Delayed Hyperenhancement MR Coronary Vessel Wall Imaging in Patients with Stable and Unstable Coronary Artery Disease

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Background: Contrast enhanced MR vessel wall imaging has been found useful for assessment of fibrous plaque in patients scheduled for carotid endarterectomy as well as in the setting of inflammatory vascular disease such as giant cell arteritis.[1] In a preliminary study of contrast enhanced coronary plaque imaging we found that Gd-DTPA can be used for selective plaque imaging.[2] In addition, we found progressive contrast uptake with progressive coronary artery disease as assessed by x-ray angiography and MSCT.[3]

Purpose: In this study, we sought to examine delayed hyperenhancement coronary MRI (DE-MRI) for coronary plaque visualization in patients with stable CAD and acute myocardial infarction (AMI). In addition, we sought to investigate the change in delayed hyperenhancement by performing serial CMR studies 1 week and 2 month post AMI.

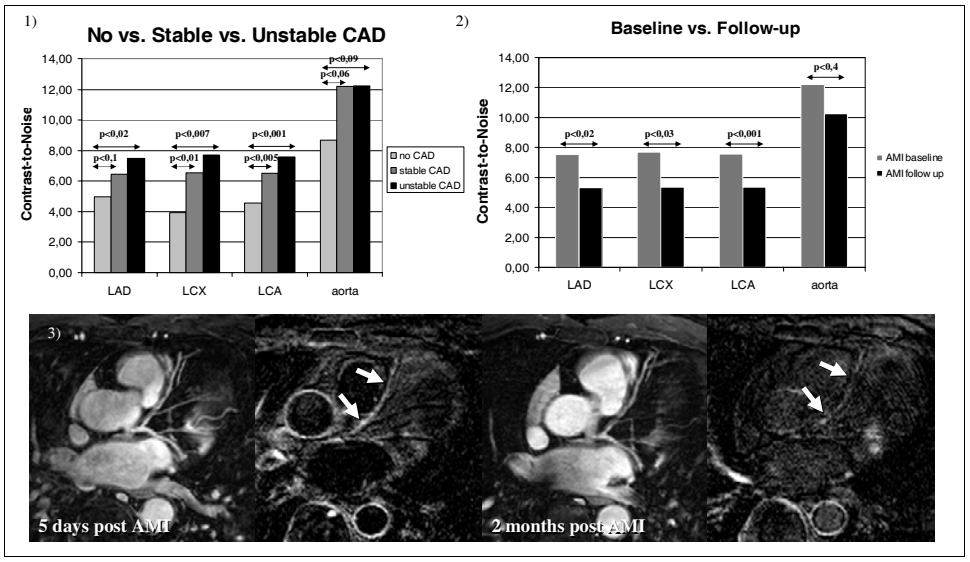
Methods: DE-MRI coronary vessel wall imaging was performed in 27 patients who had undergone x-ray coronary angiography (stable CAD (N=15), AMI (N=12)) ~40 minutes after administration of 0.2mmol/kg Gd-DTPA (Magnevist, Schering). For comparison, an age matched control group of 5 patients free of angiographically visible CAD (no-CAD) was included. In a subgroup (N=10) of patients with AMI, imaging was repeated at 2 months post MI. DE-MRI was performed using an ECG triggered free-breathing T1-weighted inversion recovery 3D fast gradient echo technique in concert with navigator gating and correction for respiratory motion correction. Contrast-to-noise (CNR) between the vessel wall and aortic blood was calculated for 5 coronary segments (LM, prox and mid LAD, prox and mid LCX) and the descending aorta using dedicated post processing software. Strong enhancement was defined as CNR > 9. Results were then compared to x-ray angiographic findings and clinical presentations.

Results:

Stable CAD vs. AMI: In both stable CAD and AMI groups, the number of enhancing coronary segments (%) correlated with the severity of CAD while there was a small trend of greater extent (25% vs. 29%; p=NS) and intensity of strong enhancement (CNR: 11.9 vs. 12.9; p=NS) in patients with AMI. Strong enhancement was not observed in the control group. Left coronary wall (LCA) contrast uptake was higher both in patients with stable CAD and AMI compared to the no-CAD subjects. Mean CNR was 6.5 ± 3.3 (stable CAD) vs. 7.2 ± 3.9 (unstable CAD) vs. 4.6 ± 2.1 (no CAD), respectively (p<0,005 and p<0,001) (Figure 1). There was no significant difference in CNR between the AMI and stable CAD groups (p<0,34). Similar to coronary enhancement, aortic enhancement was also greater in patients with stable and AMI, compared to the control group (Figure 1) with no difference between the AMI and stable CAD groups (p=NS).

Follow-up imaging: Two month post AMI, the number of all coronary segments with strong enhancement decreased from 36% to 18% and the median CRP value decreased from 1.6 to 0.0 mg/dL. This decrease was most prominent in segments without angiographic CAD or wall irregularities (34% to 11%). In contrast, the number of stenotic segments with strong contrast uptake remained unchanged (40% to 40%). There was a trend for decreased (12.2 ± 5.0 vs. 10.3 ± 6.4; p<0.4) aortic enhancement at the 2 month scan (Figure 2). Representative images of the LAD, LCX, and aorta are shown in Figure 3.

Conclusion: We demonstrate the feasibility of DE-MRI coronary wall imaging for the assessment of coronary plaque burden in patients with stable and unstable CAD. The small trend of greater extent and intensity of coronary wall hyperenhancement in patients with AMI may reflect vascular inflammation associated with the acute event. In addition, we could demonstrate that aortic vessel wall enhancement correlates with x-ray angiographic apparent CAD but is independent on stable or unstable CAD. Serial DE-MRI of the coronary arteries and aorta in patients with AMI revealed a decrease of the spatial extent and intensity of strong contrast enhancement at the 2 month post MI scan. This decrease in hyperenhancement may be related to the overall reduction of systemic inflammation and appears to parallel declines in CRP. Larger follow-up studies as well as studies in patients undergoing carotid endarterectomy are now warranted to better understand the spatial and temporal extent of enhancement and its underlying pathophysiology.



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