Vascular Hyperintensity on Fluid-Attenuated Inversion Recovery Indicates the Severity of Hypoperfusion in Acute Stroke

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Background and Aim: Although fluid-attenuated inversion recovery vascular hyperintensities may be frequently seen in acute large-artery ischemic stroke, reports on their prognostic utility had been conflicting due to lack of quantitative evaluation of the perfusion status based on the signal intensity. We hypothesized that greater hyperintensity represents more severe hypoperfusion. *Methods:* Overall, 27 patients with acute occlusion of the proximal middle cerebral artery were divided into 2 groups, based on their signal intensity in the insular segment of middle cerebral artery on the affected side, relative to that of the insular cortex: the low signal intensity group (hypo- or isointense signals, n = 12) and the high signal intensity group (hyperintense signals, n = 15). Using dynamic susceptibility contrast magnetic resonance imaging, we assessed the time of the maximum value of the residue function and mean transit time, in the entire middle cerebral artery cortical area and diffusion-weighted imaging-Alberta Stroke Program Early Computed Tomography Score regions, including the corona radiata. *Results:* The high signal intensity group had significantly longer time of the maximum value of the residue function in all the diffusion-weighted imaging-Alberta Stroke Program Early Computed Tomography Score regions, except the M3 and M6 regions, and significantly longer mean transit time in the M1 and M4 regions. *Conclusions:* Quantitative analysis of the perfusion parameters revealed more severely compromised and widely disturbed perfusion status in the high signal intensity group than in the low signal intensity group.

Key Words: Ischemic stroke—brain infarction—magnetic resonance imaging—magnetic resonance angiography—perfusion imaging

Introduction

Under normal blood flow conditions, major cerebral arteries exhibit low signals, such as those seen in the cerebrospinal fluid on magnetic resonance imaging (MRI) fluid-attenuated inversion recovery (FLAIR). In acute ischemic stroke, bright signals in the distal branch of the affected vascular territory are frequently seen with severe stenosis or

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occlusion in the anterior circulation territory, especially in the main trunk of the middle cerebral artery (MCA). This MRI finding had been termed FLAIR vascular hyperintensity (FVH), which reflects stagnant blood flow.[1-13]

FVH had been used as a surrogate, in place of perfusion-weighted imaging (PWI), to reflect a large mismatch with the findings on diffusion-weighted imaging (DWI).[1] However, reports on the prognostic value of the presence of FVH had been controversial,[2-8] likely due to the differences among the studies in terms of the study population, time from onset to imaging, FLAIR imaging conditions, treatment modalities, study endpoints, and definitions and assessments of FVH. Based on the fact that the signal intensity of FVH mainly reflects blood flow velocity, which is closely related with the perfusion status, we hypothesized that a relatively high FVH signal intensity in the insular segment of the MCA (MCA-M2)[14] would reflect a relatively more severe hypoperfusion in patients with acute occlusion of the horizontal segment of the MCA (MCA-M1).[15] The MCA-M2 is the most proximal portion that runs almost perpendicularly in the axial plane to an MCA-M1 occlusion; this anatomy potentially makes the signal intensity of the MCA-M2 the most sensitive to an inflow effect.

In this study, we conducted a quantitative analysis of cerebral perfusion in patients with acute ischemic stroke using dynamic susceptibility contrast-MRI to test our hypothesis. Moreover, we investigated the ability of the FVH signal intensity in the MCA-M2 to estimate hypoperfusion of the entire MCA cortical area and the DWI-Alberta Stroke Program Early Computed Tomography Score (DWI-ASPECTS) regions,[16] including the corona radiata (CR).

Materials and Methods

Subjects

This retrospective study included consecutive patients with MCA-M1 occlusion who underwent MRI within 24 hours of the stroke onset but before treatment at Kuwana Hospital between April 2010 and December 2015. The patients were excluded if they had occlusion in the internal carotid artery, occlusion of other MCA segments with poor DWI quality, or differences in the imaging slice levels and angles between DWI and PWI. The ethics committee of Kuwana Hospital approved the use of patient data for this retrospective analysis and waived the need for patient consent, but written informed consent for MRI was obtained from the patients or their legal representative.

MRI Protocol

At our institution, the standard MRI protocol for acute ischemic stroke patients comprised DWI, FLAIR, time-of-flight magnetic resonance angiography (TOF-MRA), and dynamic contrast susceptibility PWI. A 1.5-T clinical MRI unit (MAGNETOM ESSENZ- Dot, Siemens, Erlangen, Bavaria, Germany) was used with a 6-channel head receiving coil. The parameters were as follows:

- *DWI*: b, 1000 s/mm^2; echo time (TE), 81 milliseconds; repetition time (TR), 3900 milliseconds; field of view (FOV), 230 mm; matrix, 142 × 142; slice thickness, 5 mm; slice interval, 1 mm; number of slices, 22; number of excitations (NEX), 1; and acquisition time (TA), 1 minute 45 seconds.
- *FLAIR imaging*: TE, 86 milliseconds; TR, 9000 milliseconds; inversion time, 2500 milliseconds; FOV, 230 mm; matrix, 150 × 320; slice thickness, 5 mm; slice interval, 1 mm; number of slices, 22; NEX, 1; and TA, 2 minutes 42 seconds.
- *TOF-MRA*: TE, 7.15 milliseconds; TR, 27 milliseconds; FOV, 150 mm; matrix, 225 × 256; slice thickness, .75 mm; number of slices, 142; slab thickness, 106.5 mm; NEX, 1; and TA, 6 minutes 43 seconds.
- *Dynamic contrast susceptibility PWI using the gradient-echo echo-planar imaging method*: TE, 32 milliseconds; TR, 1740 milliseconds; flip angle, 90°; FOV, 230 mm; matrix, 128 × 128; slice thickness, 5 mm; slice interval, 1 mm; number of slices, 19; and NEX, 1. Using a power injector, a 4-mL/s bolus of the contrast agent gadopentetate dimeglumine (.2 mL/kg) was administered for a 94-second serial imaging.

Assessment and Classification of the MRI and MRA Findings

Occlusions were evaluated by TOF-MRA. The MCA was divided into 4 segments: MCA-M1,[15] MCA-M2, opercular (MCA-M3), and cortical (MCA-M4).[14]

FVHs were defined as focal, tubular, or serpentine hyperintensities in the subarachnoid space, relative to those in the cerebrospinal fluid and corresponding with the typical arterial course.[1] Based on the signal intensity in each volume of region of interest (ROI) on SYNAPSE STD ver. 4.1 (FUJIFILM Medical Corp, Tokyo, Japan), FVHs were classified into 3 grades: FVH_{none}, which indicated the absence of a signal intensity in the suspected M2 portion; FVH_{low}, which indicated hypo- or iso-signal intensity, relative to that in the insular cortex; and FVH_{high}, which indicated higher signal intensity, relative to that in the insular cortex. The FVH_{none} was considered FVH negative, whereas the FVH_{low} and FVH_{high} were considered FVH positive. The FVHs of the MCA-M2 were assessed on both the affected and unaffected sides, at the level of the basal ganglia, according to the DWI-ASPECTS regions.[16] The entire cohort was divided into 3 groups, the no signal intensity group (FVH_{none} group), the low signal intensity group (FVH_{low} group), and the high signal intensity group (FVH_{high} group), according to the FVH signal intensity grade in the MCA-M2 of the affected cerebral hemisphere (Fig 1). Patients with multiple MCA-M2 areas with higher

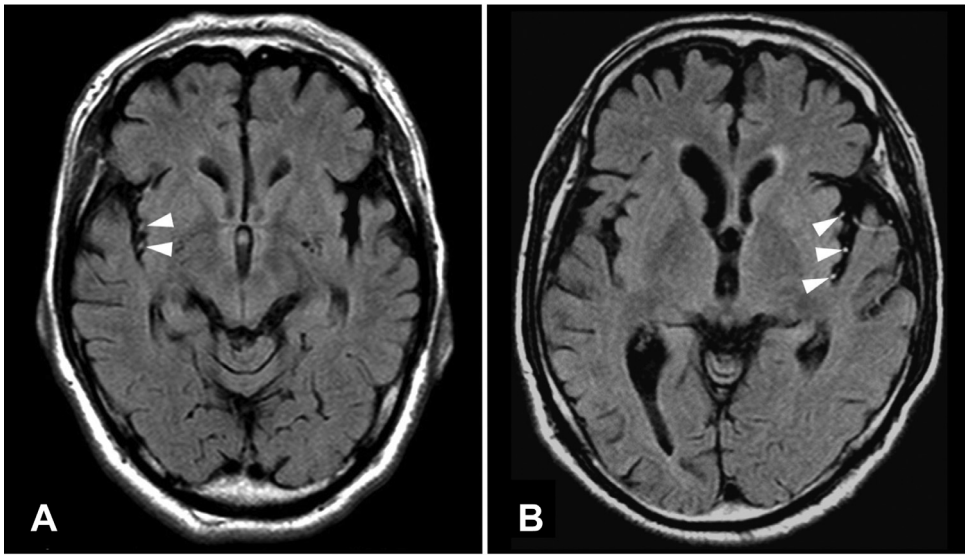

Figure 1. *FVH grading. (A) FVH_{low} grading: hypo- or iso-signal intensity relative to the insular cortex. The areas of FVH are indicated by arrowheads. (B) FVH_{high} grading: higher signal intensity relative to the insular cortex. The areas of FVH are indicated by arrowheads. Abbreviation: FVH, vascular hyperintensities on fluid-attenuated inversion recovery images.*

signal intensity relative to that in the insular cortex were assigned to the FVH_{high} group. The FVH-positivity rates were assessed for each MCA segment.

A board-certified neurosurgeon (T.N.) and a board-certified neuroradiologist (K.O.) performed the assessments and classifications, including the presence of MCA-M1 occlusion, occluded site, FVH signal intensity, and DWI-ASPECTS. FVH signal intensity was assessed by reviewers who were blinded to the DWI and PWI information. Any disagreement over conflicting conclusions was resolved by consensus.

PWI Software, Perfusion Parameters, and Procedures for Analysis

A Perfusion Mismatch Analyzer (PMA Ver. 5.2.0.2, ASIST-JAPAN; http://asist.umin.jp/), which automatically selected the arterial input functions based on the histograms of the maximum concentration, was used for PWI analysis. Using the perfusion maps calculated by the software, we analyzed the parameters, including time to the maximum of the residue function (Tmax), which reflected cerebral blood flow, and mean transit time (MTT), which reflected circulation time throughout the tissue.[17] The ROI for each parameter was manually set in the unaffected cerebral hemisphere on DWI-ASPECTS slices and in the mirror ROI in the affected hemisphere. In the current study, the perfusion parameters were measured in both the affected and unaffected cerebral hemispheres, and the corrected parameters were automatically calculated. The Perfusion Mismatch Analyzer software had been used in several recent PWI studies.[18-20]

To calculate parameters other than the Time to peak, we used block-circulant singular value decomposition to overcome the influence of delayed vascular transit time.[18] The DWI-ASPECTS regions used for ROI analyses were the lentiform (DWI-ASPECTS-L), insular ribbon (DWI-ASPECTS-I), and MCA cortical regions (ie, DWI-ASPECTS-M1, -M2, -M3, -M4, -M5, and -M6). In the PWI studies, we excluded small regions, such as the caudate and internal capsule, for which symmetrical ROI settings were challenging to obtain (Fig 2).

Statistical Analysis

Mann-Whitney U test was performed to compare the DWI-ASPECTS between the FVH_{low} and FVH_{high} groups. The Tmax and MTT of each DWI-ASPECTS region were compared between the 2 groups using unpaired t test. SPSS ver. 25 (IBM Corp., Armonk, New York, USA) was used for all statistical analyses, and P values less than .05 were considered statistically significant.

Results

Of the 37 consecutive patients enrolled, 10 were excluded due to poor DWI quality caused by head motion (n = 2) and differences in the imaging slice levels and angles between DWI and PWI (n = 8). The remaining 27 patients had MCA-M1 occlusion, DWI lesions, and MCA-M2 FVHs on the affected side. DWI-ASPECTS according to time after the onset is shown in Supplemental Figure 1. Five patients with unknown time of onset within 24 hours from last seen well were excluded. In the remaining 22 patients, there was no correlation between the time after the onset and DWI-ASPECTS (R = .355; P = .947). Fourteen patients were examined MRI within tissue plasminogen activator and 15 patients were within interventional radiology treatable times. There were 12 patients (44.4%) in the FVH_{low} group and 15 patients (55.6%) in the FVH_{high} group. Only 2 patients (7.4%) had FVH in the MCA-M2 on the unaffected side without severe M1 stenosis

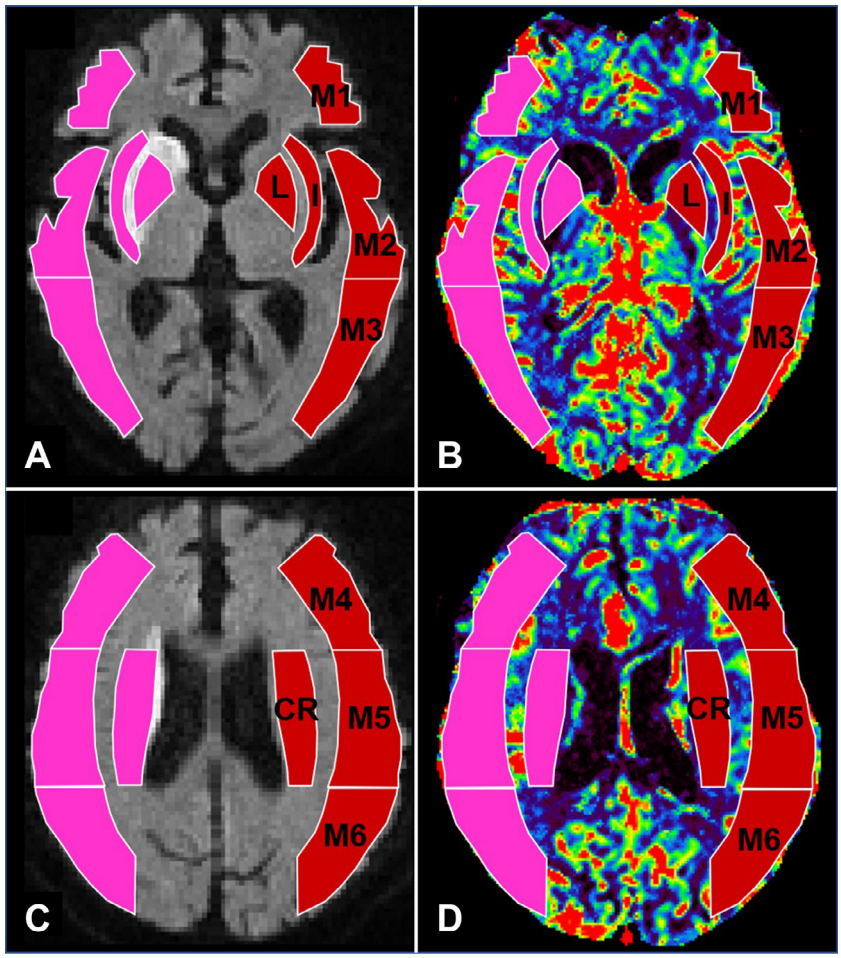

Figure 2. *Setting of the ROIs for the DWI-ASPECTS regions. (A) The ROIs of the L, I, and M1-M3 regions are manually set in the unaffected hemisphere (red). DWI with the automatically mirrored ROIs in the affected hemisphere (pink), at the level of the basal ganglia, is shown. (B) The ROIs are automatically transferred on the PWI at the level of the basal ganglia. (C) The ROIs of the M4-M6 and CR regions are manually set in the unaffected hemisphere (red). DWI with the automatically mirrored ROIs in the affected hemisphere (pink), at the level of the lateral ventricle above the basal ganglia, is shown. (D) The ROIs are automatically transferred on the PWI at the level of the lateral ventricle. Abbreviations: ASPECTS, Alberta Stroke Program Early CT Score; CR, corona radiata; DWI, diffusion-weighted imaging; I, insular ribbon; L, lentiform; M1-M6, cortical regions; PWI, perfusion-weighted imaging; ROIs, regions of interest.*

or DWI lesions, one is 77-year-old woman with subtle tubular hyperintensity, and the other is 66-year-old woman with subtle focal hyperintensity.

The clinical characteristics are shown in Table 1. The percentage of atrial fibrillation and embolism of the FVH_{high} group are significantly higher than that of the FVH_{low} group. Patients were classified into 2 groups according etiology: Embolism group (19 patients); Atherosclerosis group (8 patients).

Etiology and Tmax

The Tmax values in the entire MCA cortical region (P = .0297), DWI-ASPECTS-L (P = .0163), -I (P = .0019), -M1 (P = .0050), -M2 (P = .0068), -M4 (P = .0036), -M5 (P = .0241), and -CR (P = .0307) regions were significantly longer in the Embolism group than in the Atherosclerosis group (Supplemental Fig 2).

Etiology and MTT

The MTT values in the entire MCA cortical region (P = .0336), DWI-ASPECTS-I (P = .0058), -M1 (P = .0435), -M2 (P = .0185), -M4 (P = .0371), -M5 (P = .0154) regions were significantly longer in the Embolism group than in the Atherosclerosis group (Supplemental Fig 3).

MCA-M2 FVH Signal Intensity and the DWI-ASPECTS

Although not statistically significant, the mean score for the DWI-ASPECTS tended to be higher in the FVH_{low} group than in the FVH_{high} group (7.0 versus 5.4). The positive ratios of the DWI-ASPECTS-defined region in the FVH_{low} and FVH_{high} groups are shown in Figure 3. In all regions, except the DWI-ASPECTS-M6, DWI lesions were more frequently observed in the FVH_{high} group than in the FVH_{low} group.

Table 1. *Clinical characteristics*

	All patients (n = 27)	FVHlow group (n = 12)	FVHhigh group (n = 15)	*P* value (FVHlow vs FVHhigh)
Demographic data and comorbidities				
Gender (F/M)	15/12	7/5	8/7	
Age	77.1 ± 11.2	75.1 ± 10.6	78.7 ± 11.3	.509
Hypertnsion	15 (55.6%)	6 (50.0%)	9 (60.0%)	.603
Diabetes mellitus	6 (22.2%)	3 (25.0%)	3 (20.0%)	.756
Dyslipidemia	6 (22.2%)	4 (33.3%)	2 (13.3%)	.214
Atrial fibrillation	16 (59.3%)	4 (33.3%)	12 (80.0%)	**<.05 (.0142)**
Etiology				
Embolism	19 (70.3%)	6 (50.0%)	13 (86.7%)	**<.05 (.0381)**
Cardioembolism	16 (59.3%)	4 (33.3%)	12 (80.0%)	**<.05 (.0142)**
Artery-to-artery embolism	2 (7.4%)	2 (16.6%)	0 (0.0%)	.13
Embolic stroke of undetermind source	1 (3.7%)	0 (0.0%)	1 (6.7%)	.36
Atherosclerosis	8 (29.6%)	6 (50.0%)	2 (13.3%)	**<.05 (.0381)**

Abbreviation: FVH, vascular hyperintensities on fluid-attenuated inversion recovery images.
Data are numbers with percentages in parenthesis or means ± standard deviation.
P values less than 0.05 were indicated in bold.

MCA-M2 FVH Signal Intensity and Tmax

The mean Tmax values in the entire MCA cortical region (*P* = .046), DWI-ASPECTS-L (*P* = .0311), -I (*P* = .0285), -M1 (*P* = .0448), -M2 (*P* = .0154), -M4 (*P* = .0297), -M5 (*P* = .0297), and -CR (*P* = .0374) regions were significantly longer in the FVH_{high} group than in the FVH_{low} group (Fig 4).

MCA-M2 FVH Signal Intensity and MTT

The mean MTT values in the DWI-ASPECTS-M1 (*P* = .0018) and -M4 (*P* = .0264) regions were significantly longer in the FVH_{high} group than in the FVH_{low} group (Fig 5).

Discussion

In the current investigation, quantitative analysis of the perfusion parameters in each DWI-ASPECTS region revealed more severely compromised and widely disturbed perfusion status in the FVH_{high} group than in the FVH_{low} group. The Tmax, commonly used as a surrogate marker of cerebral blood flow[21] in almost all regions, except DWI-ASPECTS-M3

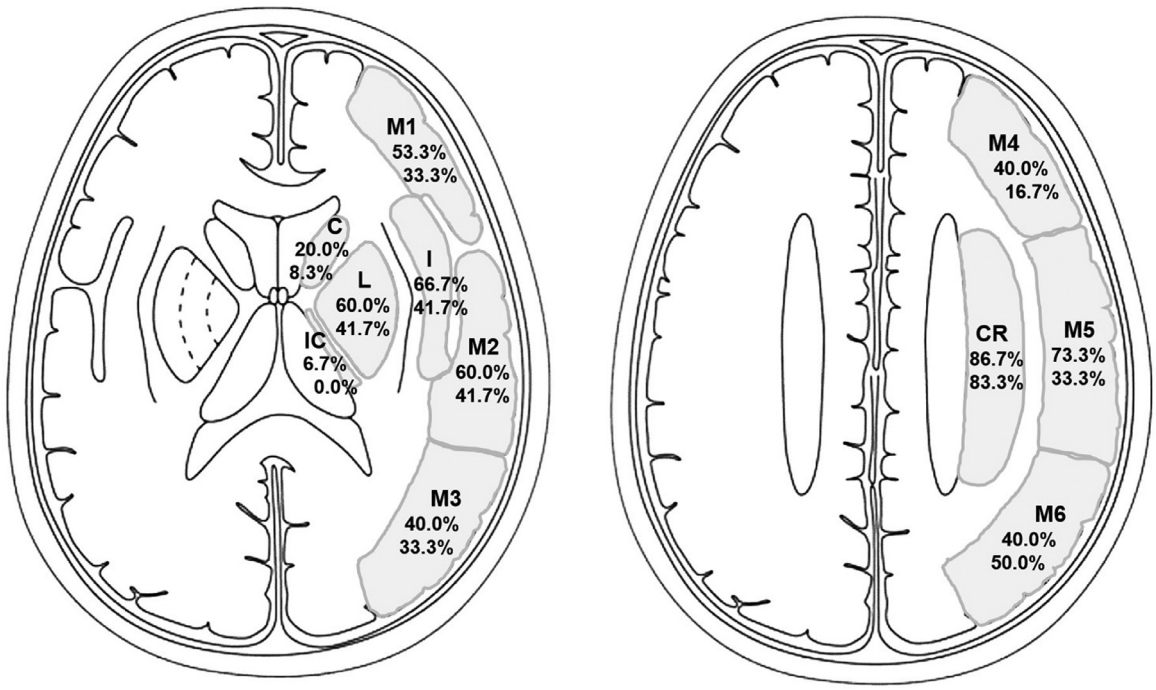

Figure 3. *Positive rate of each DWI-ASPECTS region, according to the FVH signal intensity. The positive rate in each region is shown in the middle column for the FVH_{high} group and in the lower column for the FVH_{low} group. In all regions, except the DWI-ASPECTS-M6, DWI lesions were more frequently observed in the FVH_{high} group than in the FVH_{low} group. Abbreviations: ASPECTS, Alberta Stroke Program Early CT Score; DWI, diffusion-weighted imaging; FVH, vascular hyperintensities on fluid-attenuated inversion recovery images.*

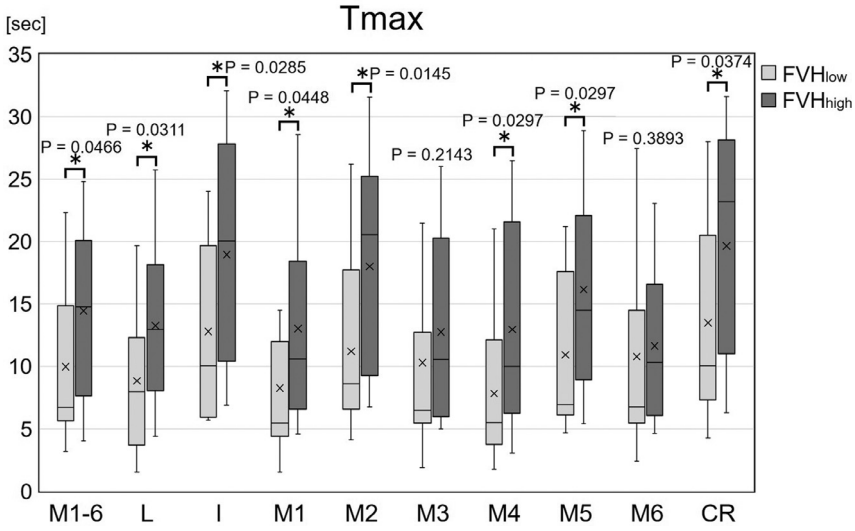

Figure 4. *Boxplots of the Tmax of the FVH_{low} and FVH_{high} groups, according to the DWI-ASPECTS region. Horizontal line inside the box indicates the median values, and the X symbol indicates the mean values. The Tmax values in the entire MCA cortical region, DWI-ASPECTS-L, -I, -M1, -M2, -M4, -M5, and -CR regions were significantly longer in the FVH_{high} group than in the FVH_{low} group. (*: P < .05.). Abbreviations: ASPECTS, Alberta Stroke Program Early CT Score; DWI, diffusion-weighted imaging; FVH, vascular hyperintensities on fluid-attenuated inversion recovery images; Tmax, time of the maximum value of the residue function.*

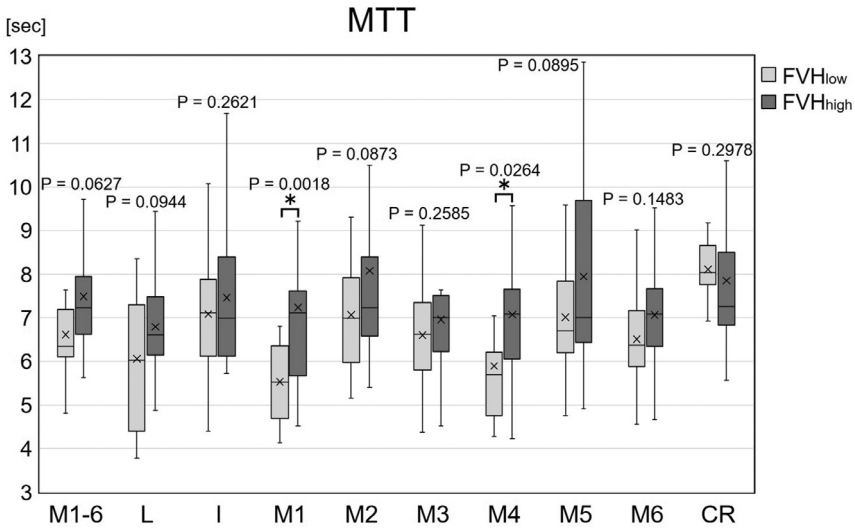

Figure 5. *Boxplots of the MTT of the FVH_{low} and FVH_{high} groups, according to the DWI-ASPECTS region. Horizontal line inside the box indicates the median values, and the X symbol indicates the mean values. The MTT values in the DWI-ASPECTS-M1 and -M4 regions were significantly longer in the FVH_{high} group than in the FVH_{low} group. (*: P < .05.). Abbreviations: ASPECTS, Alberta Stroke Program Early CT Score; DWI, diffusion-weighted imaging; FVH, vascular hyperintensities on fluid-attenuated inversion recovery images; MTT, mean transit time.*

or -M6 region, was longer in the FVH_{high} group than in the FVH_{low} group. This was also the case for the more proximal DWI-ASPECTS-M2 and -M5 regions, which were less likely to be reached by the collateral circulation.

However, in patients with acute MCA-M1 occlusion, FVH was detected at a much higher rate of 97% to 100%.[1-3] In the current study, the FVH-positivity rate was 100% in the MCA-M2 and MCA-M3. Relative to the axial plane, the MCA-M3 runs parallel, whereas the MCA-M2 runs perpendicularly. Therefore, the sensitivity of the FVH signal intensity to the inflow effects would be higher in the MCA-M2 than in the MCA-M3.

An experimental study, which used flow phantom analysis, showed that the FVH signal intensity was affected by the flow velocity on imaging parameters; in particular, a slower flow was associated with higher signal intensity.[22] Similarly, our quantitative analysis showed that the delays in Tmax and MTT were more severe in the FVH_{high} group than in the FVH_{low} group. Therefore, in MCA-M1 occlusion, high MCA-M2 FVH signals would reflect slower blood flow or retrograde collateral circulation in the affected vascular territory. In our study, subtle FVH was observed in the unaffected side in 2 elderly patients (77 years and 66 years) without severe M1 stenosis or DWI

lesions. Previous studies have confirmed that FVH was rarely detected at rate of 0% to 5.2% in patients who do not have stenosis and occlusion of cerebral vessels.[9,12,23]

Recent studies suggested that identification of FVHs beyond the boundaries of a DWI cortical lesion (ie, FVH-DWI mismatch) might be an easy and reproducible way to identify patients with a large penumbra (ie, a large PWI-DWI mismatch indicated large infarct growth).[1,2,6] In support of the previous research, our quantitative analyses showed extensive hypoperfusion in the DWI-ASPECTS regions, including the CR region, beyond the DWI lesions. We could not find any reports that used the Tmax or MTT parameters for quantitative assessment of brain perfusion status[17] in the DWI-ASPECTS regions, including the CR. Furthermore, no studies have quantitatively compared FVH-based hypoperfusion between the FVH_{low} and FVH_{high} groups. Our analyses suggested more severe hypoperfusion of a more extensive area in patients with FVH_{high} in the MCA-M2 than in those with FVH_{low} in the MCA-M2.

Therefore, failure to improve hypoperfusion by recanalization early after imaging can result in relatively worse outcomes in the FVH_{high} group, if the DWI lesions expand or additional DWI lesions appear over time. In the FVH_{low} group, the high DWI-ASPECTS indicated relatively less severe hypoperfusion in the FVH-DWI mismatch regions; these results implied more potentially salvageable regions in the ischemic penumbra, compared with those in the FVH_{high} group. Based on these results, categorizing patients as FVH_{high} or FVH_{low}, in the setting of acute MCA-M1 occlusion, might be helpful in quantitatively estimating the degree of hypoperfusion.

Summary and Conclusion

In acute cerebral ischemic stroke with occlusion of the MCA-M1, hypoperfusion of the MCA territory was more severe and extensive when the FVH signal intensity was higher in the MCA-M2 than in the cerebral cortex. Our results suggested that hypoperfusion can be quantitatively estimated based on the FVH signal intensity in the MCA-M2.

Conflict of Interest

None.

Acknowledgments

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Author Contribution Statement

Toshiharu Nomura, Kouichirou Okamoto, Hironaka Igarashi, and Yukihiko Fujii designed the study. Toshiharu Nomura, Kouichirou Okamoto, and Masato Watanabe contributed to the data collection. Toshiharu Nomura, Kouichirou Okamoto, Hitoshi Hasegawa, Makoto Oishi, and Yukihiko Fujii contributed to the analysis and interpretation of data. Toshiharu Nomura, Hironaka Igarashi, and Kouichirou Okamoto wrote the manuscript. Hironaka Igarashi, Masato Watanabe, Hitoshi Hasegawa, Makoto Oishi, and Yukihiko Fujii assisted in the preparation and critically reviewed the manuscript. All authors have approved the final version of the manuscript and agree to be accountable for all aspects of the work, ensuring that the questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Supplementary materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.jstrokecerebrovasdis.2019.104467.

References

1. Legrand L, Tisserand M, Turc G, et al. Do FLAIR vascular hyperintensities beyond the DWI lesion represent the ischemic penumbra? AJNR Am J Neuroradiol 2015;36:269-274.
2. Legrand L, Tisserand M, Turc G, et al. Fluid-attenuated inversion recovery vascular hyperintensities-diffusion-weighted imaging mismatch identifies acute stroke patients most likely to benefit from recanalization. Stroke 2016;47:424-427.
3. Liu D, Scalzo F, Rao NM, et al. Fluid-attenuated inversion recovery vascular hyperintensity topography, novel imaging marker for revascularization in middle cerebral artery occlusion. Stroke 2016;47:2763-2769.
4. Pérez de la Ossa N, Hernández-Pérez M, Domènech S, et al. Hyperintensity of distal vessels on FLAIR is associated with slow progression of the infarction in acute ischemic stroke. Cerebrovasc Dis 2012;34:376-384.
5. Hohenhaus M, Schmidt WU, Brunecker P, et al. FLAIR vascular hyperintensities in acute ICA and MCA infarction: a marker for mismatch and stroke severity? Cerebrovasc Dis 2012;34:63-69.
6. Kufner A, Galinovic I, Ambrosi V, et al. Hyperintense vessels on FLAIR: hemodynamic correlates and response to thrombolysis. J Neuroradiol 2015;36:1426-1430.
7. Girot M, Gauvrit JY, Cordonnier C, et al. Prognostic value of hyperintense vessel signals on fluid-attenuated inversion recovery sequences in acute cerebral ischemia. Eur Neurol 2007;57:75-79.
8. Cheng B, Ebinger M, Kufner A, et al. Hyperintense vessels on acute stroke fluid-attenuated inversion recovery imaging: associations with clinical and other MRI findings. Stroke 2012;43:2957-2961.
9. Sanossian N, Saver JL, Alger JR, et al. Angiography reveals that fluid-attenuated inversion recovery vascular hyperintensities are due to slow flow, not thrombus. AJNR Am J Neuroradiol 2009;30:564-568.
10. Kamran S, Bates V, Bakshi R, et al. Significance of hyperintense vessels on FLAIR MRI in acute stroke. Neurology 2000;55:265-269.
11. Noguchi K, Ogawa T, Inugami A, et al. MRI of acute cerebral infarction: a comparison of FLAIR and T2-weighted fast spin-echo imaging. Neuroradiology 1997;39:406-410.
12. Toyoda K, Ida M, Fukuda K. Fluid-attenuated inversion recovery intraarterial signal: an early sign of hyperacute

cerebral ischemia. AJNR Am J Neuroradiol 2001;22:1021-1029.
13. Liu W, Xu G, Yue X, et al. Hyperintense vessels on FLAIR: a useful non-invasive method for assessing intracerebral collaterals. Eur J Radiol 2011;80:786-791.
14. Gibo H, Carver CC, Rhoton AL, et al. Microsurgical anatomy of the middle cerebellar artery. J Neurosur 1981;54:151-169.
15. Zaidat OO, Yoo AJ, Khatri P, et al. Recommendations on angiographic revascularization grading standards for acute ischemic stroke: a consensus statement. Stroke 2013;44:2650-2663.
16. Barber PA, Hill MD, Eliasziw M, et al. Imaging of the brain in acute ischaemic stroke: comparison of computed tomography and magnetic resonance diffusion-weighted imaging. J Neurol Neurosurg Psychiatry 2005;76:1528-1533.
17. Copen WA, Schaefer PW, Wu O, et al. MR perfusion imaging in acute ischemic stroke. Neuroimaging Clin N Am 2011;21:259-283.
18. Zaro-Weber O, Livne M, Martin SZ, et al. Comparison of the 2 most popular deconvolution techniques for the detection of penumbral flow in acute stroke. Stroke 2015;46:2795-2799.
19. Huang YC, Liu HL, Lee JD, et al. Comparison of arterial spin labeling and dynamic susceptibility contrast perfusion MRI in patients with acute stroke. Plos One 2013;8:e69085.
20. Wang D, Zhu F, Fung KM, et al. Predicting cerebral hyperperfusion syndrome following superficial temporal artery to middle cerebral artery bypass based on intraoperative perfusion-weighted magnetic resonance imaging. Sci Rep 2015;5:14140.
21. Zaro-Weber O, Moeller-Hartmann W, Siegmund D, et al. MRI-based mismatch detection in acute ischemic stroke: optimal PWI maps and thresholds validated with PET. J Cereb Blood Flow Metab 2017;37:3176-3183.
22. Ahn SJ, Lee KY, Ahn SS, et al. Can FLAIR hyperintense vessel (FHV) signs be influenced by varying MR parameters and flow velocities? A flow phantom analysis. Acta Radiol 2016;57:580-586.
23. Zaro-Weber O, Moeller-Hartmann W, Siegmund D, et al. MRI-based mismatch detection in acute ischemic stroke: optimal PWI maps and thresholds validated with PET. J Cereb Blood Flow Metab 2017;37:3176-3183.