Quantifying Peripheral Vascular Calcifications with Quantitative Susceptibility Mapping

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Introduction: The presence of arterial wall calcifications substantially impacts patient management and prognosis[1]. Magnetic resonance angiography is an excellent imaging modality for identifying arterial diseases, however, it does not reliably identify or quantify vascular calcifications. The current landscape of using MRI to detect peripheral calcifications is limited to just a few reports[2,3]. Moreover, existing MR techniques such as SWI[3] does not provide quantitative measurements and cannot distinguish paramagnetic (e.g. calcium) from diamagnetic (e.g. surgical clips) materials. In this study, we applied quantitative susceptibility mapping (QSM) to quantify vessel calcification in patients with peripheral vascular diseases (PVD).

Methods: This study was approved by the local institutional review board and written informed consent was provided for all participants. A total of 6 patients (5 male, 1 female; 72.1 ± 7.5 years of age; age range, 57 – 78) with peripheral arterial disease and known vascular calcifications were recruited for MR imaging. All imaging was performed on a 32-channel 1.5 T scanner (MAGNETOM Avanto, Siemens Healthcare, Germany) equipped with high performance gradient coils (45 mT/m maximum gradient strength, 200 mT/ m/ms slew rate). The body coil was used as the transmitter, and spine array (4 channels) and body array coils were used as the receiver. Subjects were positioned feet first and all imaging was performed at magnet isocenter.

A three-dimensional, T2*-weighted, dual-echo, flow compensated, spoiled gradient echo sequence was used for QSM data collection with the following imaging parameters: coronal oblique imaging plane; echo times, 4.59 and 9.54 ms; repetition time, 15 ms; field of view, 384 mm; imaging matrix, 384 x 384; slab encoding thickness, 0.5 mm; flip angle, 10 degrees; parallel acceleration factor, 2 with iPAT. The total imaging time was 7 minutes and 18 seconds. QSM data was reconstructed offline using the MEDI algorithm[4].

Results: Peripheral vascular calcifications were clearly identifiable on QSM images. Sample images from one patient with a starclose nitinol ring implant are shown in Figure 1. The calcification shape and location on QSM images well correlated with the corresponding CT images. The mean susceptibility values for the calcifications ranged from -0.99 to -1.75 ppm, with an average of -1.23 ± 0.35 ppm. The susceptibility of the calcification was lower than surrounding muscle (0.02 ppm) and easily distinguishable from the paramagnetic nitinol ring (1.45 ppm).

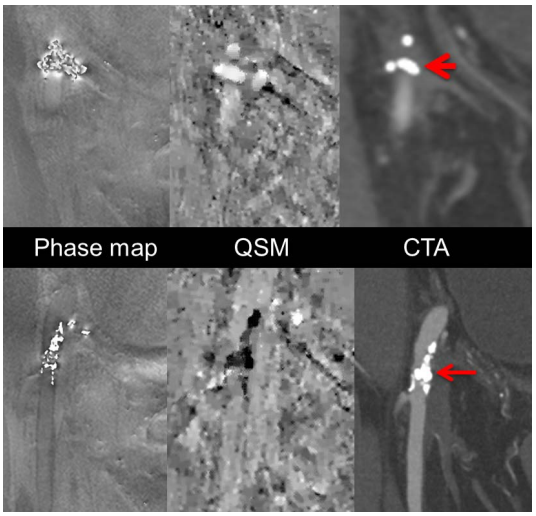

Figure 1. Phase maps and QSM-derived magnetic susceptibility measurements for a patient with PVD. Top row corresponds to a coronal slice near the anterior margin of the right CFA; bottom row corresponds to a slice near the posterior margin. Long arrow – calcifications along the posterior wall of the right CFA. Short arrow – Starclose nitinol ring that was deployed along the anterior aspect of the right CFA after a cardiac catheterization three years earlier.

Discussion: In this study, a dual-echo 3D gradient echo sequence with in-phase echo timing and flow compensation for eliminating flow-related phase shifts was used to generate QSM maps of peripheral vascular calcifications. While we aim to improve the quality of the QSM reconstruction, the concept of mapping large susceptibility sources in presence of fat, bones, and rapid flow proves to be possible. Measurement of the magnetic susceptibility may also prove helpful should a plaque have an intermediate value for T2*. In such a hypothetical situation, measurements of the magnetic properties of the lesion might facilitate this distinction between a calcification and intra-plaque hemorrhage.

Conclusion: The preliminary result has demonstrated the capabilitiy for in-vivo measurements of the diamagnetic suscepbilility associated with vascular calcifications. Further optimzation of the reconstruction parameters is warranted in future investigations.

References: 1) Demer et al, Circulation, 2008. 2) Li et al, JCMR, 2010. 3) Yang et al, JMRI, 2009. 4) Liu et al. MRM. 2010.