Assessing myocardial infarct using $T_{1\rho}$ and Late Gadolinium Enhancement in vivo

Elias Ylä-Herttuala[1], Svetlana Laidinen[1], Maarit Pulkkinen[1], Hanne Hakkarainen[1], and Timo Liimatainen[1]
[1]*Biomedical Imaging Unit, University of Eastern Finland, A. I. Virtanen instute, Kuopio, Finland*

Target audience: Physicians and researchers interested in imaging biomarkers of heart disease with MRI, especially for quantitative assessment of myocardium infarction.

Purpose: When perfusion to the myocardium is limited, the myocardium undergoes a loss of myocytes, inflammation, granulation tissue formation, and repair or remodeling of damaged tissues [1]. It has been shown that 2 days after infarction, the infarct consist mostly necrotic tissue and is transformed first to granulation tissue and finally to scar in 14 days after onset of ischemia in mouse infarct model [2]. T_2 has been shown to increase due to edema i.e. increased water content which is associated with necrosis in the ischemic myocardium in the acute phase (1 day after onset) while increased longitudinal rotating frame relaxation time ($T_{1\rho}$) has been related with later tissue remodeling i.e. granulation tissue formation and fibrosis [3,4]. Late gadolinium enhancement has been a standard MR method to assess chronic infarct volume [5]. In this study we compared infarct sizes obtained based on $T_{1\rho}$–relaxation time maps and LGE images to validate the $T_{1\rho}$–mapping as method for sizing the chronic phase of myocardial infarct.

Methods: In this *in vivo* research the infarct was induced by ligation of left anterior descending (LAD) artery in 9 c57bl mice. All experiments were done with 9.4 T magnet interfaced with Bruker BioSpec console. Gradient echo cine (TR=8.0ms, TE=1.9ms, slice thickness=1.0mm, matrix size=192x192, number of cine frames= 10-11) and $T_{1\rho}$ (continuous wave (CW) pulse power 1250 Hz, duration 0-57 ms) relaxation time was acquired 3 weeks after LAD occlusion. Fast imaging with steady state precession readout (TR=4.89ms, TE=1.9ms, 4 excitations after signal weighting) was used. LGE images were measured using fast imaging with steady state precession readout (TR=5.55ms, TE=1.99ms, and 300 ms delay after inversion pulse) 30 minutes after Gadovist injection (1.0 mmol/ml) into tail vein at 3 week time point.

Results and discussion: Infarct sizes were drawn based on elevated $T_{1\rho}$ in $T_{1\rho}$–map (Figure 1) and enhanced area in LGE images and the results were compared. Infarct sizes obtained based on $T_{1\rho}$ and LGE measurements showed high linear correlation (R^2=0.94, p<0.001) (Figure 2). For quality control of $T_{1\rho}$, B_1 was measured being 610 ± 30 Hz (mean±std) over all mice. High correlation between infarct sizes obtained with $T_{1\rho}$–map and LGE images indicates applicability of $T_{1\rho}$–mapping for infarct sizing in mouse models. The result is in line with the previous studies done in swine model and human cardiac infarcts. Infarct was easily discriminated from the viable myocardium in both $T_{1\rho}$–map and LGE images.

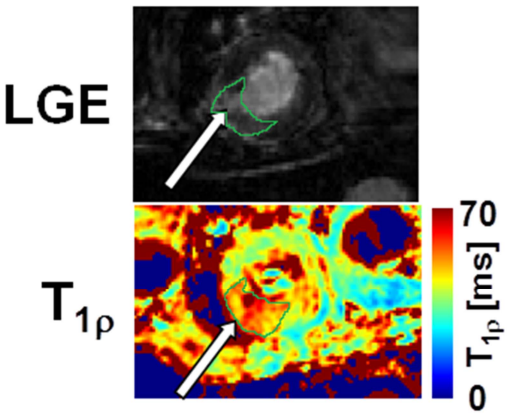

Figure 1. LGE image and T1ρ –map from one animal. White arrows are showing the infarct areas.

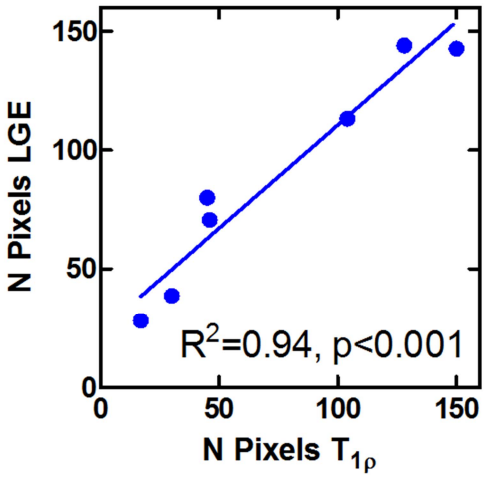

Figure 2. Comparison of LGE and $T_{1\rho}$ infarct sizes in imaging slice.

Conclusion: Based on this study, infarct areas determined by $T_{1\rho}$–mapping and LGE images was consistent with each other. Therefore, $T_{1\rho}$–mapping can be used to determine chronic infarct area in mouse model.

References: [1] Ertl G *et al. Cardiovasc Res* 66:22–32 (2005), [2] Virag JI *et al. Am. J. Pathol.* 163:2433–2440 (2003), [3] Mustafa HSN *et al. Magn Reson Med* 69:1389-1395 (2013), [4] Witschey WR *et al. Magn Reson Med* 64: 1453–1460 (2010), [5] Witschey WR *et al. J Cardivasc Magn Reson* 15:14-37 (2012).