Journal of the Neurological Sciences 368 (2016) 160–164

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Characteristics of subcortical infarction due to distal MCA penetrating artery occlusion☆

Kentaro Suzuki [a,*], Junya Aoki [a], Yoshio Tanizaki [b], Yuki Sakamoto [a], Satoshi Takahashi [c], Arata Abe [a], Hiroaki Kimura [d], Tadashige Kano [b], Satoshi Suda [a], Yasuhiro Nishiyama [a], Kazunori Akaji [b], Ban Mihara [d], Kazumi Kimura [a]

[a] Department of Neurological Science, Nippon Medical School Hospital, Tokyo, Japan
[b] Department of Neurosurgery, Institute of Brain and Blood Vessels, Mihara Memorial Hospital, Gunma, Japan
[c] Department of Neurosurgery, Keio University, Tokyo, Japan
[d] Department of Neurology, Institute of Brain and Blood Vessels, Mihara Memorial Hospital, Gunma, Japan

ARTICLE INFO

Article history:
Received 30 November 2015
Received in revised form 29 June 2016
Accepted 7 July 2016
Available online 8 July 2016

Keywords:
Ischemic stroke
Clinical outcome
Diffusion-weighted imaging
Infarct evolution

ABSTRACT

Objective: Isolated deep subcortical infarcts develop as a result of occlusion of the penetrating arteries from the internal carotid artery (ICA) and the proximal (M1) and distal middle cerebral artery (MCA). However, the clinical and neuroimaging characteristics of infarcts due to the occlusion of the distal MCA penetrating artery are unclear.

Methods: Consecutive patients with ischemic stroke or transient ischemic attack with magnetic resonance imaging (MRI) performed within 2 days of onset were studied retrospectively. Using coronal MRI data, isolated deep subcortical infarcts were classified into two groups: 1) proximal group, described as being longer than they are wide, which were expected to be related to the occlusion of the ICA or M1 penetrating artery; and 2) distal group, described as oblong, which were expected to be associated with the occlusion of penetrating arteries from the distal MCA (M2/M3/M4).

Results: A total of 653 consecutive acute ischemic stroke patients (proximal group, 50 [7.7%]; distal group, 14 [2.1%]) were enrolled. Baseline clinical characteristics were not different between the 2 groups. Modified Rankin Scale scores were lower in the distal group than in the proximal group 3 months after stroke onset (1.43 ± 0.36 vs. 2.26 ± 1.35, $p = 0.023$). We measured the lengths of the infarcts in the X and Y directions using axial MRI. The X/Y ratio was larger in the distal group than in the proximal group (1.3 ± 0.6 vs. 0.7 ± 0.2, $p < 0.01$), which indicated that distal MCA penetrating artery infarcts appear more oblong on axial MRI.

Conclusions: One cause for deep subcortical infarction is the occlusion of the distal MCA penetrating arteries, which occurs in 22% of patients with deep subcortical infarctions. These patients had better clinical outcomes than those with ICA and M1 penetrating artery infarctions. Distal MCA penetrating artery infarctions appear oblong on axial MRI.

1. Introduction

Deep subcortical infarcts caused by the occlusion of penetrating arteries, such as the anterior choroidal artery and the lenticulostriate artery (LSA), are representative of strokes affecting the anterior circulation [1–3]. These penetrating arteries arise from the internal carotid artery (ICA) and the middle cerebral artery (MCA). The development of magnetic resonance imaging (MRI) coronal sequencing techniques have enabled us to anatomically differentiate infarctions due to the occlusion of a penetrating arteries from the distal portion of the MCA (M2/M3/M4) from those due to the occlusion of penetrating arteries from the ICA and the proximal (M1) MCA [4]. However, the frequency and clinical and neuroimaging characteristics of distal MCA penetrating artery infarctions are unclear.

We suspected that there may be differences in the clinical characteristics of stroke patients with or without distal MCA penetrating artery infarctions. We hypothesized that these clinical characteristics would include patient outcome and neuroimaging findings. We therefore examined the characteristics of distal MCA penetrating artery infarctions.

2. Methods

We reviewed data from consecutive acute ischemic stroke patients admitted to Mihara Memorial Hospital between October 2012 and

☆ The authors have no conflicts of interest or funding sources to disclose.

* Corresponding author at: Department of Neurological Science, Nippon Medical School Hospital, 1-1-5 Sendagi, Bunkyo-ku, Tokyo 113-8603, Japan.
E-mail address: kentarow@nms.ac.jp (K. Suzuki).

http://dx.doi.org/10.1016/j.jns.2016.07.005
0022-510X/

September 2014 and imaged with MRI and MR angiography (MRA) within 2 days of stroke onset. Only patients who had an isolated MRI-proven infarct localized in the territory of the deep subcortex were analyzed in this study. An isolated deep subcortical infarct was defined as a singular infarct on MRI localized to the corona radiata, the basal ganglia, the centrum semiovale, the internal capsule, or other deep white matter regions [5].

In this study, two neurologists (K.S. and Y.T.), who were blinded to clinical data, reviewed the MRI images at admission and classified the isolated deep subcortical infarcts into two groups based on their shapes on coronal diffusion-weighted imaging (DWI) images: 1) proximal infarcts, described as being longer than they are wide, which were expected to be related to the occlusion of the penetrating artery from the ICA or the M1 (Figs. 1-A and 2-A), and 2) distal infarcts, described as oblong, which were expected to be associated with the occlusion of the penetrating artery from the distal MCA (M2/M3/M4) (Figs. 1-B and 2-B). We also measured the lengths of the infarcts in the X and Y directions on the 5-mm-thick axial DWI slice that depicted the largest infarct and calculated the X/Y ratio. None of the patients had ICA or MCA occlusions, as determined by MRA. A follow-up MRI with fluid-attenuated inversion recovery (FLAIR) was conducted on the patients during their hospitalization.

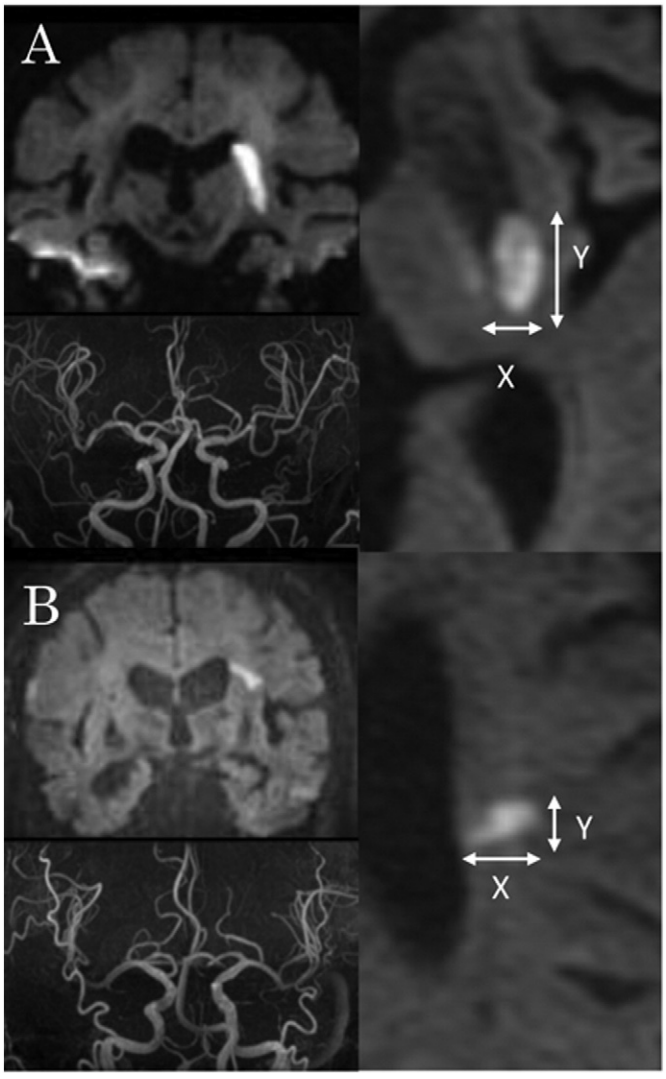

Fig. 1. Two types of deep subcortical infarction in the middle cerebral artery perforator territory. A, Proximal group: ICA or M1 penetrating artery infarction. (X-axis, 7.7 mm; Y-axis, 15.9 mm; X/Y ratio, 0.48) B, Distal group: Distal MCA penetrating artery infarction (X-axis, 10.1 mm; Y-axis, 7.1 mm; X/Y ratio, 1.42).

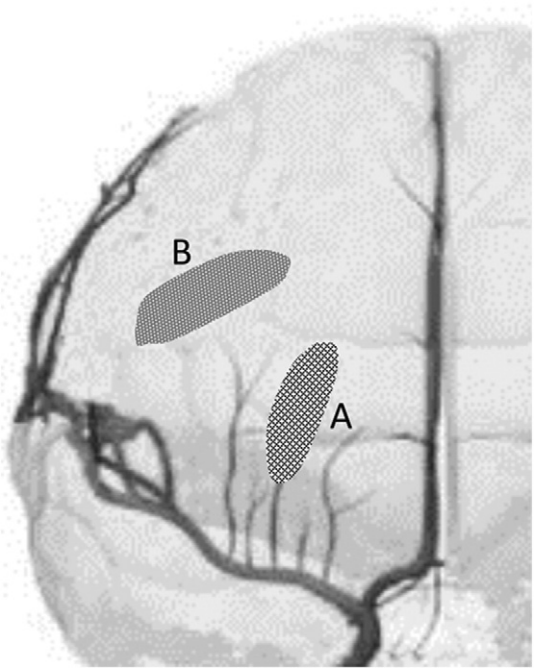

Fig. 2. Two types of deep subcortical infarction in the middle cerebral artery perforator territory. A, Proximal group: ICA or M1 penetrating artery infarction. B, Distal group: Distal MCA penetrating artery infarction.

The following clinical information was also obtained: age, sex, neurological deficits, vascular risk factors, chronic heart failure, atrial fibrillation, frequency of classic lacunar syndrome, significant artery stenosis, size of the deep subcortical infarcts, deterioration of neurological symptoms, and clinical outcome at 3 months. Neurological deficits were assessed using the National Institutes of Health Stroke Scale (NIHSS) score [6] at admission. Vascular risk factors were identified as: 1) hypertension, a history of using antihypertensive agents, a systolic blood pressure ≥ 140 mmHg, or a diastolic blood pressure ≥ 90 mmHg at hospital discharge; 2) diabetes mellitus, use of hypoglycemic medicines, random glucose level ≥ 200 mg/dL, or glycosylated hemoglobin >6.4% on admission; 3) hyperlipidemia, use of antihyperlipidemic agents, or serum cholesterol level > 220 mg/dL on admission; and 4) smoking, any cigarette usage within the 28 days preceding the index stroke. To detect arterial fibrillation, all patients underwent 12-lead electrocardiography (ECG), 24-hour ECG monitoring, and transthoracic echocardiography. Classic lacunar syndrome includes pure motor hemiparesis, pure sensory stroke, sensorimotor stroke, ataxic hemiparesis, and dysarthria-clumsy hand syndrome, according to previous reports [7,8]. Color-flow duplex carotid ultrasonography (Toshiba SSA 770A, Toshiba Inc., Tokyo, Japan) was performed in all cases. Conventional cerebral angiography was also conducted if appropriate. The extent of extracranial carotid stenosis was determined according to the criteria used in the North American Symptomatic Carotid Endarterectomy Trial [9]. To determine the degree of intracranial stenosis, the vessel being evaluated was measured according to the criteria used in The Warfarin Aspirin Symptomatic Intracranial Disease (WASID) trial [10]. Arterial disease was considered significant when stenosis was >50% or when occlusion was evident in the ipsilateral carotid system.

The MRI protocol included DWI, FLAIR, and intracranial MRA performed on a 3.0-T scanner (GE Sigma HDxt 3.0T HD, General Electric, Fairfield, USA) on admission. DWI was obtained with two protocols using the following parameters: 1) repetition time (TR), 6000 ms; echo time (TE), 83 ms; b-values, 0 and 1500 s/mm^2; field of view, 26 cm; acquisition matrix, 128 × 192; and slice thickness, 2.0 mm without gap; 2) TR, 6000 ms; TE, 80 ms; b-values, 0 and 1500 s/mm^2; field of view, 23 cm; acquisition matrix, 256 × 128; and slice thickness, 5.0 mm