Letter to the Editor

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Letter by Shang et al Regarding Article, “High-Resolution Magnetic Resonance Wall Imaging Findings of Moyamoya Disease”

To the Editor:

Our group read the study by Ryoo et al with great interest. They applied a vessel wall high-resolution MRI technique and were able to distinguish the vessel wall enhancement pattern in Moyamoya disease (MMD) from intracranial atherosclerotic disease. Concentric or focal eccentric enhancement of the internal carotid artery or middle cerebral artery was observed in patients with MMD or patients with symptomatic intracranial atherosclerotic disease, respectively. Authors proposed that the concentric enhancement in MMD could represent the hyperproliferation of vessel wall components, whereas the focal eccentric enhancement in intracranial atherosclerotic disease could represent atherosclerotic plaques.

We agree that different vessel wall enhancement patterns could represent distinct underlying pathology in various intracranial artery diseases. A more in-depth discussion elucidating the patterns and mechanisms of enhancement may be of interest. It is well accepted that intracranial arteries are distinguished from extracranial arteries by the absence of vasa vasorum. When intracranial arteries are diseased, such as in atherosclerosis, vasa vasorum develop in the vessel wall and contribute to the inflammation and eccentric enhancement in intracranial atherosclerotic disease. However, such pathological changes have not been observed in MMD, and the enhancement pattern in MMD has not been consistent. Prior studies have demonstrated that ≈70% of MMD cases lacked any enhancement, which is in contrast to the results of the current study. The reason of this discrepancy could be related to the presence of slow-flow artifacts or differences in MRI acquisition protocols between studies. Of note, the study from Kim et al measured enhancement at the middle cerebral arteries while the current study described enhancement predominantly in the supraclinoid internal carotid arteries. The study from Aoki et al focused mainly on the internal carotid arteries but used 2-dimensional acquisitions with likely thicker slice profiles, creating a potential limitation to the sensitivity of enhancement detection because of partial volume artifacts.

The nature of vessel wall enhancement in MMD is another point of debate. An inflammatory process has been proposed in the pathogenesis of MMD but remains controversial. Whether inflammation could result in vessel wall enhancement in MMD requires further investigation. The hyperproliferation of vessel wall components is likely the mechanism of enhancement, and it is specifically the proliferation of moyamoya vessels in the vessel wall. This vascular network decreases in Suzuki stage 5 and disappears in stage 6. In the current study, the enhancement patterns observed between Suzuki stages 1 to 3 and stages 4 to 6 were not significantly different. It would have been insightful for the authors to compare the differences of enhancement patterns between Suzuki stages 1 to 4 and stage 5 to 6, or between stage 1 to 5 and stage 6. It would be also interesting to study patterns of vessel wall enhancement in MMD associated with the ACTA2 mutation. Lack of moyamoya vessels, despite classic large vessel steno-occlusion, was observed in patients with ACTA2 mutation. Decreased vessel wall enhancement at Suzuki stage 5, or absence of enhancement at stage 6 in ACTA2 mutation, would further support the contribution of moyamoya vessels to vessel wall enhancement. Because of the complexity of MMD pathogenesis, more research is needed for reliable analysis of vessel wall enhancement, and as the authors suggested, encourage the use of double-inversion recovery techniques to more confidently mitigate slow-flow effects.

Disclosures

None.

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