MR QUANTIFICATION OF LONGITUDINAL AND CIRCUMFERENTIAL STRAIN IN LEFT AND RIGHT VENTRICLES SUBJECTED TO PATCHY MICROINFARCT, LARGE INFARCT AND COMBINED INSULT

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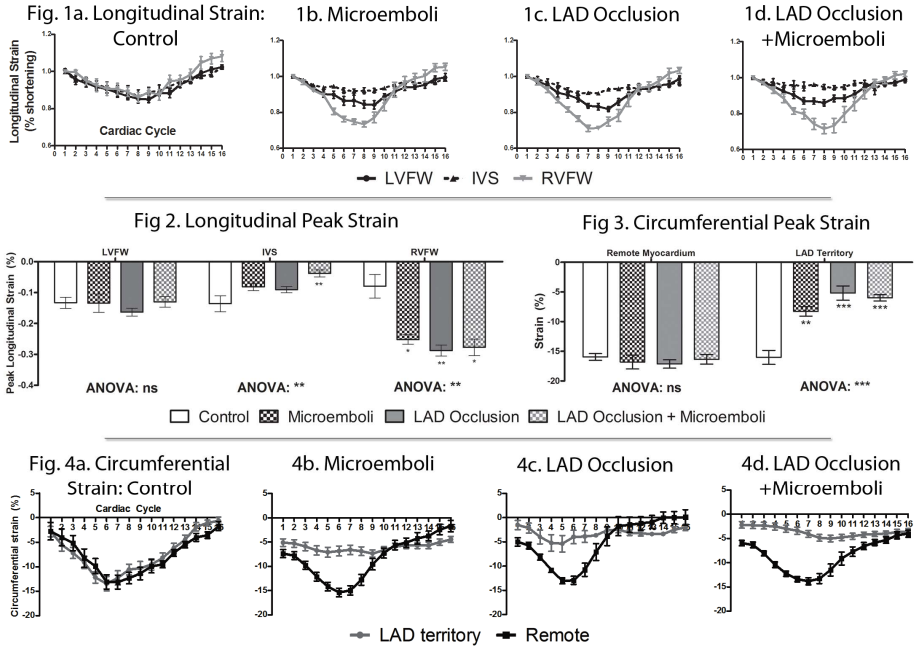
Background: The quantification of regional myocardial function serves as a valuable tool for the diagnosis and monitoring of cardiac function in patients with myocardial infarction and/or heart failure. A recent study reported that longitudinal strain after primary reperfusion therapy is an excellent predictor of LV adverse events in patients with anterior wall acute myocardial infarction[1]. Investigators also found that the combined assessment of both long- and short-axis function using 2D strain may be useful to identify the transmural extent of myocardial infarction[2]. In the current study, we used 4-chamber view cine MRI to measure longitudinal length of biventricular free walls and interventricular septum (IVS); a method routinely used in echocardiography[3]. We aimed to quantify longitudinal and circumferential strain in left and right ventricles in animals subjected to solely LAD microembolization and LAD occlusion/reperfusion with and without microembolization.

Methods: In swine (n=32), the LAD coronary artery was catheterized, using 3F balloon catheter, under X-ray fluoroscopy. The animals were subjected to 90min LAD occlusion followed by reperfusion (n=8), delivery of microemboli (32mm^3 volume and average diameter 80 µm, n=8), 90min LAD occlusion followed by delivery of microemboli and reperfusion (32mm^3 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. For longitudinal strain, cine MR images in the long axis view were acquired using a steady-state free precession sequence (pulse repetition time/TE/flip angle = 3.5 ms/1.75 ms/70). For circumferential strain, tagged MR images in the short axis view were acquired using a tagged turbo-field echo-planar sequence (TR/TE/flip angle=35/6.1ms/25). Phasic and peak longitudinal and circumferential strains were analyzed using *HARP*. Presence of myocardial infarct was documented on triphenyltetrazolium chloride. Paired and unpaired nonparametric t-tests and nonparametric ANOVA with Dunn's Multiple comparison tests were used as appropriate.

Results: Phasic longitudinal strain in the LV free wall (LVFW) and RV free wall (RVFW) of ischemic regions showed marked differences in temporal strain than controls (Fig. 1). All ischemic interventions (microemboli and LAD occlusion/reperfusion with and without microemboli) caused significant compensatory increased peak longitudinal strain in the RVFW (Fig. 2). LAD occlusion/reperfusion with microemboli caused severe reduction in IVS longitudinal strain compared with controls (Fig. 2). All ischemic interventions caused significant suppression in circumferential strain in LAD territory (corresponding to IVS in swine) compared with remote myocardium (corresponding to LVFW) and controls (P<0.01), although no significant difference was noted between intervention types (Fig. 3). The infracted regions of all groups showed akinesis (no contraction, Figs. 4b-d). In control animals, there was no significant difference in circumferential strain between LAD territory and remote myocardium (Fig. 4a).



Conclusion: Cine and tagging MRI show that infarcted segments in the left ventricle caused by LAD occlusion/reperfusion with microemboli have the most severe impairment in both longitudinal and circumferential strain, while solely microemboli or LAD occlusion intervention affected only circumferential strain. The interaction between right and left ventricles after ischemic interventions is clearly demonstrated by the compensatory increase in right ventricular free wall contraction.

References:

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