Evaluating recanalization of relevant lenticulostriate arteries in acute ischemic stroke using high-resolution MRA at 7T

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Abstract

Background: Occluded major intracranial arteries can spontaneously recanalize in patients with acute ischemic stroke mainly due to embolic mechanisms. However, it remains unknown whether recanalization can occur in perforating arteries, such as lenticulostriate arteries (LSAs). Therefore, in the present study, we assessed changes suggesting recanalization of the LSAs in patients with acute ischemic stroke of the LSA territory using high-resolution magnetic resonance angiography (HR-MRA) at 7T.

Methods: We prospectively examined 39 consecutive patients with acute infarcts confined within the LSA territory. Using a 7T scanner during the acute period and 1 month thereafter, we evaluated imaging findings indicating the recanalization of the relevant LSAs, following which we examined differences in other imaging findings and clinical characteristics between patients with/without recanalization.

Results: HR-MRA findings suggestive of recanalization (i.e., patent LSAs within acute infarct lesions with/without hemorrhagic changes) were observed in eight (25%) of 32 patients who were eligible for analyses. These findings were detected in three and five patients on the baseline and follow-up images, respectively. The lengths of relevant LSAs on the follow-up MRA were significantly larger in patients with recanalization than in those without (P = 0.01). However, there were no significant differences in the

infarct volume or clinical outcomes between the recanalization and non-recanalization groups.

Conclusion: HR-MRA at 7T revealed that recanalization of the relevant LSAs can occur in patients with acute ischemic stroke confined to the LSA territory.

Introduction

Spontaneous recanalization occasionally occurs in occluded major intracranial arteries or cortical branches in patients with acute ischemic stroke mainly due to embolic mechanism, which can contribute to the preservation of brain tissue or induce hemorrhagic transformation (1). However, it remains unknown whether recanalization can occur in occluded perforating arteries such as the lenticulostriate arteries (LSAs), although previous research has suggested that embolic mechanisms can cause infarcts confined to the territory of the perforating arteries (2-9). Several studies have indicated that embolic mechanisms play a role in patients with acute infarcts in the LSA territory (3-5,8,10); however, there has been no direct evidence of LSA recanalization, mainly because conventional imaging techniques fail to detect changes in LSAs during the acute stage due to limited spatial resolution.

Recently, ultra-high-field magnetic resonance (MR) scanners have been applied for direct visualization of perforating arteries, including the LSAs (11-15). High-resolution (HR) MR angiography (MRA) at 7T has been reported to readily depict LSAs and their changes in patients with hypertension, chronic stroke, and acute stroke (12,14,15). However, no studies have investigated temporal changes in relevant LSAs and their clinical implications during treatment for acute ischemic stroke.

Therefore, we attempted to evaluate longitudinal changes in the LSAs relevant to acute infarcts confined in the LSA territory using HR-MRA at 7T, in order to determine whether recanalization of relevant LSAs can occur in patients with acute ischemic stroke. We further aimed to examine whether recanalization is associated with infarct size and clinical outcomes.

Methods

Patients

This study was performed after obtaining the approval of the institutional ethics committee (H24-68), and written informed consent was obtained from all participants. From October 2012 to October 2018, we prospectively recruited 39 consecutive patients (age range: 37–82 years; median age: 66 years; 27 men and 12 women) with acute ischemic stroke confined within the unilateral LSA territory on baseline diffusion-weighted images (DWIs) obtained using 1.5/3T MR scanners. Patients with arterial dissection, vasculitis, moyamoya disease, or hypercoagulation; those with modified Rankin scale (mRS) scores ≥2 before onset; those who had undergone thrombolytic therapy or mechanical thrombectomy; and those with contraindications for MR examination were excluded.

Patient history prior to hospitalization included hypertension in 19 patients, hyperlipidemia in 13 patients, diabetes mellitus in six patients, and ischemic stroke in two patients. No patients exhibited atrial fibrillation. Medication history was as follows: angiotensin II receptor blockers (n = 6), statins (n = 6), oral hypoglycemic mediations (n = 4), antiplatelet agents (n = 3), and anticoagulant agents (n = 1). After hospitalization, all patients received standard treatment for ischemic stroke, including antiplatelet, neuroprotective, transfusion, and statin therapies. The presence of atrial fibrillation was assessed via 24-h bedside electrocardiogram monitoring for at least 7 days, while steno-occlusive lesions of the major cervical/intracranial arteries were examined via cervical ultrasound and MRA. Clinical examination also included National Institute of Health Stroke Scale (NIHSS) and mRS scores at the time of admission and 3 months after onset.

Imaging Protocols

MR examinations were performed using a 7T scanner (Discovery MR950, GE Healthcare, Waukesha, USA) with a quadrature transmission and 32-channel receive head coil system. Patients underwent MR examinations during the acute period within 2 weeks after stroke onset and at 1 month after the baseline scans. Three-dimensional (3D) time-of-flight MRA scans were acquired using 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; 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 1,000 s/mm^2 were as follows: TR, 12,000 ms; TE, 70.5 ms; FOV, 20 cm; matrix size, 128×128; slice thickness, 1.8 mm; number of slices, 80; NEX, 6; and acquisition time, 5 min. Parameters for 3D-FLAIR images were as follows: TR, 7,000 ms; TE, 101 ms; FOV, 25.6 cm; matrix size, 512 × 256; slice thickness, 0.5 mm (after ZIP); partition, 384; NEX, 1, and acquisition time, 12 min 4 s.

Image Analyses

Using a workstation (Advantage Workstation 4.5; GE Healthcare, Waukesha, USA), one of the authors (T.S.) generated slab maximum intensity projection (MIP) images focused on the LSAs at the oblique coronal planes, which were created parallel to the LSAs and the bilateral sagittal planes (thickness: 20 mm; interval, 1 mm; partitions, 35–40; FOV, 90 mm). Contiguous coronal images with a thickness of 2 mm were also generated from the MRA source images, DWIs, and FLAIR images.

Using a software program (VOX-BASE II; J-MAC SYSTEM, Sapporo, Japan), two board-certified neurologists (T.N., H.M.), who were unaware of patient information, visually determined the LSAs relevant to the infarcts according to the spatial relationships on the MRA, DWI, and FLAIR images. They then assessed whether the relevant LSAs had recanalized based on the following imaging criteria: (a) Relevant LSAs were patent and passed through the infarct lesions with/without hemorrhagic changes on the baseline images; (b) relevant LSAs that had been occluded at baseline had become patent and passed through the infarcts on the follow-up images. They also evaluated occlusion sites of the relevant LSAs (i.e., origin, proximal portion beneath the basal ganglia, distal portion within the basal ganglia, or no apparent occlusion). In addition, they assessed stenosis or wall irregularity of intracranial major arteries proximal to the LSAs. All visual assessments were performed twice in randomized order, with an interval of 2 weeks between assessments. Differences between the visual assessments of the two observers were resolved via consensus.

For quantitative analyses, one of the authors (T.S.), who was blinded to patient information, measured the straight lengths of the relevant LSAs on the coronal slab MIP images, in accordance with previously described methods (14, 15). The same author measured the infarct volume on FLAIR images using a software package (3D-Slicer,

http://www.slicer.org) (16). The measurements were performed twice, and the values were then averaged.

Statistical Analyses

We compared demographic and clinical characteristics between patients with recanalization of the relevant LSA (recanalization group) and those with no apparent recanalization (non-recanalization group) using Mann-Whitney U-tests or Fisher's exact tests. Differences in imaging findings (length of the relevant LSAs and infarct volume) and clinical outcomes (NIHSS and mRS scores at 3 months) were compared between the two groups using Mann-Whitney U-tests or Fisher's exact tests. Intra/inter-rater agreement values for the visual assessments and measurements were determined using the kappa value or intraclass correlation coefficient (ICC). The level of statistical significance was set at $P < 0.05$.

Results

Baseline MR images were obtained from all patients; however, follow-up MR scans were unavailable for six patients due to declination, transportation issues, and hospitalization for other diseases. In addition, one patient was excluded due to profound motion artifacts on MR images. The images of 32 patients (82.1%) were thus eligible

for further analyses. In these patients, the baseline and follow-up MR examinations were performed 7–13 days (median: 9 days) and 34–45 days (median: 41 days) after the onset, respectively, with an interval of 27–35 days (median: 31 days).

Occlusion of LSAs relevant to the infarcts was observed on baseline HR-MRA images in 25 patients (78.1%). Among these, proximal and distal occlusions were observed in six and 19 patients, respectively; however, no patient exhibited occlusion at the origin. On follow-up images, revisualization of the LSAs due to recanalization was observed in five patients (20.0%) (proximal occlusion: 1 [16.7%]; distal occlusion: 4 [21.1%]) (Fig. 1, 2). Among the remaining seven patients with no apparent LSA occlusion (23.3%), three patients exhibited findings indicative of recanalization (i.e., relevant LSAs passing through the infarct core) on the baseline images (Fig. 1, 3). In other four patients, the infarct cores, confined within the corona radiata, were located distal to the visible LSAs on the baseline images as well as the LSAs appeared unchanged and no hemorrhagic changes occurred on the follow-up images, suggesting no evidences of recanalization. Thus, in total, eight patients (25.0%) exhibited findings indicative of recanalization of the relevant LSAs (recanalization group), including two patients (baseline: 1; follow-up: 1) who exhibited hemorrhagic changes in the surrounding territory. In these patients, there were no episodes suggestive of

recanalization, such as improved neurological symptoms.

Hypertension was more frequently observed and age was higher in the recanalization group than in the non-recanalization group ($P = 0.04$). However, there were no significant differences in other patient characteristics and clinical outcomes ($P = 1.00–0.19$) (Table 1). Atrial fibrillation was detected only in one patient of the non-recanalization group during the follow-up period. No patients exhibited stenotic changes or wall irregularity in cervical/intracranial parent arteries proximal to the LSAs, which can cause artery-to-artery embolism or other diseases related to the stroke event.

The lengths of the relevant LSAs on the follow-up images were significantly larger in the recanalization group (28.1–34.0 mm [median, 30.4 mm]) than those in the non-recanalization group (19.9–27.9 mm [23.3 mm] ($P = 0.01$), although there were no significant differences in these values at the baseline ($P = 0.51$). In contrast, there were no significant differences in the infarct volume between the two groups ($P = 0.54$).

The kappa values for the visual assessments ranged 0.74–1.00, indicating substantial agreements, while the ICC values for quantitative measurements ranged 0.82–0.99, indicating excellent agreement.

Discussion

In the present study, we successfully assessed changes in relevant LSAs and surrounding structures in patients with acute ischemic stroke confined to the LSA territory using 7T HR-MRA. We observed that approximately 25% of patients exhibited findings indicative of spontaneous LSA recanalization on baseline or follow-up images—a greater frequency than expected. Direct detection of LSA recanalization has been difficult using conventional imaging techniques due to the limited spatial resolution of 1.5/3T MRA and computed tomography angiography (CTA). Although catheter angiography can depict perforating arteries, it appears too invasive for repeated use in the diagnosis of stroke events. In contrast, 7T HR-MRA is a non-invasive technique with excellent spatial/contrast resolution, making it suitable for evaluating changes in perforating arteries in patients with acute ischemic stroke.

In this study, recanalized, normal-appearing LSAs traversed at the center of the acute infarct cores and were occasionally accompanied by hemorrhagic changes, even though no patient had undergone thrombolytic therapy. Hence, we assumed that the spontaneous recanalization of the LSAs occurred in the infarcts mainly due to embolic mechanisms, although no patients had atrial fibrillation or other pathological conditions that can cause embolisms. The spontaneous recanalization may occur in other various conditions such as micro-atheroma, vasospasm, and the development of collateral

circulation. In this study, however, we found no supportive imaging findings, such as stenosis or wall irregularity of relevant LSAs and appearance of collaterals.

Infarction confined in the LSA territory, particularly lacune, is said to be mainly caused by small vessel disease mechanisms, which are recently considered as complex processes based on pathological changes such as arteriolosclerosis, lipohyalinosis, and fibrinoid necrosis as well as dysfunction of the blood-brain barrier (BBB) (17). On the other hand, several studies have suggested that micro-embolisms may occur in the perforating artery territory, although conventional imaging techniques have been unable to provide direct evidence (3-5,8,10). One primate study demonstrated that micro-emboli less than 80 microns can enter perforating arteries, subsequently resulting in lacunar infarctions (18). In addition, several studies have suggested that small infarcts in the LSA territory can occur due to cardio-embolism (3,5,9,10,19-21) or artery-to-artery embolism (22,23). Furthermore, hemorrhagic infarction is generally observed after spontaneous recanalization in patients with embolic stroke due to the leakage of red blood cells from the damaged arterial walls during reperfusion, but not in patients with lacunar or atherothrombotic stroke (1). In two of eight patients with recanalized LSAs (25%), we observed hemorrhagic infarction, suggesting that LSA recanalization can occur in patients with acute stroke due to embolic mechanisms.

We were unable to determine embolic sources in the present study. No patients exhibited atrial fibrillation during the follow-up period in the recanalization group although one patient in the non-recanalization group did. Atherosclerotic plaques of major cervical or intracranial arteries are said to cause lacunar-type infarcts due to artery-to-artery embolism (8,22,23). However, no patients in our study exhibited any stenotic changes or wall irregularities of these arteries. Recent studies have indicated that vessel wall imaging (VWI) techniques can detect atherosclerotic plaques in major intracranial arteries without stenosis (24), suggesting that atherosclerosis with outward remodeling can occur in intracranial arteries, similar to that in coronary arteries, which can cause acute coronary syndrome (24-26). Thus, plaques of intracranial major arteries without stenosis may cause artery-to-artery embolism to the LSAs and may result in recanalization. Small vessel disease effects including the BBB dysfunction can be another possible cause for LSA occlusion. Leakage through the BBB and perilesional interstitial edema can be assessed by using advanced MR imaging methods such as dynamic contrast-enhanced imaging and T1 mapping (17); however, these images were not included in this study. Further studies involving VWI and BBB-leakage imaging are required to determine mechanisms of the LSA occlusion and recanalization.

In this study, there was no significant difference in the infarct volume between the

recanalization and non-recanalization groups. In addition, there were no correlations between LSA recanalization and clinical outcomes, although the presence of recanalization is thought to be related to good functional outcomes (27). These results indicate that LSA recanalization may occur particularly in relatively large LSAs but does not strongly contribute to the preservation of ischemic brain tissues. In addition, the lengths of the relevant LSAs were significantly larger in the recanalization group than in the non-recanalization group at the 1-month follow-up, which may reflect additive effects of recanalization and vasoparalysis on improved visualization on MRA.

This study had several limitations. First, we were able to enroll a relatively small number of patients, mainly because of the strict exclusion criteria (e.g., infarct location confined within the LSA territory) and issues with transportation to the facility containing the 7T MR scanner, which is located approximately 15 km from the hospital. Such issues also resulted in varied and late timing of the baseline scans. There were no patients who underwent the baseline MR examination in the acute phase less than 7 days after onset, which may have resulted in substantial bias to the recanalization rate. We hardly compare our results with those in previous studies, although the recanalization rate in this study appears comparable to that of large artery occlusion, approximately 20% (27). In addition, the LSA recanalization rate after thrombolytic

therapy remains unknown. We excluded these patients from this study because it was difficult to perform 7T scans before the therapy. Second, it remains unknown when LSAs were recanalized. No major changes in clinical symptoms due to recanalization were detected, and we were unable to perform repeated 7T scans during the acute period. Third, we cannot deny the possibility of overlooking partial recanalization with severe stenosis and slow flow, because the 3D-TOF technique generally overestimates stenotic severity and is insensitive to slow flow. Furthermore, we hardly assess occlusion or recanalization of arterioles that extend from distal LSAs, which are too small to be visualized by 7T HR-MRA. Fourth, we did not directly assess vessel wall lesions such as atherosclerotic plaques of major intracranial arteries, although there were no patients with visible atherosclerotic changes, i.e., stenosis or wall irregularity, in parent arteries proximal to the relevant LSAs on HR-MRA. Hence, it remains unclear whether intracranial plaques are associated with LSA territorial infarcts and LSA recanalization. Further studies should aim to resolve this issue via combined use of HR-MRA and VWI at 7T. Our results are also limited by technical issues related to the postprocessing methods. We used axial source images and slab MIP images for qualitative and quantitative assessments of changes in LSAs, which can include substantial errors because LSAs have a three-dimensional configuration. Sophisticated

methods such as a curved planar reconstruction are required for more accurate analyses of microvessels such as LSAs (28).

In conclusion, our findings demonstrate that HR-MRA at 7T can detect findings that indicate recanalization of the relevant LSAs in patients with acute ischemic stroke confined to the LSA territory.

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Disclosures

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

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Figure legends

Fig. 1. Flowchart of patient enrollment and MR examinations at 7T

Fig. 2. Longitudinal imaging findings indicating LSA recanalization in patients with acute ischemic stroke of the LSA territory.

A 74-year-old woman with an acute infarct in the left LSA territory. The infarct lesion involves the left corona radiata and the upper basal ganglia on coronal FLAIR images (arrows). The left, relevant LSA exhibits occlusive changes at the distal portion on baseline HR-MRA images (7 days after onset) (A, arrowheads). The LSA appears patent without any steno-occlusive changes on follow-up HR-MRA images (42 days after onset) (B, arrowheads).

Fig. 3. Baseline imaging findings suggesting LSA recanalization in patients with acute ischemic stroke of the LSA territory.

A 57-year-old man with an acute infarct in the right LSA territory (13 days after onset). The infarct lesion involves the right corona radiata and upper basal ganglia on coronal FLAIR images (arrows). On baseline HR-MRA images, the right LSA relevant to the infarct is apparently intact and passes through the infarct core, which is

surrounded by hemorrhagic changes (arrowheads).

Table 1. Characteristics of patients with acute infarcts confined within the LSA territory

Patient characteristics	Total (n = 32)	LSA Recanalization (n = 8)	LSA Non-recanalization (n = 24)	*P*-value*
Age	60–71 (66)	62–81 (72)	58–68 (63)	**0.04**
Men	23 (71.9%)	5 (62.5%)	18 (75.0%)	0.65
NIHSS at admission	2–4 (3)	3–5 (3)	2–4 (3)	0.65
mRS at admission	2–3 (2)	2–2 (2)	1–3 (2)	0.85
NIHSS at 3 months	1-2 (2)	1–4 (2)	1–2 (2)	0.19
mRS at 3 months	1-1 (2)	1–1 (1)	1–1 (2)	0.98
Hypertension	18 (56.3%)	8 (100%)	10 (41.7%)	**0.04**
Dyslipidemia	12 (37.5%)	2 (25.0%)	10 (41.7%)	0.23
Diabetes mellitus	6 (18.8%)	1 (12.5%)	5 (20.8%)	1.00
History of stroke	2 (6.3%)	1 (12.5%)	1 (4.2%)	0.44
Atrial fibrillation	1 (3.1%)	0	1 (4.2%)	1.00
Major artery stenosis/irregularity	0	0	0	1.00
Ankle brachial index	1.10-1.19 (1.17)	1.12-1.17 (1.13)	1.07-1.22 (1.18)	0.44
Max IMT	1.21-2.31 (1.53)	1.45-2.48 (1.85)	1.10-2.00 (1.5)	0.23

LSA, lenticulostriate artery; mRS, modified Rankin scale; NIHSS, National Institutes of Health Stroke Scale; IMT, intima-media thickness; *Fisher's exact tests or Mann-Whitney U-tests; parentheses, median or percentage.

Table 2. Imaging findings of the relevant LSAs and infarct lesions in patients with acute infarcts confined within the LSA territory

Imaging findings		LSA Recanalization (n = 8)	LSA Non-recanalization (n = 24)	*P*-value*
Lengths of relevant LSAs	Baseline [mm]	19.1–26.2 (25.2)	20.6–24.6 (22.8)	0.51
	Follow up [mm]	28.1–34.0 (30.4)	19.9–27.9 (23.3)	**0.01**
Infarct size	Volume [mm^3]	646–1997 (794)	489–1644 (764)	0.54

*Mann-Whitney U-tests; parentheses, median.