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Poster presentation

Coronary microemboli have long-term effects on regional left ventricular function: MRI 3D strain analysis

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Introduction

Microembolization is common during coronary intervention in patients. The long-term effects of microemboli on regional 3D strain and global left ventricular (LV) function have not been examined.

Purpose

This experimental study investigated whether coronary microemboli have long-term deleterious effects on LV function using MRI strain analysis.

Methods

A hybrid X-ray and MRI system was used to catheterize the LAD in 6 pigs and deliver the embolic materials (40-120 μ m, 250,000 count). The area at risk (AAR) was determined on first-pass perfusion (FPP) MRI before and after microembolization. Delayed enhancement (DE)-MRI and histochemical staining were used to visualize and measure microinfarcts. Analysis of longitudinal strain was performed on phase-contrast MRI and radial strain as well as global function on ssfp MRI using Segment <u>http://segment.heiberg.se</u>. Tagged MRI were analyzed to measure circumferential strain using HARP. The Student's t-test was used to determine if strain over the cardiac cycle differed between baseline, acute (1 h) and chronic phases (7-8 weeks).

Results

Coronary microemboli caused an acute reduction in ejection fraction $(32 \pm 3\% \text{ vs. } 50 \pm 3\%, P < 0.05)$ compared to baseline. Longitudinal strain in the AAR and remote

acutely declined compared to baseline (P < 0.001, Figure), suggesting that microemboli has acute global effects. At the chronic phase the AAR displayed a partial and remote a full recovery of function compared to baseline (Figure). Radial strain also declined in the AAR ($1.0 \pm 7.2\%$ vs. 58.1 \pm 6.3%, P < 0.004) and remote (17.8 \pm 6.8% vs. 51.1 \pm 5.8%, P < 0.01) at the acute phase compared to baseline. There was a persistent decrease in the AAR but not in remote at the chronic phase (P < 0.001 and P = 0.18). On the contrary, there was a persistent decline in circumferential strain at the acute and chronic phase in the AAR compared to baseline, as well as in remote area (P < 0.001, Figure 1). FPP, DE-MRI and histopathology at postmortem confirmed the presence of microembolization and microinfarction. Microinfarcts could be detected in the acute phase as a perfusion defect on FPP but not on DE-MRI ($0.1 \pm 0.0\%$ LV). No perfusion defect could be visually detected on FPP at the chronic phase but DE-MRI showed speckled hyperenhancement in the AAR. The microinfarct size was $6.6 \pm 0.5\%$ LV mass.

Conclusion

This serial MRI study demonstrated a persistent regional LV dysfunction in the microembolized area. Regional 3D strain measurements may be useful in predicting decreased contractility in the LV in patients with biomarker release after coronary intervention.

* P < 0.05, *** P < 0.001 compared to baseline and $\dagger p < 0.05$ compared with AAR at the same stage.



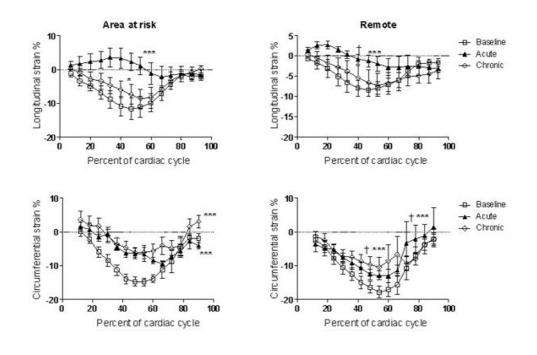


Figure I