Observer performance with state-of-the-art T2 weighted sequences (T2p ssSSFP, T2w ACUTE, T2 DB TIRM, T2 DB BLADE) for the detection of area-at-risk (AAR) in acute coronary syndromes (ACS).

M. Viallon[1], N. Mewton[2], D. Revel[2], P. Kellman[3], and P. Croisille[2]

[1]Radiology, Geneva university hospital, GENEVA, Switzerland, [2]Radiology, Hospices Civils de Lyon, Université de lyon, Lyon, France, [3]Radiology, Laboratory of Cardiac Energetics, NIH/NHLBI, Bethesda, Maryland, United States

Introduction

Determining the area-at-risk (AAR) in acute coronary syndromes (ACS) after Percutaneous Coronary Intervention (PCI) is remaining a challenge and is crucial to estimate the myocardial salvage and especially the AAR/infarct-size ratio. Depicting the AAR accurately maximizes then ability to test acute imaging paradigms. In addition, evaluating the occurrence of early hemorrhagic transformation or microvascular defects (no/low reflow) strongly influences the appropriate predictive analysis of final tissue outcome. All attempts to evaluate AAR using MRI involved T2 weighted (T2w) imaging because of its sensitivity to edema which is caused by the infarct and/or the reperfusion and has been shown to match the AAR. T2w dark blood (DB) TSE with inversion recovery (TIRM) is commonly set as a reference [1] and is supposed to show the AAR as a contrasted bright area within the myocardium but is often subject to artifacts. The spatial resolution is constrained by the breath hold duration and remains low. Recently white blood (WB) free breathing single-shot T2-prepared SSFP (T2p ssSSFP) readout and hybrid TSE-SSFP (ACUTE) breath hold provided alternate solutions to DB TSE in acute MI [2-3]. WB techniques circumvent flow artefactual subendocardial hypersignals but the ultra bright signal of the cavity imposes aggressive windowing to be able to indentify the edema area. Finally, BLADE k-space coverage for DB TSE sequence represents another free breathing alternative with increased spatial resolution and improved detailed depiction of morphology [4]. No clear consensus exists on the most appropriate sequence to characterize AAR. Our objective was to determine the performance of each technique to clearly identify regions of hyper/hypo enhancement (edema/no reflow) as a surrogate to AAR.

Methods

T2p ssSSFP with (Norm) and without (Unnorm) careful normalization for coil sensitivity, T2w ACUTE (Norm+Unnorm), T2 DB TIRM (Unnorm) and T2 DB BLADE (Unnorm) images (Fig.1) were systematically acquired in 27 patients with ACS (21/27 Acute Myocardial Infarct (AMI)+PCI within 5 days and 6/27 myocarditis) on a Siemens 1.5T Avanto scanner in addition to a conventional ACS protocol. Reading of all available data (cine, T2, delayed enhancement and x-ray coronary angiography) was performed independently by an experienced radiologist to determine abnormal segments. Presence and extent of the AAR were defined as regions in hypersignal (edema) from each available T2W images, with rest dysfunction (defined on cine), and located in the territory of the culprit artery AAR (defined by coronary angiography). Segments satisfying these criteria are considered as abnormal when establishing the reference. Readings of T2 images were then performed independently by two observers that were blinded to all corresponding clinical and imaging data of the patients. Patients and sequences were presented randomly, with only a single T2 image presented at a time. Readers were allowed to adjust freely W/L settings and asked to grade their confidence in the presence of any hypersignal/hyposignal within AHA myocardial segments according to a 5-points rating scale (0: absence up to 4: definite abnormality=hypo/hyper enhancement). Receiver Operating Characteristics (ROC) analysis were then conducted (Stata V10, College Station, Texas) to assess the performance of each technique to accurately classify segments.

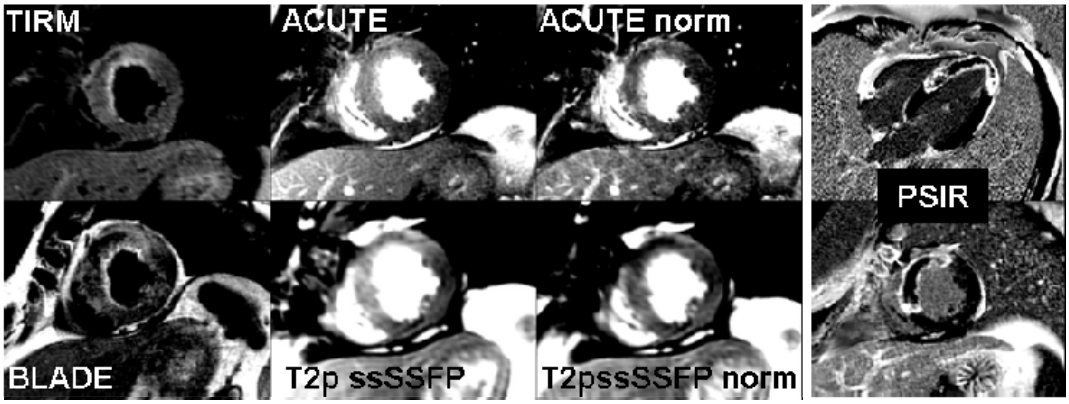

Figure 1. Illustration of typical images obtained on 1 AMI patient using all T2 weighted techniques evaluated here + late Gd enhanced (LGE) PSIR sequence. Images show localized edema (hypersignal in infero septal segment) and no reflow (hyposignal within edema on T2 images correlated with hyposignal within the hyper enhanced area on PSIR images). Clinical signs differ from one T2 sequence to another, but all techniques showed edema/no reflow matching more or less with LGE myocardium.

Results and Discussion

	Hyper versus Damage (AAR reference)	
Technique	ROC area	95% CI
T2 DB TIRM	0.83	0.77-0.89
T2 ACUTE Norm	0.82	0.76-0.88
T2 ACUTE Unnorm	0.83	0.78-0.89
T2 DB BLADE (free breathing)	0.72	0.64-0.80
T2p ssSSFP Norm (free breathing)	0.89	0.85-0.94
T2p ssSSFP Unnorm (free breathing)	0.85	0.80-0.91

Table 1. Results of ROC analysis based on reader 1 data that scale the accuracy of each T2 technique to detect hyper enhancement (edema) as a criterion for AAR determination, in all ACS patients. Significant differences across all techniques were found (p=0.0087 chi2=15.43), with the highest accuracy obtained with the T2p-ssSSFP (area under the curve, AUC=0.89), whereas T2 DB TIRM (AUC=0.83), ACUTE (AUC=0.82) and T2 DB BLADE (AUC=0.72) appeared less accurate.

ROC analysis allowed us to classify T2 techniques regarding to their relative performance to depict reperfusion injury with regards to: 1) capacity of detecting hyper enhanced regions likely to correspond to edema and 2) capacity of detecting no reflow. This analysis allowed us also to quantify the actual impact of coil sensitivity normalization (negligible). Diagnostic power of all techniques was increased (e.g. for T2p ssSSFP area=0.92/CI: 0.87-0.96) when considering AMI patients only, but decreased in our myocarditis population (e.g. for T2p ssSSFP area=0.82/CI: 0.69-0.94). On simple visual qualitative analysis, clear hypersignal (edema) was often seen on the images, and no reflow less evidently visually detected on T2 pre-contrast images. Nethertheless, specificity of 96% & 98% was reached regarding no reflow for the established rating scale of 1&2 respectively whereas specificity is much lower (94% & 95% for rating scale 1&2). And the more pathophysiological mechanisms are intricate (hemorrhagic lesions/no reflow/edema), the more clinical signs differ with the technique but always at the advantage of free breathing T2p ssSSFP. Natural sensitivity of GRE sequence to susceptibility helped showicing no reflow and hemorrhage lesion. DB Blade and TIRM are less sensitive when edema is combined to no reflow. Moreover the higher the resolution, the lower the sensitivity and low resolution technique such as TIRM and T2p ssSSFP were therefore able to highlight very mild hypersignal. All of these techniques rely anyway on the presence of edema but in cases where edema is reduced (e.g when induced by the therapy itself (cyclosporine, aspirin, etc) then sensitivity to no reflow might be of interest. T2p ssSSFP is then appearing to be the state-of-the-art method of choice to qualify and quantify the AAR, and is in addition very robust regarding to patient compliance and arrhythmia.

References: [1] Pennell D. Eur Heart J. 25: 1940–1965 (2004). [2] Kellman P. MRM. 2007;57(5):891-7. [3] Aletras A et al. MRM 59:229–235 (2008) [4] Viallon M, et al. Proceedings ISMRM, 16: 1008 (2008).