MICROEMBOLIZATION CONTRIBUTES TO ACUTE AND SUBACUTE LEFT VENTRICULAR DYSFUNCTION: MR ASSESSMENT OF LEFT VENTRICULAR FUNCTION AND STRAIN

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INTRODUCTION: Microembolization following unstable angina pectoris or PCI and CABG treatment causes microinfarction. It is thought to be one of the factors of mismatch between blood flow in the epicardial coronaries and left ventricular (LV) function, in patients. To our knowledge there is no study using MR to serially and non-invasively study the effects of microembolization on global LV function and regional strain.
PURPOSE: To link microinfarction caused by a predetermined size of embolic agents to the acute global and regional LV dysfunction and to noninvasively monitor the changes in LV volumes, ejection fraction, wall thickening and strain for 1 week after deposition of embolic agents.
MATERIALS AND METHODS: The studies were performed in hybrid XMR suite (Philips Medical Systems). Six pigs (33±3kg) were injected with embolic agents (n=10^4, 100-300 μm, Embosphere®, Biosphere Medical, Rockland, MA, USA) after selective catheterization of the LAD coronary artery. The embolic agents were injected into LAD coronary artery distal to first diagonal branch. Serial MR imaging was performed for assessment of the effect of microinfarction on global and regional LV function. Images were acquired prior to the injection (baseline), 1hr and 1week after the selective delivery of embolic agents. Short axis images covering the whole LV were acquired using 1) IR-GRE imaging for viability, 2) cine (steady state free precession, α=70°, TE=1.8 ms, TR=3.6 ms, Image resolution=1x1x10 mm, no slice gap, retrospective ECG-triggering with 16 time phases) and 3) tagging MR images (CSPAMM, α=25°, TE=6 ms, TR=37 ms, Image resolution=1x1x10 mm, no slice gap, prospective triggering with 16 heart phases) using a 1.5 T Philips Intera. Cine images were acquired and used to calculate end diastolic volume (EDV), end systolic volume (ESV) and ejection fraction (EF). Regional function was assessed by measurement of systolic wall thickening. Circumferential strain analysis was performed using the HARP software (Diagnosoft Inc. USA). At the conclusion of the second imaging session, the animals were sacrificed and TTC and histopathology was used to confirm delivery of the embolic agent and microinfarction.
RESULTS: Under X-ray fluoroscopy, all animals were successfully catheterized and a 3F catheter was precisely positioned in the desired segment of LAD coronary artery. After moving of the animal on floated table to MR scanner, selective injection of Gd-DOTA (6ml, 10%) was performed to map the territory of distribution of the embolic agent in myocardium (**Fig. 1**). Embolic agents delivered into the coronary artery caused persistent decreased ejection fraction over the course of one week (51±1% at baseline, 30±4% at 1hr and 35±3% at 1 week) (**Fig.1**). EDV was progressively increased from 86±8 ml at baseline, 96±8 ml at 1 h, and 107±12 at 1 week, likewise ESV also increased from and 42±3 ml, 67±5 ml and 70±9 ml, respectively, whereas systolic wall thickening significantly (P<0.05) decreased in the embolized and to a lesser extent in remote myocardium (**Fig. 2**). Furthermore, circumferential strain in the embolized region decreased from -18±1% during peak contraction at baseline to -8±2% 1hr after delivery of the embolic agent (**Fig. 2**). At 1 week there was no recovery of circumferential strain in the embolized region (-7±1%). Circumferential strain in the remote myocardium showed a trend of deterioration from -18±1% to -14±1% at 1hr and -15±1% at 1 week. TTC and histopathology confirmed the presence of microinfarction.

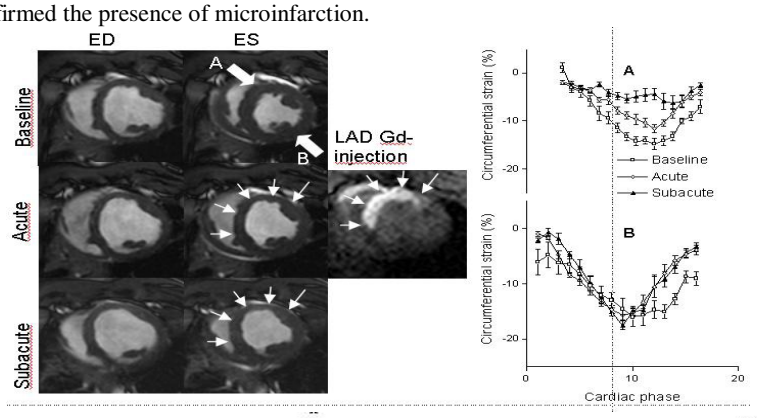

Fig. 1. End-diastolic (ED) and end-systolic (ES) images at baseline, acute and subacute phases of microinfarction showing the changes in LV chamber. Thick arrows indicate the areas of the strain measurements; namely (A) embolized region and (B) remote myocardium. White arrows show the embolized region with decreased systolic thickening on acute and subacute images. The plots show the circumferential strain throughout the cardiac cycle in the embolized region and remote myocardium at baseline, acute and subacute. Note the decrease in strain in the embolized region 1hr and 1 week after microembolization.

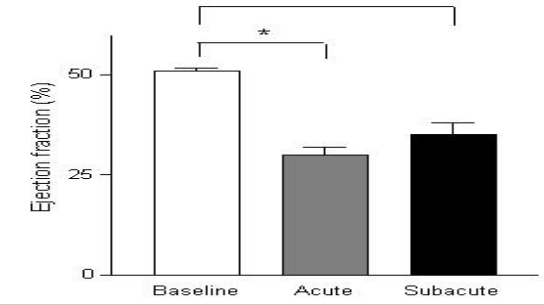

Fig. 2. LV ejection fraction before and after intracoronary delivery of embolic agent. The agents caused acute (1hr) and subacute (1 week) decline in ejection fraction. * P<0.05.

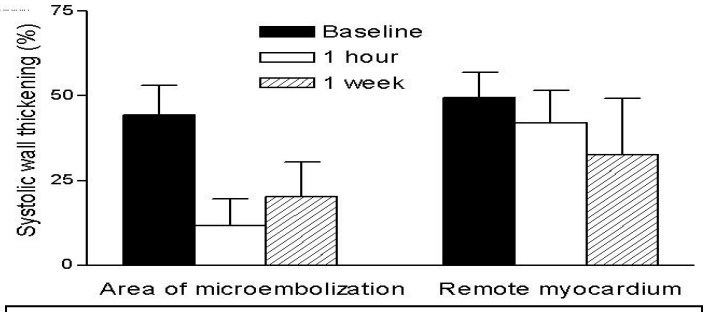

Fig. 3. Systolic wall thickening in the embolized and remote myocardium before and after (1hr and 1 week) of the delivery of the embolic agents. Both regions showed dysfunction at 1 week, linking microinfarction to LV dysfunction.

CONCLUSION A link between microinfarction and LV dysfunction has been established in swine model. Analysis of LV volumes and ejection fraction indicate acute and subacute LV remodeling. The data showed persistent decline in wall thickening and strain after embolization. Microembolization after coronary intervention and CABG in patients could be a cause of LV dysfunction.

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