On MR Imaging of the Intracranial Vessel Wall

Intracranial arterial stenosis or occlusion accounts for 9-15% of ischemic strokes in the United States, and an even higher proportion of strokes in Asian populations. Conventionally, intracranial steno-occlusive disease has been characterized using CT angiography, MR angiography, and catheter angiography. These techniques depict the effect of disease on the arterial lumen, but do not directly image the underlying pathology which most commonly involves the vessel wall. There has been burgeoning interest in MR imaging of the carotid vessel wall to differentiate between stable and unstable atherosclerotic plaque. For the (relatively smaller) intracranial arteries, 3 Tesla MR systems are much better than 1.5 Tesla MR systems, as the former provide greater signal-to-noise, enabling imaging with the spatial resolution needed to characterize the intracranial arterial wall. The MR vessel wall imaging may allow differentiation between stable and unstable intracranial atherosclerotic plaque. Furthermore, intracranial stenosis occurs secondary to a broad range of pathologies (such as atherosclerosis, vasculitis, drug-induced vasculopathy, and Moyamoya disease), with differing management, and vessel wall imaging may aid in differentiating among these.

In this issue of the Canadian Journal of Neurological Sciences, Payne et al present contrast-enhanced T1-weighted images to depict vessel wall pathology, but a variety of non-enhanced sequences may be used as well. Carotid vessel wall MR imaging has used a combination of tissue weightings (T1, T2, proton density) to identify fibrous cap, intraplaque hemorrhage, calcification, and lipid core, that is, the components of atherosclerotic plaque. In the carotid arteries, there is evidence that intraplaque high signal intensity corresponds with recent intraplaque hemorrhage, and indicates increased risk of future transient ischemic attack or stroke. In a post-mortem study, MCA plaques associated with infarction had a higher prevalence of neovascularisation and intraplaque hemorrhage than plaques not associated with infarction. Ryu et al demonstrated focal areas with differing MR signal intensities within MCA plaques in vivo, but without histopathological correlation. High resolution non-enhanced T1-weighted images are certainly helpful for the diagnosis of intracranial arterial dissection.

Basic implementation of intracranial vessel wall imaging is not difficult, and may be accomplished using standard MR pulse sequences. This is more easily accomplished on 3 Tesla MR systems due to higher intrinsic signal-to-noise than on 1.5 Tesla systems. For an institute interested in using this technique, a starting point might be to run a T1-weighted 2D FLAIR fast spin echo sequence (slice thickness 3 mm, matrix size 512 x 512) in plane and perpendicular to the vessel of interest, before and after intravenous administration of gadolinium.

Unlike carotid artery disease, for which endarterectomy specimens are available, intracranial vessel wall specimens are rare, and progress may largely depend on detailed correlation of serial vessel wall imaging with clinical and laboratory findings. Further development of dedicated MR pulse sequences, and further study of the breadth of patients with intracranial disease will almost certainly lead to both a better understanding of those
diseases affecting the intracranial arteries, and a clinically important diagnostic tool.

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REFERENCES