

POSTER PRESENTATION

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Acute reperfusion intramyocardial hemorrhage leads to regional chronic iron deposition in the heart

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Background

Intramyocardial hemorrhage commonly occurs in large reperfused myocardial infarctions. However, its long-term fate remains unexplored. We hypothesized that acute reperfusion intramyocardial hemorrhage leads to chronic iron deposition.

Methods

Fifteen patients (mean age = 58±8 years; 3 women), who underwent successful angioplasty for first STEMI, were recruited following informed consent. Cardiovascular Magnetic Resonance (CMR) imaging (1.5T) was performed on day 3 and month 6 post-angioplasty. 2D T2* maps (6 TEs = 2.6-13.7 ms; ΔTE=2.2ms) and Late Gadolinium Enhancement (LGE) images of the entire left ventricle (LV) were acquired. Threshold-based image analysis was performed to identify remote, hemorrhagic (Hemo+) and non-hemorrhagic (Hemo-) myocardium.

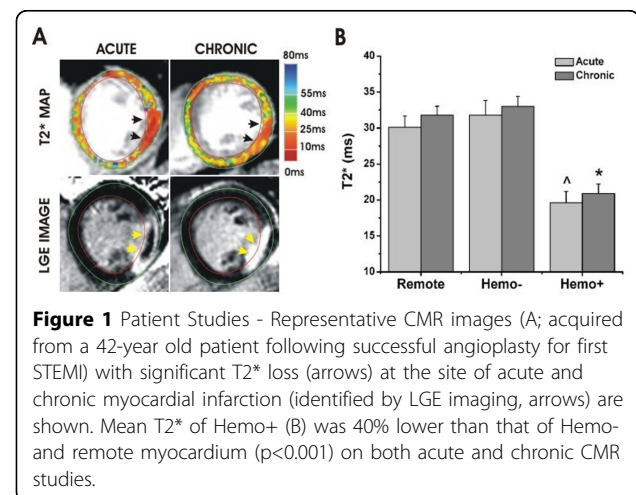
Fourteen canines, subjected to ischemia-reperfusion (I-R) injury (3 hours of LAD occlusion followed by reperfusion), underwent CMR (1.5T) on days 3 and 56 post-I-R injury. Three sham-operated animals (Shams) were also studied using CMR at similar time points. 2D T2* maps (6 TEs = 3.4-18.4 ms; ΔTE=3.0ms) and LGE images of the entire LV were acquired. Threshold-based image analysis was performed to identify remote, Hemo+ and Hemo- myocardium. Subsequently, animals were euthanized (day 56), hearts were excised and imaged ex-vivo. Sections of Hemo+, Hemo-, remote and Sham myocardium were isolated and histology was performed. The concentration of iron

([Fe]) within each type of tissue was measured using mass spectrometry.

Results

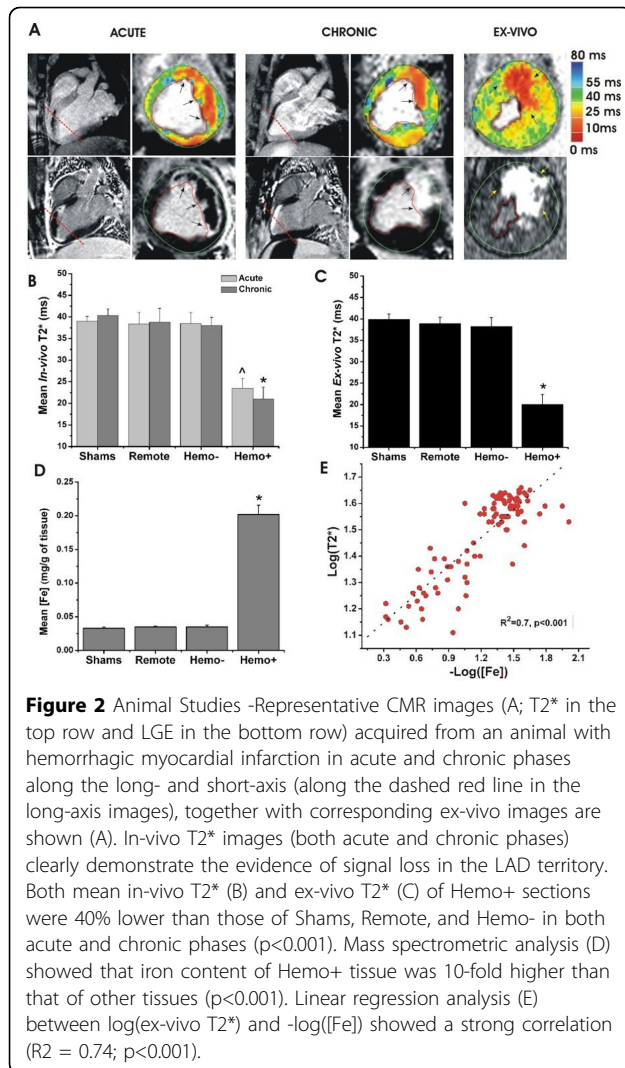
Six months post-angioplasty, mean T2* of the scar tissue in patients with Hemo+ infarctions (n=11 as determined by T2* losses within the infarct on day 3 CMR; Figure 1) was 40% lower than that of remote myocardium, suggesting chronic iron deposition (p<0.001). In contrast, mean T2* of Hemo- infarctions (n=4) was not significantly different from that of remote myocardium at both 3 days and 6 months post-angioplasty (p=0.51).

In canines, in-vivo mean T2* of Hemo+ myocardium was 40% lower than those of Sham, remote and Hemo-myocardium (p<0.001) at both 3 days and 56 days post-I-R injury (Figure 2B). Similarly, mean ex-vivo T2* of Hemo+ myocardium was 40% lower than those of Sham, remote



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and Hemo- myocardium ($p < 0.001$; Figure 2C). Perl's stain confirmed localized chronic iron deposition only within Hemo+ infarctions. Mean [Fe] of Hemo+ infarctions was nearly 10-fold higher than those of Sham, remote and Hemo- myocardium ($p < 0.001$; Figure 2D). A strong linear relationship was observed between $\log(\text{ex-vivo } T2^*)$ and $-\log([\text{Fe}])$ ($R^2 = 0.7$; $p < 0.001$; Figure 2E).

Conclusions

Acute reperfusion intramyocardial hemorrhage leads to regional chronic iron deposition within the infarct zones. T2* CMR can accurately characterize localized chronic iron deposition following reperfusion-induced myocardial hemorrhage. The clinical significance of this finding remains to be investigated.

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