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Reply:

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REPLY:

We appreciate the comments by Drs Mori and Mugikura and thank the editor for the opportunity to reply to those comments.

As suggested by Drs Mori and Mugikura, our results may "revealed different underlying pathophysiologies" between the two imaging modalities. Until today, most studies support the hypothesis that FLAIR vascular hyperintensity (FVH) represents slow or turbulent flow through the engorged leptomeningeal collaterals (LMCs).[1] As to contrast-enhanced MR imaging (CEMR), a previous study suggested that the pial vascular enhancement in Moyamoya disease could be attributed to a decrease of flow velocity–related signal losses and spin-dephasing effects, which consequently induce high intensity of the vessel from the mixture of blood and contrast medium.[2] The underlying pathophysiology of the ivy sign on CEMR and FLAIR still needs future validation to explore the association of visible imaging findings and physical mechanisms.

The main objective of our study was to develop a quantitative method on both CEMR and FLAIR and to compare the ability of these two imaging modalities in reference to DSA. As mentioned by Drs Mori and Mugikura in the letter, they found that the distribution of FVH was more frequently seen in the anterior MCA region. Furthermore, they found that LMCs in the anterior and posterior regions could also change along with the Suzuki stages in Moyamoya disease.[3] ASPECTS is a widely accepted approach for brain region segmentation in cerebrovascular diseases. In this study, we used this approach and designed a total ivy sign score. As a result, no statistical comparison concerning the distribution of the ivy sign was performed. Nevertheless, in the process of scoring, no obvious visible difference in the distribution of the ivy sign between CEMR and FLAIR was found.

To our knowledge, the relationship between FVH and hemodynamic indicators, cerebrovascular reserve (CVR) and CBF for example, showed different results in previous literature.[4,5] One possible explanation is the result of our study. Because the presence of FVH seems to be associated with the clinical phase of the ischemic lesion in Moyamoya disease, patients with different clinical phases enrolled in the previous studies may affect the consistency. Another possible reason is the different hemodynamic indicators used. Compared with CBF, which was used in our study, CVR may be more sensitive for reflecting hemodynamic changes. Physically, the CBF value will decrease when the cerebral vessels reach the maximum of vasodilatory ability.

Another comment was about the correlation between the ivy sign score on CEMR and CBF in the late Suzuki stage. Drs Mori and Mugikura mentioned that posterior circulation involvement is often seen in this phase. In our study, we used posterior cerebral artery involvement as a covariant to address the impact of this confounding factor. We completely agree that the ivy sign on CEMR may be attributed to the maximally dilated pial vasculature, which includes both arterioles and venules. Although we assume that the imbalance between LMCs and abnormal ICA and Moyamoya vessels may be one possible explanation, the underlying mechanism of the negative relationship between the ivy sign score on CEMR and CBF in the late Suzuki stage is still to be determined. Future studies with larger sample sizes, sensitive hemodynamic indicators and advanced imaging processing approach are needed to confirm the current results.

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