ORIGINAL ARTICLE

Lesions on DWI and the Outcome in Hyperacute Posterior Circulation Stroke

Hye Mi Lee, Minjik Kim, Sang-il Suh, Ji Hyun Kim, Kyungmi Oh, Seong-Beom Koh, Woo-Keun Seo

ABSTRACT: ***Background:*** Few studies have addressed the association between the characteristics of ischemic lesions detected by diffusion-weighted imaging (DWI) and the clinical outcome in patients with hyperacute posterior circulation ischemic stroke. This study demonstrates a relationship between the findings assessed by DWI and the outcome in patients with hyperacute posterior circulation ischemic stroke. ***Methods:*** We reviewed data from 118 patients who had posterior circulation ischemic stroke within six hours from the onset of their symptoms. The clinical outcome included early neurological deterioration (END) and a favorable outcome at three months after the onset of symptoms. Using DWI, the lesion volume and the number and location of injured anatomical regions were analyzed to evaluate whether the results correlated with the clinical outcome measures. ***Results:*** The number of injured anatomical regions assessed by DWI was associated with the initial and delayed neurological status. Both the total volume and the number of injured anatomical regions associated with END and a favorable outcome. Analysis of the location of the injured regions determined that only a pontine lesion independently associated with END. Interestingly, four out of five patients who underwent decompressive craniectomy exhibited a large infarction volume but minor symptoms. ***Conclusions:*** In patients with hyperacute posterior circulation ischemic strokes, the lesions assessed by DWI were associated with the clinical outcome, regardless of the initial neurological status. DWI is an effective initial imaging tool for assessing the extent of lesions and clinical outcomes in patients with hyperacute posterior circulation ischemic stroke.

RÉSUMÉ: Lésions détectées à l'imagerie pondérée en diffusion (IPD) et issue d'un accident vasculaire hyperaigu au niveau de la circulation postérieure. ***Contexte :*** Peu d'études ont examiné l'association entre les caractéristiques des lésions ischémiques détectées par imagerie pondérée en diffusion (IPD) et les conséquences cliniques chez les patients présentant un accident vasculaire cérébral (AVC) hyperaigu au niveau de la circulation postérieure. Cette étude démontre qu'il existe une relation entre les constatations faites à l'IPD et l'issue chez des patients atteints d'un AVC ischémique hyperaigu de la circulation postérieure. ***Méthode :*** Nous avons revu les données recueillies chez 118 patients atteints d'un AVC ischémique de la circulation postérieure dans un délai de 6 heures du début des symptômes. Au point de vue clinique, les patients présentaient une détérioration neurologique précoce suivie d'une issue favorable trois mois après le début des symptômes. À l'IPD, le volume de la lésion, le nombre et les régions anatomiques touchées ont été analysés pour évaluer si les résultats étaient corrélés à l'issue clinique. ***Résultats :*** Le nombre de régions anatomiques touchées à l'IPD était associé au statut neurologique initial et à 3 mois de l'événement. Tant le volume total que le nombre de régions anatomiques touchées étaient associés à la détérioration neurologique précoce et à l'issue favorable. L'analyse de la localisation des régions touchées a révélé que la présence de seulement une lésion située au niveau du pons était associée de façon indépendante à la détérioration neurologique précoce. Il était intéressant de constater que 4 patients sur 5 qui ont une craniectomie de décompression avaient un infarctus étendu mais peu de symptômes. ***Conclusions :*** Chez les patients atteints d'un AVC ischémique hyperaigu de la circulation postérieure, les lésions détectées par IPD étaient associées à l'issue clinique, quelque soit le statut neurologique initial. L'IPD est une méthode d'imagerie initiale efficace pour évaluer l'étendue des lésions et l'issue clinique chez les patients atteints d'un AVC hyperaigu de la circulation postérieure.

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Findings from diffusion-weighted imaging (DWI) have been used as indicators of early changes in ischemic brain tissue; several studies have suggested that initial DWI findings can predict the clinical severity and clinical outcomes for a patient.[1-8] A large proportion of these studies included patients with anterior circulation ischemic stroke.[1-6] However, DWI is more useful in detecting infarctions and estimating the severity of ischemic change in patients with posterior circulation ischemic stroke; DWI is more sensitive than computed tomography (CT) for identifying structures in the posterior fossa.[9,10] Few studies have addressed whether lesions assessed by DWI are associated with the clinical outcomes of patients with acute posterior circulation ischemic stroke.[7,8,11] In previous studies, the lesion volume and the number and distribution of

From the Department of Neurology (HML, MK, JHK, KO, SBK, WKS), Department of Radiology (SIS), College of Medicine, Korea University, Seoul, Republic of Korea.

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Correspondence to: Woo-Keun Seo, Department of Neurology, Korea University Guro Hospital, 148 Gurodong-ro, Guro-gu, Seoul, [152-703], South Korea.
Email: nukseo@korea.ac.kr.

lesions assessed by DWI did not correlate with the National Institutes of Health Stroke Scale (NIHSS) score and outcome scales.[8,11] However, the number of lesions in ischemic posterior circulation regions was a predictor of their functional outcome.[7] These studies involved patients with acute posterior circulation stroke and to our knowledge, studies that address the correlation between lesions assessed by DWI and clinical outcomes in patients with hyperacute posterior circulation ischemic stroke have not yet been reported. Therefore, in this study, we investigated predictors of clinical severity and clinical outcomes in patients with hyperacute posterior circulation ischemic stroke based on DWI lesion volume and anatomical location data. If DWI findings scanned before thrombolytic treatment have the potential to predict the clinical outcome for hyperacute posterior circulation ischemic stroke patients, it would provide meaningful information to help neurologists determine proper course of medical treatment.

Methods

Study Population

We reviewed data from the Korea University Stroke Registry - Guro Arm (KUSR-G), which were collected between January 1, 2008, and February 29, 2012. Among 1730 patients with acute ischemic stroke, clinical diagnosis and/or DWI determined that 371 patients (21.4%) had posterior circulation ischemic stroke. Patients with hyperacute posterior circulation ischemic stroke were defined as patients who arrived at emergency department within six hours of the onset of symptoms and 118 patients (6.8%) had hyperacute posterior circulation ischemic stroke. A diagnosis of acute ischemic stroke was based on the presence of focal or global neurological deficits and on the brain lesions detected by DWI or by subsequent magnetic resonance imaging (MRI) or CT. This study protocol was approved by the ethics committee of Korea University, Guro Hospital.

Clinical Assessment

Baseline demographic and clinical information was collected from patients' medical records and included age, gender, stroke characteristics and cardiovascular risk factors, such as hypertension, diabetes, atrial fibrillation, smoking and a previous history of stroke/transient ischemic attack (TIA) or coronary heart disease. Patient laboratory information was collected after at least eight hours of fasting, within 24 hours from admission, and included levels of total cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, white blood cell count (WBC) and C-reactive protein (CRP). Inclusion and exclusion criteria for the safe implementation of thrombolytic treatment in hyperacute ischemic stroke patients were verified according to clinical practice guidelines.[12] Stroke characteristics included symptom-to-door time and the subtypes of stroke, which were classified according to the Trial of ORG 10172 in Acute Stroke Treatment.[13] Stroke severity was assessed using the NIHSS score at the time of presentation and seven days after stroke. Functional status was assessed using a modified Rankin Scale (mRS) score at the time of presentation and three months after stroke. The clinical scales were determined prior to the first imaging study, and all patients underwent DWI before deciding whether to proceed with thrombolytic therapy. The clinical outcome measure was assessed by early neurological deterioration (END) and the presence or absence of a favorable outcome. Early neurological deterioration was defined as an increase in the NIHSS score by ≥2 points, the enforcement of a decompressive craniectomy or a stroke-related death between admission and seven days after stroke.[14,15] A favorable outcome was defined as an mRS score of ≤2 at three months after stroke.

Acquisition and Assessment of Brain Imaging

Magnetic resonance imaging examination was performed using 1.5 Tesla MR equipment (Sonata, Siemens, Erlangen, Germany) or 3.0 Tesla MR equipment (TrioTim, Siemens, Erlangen, Germany) with computer workstations (Syngo VA30 for 1.5 Tesla MR and Syngo VB17 for 3.0 Tesla MR). All patients underwent diffusion-weighted imaging (TR 3100 ms; TE 84 ms; slice thickness 5 mm; FOV 230 x 230; no gap for 1.5 Tesla MR or TR 3300 ms; TE 87 ms; slice thickness 5 mm; FOV 230 x 230; no gap for 3.0 Tesla MR) and T2* gradient echo imaging (TR 700 ms; TE 26 ms; slice thickness 5 mm; FOV 175 x 230 for 1.5 Tesla MR or TR 500 ms; TE 15 ms; slice thickness 5 mm; FOV 187 x 230 for 3 Tesla MR) before thrombolysis. Final infarctions and hemorrhagic transformation were checked by DWI or FLAIR imaging at least 24 hours after the onset of symptoms. The final vascular status was assessed using contrast-enhanced MR angiography or CT angiography third or fourth days after the onset of stroke. Lesions detected by DWI with high signal intensity on b factor – 1000 s/mm^2 and low signals on corresponding ADC maps were blindly reviewed by a single experienced board certificated neuroradiologist (SSI) and volumes of lesions were recorded by a single blinded experienced observer (LHM). The observer manually outlined the area of diffusion and hyperintensity on each slice, twice per measurement, and recorded the average value. Because there was no inter-slice gap, the volume of the lesion was determined by multiplying the area of lesions by the section thickness (5 mm). In addition, brain structures were divided into six parts (cortex and/or subcortical white matter, thalamus, midbrain, pons, medulla and cerebellum), and the regions with ischemic lesions were counted. In terms of volume measure of the lesions on DWI, the intra-observer correlation was excellent (Spearman's rho = 0.94, $P < 0.001$).

Statistical Analysis

Statistical analysis was performed using SPSS (version 17.0; SPSS Inc., Chicago, IL, USA), and P values <0.05 were regarded as significant. Descriptive statistics are presented as the mean ± standard deviation (SD) and as a proportion for categorical variables. Differences between groups were tested with the Mann-Whitney U test for continuous variables and the Chi-square test for categorical variables. Multivariate logistic regression analysis was performed to determine which anatomical regions could independently predict END or a favorable outcome after adjusting for covariates. The results of logistic regression analysis were presented as an odds ratio (OR) with a 95% confidence interval (CI).

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Table 1: Demographics and clinical characteristics of the study population

Variables	END (n=106)		Favorable outcome (n=110)	
	Y (n=16)	N (n=90)	Favorable (n=92)	Unfavorable (n=18)
Age (years)	66.31 ± 10.24	60.82 ± 13.52	60.71 ± 13.32	67.00 ± 12.95
Female:Male	5 : 11	31 : 59	32 : 60	5 : 13
NIHSS at presentation†	7.56 ± 7.71* (4.50, 2.00 - 12.75)	2.99 ± 4.83 (2.00, 1.00 - 3.00)	2.18 ± 2.58** (2.00, 1.00 - 3.00)	14.72 ± 9.01 (13.00, 6.00 - 23.50)
Symptom-to-door time (min)	193 ± 147	189 ± 215	179 ± 131	241 ± 261
Thrombolysis (IV and/or IA)	4 (25.0)	8 (8.9)	6 (6.5)**	9 (50.0)
Major vessel occlusion (BA, VA and/or PCA)	5 (31.3)	12 (13.3)	11 (12.0)*	7 (38.9)
Decompressive surgery	5 (31.3)**	0 (0.0)	2 (2.2)	2 (11.1)
Stroke subtypes				
LAA	7 (43.8)	26 (28.9)	25 (272.2)	8 (44.4)
CE	6 (37.5)	17 (18.9)	20 (21.7)	7 (38.9)
SVO	1 (6.3)	21 (23.3)	22 (23.9)*	0 (0.0)
UDE	1 (6.3)	22 (24.4)	21 (22.8)	1 (5.6)
ODE	1 (6.3)	4 (4.4)	4 (4.3)	2 (11.1)
Risk factors				
Hypertension	14 (87.5)**	42 (46.7)	47 (51.1)	11 (61.1)
Diabetes	6 (37.5)	18 (20.0)	17 (18.5)*	10 (55.6)
Atrial fibrillation	6 (37.5)*	11 (12.2)	12 (13.0)*	8 (44.4)
Smoker	4 (25.0)	26 (28.9)	25 (27.2)	5 (27.8)
Stroke/TIA	7 (43.8)*	17 (18.9)	19 (20.7)**	8 (44.4)
Total cholesterol (mmol/L)	178.44 ± 41.67	179.53 ± 38.41	179.11 ± 37.93	182.89 ± 41.90
LDL (mmol/L)	113.38 ± 35.47	112.66 ± 32.81	112.91 ± 32.52	115.89 ± 36.48
TG (mmol/L)	145.75 ± 107.09	123.61 ± 83.63	123.48 ± 84.01	165.67 ± 128.02
HDL (mmol/L)	42.88 ± 13.99	46.18 ± 10.38	46.19 ± 10.86	42.28 ± 10.60
CRP (mmol/L)	3.62 ± 6.49	5.34 ± 14.85	5.11 ± 14.72	5.28 ± 6.85
WBC (10^3/µl)	8.14 ± 2.91	8.94 ± 3.21	9.05 ± 3.25	7.58 ± 2.28
Systolic BP (mmHg)	165.00 ± 34.06	147.94 ± 30.14	150.27 ± 31.36	148.33 ± 28.13
Diastolic BP (mmHg)	96.88 ± 17.78	88.99 ± 15.00	89.77 ± 15.70	89.44 ± 13.92
DWI lesion volume (Cm^3) †	8.85 ± 8.98* (6.33, 0.56 - 18.56)	2.70 ± 4.81 (0.39, 0.13 - 3.30)	2.90 ± 5.30* (0.37, 0.12 - 3.27)	6.14 ± 7.14 (2.81, 1.11 - 9.74)
N of injured regions	2.00 ± 1.41*	1.11 ± 0.73	1.02 ± 0.55**	2.78 ± 1.31

†Median and interquartile range are presented. END, early neurological deterioration; NIHSS, National Institute of Health stroke scale; min, minute(s); IV, intra-venous; IA, intra-arterial; BA, basilar artery; VA, vertebral artery; PCA, posterior cerebral artery; LAA, large artery atherosclerosis; CE, cardioembolism; SVO, small vessel occlusion; UDE, undetermined etiology; ODE, other determined etiology; TIA, transient ischemic attack; CHD, coronary heart disease; mmol, millimole(s); L, liter(s); LDL, low-density lipoprotein; TG, triglycerides; HDL, high-density lipoprotein; CRP, C-reactive protein; WBC, white blood cell; µl, microliter(s); BP, blood pressure; mmHg, millimeter(s) of mercury; DWI, diffusion-weighted imaging; Cm, centimeter(s); N, number. The Chi-square test (for categorical variables) and the Mann-Whitney U test (for continuous variables) were used to assess the significance of inter-group differences. *P < 0.05, **P < 0.01

Results

Among 118 patients with hyperacute posterior circulation ischemic stroke, all patients (including 39 females, mean age = 62.24 ± 13.57 years) underwent DWI within the hyperacute period and subsequent MRI with angiography in a few days after the onset of stroke. Mean time interval between the onset of symptoms to emergency department was 145 ± 101 minutes and the interval between the arrival time at emergency department and the time DWI had been taken was 74 ± 149 minutes (median= 39, interquartile range= 28 - 72). In moderate to severe hyperacute posterior circulation ischemic stroke patients, targets

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Table 2: Association between factors representing clinical severity/outcome and diffusion-weighted imaging lesion features in stroke patients

	Total lesion volume†		N of injured parts				
			Total†		≤ 1 versus ≥ 2 ‡		
	r	*P*	*r*	*P*	≤ 1 (Mean ± SD) (Median, IQR)	≥ 2 (Mean ± SD) (Median, IQR)	*P*
Initial NIHSS	0.112	0.225	0.535	<0.001*	2.36 ± 3.040 (2.00, 0.75 - 3.00)	11.93 ± 9.189 (11.50, 4.50 - 21.25)	<0.001*
NIHSS at 3 months	0.042	0.674	0.390	<0.001*	0.75 ± 0.980 (0, 0 - 1.00)	9.10 ± 13.674 (3.00, 1.00 - 17.50)	<0.001*
mRS at 3 months	0.133	0.165	0.480	<0.001*	0.83 ± 1.043	2.75 ± 1.984	<0.001*

N, number; r, correlation coefficient; SD, standard deviation; IQR, interquartile range; NIHSS, national institute of health stroke scale; mRS, modified Rankin scale. † Spearman's correlation test was performed. ‡ The Mann-Whitney U test was performed for analysis of intergroup differences. *P < 0.05

for thrombolytic treatment, time from the arrival at emergency department to DWI acquisition was 30 ± 11 minutes.

Lesions were detected by DWI in 108 patients, while the lesions for the remaining ten patients were detected by subsequent T2-weighted imaging. Among the 118 patients, 12 patients had no records regarding END and eight patients had no records regarding favorable outcome. Therefore, the study population consisted of 105 patients for the evaluation of END within seven days from the onset of stroke symptoms and 110 patients for the evaluation of a favorable outcome three months later.

Table 1 summarizes the demographics and clinical characteristics for stroke patients with or without END and a favorable outcome and illustrates the significant differences between patients' NIHSS scores measured at the time of presentation. Hypertension (P = 0.003), atrial fibrillation (P = 0.011) and a previous history of stroke/TIA (P = 0.003) were significantly associated with END. Stroke subtype (small vessel occlusion) (P = 0.020), diabetes (P = 0.001), atrial fibrillation (P = 0.002) and a previous history of stroke/TIA (P = 0.019) were significantly associated with favorable outcome and thrombolytic therapy was associated with unfavorable outcome (P < 0.001). There were statistically significant differences in the total volume of lesions between patients developing or not developing with END (P = 0.005). The total lesion volume was negatively associated with a favorable outcome (P = 0.001). The association between lesion features assessed by DWI (lesion volume and the number of injured regions) and clinical variables are summarized in Table 2. The number of injured regions positively correlated with the END and negatively correlated with a favorable outcome (Figure 1). By comparing patients with one or no lesion and patients with two or more lesions, the patients with two or more lesions were shown to have significantly higher initial NIHSS scores and a higher mRS score at three months. However, neither the initial NIHSS scores, NIHSS scores at three months, nor mRS scores at three months correlated with the total lesion volume. Interestingly, four among

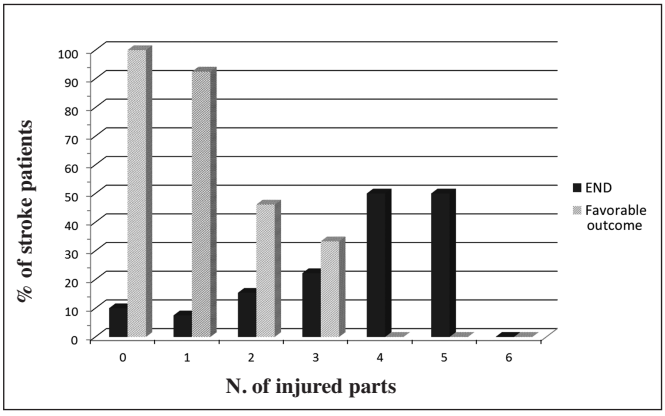

Figure 1: *Bar graph depicting the proportion of posterior circulation ischemic stroke patients that exhibited END or a favorable outcome with respect to the number of injured anatomical regions.*

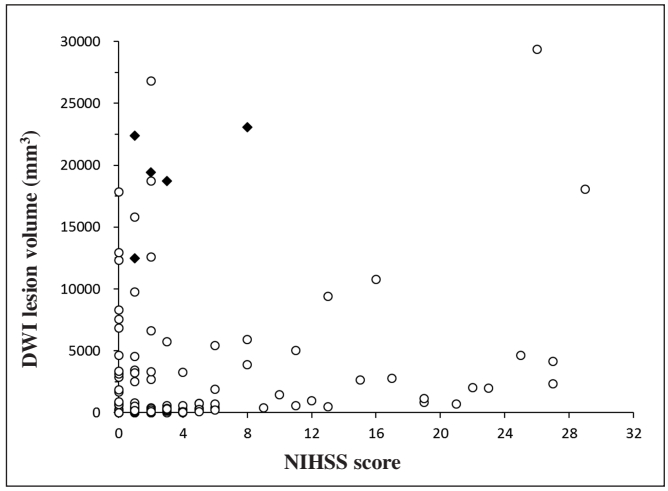

Figure 2: *Scatter plot depicting the correlation between DWI lesion volumes and the corresponding NIHSS scores in 118 patients with hyperacute posterior circulation ischemic stroke. Five of the 118 patients underwent decompressive craniectomy due to symptom aggravation (black diamonds).*

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Table 3: Logistic regression of lesion locations for END and a favorable outcome

Lesion location	END†			Favorable outcome‡		
	OR	95% CI	*p*	OR	95% CI	*p*
Supratentorium	0.400	0.064 - 2.522	0.330	21.122	0.289- 1542.705	0.164
Thalamus	0.108	0.010 - 1.103	0.060	0.345	0.018 - 6.765	0.483
Midbrain	0.843	0.066 - 10.813	0.896	0.392	0.016 - 9.411	0.563
Pons	8.712	1.700 - 44.645	0.009*	0.639	0.038 - 10.625	0.755
Medulla	1.523	0.179 - 12.962	0.700	0.112	0.003 - 4.438	0.244
Cerebellum	1.014	0.216 - 4.751	0.986	2.322	0.136 - 39.760	0.561

END, early neurological deterioration; OR, odds ratio; CI, confidence interval. † Variables are adjusted for age, NIHSS at day 1, hypertension, history of Stroke/TIA, atrial fibrillation and total number of injured locations. ‡ Variables are adjusted for age, NIHSS at day 1, thrombolysis, major vessel occlusion, stroke subtype (SVO), diabetes, history of stroke/TIA, atrial fibrillation and total number of injured locations. *P < 0.05

five patients who underwent decompressive craniectomy showed initial NIHSS scores of less than 4, whereas lesion volumes assessed by DWI were larger than 10,000 mm^3 (Figure 2). Multivariate logistic regression analyses were used to identify independent predictors for END or a favorable outcome. It demonstrated that pons lesions independently associated with END (OR 8.712, 95% CI 1.700 - 44.645, P = 0.009) after adjusting for covariates (Table 3). No specific location of lesions assessed by DWI was shown to function as independent predictors of favorable outcomes.

Discussion

In the present study, multiple lesions or a large total volume of lesions assessed by DWI were determined to be sufficient predictors of END. In addition, less lesions or a small total volume of lesions assessed by DWI were determined to be sufficient predictors of favorable outcomes in patients with hyperacute posterior circulation ischemic stroke, although these lesions did not significantly correlate with the initial neurological status measured by the NIHSS. Among the anatomical locations examined, presence of lesion in pons functioned as an independent predictor for END.

The rapid identification and evaluation of a patient's neurological status is critical for the proper management of acute ischemic stroke, such as with thrombolytic therapy. NIHSS was developed and validated for the assessment of clinical stroke severity.[16,17] It is a standard scale that is suitable for brief bedside use and can be performed by non-neurologists after training.[16,18] However, the NIHSS scale is weighted toward the assessment of anterior circulation stroke and does not reflect vague posterior circulation stroke-related symptoms, such as headache, vertigo or truncal ataxia.[2,11,19,20] The poor association between the lesion volume and the NIHSS score, reported here and in previous studies, could be a result of the limitations of the NIHSS as well as a relatively small functional deficit compared with the extent of lesions in the posterior circulation area.[11] However, patients with isolated posterior circulation stroke symptoms may worsen later. Among the patients with low initial NIHSS scores (<4), four patients underwent a decompressive craniectomy because of brain edema and severe symptom aggravation (Figure 1), and one of the four patients exhibited an unfavorable outcome. These patients were not given thrombolytic therapy, despite having arrived at the hospital within six hours from the onset of symptoms because their initial symptoms were not severe enough to warrant thrombolytic therapy, even though the initial lesion volumes assessed by DWI were relatively large for these patients. Therefore, patients with large, initial ischemic lesions assessed by DWI should be carefully monitored by a medical team, regardless of clinical severity, because of the possibility of early aggravation of neurological symptoms and brain edema. Furthermore, patients with hyperacute posterior circulation ischemic stroke who have had large ischemic lesions assessed by DWI should be under vigorous monitoring for neurological worsening even if their initial symptoms are not severe.

To date, most centers have used non-contrast brain CT as a primary diagnostic imaging tool for the evaluation of hyperacute stroke; however, CT is not sufficiently sensitive for the detection of structures of posterior circulation.[10,12,21] DWI is more useful than CT for detecting posterior circulation ischemic stroke and identifying the extent and nature of ischemic stroke.[9,10] In the present study, we found that the more ischemic areas detected by DWI, the more chances of END and poor prognoses were given to hyperacute posterior circulation ischemic stroke patients. Notably, DWI showed considerable predictive power for assessing the need for decompressive craniectomy. In addition, patients with two or more injured anatomical regions were more likely to exhibit END and a less favorable outcome than patients with one or no lesions (Figure 2). These results provide evidence that the number of acute ischemic anatomical areas determined by DWI could help predict the worsening of the initial symptoms and the prognosis in a short time frame.

An analysis of lesion location demonstrated that a pontine lesion is a reliable independent predictor of END. It might be explained by many important tracts and nuclei concentrated with in a small area of pons; severe cases such as basilar atherosclerosis-related stroke often emerge as pontine infarctions with or without lesions in other regions during the hyperacute

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stage. Thus, the possibility of symptom aggravation in patients with hyperacute ischemic stroke in the pons region is very likely. The present study has some limitations because of its retrospective approach to data collection. For example, collecting data that resulted in a clinical or stroke outcome was not always possible. Additionally, a lack of patients with large lesion volume and higher NIHSS scores might have influenced the outcome of our analyses. The volumetric assessment may lack precision because ischemic lesions located in the posterior fossa are typically small and scattered, which allows for distortion during the measuring process. Nevertheless, it is a useful assay because intra-observer reliability is high and the association between lesion number and volume and clinical outcome is significant. Magnetic resonance scanners with different magnetic field strengths were another limitation because the detection rate of acute ischemic lesion on DWI by 3.0 Tesla MR equipment is superior to that by 1.5 Tesla MR equipment. Finally, we presented ten patients (8.47%) with stroke symptoms that initially displayed no restricted diffusion; it is possible that the duration was too short to generate cytotoxic edema, even though hypoperfusion and acute neurological deficits have appeared.[22] In this case, it would be useful to have performed additional imaging for identifying acute infarction. Furthermore, although all ten patients showed a favorable outcome, false negatives should be avoided whenever possible, even if no lesions were initially found by DWI in patients diagnosed with a posterior circulation infarction.

In conclusion, the clinical outcome of the hyperacute posterior circulation ischemic stroke could be predicted based on the total lesion volume and/or the number of injured anatomical regions by using DWI. These results place importance on the role of DWI as an effective diagnostic tool for assessing the severity of lesions in patients with hyperacute posterior circulation ischemic stroke.

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