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On the mechanism of myocardial edema contrast in T2-STIR images

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Introduction

Acute myocardial infarcts (AMI) are typically discriminated with T2-STIR imaging [1], albeit with limited specificity [2]. Guided by the association that T2-STIR images identify AMI territories on the basis of edema-related T2 changes, even some of the recently proposed improvements [2,3] have relied on preferential sensitization of magnetization to T2-weighting. However, whether T2-STIR imaging itself may also be sensitive to other sources of contrast have not been fully investigated.

Purpose

To demonstrate that in addition to T2-weighting, edema detection with T2-STIR imaging has substantial weighting from proton density (PD) changes.

Methods

Dogs (n = 3) subjected to an ischemia-reperfusion injury (LAD occlusion for 3 hours followed by reperfusion) were studied 2-hours post reperfusion (day 0), and on days 2, 5, and 7. Multiple breath-held and ECG-triggered T2-STIR images, and T2- and T1-maps were acquired using a Siemens Espree (1.5 T) system. All studies were terminated with PSIR late-gadolinium-enhancement (LGE) acquisitions to confirm LAD infarction. STIR images were acquired at the minimum TE(7.1 ms) and at 64 ms with body coil to limit potential surface coil bias. Other T2-STIR scan parameters were: TR = 2 R-R interval; TI = 170 ms; spatial-resolution = 0.9 × 0.9 × 8.0 mm^3. Affected territories on STIR images and T1- and T2-maps were identified using a semi-automated approach[1]. Mean signal intensities from STIR images, corresponding mean relaxation values, and residual longitudinal magnetization between the segmented TSE readouts were used to compute the relative changes in PD between affected and normal territories.

Results

Affected territories were identifiable on STIR (at both TEs) and LGE images, and on T2- and T1-maps as hyperintense zones (Figure 1). Table 1 lists the source of contrast in STIR images obtained at the different TEs. STIR signal changes at TE = 7.1 ms were primarily attributable to PD. However, at TE = 64 ms, the contrast was largely based on

Table 1: Mean T1, T2 and PDF contributions to image contrast between affected and healthy myocardial territories assess during each study.

Source	STIR (TE = 7.1 ms)	STIR (TE = 64 ms)
T1 effects	-25.3.7 ± 12.7%	-13.0 ± 6.7%
T2 effects	15.1 ± 3.7%	86.2 ± 25.7%
PD effects	115.9 ± 15.6%	25.5 ± 17.3%

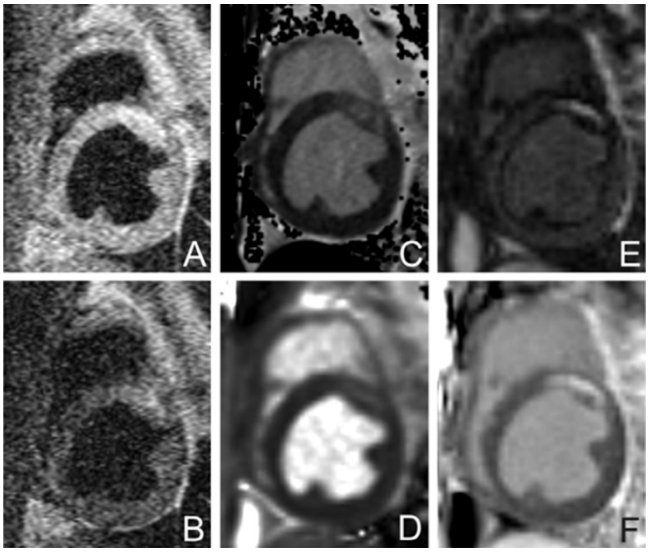

Figure 1
Representative short-axis images obtained from a dog subject to ischemia-reperfusion injury in the LAD territory. **A** STIR (TE = 7.1 ms); **B**: T2-STIR (TE = 64 ms); **C**: T1 map; **D**: T2 map; **E**: PSIR LGE magnitude image; **F**: PSIR LGE phase image. **A** and **B** were obtained with body coil, while **C-F** were ontained with surfance coil. Note that the LAD territory is hyperintense in **A** and **B**, indicating that even in the presence of a low T2-weighting (**A**), the affected region can be readily discriminated. Corresponding myocardial territories that appear hyperintese in **A** and **B**, also show elevations in T1 and T2 values (**C** and **D**, respectively). **E** and **F** confirm the presence of LAD infact.

T2 changes, along with significant contribution from PD. Mean signal intensity changes between affected and healthy territories from STIR images over all studies were: 33.4 ± 7.1% (TE = 7.1 ms) and 65.7 ± 12.9% (TE = 64 ms).

Conclusion

In addition to the T2 changes, edematous territories identified on the basis of long TE STIR (T2-STIR) images, have substantial contribution from PD. Our findings suggest that methods that rely on the identification of edematous territories solely on the basis of T2 changes may compromise sensitivity. Approaches that combine PD and T2 contrast in a synergistic manner are expected to be the most sensitive for detecting myocardial edema.

At the low TE (7.1 ms), PD effect dominates, while at the higher TE, T2 contributions are dominant with a reduced, but a significant, contribution from proton density. Note that the T1-effects are always in the opposite direction to PD and T2 effects, as expected.

References

1. Hassan , *et al.*: *Circulation* 2004.
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