Topographic Location of Acute Pontine Infarction Is Associated With the Development of Progressive Motor Deficits

Semi Oh, MD; Oh Young Bang, MD, PhD; Chin-Sang Chung, MD, PhD; Kwang Ho Lee, MD, PhD; Won Hyuk Chang, MD, PhD; Gyeong-Moon Kim, MD, PhD

Background and Purpose—Motor weakness progression is relatively common in acute pontine infarction and frequently associated with increased functional disability. We designed this study to identify the predictors of progression of motor weakness in patients with pontine infarction during the acute phase.

Methods—We identified consecutive patients with acute ischemic stroke in the pons. Patients were defined as having progressive motor deficits (PMD) if their motor National Institutes of Health Stroke Scale scores increased by ≥1 unit between the maximal and initial neurological deficits. To define the predictors of PMD in patients with a pontine infarct, clinical, laboratory, diffusion-weighted imaging lesion location, and magnetic resonance angiographic variables were investigated.

Results—A total of 190 patients (male:female=112:78, 66.4±10.6) were identified, and 49 (25.8%) patients were diagnosed with progressive motor deficits. Logistic multiple regression analysis identified lesion involvement of the lower pons (odds ratio, 3.768; 95% confidence interval, 1.696–8.371) as an independent risk factor contributing to motor progression. Although 34 patients (17.9%) had significant basilar artery stenosis, there was no relationship between PMD in pontine infarct patients and the presence of basilar artery stenosis. Additionally, female and previous hypertension were associated with PMD (odds ratio, 2.651, 95% confidence interval, 1.211–5.802; odds ratio, 3.051, 95% confidence interval, 1.087–9.673).

Conclusions—Our results suggest that lower pons lesions may contribute to progressive motor deficits in patients with isolated acute pontine infarction. Infarct topography is therefore a potential prognostic factor of PMD, because the location of the infarct can affect the extent of ischemic degeneration of the corticospinal tract. (Stroke. 2012;43:708-713.)

Key Words: acute stroke ▪ brain stem stroke ▪ diffusion-weighted imaging ▪ prognosis ▪ stroke in evolution

Worsening of neurological symptoms after acute cerebrovascular disease is associated with increased mortality and more severe functional disabilities. The frequency of deterioration has been reported to range between 23.9% to 31% in prior studies that investigated neurological symptoms 24 hours from onset. Neurological deterioration could occur early after acute cerebrovascular disease onset (generally within 72 hours from onset) because of disruption of function in the infarcted area, or it could be delayed due to systemic causes. Classic mechanisms underlying neurological worsening include a propagating thrombus rising from an atheromatous lesion in the basilar artery, narrowing of arterial stenosis, development of brain edema, recurrent artery-to-artery embolism, or failure of collateral circulation. Pontine infarctions account for about 7% of all ischemic strokes. Unilateral pontine infarctions usually manifest with lacunar syndromes, including pure motor stroke, ataxic hemiparesis, or dysarthria–clumsy hand. Progression of motor weakness is relatively common in acute pontine infarction cases and is frequently associated with increased functional disability. Recently, basilar artery (BA) stenosis was reported to be related to the subacute increase in lesion volume seen in pontine infarctions but was not correlated with clinical outcomes. Factors that affect the progression of clinical motor weakness are largely unknown. Therefore, we evaluated potential predictors of progression of motor weakness in pontine infarction cases during the acute phase, such as the presence of BA stenosis and the location of the
infarction. The results presented in the present report could lead to strategies to predict stroke extent and improve clinical outcomes.

**Methods**

**Patient Selection**

We analyzed demographic, clinical, laboratory, and radiographic data collected prospectively from consecutive patients admitted to the Stroke Center of Samsung Medical Center for acute cerebral infarction between January 2002 and December 2009.

Inclusion criteria for this study were (1) admittance to the Stroke Center within 48 hours of the onset of symptoms, (2) acute ischemic lesions within the unilateral pons on diffusion-weighted imaging (DWI), and (3) patients who presented with motor deficits during the admission period. The exclusion criteria were patients who (1) did not have DWI performed within 72 hours of initial presentation, (2) had cortical symptoms, and (3) had deterioration caused by extracerebral illnesses such as infections, bronchoaspirative pneumonia, dehydration, metabolic changes, hypotension, or respiratory or heart failure. Our institutional review board approved this study.

**Data Collection**

All patients underwent MRI (3.0T, Achieva, Philips Medical Systems), using a protocol that also included DWI, gradient-recalled echo, fluid-attenuated inversion recovery, and T1-weighted imaging (WI) angiography imaging of the cervical and intracranial vessels.

Demographic features and risk factors were investigated. Those included sex, age, hypertension (patient prescribed antihypertensive medication or had a blood pressure ≥140/90 mm Hg on repeated measurements 1 week after stroke onset), diabetes mellitus (patient taking diabetes medication or a fasting blood glucose level ≥126 mg/dL), hyperlipidemia (patient taking a cholesterol-reducing agent or a fasting cholesterol level ≥220 mg/dL), atrial fibrillation, and previous stroke or transient ischemic attack.

To assess clinical progression, the National Institutes of Health Stroke Scale (NIHSS) score was measured at the time of admission, discharge, and maximal neurological deficits if the maximal neurological deficits occurred between the admission and discharge dates. Patients were defined as having progressive motor deficits (PMD) if their motor NIHSS scores increased by ≥1 unit between the maximal and initial neurological deficits.15

After the onset of symptoms, the following information was recorded: antplatelet agent use, anticoagulation therapy (including anticoagulation plus antplatelet agents), and thrombolysis. All patients underwent vital signs check-ups, routine blood tests, electrocardiography, and cardiac telemetry for at least 24 hours to evaluate the presence of atrial fibrillation.

**Brain MRI and Magnetic Resonance Angiography Parameters**

DWI was obtained using 2 levels of diffusion sensitization (b values of 0 and 1000 s/mm², respectively; 5–7-mm slice thickness; no gap). We investigated the laterality and extent of the pontine DWI lesion. We divided the patients into 3 groups, based on the rostrocaudal location of the observed DWI lesion: (1) upper (rostral) pons, characterized by a relatively round shape with a small, round-shaped aqueduct; (2) middle pons, characterized by its square-shaped fourth ventricle, large middle cerebellar peduncles, and silhouettes of trigeminal nerves; and (3) lower (caudal) pons, characterized by its similar shape to the middle pons but with images of facial/acoustic nerves and grooves instead of trigeminal nerves.12,16 We also divided the patients into 2 groups according to axial lesion location and extent: (1) paramedian pontine infarcts and (2) extended pontine infarcts. The mapping of lesions on the T1-MRI template and the visual comparative analysis of MRI data were achieved by using free software (MRICro, version 1.04 build 1). BA stenosis was defined as a reduction in the caliber of the basilar artery by at least 50% or occlusion of the basilar artery. The DWI lesion extent and the presence of BA stenosis were assessed by 2 neurologists who were blind to the MRI findings. If there was a discrepancy, a third neurologist’s opinion was obtained.

**Statistical Analysis**

Baseline characteristics and vascular risk factors associated with PMD were compared between those 2 groups. Student t test was used for continuous variables, and the χ² test or Fisher exact test was used for categorical variables. Multivariate analyses were performed using multiple logistic regression to determine independent factors associated with PMD. Variables from univariate analyses with probability values <0.2 were considered to represent explanatory variables and entered into multivariate analysis. A 2-sided probability value of <0.05 was considered statistically significant. All statistical analyses were performed with the use of SPSS version 15.0 software (SPSS, Chicago, IL).

**Results**

**General Characteristics of Patients**

Among 3700 patients admitted for acute ischemic stroke during the study period, 272 (7.3%) consecutive patients had acute ischemic pontine infarctions. A total of 190 consecutive patients (112 men and 78 women; age, 66.4 ± 10.6 years) with acute ischemic pontine infarctions were studied retrospectively. Reasons for exclusion of the other patients were as follows: presented 48 hours or later after stroke onset (n=33), did not have DWI performed within 72 hours of initial presentation (n=22), cortical symptoms (n=8), and deterioration caused by extracerebral illnesses (n=19).

Forty-nine patients (25.8%) had PMD, and 141 patients were assigned to the stable motor deficit group without PMD. Among the total of 190 patients, 116 patients (61.1%) were admitted within 24 hours. In the PMD group, 44 patients (89.8%) deteriorated within the first 4 days (median, 1 day; range, 1–9 days), especially 11 patients within 24 hours (Figure 1).

There was no difference in admission motor NIHSS between the 2 groups. An examination of the demographic data revealed that female sex was significantly more frequently related to PMD than male sex (P=0.013) (Table 1). More patients in the PMD group had hypertension than those in the group without PMD (P=0.027). The use of statin and antihypertensive agents were not related to the PMD.

Patients were initially treated as follows: 121 patients (63.7%) were given anticoagulants (heparin or warfarin), 60 patients (31.6%) were given antplatelet agents, 4 (2.1%) were given anticoagulants plus antplatelet agents, and 5 patients received thrombolytic agents. There were no differences in medications after admission between the 2 groups.

The patients stayed for a median of 9.5 days in the hospital. The mean length of hospital stay was significantly longer in the PMD group than without PMD group (21.8 versus 12.9 days, P<0.001). The PMD patients stayed for a median of 24 days (range, 4–42 days), and more PMD patients were transferred to the Department of Rehabilitation Medicine than those without PMD (73.4% versus 32.6%, P<0.001).

The level of C-reactive protein (P=0.022) was higher in patients with PMD than those without PMD. The other variables investigated showed no relationship to stroke phenotype.
Lower pontine lesions (51.8%) showed deterioration of neurological symptoms within the first 2 days from symptom onset.

**Topographic Classification**

DWI revealed upper pontine infarcts in 63 patients, mid pontine infarcts in 62, and lower pontine infarcts in 65 patients. Paramedian pontine infarcts were present in 102 patients, and extended pontine infarcts were present in 24 patients (Table 2). The incidence of lower pontine infarcts was significantly higher in the PMD group than in the non-PMD group (P=0.004); 26 patients (53.1%) in the PMD group had lower pontine infarcts, whereas 39 patients (78.4%) in the non-PMD group had lower pontine infarcts. In contrast, the incidence of upper pontine lesions was significantly higher in patients without PMD (P=0.028). DWI lesion overlap on MRI template suggested anteromedial location in lower pons has a probable association with PMD (Figure 2). There was no difference in DWI lesion volumes between the 2 groups (Table 2).

Forty-one patients (83.7%) in the PMD group and 99 patients (70.2%) in the non-PMD group had old lacunar infarctions in bilateral basal ganglia or cerebral hemispheres. The presence of old lacunar infarctions was not significantly related to the PMD (P=0.065).

Although significant BA stenosis was observed in 34 patients (17.7%), there was no relationship between the progression of motor deficits and the presence of BA stenosis.

In the PMD group, 17 patients (34.6%) received a repeat both MRI scan and CT scan, and 40 patients (81.6%) underwent only repeat CT scan. On follow-up CT or MRI, none of the patients with PMD had acute intracerebral hemorrhage or hemorrhagic transformation of the initial pontine infarction.

**Multivariate Analysis**

Multiple logistic regression analysis was conducted to further evaluate the independent predictors of PMD. Lower pontine infarctions and PMD were significantly associated (odds ratio, 3.768; 95% confidence interval, 1.696–8.371, P=0.001) after adjusting for covariates (Table 3). The sensitivity, specificity, positive predictive value, and negative predictive value of lower pontine lesions for predicting PMD in patients with acute pontine infarctions was 53.0%, 72.3%, 66.6%, and 81.6%, respectively. Additionally, female sex and previous hypertension were associated with PMD (odds ratio, 2.651; 95% confidence interval, 1.211–5.802; odds ratio, 3.051; 95% confidence interval, 1.087–9.673).

**Table 1.** Clinical Characteristics Between Patients With Progressive Motor Deficits and Those Without PMD in Acute Pontine Infarction

<table>
<thead>
<tr>
<th>Patients Without PMD (n=141), (%)</th>
<th>Patients With PMD (n=49), (%)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y, mean±SD</td>
<td>66.5±10.7</td>
<td>66.2±10.3</td>
</tr>
<tr>
<td>Female sex, n (%)</td>
<td>51 (36.2%)</td>
<td>27 (55.1%)</td>
</tr>
<tr>
<td>Stroke risk factors</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prior stroke or TIA</td>
<td>58 (41.1%)</td>
<td>20 (40.8%)</td>
</tr>
<tr>
<td>Previous hypertension</td>
<td>105 (74.5%)</td>
<td>44 (89.8%)</td>
</tr>
<tr>
<td>Known diabetes</td>
<td>69 (48.9%)</td>
<td>28 (57.1%)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>5 (3.5%)</td>
<td>1 (2.0%)</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>29 (20.6%)</td>
<td>12 (24.5%)</td>
</tr>
<tr>
<td>Medications before onset</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antihypertensive agents</td>
<td>62 (48.4%)</td>
<td>25 (56.8%)</td>
</tr>
<tr>
<td>Statin user</td>
<td>10 (7.7%)</td>
<td>6 (13.6%)</td>
</tr>
<tr>
<td>Initial treatment after admission</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antiplatelet agent</td>
<td>45 (31.9%)</td>
<td>15 (31.3%)</td>
</tr>
<tr>
<td>Anticoagulation plus</td>
<td>92 (67.2%)</td>
<td>33 (68.8%)</td>
</tr>
<tr>
<td>Motor NIHSS at admission, mean±SD</td>
<td>2.31±1.3</td>
<td>2.0±1.2</td>
</tr>
<tr>
<td>Hospital days</td>
<td>12.9±10.6</td>
<td>21.8±9.8</td>
</tr>
<tr>
<td>Transferred patients to rehabilitation</td>
<td>46 (32.6%)</td>
<td>36 (73.5%)</td>
</tr>
</tbody>
</table>

**Biochemical and clinical parameters**

<table>
<thead>
<tr>
<th></th>
<th>Patients Without PMD (n=141), mean±SD</th>
<th>Patients With PMD (n=49), mean±SD</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma glucose, mg/dL</td>
<td>160.6±77.4</td>
<td>156.3±67.0</td>
<td>0.915</td>
</tr>
<tr>
<td>Hematocrit, %, mean±SD</td>
<td>41.9±4.9</td>
<td>40.6±5.6</td>
<td>0.221</td>
</tr>
<tr>
<td>Leukocyte count, 10^3/mm³, mean±SD</td>
<td>7611±2820</td>
<td>8927±7589</td>
<td>0.271</td>
</tr>
<tr>
<td>Systolic BP, mm Hg, mean±SD</td>
<td>155.7±25.5</td>
<td>158.1±26.0</td>
<td>0.372</td>
</tr>
<tr>
<td>Body temperature, °C, mean±SD</td>
<td>36.1±0.4</td>
<td>36.3±0.5</td>
<td>0.258</td>
</tr>
<tr>
<td>Hemoglobin A1c, mean±SD</td>
<td>6.8±1.6</td>
<td>7.0±1.8</td>
<td>0.682</td>
</tr>
<tr>
<td>ESR, mm/h, mean±SD</td>
<td>23.1±19.8</td>
<td>29.7±25.9</td>
<td>0.212</td>
</tr>
<tr>
<td>CRP, mg/dL, mean±SD</td>
<td>0.59±1.4</td>
<td>1.14±2.1</td>
<td>0.022</td>
</tr>
<tr>
<td>Total cholesterol, mg/dL, mean±SD</td>
<td>190.1±42.1</td>
<td>182.3±47.0</td>
<td>0.652</td>
</tr>
</tbody>
</table>

Data represent mean±SD.

PMD indicates progressive motor deficits; TIA, transient ischemic attack; NIHSS, National Institutes of Health Stroke Scale; BP, blood pressure; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein.
Discussion
Progression of motor weakness is relatively common in patients with acute pontine infarctions. In the present study, 49 (25.8%) patients with acute pontine infarctions had progressive motor deficits, consistent with the previous study of Kim et al., who reported motor progression in 27% of patients with pontine infarctions.

Table 2. Neuroradiologic Parameters Between Patients With Progressive Motor Deficits and Those Without PMD in Acute Pontine Infarction

<table>
<thead>
<tr>
<th>Location of Infarction</th>
<th>Patients Without PMD (n=141), (%)</th>
<th>Patients With PMD (n=49), (%)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left</td>
<td>75 (53.2%)</td>
<td>31 (63.3%)</td>
<td>0.419</td>
</tr>
<tr>
<td>Lesion location, rostrocaudal</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upper</td>
<td>53 (37.6%)</td>
<td>10 (20.4%)</td>
<td>0.028</td>
</tr>
<tr>
<td>Middle</td>
<td>49 (34.8%)</td>
<td>13 (26.5%)</td>
<td>0.290</td>
</tr>
<tr>
<td>Lower</td>
<td>39 (27.7%)</td>
<td>26 (53.1%)</td>
<td>0.001</td>
</tr>
<tr>
<td>Lesion extent, transverse in axial plane</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paramedian</td>
<td>72 (51.1%)</td>
<td>30 (61.2%)</td>
<td>0.425</td>
</tr>
<tr>
<td>Extended</td>
<td>18 (12.8%)</td>
<td>6 (26.5%)</td>
<td></td>
</tr>
<tr>
<td>DWI lesion volume, mL</td>
<td>0.95±0.86</td>
<td>0.93±0.79</td>
<td>0.854</td>
</tr>
<tr>
<td>Basilar artery stenosis ≥50%</td>
<td></td>
<td>24 (17.0%)</td>
<td>0.594</td>
</tr>
<tr>
<td>Old lacunar infarctions*</td>
<td>99 (70.2%)</td>
<td>41 (83.7%)</td>
<td>0.065</td>
</tr>
</tbody>
</table>

PMD indicates progressive motor deficits; DWI, diffusion-weighted imaging.

*Old lacunar infarctions indicates multiple lacunar infarctions in both basal ganglia or cerebral hemispheres.

Table 3. Multivariate Logistic Regression Analysis of Predictors for PMD in Acute Pontine Infarction

<table>
<thead>
<tr>
<th>Estimated OR for PMD</th>
<th>Multivariate OR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female sex</td>
<td>2.651 (1.211–5.802)</td>
<td>0.015</td>
</tr>
<tr>
<td>Previous hypertension</td>
<td>3.051 (1.087–9.673)</td>
<td>0.048</td>
</tr>
<tr>
<td>Motor NIHSS, per 1 increment</td>
<td>0.786 (0.548–1.127)</td>
<td>0.100</td>
</tr>
<tr>
<td>C-reactive protein, per 1-mg/dL increment</td>
<td>1.194 (0.962–1.482)</td>
<td>0.107</td>
</tr>
<tr>
<td>Lower pontine lesion</td>
<td>3.768 (1.696–8.371)</td>
<td>0.001</td>
</tr>
<tr>
<td>Basilar artery stenosis ≥50%</td>
<td>0.805 (0.286–2.264)</td>
<td>0.681</td>
</tr>
<tr>
<td>Old lacunar infarctions*</td>
<td>0.894 (0.375–2.216)</td>
<td>0.799</td>
</tr>
</tbody>
</table>

PMD indicates progressive motor deficits; OR, odds ratio; CI, confidence interval; NIHSS, National Institutes of Health Stroke Scale.

*Old lacunar infarctions indicates multiple lacunar infarctions in both basal ganglia or cerebral hemispheres.

BA branch disease is the most common cause of stroke in patients with isolated pontine infarctions, with a relative frequency of about 40%18,19; furthermore, these patients have a worse prognosis than patients with lacunar pontine infarctions.20 Significant basilar artery stenosis, defined as reduction of the caliber of the basilar artery by at least 50% or occlusion of the basilar artery, was observed in 17.7% of our patients. However, the presence of BA stenosis was not related to PMD, even in patients (n=85, 44.5%) with BA luminal irregularities, including mild stenosis (<50%). This is consistent with a previous report that basilar artery disease is only related to the subacute increase in lesion volume of

Figure 2. MR images of patients with progressive motor deficits (PMD) or without PMD and frequency map of region of interest (ROI) of diffusion-weighted images (DWI) in acute pontine infarction. A 66-year-old male patient had an acute cerebral infarct in the lower pons detected by DWI (A). Basilar artery stenosis was not observed, and his motor deficit was deteriorated from MRC grade IV–I after 18 hours from the first symptom. A 73-year-old male patient had an acute upper to middle pontine infarct without basilar artery stenosis (C); his motor deficit improved during hospitalization. In PMD patients, the ROI density color bar image showed that the anteromedial area of the lower pons was more frequently involved than the upper or mid pons in PMD patients (n=26) (B). Otherwise, in non-PMD patients, the ROI density color bar image showed less frequent involvement of the lower pons (n=59) (D).
pontine infarctions and not to neurological progression.\textsuperscript{14} Thus, progression of motor deficits is unlikely to be caused by hemodynamic compromise related to BA stenosis.

We hypothesized that the stroke topography could be related to the PMD. Deep subcortical infarctions are known to have similar causes as pontine infarctions. The location of the infarction was found to be a predictor of motor progression in subcortical infarct patients.\textsuperscript{21,22} The involvement of the posterolateral striatum was related to early motor deterioration because of its anatomic proximity to the corticospinal tract.\textsuperscript{21,23} In the present study, lower pontine infarctions were significantly associated with PMD in patients with acute pontine infarctions. In pure motor pontine infarcts, the topography of the infarct lesion has been reported to be related to the prognosis; lesions causing severe hemiparesis were generally large and involved the ventral surface of the paramedian caudal or middle pons.\textsuperscript{12,24} Moreover, within the PMD group in our study, 14 patients with lower pontine lesions (51.8\%) showed deterioration of neurological symptoms within the first 2 days from symptom onset, suggesting that the PMD seen in those patients was related to a change in the infarcted area caused by the development of brain edema or an increase in infarct size.\textsuperscript{12,24} The diffusion tensor imaging and diffusion tensor tractography of the PMD patient more easily explained the more damage of the corticospinal tract than without PMD (Figure 3).

In the present study, being female was significantly more frequently related to PMD than being male, consistent with a previous study of Barber et al.\textsuperscript{25} who reported that a higher prevalence of female gender in the progressing stroke group. The sex effect in progressive stroke is still unsettled; however, several studies have shown that women have more severe strokes than men.\textsuperscript{26-28} That might be explained by estrogen withdrawal effects; estrogen has multiple vascular effects, all of which could contribute to salvage of neural tissue during ischemic episodes, and increases cerebral perfusion in women with or without known cerebrovascular disease.\textsuperscript{26-28} In addition to vascular effects, estrogen has anti-inflammatory effects that might be modulated by antioxidant and antiapoptotic effects.\textsuperscript{26,29} After menopause, there is an estrogen alteration,\textsuperscript{30} and the inflammation around the lesion, brain edema, and failure of collateral circulation could promote progression in pontine infarction patients. More research is needed to assess the relationship between gender effect and progressive stroke.

We also found that previous hypertension was associated with PMD in acute pontine infarction cases. Chronic hypertension may impair microvascular function and blood flow. This probably reduces the potential of the microvasculature to provide collateral circulation to ischemic areas. Progression could be related to decreased perfusion and/or decreased potential for rapid development of adequate collateral blood flow to ischemic zones.\textsuperscript{31} Further studies are required to investigate that relationship in more detail.

A progressive course after an acute stroke could be explained by the extension of ischemic edema due to propagating intra-arterial thrombi.\textsuperscript{32} Caplan\textsuperscript{33} suggested that patients with progressive stroke should be treated with aspirin or other agents that decrease platelet adhesion and aggregation. However, the effects of anticoagulation agents or aspirin

\begin{figure}
\centering
\includegraphics{stroke.ahajournals.org}
\caption{The diffusion tensor images (DTI) and tractography (DTT) of patients with progressive motor deficits (PMD) and without PMD. The DTI and DTT of the PMD patient involving the lower pontine lesion (A) showed more damage of the corticospinal tract than without PMD patient involving the upper pontine lesion (B). For the methods of diffusion tensor imaging and tractography, see online-only Supplemental Methods (http://stroke.ahajournals.org).}
\end{figure}
on the progressive or fluctuating course of stroke remain controversial. In our study, there was no difference in the initial management of patients who were later assigned to the PMD and non-PMD groups. Prospective, randomized studies are required to clarify the value of anticoagulation agents and aspirin in stroke patients.

The present study had several limitations. First, it was a retrospective study with a modest sample size. Neurological progression or an increase in lesion volume can occur in the very early stage of stroke; we may therefore have underestimated the rate of neurological progression. Second, we included patients who had acute cerebral infarct lesions in the unilateral pons. We may therefore have underestimated BA stenosis. Third, repeat MRI was not performed after deterioration in the majority of patients, and the reason for progression was not obvious on imaging studies. Future longitudinal studies based on diffusion tensor imaging could potentially help determine the extent of pyramidal tract damage and functional recovery.

In conclusion, lower pontine lesions were significantly associated with progressive motor deficits in acute pontine infarction patients. Infarct topography may therefore be a useful prognostic factor for progressive motor deficits, because the topology of the infarct may be related to ischemic degeneration of the corticospinal tract. Female sex and chronic hypertension also were significant predictors of PMD in acute pontine infarction patients in our study.

Disclosures

None.

References

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Data Supplement (unedited) at:
http://stroke.ahajournals.org/content/suppl/2012/02/17/STROKEAHA.111.632307.DC1
SUPPLEMENTAL MATERIAL

Methods

Diffusion tensor imaging and tractography

46 directional diffusion-weighted images were obtained using the single-shot EPI sequences (number of slices = 60, slice thickness = 2.25 mm, matrix size = 112 × 112, in-plane resolution = 1.96 mm × 1.96 mm). The 46 images consisted of 1 image volume acquired without diffusion gradients and 45 image volumes acquired with diffusion gradients along respective directions. In addition, a high-resolution T1-weighted structural image was acquired using a 3D gradient echo sequence (TR = 13.914 ms, TE = 6.89 ms, number of slices = 124 slices, slice thickness = 1.6 mm, matrix size = 512 × 512, in-plane resolution = 0.47 mm × 0.47 mm). We evaluated fiber connectivity using the FACT (fiber assignment by continuous tracking) technique with 3D-fiber reconstruction algorithm PRIDE® software ((Philips Medical Systems, Best, Netherlands). The termination criteria used for fiber tracking were FA < 0.2 and an angle change > 45°. To reconstruct the corticospinal tract (CST) we used the 2 region-of-interest (ROI) method. The analysis was started with an ROI in the motor cortex. A second ROI was drawn in the lower anterior pons. A logical AND-function was added so that only fibers passing through both the motor cortex and basis pontis were considered for further analysis. Fibers that went to the cerebellum were excluded by drawing ROIs in the superior cerebellar peduncle and the middle cerebellar peduncle and applying a logical NOT-function in conjunction with the other ROIs.  

1 2
References
