Arterial Stiffness and Progressive Neurological Deficit in Patients With Acute Deep Subcortical Infarction

Naoki Saji, MD, PhD; Kazumi Kimura, MD, PhD; Toshitaka Kawarai, MD, PhD; Hirotaka Shimizu, MD, PhD; Yasushi Kita, MD

Background and Purpose—The mechanism of progressive neurological deficit (PND) in patients with ischemic stroke remains unclear. The aim of this study was to clarify whether arterial stiffness, a marker of vascular endothelial impairment and atherosclerosis, is associated with PND in patients with acute deep subcortical infarction.

Methods—We evaluated 156 consecutive first-ever ischemic stroke patients with acute deep subcortical infarction. PND was defined as an increment of ≥2 points in the National Institute of Health Stroke Scale score or an increase of ≥1 point in the limb weakness score within 7 days of stroke onset. Patients were assessed for risk factors, and infarct size was measured on initial diffusion-weighted magnetic resonance imaging. We measured brachial-ankle pulse wave velocity (baPWV) as a marker of arterial stiffness. We divided patients into 2 groups according to the presence or absence of PND to compare their clinical characteristics.

Results—Fifty-two patients (33%) had PND, and baPWV was significantly higher in patients with than in those without PND. The baPWV cut-off value for PND was 18.24 m/s, with 90% sensitivity and 47% specificity. In multivariable logistic regression analysis, high baPWV (≥18.24 m/s; odds ratio, 8.22; 95% confidence interval, 2.55–31.9), large infarct size (≥15 mm; odds ratio, 2.76; 95% confidence interval, 1.01–7.92), and ≥3 infarct slices on serial axial diffusion-weighted imaging (odds ratio, 3.38; 95% confidence interval, 1.22–10.0) were independently associated with PND.

Conclusions—Arterial stiffness indicated by baPWV is independently associated with PND in patients with acute deep subcortical infarction. (Stroke. 2012;43:00-00.)

Key Words: acute stroke • arterial stiffness • atherosclerosis • blood–brain barrier • deep subcortical infarct • progressive neurological deficit • pulse wave velocity

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Methods—We evaluated 156 consecutive first-ever ischemic stroke patients with acute deep subcortical infarction. PND was defined as an increment of ≥2 points in the National Institute of Health Stroke Scale score or an increase of ≥1 point in the limb weakness score within 7 days of stroke onset. Patients were assessed for risk factors, and infarct size was measured on initial diffusion-weighted magnetic resonance imaging. We measured brachial-ankle pulse wave velocity (baPWV) as a marker of arterial stiffness. We divided patients into 2 groups according to the presence or absence of PND to compare their clinical characteristics.

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Key Words: acute stroke • arterial stiffness • atherosclerosis • blood–brain barrier • deep subcortical infarct • progressive neurological deficit • pulse wave velocity

Deep subcortical infarction sometimes presents with progressive neurological deficit (PND). The etiology of PND is the occlusion of perforating arteries attributable to cerebral small vessel disease.1–8 Small vessel disease is associated with arterial stiffness, a marker of both atherosclerosis and vascular endothelial impairment, and can cause neurological damage.8–13 A noninvasive measurement of arterial stiffness, brachial-ankle pulse wave velocity (baPWV), is independently associated with cerebral small vessel disease such as silent lacunar infarction12 and white matter disease.13

The mechanism of PND in patients with acute deep subcortical infarcts is still unclear. Arterial stiffness indicates vascular endothelial impairment, which causes blood–brain barrier failure and leads to neurological damage.8 This mechanism could increase the risk of PND. Our hypothesis was that arterial stiffness is independently associated with PND in patients with acute deep subcortical infarcts. To test this hypothesis, we investigated the association between arterial stiffness, conventional risk factors, and PND in patients with a first-ever deep subcortical infarct.

Materials and Methods

This study was approved by the Institutional Review Board at the Hyogo Brain and Heart Center at Himeji. Between October 2003 and March 2010, we enrolled 156 consecutive patients presenting with clinical lacunar stroke syndrome who were admitted to our hospital within 48 hours after stroke onset. All patients had a first-ever ischemic stroke attributable to an acute deep subcortical infarct detected on initial diffusion-weighted imaging.4 Informed consent was obtained from all patients.

Infarcts <15 mm in diameter on diffusion-weighted imaging scans were considered small infarcts and those >15 mm were considered large infarcts.5 Infarct slice number was first defined as the number of slices accompanied by visible infarcts on serial axial diffusion-weighted imaging scans and then used to evaluate the vertical extension of those infarcts.3 We measured baPWV at 7 days after the onset or stabilization of the neurological deficit using an oscillometric device (Form PWV/ABI; Omron Colin, Tokyo, Japan).10–13 PND was defined as an increase of ≥2 points in the National Institutes of Health Stroke Scale score or increase of ≥1 point in limb weakness in the National Institutes of Health Stroke Scale score during the 7 days after stroke onset.3–7 Excellent outcome after stroke onset was defined as a modified Rankin Scale score of 0 to 1 at discharge.9 Univariable and multivariable logistic regression models were used to identify the variables independently associated with PND. Odds
ratios are presented along with 95% confidence intervals. All comparisons were 2-tailed and $P < 0.05$ was considered significant. Detailed and Methods are provided in the Online Supplement.

**Results**

Fifty-two patients (33%) had PND. The average time to detect PND from the onset of ischemic stroke was 24 hours (interquartile range, 17–30 hours). Table 1 summarizes the characteristics of these patients. The baPWV cut-off value to detect PND was $18.24 \text{ m/s}$ with 90% sensitivity and 47% specificity. Patients with high baPWV ($\geq 18.24 \text{ m/s}$), those with large infarct ($\geq 15 \text{ mm}$), and those with infarct slice number $\geq 3$ had more PND than the rest of the patients (Figure).

In multivariable analysis (Table 2), high baPWV ($\geq 18.24 \text{ m/s}$), infarct size ($\geq 15 \text{ mm}$), and infarct slice number $\geq 3$ were significant predictors of PND.

![Figure](https://example.com/figure.png)

Figure: Association among progressive neurological deficit (PND) and infarct size, infarct slice number, and brachial-ankle pulse wave velocity (baPWV). Prevalence of PND in patients based on baPWV, infarct size, and infarct slice number. **A**, The baPWV ($< 18.24 \text{ m/s}$ and $\geq 18.24 \text{ m/s}$). **B**, Infarct size based on diffusion-weighted imaging (DWI) scans ($< 15 \text{ mm}$ and $\geq 15 \text{ mm}$). **C**, Infarct slice number ($< 3$ and $\geq 3$). The y-axis in each panel shows the prevalence of PND.

### Table 1. Characteristics of Patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>Present (n=52)</th>
<th>Absent (n=104)</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female, n (%)</td>
<td>26 (50)</td>
<td>35 (34)</td>
<td>0.057</td>
</tr>
<tr>
<td>Age, y</td>
<td>73 (12)</td>
<td>67 (11)</td>
<td>0.006</td>
</tr>
<tr>
<td>Body temperature, °C</td>
<td>36.4 (36.2 to 36.7)</td>
<td>36.4 (36.2 to 36.7)</td>
<td>0.273</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>163 (148–181)</td>
<td>161 (142–165)</td>
<td>0.044</td>
</tr>
<tr>
<td>NIHSS score on admission</td>
<td>4 (2–5)</td>
<td>2 (1–4)</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>mRS 0 to 1 at discharge, n (%)</td>
<td>6 (12)</td>
<td>74 (71)</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>mRS 4 to 5 at discharge, n (%)</td>
<td>32 (62)</td>
<td>8 (8)</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>Leukocytes, $\times 10^{+9}/\mu L$</td>
<td>60 (49–74)</td>
<td>63 (54–71)</td>
<td>0.519</td>
</tr>
<tr>
<td>CRP, mg/dL</td>
<td>0.1 (0–0.3)</td>
<td>0.1 (0–0.2)</td>
<td>0.355</td>
</tr>
<tr>
<td>eGFR, ml/min/1.73 m²</td>
<td>43.4 (30.1 to 51.7)</td>
<td>50.3 (40.4 to 62.2)</td>
<td>0.005</td>
</tr>
<tr>
<td>Serum glucose, mmol/L</td>
<td>66.0 (58.3 to 81.6)</td>
<td>66.6 (57.7 to 88.8)</td>
<td>0.916</td>
</tr>
<tr>
<td>baPWV, m/s</td>
<td>23.79 (19.37 to 27.61)</td>
<td>19.04 (19.37 to 27.61)</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>Time to MRI from onset, hour</td>
<td>20 (12–38)</td>
<td>20 (12–38)</td>
<td>0.976</td>
</tr>
<tr>
<td>DWI $\geq 15 \text{ mm}$, n (%)</td>
<td>26 (50)</td>
<td>18 (17)</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>Infarct slice No.</td>
<td>3 (3–4)</td>
<td>2 (1–3)</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>Stroke, n (%)</td>
<td>29 (56)</td>
<td>56 (54)</td>
<td>0.866</td>
</tr>
<tr>
<td>WMD, n (%)</td>
<td>36 (69)</td>
<td>62 (60)</td>
<td>0.293</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>47 (90)</td>
<td>88 (85)</td>
<td>0.456</td>
</tr>
<tr>
<td>Hypercholesterolemia, n (%)</td>
<td>27 (52)</td>
<td>54 (52)</td>
<td>1.000</td>
</tr>
<tr>
<td>Diabetes mellitus, n (%)</td>
<td>12 (23)</td>
<td>38 (37)</td>
<td>0.103</td>
</tr>
<tr>
<td>Ischemic heart disease, n (%)</td>
<td>2 (4)</td>
<td>13 (13)</td>
<td>0.147</td>
</tr>
<tr>
<td>Smoking, n (%)</td>
<td>16 (31)</td>
<td>52 (50)</td>
<td>0.026</td>
</tr>
<tr>
<td>Antihypertensives, n (%)</td>
<td>23 (44)</td>
<td>43 (41)</td>
<td>0.735</td>
</tr>
<tr>
<td>Statins, n (%)</td>
<td>13 (25)</td>
<td>20 (19)</td>
<td>0.413</td>
</tr>
<tr>
<td>Aspirin, n (%)</td>
<td>36 (30)</td>
<td>84 (70)</td>
<td>0.113</td>
</tr>
<tr>
<td>Cilostazol or clopidogrel, n (%)</td>
<td>15 (29)</td>
<td>17 (16)</td>
<td>0.092</td>
</tr>
<tr>
<td>Edaravone, n (%)†</td>
<td>33 (63)</td>
<td>70 (67)</td>
<td>0.720</td>
</tr>
<tr>
<td>Argatroban hydrate, n (%)*</td>
<td>25 (48)</td>
<td>26 (25)</td>
<td>0.006</td>
</tr>
<tr>
<td>tPA, n (%)</td>
<td>0 (0)</td>
<td>2 (2)</td>
<td>0.553</td>
</tr>
</tbody>
</table>

baPWV indicates brachial-ankle pulse wave velocity; CRP, C-reactive protein; DWI, diffusion-weighted imaging; eGFR, estimated glomerular filtration rate; MRI, magnetic resonance imaging; mRS, modified Rankin Scale; NIHSS, National Institutes of Health Stroke Scale; SBP, systolic blood pressure; SLI, silent lacunar infarction; tPA, tissue plasminogen activator; WMD, white matter disease.

*Free radical scavenger.

†Antithrombotic agent.
Discussion

The main finding of the present study was that arterial stiffness indicated by baPWV was independently associated with PND in patients with acute deep subcortical infarcts. The association between arterial stiffness and PND had not yet been reported before this study. Several mechanisms might explain this association. Vascular endothelial impairment, which is indicated by arterial stiffness,11–13 causes blood–brain barrier dysfunction and leads to cerebral parenchyma damage,8,14 possibly inducing PND. Decreased production of protective factors such as nitric oxide and endogenous tissue plasminogen activator,7,15 lack of collateral blood flow,1 arteriosclerotic microcirculatory impairment,16 and stepwise occlusion of the cerebral perforating arteries3–7 may accelerate endothelial dysfunction. However, high baPWV could be merely a result of high blood pressure in the acute phase of ischemic stroke.11

The cut-off value of baPWV for PND (18.24 m/s) in the present study was the same as the cut-off of baPWV for cerebral small vessel disease shown in previous studies (cerebral small vessel disease, 17 m/s;13 silent lacunar infarction, 17.24 m/s;12 and white matter disease, 18.29 m/s13).

These results support the hypothesis that baPWV is a marker for cerebral microvascular damage.10–13

Arterial stiffness is independently associated with functional outcome after stroke. High carotid-femoral PWV, a conventional measurement of arterial stiffness, is inversely associated with excellent functional outcome in patients with acute ischemic stroke.9 This finding is consistent with our results. The inverse association of baPWV with functional outcome is reasonable, because we also found an association between baPWV and PND.

There were several limitations of this study. A causal relationship between baPWV and PND could not be established, and selection bias was possible because this was an observational study performed in a hospital-based cohort. The low specificity (47%) of elevated baPWV to detect PND suggests that other mechanisms, such as neuroexcitotoxic amino acids, could also play a role in PND. This will need to be addressed in future studies.

Sources of Funding

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Disclosures

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References

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SUPPLEMENTAL MATERIAL

Arterial stiffness and progressive neurological deficit in patients with acute deep subcortical infarction
Supplemental methods

Patients
We enrolled patients with clinical lacunar stroke syndrome, with first-ever ischemic stroke due to an acute deep subcortical infarct. Patients who had a potential cardiac source of embolism such as atrial fibrillation, >50% stenosis of the extracranial carotid or vertebral arteries or intracranial artery occlusion were excluded. Patients who had cortical infarcts, border-zone infarcts or acute multiple infarcts were also excluded.

Assessment
Patients underwent a 1.5T MRI of the brain (Intera, Philips Medical Systems, Best, The Netherlands), including diffusion-weighted imaging (DWI), fluid-attenuated inversion recovery (FLAIR) imaging, and T2-weighted imaging (T2WI). Slice thickness was 0.7 mm. The image acquisition parameters were as described previously. The average time of MRI assessment from the onset of ischemic stroke was as follows: mean; 20 h, inter-quartile range (IQR); 10–38 h. Infarcts <15 mm in diameter on DWI scans were considered small infarcts and those >15 mm were considered large infarcts. Infarct slice number was defined as the number of brain slices with infarcts visible on DWI scans to evaluate the vertical extension of the infarcts. Mass effect due to edema and hemorrhagic transformation were evaluated on the initial and/or second MRI scans. Silent lacunar infarction (SLI) was defined as a focal lesion of at least 3 mm in diameter, with hyperintensity on T2WI and hypointensity on FLAIR. White matter disease (WMD) was defined as an irregular periventricular hyperintensity and/or early confluent or confluent separate deep white matter hyperintense lesion on T2WI and FLAIR, based on the rating scales for ischemic tissue damage. We measured the far wall common carotid artery intima–media thickness, ankle-brachial pressure index, and brachial-ankle pulse wave velocity (baPWV). Blood biochemical tests were performed on admission including leukocytes and serum C-reactive protein (CRP) as inflammatory markers in the acute-phase of stroke. Body temperature and serum glucose on admission were also recorded.

Risk factors
Hypertension was defined as a systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg and/or the use of antihypertensive agents. Hypercholesterolemia was defined as serum total cholesterol ≥ 5.69 mmol/L (220 mg/dL) and/or the use of statins. Diabetes mellitus was defined as hemoglobin A1c level ≥ 6.5% and/or the use of oral hypoglycemic agents or insulin, and/or a serum fasting glucose level ≥ 69.9 mol/L (126
mg/dL). Ischemic heart disease was defined as a history of physician-diagnosed angina pectoris, evidence of prior myocardial infarction or a prior coronary revascularization procedure (percutaneous coronary intervention or coronary artery bypass surgery). Current smoking habits were assessed on admission and estimated glomerular filtration rate (eGFR), a marker of renal function, was determined based on serum creatinine.

**Statistical Analysis**
Continuous variables are expressed as the mean ± standard deviation and were compared using unpaired Student’s t-tests. Variables exhibiting skewed distributions are expressed as medians and the inter-quartile range (IQR), and were compared using Wilcoxon rank-sum tests. Categorical variables are expressed as frequencies and percentages and were compared using $\chi^2$ tests. Receiver operating characteristic curve analysis was performed to determine the optimal cutoff values for the presence of progressive neurological deficit (PND). Potential variables with a $P< 0.10$ in univariable analysis were entered into a multivariable logistic regression model to identify the variables independently associated with PND. Odds ratios (ORs) are presented along with 95% confidence intervals (CIs). The data were analyzed using the JMP 8.0.2 software package (SAS Institute Inc., Cary, NC).
Supplemental results

A total of 1503 patients with acute ischemic stroke were admitted during the study period. Among 276 patients who met the inclusion criteria, we excluded 62 patients (24 did not have complete data; 20 had 3T MRI; and 18 were complicated by peripheral artery disease) and 58 patients who had infratentorial infarcts. The final analysis included 156 patients. Table S1 is the additional data of the characteristics of these patients. There were no patients who developed mass effect due to edema and hemorrhagic transformation on MRI scans. The distributions of the lacunar stroke syndrome were as follows: pure motor hemiparesis, 97 (62%); sensorimotor syndrome, 20 (13%); pure sensory syndrome, 16 (10%); dysarthria-clumsy hand, 9 (6%); ataxic hemiparesis, 8 (5%); and others, 6 (4%).

There were no significant differences in baPWV or the proportion of PND between 139 patients who were admitted within 24 h of onset and 17 patients who were admitted within 24–48 h (20.4 m/s vs. 20.8 m/s; 35% vs. 33%, respectively). Patients with high baPWV (≥18.24 m/s; 47/102 [46%] vs. 5/54 [9%]; relative risk 4.98, 95% CI 2.10–11.77, Figure 1A), those with a large infarct (≥15 mm; 26/44 [59%] vs. 26/112 [23%]; relative risk 2.55, 95% CI 1.68–3.86, Figure 1B), and those with infarct slice number ≥3 (40/80 [50%] vs. 12/76 [16%]; relative risk 3.17, 95% CI 1.80–5.56, Figure 1C) had more PND than the rest of the patients.
### Supplemental tables

#### Table S1. Additional data of patient characteristics

<table>
<thead>
<tr>
<th></th>
<th>Progressive neurological deficit</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Present (n = 52)</td>
<td>Absent (n = 104)</td>
<td></td>
</tr>
</tbody>
</table>
| DBP, mm Hg*              | 82 (81–86) | 83 (81–84) | 0.628  
| PP, mm Hg*               | 79 (63–85) | 78 (60–82) | 0.115  
| CCAIMT, mm               | 1.45 (1.10–1.78) | 1.40 (1.10–1.68) | 0.803  
| Ankle-brachial pressure index | 1.1 (1.05–1.16) | 1.12 (1.05–1.19) | 0.294  
| SBP, mm Hg†              | 149 (135–164) | 146 (135–158) | 0.271  
| DBP, mm Hg†              | 85 (79–92) | 87 (78–94) | 0.846  
| PP, mm Hg†               | 63 (55–75) | 59 (53–66) | 0.086  
| Days‡                    | 10 (8–15) | 7 (5–9) | <0.001  
| Calcium channel blocker, n (%) | 14 (27) | 19 (18) | 0.220  
| ACEI or ARB, n (%)       | 16 (31) | 31 (30) | 1.000  

*variables measured on admission; †variables measured at the time of baPWV measurement; ‡days from baPWV measurement to stroke onset.

Abbreviations: DBP, diastolic blood pressure; PP, pulse pressure; CRP, C-reactive protein; CCAIMT, intima–media thicknesses of the common carotid artery; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker.
Table S2. Association between high baPWV and PND

<table>
<thead>
<tr>
<th>Variables</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>baPWV, every 1 m/s increase†</td>
<td>1.15 (1.08–1.24)**</td>
</tr>
<tr>
<td>baPWV, every 1 m/s increase‡</td>
<td>1.14 (1.05–1.25)**</td>
</tr>
<tr>
<td>baPWV, ≥18.24 m/s†</td>
<td>8.37 (3.34–25.6)**</td>
</tr>
<tr>
<td>baPWV, ≥18.24 m/s§</td>
<td>8.22 (2.55–31.9)**</td>
</tr>
<tr>
<td>baPWV, ≥18.24 m/s∥</td>
<td>8.47 (2.52–34.5)**</td>
</tr>
</tbody>
</table>

PND was the dependent variable. *P< 0.01, **P< 0.001

†unadjusted OR.

‡adjusted OR for age and sex.

§adjusted OR for variables with P< 0.10 in univariate analysis: age ≥65 years, female sex, SBP ≥165 mm Hg, eGFR <48.7 mL/min/1.73m², infarct size ≥15 mm, infarct slice number ≥3, NIHSS ≥4 points, diabetes mellitus, ischemic heart disease, and smoking as shown in Table 2 (model 1).

∥adjusted OR for variables entered in the model 1, the use of cilostazol or clopidogrel (yes vs. no), and the use of argatroban hydrate (yes vs. no).
Table S3. Association between high baPWV and excellent functional outcome after ischemic stroke (mRS 0–1)

<table>
<thead>
<tr>
<th>Variables</th>
<th>OR</th>
<th>(95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>baPWV, every 1 m/s increase†</td>
<td>0.87</td>
<td>(0.81–0.93)***</td>
</tr>
<tr>
<td>baPWV, every 1 m/s increase‡</td>
<td>0.90</td>
<td>(0.83–0.97)**</td>
</tr>
<tr>
<td>baPWV, ≥18.24 m/s†</td>
<td>0.26</td>
<td>(0.12–0.52)***</td>
</tr>
<tr>
<td>baPWV, ≥18.24 m/s§</td>
<td>0.32</td>
<td>(0.11–0.86)*</td>
</tr>
<tr>
<td>baPWV, ≥18.24 m/s∥</td>
<td>0.32</td>
<td>(0.11–0.86)*</td>
</tr>
</tbody>
</table>

Modified Rankin Scale (mRS) 0 to 1 at discharge was the dependent variable. *P< 0.05, **P< 0.01, ***P< 0.001

†unadjusted OR.
‡adjusted OR for age and sex.
§adjusted OR for variables with P< 0.10 in univariate analysis: age ≥65 years, female sex, SBP ≥165 mm Hg, eGFR <48.7 mL/min/1.73m², infarct size ≥15 mm, infarct slice number ≥3, NIHSS ≥4 points, diabetes mellitus, ischemic heart disease, and smoking as shown in Table 2 (model 1).
∥adjusted OR for variables entered in the model 1, the use of cilostazol or clopidogrel (yes vs. no), and the use of argatroban hydrate (yes vs. no).
Supplemental references


