High-Resolution Vessel Wall MRI of Chronic Unilateral MCA Occlusion

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Introduction: There are few reports that high-resolution intracranial image by 3T MRI may be able to differentiate patterns of intracranial vascular wall pathologies (1-3). Like as intracranial artery stenosis, high-resolution vessel wall MRI may be useful for differentiating other causes of intracranial artery occlusion from atherosclerosis. An our previous study presented that MCA occlusion seen incidentally on TOF-MRA and any significant vascular lumen in the sylvian fissure were not seen on high-resolution vessel wall MRI (2). The etiology of MCA occlusion in this case may be different to atherosclerosis based on the surgical resection or pathologic findings of non-atherosclerotic cause of chronic MCA occlusion. Therefore, we hypothesized that, when high-resolution vessel wall imaging reveals the occluded MCA segment, MRI finding might characterize the morphology of occluded segment and be useful to evaluate the etiology of the chronic occlusion. In this study, we prospectively acquired the high resolution vessel wall MRI of MCA in patients with chronic unilateral MCA occlusion and evaluated the characteristics of MRI and clinical findings.

Methods and Materials: *Subjects*: We selected 17 consecutive patients (M:F = 8: 9; mean age = 63.7 years) who presented with unilateral MCA occlusion as documented by time-of-flight MR angiography (TOF-MRA). *MRI Scan*: High resolution vascular wall images were acquired both occluded MCA and contralateral MCA simultaneously in all patients using a 3T MRI. As the reference of TOF-MRA, the oblique sagittal planes were used with perpendicular to the axes of contralateral MCA or mirrored perpendicular of the occluded MCAs. The PD-weighted TSE sequence was used with imaging parameters: TR/TE = 2,500/30, FOV = 120 x 105 mm, matrix size = 320 x 220, ETL = 16, and NEX = 4. The black-blood (BB) technique with pre-regional saturation pulses of 80 mm thickness was used to saturate incoming arterial flow. The longitudinal coverage of each artery was 18–20 mm (9–10 slices). The voxel size for the BB-MR sequences was 0.38 x 0.48 x 2 mm; the reconstructed voxel size was 0.23 x 0.23 x 2 mm. *Imaging Analysis*: We measured the outer diameter of MCA at occluded segment and the same segment of contralateral MCA. The outer diameter was defined as the distance between outer interfaces of the vessels on vessel wall MRI. On TOF-MRA, occluded segment of MCA and the presence of steno-occlusive lesion in intracranial arterial segment were evaluated.

Results: Of 17 patients, 13 patients showed the vascular structure representing MCA, which was measurable with an electronic caliber. The mean diameters were 3.17±0.92mm and 3.46±0.65 mm for 13 occluded MCA and13 contralateral MCA, respectively. The range of diameter difference between occluded MCA and contralateral MCA was -23 to 46%. The other 4 patients did not have any significant vascular structure that represents MCA trunk in the sylvian fissure except small (diameter less than 1mm) lumen of vessels, although there was not any artifact or problem during imaging acquisition. According to the presence of MCA on vessel-wall MRI, MCA occlusion was classified two types: the plaqued MCA group that is shown in the clear demonstration of occluded MCA and atrophic MCA group that is no MCA in the sylvian fissure. In 13 plaqued MCA group, occluded segments of MCA were os 4, proximal 3, medial 2 and distal segment 2. Nine patients had stenoses in other intracranial arteries and 11 patients had the risk factors of atherosclerosis. Six patients presented symptom related occluded MCA and 2 patients showed small size of recent infarction. In all of atrophic MCA group, the occlusion segments were ostium 3 and proximal segment 1. There was no atherosclerosis at other intracranial arteries on TOF-MRA, but two patients had risk factors of atherosclerosis. They did not present any symptom or recent infarction relied on occluded MCA. Two patients showed old lacunar infarction or small size, subcortical infarction in the ipsilateral side.

Discussions: In present study, MRI was able to demonstrate clearly the occluded segment of intracranial artery and to measure the outer diameter of occluded vessel. This result indicated that vessel wall MRI might be used to evaluate the characteristics of the intracranial arterial occlusion. We suggest that four patients who were classified atrophic MCA group might have a different pathogenesis of MCA occlusion to atherosclerosis. There are few surgical and pathological findings to support our results (4,5). Some of chronic MCA occlusion showed hypotrophic or atretic proximal MCA trunk in operative finding, and these may be a manifestation of acquired or congenital etiology and different to atherosclerosis and moyamoya disease. We suggest that MRI finding of atrophic MCA occlusion group in present study would be consistent with the surgical findings that were previously described as non-atheromatous atrophic (or atretic) MCA occlusion. Non-atherosclerotic unilateral MCA occlusion with collateral arterial networks can manifest as a rupture of flow-related aneurysm in collateral arterial networks around occluded MCA.

Conclusions: This study proposed that vessel wall MRI might be a useful imaging tool that characterizes the MCA occlusion in vivo. We regarded the MCA occlusion with non-visualization of main MCA trunk on vessel wall MRI as the atrophic (atretic) MCA occlusion. This disease entity would be different from atherosclerotic occlusion, and has not been well known. When a patient had complex risk factors for multiple diseases or insufficient risk factors for atherosclerosis, comprehensive analysis including vessel wall MRI would be useful to diagnose the etiology of MCA occlusion.

Acknowledgement:

References: 1) Swartz RH, et al. *Neurology* 2009;72:627-634; 2) Ryu CW, et al. *Cerebrovasc Dis* 2009;27:433-442; 3) Kuker W, et al. *Cerebrovasc Dis* 2008;26:23-29; 4)Fukawa O, et al. *No shinkei geka* 1982;10:1303-1310; 5)Seki Y, et al. *Surgical neurology* 2001;55:58-62;

Fig.1. Atrophic MCA occlusion

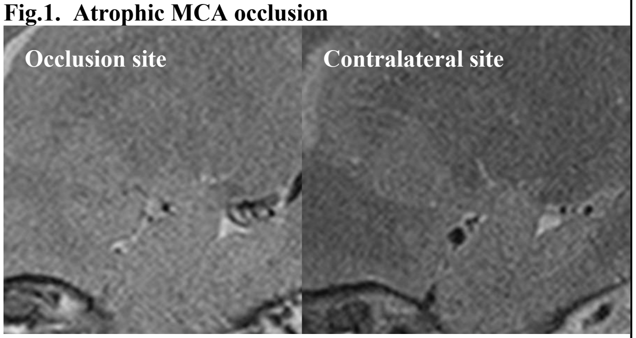

Fig.2. Plaqued MCA occlusion