Topographic Patterns and Stroke Subtypes According to Progressive Motor Deficits in Lacunar Syndrome

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Although progressive hemiparesis occurs frequently in acute ischemic stroke, the topography and mechanisms associated with progressive motor deficit (PMD) remain unclear. The aim of this study was to identify the differences in PMD according to lesion location and the presumed underlying pathogenesis in patients with lacunar motor syndrome. Consecutive patients experiencing acute lacunar motor syndrome within 24 hours of stroke onset were included. Topographic patterns, risk factors, and presumed stroke mechanisms were compared between patients with PMD and those without PMD. Of the 168 patients in the study group, 47 (28.0%) had PMD. Baseline National Institutes of Health Stroke Scale score ($P = .034$) and female sex ($P = .005$) were associated with PMD on univariate analysis. Deep perforating artery infarct was more frequently associated with PMD (35.8%) compared with large artery disease (27.3%) and cardioembolism (5.3%). Multiple logistic analysis found that deep perforating artery infarct was independently associated with PMD (odds ratio, 2.87; 95% confidence interval, 1.26-6.5; $P = .012$). Deep perforating artery infarct is the major cause of PMD. In patients with lacunar syndrome, the pattern of PMD varies according to the location and etiology of stroke.
Key Words: Progressive motor deficit—lacunar syndrome—stroke subtype—topography.

Neurologic deficits frequently progress during the acute phase of cerebral infarct. Progressive neurologic deficit has been reported in 16%-43% of patients, depending on the diagnostic criteria for neurologic progression and the time interval from onset of symptoms to evaluation.[1-6] There is a lack of standardization in the terminology and assessment of progressing stroke, however,[7] with various mechanisms of neurologic worsening, including progression of focal ischemia, brain edema, systemic infection, and general medical deteriorations, considered neurologic progression in different reports. Although investigation of progressive motor deficit (PMD) has an advantage over the study of broad range neurologic deterioration in identifying the mechanism of stroke progression, PMD has been investigated only rarely.[8-12] Most studies of progressive hemiparesis have been conducted in patients with lacunar infarct, and lacunar stroke caused by small-vessel disease is known to be the major cause of PMD.[8] The aim of the present study was to identify the differences in PMD according to lesion location and the presumed underlying pathogenesis.

Patients and Methods

Between January 2005 and September 2007, we retrospectively studied consecutive patients with acute

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ischemic stroke from a prospectively collected acute stroke registry. The patients included in this study had suffered acute lacunar motor syndrome with motor deficits, with symptom onset in the preceding 24 hours, and had no cortical symptoms, such as aphasia, neglect, visual field defect, apraxia, or decreased level of consciousness. All patients had an acute infarction detected by diffusion-weighted magnetic resonance imaging (DWI) and underwent brain magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) covering the cerebral and carotid arteries within 3 days of admission.

MRI examination was performed using a 1.5-T scanner (Intera; Philips Medical Systems, Best, The Netherlands). Axial and sagittal T1-weighted spin-echo (repetition time [TR], 420 ms; echo time [TE], 11 ms), axial T2-weighted fast spin-echo (TR, 4000 ms; TE, 100 ms), axial fluid attenuation inversion recovery (FLAIR) (TR, 6000 ms; TE, 120 ms), and DWI (b-values, 0 and 1000 s/mm^2; TR, 6000 ms; TE, 74 ms) were included in the routine MRI protocol.

Each patient's medical history, general physical and neurologic examination findings, and laboratory test results were reviewed. The following risk factors were considered: hypertension, defined as having received antihypertensive medication or a blood pressure >140/90 mm Hg on repeated measurement after 1 week of stroke onset; diabetes mellitus, defined as taking diabetes medication or a fasting blood glucose level ≥126 mg/dL (7.0 mmol/L); current cigarette smoking; and hypercholesterolemia, defined as receiving a cholesterol-reducing agent or a fasting cholesterol level ≥220 mg/dL (5.69 mmol/L). Cardioembolism was considered in patients with a high- or medium-risk source of cardioembolism according to the Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification.[13] Electrocardiography and transthoracic echocardiography were performed in all patients.

DWI lesion patterns were analyzed by an investigator (P.-W.C) blinded to clinical and MRA data. Infarct topography was classified according to the criteria of Tatu and colleagues.[14,15] DWI lesion patterns were divided into cortical, subcortical, and multiple (cortical and subcortical) infarctions. Striatocapsular infarcts encompassed infarcts confined to the lenticular nucleus or internal capsule, as well as those partially extending into the corona radiata. Corona radiata infarcts was defined as infarcts strictly confined to the subcortical corona radiata. Each patient's National Institutes of Health Stroke Scale (NIHSS) score[16] was recorded at admission as well as on days 1, 2, 3, 4, 5, and 7 and on the day of discharge. Any change in NIHSS score between recording days was noted. The NIHSS was scored by neurologists certified in NIHSS, and each patient was examined by the same investigator during follow-up. The presumed mechanisms of the ischemic strokes were classified into 5 categories according to the modification of TOAST classification. Large-artery atherosclerosis was defined if there was corresponding artery stenosis (>50%) with no evidence of cardioembolism. Cardioembolism was diagnosed if emboligenic heart disease was detected according to the TOAST stroke classification criteria. Perforating artery disease was diagnosed if there was an infarct confined to the area of the perforating artery in the brainstem and basal ganglia with no evidence of large-artery disease or cardioembolism, regardless of lesion size. Perforating artery disease was further divided into 2 groups according to lesion size: small deep infarct for lesions <20 mm and large deep infarction for lesions located in the basal ganglia, internal capsule, or corona radiata with a diameter of 20-40 mm.[17]

The study population was divided into 2 groups according to the course of stroke: patients with PMD and those without PMD. PMD was defined as an increase of at least 2 points on the motor item of the NIHSS score persisting for at least 24 hours within 5 days of stroke onset. The study design was approved by Kangbuk Samsung Hospital's Institutional Review Board.

Statistical analyses were performed to identify the topographic and etiologic differences between the patients with PMD and those without PMD. Baseline characteristics and vascular risk factors associated with motor progression also were compared between the 2 groups. The Student t test was used for continuous variables, and the χ^2 or Fisher's exact test was used for noncontinuous variables. Multivariate analyses were performed with multiple logistic regression to determine independent factors associated with PMD. Factors including age, sex, hypertension, hypercholesterolemia, diabetes, admission NIHSS score, and previous stroke were included in the multivariate analysis. All statistical analyses were performed with SPSS version 10.0 (SPSS, Chicago, IL).

Results

Of the 603 patients with acute ischemic stroke who were admitted to our neurology clinic during the study period, 168 met the inclusion criteria. The study group comprised 97 men (57.7%) and 71 women (42.3%), ranging in age from 29 to 91 years (mean age, 64.9 ± 12.3 years). Forty-seven patients (28.0%) had PMD, and 121 patients were in the stable motor deficit group without PMD.

An examination of the demographic data shows that female sex was more frequently related to PMD than male sex (Table 1). Cardioembolic sources were more frequent in the patients without PMD. However, comparison of other vascular risk factors demonstrated no significant differences between the patients with PMD and those without PMD. There also was no difference in admission delay after stroke onset between the 2 groups. The initial NIHSS score was higher in the patients with PMD (4.1 ± 2.4 vs 3.3 ± 2.3; $P = .034$).