EVALUATION OF ATHEROSCLEROTIC PLAQUES IN ACUTE ISCHEMIC STROKE OF MIDDLE CEREBRAL ARTERY TERRITORY USING HIGH-RESOLUTION INTRACRANIAL VESSEL WALL IMAGING

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Target audience: Neuroradiologist, neurologist, neurosurgeon

Purpose: Intracranial magnetic resonance (MR) angiography is widely used to diagnose cerebrovascular diseases such as infarction, vasculitis, and moyamoya disease. MR angiography noninvasively shows luminal narrowing of the intracranial arteries. However, most vascular diseases, especially atherosclerosis, do not show luminal narrowing in early stage. Recently, high-resolution (HR) intracranial vessel wall imaging is used to demonstrate intracranial arterial wall abnormality. HR intracranial vessel wall imaging clearly shows arterial wall pathologies such as wall thickening or enhancement at atherosclerosis, do not show luminal narrowing in early stage. Recently, high-resolution (HR) intracranial vessel wall imaging is used to demonstrate moyamoya disease. MR angiography noninvasively shows luminal narrowing of the intracranial arteries. However, most vascular diseases, especially intracranial atherosclerosis with stenosis is associated with acute ischemic stroke. However, not all atherosclerotic plaque is associated with acute ischemic stroke and even acute ischemic stroke occurred with non-stenotic atherosclerotic plaque. On the vessel wall imaging, plaque enhancement is considered as vulnerable plaque, and highly associated with acute ischemic stroke. However, in this study, enhancing wall thickening was frequently noted on the contralateral MCA. However, the plaque burden was larger on the ipsilateral MCA and the involved site was different. Ipsilateral MCA showed circumferential and superior wall thickening more frequently, but contralateral MCA showed anterior and posterior wall thickening more frequently. It is probably due to the MCA perforator is most commonly originate from superior wall of MCA, therefore, superior wall or circumferential atherosclerotic plaque may occlude perforator more likely.

Conclusion: High-resolution intracranial vessel wall imaging may reveal atherosclerotic plaque of vessel wall even the stenosis is not evident on the MR angiography. Atherosclerosis with luminal stenosis, circumferential or superior wall involvement and larger plaque burden were correlated with ipsilateral acute ischemic stroke.

Methods: Between May 2012 and October 2013, there were 225 patients with HR intracranial arterial wall imaging in our institution. Of them, 28 patients who had unilateral noncardiogenic acute ischemic stroke of MCA territory were included in this study (13 males; ages, 32-82 years; mean, 60 years). MRI was performed at 3.0 T (Achieva, Philips Medical Systems, Best, The Netherlands) with an 8-channel sensitivity-encoding (SENSE) head coil. Three-dimensional (3D), TOF MR angiography and contrast-enhanced T1-weighted volumetric isotropic TSE acquisition (VISTA) image were acquired with 0.5 mm isotropic voxels. Black-blood contrast was acquired by applying saturation band below the imaging slab. The type of infarction (striatocapsular or borderzone), presence of stenosis, location (proximal/middle/distal MCA), and type (anterior/posterior/inferior/superior wall or circumferential) of enhancing wall thickening of the ipsilateral and contralateral MCA were analyzed. Atherosclerotic plaque burdens were calculated from the sum of involved segment on the MCA.

Results: 67.9% (19/28) showed ipsilateral MCA stenosis. Stenosis of distal MCA was most common (47.4%). Borderzone infarction was more common than striatocapsular infarction with MCA stenosis (P=0.019). In the stenotic segment, circumferential wall thickening (11/19) was most common followed by superior wall thickening (4/19). In case of striatocapsular infarction, superior (43.8%) wall thickening was most common followed by circumferential (25%) wall thickening (Fig. 1, 2).

100% of ipsilateral MCA and 85.7% of contralateral MCA shows enhancing wall thickening. However, the burden of atherosclerotic plaque on the ipsilateral MCA was significantly larger than contralateral MCA (2.21 vs 1.68, P<0.008). On the ipsilateral MCA, the most significant wall thickening was shown on the middle segment (39.3%) and distal segment (39.3%). On the middle segment, circumferential (36.4%), superior (27.3%), and posterior (27.3%) wall thickening were frequently found, however, on the distal segment, circumferential (54.5%) and superior (36.4%) wall thickening were frequently found.

On the contralateral MCA, the most significant wall thickening was shown on the distal segment (37.5%) and proximal segment (33.3%). On the distal segment, posterior (77.8%) wall thickening were frequently found, however, on the proximal segment, anterior (62.5%) wall thickening were frequently found.

Discussion: Intracranial atherosclerosis with stenosis is associated with acute ischemic stroke. However, not all atherosclerotic plaque is associated with acute ischemic stroke and even acute ischemic stroke occurred with non-stenotic atherosclerotic plaque. On the vessel wall imaging, plaque enhancement is considered as vulnerable plaque, and highly associated with acute ischemic stroke. However, in this study, enhancing wall thickening was frequently noted on the contralateral MCA. However, the plaque burden was larger on the ipsilateral MCA and the involved site was different. Ipsilateral MCA showed circumferential and superior wall thickening more frequently, but contralateral MCA showed anterior and posterior wall thickening more frequently. It is probably due to the MCA perforator is most commonly originate from superior wall of MCA, therefore, superior wall or circumferential atherosclerotic plaque may occlude perforator more likely.

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References:

Fig. 1. 76-year-old female with left striatocapsular infarction. Left distal MCA (A) shows enhancing wall thickening of superior wall. Contralateral proximal MCA (B) shows anterior wall thickening, and middle MCA (C) shows no wall thickening.

Fig. 2. 60-year-old female with left borderzone infarction. Left proximal MCA (A) shows tight stenosis with circumferential enhancing wall thickening.