QUANTITATIVE ASSESSMENT OF REPERFUSION SIX MONTHS AFTER ACUTE MYOCARDIAL INFARCTION USING GD-DTPA DCE-MRI UNDER ADENOSINE INDUCED STRESS

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Introduction: In patients with acute myocardial infarction (AMI) dynamic contrast enhanced MRI (DCE-MRI) could provide important diagnostic information about the possible presence of ischemic areas outside of the infarct zone. Furthermore, successful reperfusion of the myocardium following AMI is essential for the restoration of normal cardiac function. Quantitative analysis of first pass DCE-MRI can be performed by measuring various timing parameters which characterise contrast kinetics: the time of contrast arrival into the microvascular bed, the microvascular transit time and the extravasation time. To assess the utility of this technique in the assessment of reperfusion following AMI we performed quantitative analysis of the time course of Gd-DTPA arrival and uptake in first pass DCE-MRI studies of myocardial perfusion under adenosine induced stress in patients with AMI at baseline and six months after the index event.

Methods: Twenty nine patients (mean age = 56 ± 8.7 years, range 41-74 years, male = 26, female = 3) presenting with a confirmed first AMI were recruited. During the index admission (1-9 days after presentation) all patients underwent rest/stress DCE-MRI and late gadolinium hyperenhancement MRI, followed by x-ray angiography. Scanning was preformed on a whole body 1.5T MR scanner (Gyroscan Intera CV, Philips Medical Systems). Seventeen patients underwent PCI and further two underwent CABG. An identical MRI protocol was carried out at follow-up (six months after the index admission). First pass DCE-MRI myocardial perfusion imaging was performed at rest and during a 5-minute adenosine infusion (140µg/kg/min)[1]. The time course of enhancement was analysed in 16 myocardial segments, defined according to AHA guidelines[2]. The same 16-segment model was used to record locations of scar tissue (detected on baseline late gadolinium hyperenhancement MRI) and segments affected by significant stenoses (≥70%) determined by baseline x-ray coronary angiography. Arterial input function (AIF) was derived from a region of interest placed within the left ventricular cavity of the most superior (4th) slice.

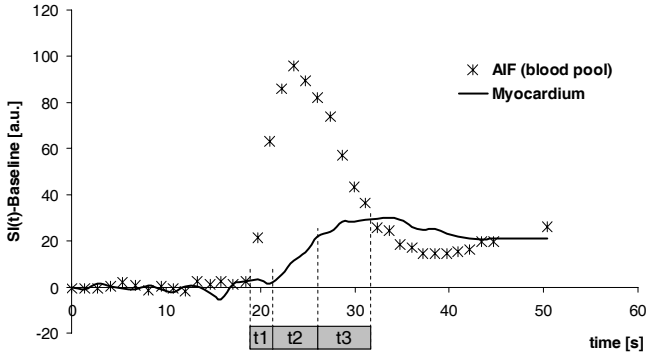

Figure 1. Definition of timing parameters extracted from individual myocardial segments

Three timing parameters were extracted from each segment at stress (Figure 1). Timing parameters were defined as follows:

t1 delivery phase: the time delay between the arrival of the contrast into the basal slice of the left ventricle and the arrival of the contrast into the myocardial segment

t2 initial microvascular phase: the time between the arrival of the contrast into the microvascular bed and the time when 50% of the maximal signal intensity is reached

t3 late microvascular phase: the time required for SI to increase from 50% to 100% of the maximal value.

Results: The statistical analysis (paired samples t-test, with $\alpha = 0.05$ confidence level) was performed on 464 pairs of measurements, divided into three groups: "Normal" (n = 239), "≥70% stenosis" (n = 121), and "Scar" (n = 104), according to the baseline classification. A summary of the baseline and follow-up measurements is presented in Figure 2.

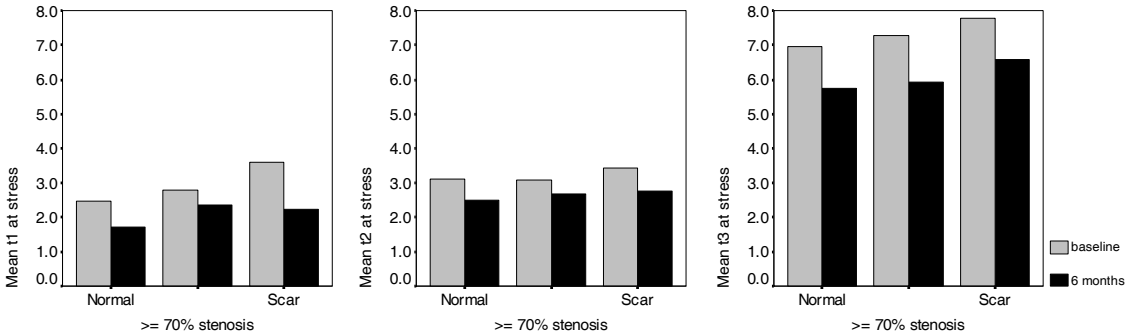

Figure 2. Mean values of DCE-MRI timing parameters t1, t2 and t3 (in seconds) measured in three myocardial segment subgroups under adenosine stress. All differences between baseline values (grey) and six-months follow-up values (black) are statistically significant ($p < 0.05$).

Conclusion: Six months after AMI all timing parameters measured under adenosine stress were significantly reduced in all three segment groups (Figure 2). Shortening of the timing parameters is indicative of improved delivery of blood to the myocardium (t1) and improved perfusion of the microvascular bed (t2 and t3). Our results suggest that the improvement in perfusion six months after AMI affects all segments of the myocardium, including the apparently normal segments outside of the infarcted territory and areas supplied by arteries with flow limiting stenoses. For example, parameter t2 measured in 239 "Normal" segments was 3.11 ± 0.97s at baseline and 2.49 ± 0.75s at follow-up (mean ± SD) , indicating a significant improvement in perfusion in this group of segments ($p<0.0005$). This implies that there is a global reduction of myocardial perfusion in the immediate aftermath of AMI.

References: [1] Plein S et al. Radiology 2005;235:423-430., [2] Cerqueira MD at al. Circulation 2002;105:539-42.