COMPARISON OF LEFT VENTRICLE CIRCUMFERENTIAL STRAIN IN ANIMALS SUBJECTED TO CORONARY ARTERY MICROEMBOLI OR OCCLUSION/REPERFUSION

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Hypothesis: It has been well recognized that left ventricular (LV) systolic function is a major predictor of outcome after acute myocardial infarction (AMI)1. The most commonly used and recommended measurements for echocardiographic quantification of global and regional LV systolic function are LV ejection fraction and wall motion score, respectively2. However, these measurements have limitations. Strain and strain rate have been introduced as novel quantitative measurements reflecting LV function3. These novel parameters use 2D speckle-tracking imaging and enable angle-independent quantification of myocardial deformation. This MRI study was designed to compare 2D strain curves, peak strain and time to peak strain (TTPS) after acute myocardial infarction (AMI) caused by LAD microembolization and occlusion/reperfusion.

Methods: The left coronary artery in swine was catheterized and the tip of the catheter was placed in the LAD distal to the 2nd diagonal branch. The animals were subjected to 90 min LAD occlusion followed by reperfusion (n=8), delivery of microemboli (16mm3 volume and average diameter 80 µm, n=8) or served as controls (n=8). Cardiac MRI was performed using 1.5-T scanner (GE Medical Systems, Milwaukee, WI, USA) three days after coronary interventions. Tagged MR images for circumferential strain in the short axis plane were acquired using a tagged turbo-field echo-planar sequence (TR/TE/flip angle=35/6.1ms/25°. Delayed enhanced MRI (inversion recovery gradient-echo sequence) was used to measure infarct size after 0.15mmol/kg Gd-DTPA, using the following parameters: TR/TE/flip angle=5ms/2ms/15°. Circumferential strain and infarct measurements were analyzed using HARP and ImageJ respectively. Paired and unpaired nonparametric t-tests and ANOVA with Dunn’s multiple comparison tests were used as appropriate.

Results: In control animals, delayed contrast enhanced MRI revealed no evidence of infarction or differential strain between LAD territory and remote myocardium (Fig. 1a, P=0.72). The infarct size was significantly smaller (8.8 ± 0.5% LV mass) in animals subjected to microembolization compared with animals subjected to LAD occlusion/reperfusion (12.4±1.2%, P<0.01). Delivered microemboli and LAD occlusion significantly decreased peak strain when compared to remote myocardium (Fig. 1b and c, both P<0.0001) or control animals (Fig. 2a). However, the difference in peak strain between animals subjected to either intervention did not reach the significant level, suggesting that there is disproportional correlation between infarct size and regional dysfunction in microembolized hearts.

At 3 days after interventions, remote myocardium showed no compensatory increase in strain compared to controls (Fig. 2), but a decrease in TTPS (strain rate). In contrast the TTPS of infarcted myocardium significantly increased compared to controls (Fig. 3), indicating regional dyssynchrony in the LV.

Conclusion: The pattern of acute myocardial infarct plays an important role in regulating strain and strain rate, which may affect mechanical synchronization within and between the ventricles. Furthermore, this study shows that there is disproportion between infarction size and circumferential strain in animals subjected to microembolization and LAD occlusion/reperfusion.

References: