

POSTER SESSION

1167 Microvascular Obstruction and Myocardial Viability by Magnetic Resonance Imaging

Tuesday, April 01, 2003, 9:00 a.m.-11:00 a.m.

McCormick Place, Hall A

Presentation Hour: 10:00 a.m.-11:00 a.m.

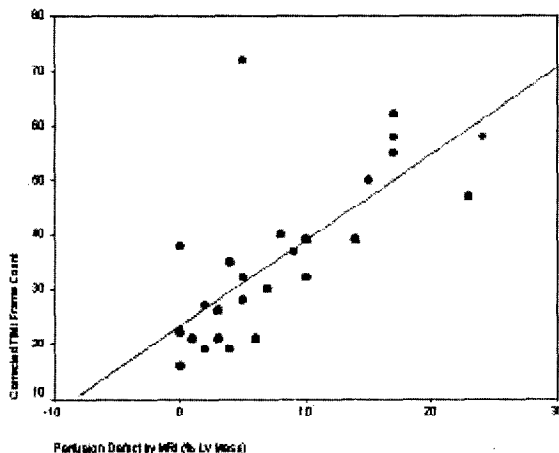
1167-35 First-Pass Magnetic Resonance Imaging Myocardial Perfusion Can Determine Microvascular Dysfunction in Acute Myocardial Infarction: Validation Against Angiographic Thrombolysis in Myocardial Infarction Frame Count and Myocardial Blush Score

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Background: Microvascular dysfunction (MD) in acute myocardial infarction (AMI) following percutaneous intervention (PCI) can be assessed by TIMI frame count (cTFC) and myocardial blush grade (MBG). We evaluated First-Pass gadolinium MRI perfusion to determine MD noninvasively against angiographic measures. **Methods:** 28 pts underwent primary PCI for first AMI followed by MRI perfusion in <72 hrs. Artery-specific myocardial perfusion was determined (qualitative and quantitative) by 2 blinded readers for the defect size (% LV mass; score 0=none to 3=large) and severity (CNR=contrast-noise-ratio). **Results:** Of 28 pts (age 59±10yr, 82% male, CK 617±1077 mg/dL, LVEF 53±11%), PCI achieved TIMI 3 flow in 85%, reduced % stenosis from 91.4±15 to 16.4±15, increased minimal diameter from 0.27±0.46 to 2.69±0.57 mm, improved cTFC from 67±28 to 36±15 and MBG from 2.0±0.9 to 1.0±0.9. Size (7.9±7% of LV mass) and severity (CNR 23.0±20) of MD by MRI correlated with cTFC ($r^2=0.72$, $p<0.0001$), MBG ($r^2=0.75$, $p<0.0001$), post TIMI antegrade flow ($r^2=-0.48$, $p=0.01$) and angiographic no-reflow ($r^2=0.81$, $p<0.0001$). Of interest, qualitative scoring alone of defect size & severity correlated with cTFC ($r^2=0.61$, $p=0.001$) and MBG ($r^2=0.57$, $p=0.002$).

See Figure.

Conclusion: MD determined noninvasively by MRI correlates with angiographic surrogates of microvascular dysfunction. Furthermore, role of qualitative analysis of perfusion to provide a rapid and reliable assessment of MD is validated.



1167-36 Detection of Acutely Impaired Microvascular Reperfusion Following Infarct Angioplasty With Magnetic Resonance Imaging

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Background: Restoration of microvascular flow is critical to reperfusion strategies in acute myocardial infarction (AMI). Despite the reopening of the infarct related artery (IRA) with infarct angioplasty, complete microvascular reperfusion does not always ensue.

Methods: We performed cardiovascular magnetic resonance imaging (CMRI) in 11 AMI patients within 24 hours of successful infarct angioplasty (TIMI grade III flow in the IRA), and also in 9 control patients, on a clinical 1.5T CMRI scanner. Three-month follow-up CMRI in AMI patients evaluated the impact of abnormal reperfusion on recovery of segmental wall motion. Microvascular perfusion was assessed at rest by first-pass perfusion CMRI following a bolus of Gadolinium-DTPA (0.1mmol/kg). Semi-quantitative perfusion analysis calculated the time to 50% maximum myocardial enhancement ($T_{50\%max}$) during the first pass of contrast. Left ventricular (LV) systolic function was assessed by a steady state free precession pulse sequence to quantify percent myocardial thickening. In all AMI patients, regional perfusion and wall thickening were evaluated in 3 standard short axis slices (apical, mid and basal), utilizing a 16-segment LV model.

Results: The mean $T_{50\%max}$ did not differ between AMI and control patients (8.5 ± 0.5

seconds vs. 8.1 ± 0.9 seconds, $P=NS$). However, in comparison to the homogenous wash-in pattern of contrast in control patients, AMI patients exhibited heterogenous contrast wash-in, with slower contrast wash-in corresponding to the infarct location. When the $T_{50\%max}$ of acutely dysfunctional segments in AMI patients was compared with remote segments, there was a mean contrast delay of 1.0 ± 0.2 seconds in infarct regions (95% confidence interval 0.7-1.4 seconds). At follow up, the mean recovery of percent systolic thickening was lower in segments with a contrast delay of 2 or more seconds (11 ± 7% vs. 31 ± 6%, $P<0.05$).

Conclusion: CMRI detects impaired microvascular reperfusion in AMI patients despite successful infarct angioplasty, which is associated with a lack of recovery of wall motion. This modality can assist the future development and assessment of strategies aimed at optimizing microvascular reperfusion in AMI.

1167-37 Infarct Imaging in a Single Heart Beat: Could LESS Be More?

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Background. Gadolinium-DTPA (Gd-DTPA) late enhanced (LE) magnetic resonance (MR) imaging using inversion recovery (IR) segmented turboFlash (IR-TFL) accurately measures the volume of infarcted myocardium. Each short axis slice of the left ventricle (LV) is acquired during breath holding over 8-10 heartbeats. We aimed to validate the LE single shot IR trueFISP sequence (LE-SS) that acquires images in a single heart beat without need for breath holding or a regular heart rhythm.

Methods. 13 male and 6 female (mean (range) age=60 (37-83)) incident hospital admissions with acute coronary syndromes were consecutively recruited. MR was performed at a median (range) of 69 (16-120) hrs from chest pain onset on a Siemens Sonata 1.5T system with a phased array chest coil. LV dimensions were evaluated by cinematographic (TrueFISP) breath hold sequence. LE MR was performed 15 mins after injection of 0.2 mmol/kg Gd-DTPA using 1) IR-TFL (ECG triggered, 23 segments, field of view (FoV)=340mm, slice=8mm, TE/TR=4.3/11ms, TI=230-330ms, flip angle (FA)=30°) and 2) LE-SS (ECG triggered, single shot, FoV=340mm, slice=8mm, TE/TR=1.2/2.7ms, TI=260-360ms, FA=30°). Sampling for troponin I (TnI), creatine kinase (CK) and CKMB took place 12 hours after chest pain onset. Images were evaluated by 2 independent and blinded observers.

Results. LE volume by LE-SS (mean (SD)) 13 (14) ml and by IR-TFL (13 (15) ml) correlated strongly ($r=0.83$, $p<0.0001$). Bland-Altman limits of agreement ($\pm 2SD$) for the difference between LE volume by LE-SS and IR-TFL were -8.55 < -0.02 < 8.51 ml. LE-SS volumes strongly correlated with TnI ($r=0.83$, $p<0.0001$) and CK ($r=0.82$, $p<0.0001$) and less so with CKMB ($r=0.63$, $p=0.009$). LE-SS volumes correlated with LV ejection fraction (mean (SD) 59 (9.7%)) and LV end-systolic (58 (19) ml) volume: $r=-0.48$, $p=0.004$ and $r=0.46$, $p=0.005$, respectively, but was unrelated to LV end-diastolic volume (140 (36) ml) and LV mass (134 (42) g).

Conclusion. The volume of myocardium showing LE measured by LE-SS strongly correlates with volumes measured by segmented IR-TFL and with standard serum markers for myocardial necrosis. Given its speed and ease of handling, LE-SS therefore appears to be a promising alternative to IR-TFL.

1167-38 Delayed-Enhanced Magnetic Resonance Imaging Detects Myocardial Fibrosis in Patients With Chronic Aortic Valve Disease

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Background: The new delayed-enhanced MRI technique has been shown to delineate precisely myocardial infarction. Chronic aortic valve disease is associated with left ventricular hypertrophy and myocardial fibrosis (MF).

Objectives: Our objectives were to evaluate the ability of a new MRI technique to detect MF in patients with severe chronic aortic regurgitation (AR) or stenosis (AS).

Methods: We evaluated 50 patients with severe aortic valve disease (25 stenosis and 25 regurgitation), with 48.3±16.2 years old, 35 males, 37 patients with congestive heart failure, New York Heart Association (NYHA) functional class III, 11 in class II, and 02 in class IV. All patients underwent MRI examination and myocardial biopsy during valve replacement surgery. MRI was performed using a gradient echo with an inversion-recovery prepulse (delayed enhancement) to detect the presence of fibrosis after injection of 0.2mmol/kg of gadolinium-DTPA. Biopsy samples were stained by hematoxyline-eosine and Masson's trichrome technique to investigate MF and were qualitatively classified as presence or absence of MF.

Results: MRI detected MF in 18 patients (72%) with severe chronic AR and 15 of those (83%) were confirmed by myocardial biopsy. Sensitivity and specificity were: 88% and 63%, respectively. Functional class III (NYHA) was predominant in patients with MF by MRI (14/18 patients, 83%). MF most frequent locations were apical anterior (44%), basal inferior (22%) and basal anterolateral (16.5%). MF areas were small, focal or linear fibrosis.

In the AS, MRI detected fibrosis in 16 patients (64%) and 14 of those (87%) were confirmed by myocardial biopsy. Sensitivity and specificity were: 82% and 75%, respectively. Eleven patients (11/16, 69%) had functional class III. The most frequent location of fibrosis were apical anterior (50%), basal inferior (25%) and lateral anterior (12.5%). MF characteristics were similar to the AR fibrosis.

Conclusion: Delayed-enhanced MRI is able to detect small areas of myocardial fibrosis in patients with a severe chronic aortic valve disease. This may provide prognostic information.