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Effect of blood pressure variability on early neurological deterioration in single small subcortical infarction with parental arterial disease

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ABSTRACT

Background and purpose: Early neurological deterioration (END) is not uncommon in acute single small subcortical infarct (SSSI), especially in those with parental arterial disease (PAD). The purpose of this study was to elucidate the effect of BP variability on the development of END as well as functional outcome at 90 days in SSSI and to determine whether the effect is linked to the status of parent artery.

Methods: Consecutive patients with acute SSSI were prospectively recruited from the First People's Hospital of Yangzhou between Aug 2013 and Jul 2016. END was defined as an NIHSS score increased ≥ 2 during the first 72 h compared with the initial NIHSS score. Functional outcome at 90 days after onset was assessed using the modified Rankin Score (mRS) and dichotomized as good (0–2) and poor (≥ 3). During this period, the parameters of BP variability such as $BP_{max–min}$, BP_{SD}, and BP_{CV} (equal to [SD × 100] / mean) were calculated.

Results: A total of 296 patients were included in the analysis. Of these, 30 (38.5%) SSSI associated with PAD and 53 (24.3%) without developed END respectively. Logistic regression analysis demonstrated that SBP_{max} (OR 1.036, 95% CI 1.005–1.069), SBP_{SD} (OR 1.177, 95% CI 1.021–1.356), SBPcv (OR 1.306, 95% CI 1.049–1.626), DBP_{max} (OR 1.141, 95% CI 1.042–1.250), $DBP_{max–min}$ (OR 1.085, 95% CI 1.015–1.160), DBP_{SD} (OR 1.369, 95% CI 1.032–1.816), and DBP_{CV} (OR 1.281, 95% CI 1.028–1.597) were all the independent predictors of END after acute SSSI associated with PAD. However, for those without PAD, none of the BP parameters was found significantly associated with END. Also, BP parameters were not related to the poor outcome at 90 days after onset.

Conclusions: Our study demonstrated that the acute in-hospital BP variability was associated with the development of END in patients with acute SSSI. However, its impact varies depending on the status of parent artery.

1. Introduction

Traditionally, single small subcortical infarction (SSSI) has been considered to be caused by small vessel disease and is pathologically characterized by fibrinoid degeneration or lipohyalinosis [1]. However, SSSI caused by atherosclerosis occurring in the parent artery is relatively common, especially in Asian populations where intracranial atherosclerosis is prevalent [2]. Generally, SSSI was considered to have a favorable outcome. However, SSSI caused by parent arterial disease (SSSIPAD) is reported to be more often associated with neurological progression or an unstable clinical course, which usually leads to unexpectedly severe disability and even death [3–5].

In the field of acute stroke, early neurological deterioration (END) remains an important unresolved practice problem, because as yet pathophysiology of deterioration is yet incompletely understood. Neither evidence-based nor consensus-based guidelines exist recommending how to prevent or halt END [6]. Efforts have been made to establishing presentation features that may help identifying patients at risk of deterioration [6–12]. Currently, hemodynamic factors have been proposed as one of possible mechanisms of progression after acute stroke [13,14]. As one of the hemodynamic factors, blood pressure (BP) variability was reported to be associated with vascular events, poor functional outcome and death during long-term follow-up [15,16]. There were also studies on the association between BP variability and early edema, lesion growth, symptomatic hemorrhagic transformation after acute stroke treated with thrombolytic agent [17–19]. Thus, we hypothesized that the BP variability might be associated with END as well as poor outcome at 90 days after acute SSSIPAD.

The purpose of this study was to elucidate the effect of acute in-hospital BP variability on the development of END and poor outcome at

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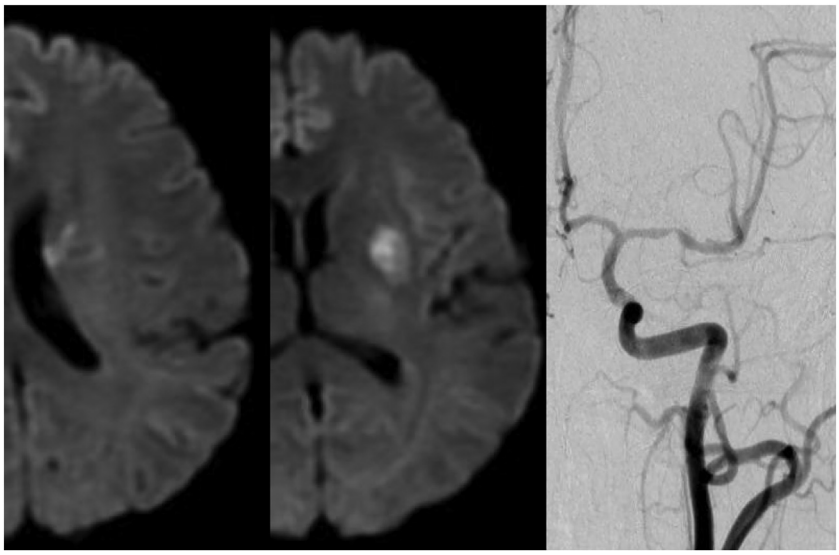

Fig. 1. Example of SSSIPAD: a 67 years old man, END was occurred during the first 17 h after onset.

90 days after onset in patients with acute SSSI and to determine whether the effect is linked to the status of parent artery.

2. Subjects and methods

2.1. Patient selection

Between Aug 2013 and Jul 2016, consecutive patients with acute ischemic stroke were prospectively registered from the First People's Hospital of Yangzhou. Patients were enrolled into our study if they fulfilled the inclusion criteria: (1) age older 18 years, (2) time from symptom onset to admission of 24 h or less, (3) magnetic resonance imaging (MRI) performed within 24 h after admission; (4) acute isolated infarction in the perforator territory of the MCA, (5) the largest diameter of lesion on axial diffusion-weighted imaging (DWI) ≤ 20 mm. We excluded patients who met the following criteria: (1) history of stroke, (2) patients with potential cardiac embolic sources, (3) treated with thrombolytic agents and interventional therapy, (4) significant stenosis (≥ 50%) in responsible extracranial large artery, (5) early discharge or had inadequate BP data, defined as BP measured fewer than 30 times during the first 72 h. The study was approved by Ethics Committees of the First People's Hospital of Yangzhou and written informed consent was obtained from each patient.

3. Clinical assessment and treatment

Detailed demographic, clinical and laboratory parameters were recorded and analyzed in our study. Hypertension was defined as blood pressure ≥ 140/90 mm Hg, or use of antihypertensive medications. Diabetes mellitus was defined as a fasting blood glucose ≥ 126 mg/dl, positive ≥ 75 g oral glucose tolerance test result, or use of insulin or oral hypoglycemic agents. Hyperlipidemia was defined as a serum total cholesterol level ≥ 240 mg/dl, or use of cholesterol-reducing medications. Current cigarette smoking was defined as current or quit smoking ≤ 6 months prior. Drinking was defined as intake > 80 g/day or quit drinking ≤ 6 months prior. Hypertension, diabetes, hyperlipidemia, smoking, drinking and ischemic heart disease were defined as stroke risk factors.

Once patients had been admitted to the stroke unit, antithrombotic therapies (including mono-antiplatelet or dual antiplatelet therapy), stain and management of blood pressure, glucose, and lipids were carried out based on the stroke unit's therapeutic and diagnostic protocol [20]. Clinical status was assessed using the National Institutes of Health Stroke Scale (NIHSS) at admission and continued at the following 72 h 1–3 times a day. END was defined as an NIHSS score increased by 2 or more points during the first 72 h compared with the initial NIHSS score [17,21,22]. The functional outcome at 90 days after onset was noted as the modified Rankin Score (mRS), which was dichotomized as good (0–2) and poor (≥ 3). The evaluation of END and the functional outcome was conducted by certificated investigators who were blinded to the clinical features.

3.1. Imaging analysis

According to our imaging protocol, all patients underwent an MRI scanning during the first 24 h after admission with 1.5-T (Signa; GE, Fairfield, CT, USA) or 3.0-T (Magnetom Avanto; Siemens, Munich, Germany) MRI largely depending on which one was available to achieve a quick evaluation. The imaging protocol included T1-weighted imaging, T2-weighted imaging, DWI, fluid-attenuated inversion recovery (FLAIR), and 3D time-of-flight magnetic resonance angiography (MRA). The diagnosis of infarcts on the MCA perforating territory was made with the use of a previously published template [23]. The size of the infarction was also analyzed and represented by the largest diameter of the lesion on DWI. Severity of white matter hyperintensity (WMH) (assessed according to the grading scales reported by Fazekas) was also evaluated and detailed documented from the MRI imaging and the deep white matter hyperintensities with scores of 2 and 3 were considered to be significant WMH in this study [24].

The status of the parent artery was evaluated using either MRA or computed tomographic angiography (CTA) and categorized as normal, mild (< 50%) stenosis, and moderate to severe (≥ 50% to occlusion) stenosis. In our study, stenosis of any degree was regarded as a significant cause of SSSI. According to the status of parent artery, two patterns of SSSI were shown: SSSI associated with PAD and those without (as shown in Figs. 1 and 2). Concomitant intracranial atherosclerotic stenosis (ICAS) and extracranial atherosclerotic stenosis (ECAS) that unrelated to the new SSSI were also evaluated according to WASID criteria and NASCET criteria, respectively [25,26]. The presence and the degree of cerebrocervical artery stenosis were analyzed by consensus among two physicians who were blinded to clinical status and the interrater variability (κ) was 0.89 for the identification of the artery status.

3.2. BP measurements and management

For each patient, systolic BP (SBP) and diastolic BP (DBP) were