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Balanced steady-state free precession cardiovascular magnetic resonance imaging of edema in reperfused acute myocardial infarcts - a translational study in animals and men

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Introduction

Edema imaging is usually performed using T2-weighted STIR imaging. We hypothesized that balanced steady-state-free-precession (b-SSFP) sequences also have sensitivity to detect edema in acute reperfused ST-elevation myocardial infarction (STEMI).

Methods

The study was conducted in mini-pigs and patients with acute reperfused STEMI. In the mini-pigs, myocardial infarction was created by angiographically guided balloon occlusion of the proximal left circumflex coronary artery for ninety minutes. The animals were imaged on day 2 or 3 after experimental ischemia/reperfusion.

For the clinical arm, patients from the coronary care unit were included within four days after successful percutaneous coronary intervention for STEMI.

All CMR images were obtained on a 1.5 T clinical system (Siemens, Germany), using the following sequences in a short axis orientation (slice thickness 10 mm, 0 gap): conventional cine SSFP, T2-STIR (patients only), late enhancement (10 min after injection of 0.2 mmol/kg Gd-DTPA), applying typical sequence parameters. Semi-quantitative threshold-based image analysis of late enhancement images (LE) identified the infarct region and infarct area was calculated. In the infarction zone and remote myocardium, on corresponding T2-STIR and SSFP images, signal and contrast, as well as the area of edema were measured and compared using paired t-tests, correlation statistics.

Results

In 13 pigs, the area of high SSFP signal and the area of LE correlated with R = 0.83. Signal intensity in the infarction zone on SSFP was higher than in the remote zone (203.5 ± 28.7 (edema) vs. 148 +/- 19.8 (remote), $p < 0.001$), with a contrast-to-noise ratio of 37 ± 13.

In 16 patients (age 57 ± 8 years, 3 female, STEMI location anterior/septal n = 9, lateral n = 1, inferior n = 6) on T2-STIR images, the signal intensity in the infarct zone was higher than signal in remote myocardium (351 ± 109 (edema) vs. 222 ± 81 (remote), $p < 0.001$), and the same was observed on SSFP (252 ± 35 (edema) vs. 163 ± 32 (remote), $p < 0.001$). Contrast-to-noise ratio efficiency (CNR_{eff}), corrected for voxel size, was not different between T2-STIR and b-SSFP (CNR_{eff} T2-STIR 77 ± 37 vs. CNR_{eff} b-SSFP 65 ± 30, $p = 0.30$). The edematous volumes as measured by T2-STIR correlated well with the volumes measured by b-SSFP ($R = 0.78$, $p < 0.001$), but on T2-STIR were little larger than on b-SSFP (volume of edema T2-STIR 6.4 ± 2.1 ml vs. SSFP 4.9 ± 1.9 ml, $p = 0.03$). Infarct volumes on LGE were 4.2 ± 1.6 ml Figure 1.

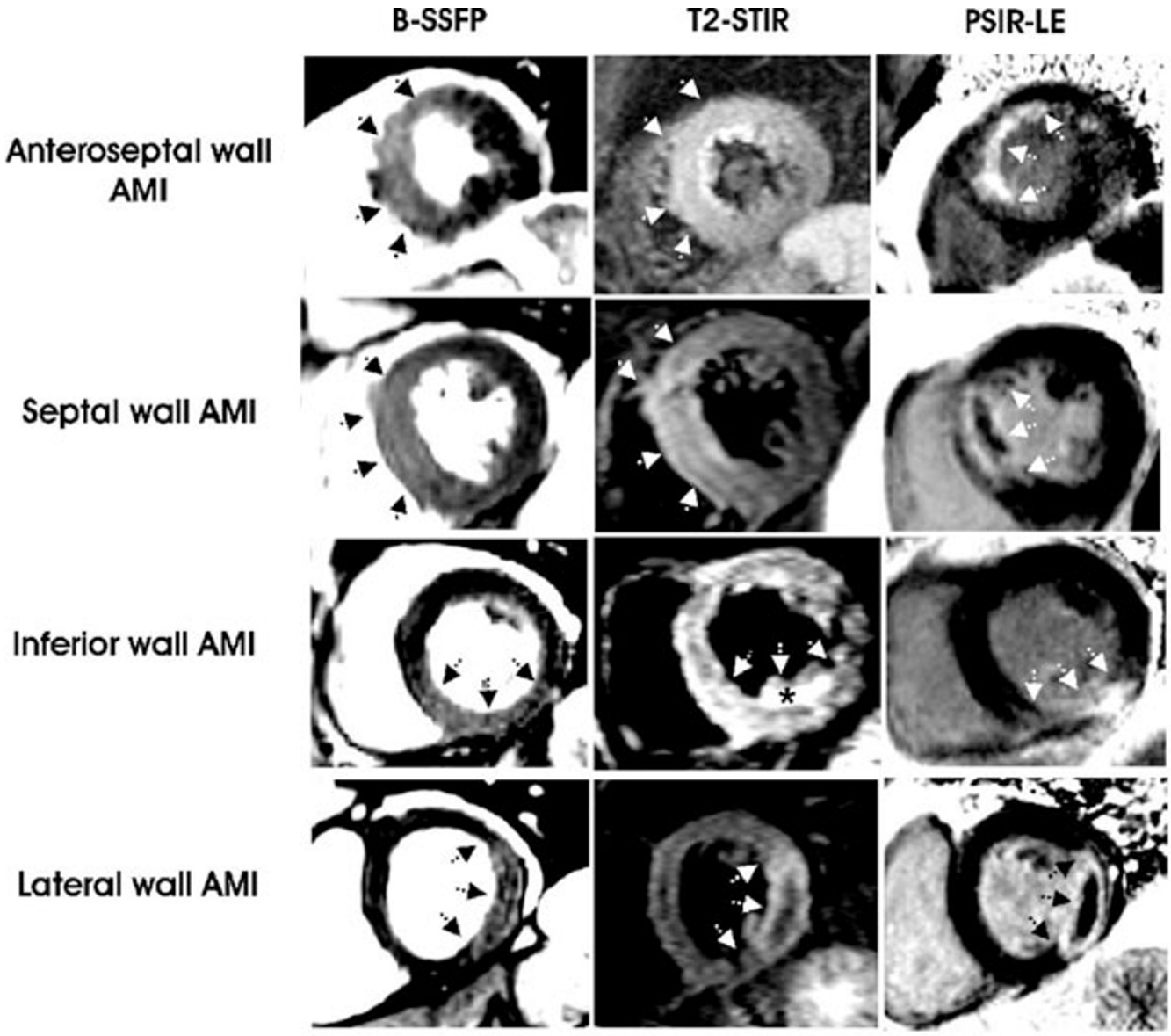

Figure 1
A representative set of edema weighted B-SSFP and T2-STIR images with difference STEMI locations in patients. Note the close correspondence between hyperintense territories identified by B-SSFP and T2-STIR acquisitions and the confirmation of infarcted territories on late-enhancement images (PSIR-LE).

Conclusion

Myocardial Edema in both, experimental and clinical STEMI, can be detected using SSFP imaging with contrast similar to T2-STIR.