MRI of Plaque Components and Fibrous Cap Inflammation in Abdominal Aortic Aneurysm

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Introduction
The ability to image plaque components including inflammation would be a major advance in the identification of the vulnerable plaque. MRI has recently shown potential for the non-invasive evaluation of plaque constituents such as the fibrous cap (1,2), lipid core, calcified plaque, intimal-medial hyperplasia, and the site of plaque rupture (3). A previous study suggests that the extent of gadolinium (Gd-DTPA) enhancement is related to the amount of inflammatory activity in the plaque (4). We hypothesized that MRI could identify components of atherosclerotic plaque in patients with an abdominal aortic aneurysm (AAA) and that imaging techniques including contrast-enhancement could identify regions of inflammation as validated by histopathology.

Methods
Patients undergoing grafting of abdominal aortic aneurysms were recruited into the study prior to surgery. Imaging was performed on a Siemens Vision 1.5T MR scanner using a 4-element phased-array coil around the abdomen. After scout imaging to define a transverse plane demonstrating significant plaque burden within the aneurysm, T1-weighted (T1-W) images were acquired in the plane with a cardiac-gated, breathhold, segmented, turbo spin echo (TSE) sequence with TR of 1 x RR interval, TE 32 ms, 180° refocusing RF pulse. T2-W images were obtained in the same plane using a similar TSE sequence but with a longer effective TE (80 msec), and a longer TR (2 x RR interval). All sequences contained a dark blood preparation pulse (selective 180°/non-selective 180°) to remove artifacts from blood and were performed with and without fat saturation (FS). Field of view was 320 mm and matrix 204 x 512, yielding an interpolated pixel size of 0.62 x 0.62 mm2 with a 7 mm slice thickness. A 3D-Gd-DTPA enhanced MRA of the aneurysm (TR 4 ms, TE 1.6 ms, slab thickness 96 mm with 48 partitions, 30° flip angle, 360 mm FOV, 160 x256 matrix and 2 NEX) was performed during infusion of 0.1mMol/kg Gd-DTPA. The T1-W images were repeated after Gd-DTPA infusion, both with and without fat saturation (FS). The surgeon was shown the site of MR imaging relative to landmarks (e.g. renal arteries) in order to localize the region of the aneurysm to resect at the time of surgery. During surgery, the aneurysm was opened longitudinally. A 1 cm thick cross-section of the aorta at the landmarked location was resected for histopathologic examination. The surgeon marked the anterior surface of the aneurysm with a suture. Each specimen was hung on a whole tissue mount and photographed. Tissue sections were fixed and stained for various plaque constituents. The pathologist blinded to the MRI findings assessed the plaques histologically for the presence of a fibrous cap, inflammation in the cap and adventitia, number of layers of thrombus and plaque, and extent of lipid. Two investigators blinded to the pathologic findings analyzed the number of visualized layers within the plaque by MRI. One investigator then analyzed the MR images with Image J software (NIH). Signal intensity (SI) was averaged from 3 ROIs within each visualized layer on the MRI from each of 6 images (T1±FS, T2±FS, post Gd-DTPA T1±FS). The ratio of SI between layers within the plaque on T2-W imaging and for luminal layers post Gd-DTPA to pre Gd-DTPA T1-W imaging was measured.

Results
9 patients (8 male, mean age 73±6) were imaged prior to grafting of AAA. Histopathology demonstrated homogenous thrombus in 1 patient, thrombus and lipid layers in 4, fibrous cap and lipid in 1, and fibrous cap, thrombus, and lipid in 3 (Figure 1). Of the 4 identified with a clearly demarcated fibrous cap, 3 had evidence of acute inflammation as defined by polymorphonuclear cell infiltration (Fig. 1). MRI identified the same number of layers in all patients. Thrombus was seen as regions of high signal and lipid as regions of lower signal on T2-W imaging (Fig. 1). In the 7 patients with both thrombus and lipid seen, the ratio of SI of thrombus to lipid layers was 2.10±0.59. For 4 patients with a fibrous cap, SI on T2-W imaging of the luminal layer was 1.71±0.35 x that of the inner layers of the plaque (Fig. 1). No difference in T2 SI ratio was seen between the 3 patients with inflammation in the cap and the 1 without. Another patient had high SI in a luminal layer on T2-W images, simulating a fibrous cap, but the signal was not uniform over the entire layer. In the 4 patients with a fibrous cap on pathology, the ratio of SI on post-Gd T1-W + FS images to the same images before Gd-DTPA infusion in the luminal layer was 3.29±1.93 (3.86±2.34 in the 3 with evidence of inflammation, 2.16 in the 1 without).

Discussion
MRI can distinguish between components of atherosclerotic plaque in abdominal aortic aneurysms noninvasively. Signal intensity is higher within thrombus than lipid layers on T2-weighted imaging. The presence of a fibrous cap with or without inflammation can be demonstrated by a combination of uniform high signal in the luminal layer on T2-W imaging, with increased SI on T1-W imaging with fat saturation after Gd-DTPA infusion. A higher SI post Gd-DTPA in the fibrous cap may be a marker of acute inflammation. With the ability to differentiate these components, especially with higher resolution in smaller arteries, MRI may become useful in the identification of the vulnerable plaque.

References