

COMPREHENSIVE AND SERIAL EVALUATION OF MYOCARDIAL STRUCTURE, FUNCTION AND PERFUSION IN REPERFUSED INFARCT

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Purpose

Previous studies investigated myocardial structure, function and perfusion individually at different time points. The purpose of this study was to 1) assess the changes in LV volumes, ejection fraction, LV mass, regional wall thickness, and 3D wall strain as well as myocardial structure, edema, microvascular obstruction and intramyocardial haemorrhage in reperfed infarct in a single imaging session, to ensure image co-registration and 2) compare the LV changes over the course of 10 weeks.

Methods

A percutaneous balloon catheter (Boston Scientific, Natick, MA) was advanced distal to the 2nd diagonal branch under X-ray and the artery was occluded for 90min. Coronary angiograms were repeatedly acquired to ensure complete occlusion and reperfusion. MR imaging was performed at 3 days (n=15 pigs), 5 weeks (n=13), and 10 weeks (n=6) after infarction using 1.5T MR scanner (Phillips, The Netherlands). The following imaging sequences were used: 1) Cine MR imaging for measuring LV volumes, ejection fraction, cardiac output, stroke volume, LV mass, wall thickness and radial strain (% systolic wall thickening in remote and infarcted myocardium); 2) T2 weighted turbo spin echo for measuring the extent of edema in the area at risk of infarct; 3) T2* weighted turbo spin echo for detecting intramyocardial hemorrhage; 4) Tagged MR imaging for measuring regional circumferential strain and absolute LV rotation; 5) Phase-contrast velocity-encoded MR imaging for measuring regional longitudinal strain; 6) First-pass perfusion MR imaging for detecting ischemic myocardium and measuring maximum upslope, peak signal intensity and time to peak; and 7) Contrast enhanced MR imaging for measuring infarct size and transmuralty. After imaging, animals were euthanized and the freshly excised hearts were sliced and stained with triphenyltetrazolium chloride (TTC) to delineate the infarct. The MR images were registered with TTC slices using anatomical landmarks. Hematoxylin and eosin and Masson trichrome were used to stage the infarct.

Results

Myocardial structural

T2 and T2* MR images demonstrated the edema in the area at risk of infarct as bright region in all animals and intramyocardial haemorrhage as dark region in 30% of the animals. Microvascular obstruction was observed in all animals with intramyocardial haemorrhage. Cine, tagged and phase-contrast velocity-encoded images showed significant wall thinning in the infarcted wall. The infarct wall thickness during diastole were 5.7 ± 0.4 mm at 5 weeks and 5.4 ± 0.6 mm at 10 weeks compared to remote myocardium (8.5 ± 0.5 mm and 8.7 ± 0.2 mm, respectively as well as to 3 days (8.4 ± 0.3 mm infarct and 8.3 ± 0.3 mm remote myocardium). On contrast enhanced MR imaging, the infarct was observed as a hot-spot (hyperenhanced) surrounding microvascular obstruction zone. Mean values of infarct size at 3 days ($16.0 \pm 0.7\%$), 5 weeks (12.4 ± 1.1 P<0.01), 10 weeks (12.7 ± 0.2 , P<0.01) and at post-mortem TTC (12.1 ± 0.7). There was no significant difference in infarct size on contrast enhanced MR imaging at 5 weeks and 10 weeks after infarction compared to TTC at 10 weeks, suggesting that the infarct size reached plateau level. The 35% decrease in infarct size between 3 days and 5 weeks after infarction reflect natural infarct resorption (healing) seen previously in swine.

Cardiac contractility

At the global level, there was no significant change in LV ejection fraction between 3 days ($37 \pm 1\%$) 5 weeks (37 ± 1) and 10 weeks (37 ± 1) after infarction. However, there was progressive increase in end diastolic volume between 3 days (75 ± 4 ml), 5 weeks (92 ± 3) and 10 weeks (114 ± 3) after infarction. A similar increase was observed in end systolic LV volumes (48 ± 2 ml, 56 ± 2 , 71 ± 2 , respectively), indicating the continuation of LV remodeling. At the regional level, cine, tagged and phase-contrast velocity-encoded MR imaging showed the severe and persistent dysfunction in the infarct compared to remote myocardium at all time points. Figure 1 shows the percent changes in peak radial, circumferential and longitudinal wall strains as a function of time.

Perfusion

First pass perfusion MR imaging showed the infarct as hypoenhanced region compared to remote myocardium. Quantitative measurements of maximum upslope and peak signal intensity showed significant decline and increase in time to peak. Histopathology showed the complete healing of the infarct at 10 weeks.

Conclusion

MR imaging provides comprehensive and serial characterization of evolved infarct. Myocardial edema, microvascular obstruction and hemorrhage are transient features of reperfusion injury that can be detected on MR imaging. Based on the MR pulse sequences used in this study, five weeks is enough time to arrest fibrosis, but not LV dilation, in reperfed infarct.

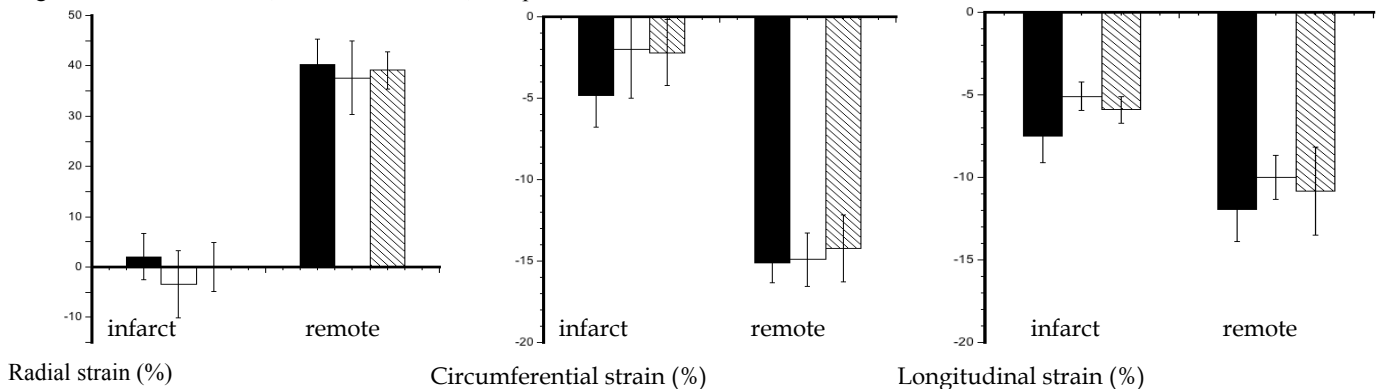


Figure 1 shows the changes in radial, circumferential and longitudinal strain at 3 days (Black bar), 5 weeks (white bar) and 10 weeks (stripped bar) in remote and infarct.