: $P < 0.05$; **: $P < 0.1$

4. Discussion

The main findings of this study indicate that early arrival from symptom onset, multiple infarctions reflected in less than six pc-ASPECTS, high initial systolic blood pressure, presence of any stenosis or occlusion in the BA may be independently predictive of ND in AIS of posterior circulation.

ND occurred in 14.8% of patients in our study, which is not significantly different from the previous results (17). Given that the estimated incidence of ND decreases steeply as time passed after stroke onset with the highest incidence within six hours of stroke (2), some deterioration might have occurred before arrival. The patients with short delay to arrival after stroke onset are likely to have higher rates of ND, and the present study showed consistent results.

We did not find any significant association between ND and LDL or HDL level. This is contrasted result of previous studies (18-20), albeit those studies covered AIS throughout anterior and posterior circulation. Although not significant in multivariable regression analysis, the fasting blood glucose level was higher amongst patients with ND. Previous studies reported association about glucose level, such as elevated blood sugar concentration at admission (21), high HbA1c level on admission (19), and history of DM (14, 22).

However, this study found an association with old age, history of stroke and history of anticoagulation. Also, patients with ND had low platelet counts and high CRP levels, while those did not independently predict ND. CRP has been consistently associated with ND or prognosis of stroke in previous studies (18, 23, 24). The low platelet counts may contribute to more susceptible to hemorrhagic transformation of ND.

The patients with higher stroke severity were more likely to have early neurological deterioration. This result is in agreement with most previous studies (19, 25, 26). The stroke severity was represented clinically in NIHSS score and radiologically in pc-ASPECTS score, though the NIHSS score was not identified to be an independent predictor as it was marginally not statistically significant. Pc-ASPECTS of six or more was predicted to be less likely to develop ND, in line with a previous study (8). This study also showed that patients with ND had initially high NIHSS score and associated with poor functional outcome than those without ND.

Most of the previous studies in the field of ND have focused on anterior circulation stroke, while a few studies specialized in predictors of ND and outcome in AIS of posterior circulation (8-10). We found that in contrast to BA, stenosis or occlusion of PCA or VA was not associated with ND. In addition, this study showed BA occlusion had a higher association with risk of ND than in BA stenosis. In particular, as the largest artery of the posterior circulation, BA occlusion was identified to be the strongest independent predictor amongst all significant factors. This finding suggests similarities with a previous findings that BA occlusion from in situ atherosclerotic thrombosis had worse clinical outcome compared to embolism without VA stenosis (27). The presence of stenosis of arteries was detected based on magnetic resonance angiography (MRA) images, the degree of stenosis was not measured here. MRA images tended to show an overestimation of the degree of stenosis relative to digital subtraction angiography (28), and the number of patients with BA stenosis of any degree was small with only 88 in our study sample. Therefore, the association with ND was not compared according to the degree of stenosis.

A significant strength of our study is the use of clinical database of

large population, where the examination score or outcome was prospectively obtained. However, this study has some limitations. First, analysis according to the location of lesion was not performed. While pc-ASPECTS calculated here reflected the severity of ischemic damage, the locations of lesions were not recorded. There are a few previous studies which analyzed the locations of lesions of posterior circulation (9, 19), but the locations were not found to be independent predictors of ND or outcome in both studies. Second, the lesion volumes which means the size of infarct cores were not measured. The pc-ASPECTS was calculated preferentially based on the initial MRI. Considering the pc-ASPECTS system, it does not quantify lesion volumes, and the same score was given regardless of the size of the same lesion location. In other words, the score of a dot-like infarction lesion of the cerebellum or a large lesion involving the entire cerebellar hemisphere was equal to one point, although the latter case could even cause obstructive hydrocephalus and increase mortality. Nonetheless, lesion location rather than lesion volume may be salient for functional outcome in posterior circulation because the anatomical architecture of the posterior circulation has a high density of motor and sensory pathways and nuclei compared to the supratentorial hemisphere (29). After all, it is an unsolved issue in this study. In addition, the pc-ASPECTS on follow-up images were not calculated. One study found that more extensive hypoattenuation on CT angiography source images, quantified with the pc-ASPECTS, is associated with a larger final infarct extent in patients with BA occlusion (30). Here measuring the infarct growth through pc-ASPECTS on follow-up images was not performed. Third, this is a single-center retrospective study, which may limit the generalizability of results. It was insufficient to draw convincing results in some parts, especially the vascular stenosis was subdivided according to the degree

and the sample size of each was too small, as described earlier. Further larger scale studies are needed to verify our findings.

5. Summary

The predictors of ND in AIS of posterior circulation are multifaceted. Early arrival, poor neuroimaging findings, any stenosis or occlusion in the BA and high initial blood pressure are identified to be independent predictors of ND. Occlusion in the BA is the strongest predictor of the prognosis among the predictors of ND. However, occlusion in the PCA or VA is not related to ND. In addition, ND in AIS involving posterior circulation was disclosed to lead to bad prognosis on short-term or long-term clinical outcome.

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Predictors of Early Neurological Deterioration in Patients with Acute Ischemic Stroke of Posterior Circulation

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(Abstract)

We aimed to evaluate the predictive factors of neurological deterioration (ND) in patients with acute ischemic stroke (AIS) involving posterior circulation. Using a single-center registry, we retrospectively investigated 3,501 AIS patients who were admitted between May 2014 and December 2019. We analyzed data on demographic and clinical characteristics, neuroimaging findings and acute treatment. Of 900 (25.7%) AIS patients involving posterior circulation (mean age 67 years, 41% female), 133 (14.8%) experienced ND. In univariate analysis, there was a significant difference between groups with and without ND in age, onset to arrival time, previous history of stroke or anticoagulation, initial National Institutes of Health Stroke Scale (NIHSS) score, the posterior circulation Acute Stroke Prognosis Early CT Score

(pc-ASPECTS), recanalization therapy, fasting blood glucose, and initial systolic blood pressure, and stenosis in the basilar artery (BA). In multivariate logistic regression analysis, predictors of ND were early arrival from symptom onset, multiple infarctions reflected in low pc-ASPECTS, high initial systolic blood pressure, and any stenosis or occlusion of the BA. The predictors of ND in patients with AIS of posterior circulation are multifaceted. Early arrival, poor neuroimaging findings, any stenosis or occlusion in the BA and high initial blood pressure are identified to be independent predictors of ND. Occlusion in the BA is the strongest predictor of the prognosis among the predictors of ND. However, occlusion in the PCA or VA is not related to ND. In addition, ND in AIS involving posterior circulation was disclosed to lead to bad prognosis on short-term or long-term clinical outcome.

후순환계 허혈성 뇌졸중에서의 초기 신경학적 악화의 예측 인자

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(초록)

급성기 뇌경색 환자에서 초기 신경학적 악화(ND)는 흔히 발생하며 장기적으로 불량한 예후와 연관되는데, 이러한 장기 예후에 대해 초기에 예상하는 것은 의학적인 판단과 치료 방침 결정을 할 때 매우 중요하다. 실제적으로 ND 의 상당한 발생률에도 불구하고 인구학적 자료가 부족하여 어떠한 인자가 후순환계 뇌경색 환자에게서 ND 를 발생시키고 예후에 영향을 주는지에 대한 연구는 부족하다. 현재까지 밝혀진 ND 의 임상적인 예측 인자로는 나이, 고혈압과 심방 세동 등의 동반 질환, 치료까지의 소요 시간, 병변의 크기, 병변의 출혈성 변환 등이 있으나 전순환계를 포함한 연구가 대부분으로 후순환계 뇌졸중에는 적용되지 않는 인자도 많다. 발생하는 ND 를 예측, 발견하고 이를 조기에 대처하는 것이 급성기 뇌졸중의 치료 중 하나가 된 바, 후순환계 뇌졸중에서의 ND 의 예측 인자에 대해 연구하였다. 2014 년부터 2019 년까지 계명대학교 동산병원

신경과에 입원한 급성기 뇌경색 환자들을 대상으로 병력, 신경학적 소견, 영상소견, 검사실 소견을 후향적으로 분석하였다. ND 의 예측 인자를 찾기 위해 연속형 변수에 대해서는 t 검정을 시행하였고 범주형 변수에 대해서는 카이제곱 검정을 시행하였다. 단일변량분석을 시행하였을 때, 증상 발생으로부터의 소요 시간, 이전의 뇌졸중이나 일과성 뇌허혈 발작의 병력, 초기 뇌졸중척도(NIHSS) 점수, pc-ASPECTS 점수, 재관류 치료, 공복 혈당, 수축기 혈압, 뇌기저동맥의 협착에 대해 ND 의 유무에 따라 통계적으로 유의한 차이가 관찰되었다. 연관성이 있을 것으로 확인된 인자들을 추출하여 다변량회귀분석을 시행하였으며, 증상 발생으로부터의 소요 시간이 짧을수록, 초기 영상학적 검사에서 허혈 손상이 심할수록, 초기 수축기 혈압이 높을수록, 그리고 뇌기저동맥의 협착이나 폐색이 있을 때 ND 가 발생할 위험도가 높음이 독립적으로 예측되었다. 뇌기저동맥에 반해 후대뇌동맥이나 척추동맥은 ND 에 대한 예측성이 없었으며, 후순환계 뇌졸중에서의 ND 는 예후가 불량한 경향을 보였다.