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

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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 US [1]. Histologic studies of postmortem specimens [2-3] 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 delineation[4]. 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.

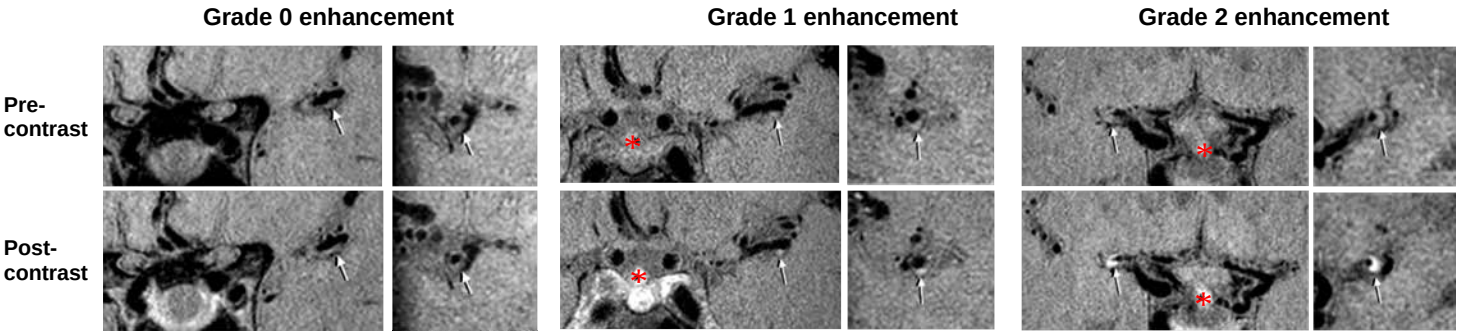

Figure 1. Categorize plaque enhancement on pre-contrast and post-contrast 3D VISTA images. Grade 0 enhancement, enhancement ≤ that of normal arterial walls seen elsewhere; grade 1: enhancement above grade 0 but less than that of the pituitary infundibulum (*); grade 2: enhancement ≥ that of the infundibulum (*).

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.

Table 2. Enhancement Grade of Non-culprit, Probably culprit and Culprit Plaques in Acute Stroke Patients

	Grade 0	Grade 1	Grade 2	Total
Non-culprit	13	18	9	40
Probably-Culprit	0	9	3	12
Culprit	0	2	16	18

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.

REFERENCES: [1] Wityk RJ, et al., Stroke 1996; 27:1974-1980. [2] Chen XY., Cerebrovascular Dis 2008; 25:74-80. [3] Labadzhyan A et al., J Neurol Sci 2011; 97-99. [4]. Qiao Y, et al., JMRI 2011; 72:627-634**.**