Identification and quantification of atherosclerosis in arterial vessels using an interventional 3T loopless detector

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Introduction. Fibrous cap thickness is a key factor in determining the vulnerability of atherosclerotic plaques to rupture, which has dire consequences for the patient. MRI's high soft tissue contrast and lack of ionizing radiation suit it well for plaque imaging, if only its resolution is sufficient to resolve the cap and other pathologies. At 3T the loopless interventional MRI (iMRI) detector offers ~4-fold higher signal-to-noise ratio than at 1.5T [1]. It can be made safe and fabricated into a small-diameter biocompatible intravascular guidewire. Here we investigate use of a 3T loopless iMRI detector in a protocol wherein plaques are identified at a coarse resolution, followed by high-resolution imaging of plaque, vessel wall morphology, and measures of fibrous cap thickness. We identify plaques and perform 80μm iMRI measurements of cap thickness in diseased human arterial specimens *in vitro*, compare the results with histology, and demonstrate feasibility of this approach in a rabbit aorta *in vivo*.

Methods. A 3T 2.2mm diameter, 40cm long loopless iMRI detector with an 40mm whip was fabricated, tuned, matched, and decoupled as described previously [1]. A fresh ~10 cm long human iliac specimen harvested at autopsy within 3 hr of death was secured in a 0.35% saline phantom. The iMRI detector was inserted into the lumen, and the vessel imaged in the coronal plane (3D FFE; FA=20°; TR/TE = 25/2.6 ms; FOV =200x120 mm; resolution=0.5mm; slice thickness =1mm; fat suppression; scan time =2.5min; Philips 3T Achieva scanner). Axial high-resolution MRI was then performed at suspect sites identified on the coronal images (3D TSE, factor=9; TR/TE=1500/32ms; 80μm resolution; NSA=4; 25min scan-time).

To evaluate its ability to measure fibrous cap thickness, iMRI was applied with the same MRI sequences, to 30 carotid artery specimens obtained from cadavers and fixed in 10% formalin at 3°C for 24 hr. Vessels were sectioned for histology at locations co-registered with MRI, and the fibrous cap thickness measured at 60° intervals around the vessel lumen on both iMRI and histology (Fig. 1e,f), averaging 6 data points for each image. The results were analyzed by linear regression and Bland-Altman statistical analysis.

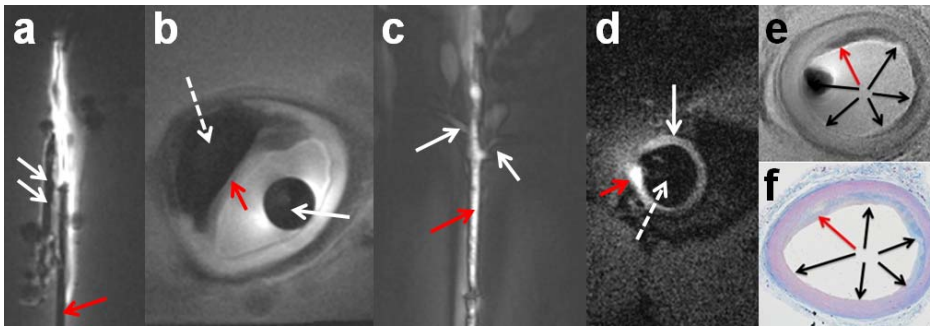

Fig. 1. (a) *in vitro* coronal MRI of iliac artery (red: iMRI coil, white: plaque). (b) *in vitro* axial MRI of iliac(red, fibrous cap; solid white,iMRI coil; dashed, plaque). (c) *in vivo* coronal MRI of rabbit aorta (red: aorta, white: renal branches). (d) *in vivo* axial MRI of rabbit aorta (red, coil; solid white, wall, dashed white, lumen). Image registration between 80μm MRI (e) and histology (f) of carotid with plaque (red, start of plaque; black, data points acquired at every 60°)

Finally, *in vivo* iMRI of a New Zealand white rabbit targeting the aortic renal bifurcation accessed via the femoral artery was performed using the same MRI sequences and an 0.8mm OD, 1.2m biocompatible nitinol loopless detector.

Results. Calcified plaque are seen on coronal iMRI of the vessel wall (Fig. 1a), confirmed by the axial 80μm TSE scan (Fig. 1b). *In vivo*, the renal bifurcation was clearly revealed on the coronal scan (Fig. 1c), with the vessel's 3.3mm lumen and 0.5mm wall matching post-mortem measurements (Fig. 1d).

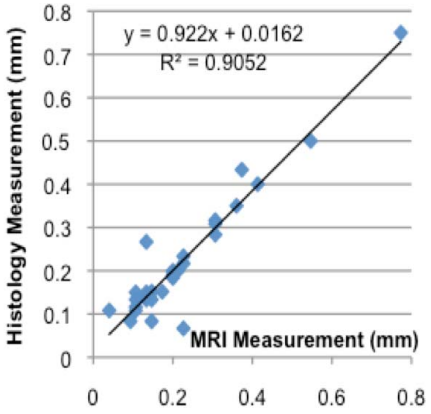

Fig. 2: Comparison of fibrous cap thickness measured by MRI vs. histology.

In the carotid specimens, the averaged fibrous cap thickness measured from 80μm iMRI correlated with histology (r^2=0.905, Fig. 2). Bland-Altman testing shows good agreement between the MRI and histological measurement (2 ± 40 μm).

Conclusion. We have demonstrated that 3T iMRI with a loopless detector can identify suspect vessel pathology for subsequent high-resolution, 80μm imaging of arterial wall morphology and pathology *in vitro,* and *in vivo*. By providing accurate measurements of fibrous cap thickness, 3T iMRI may find use for the detection and quantification of vulnerable plaque in experimental models of atherosclerosis and its treatment, potentially extending to the clinic.

Reference: [1] AM El-Sharkawy et al. Med Phys 2008;35: 1995.
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