Response to Letter Regarding Article, “High-Resolution Magnetic Resonance Wall Imaging Findings of Moyamoya Disease”

We appreciate the constructive comments Shang et al provided on our report in Stroke, which evaluated high-resolution wall MRI (HR-MRI) findings of Moyamoya disease (MMD) compared with intracranial atherosclerotic disease. MMD exhibited distinct HR-MRI findings, concentric enhancement on bilateral distal internal carotid arteries, and shrinkage of middle cerebral artery, regardless of symptoms (presence or severity) or stage (Suzuki grade).

As Shang et al pointed out, the wall enhancement observed in our patients may have been because of luminal (and not wall) enhancement from slow-moving blood adjacent to the wall of the vessel (slow flow artifact). The suppression of intravascular signals is important in vessel wall imaging and various black-blood imaging techniques are used to achieve this, including spatial presaturation (commonly used with T1 fluid-attenuated inversion recovery or volumetric isotropic turbo spin-echo acquisition) and double inversion recovery preparation. The double inversion recovery preparation sequence is used widely for black-blood imaging and generally provides good image quality. However, the multisection double inversion recovery turbo spin-echo sequence requires a complicated section-interleaving implementation, which can compromise black-blood imaging efficacy. Furthermore, the double inversion recovery method is not easily adaptable for large field-of-view 3-dimensional black-blood imaging because of outflow limitations. To address this problem, new techniques such as improved motion-sensitized driven equilibrium and delay alternating with nutation can be used.

Recently, we switched from spatial presaturation to improved motion-sensitized driven equilibrium sequences for black-blood imaging, and further studies with this improved technique are ongoing. In addition, several findings of our study suggest that enhancement on distal internal carotid artery/middle cerebral artery was caused by true wall enhancement rather than slow flow artifacts. First, in patients with intracranial atherosclerotic disease, enhancement was rarely observed on the asymptomatic stenosed segment. Second, in patients with MMD, enhancement was often observed on the nonstenosed segment on time of flight MRA, usually with mild concentric wall thickening either on the asymptomatic side or on the nonstenosed distal internal carotid artery in conjunction with bright signal, which suggests fast arterial flow, not slow flow.

The mechanisms of vessel wall enhancement in MMD remain controversial. Circumscribed enhancement of the distal internal carotid artery on symptomatic or asymptomatic segments may represent active angiogenesis or hyperproliferation of the vessel wall component, such as smooth muscle cell migration/proliferation or fine vascular networks. Other data indicate that far advanced MMD (Suzuki grade 5 or 6) also exhibited a similar pattern of enhancement. Therefore, increased angiogenesis may not be the only cause of vessel wall enhancement in MMD. In addition, blood–brain barriers were fragile in patients with MMD, and enhancement may be related to fragile blood–brain barriers. Further studies with histological confirmation are required. Shang et al also suggested studying the association between HR-MRI findings and ACTA2 mutation. Although ACTA2 mutation analysis was not performed in our study, it would be helpful to establish the mechanism of vessel enhancement in MMD in future studies.

The diagnosis of MMD is reliant on angiography-based criteria, and this has inherent limitations, including the absence of basal collaterals in early or far advanced MMD and a lack of biomarkers. Our study revealed that HR-MRI could be an imaging biomarker specific to MMD. Further study is warranted to develop new diagnostic criteria for MMD using more objective and consistent factors such as HR-MRI findings and genetic variation (ongoing trial, NCT02074111).

Sources of Funding

This study was supported by the National Research Foundation of Korea, Ministry of Science, ICT (Information, Communication, Technology) and Future Planning (2011-0019389).

Disclosures

None.

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*Stroke*. 2014;45:e300; originally published online October 14, 2014;
doi: 10.1161/STROKEAHA.114.007219

*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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