

MRI can detect intraplaque hemorrhage in ex-vivo coronary arteries

G. Leung¹, N. Munce¹, R. Bitar², J. Butany³, G. A. Wright¹, A. R. Moody^{1,2}

¹Medical Biophysics, Sunnybrook and Women's College Health Sciences Centre, Toronto, Ontario, Canada, ²Medical Imaging, Sunnybrook and Women's College Health Sciences Centre, Toronto, Ontario, Canada, ³Laboratory Medicine and Pathobiology, Toronto General Hospital, Toronto, Ontario

Introduction: Intraplaque hemorrhage (IPH) has been seen as a critical factor in the destabilization and rupture of a vulnerable arterial plaque [1]. Compared to fibrous cap thinning, this hallmark can be easily detected using magnetic resonance imaging [2-3]. While most of this work has been done in the carotid arteries, detecting IPH in the coronary arteries is of great interest as a prognostic indicator of future cardiac events. This paper presents initial results of ex-vivo MRI of coronary arteries following acute myocardial ischaemic death with histological correlation demonstrating intraplaque hemorrhage in the coronary vessel wall.

Methods: 4 coronary arteries and 2 vein grafts were obtained from patients who died of acute myocardial infarction. These arteries were imaged after being immersed in 10% buffered formalin for 24 hours. Subsequently, the samples were transferred into a container of pure olive oil which was evacuated of air in a vacuum chamber for one hour to remove air bubbles avoiding susceptibility artefacts. MR imaging was performed on a GE 3T Signa scanner (Michigan, USA) using a fast spoiled gradient echo sequence and an in-house built dedicated specimen scanning coil was used to generate T1 weighted images with voxel size of 300 x 300 x 200 micron. A fat suppression pulse was used to remove the surrounding epicardial fat as well as the olive oil medium. Finally, the specimen was sent for histological processing with H&E staining.

Results: Figure **a**) shows a curved reformat of a bypass vein graft. This vessel is unremarkable other than some luminal stenosis, with calcium deposits in vessel walls. Figure **b**) is a curved reformat along the length of a symptomatic right coronary artery. While the vessel is occluded proximally, bright signal indicative of IPH is easily visible in the vessel wall of the native coronary. Figure **c**) shows a reformat cut at the proximal portion of the coronary artery as indicated in **b**) (red, above). The vessel is heavily stenosed with an area of signal hyper-intensity just beyond the dark lumen between 9 and 12 o'clock (*). **d**) a reformat cut at the distal end of the coronary artery, with no visible lumen, and signal hyper-intensity filling the middle of the vessel. **e**) shows an H&E stained cross section of the proximal branch of the native coronary artery in the vicinity of the section shown in **c**). IPH is seen (*) beneath an eroded, thick fibrous cap (x). Additionally, a core of cholesterol crystals is visualized adjacent to the IPH (+). All two foci of IPH found in the six ex-vivo specimens through histopathology were also detected using MR imaging.

Discussion and Conclusions: This paper demonstrates the use of MR imaging in ex-vivo samples and the correlation to histopathologic findings. Initial results suggest that MRI can be used to aid in post mortem identification of culprit atherosclerotic lesions. Additionally, we show that MRI can detect IPH in an ex-vivo setting, motivating the need to investigate improved motion compensation techniques to visualize this important hallmark of plaque vulnerability in-vivo.

References: [1] Virmani Arterioscler Thromb Vasc Biol. 2005 Oct;25(10):2054-61. [2] Moody AR Circulation. 2003 Jun 24;107(24):3047-52 [3] Yamada et al. Proc ISMRM 2005;23

