Detecting lenticulostriate artery lesions in patients with acute ischemic stroke using high-resolution MRA at 7 T

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Abstract

Background: Recent advances in high-resolution (HR) magnetic resonance angiography (MRA) using ultrahigh-field systems enables direct visualization of the lenticulostriate arteries (LSAs), which had been hardly achieved by conventional MRA. Hence, by using HR-MRA at 7 T, we attempted to assess occlusive changes in the LSAs in patients with LSA territorial infarcts.

Methods: We prospectively examined 34 consecutive patients with acute ischemic stroke in the LSA territory using a 7 T scanner. We measured the lengths of the relevant LSAs on HR-MRA and the diameters/volume of the infarcts and compared these between the patients with/without occlusive changes in the LSAs.

Results: On HR-MRA, occlusion of the LSAs were observed in 19 (59%) of 32 patients who were eligible for the analyses. The curved/straight lengths of the LSAs in the patients with LSA occlusion (23.1–31.1/17.8–24.3 mm) were significantly shorter than in those without apparent LSA occlusion (25.8–39.5/24.0–30.4 mm) (P = 0.027 /0.003). The anteroposterior/superoinferior diameters of the infarcts were significantly larger in the occluded-LSA group (14.5–21.4/14.9–22.2 mm) than in the intact-LSA group (10.9–16.8/10.8–16.2 mm) (*P* = 0.041/0.011). In addition, the curved lengths of the relevant LSAs showed significant correlations with the superoinferior diameters of the infarcts (*r*

= 0.38, *P* = 0.034).

Conclusion: Occlusive changes in the LSAs were frequently found in patients with acute ischemic stroke within the LSA territory when using HR-MRA at 7 T and were substantially related to superoinferior extension of the infarcts.

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Introduction

The lenticulostriate arteries (LSAs) are perforating arteries originating from the proximal portion of the middle cerebral artery (MCA) and mainly supply the basal ganglia and the corona radiata (1–3). Ischemic stroke confined in the LSA territories, which is often of limited size (4), is reportedly caused by various mechanisms such as LSA occlusion due to lipohyalinosis or micro-atheroma, obstruction at the LSA origin due to MCA plaques (i.e., branch atheromatous disease), and micro-embolism (5–7). However, it has been difficult to determine the pathogenesis of LSA-territorial infarcts mainly because conventional imaging modalities, such as magnetic resonance angiography (MRA) and computed tomography angiography (CTA), have so far been incapable of properly visualizing with LSAs and their abnormalities.

Recently, ultrahigh-field magnetic resonance (MR) scanners have become available in several research institutes. High-resolution (HR) imaging at 7 T has been applied for the direct assessment of perforating arteries using MRA. Several studies using HR-MRA at 7 T have revealed that the LSAs present with anatomical variations among healthy subjects (8–9) and are poorly visualized in patients with hypertension and chronic stroke (10-11). However, LSA imaging findings in patients with acute ischemic stroke in the LSA territory remain unknown. Therefore, we attempted to qualitatively

and quantitatively assess whether steno-occlusive changes in the LSAs occurred in patients with acute ischemic stroke in the LSA territories using HR-MRA at 7 T and to examine their correlations with infarct size and clinical outcome.

Method

Subjects

This study was approved by the appropriate institutional ethical committee (H24-68) and all patients provided informed consent prior to inclusion in this study. From October 2012 to July 2017, we prospectively recruited 34 consecutive patients (age range, 37–82 years; median age, 66 years; 23 men and 11 women) with acute ischemic stroke confined in the LSA territory, i.e., the basal ganglia and/or corona radiata, on baseline diffusion-weighted images (DWIs) obtained using 1.5T or 3T MR scanner, after exclusion based on the following criteria: 1) the patients showing infarcts in the territories of the medial striate arteries, recurrent artery of Heubner, anterior choroidal artery, medullary artery from cortical branch of the MCA, or long insular artery, 2) those who had steno-occlusive changes in major cervical and/or intracranial arteries, 3) those with possibility of cardioembolic stroke, according to electrocardiogram findings including a 24h monitoring during at least 7 days, and other disorders such as arterial

dissection, artery to artery embolism, vasculitis, moyamoya disease, and hypercoagulation state, 4) those with modified Rankin scale (mRS) score before onset of the stroke event of ≥ 2, and 5) those who had contraindications for MR scanning.

The patient characteristics before hospitalization included hypertension present in 19, hyperlipidemia in 15, and diabetes mellitus in six patients. Regarding medication use, seven patients were receiving angiotensin-2 receptor blockers, five statins, four oral hypoglycemic mediations, three anti-platelet agents, and one an anticoagulant agent. After hospitalization, all patients received standard treatment for ischemic stroke, such as antiplatelet, neuroprotection, transfusion, and statin therapies. There were no patients who received thrombolytic therapy or mechanical thrombectomy. Regarding the clinical assessment of the patients, the National Institute of Health Stroke Scale (NIHSS) and the mRS were evaluated at the time of admission and 3 months after onset.

Imaging Protocols

MR examinations were performed using a 7 T scanner (Discovery MR950, GE Healthcare, Milwaukee, WI) with quadrature transmission and 32-channel receive head coils. The patients underwent MR examinations 5–21 days after stroke onset (median, 8.5 days). Three-dimensional (3D) time-of-flight MRA images were acquired with the following scanning parameters: repetition time (TR), 15 ms; echo time (TE), 3.4 ms;

flip angle (FA), 15°; field of view (FOV), 12 cm; acquisition matrix size, 512 × 320; slice thickness, 0.6 mm; voxel size, 0.23 × 0.23 × 0.3 mm after zero-fill interpolation (ZIP); partitions, 192; parallel imaging factor, 2; number of excitations (NEX), 1; and acquisition time, 16 min 4 s.

Quasi-3D DWIs and 3D fluid attenuated inversion recovery (FLAIR) images were obtained to assess infarct size and extent. The parameters of the DWIs with b-values of 1000 s/mm^2 were as follows: TR, 12,000 ms; TE, 70.5 ms; FOV, 20 cm; acquisition matrix size, 128×128; slice thickness, 1.8 mm; number of slices, 80; NEX, 6; and acquisition time, 5 min. Those for the 3D-FLAIR were as follows: TR, 7,000 ms; TE, 101.3 ms; FOV, 25.6 cm; acquisition matrix size, 512 × 256; slice thickness, 0.5 mm (after ZIP); partition, 384; NEX, 1, and acquisition time, 12 min 4 s.

Image Analyses

Image reconstructions were performed by one of the authors (H.M.) using a commercially available workstation (Advantage Workstation 4.5; GE Medical Systems, Milwaukee, WI). The slab maximum intensity projection (MIP) images focused on the LSAs were reconstructed at the oblique coronal planes being parallel to the LSAs and the bilateral sagittal planes (thickness, 20 mm; interval, 1 mm; partitions, 35–40; FOV, 90 mm). In addition, contiguous coronal images with 2-mm thickness were generated

from the MRA source, DW, and FLAIR images.

Two board-certified experts, a neuroradiologist (M.S.) and a neurologist (T.N.), who were blinded to patient information, visually determined the LSAs that were relevant to the infarcts according to the spatial relationships on the MR source, DWI, and FLAIR images using a software program (VOX-BASE II; J-MAC SYSTEM, Sapporo, Japan). They then assessed the locations of the occlusive changes in the relevant LSA using the categories as follows; origin, proximal portion beneath the basal ganglia, distal portion within the basal ganglia, and no apparent occlusion. The presence of recanalization in the relevant LSAs was also speculated when the LSAs passed through the infarct lesions with/without hemorrhagic changes (12). In addition, one of the authors (H.M), who was blinded to patient information, measured the curved/straight lengths and the tortuosity of the relevant LSAs from the origin on the coronal slab MIP image, according to previous studies (8, 10-11).

Regarding the infarct size, two authors (M.S., T.N.) visually assessed the extent of the infarcts in terms of involvement of five areas: the corona radiata, the upper, middle, and lower thirds of the basal ganglia, and the anterior perforating substance, on DW and FLAIR images. All the visual assessments were performed twice with randomized orders within a 2-week interval. Differences in the visual assessment between two

observers were solved by consensus.

In addition, another author (H.M.) measured the maximum anteroposterior (AP), dextrosinistral (DS), and superoinferior (SI) diameters on axial and coronal DWIs (or FLAIR images when DWIs were unavailable) as well as the volume of the infarcts on DW source images (or FLAIR images) using a free software package (3D-Slicer, http://www.slicer.org) (13). For the volume measurements, a small circular region of interest (ROI) was manually set on the infarcts, and the areas showing hyperintensity more than 2 standard deviations of the ROI were defined as infarct lesions. The infarct volume was then calculated. The measurements were performed twice and the values were then averaged.

Statistical Analyses

The patient characteristics were compared between the patients with LSA occlusion (occluded-LSA group) and those with no apparent occlusion (intact-LSA group) using the Mann-Whitney or Fisher's exact test. Differences in imaging findings including lengths/tortuosity of the relevant LSAs, extent of infarcts, and infarct volume/diameters as well as clinical outcome were compared between the occluded-LSA and intact-LSA groups using the Mann-Whitney, Fisher's exact, or chi-squared test. Further, the correlations between the lengths/tortuosity of the relevant LSA and the

diameters/volume of the infarcts were examined by linear regression analysis. Intrarater and interrater agreements of the visual assessments and measurements were determined using the kappa value or intraclass correlation coefficients (ICCs). Statistical values of $P < 0.05$ were considered significant.

Results

MR images were obtained from all patients; however, the MRA source images of two patients had profound motion artifacts and had to be excluded. Hence, the images of the remaining 32 patients (94.1%) were eligible for further analyses.

On HR-MRA, occlusion of the LSA relevant to the infarcts was observed in 19 patients (59.3%). Among these, proximal and distal occlusions were found in six and 13 patients, respectively; however, no LSA was occluded at the origin (Table 1, Fig. 1A). The remaining 13 patients (40.6%) showed no apparent LSA occlusion, including suggestive of recanalization in two patients (Fig. 1B). No significant differences in patient characteristics were observed between the occluded-LSA and intact-LSA groups (P = 1.00–0.07, Fisher's exact tests or Mann-Whitney test) (Table 1). In addition, there were no significant differences in NIHSS and mRS scores at 3 months after onset between the groups (P = 0.12 and 0.28, respectively; Mann-Whitney test) (Table 2).

The curved/straight lengths of the relevant LSA in the occluded-LSA group (23.1–31.1mm [median, 27.1 mm] and 17.8–24.3 mm [21.8 mm], respectively) were significantly shorter than those in the intact-LSA group (25.8–39.5mm [34.2 mm] and 24.0–30.4mm [29.2 mm], respectively] (P = 0.027 and 0.030, respectively; Mann-Whitney test). The tortuosity of the relevant LSAs showed no significant differences between the two groups (Table 2).

Regarding the extent of the infarcts, lesions confined within the corona radiata (CR) were found in nine patients (28.1%) and those extending from the CR to the upper, middle, and lower basal ganglia were observed in 15 (46.9%), five (15.6%), and three (9.4%) patients, respectively (Table 1). The extent was significantly larger in the occluded-LSA than in the intact-LSA group (P = 0.014, chi-squared test). The AP and SI diameters of the infarcts were also significantly larger in the occluded-LSA group (14.5–21.4 mm [median, 16.3 mm] and 14.9–22.2 mm [17.1 mm], respectively) than in the intact-LSA group (10.9–16.8 mm [12.5 mm] and 10.8–16.2 mm [12.6 mm], respectively) (P = 0.041 and 0.011, respectively; Mann-Whitney test), although the DS diameter and volume showed no significant differences between the occluded-LSA group (8.6–13.9 mm [11.3 mm] and 504–1611 mm^3 [median, 671 mm^3], respectively) and the intact-LSA group (8.2–12.5 mm [9.3 mm] and 348–792 mm^3 [511 mm^3],

respectively) (P = 0.29 and 0.16, respectively) (Table 2).

Regarding the correlations between LSA lengths/tortuosity and infarct diameters/volume, the curved LSA lengths showed a significant correlation with the SI diameters (r = 0.375, P = 0.034), whereas there were no significant correlations between other combinations (Table 3, Fig. 2).

The intra/inter-rater agreements of the visual assessments in terms of kappa values ranged within 0.42–0.62/0.75–0.88, indicating moderate to substantial agreements, while the ICC value of the quantitative measurement ranged within 0.96–1.00, indicating excellent agreements.

Discussion

In the present study, we successfully assessed alterations in LSAs in patients with acute ischemic stroke confined in the LSA territory by using HR-MRA at 7 T. We found that approximately 60% of the patients showed proximal or distal occlusions of the relevant LSAs and had substantially large infarcts along the SI direction involving the corona radiata and partially the basal ganglia. These results suggest that ischemic stroke in the LSA territory can occur due to occlusion of the LSA at the level passing through the basal ganglia or more proximal areas. These occlusive changes could be caused by

micro-atheroma rather than lipohyalinosis because micro-atheroma reportedly appears in perforating arteries 200–800 μm in diameter while lipohyalinosis occurs in arteries 40–300 μm in diameter (15).

Among the patients with LSA occlusion, three patients showed large infarcts along the SI direction extending from the corona radiata to the lower one third of the basal ganglia, which were compatible to the distribution caused by a branch atheromatous disease (BAD) mechanism, i.e., obstruction at the origin of perforating arteries by atherosclerotic lesions of the parent artery (15). However, these patients showed occlusive findings not at the origin but at the proximal portion of the LSAs. These results suggest that infarcts with BAD-type distribution in the LSA territory can be caused by LSA occlusion per se, presumably due to micro-atheroma.

We found that most patients with apparently intact LSA had relatively small infarcts confined within the corona radiata, which could be caused by lipohyalinosis rather than micro-atheroma. However, two of them showed findings suggestive of recanalization of the relevant LSAs. In general, recanalization of occluded arteries in acute infarction can occur in embolisms to major intracranial arteries or cortical branches (11). Several studies assumed possibilities of embolic mechanisms for ischemic stroke in the LSA territory, although conventional imaging techniques failed to

detect changes in relevant LSAs (16–18). Recanalization of the LSAs in this study may have been caused by artery-to-artery micro-embolism from intracranial atherosclerotic plaques with positive remodeling which have no substantial stenosis on MRA because no evidence of atrial fibrillation or cervical carotid plaques were observed.

In this study, occlusive locations or lengths of the relevant LSAs were substantially related with the extension or size of the infarcts along the SI direction, indicating that LSA occlusion can cause ischemia in the areas distal to the occlusion site within the territory. However, these characteristics of the occluded LSAs showed no apparent relationships with horizontal dimensions and volume of the infarcts that were mainly located in the corona radiata. In addition, there were no significant differences in clinical outcome between the patients with/without LSA occlusion. These results suggest that the infarct size in the corona radiata depends on both the location of the LSA occlusion and on other factors including variations of territorial size and collaterals.

This study had several limitations. First, we were able to enroll a relatively small number of patients, mainly because of the strict exclusion criteria. In addition, the date of the MR examination after stroke onset substantially varied because of a transportation issue to the 7 T MR scanner that is situated approximately 10 km away

from the hospital, which could have affected the results of this study to some extent. Second, the precision in the measurement of LSA lengths has not been fully determined. The measurements using coronal MIP images can include substantial errors because the trajectories of the LSAs are three-dimensional and their angulations vary. Sophisticated techniques such as a curved planar reconstruction are needed to further improve measurement accuracies. Third, in this study, we evaluated LSA changes only in the acute period. Hence, the time courses of the LSA lesions during medical treatment remain unknown, although this issue was beyond the scope of this study. Another limitation of the present study involves technical issues related to the imaging methods used. The MR scans with high spatial resolution at 7 T we used require a substantially long examination time, as compared with routine MR scans for acute stroke in clinical practice. Time reduction techniques such as parallel imaging, compressed sensing, and multiband excitation are needed to facilitate scan use even in patients with acute stroke. Finally, we did not assess vessel wall lesions such as atherosclerotic plaques in LSAs and their parent artery, so that relationships between LSA occlusive changes and atherosclerotic changes remain unclear. This issue can be investigated by the combined use of HR-MRA and HR vessel wall imaging at 7 T (19-20).

In conclusion, we readily elucidated that occlusive changes in the LSAs were frequently observed in patients with acute ischemic stroke confined in LSA territories and were significantly related with SI extension of the infarcts by using HR-MRA at 7 T.

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Disclosures

The authors declare no conflict of interest associated with this manuscript.

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Figure legends

Fig. 1. High-resolution magnetic resonance angiography (HR-MRA) of patients with acute stroke in the lenticulostriate artery (LSA) territory obtained by a 7T scanner

A: A 66-years-old man with acute infarct in the left LSA territory (6 days after onset). The LSA relevant to the infarct is occluded at the distal portion on HR-MRA (arrowheads). The infarct lesion involves the corona radiata and the upper basal ganglia on diffusion-weighted images (DWIs) (arrows).

B: A 37-years-old man with acute infarct in the right LSA territory (17 days after onset). The relevant LSA appears intact on HR-MRA (arrowheads). The infarct is confined within the corona radiata on DWIs (arrows).

Fig. 2. Correlations between the LSA lengths and the infarct size in patients with LSA-territorial infarct

There are significant, substantial negative correlations between the curved lengths of the relevant LSAs and the superoinferior (SI) diameter of the infarcts (r = -0.38; P = 0.034).

Closed circles, patients with occlusion of the relevant LSAs (occluded-LSA group); open circles, those with apparently intact LSAs (intact-LSA group).

Table 1. Patient characteristics in the patients with occluded LSA and those with apparently intact LSA

Patient characteristics	Occluded LSA (n = 19)	Intact LSA (n = 13)	Total (n = 32)	*P*-value*
Age (median)	63.5–74.8 (68)	57–67 (61)	60.3–74.3 (66)	0.07
Men	12 (37.5%)	11 (34.4%)	23 (71.9%)	0.25
NIHSS at admission (median)	3–5 (3)	2–4 (2)	2–4 (3)	0.11
Hypertension	11 (34.4%)	8 (25%)	19 (59.4%)	1.00
Dyslipidemia	7 (21.9%)	8 (25%)	15 (46.9%)	0.28
Diabetes mellitus	4 (12.5%)	2 (6.3%)	6 (18.8%)	1.00
Old ischemic stroke	2 (6.3%)	0	2 (6.3%)	0.50
Old intracerebral hemorrhage	1 (3.1%)	0	1 (3.1%)	1.00
Atrial fibrillation	1 (3.1%)	0	1 (3.1%)	1.00
Cardiovascular disease	1 (3.1%)	0	0 (3.1%)	1.00
Chronic kidney disease	0	1 (3.1%)	1 (3.1%)	0.41
Smoking	9 (28.1%)	7 (21.9%)	16 (50%)	0.72

LSA, lenticulostriate artery; NIHSS, National Institutes of Health Stroke Scale;

*Fisher’s exact tests or Mann-Whitney test

Table 2. Imaging findings and clinical outcomes in the patients with occluded LSA and those with apparently intact LSA

Imaging findings and clinical outcome		Occluded LSA (n = 19)	Intact LSA† (n = 13)	*P*-value*
Lengths of relevant LSA	Curved length [mm]	23.1–31.1 (27.1)	25.8–39.5 (34.2)	**0.027**
	Straight length [mm]	17.8–24.3 (21.8)	24.0–30.4 (29.2)	**0.003**
	Tortuosity	1.15–1.31 (1.25)	1.10–1.29 (1.17)	0.182
Extent of infarcts	CR only	2 (11%)	7 (54%)	**0.014**
	CR – upper 1/3 of BG	9 (47%)	6 (46%)†	
	CR – middle 1/3 of BG	5 (26%)	0	
	CR – lower 1/3 of BG	3 (16%)	0	
Infarct size	Volume [mm^3]	504–1611 (671)	348–792 (511)	0.16
	AP diameter [mm]	14.5–21.4 (16.3)	10.9–16.8 (12.5)	**0.041**
	DS diameter [mm]	8.6–13.9 (11.3)	8.2–12.5 (9.37)	0.29
	SI diameter [mm]	14.9–22.2 (17.1)	10.8–16.2 (12.6)	**0.011**
Clinical outcome	NIHSS at 3 months	1–3 (2)	0–2 (1)	0.12
	mRS at 3 months	1–1 (1)	1–1 (1)	0.28

AP, anteroposterior; APS, anterior perforated substance; BG, basal ganglia; CR, corona radiata; DS, dextrosinistral; LSA, lenticulostriate artery; mRS, modified Rankin scale; NIHSS, National Institutes of Health Stroke Scale; SI, superoinferior; parentheses, median; *Mann-Whitney test, Fisher's exact tests, or chi-squared test; †, including two patients with recanalized LSAs.

Table 3. Correlations between the lengths/tortuosity of relevant LSAs and the infarct volume/diameters

Infarct size	Total LSA lengths		
	Curved length	Straight length	Tortuosity
Volume	-0.15 (0.40)*	-0.12 (0.51)	0.04 (0.83)
AP diameter	-0.21 (0.24)	-0.19 (0.30)	0.15 (0.40)
DS diameter	-0.05 (0.80)	0.01 (0.94)	-0.02 (0.92)
SI diameter	**-0.38 (0.034)**	-0.28 (0.13)	-0.21 (0.24)

AP, anteroposterior; DS, dextrosinistral; LSA, lenticulostriate artery; SI, superoinferior;

*Pearson's correlation coefficient (*P*-value).