

Using T2*-maps as a quantitative indicator for myocarditis

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Introduction

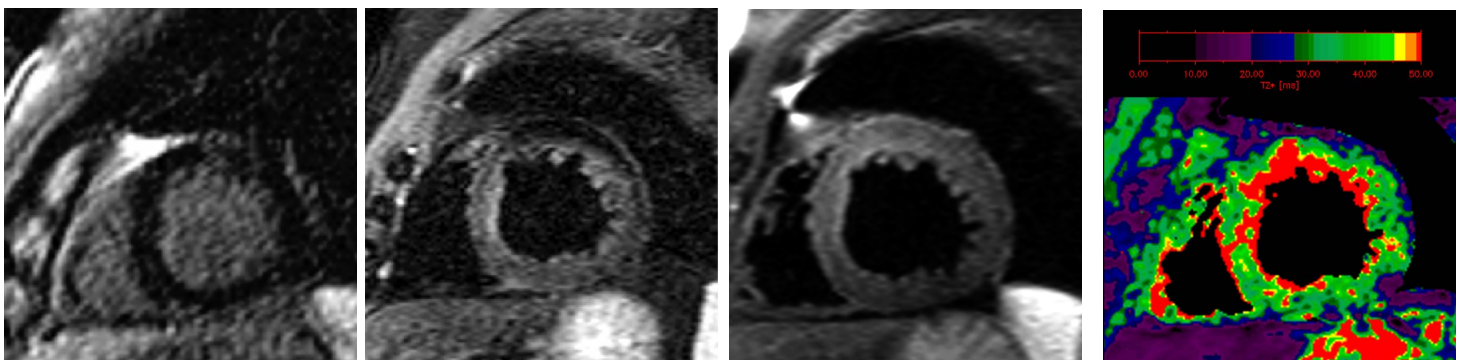
Diagnosis of myocarditis using cardio-MRI is currently based on indicators like late contrast agent enhancement and specular hyperintense areas in T2-weighted images. However, this standard procedure can lead to difficulties in cases of myocarditis which do not show accumulation of contrast agent due to severe damage in the myocardium or in cases of homogeneous or very diffuse edematous tissue [1]. In such cases, T2-weighted images can reveal a smooth, slightly increased signal level which is not easily interpretable as a clear indicator for myocarditis. Diagnosis can be furthermore hampered by technical limitations caused by the large time delays between preparation pulses and acquisition due to motion. Additionally, for patients with high pulse rates it becomes difficult to acquire data within a motion free phase for an echo train length of typically 150-200 ms. While T2* measurements are inherently prone to artifacts with longer echo times, a T2*-map calculated from a multi-echo gradient-echo acquisition may be sufficiently stable for quantitative analysis of myocardial edema. The aim was to assess the potential of T2* maps in order to differentiate normal myocardium from edematous tissue.

Material and Methods

T2*-maps were calculated from data obtained with a single slice, single breathhold, segmented (echo train length 7) multi-echo gradient-echo sequence (TE=4.76/9.52/14.3/19.0/23.8/28.6/33.3 ms) with a dark blood preparation pulse. Baseline T2*-values for healthy volunteers were determined at 1.5 and 3 T [2]. Although these normal values show some variation over different myocardium areas (septum, anterior and posterior wall) due to susceptibility artifacts [2], they were used for comparison with patients suffering from myocarditis. The color coding of the calculated maps was adjusted to display normal myocardium in green and all values above 49ms in red, indicating myocardial edema.

Results

Fig. 1 demonstrates the case of a patient showing images with the same midventricular slice position illustrating some of the common problems associated with the disease. No pathological late enhancement was seen (Fig. 1a), whereas in the T2-weighted IR recovery images (1b) an edematous involvement was detected in the septum. However, the anterior area is superimposed by a commonly seen motion induced artifact. The T2 TSE sequence in (c) shows a hyperintense myocardium in anterior, antero- and inferoseptal segments. The T2*-map in (d) clearly identifies the same area with suspiciously high T2*-values. In addition, the map shows an improved delineation of the edematous areas which is slightly larger than in (c).



(a) Segmented FLASH 3D, TE/TI/ α = 1.23ms/ 300ms/ 10°, TR each trigger (late enhancement)

(b) TIRM TE/TI=60ms/170ms, TR every other trigger, ETL 15, dark blood prepulse.

(c) T2 weighted TSE sequence, TE=69ms, TR every other trigger, ETL 15.

(d) TE=69ms, TR every other trigger, dark blood prepulse, map calculated from mono-exponential fit.

Figure 1: *In vivo* results of a 45-year-old patient with streptococcus associated myocarditis.

Discussion

Quantitative T2*-maps may yield valuable additional information in the diagnosis of myocarditis. Since all routine contrasts in Fig.1 a-c rely on repeated slice selective inversion or excitation any through plane motion will lead to signal dropouts. Although gradient echo images are prone to susceptibility artifacts, no further motion induced inhomogeneities are introduced once the slice selective excitation has occurred. The signal decay sampled by seven gradient echoes and the calculated T2*-values are free of motion induced signal inhomogeneities. It is anticipated that the quantitative nature of the T2* maps will help to resolve signal level interpretation ambiguities in patients with suspected myocarditis, which certainly has to be proven in a larger cohort of patients

[1] NS Yelgec et al. Eur Radiol 2007;17:2211-2217

[2] KH Herrmann, et al., In Proc. Int. Soc. Magn. Reson. Med., 2006. (#2563).