**T_1** and **T_2** mapping for early diagnosis of dilated non-ischaemic cardiomyopathy in middle-aged patients and differentiation from normal physiological adaptation

Ify Mordi, David Carrick, Hiram Bezerra, and Nikolaos Tzemos

Aims

The differential diagnosis of patients with early non-ischaemic dilated cardiomyopathy (DCM) and those with physiological adaptation to exercise ('athlete's heart') may be difficult as many of the morphological adaptations are shared in the two conditions. Increased physical fitness is becoming more common in later adulthood, a group in whom there may be even more diagnostic difficulty. We hypothesized that tissue characterization using cardiovascular magnetic resonance (CMR) **T_1** and **T_2** mapping would be able to differentiate between patients with left ventricular (LV) dilatation due to early DCM and exercisers.

Methods and results

Fifty-eight middle-aged males [21 healthy controls, 21 males with a history of aerobic exercise and LV ejection fraction (LVEF) 45–55%, and 16 patients with DCM and LVEF 45–55%] underwent a CMR protocol including **T_1** and **T_2** mapping and calculation of extracellular volume (ECV) using a 1.5 T MRI scanner. Native **T_1**, ECV, and **T_2** relaxation times were significantly increased in DCM patients compared with controls (native **T_1** 1017 ± 42 vs. 952 ± 31 ms, **P** = 0.001; ECV 31.2 ± 4.1 vs. 26.2 ± 2.9%, **P** = 0.003; **T_2** 55.9 ± 4.4 vs. 52.9 ± 3.3 ms, **P** = 0.05) and exercisers (native **T_1** 957 ± 32 ms, **P** < 0.001; ECV 26.3 ± 3.6%, **P** = 0.004; **T_2** 52.8 ± 3.2 ms, **P** = 0.042). Using multivariable logistic regression, native **T_1** gave the best differentiation between exercisers and sedentary patients with early DCM (area under the curve 0.91).

Conclusion

**T_1** and **T_2** mapping are potentially useful tools for differentiating between athlete’s heart and patients with early DCM, and could be used whenever differentiation between these two phenotypes is inconclusive using standard imaging techniques.

Keywords

**T_1** mapping • **T_2** mapping • Cardiovascular magnetic resonance • Athletes • Dilated cardiomyopathy

**Introduction**

Cardiovascular magnetic resonance (CMR) imaging is increasingly becoming an integral part of the assessment of myocardial function and structure due to its accuracy and reproducibility in the measurement of left ventricular (LV) dimensions and function and its unique ability to non-invasively characterize myocardial tissue.**1,2** The assessment of LV ejection fraction (LVEF) is a particularly important indication for CMR, for example in non-ischaemic dilated cardiomyopathy (DCM). DCM is the final common pathway of a number of cardiomyopathies, but is typically characterized by a dilated left ventricle with an impairment of systolic function in the absence of significant obstructive coronary artery disease and in the presence of normal wall thickness.**3,4**

LV dilatation, despite being considered as the hallmark of DCM, is not always diagnostic of a pathological cardiomyopathy; it can develop as an adaptive response to chronically sustained physical exertion, known as ‘athlete’s heart’. Intensive aerobic (endurance)
exercise typically causes LV dilatation with significantly increased bi-
ventricular diameters and volumes, and consequently a mildly re-
duced LVEF (typically 45–55%). These changes have also been seen in older patients undergoing marathon training.8 Using simple parameters, such as LVEF, could potentially lead to diagnostic prob-
lems in the differentiation of early DCM and physiological adapta-
tion to exercise, especially in middle-aged and older patients, in whom the pretest probability of cardiomyopathy is probably higher. This is compounded by the fact that an increasing number of older adults are taking up more intense exercise.9 Correct early diagnosis of cardiomyopathy is of vital importance for future prognosis and management.

Late gadolinium enhancement (LGE) imaging during CMR can also add diagnostic confidence, allowing the identification of specific aetiologies of DCM.10 The presence of LGE, which is typically mid-
wall and occurs in ~30% of patients with DCM,11 is also an adverse prognostic indicator.12,13 Nevertheless, healthy older marathon runners have also been shown to have the present of LGE, which might again cause some diagnostic uncertainty.9,14

Recently, T1- and T2-mapping techniques have been utilized to provide further myocardial tissue characterization information in addition to LGE. These techniques rely on the fundamental magnet-
ic properties of the myocardium in order to identify (among others) diffuse fibrosis and oedema.15–17 To the best of our knowledge, the utility of T1 and T2 mapping in the diagnosis of physiological adaptation to exercise has not yet been evaluated.

In this study, we wished to evaluate the utility of T1 and T2 mapping in differentiation of athletes and patients with mild DCM, po-
tentially allowing for improvement in diagnostic confidence where the assessment of LVEF and LGE may still leave doubt.

**Methods**

**Patient selection**

We evaluated three separate cohorts of middle-aged male patients who all underwent CMR examination. The first cohort, who acted as the control group, consisted of 21 healthy control patients without any his-
tory of cardiovascular disease, on no medications, and normal resting electrocardiograms. The second group consisted of 21 males, prospect-
ively recruited, with a history of regular aerobic exercise (‘exercisers’) and mildly impaired or borderline LVEF using echocardiography (LVEF using Simpson’s biplane 45–50%). All patients in this group undertook over 6 h of intensive aerobic exercise per week at an amateur level (predom-
nantly running). These patients also had no history of cardiac dis-
ease, hypertension, and were on no medications, with diagnosis of cardiomyopathy excluded using case note review and the absence of an ischaemic pattern of LGE or significant valvular disease on CMR examination. The final group consisted of 16 patients with confirmed DCM (LVEF 40–50% by echocardiography) and without a history of en-
gagement in significant aerobic exercise and no family history of cardio-
myopathy. All of these patients had also undergone comprehensive evaluation to exclude ischaemic cardiomyopathy with invasive or CT coronary angiography or SPECT imaging (modality chosen at the discre-
tion of their clinician). Additionally, other causes of cardiomyopathy, such as hypertension or significant valvular heart disease, were also excluded based on clinical evaluation. As a final discriminator, any pa-
tients with a subendocardial or transmural pattern of LGE, suggestive of ischaemic cardiomyopathy, were also excluded. All DCM patients were on at least an ACE inhibitor and beta-blocker as per the current guidelines for management of heart failure. The study was approved by our local ethics committee and all patients gave written informed consent to undergo CMR examination.

**CMR protocol and analysis**

All patients underwent a systematic CMR examination using a 1.5 T MRI scanner (Magnetom Avanto, Siemens, Erlangen, Germany). Following the acquisition of localizers, cine imaging was carried out using steady-state free precession imaging (SSFP) to acquire a stack of short-axis slices and three long-axis views to fully assess LV mass, dimensions, and function, as previ-
ously described.18 Following this, we acquired native T1 maps in three slices representing the basal, mid, and apical slices using a modified Look-Locker inversion recovery (MOLLI) sequence.19 Several TrueFISP images were ac-
quired at various inversion times (TI) during a single breath-hold, allowing the measurement of the T1 time of each voxel to be displayed in a colour-
coded ‘map’. Typical parameters of the MOLLI sequence were: (echo time) TE 1.1 ms, initial TI 100 ms; TI increment 80 ms, flip angle 35°, matrix 192 × 124 pixels, spatial resolution 2.1 × 1.1 × 8.0 mm, slice thickness 8 mm, and scan time 17 heartbeats. A motion-correction algorithm was applied to compensate for any patient movement.

We then acquired T2 maps in the same three short-axis slices immedi-
ately after using an SSFP-prepared sequence.20 Three T2-weighted images were obtained during one breath-hold at 0, 24, and 55 ms, allowing gener-
ation of the T2 time for each individual voxel. Again, a motion-
correction algorithm was applied to allow for movement artefact. Typical imaging parameters were TE 1.1 ms, repetition time 260 ms, flip angle 70°, bandwidth ~947 Hz/pixel, matrix 160 × 105 pixels, spatial reso-
lution 2.6 × 2.1 × 8.0 mm, and slice thickness 8 mm.

After acquisition of these images, an intravenous injection of gadolin-
ium contrast was given (0.15 mmol/kg of gadobutrol; Gadovist, Bayer, Berlin, Germany). Ten minutes after this injection, an MOLLI sequence was acquired to obtain three short-axis post-contrast T1 maps. Immedi-
ately following acquisition of post-contrast T1 maps, LGE images were ac-
quired as previously described.18

**Image analysis**

All analysis was carried out using the proprietary software (Argus, Sie-
mens, Erlangen, Germany). LV mass and function was assessed by manu-
ally tracing round the endocardial (excluding papillary muscles) and epicardial borders to obtain ejection fraction.

To assess myocardial T1 and T2 times, as wide a region of interest as possible was drawn within the septum of the basal and mid-ventricular slices, taking care to avoid blood pool signal. The average of the two va-
ues was taken in order to have as a representative value of the myocard-
ium as possible. The apical slice and the lateral walls were excluded as we found that these areas were more likely to be affected by artefact.

Once the region of interest was drawn, T1 and T2 times were automatic-
ally displayed on the workstation. On T1-weighted images, a large rep-
resentative region of interest was also drawn within the blood pool. Motion correction was not used for analysis of the maps.

The presence of LGE was determined by the presence of areas of en-
hanced myocardium with signal intensity 5 standard deviations higher than non-enhanced myocardium.

**Calculation of extracellular volume**

A blood sample was taken immediately prior to CMR scanning to ana-
ylse the full blood count. The haematocrit was obtained, allowing for
calculation of extracellular volume (ECV) using the following equation: 
ECV = (1 − haematocrit) × (ΔR1myocardium/ΔR1blood). 21

Statistical analysis
All continuous variables are presented as mean ± SD, whereas non-continuous variables are expressed as percentages. Comparisons between multiple groups were carried out using a one-way ANOVA with Bonferroni correction applied for multiple group testing. Statistical significance was set at <0.05. Correlations were assessed by Pearson’s correlation coefficient. Binary logistic regression was used to assess the value of native T1 and T2 mapping parameters for identification of DCM patients. Native T1 and T2 values and calculated ECV were incorporated into univariable analysis as continuous variables, and significant predictors (P < 0.05) were then included into multivariable analysis. Sensitivity and specificity was analysed using receiver-operator characteristic (ROC) curves, in order to obtain the area under the curve (AUC). All statistical analysis was carried out using SPSS 22.0 (IBM, Armonk, NY, USA).

Results
In total, 58 males were evaluated—21 with a history of aerobic exercise, 16 with mild DCM, and 21 age-matched healthy controls. The average age of the cohort was 47.9 ± 14.5 years.

Correlations between tissue characteristics and LV function
Myocardial T2 times were significantly correlated with both native T1 times (r = 0.53, P < 0.001) and ECV (r = 0.56, P < 0.001). There was a strong correlation between native T1 and ECV (r = 0.69, P < 0.001).

LVEF was also correlated with both native T1 (r = −0.40, P < 0.001) and ECV (r = −0.43, P < 0.001). T2 values were not significantly correlated with either LVEF. There were no significant correlations between age, native T1, ECV, or T1 values.

Differentiation of aerobic exercisers, DCM patients, and controls
Both exercisers and DCM patients had significantly increased LV volumes and reduced LVEF compared with control (Table 1). Exercisers also had an increased LV mass, compared with both controls and DCM patients.

DCM patients were more likely to have LGE than both controls and exercisers (P = 0.027). There was no subendocardial or transmural LGE in any group to suggest prior myocardial infarction or ischaemia—four patients in the DCM group had midwall the present of LGE, whereas two exercisers had small amounts of the present of LGE at the right ventricular insertion points.

Both native T1 and ECV were significantly different between athletes and DCM patients (native T1 957 ± 32 vs. 1017 ± 42 ms, P < 0.001; ECV 26.3 ± 3.6 vs. 31.2 ± 4.1%, P < 0.001, respectively). Athletes had a similar native T1 and ECV to controls (Figures 1 and 2). T2 relaxation time was also similar between controls and athletes (52.9 ± 3.3 vs. 52.8 ± 3.2 ms, respectively) and significantly lower than DCM patients (55.9 ± 4.4 ms, P = 0.024 between all three groups).

Native T1 (OR 1.05; 95% CI 1.02–1.08, P = 0.002), ECV (OR 1.38; 95% CI 1.09–1.75, P = 0.009), and T2 (OR 1.23; 95% CI 1.01–1.49, P = 0.042) were all significantly associated with the presence of DCM in univariable analysis. Using multivariable logistic regression analysis, native T1 was the only independent discriminator between athletes and DCM patients (OR 1.06; 95% CI 1.01–1.10, P = 0.015; Table 2). Using ROC analysis, the AUC for differentiation between athletes and DCM patients was 0.91 (95% CI 0.82–1.00, P < 0.001), which was significantly better than both ECV (AUC 0.82; 95% CI 0.67–0.98, P = 0.002) and T2 relaxation time (AUC 0.74; 95% CI 0.56–0.92, P = 0.02; Figure 3).

Discussion
In this study, we identified several important findings. First, in the largest cohort so far evaluated, we have shown the incremental diagnostic utility of both T1 and T2 mapping to identify changes in the myocardium in patients with DCM. Secondly, we have shown that native T1 especially might be used to differentiate between middle-aged exercisers with mildly impaired LV function due to physiological adaptation and middle-aged patients with early DCM.

Both T1 and T2 mapping have emerged as valuable tools in the CMR assessment of non-ischaemic cardiomyopathies.22,23 T1 mapping identifies myocardial oedema secondary to acute myocardial injury.17 Native T1 times reflect both interstitial and myocyte signals, complementing T2 imaging with a high correlation between native T1 and T2 times. Finally, the assessment of T1 values after the administration of intravenous gadolinium (which shortens tissue T1 time) allows the measurement of the ECV, which can be a measure of diffuse fibrosis. We have shown, similar to other studies, that these parameters are associated with the presence of DCM and are correlated with adverse remodelling in this condition.15,16 Additionally, we have also shown, for the first time, that these parameters can provide diagnostic confidence at the 1.5 T field strength, which is more commonly used in routine clinical practice.24

Puntmann et al.15 studied 27 patients with DCM (included in a cohort with 25 HCM patients) and found that native T1 times were longer in DCM patients compared with controls, while postcontrast T1 values were lower and ECV was higher. The authors found that native T1 was the best predictor of cardiomyopathy. In another study by Dass et al.,16 the authors evaluated 18 DCM patients (along with 28 HCM patients) and again found that native T1 values were increased in DCM patients compared with controls and were significantly correlated with global circumferential strain. Similar to the above studies, we also found that native T1 was significantly higher in patients with mild DCM.

ECV measurement using T1 mapping has also been shown to correlate extremely well with myocardial fibrosis measured histopathologically in patients with DCM.25 In this study by aus dem Siepem et al., the authors also showed that non-invasive ECV was even able to differentiate between patients with mild DCM and controls, similar to our study. This again would suggest that the presence of an increased ECV could be useful in risk stratification and management of these patients.

Numerous myocardial adaptations occur in endurance-trained athletes.3,7,8,14 These include an increase in end-diastolic diameter, volume, and mass, leading to a reduction in LVEF.6 These findings were also reflected in our study. LV mass tends to increase in
both endurance- and strength-trained athletes, but also increases in DCM due to the increase in LV size, potentially causing diagnostic difficulty. This has important clinical implications, particularly in the differentiation between the normal cardiac adaptations to exercise and athletes, in whom the changes in myocardial structure and function are actually the early signs of DCM. Current non-invasive techniques have limited ability to differentiate between the two, and detraining is often unattractive and impractical for athletes.

The implications of an incorrect diagnosis—either as athlete’s heart or early DCM, may lead to unnecessary exclusion from sport, or sudden cardiac death (SCD) if undertaking exercise. Additionally, the erroneous labelling of early DCM would lead to long-term treatment incorrectly directed at heart failure. Our finding that native $T_1$ mapping seems to be able to differentiate between athletes and patients with early DCM might provide an extra parameter to support the diagnosis in these cases.

Table 1  Baseline and CMR characteristics

<table>
<thead>
<tr>
<th></th>
<th>Controls ($n = 21$)</th>
<th>Exercisers ($n = 21$)</th>
<th>DCM patients ($n = 16$)</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>$47.9 \pm 16.0$</td>
<td>$45.9 \pm 10.7$</td>
<td>$46.1 \pm 13.6$</td>
<td>0.79</td>
</tr>
<tr>
<td>LV mass (g/m$^2$)</td>
<td>$60.7 \pm 12.7$</td>
<td>$84.3 \pm 28.2^a$</td>
<td>$68.8 \pm 15.5$</td>
<td>0.001</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>$64.5 \pm 4.1$</td>
<td>$51.2 \pm 5.9^a$</td>
<td>$48.1 \pm 4.8^a$</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>LVEDV (mL/m$^2$)</td>
<td>$78.6 \pm 12.4$</td>
<td>$102.2 \pm 36.0^a$</td>
<td>$101.2 \pm 21.2^a$</td>
<td>0.002</td>
</tr>
<tr>
<td>LVESV (mL/m$^2$)</td>
<td>$28.0 \pm 5.9$</td>
<td>$49.5 \pm 16.2^a$</td>
<td>$52.6 \pm 12.2^a$</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>The present of LGE (%)</td>
<td>0 (0)</td>
<td>2 (9.5)</td>
<td>4 (25)$^a$</td>
<td>0.027</td>
</tr>
<tr>
<td>Native $T_1$ (ms)</td>
<td>$952 \pm 31$</td>
<td>$957 \pm 32$</td>
<td>$1017 \pm 42^a$</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>ECV (%)</td>
<td>$26.2 \pm 2.9$</td>
<td>$26.3 \pm 3.6$</td>
<td>$31.2 \pm 4.1^a$</td>
<td>$&lt;0.001$</td>
</tr>
<tr>
<td>$T_2$ (ms)</td>
<td>$52.9 \pm 3.3$</td>
<td>$52.8 \pm 3.2$</td>
<td>$55.9 \pm 4.4^a$</td>
<td>0.024</td>
</tr>
</tbody>
</table>

DCM, dilated cardiomyopathy; LVEF, left ventricular ejection fraction; LGE, late gadolinium enhancement; ECV, extracellular volume; CMR, cardiovascular magnetic resonance; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume.

$^aP < 0.01$ vs. controls.

$^bP < 0.01$ vs. exercisers.

$^cP$ values in bold signify $P < 0.05$ (i.e. statistically significant).

Figure 1  Examples of the protocol for a healthy control (top), a patient with DCM (middle), and an exerciser (bottom). Top: a 52-year-old healthy male control with a normal-sized left ventricle (end-diastolic diameter 5.6 cm) and preserved LVEF (57.8%) ($A$). Septal $T_2$ was 51.6 ms ($B$), septal native $T_1$ 924.9 ms ($C$), and there was no LGE ($D$) or significant diffuse fibrosis (post-contrast $T_1$ 467.1 ms, ECV 27%) ($E$). Middle: a 58-year-old DCM patient with a dilated left ventricle (end-diastolic diameter 6.7 cm) and mildly impaired LVEF (48.4%) ($F$). Septal $T_2$ was 59.8 ms ($G$) and septal native $T_1$ 1017.8 ms ($H$). There was midwall LGE ($I$; arrow) also reflected by the reduced post-contrast $T_1$ and ECV (post-contrast $T_1$ 429.8 ms, ECV 35%; $J$; arrow). Bottom: a 54-year-old male exerciser with a dilated left ventricle (end-diastolic diameter 6.3 cm) and borderline impaired LVEF (53.4%) ($K$). Septal $T_2$ was 50.4 ms ($L$) and septal native $T_1$ 931.4 ms ($M$). There was no LGE ($N$) and similar post-contrast $T_1$ and ECV values to the controls (post-contrast $T_1$ 473.4 ms, ECV 28%; $O$).
The importance of these findings is perhaps their potential use in identification of athletes at risk of sudden death, in whom the first presentation of any cardiac abnormality is that of SCD. Vigorous exertion can act as a substrate for ventricular arrhythmias in people with an underlying abnormal myocardium. One theory for this is that repeated myocardial injury (causing an increase in troponin release) leads to fibrosis and scarring, which may be identified using LGE. These areas of fibrosis lead to an increased susceptibility to ventricular arrhythmias. ECV evaluation using T1 mapping may also identify these areas; however, whether the above is valid also in athletic hearts remains to be established pathologically. Nevertheless, the overwhelming majority of athletes do not have fibrosis that can be seen using LGE or ECV imaging. Native T1 mapping appears to identify other processes that are not purely related to fibrosis, for example myocardial inflammation represented by oedema (also seen using T2 mapping, for which there is a strong correlation in our study). Speculatively, native T1 mapping may also identify early changes in myocardial function and metabolism, although large studies are awaited to understand the mechanisms and their clinical impact.

The finding of increased T2 values in patients with cardiomyopathy has been recognized in other studies and is possibly due to myocardial oedema caused by injury and inflammation related to

---

**Table 2** Univariable and multivariable logistic regression of CMR variables for discrimination of exercisers and DCM patients.

<table>
<thead>
<tr>
<th>CMR variable</th>
<th>Univariable</th>
<th>Multivariable</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>P-value</td>
</tr>
<tr>
<td>Native T1 (ms)</td>
<td>1.05 (1.02–1.08)</td>
<td>0.002</td>
</tr>
<tr>
<td>ECV (%)</td>
<td>1.38 (1.09–1.75)</td>
<td>0.009</td>
</tr>
<tr>
<td>T2 (ms)</td>
<td>1.23 (1.01–1.49)</td>
<td>0.042</td>
</tr>
</tbody>
</table>

ECV, extracellular volume; DCM, dilated cardiomyopathy; CMR, cardiovascular magnetic resonance.
the cardiomyopathic process.17,31,32 Oedema is typically present within 4–6 weeks of a myocardial insult, and is thus reflective of an active or recent process. The reason for its presence in the myocardium of patients with idiopathic DCM is unclear; however, its absence in athletes in our study might perhaps suggest that the physiological adaptation seen in athletes is not related to any persistent and ongoing myocardial damage. T2 values do, however, correlate with T1 and so, the increase in T2 values may also reflect other underlying processes.

We speculate that the similarity in values seen between controls and exercisers is because these pathological processes contributing to the development of irreversible myocardial injury in DCM do not occur in athletes despite the ventricular remodelling caused by exercise. Fibrosis is not a common feature in athletes, and this may be reflected in our finding of ‘normal’ T1, T2, and ECV values in our cohort of exercisers. In a study of older athletes, Mohlenkamp et al.9 reported that only 12% of the athletes had the present of LGE. Nevertheless, LGE does represent a different pattern of fibrosis to T1 and T2 mapping, neither of which have been evaluated in a large cohort of athletes (as yet).

Both native T1 values and ECV are known to increase with age, whereas post-contrast T1 values decrease with age.33 In our study, there were no significant correlations between age and T1 or T2 mapping parameters, although we believe that this is simply due to the small cohort. Despite the fact that there is an age-related increase in T1 values, we have shown that this parameter could potentially still be used as a diagnostic tool. This is particularly important given that older healthy exercisers may still have the present of LGE, meaning that the presence (or absence) of LGE alone might not confirm or refute the diagnosis of DCM.9,34

It is important to note that there was a significant overlap in the T1 and in particular T2 values in all three groups. This is common to the mapping techniques, changes in which reflect a continuous spectrum ranging from normality to pathological, in contrast to LGE which is either present or absent.

Limitations

Our study does have some limitations. First, it is a single-centre study with a relatively small cohort, although to the best of our knowledge, this is the largest cohort studied in this area. Nevertheless, larger studies are required to confirm our findings and identify any potential prognostic benefit. Additionally, due to the small numbers, there is some degree of overlap present between the three groups, which could potentially be resolved with a larger study. A further difficulty is the lack of standardization (as yet) between vendors regarding mapping techniques. Hence, although our results suggest a high level of repeatability, larger multicentre, multivendor studies are still needed to refine the results.

Secondly, the lack of histological correlation in this study is a limitation as this could have provided further information. This does, however, reflect the decreasing use of endomyocardial biopsy in routine clinical practice. Also, biopsy itself is limited by sampling error. Associated with the lack of histological diagnostic confirmation, the patients in this study undoubtedly represent a small fraction of wide spectrum of DCM, such as those with genetic causes. Indeed, it was not possible to rule out post-inflammatory states for example. Thirdly, although measurement of T1 and T2 times in the septum is accepted in the current literature as being an adequate surrogate for the whole myocardium, both sequences still need to be optimized to allow accurate quantification in the lateral wall and apex without artefact. We also only examined males as T1 parameters show gender-specific variation.33,34 This study would need to be repeated in order to validate the findings in females. We only carried out post-contrast T1 imaging at one time point, 10 min after gadolinium administration. The optimal time for post-contrast T1 mapping has yet to be validated, although 10 min has been used in previous studies for determination of ECV.15,36 To maintain consistency, we adhered to imaging at 10 min for all patients in our protocol.

Conclusions

Both T1- and T2-mapping parameters could potentially be used to predict the presence of mild DCM. T1 mapping in particular could potentially have the ability to differentiate between patients with early non-ischaemic DCM and middle-aged exercisers.

Conflict of interest: None declared.

References

1. McMurray JJ, Adamopoulos S, Anker SD, Auricchio A, Bohm M, Dickstein K et al. ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: the Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC. Eur Heart J 2012;33:1787–1847.


