CHRONOLOGICAL CARDIAC MRI ASSESSMENT OF CIRCUMFERENTIAL AND LONGITUDINAL STRAIN FOLLOWING MECHANICAL OBSTRUCTION OF CORONARY MICROVESSELS IN PRE-EXISTING INFARCT

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Hypothesis: The main processes that are responsible for acute and chronic mechanical changes in the left ventricle (LV) post-acute myocardial infarction (AMI) include resolution of transitive phenomenon (natural progression of AMI) (1), and long-term mechanics (contraction/relaxation and dyssynchrony) (2-4). These processes are well known to affect mortality (5). Many studies have employed echocardiography to measure cardiac mechanics, but this technique is heavily operator dependent and uses indirect indices of cardiac mechanics (6). Cardiac MRI can provide 3D quantitative radial, circumferential, and longitudinal strain data (7). These parameters have been shown to have higher sensitivity and specificity than segmental systolic wall thickening in predicting area of infarction (6) and LVEF (8). Two wxperimental models were designed to monitor and compare biventricular circumferential and longitudinal strain/strain rate in acute and chronic infarction.

Methods: Balloon angioplasty catheter was guided to LAD coronary artery under X-ray fluoroscopy. Pigs underwent 90min LAD occlusion/reperfusion (n=7) or LAD occlusion/reperfusion with microvascular obstruction induced by microemboli (n=7). Cardiac MRI was performed at 3 days and 5 weeks after coronary interventions using 1.5-T scanner. 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°. 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: Quantitative strain analyses are illustrated in figures 1-6. Briefly, there was no significant improvement in circumferential strain over the course 5 weeks in the infarcted regions. In remote myocardium an increase in circumferential strain was evident in the chronic setting (Fig. 1). Time to peak strain (TTPS) was significantly increased in remote myocardium ($P<0.05$), but not in the infarcted regions after 5 weeks (Fig 2). Dyssynchrony was defined as the difference in peak strain between remote myocardium and LAD territory and was significantly decreased at 5 weeks (Fig. 3, $P<0.05$), indicating improvement in synchronous contraction of the LV. Strain rate in remote myocardium demonstrated a difference at 5 weeks compared to 3 days (Fig. 4). Fig. 5 shows the difference in longitudinal strain between the right ventricular free wall (RVFW) and LV free wall (LVFW). Peak strain in the RVFW and LVFW was improved at 5 weeks compared with 3 days (Fig. 10). Longitudinal TTPS strain was not significantly different at 5 weeks (Fig. 6). Other metrics parameters of myocardial injury were unchanged in chronic phase (5 weeks).

Conclusion: Time resolved cine and tagged MRI have high specificity in chronologically monitoring circumferential and longitudinal strain changes related to the evolution of myocardial infarction and coronary microvascular obstruction in both left and right ventricles. Coronary microvascular obstruction accentuates cardiac dysfunction in acute and chronic infarct.

Fig. 1

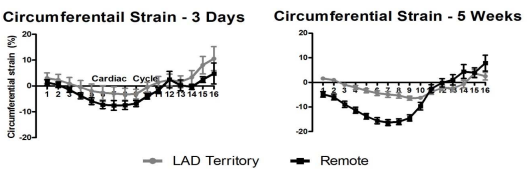


Fig. 2

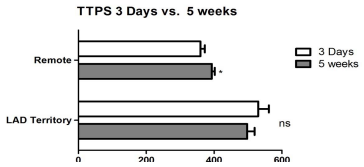


Fig. 3

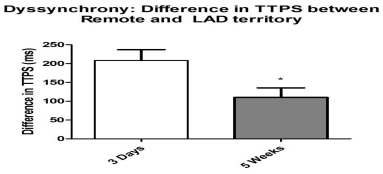


Fig. 4

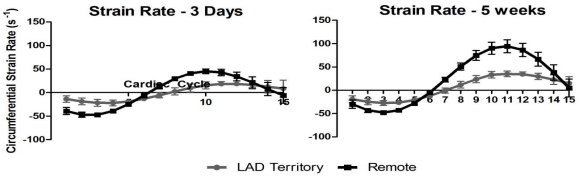


Fig. 5

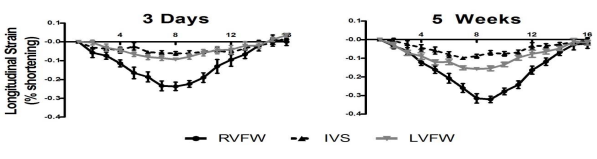
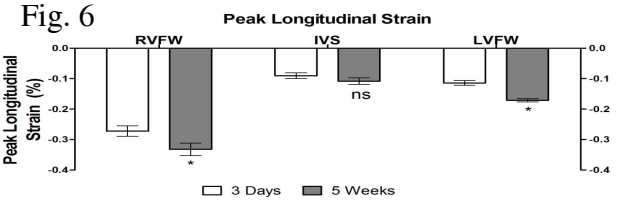


Fig. 6



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