Intracranial Arterial Wall Imaging using 3D Isotropic High Resolution Black Blood MRI at 3.0 T

Ye Qiao1, Steve R Zeiler2, Saeedeh Mirbagheri1, Richard Leigh2, Victor Urrutia2, Robert Wityk2, and Bruce A Wasserman1

1Radiology, Johns Hopkins University, Baltimore, Maryland, United States, 2Neurology, The Johns Hopkins Hospital, Baltimore, Maryland, United States

TARGET AUDIENCE: Scientists and clinicians who are interested in using MRI for identifying intracranial atherosclerosis and stroke risk.

PURPOSE: Intracranial atherosclerotic disease (ICAD) is a major cause of stroke worldwide and is responsible for 8-10% of strokes in the US1. Histologic studies of postmortem specimens2-3 have revealed a strong inflammatory response in culprit ICAD plaques (i.e., lesions responsible for ischemic events), reflecting increased macrophage infiltration and neovascularity, and that the degree of inflammation might influence the likelihood of a stroke. We sought to characterize intracranial plaque inflammation in vivo using 3D high-resolution contrast-enhanced black blood MRI imaging (BBMRI) and investigate its relation to cerebrovascular ischemic events.

METHODS: Twenty-two patients (18 male; mean age 57.7±12.4 years) with cerebrovascular ischemic events (16 acute, 1 subacute, and 3 chronic strokes; 2 transient ischemic attacks) underwent 3D time-of-flight MRA and contrast-enhanced BBMRI examinations for intracranial atherosclerotic disease at 3T. The 3D BBMRI sequence was acquired using a volumetric isotropic TSE acquisition (VISTA) in a coronal plane (40-mm-thick slab) optimized for flow suppression and intracranial vessel wall delineation4. The following parameters were used: TR/TE, 2000ms/38ms; TSE factor, 56 echoes; echo spacing, 6.1ms; sense factor, 2; number of averages, 1; acquired resolution, 0.4x0.4x0.4 mm3; scan time, 7.5 minutes. The BBMRI images were repeated 5 minutes after contrast administration. Each identified plaque was classified as culprit, probably culprit or non-culprit based on its likelihood to cause the presenting stroke or TIA symptoms. Plaque enhancement was categorized on BBMRI (Figure 1), and the degree of enhancement was calculated.

RESULTS: Seventy plaques were identified in 16 acute stroke patients (18 culprit, 12 probably culprit, and 40 non-culprit plaques). Among the 70 plaques identified in acute stroke patients, 57 enhanced (i.e., grade 1 or 2). All 18 culprit plaques enhanced (11% grade 1, 89% grade 2), all 12 probably-culprit plaques enhanced (75% grade 1, 25% grade 2), and 27 (68%) non-culprit plaques enhanced (45% grade 1, 23% grade 2) (Table 1). For these acute patients, grade 2 enhancement was associated with culprit plaques (OR 21.7, 95% CI: 2.6-178.2 compared with grade 0), and this was independent of its thickness. Plaque enhancement (grades 1 and 2) persisted beyond the acute stage (>4 weeks). A lack of enhancement (13, grade 0) was observed only in non-culprit plaques.

CONCLUSION: Contrast-enhancement of intracranial atherosclerotic plaque is associated with its likelihood to have caused a recent ischemic event and may serve as a marker of its stability, providing important insight into stroke risk.

DISCUSSION: Contrast enhancement of extracranial atherosclerotic plaque is an established feature of inflammation and risk for plaque disruption. In this study, we applied a 3D contrast-enhanced MRI technique to compare culprit and non-culprit intracranial lesions to determine if enhancement might also serve as a marker of intracranial plaque instability and stroke risk. We found that strong contrast enhancement was associated with culprit plaques whereas the lack of enhancement was detected only in non-culprit plaques. This 3D technique is easy to implement in clinical practice and has great potential as a diagnostic tool to identify intracranial plaque vulnerability and assess the effectiveness of new therapies.