Delayed Contrast Enhanced MRI in Patients with Acute Myocardial Infarction and Coronary Intervention: Sensitivity and Correlation between MRI Infarct Size, Biochemical Markers and Time to Intervention

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Background:

Recently, the quantitative assessment of myocardial infarctions using delayed contrast-enhanced magnetic resonance imaging (MRI) has been validated using post-mortem histopathological animal studies and has been shown to be superior to SPECT and PET for the detection of myocardial infarctions. The sensitivity of delayed-enhancement MRI in patients with acute myocardial infarctions (MI) and its correlation to established markers of MI are yet unknown.

Purpose:

In a prospective study we investigated 1) the sensitivity of delayed enhancement (DE) MRI for the detection of acute myocardial infarctions (MI) in patients with biochemically (CK, CK-MB, Troponin) and angiographically confirmed MI after rescue percutaneous coronary intervention (PCI) and 2) the correlation between infarct size as assessed by DE MRI and a) biochemical markers of myocardial infarction such as creatine-kinase (CK) and c-reactive protein (CRP) as well as b) the time from onset of symptoms to intervention.

Methods:

The study group consisted of 45 patients (pts) with acute myocardial infarctions (confirmed by elevated levels of CK, CK-MB and Troponin) who underwent rescue PCI within 24 hours after onset of symptoms. During a three day follow-up blood levels of CK and CRP were measured every 8 hours. Delayed enhancement MRI was performed 4 to 10 days after intervention using k-space segmented 3D inversion recovery gradient-echo MR sequences at a 1.5 Tesla magnetic resoncance imaging system with complete coverage of the left ventricular (LE) myocardium in short axis slices. In-plane resolution was 1.33x1.33 mm² and slice thickness was 5 mm. MR images were acquired 10 min after intravenous administration of Gadolinium-DTPA (0.2 mmol/kg). The inversion delay time T_i was iteratively adjusted to null the signal from normal myocardium. Areas of hyperenhancement representing non-viable myocardium were defined as those with signal intensity >2 SD above normal remote myocardium and manually planimetred on each short axis slice. The mass of infarcted tissue based on the volume of hyperenhanced myocardium was calculated assuming a myocardial specific gravity of 1.05 cm³ and expressed in absolute (g) numbers as well as in relative values in relationship to the total mass of LV myocardium (LV%). Linear regression analysis was performed to assess the correlation between absolute size of infarctions (g) as well as relative size (LV%) with peak values of CK, CRP and the time from onset of symptoms to PCI.

Results:

In all pts (n=45/45) acute myocardial infarctions were detected with the technique of delayed enhancement (sensitivity 100%). The mass of infarcted tissue varied between 1.30 g – 78.8 g, mean 24.2 +/- 17.8. Significant correlation existed between the absolute size of infarctions (g) and peak CK values (r=0.72; p<0.001) as well as the relative size (LV%) and peak CK (r=0.77; p<0.001). No correlations were found between absolute size (r=0.33) as well as relative size (r=0.27) of infarctions and peak CRP. There was also no correlation between absolute (r=0.29) as well as relative size of infarctions (r=0.27) and the time from onset of symptoms to PCI.

Conclusion:

1) DE MRI is as sensitive as established biochemical markers (CK, CK-MB, Troponin) for the detection of acute myocardial infarctions. 2) In pts with acute MI (<24h) undergoing rescue PCI, peak CK values correlate well with infarct size as assessed by delayed contrast enhanced MRI. 3) There is no correlation between infarct size and peak CRP as well as the time from onset of symptoms to intervention.

