Table 1. CMR parameters (mean ± SD)

Parameter (unit)	Controls	Excluded Myocarditis	Suspected Myocarditis
GLS (abs %)#*†	16±3	14±4	11±4
LV EF (%)#*†	67±7	58±13	44±17
LV EDV (ml/m^2)	70±12	78±24	93±37
LV ESV (ml/m^2)#*	23±7	35±21	56±36
LV Mass (g/m^2)	61±13	62±19	78±28
GCS (abs %)#*†	19±2	17±5	13±5
GRS (%)#*†	34±7	28±10	20±9
GLSR (1/s)*	0.9±0.27	0.7±0.22	0.6±0.24
GCSR (1/s)*†	1.0±0.24	1.0±0.32	0.6±0.23
GRSR(abs 1/s)*†	1.9±0.56	1.8±0.73	1.0±0.49
ECV (%)*†	29±3	30±4	34±4
T1_3T (ms)*	1226±59	1268±50	1301±40
T1_1.5T (ms)*†	975±44	1017±77	1143±74
T2_3T (ms)#*	39±3	42±4	43±2
T2_1.5T (ms)#*†	45±2	50±3	54±3

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Kiosk 8R-TA-06

Mapping the Atlhete's Heart: T1 and T2 Insights into Cardiac Remodeling

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Background: Cardiac magnetic resonance (CMR) T1- and T2-mapping techniques allow for precise quantification of myocardial tissue characteristics. A more comprehensive understanding of normal T1 and T2 mapping values in athletes and identifying factors influencing these parameters is crucial for distinguishing between physiological adaptation and cardiac conditions. The purpose of this study was to assess T1 and T2 mapping values in both healthy athletes and less active individuals and to investigate their relationship with clinical and CMR parameters.

Methods: We enrolled healthy athletes (weekly training hours >7) and less active age- and sex-matched control individuals (weekly training hours ≤6) to undergo CMR examinations. We determined ventricular volumes, masses, and function and performed T1- and T2-mapping on non-contrast short-axis images.

Results: We conducted 224 CMR examinations involving 123 athletes (76 males, 23±5 years, weekly training hours: 21±7) and 101 controls (64 males, 26±3 years, weekly training hours: 3±2). Comparing male athletes and less active individuals, we observed marked MR signs of sports-related cardiac remodeling, including increased end-diastolic volume and mass index (Left ventricular end-diastolic volume index (LVEDVi): 120±16 vs 97±12 ml/m^2, p<0.001; left ventricular mass index (LVMi): 70±14 vs 53±9 g/m^2, p<0.001). Examining the female athletes, we also noted significant sports adaptations compared to the control group (LVEDVi: 103±12 vs 86±11 ml/m2, p<0.001; LVMi: 54±9 vs 39±5 g/m2, p<0.001). The athletes' T1-mapping values were lower compared to the control group in both sexes (males: 939±25 vs 961±20 p<0.001, females: 966±21 vs 985±17, p<0.001). T1 values negatively correlated with training hours (r=-0.362, p<0.001), LVEDVi (r=-0.411, p<0.001) and LVMi (r=-0.546, p<0.001). We found a significant association between T1 values and training hours across different models. In the raw model, which considered the unadjusted association between T1 values and training hours, we observed that every additional hour of training was associated with a decrease of 0.946 ms in T1 values (p<0.001). Upon adjusting for age, sex, and heart rate (HR) in model 2, the association remained significant, albeit slightly attenuated, with each additional training hour corresponding to a T1 reduction of 0.825 ms (p<0.001). Further adjustments for LVMi in model 3 continued to exhibit a significant association, with a decrease of 0.565 ms in T1 values for each additional training hour (p=0.002).

Conclusion: Our research emphasises a robust association between increased training hours and decreased T1 values, and this relationship persisted independent of the influences of age, sex, HR, and LVMi.

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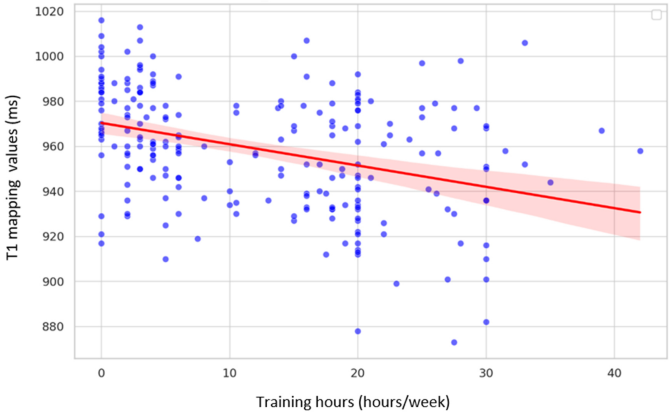

Regression analyses: prediction of T1 mapping values

Model 1					
Variable	Coefficient	Standard Error	t-value	p-value	95% CI
Training hours	-0.946	0.164	-5.774	<0.001	(-1.268, -0.632)
Model 2					
Variable	Coefficient	Standard Error	t-value	p-value	95% CI
Training hours	-0.825	0.158	-5.230	<0.001	(1.136, -0.514)
Age	0.663	0.336	1.973	0.050	(0.001, 1.325)
Sex	28.437	3.166	8.981	<0.001	(22.197, 34.678)
Heart rate	0.168	0.122	1.371	0.171	(-0.073, 0.409)
Model 3					
Variable	Coefficient	Standard Error	t-value	p-value	95% CI
Training hours	-0.565	0.179	-3.158	0.002	(-0.917, -0.212)
Age	0.516	0.334	1.544	0.124	(-0.143, 1.174)
Sex	21.943	3.819	5.746	<0.001	(14.416, 29.469)
Heart rate	0.099	0.123	0.809	0.419	(-0.142, 0.341)
LVMi	-0.393	0.134	-2.936	0.004	(-0.658, -0.129)

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Multiparametric CMR Can Detect Cardiac Amyloidosis in a Real-world Cohort of Undifferentiated Concentric Left Ventricular Hypertrophy

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Background: Hypothesis: Multiparametric CMR can detect cardiac amyloidosis (CA) in a real-world cohort of undifferentiated concentric left ventricular hypertrophy (LVH).

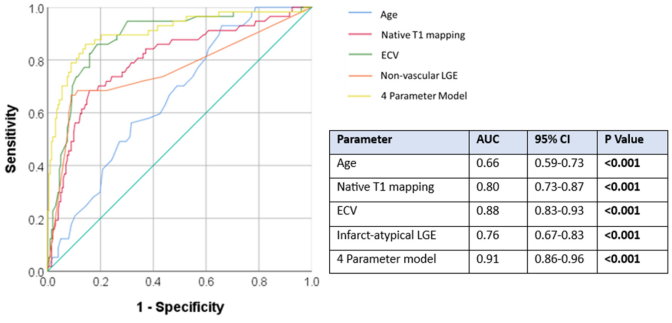
Methods: A retrospective patient search was performed for patients ≥18 years of age who received 1.5T CMR from January 2018 to August 2022 to evaluate for myocardial infiltration in the setting of known concentric LVH at a tertiary medical center. Clinical records were reviewed for positive diagnosis or exclusion of CA. A positive diagnosis included: endomyocardial biopsy positive, or PYP scan positive with negative serum immunofixation for monoclonal band, or, clinical suspicion of CA with a diagnosis of systemic AL amyloidosis with an amyloid positive biopsy at a surrogate site. CA was excluded if: endomyocardial biopsy negative, PYP scan negative with negative serum immunofixation, or, presence of clear alternative clinical diagnosis (e.g. hypertrophic cardiomyopathy).

CMR were post-processed using commercially available software (CVI42, Circle Cardiovascular Imaging) to evaluate ventricular volumes and function, native T1 and T2 mapping, and extracellular volume (ECV). Late gadolinium enhancement (LGE) was qualitatively evaluated by an experienced observer and each segment categorized into vascular or non-vascular LGE using the American Heart Association 16 segment model.

Group comparisons were made using one-way ANOVA (parametric) or Kruskal-Wallis (non-parametric) tests. Receiver operator characteristic (ROC) analysis with area under curve (AUC) values were generated for both individual and combined parameters using binary logistic regression (IBM SPSS Statistics V26.0). $P < 0.05$ was considered significant.

Results: CMR were performed in 278 patients for myocardial infiltration evaluation in the setting of known concentric LVH (mean age 63.2 ± 14.9, 46% female). Diagnostic groups were determined as follows: CA positive (n = 60), CA negative (n = 100) and CA unknown (n = 118). CA positive patients, when compared to both CA negative and CA unknown groups, respectively, demonstrated significantly higher age (69.9 ± 10.3 vs 59.7 ± 14.2 and 62.9 ± 16.4 years), native T1 (1122.4 ± 64.6 vs 1056.8 ± 69.7 and 1051.4 ± 54.0 ms), ECV (46.4 ± 11.5 vs 32.1 ± 7.2 and 32.1 ± 7.6 %) and number of segments with non-vascular LGE (10.2 ± 7.3 vs 2.7 ± 4.7 and 2.0 ± 4.3). ROC AUC values were calculated for native T1 (0.80), ECV (0.88), and number of non-vascular LGE segments (0.76). A 4 parameter model including age, native T1, ECV, number of segments with non-vascular LGE demonstrated an AUC of 0.91 for detection of CA.

Conclusion: Multiparametric CMR can differentiate CA positive patients from CA negative and undifferentiated LVH patients with excellent discrimination in a real-world tertiary center population. These findings demonstrate that CMR has significant diagnostic potential for CA.



Parameter	AUC	95% CI	P Value
Age	0.66	0.59-0.73	**<0.001**
Native T1 mapping	0.80	0.73-0.87	**<0.001**
ECV	0.88	0.83-0.93	**<0.001**
Infarct-atypical LGE	0.76	0.67-0.83	**<0.001**
4 Parameter model	0.91	0.86-0.96	**<0.001**

Multiparametric CMR evaluation of patients that are CA positive vs CA negative and CA unknown status.

Multiparametric CMR	CA positive	CA negative	CA unknown	Total	P Value
LV Volumes and Function					
LV ejection fraction (%)	47.5 ± 13.3	45.8 ± 16.4	50.4 ± 13.3	48.1 ± 14.6	0.12
LV end diastolic volume (ml)	149.7 ± 41.3	175.6 ± 79.7	165.8 ± 65.7	165.8 ± 67.4	0.20
LV end systolic volume (ml)	79.7 ± 35.0	101.8 ± 76.7	86.3 ± 54.9	90.4 ± 61.0	0.23
LV stroke volume (ml)	69.9 ± 25.9	73.8 ± 29.4	79.5 ± 28.5	75.4 ± 28.5	0.07
LV Mass (g)	158.0 ± 51.2	160.7 ± 64.0	142.5 ± 53.6	152.4 ± 57.5	0.69
LV Tissue Characterization					
Native T1 mapping (ms)	1122.4 ± 64.6	1056.8 ± 69.7	1051.4 ± 54.0	1068.7 ± 68.3	**<0.001**[a,b]
Post-Gd T1 mapping (ms)	364.6 ± 101.6	427.7 ± 81.4	423.7 ± 86.4	412.2 ± 91.4	**<0.001**[a,b]
ECV (%)	46.4 ± 11.5	32.1 ± 7.2	32.1 ± 7.6	35.3 ± 10.3	**<0.001**[a,b]
T2 mapping (ms)	53.0 ± 7.4	50.8 ± 5.1	50.7 ± 3.9	51.3 ± 5.4	0.05
Non-vascular LGE (segments, n)	10.2 ± 7.3	2.7 ± 4.7	2.2 ± 4.3	4.1 ± 6.1	**<0.001**[a,b]
Vascular LGE (segments, n)	0.4 ± 1.3	0.6 ± 1.6	0.5 ± 1.4	0.5 ± 1.4	0.72