Response by Chen et al to Letter Regarding Article, “Magnetic Resonance Imaging of Plaque Morphology, Burden, and Distribution in Patients With Symptomatic Middle Cerebral Artery Stenosis”

In Response:

We appreciate the opportunity to respond to comments by Du et al about our recent article,1 which was a hospital-based pilot study using high-resolution magnetic resonance imaging (HRMRI) to investigate the total intracranial plaque burden in patients with symptomatic middle cerebral artery stenosis. HRMRI is a promising imaging modality to visualize intracranial arterial wall pathology, yet to be validated for interpreting the clinical significance of signal changes.

Du et al suggest that quantitative MRI measurements should be performed. We agree that morphological measurements of intracranial vessels are feasible, but not compulsory for every study involving HRMRI technology. The ultimate purpose of HRMRI study is to identify proper imaging biomarker for predicting clinical outcome rather than for achieving accurate quantitative values of different measurements. Therefore, the qualitative measurements may prove a rapid visual score method that can be applied in routine clinical practice by radiologists or stroke physicians, which is a reasonable alternative for time-consuming quantitative measurements in some circumstances.

We agree that plaque vulnerability is a meaningful and hot topic when talking about atherosclerotic disease. Determining plaques prone to rupture (plaque vulnerability) might help to identify patients at risk for myocardial infarction or stroke. Pathologically, the features of plaque vulnerability include thin cap fibroatheroma, large lipid core, spotty intimal calcification, positive remodeling, and intraplaque neovascularization. Unfortunately, it is challenging to translate the pathological definition of plaque vulnerability directly to identify the vulnerable plaques by using HRMRI. For several years, the pathology of intracranial atherosclerosis had been understudied because of relatively inaccessibility of cerebral artery specimens under current treatment strategies. Acquiring postmortem cerebral vessel specimens for histology processing is the most direct method to analyze the pathological features intracranial atherosclerosis.2 We performed a series of postmortem study by collecting cerebral artery specimens and for the first time demonstrated that percent-age of lipid area and the presence of intraplaque neovasculature may play a key role in contributing to ischemic stroke, in addition to plaque-induced luminal stenosis.3

Our findings indicate that specific components within intracranial atherosclerotic plaques could predict plaque vulnerability and downstream ischemic vascular event within intracranial cerebral arteries. Our subsequent studies on the same series of cerebral artery specimens validated high signal on T1-weighted sequence to be intraplaque hemorrhage4 and low signal on T1-weighted fat-suppressed images5 to be lipid core with intracranial atherosclerotic plaques by MRI-histology comparative study. However, the definition of plaque vulnerability by using HRMRI is complex and still under exploration. Therefore, it is impossible to define plaque vulnerability only depending on the intraplaque heterogeneous signal intensity or plaque enhancement as Du et al suggested.

Considering the comment by Du et al on our seemingly inconsistent finding of no correlation between plaque enhancement and symptomatic presentation, the broad recruitment of patients with both acute and chronic stroke would like to offer reasons that might explain, as stated clearly in our discussion part1 and repeated by Du et al. We hope that more studies of validating HRMRI in assessing intracranial arterial disease and predicting stroke risk can now follow.

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Disclosures

None.

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