SCIENTIFIC EDITORIAL

T2 cardiac magnetic resonance in infarct patients: Sideman or leader?☆

Résonance magnétique cardiaque pondérée T2 en post-infarctus : instrumentiste ou leader ?

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Advances in T2-weighted cardiovascular magnetic resonance (CMR) imaging of the heart have overcome some of its limitations, such as low signal-to-noise ratio (SNR) and inconsistent image quality. T2-weighted images of the heart can be obtained in a short breath hold at each location [1] and should be acquired before gadolinium administration [2]. T2-weighted CMR is sensitive to regional or global increases in myocardial water content. T2 is the exponential time constant governing the rate of decay of transverse magnetization after the application of a radio-frequency excitation pulse. Tissues with a high water content have longer T2 values and therefore higher signals in T2-weighted images (bright signal intensity). Free water is the most significant contributor to the T2 signal intensity in muscle tissue [3]. In fast-segmented spin-echo pulse sequences [1], the double-inversion blood-nulling preparation is used to suppress the signal from flowing blood. Of note, enhanced sensitivity to tissue fluid and improved image dynamic range by fat suppression are obtained through the use of a third inversion pulse with a short inversion time inversion recovery (STIR). The STIR pulse effectively suppresses the signal from short-T1 tissues such as fat, highlighting the signal from long-T1, long-T2 species, such as fluid and oedematous tissue. Effective blood and fat signal suppression, rapid scan times and the ability to generate contrast have contributed to the widespread acceptance of black-blood

Abbreviations: CMR, Cardiovascular magnetic resonance; SNR, Signal-to-noise ratio; STIR, Short-inversion time inversion recovery.


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segmented fast-spin echo imaging. The use of surface coils for image acquisition may be limited by a progressive loss of signal in posterior and lateral myocardial segments, hindering image interpretation significantly. More specifically, the anteroseptal segments may appear hyperintense because of their proximity to the coil and a true high signal in the infarcted segments may be overlooked easily. This supports the combined use of phased array surface coils with signal intensity correction algorithms in T2 CMR sequences.

Oedema is a non-specific yet invariable pathological concomitant of acute injury. An increased myocardial water content has been found in several acute heart diseases, such as transplant rejection, acute viral myocarditis, Takotsubo cardiomyopathy, acute sarcoid lesions, pulmonary hypertension [4] and myocardial infarction [5,6]. The first report demonstrating the linear correlation between T2 relaxation time and myocardial water content in acutely infarcted myocardium was that from Higgins et al. [7] in an experimental dog model. Since then, a strong body of literature supports the hypothesis that T2-weighted CMR can identify infarct-related myocardial oedema [7—12]. Infarct-associated oedema can persist for up to 2—3 weeks after myocardial infarction and may offer a tissue memory marker for a recent acute injury after usual serum markers of tissue damage have normalized. Late enhancement CMR, although powerful in identifying irreversible injury, is unable to differentiate chronic from acute infarcts. In such cases, the presence of a high T2 signal may be very helpful for confirming a recent ischaemic injury [13,14]. High T2 signal intensity areas are consistently larger than the spatial extent of the irreversible injury, as defined by the extent of late enhancement in both animal models [11] and clinical studies [15], emphasizing the concept that the necrotic region is surrounded by an area of reversible injury, mainly characterized by oedema, and referred to as the salvageable area of risk.

In this issue of Archives of Cardiovascular Diseases, Manrique et al. report on the assessment of the spatial distribution of T2 relaxation time in 24 patients with recent acute myocardial infarction who underwent prompt prehospital thrombolysis, and rescue angioplasty in nine cases. Not surprisingly, myocardial T2 relaxation time was increased significantly in the infarct region (bright signal) compared with in the remote region, but also in the remote region compared with control subjects. In addition, patients with microvascular obstruction and no-reflow had further myocardial T2 lengthening within the subendocardium compared with patients without microvascular obstruction. The authors also found a significant correlation between peak troponin and T2 relaxation time. Interestingly, the authors were able to provide a T2 map within infarcts, remote myocardium and controls.

The correlation between myocardial oedema (long-T2) and reperfusion status reinforces the concept of a pathological role for acute oedema in the impairment of microvascular reperfusion, which is in agreement with other findings [16]. However, the explanation for increased T2 signal in no-reflow patients is complex, and it may be speculated that high T2 can also be caused by the confounding presence of acute haemorrhage in this setting. This also raises the issue of potential dynamic changes in T2 relaxation in such conditions, with varying haemoglobin degradation products over the first few days. Although the presence of a bright signal in the area of infarction is the rule, an area of low signal may appear in the core of the infarct, surrounded by peripheral high signal. The similarity of this pattern to the no-reflow areas observed early after gadolinium injection may indicate a relationship to no-reflow, and in animal models it could be related to haemoglobin degradation products [17]. More specifically, after a few hours or days, the presence of iron linked to haemoglobin may affect T2 relaxation times adversely and in a confounding way, with subsequent decrease of T2 signal. Compared with a standard recommended T2 segmented fast-spin echo sequence (STIR), the use by the authors of a half-Fourier turbo spin-echo sequence, meaning partial k space sampling, implies less density of the obtained T2 relaxation map. It should also be pointed out that the observed correlation of T2 relaxation to troponin rise is not independent, but is associated with infarct size. One should acknowledge the lack of a direct pathophysiological link between lengthening of T2, presence of oedema and irreversible tissue damage. The authors report further lengthening of T2 in areas of no-reflow, which could appear to be in opposition to experimental sodium magnetic resonance imaging data, showing delayed sodium wash-in in no-reflow areas and implicitly less water content [18]. The discrepancies between these data could be explained by different time delays between reopening of the infarct-related artery and the magnetic resonance studies. Besides the fact that spatial coverage of the left ventricle was quite limited in the study, a regular limitation of T2-weighted CMR images is that SNR and contrast are lower than in most other CMR images. Consequently, a frequent problem is misinterpretation of non-suppressed blood. Indeed, slow flowing blood, especially near hypokinetic segments and/or between trabeculae, may not be suppressed adequately and the corresponding high signal may be interpreted falsely as an affected area within the myocardium.

Overall, the main finding of this study is that T2 relaxation time in patients with myocardial infarction was elevated significantly in the remote region compared with the control population, which may be explained — as suggested by the authors — by early structural changes and widespread inflammatory infiltrate. This finding is interesting and merits further confirmation, as it may explain, at least in part, the relation of initial infarct size and initial area at risk to subsequent left ventricular remodelling, which corresponds to a more global process involving all myocardial segments. Although intriguing, these interesting data, along with other data, underline the potential and support the use of T2 CMR in patients with acute heart diseases and myocardial infarction.

Conflict of interest
None.

References


