Application of Magnetic Resonance Direct Thrombus Imaging to the Renal Vessel Wall

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Introduction: Atherosclerosis is a systemic disease that leads to raised plaques within the vessel wall of arteries such as the aorta, carotids, coronaries and renals. However, the plaques that lead to morbidity and mortality are usually modestly stenotic, often not seen by angiography. This is part of the impetus behind direct imaging of the vessel wall. By providing excellent tissue contrast and no radiation, MRI is one of the best non-invasive methods available to investigate the vessel wall. In the current literature, there are various studies imaging the aortic, carotid and recently coronary vessel wall (1), but few that image the renal arterial wall. However, there has been recent interest in the medical community in renal atherosclerosis, as evidenced by the announcement of the CORAL trial (Cardiovascular Outcomes in Renal Atherosclerotic Lesions) (2). In this study, we propose to apply magnetic resonance direct thrombus imaging (MRDTI) to the renal arterial wall, as a first step in developing a method to image the arterial wall of small-to-medium sized vessels. MRDTI has previously been optimized to image the carotid arterial wall and plaque, with correlation to histology. It exploits the T1 shortening effects of methemoglobin, allowing the visualization of intraplaque hemorrhage/thrombus (IPHT) within atherosclerotic plaque in arterial vessel wall, defining the atheroma as complicated (3,4).

Methods: Twenty-two patients (11 male, 11 female, mean age 65.9 years [24-83years]) were scanned using a 1.5T GE Twin Speed Clinical MR scanner (GE Medical systems, USA) using an 8-channel torso phased-array coil (USA Instruments, USA). Patients were being investigated for the following: renal artery stenosis (68.2%, 15 of 22 patients), abdominal aortic aneurysm (13.8%, 3 of 22 patients), aortic dissection (4.5%, 1 of 22 patients), inferior vena cava thrombosis (4.5%, 1 of 22 patients), renal infarct (4.5%, 1 of 22 patients) and renal cell carcinoma (4.5%, 1 of 22 patients). The sequence used to image the renal artery vessel wall was a 3D T1W fat-suppressed spoiled gradient echo (TR/TE/α 5.7ms/1.2ms/Fr, 15^0), with 2.2mm thickness, FOV 360mm^2, matrix size 256 x 128, and effective pixel size 1.4mm x 2.8 mm x 1.1mm (interpolated). Fat suppression was performed using the Special (Spectral Inversion At Lipids) GE proprietary technique. For those patients who could breath-hold, a NEX=1 was used with a scan time of 28 seconds. If patients could not breath-hold (resulting in suboptimal images), a free-breathing scan was used, with a NEX=5 and a scan time of 02:15 minutes. Contrast-enhanced renal MRA (CEMRA) was performed as part of the clinical imaging protocol of the patients. The sequences was a 3D fat-saturated spoiled gradient echo (TR/TE/α 4.2ms/1.1ms/Fr, 40^0), with 3mm thickness, FOV 300mm^2, matrix size 320 x 224, and effective pixel size 0.9mm x 1.3 mm x 1.5mm (interpolated), NEX=1. Intravenous contrast (Omniscan, 0.1 mmol/kg, Amersham Health, USA) was injected at a rate of 2cc/second. Maximum Intensity Projections (MIP) of the renal CEMRA were performed.

Results: The proximal vessel wall of the renal arteries was well visualized in all 22 patients. Fourteen of 22 patients (63.8%) had breath-held scans, and 8 of 22 had free-breathing scans (36.2%). No differences were noted in renal vessel wall visualization in breath-held versus free-breathing scans (Figures 1 and 2). Intraplaque hemorrhage was seen in 6 of the 22 patients (Figure 1B and 2B). It was present in 5 of the 15 patients (33.3%) being investigated for renal artery stenosis, and in 1 of the 3 patients (33.3%) being investigated for an abdominal aortic aneurysm. All patients with complicated plaques were male. Seven stenoses were noted (2 in one patient). The majority of the stenoses seen were due to complicated plaque (6 of 7, 85.8%), and this was confirmed by CEMRA of the renal arteries. In one case (the patient with bilateral stenoses), one was caused by a complicated plaque, the other was caused by thickening of the left arterial wall (Figure 2, B-D).

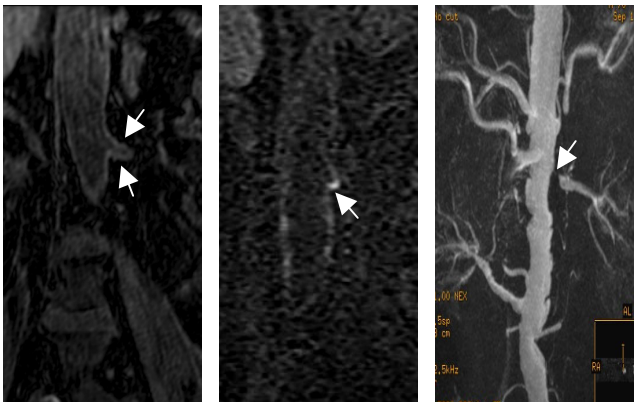

Figure 1. **Breath-held examinations. A**. Normal right arterial wall is seen (arrows). **B**. Complicated atherosclerotic plaque is noted in the aorta and at the origin of the left renal artery (arrow). **C.** Maximum Intensity Projection of renal CEMRA from patient in B shows a significant stenosis at the origin of the left renal artery, where the complicated plaque is located (arrow)**.**

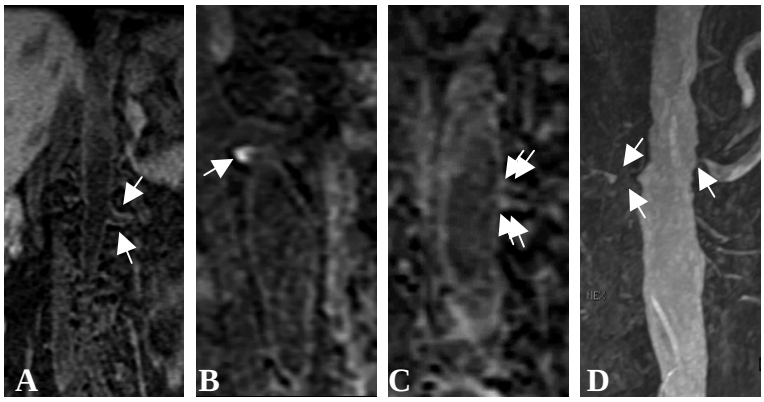

Figure 2. **Free-breathing examinations. A**. Normal left arterial wall is seen (arrows). **B**. Complicated atherosclerotic plaque is noted at the origin of the right renal artery (arrow). **C.** The origin of the left renal artery in patient from B is somewhat thickened, but no complicated plaque is seen (double arrows). **D.** Maximum Intensity Projection of renal CEMRA from patient in B and C shows complete cessation of flow within the proximal right renal artery (location of complicated plaque, double arrows), as well as significant stenosis at the origin of the left renal (single arrow).

Conclusion: We have successfully applied a sequence previously shown to identify complicated plaque to the renal arteries. This sequence demonstrates the vessel wall, as well as complicated atheromatous plaque, in the renal arteries.

References:

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