Arterial Spin Labeled MRI Detects Increase in Myocardial Blood Flow with Adenosine

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INTRODUCTION

Myocardial arterial spin labeling (ASL) is an emerging technique for the assessment of myocardial perfusion. ASL does not require any contrast agents, and therefore can be used in patients with renal failure, and can be performed continuously during an intervention. Furthermore, ASL has the potential to provide quantitative measurement of regional myocardial blood flow (MBF) in ml-blood/g-tissue/min. We recently demonstrated feasibility of myocardial ASL, including measurements of increased MBF during leg elevation and isometric handgrip [1]. In this study, we applied myocardial ASL to the measurement of MBF at rest and during an infusion of adenosine, which is a widely used pharmacologic vasodilator [2].

METHODS

Eleven patients (aged 63±6) were recruited, among those scheduled for routine cardiac MR exams at Loma Linda University that included CMR first-pass perfusion imaging with adenosine. All experiments were performed on a GE Signa 3.0 T EXCITE HDx system with a custom pulse sequence. Table 1 summarizes relevant portions of the imaging protocol. Myocardial ASL measurements were obtained from a single mid short-axis slice, using a double-gated pulse sequence with flow-sensitive alternating inversion recovery (FAIR) tagging and balanced steady-state free precession (SSFP) imaging, as described in Ref. [1]. Each ASL scan required six breath-holds, each lasting 10 seconds, for a total time of 3 minutes (including breaks). In each patient, one or two scans were performed at rest, and one scan was performed between minutes 2 and 5 of adenosine infusion (dosage: 0.14 mg/kg/min). CMR first-pass perfusion was performed between minutes 5 and 6 of the adenosine infusion.

	3 min	Rest ASL scan
	3 min	Rest ASL scan
Start adenosine infusion →	2 min	Wait
	3 min	Stress ASL scan
Stop adenosine infusion →	1 min	Stress first-pass perfusion

Table 1. Imaging protocol excerpt that includes ASL scans at rest and during adenosine infusion.

RESULTS

CMR first-pass images were read by two experienced cardiologists. Seven of the eleven subjects were classified as "normal," based on having no visible perfusion defect. Figure 1 contains myocardial ASL results from the seven "normal" subjects. Each bar represents the average MBF across the whole myocardium in the slice. The average MBF measurement across subjects was 1.09 ± 0.53 ml/g/min at rest and 3.75 ± 1.06 ml/g/min during adenosine infusion. Perfusion reserve (MBF_{stress}/MBF_{rest}) was 4.97 ± 4.64. Using a t-test, the MBF increase with adenosine was found to be statistically significant, with p=0.0008.

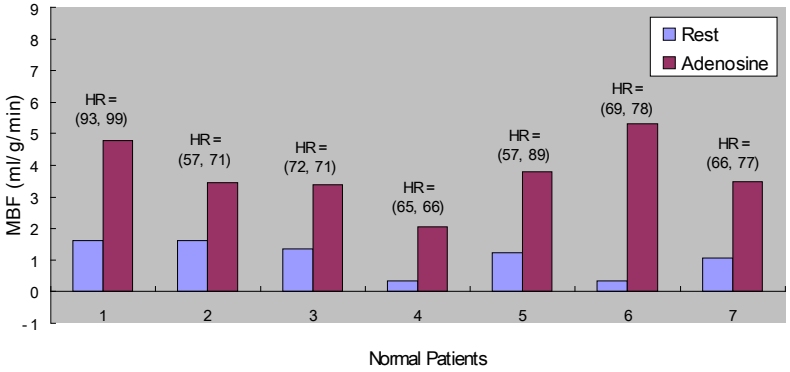

Figure 1. MBF measurements using ASL and heart rates (HR, in bpm) at rest and during adenosine infusion, from seven patients with no visible perfusion defects on CMR first-pass imaging.

DISCUSSION

Adenosine is a widely used vasodilator that produces large increases in MBF for normal myocardium. This increase has been documented to be 4.00 ± 1.10 times based on ^{15}O-H_2O PET [3]. The present study has demonstrated that myocardial ASL is able to capture these dramatic MBF changes in normal myocardium. This study is on-going, and as we scan a larger number of patients, we will be able to evaluate the ability of rest-stress myocardial ASL to detect angiographically significant coronary artery disease. Figure 2 contains a perfusion reserve map and coronary angiogram from our first patient with single-vessel disease. Lowered perfusion reserve in the anterior wall appears to correspond to a total occlusion of the left anterior descending (LAD) coronary artery in this patient.

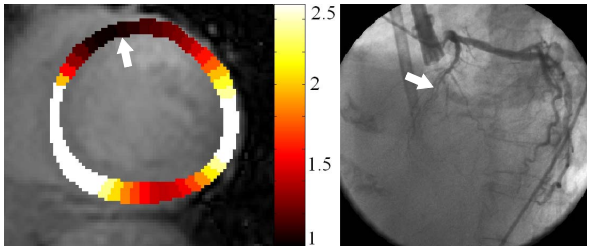

Figure 2. Left: Perfusion reserve map from a mid short-axis slice using ASL and Hamming windowing. **Right**: Coronary angiogram from the same patient. Lowered perfusion reserve on anterior wall (arrow on left) is consistent with the total LAD occlusion (arrow on right).

REFERENCES

[1] Zun Z *et al*, MRM 62:975, 2009. [2] Wacker CM *et al,* JMRI 18:555, 2003. [3] Kaufmann PA *et al,* Am J Physiol Heart Circ Physiol 293:H2178, 2007.