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Using T1 and T2 mapping and late gadolinium enhancement to accurately diagnose hypertrophic cardiomyopathy

Sinem Aydin[a,*], Mehmet Kanyilmaz[a], Arda Guler[a], Gamze Babur Guler[a], Aysel Turkvatan Cansever[a]

[a]*Istanbul Mehmet Akif Ersoy Cardiothoracic and Vascular Surgery Research Hospital, Department of Radiology, Istanbul, Türkiye*

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Abstract

Aim: Hypertrophic cardiomyopathy (HCM) is a genetic disorder characterized by thickening of the heart muscle and may have serious consequences. Diversity in the distribution and degree of hypertrophy has led to the classification of HCM types. Cardiac MR (CMR) imaging is a non-invasive tool that maintains accurate and detailed data about cardiac functions and structure. The aim of the study was to observe and compare the CMR characteristics of the three HCM morphologic types (asymmetric, concentric, and apical).

Materials and Methods: 56 patients (female/male: 29/58, mean age 46.31±15.12, body surface area (BSA) 1.88±0.19 m^2) who were referred for HCM diagnosis or follow-up between January 2021 and January 2023 were included in this single-center retrospective study. The imaging procedure was conducted using a 1.5 T device (Aera, Siemens Medical Systems, Erlangen, Germany). Cardiac volume and functions were determined quantitatively. The location and amount of late gadolinium enhancement (LGE) were evaluated qualitatively. The statistical analyses were performed using SPSS version 25.0. A p value under 0.05 was considered statistically significant.

Results: Late gadolinium enhancement revealed myocardial fibrosis in apical HCM (ApHCM), with a higher prevalence than in asymmetric and concentric HCM. Apical HCM and asymmetric HCM patients widely presented midmyocardial fibrosis, whereas most concentric HCM patients had patchy involvement. The mean native myocardial T1 values for individuals with concentric HCM were found to be higher compared to those with asymmetric and ApHCM and the healthy participants.

Conclusion: Cardiac MR imaging enables the multiparametric evaluation of HCM through the utilization of native T1 and T2 mapping techniques, as well as LGE. Both T1 and T2 values have the potential to be used in conjunction with LGE to identify regions of myocardial fibrosis.

Introduction

Hypertrophic cardiomyopathy (HCM) is a common genetic cardiac disease characterized by thickening of the heart muscle in the absence of any other potential causative factors [1, 2]. The disease affects approximately one in 500 people worldwide [3] and is a leading cause of sudden cardiac death (SCD) [4], atrial fibrilation [5], stroke [6], and heart failure (HF) [7].

The degree and pattern of hypertrophy can vary extensively in HCM cases, leading to a diverse range of clinical manifestations and outcomes [8]. Asymmetric (classical) hypertrophy, concentric (symmetric) hypertrophy, apical hypertrophy, and mixed hypertrophy are the four phenotypes of HCM. The most prevalent form is asymmetric hypertrophy, which is characterized by asymmetric septal hypertrophy that can cause left ventricular (LV) outflow tract obstruction [9]. Typically, this particular form of HCM is linked to a higher likelihood of sudden cardiac death (SCD). Concentric hypertrophy, also known as symmetric hypertrophy, is characterized by a uniform thickening of the entire LV wall and is less common than the asymmetric subtype. The predominant morphological feature of apical HCM (ApHCM) is myocardial hypertrophy confined to the apex of the LV, resulting in a spadelike shape of the LV chamber [9].

Cardiac MR (CMR) imaging is a non-invasive tool that provides accurate and detailed information about cardiac functions and structure. Cardiac MR not only allows the measurement of parameters related to ventricular function, such as ejection fraction and stroke volume, but also offers comprehensive tissue characterization. It can provide

*Corresponding author:
Email address: sinem.rad@gmail.com (Sinem Aydin)

detailed information about the composition and viability of myocardial tissue with the use of native T1 and T2 maps and late gadolinium enhancement (LGE), allowing for the detection and characterization of various pathologies. Cardiac MR is also useful in distinguishing hypertrophic cardiomyopathy (HCM) from other conditions that might cause left ventricular hypertrophy.

The objective of this study was to observe and compare the CMR characteristics of the three HCM morphologic types (asymmetric, concentric, and apical). Additionally, we sought to identify any discernible imaging patterns that could differentiate between these phenotypes.

Materials and Methods

This retrospective, single-center study received approval from the ethics committee of our institution (Istanbul Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital Clinical Research Ethics Committee, Decision no: 2023.08-78). The patients gave oral and written consent.

Patients

The calculation of the sample size was conducted using G Power 3.1.9.7 (Franz Faul, Germany) according to the data published in the article by Luca Arcari et al. [10], where the effect size was calculated as d = 0.408. To conduct statistical power with $\alpha = 0.05$ and power = 0.85, the analysis revealed that in order to conduct the research, a minimum sample size of 80 was necessary.

The patients were among 187 patients who were referred to our CMR unit between January 2021 and January 2023 for functional and volume analysis for either diagnostic work-up or follow-up. 56 patients who met the criteria for hypertrophied cardiomyopathy were included in the study. Hypertrophic cardiomyopathy is defined as a nondilated, hypertrophied left ventricle in the absence of another cardiac or systemic disease that is capable of inducing LV hypertrophy [11]. Asymmetric (septal) hypertrophy was characterized as end-diastolic interventricular septum thickness $\geq$ 15 mm. Apical hypertrophy refers to left ventricular hypertrophy that is predominantly localized to the apex of the ventricle (as to the 17-segment criteria of the American Society of Echocardiography, only the top 4 segments and the topmost portion are considered apical) and characterized by a minimum apical wall thickness of 15 mm and an end-diastole maximal apical to posterior wall thickness ratio of 1.5 [12]. The definition of concentric hypertrophy was LV that was diffusely hypertrophied in almost all segments.

31 patients who had a clinical history of atypical chest pain and normal blood samples, normal echocardiography, and CMR imaging findings were recruited for baseline value comparisons.

CMR imaging and analysis

A standardized CMR protocol was implemented to routinely assess cardiac volumes and functions in all subjects, T1 and T2 mapping, myocardial perfusion, and LGE, as well, using a 1.5 T system (Magnetom Aera; Siemens Medical Solutions, Erlangen, Germany) with an 18-channel body coil. For cardiac volume and functional analysis, steady-state free precession breath-hold cines in sequential short-axis slices (slice thickness: 8 mm, gap: 2 mm) from the atrioventricular ring to the LV apex were performed. The imaging parameters: repetition time/echo time, 32 msec/1.01 msec; flip angle, 51°; voxel size 1.85x 1.85 x 8 mm; matrix size, 440 × 320; pixel size, 1.85 mm x 8 mm; and temporal resolution, 10 heart beats depending on the patient. The LGE sequence was conducted utilizing rapid low-angle single-shot T1-weighted imaging employing the phase-sensitive inversion-recovery approach, 10 to 15 minutes after injection of 1.5 cc per 10 kg for patients > 60 kg and 2 cc per 10 kg for patients< 60 kg of gadobutrol (Gadonans, Biem, Türkiye) using an automated injector. Late gadolinium enhancement images were acquired in the LV short-axis orientation as well as in two-, three-, and four-chamber views corresponding to the slice positions of MRI cines. Inversion times were adjusted to null normal myocardium (usually 210–260 ms) using a mid-diastolic inversion prepared a 2-dimensional Trufi sequence (TE/TR/flip-angle 1.06 msec/40.16 msec/43°, acquired voxel size 1.06x1.06x8mm). Field of view and image matrix were 400 × 300 mm and 72 × 192, respectively.

T1 maps were obtained using modified Look-Locker imaging. A short axis slice was prescribed at the midventricular level using ECG-gating and motion correction techniques, with the following parameters: slice thickness: 8 mm; TE: 1.08 ms; minimum TI: 209 ms with 80 ms increments according to a 5(3)3 scheme; matrix size: 256 (phase res.: 66%); FOV (rectangular): 400 mm (phase FOV: 85%); bandwidth: 1085 Hz/px; flip angle: 35°. T2 mapping was based on a True FISP application with multiple T2 preparations and recovery periods. An ECG-gated, motion-corrected, short-axis slice was prescribed at the midventricular level with the following parameters: slice thickness: 8 mm; TE: 1.04 ms; No. of T2 preps: 3 (0, 25, 55 ms); matrix size: 192 (phase res.: 76%); FOV (rectangular): 400 mm (phase FOV: 80.2%); bandwidth: 1184 Hz/px; flip angle: 70°. These sequences were obtained before the contrast was given.

The myocardial native T1 and T2 values were quantified by applying the ConSept method, which involved placing a region of interest (ROI) within the septal myocardium [13]. Areas of LGE were excluded from the ROI in order to obtain accurate myocardial values. Care was taken to prevent the interference of signals from the blood pool due to contamination (Figure 1).

Cardiac volume and function were determined using semi-automatic segmentation in the software (Argus, Siemens Healthcare, Erlangen, Germany). Left ventricle ejection fraction (LV EF), LV end-systolic volume (LV ESV), and LV end-diastolic volume (LV EDV) were quantified using conventional volumetric methods. On cine images, the endocardium and epicardium of the LV were manually drawn in order to delineate the myocardium. The American Heart Association's 17-segment model was utilized to assess the LV chamber [14]. Maximal septal thickness, maximal LV posterior wall thickness, and maximal apical thickness were defined as the greatest dimensions at the mentioned site at the end-diastolic phase of the short axis. Late gadolinium enhancement was first described as

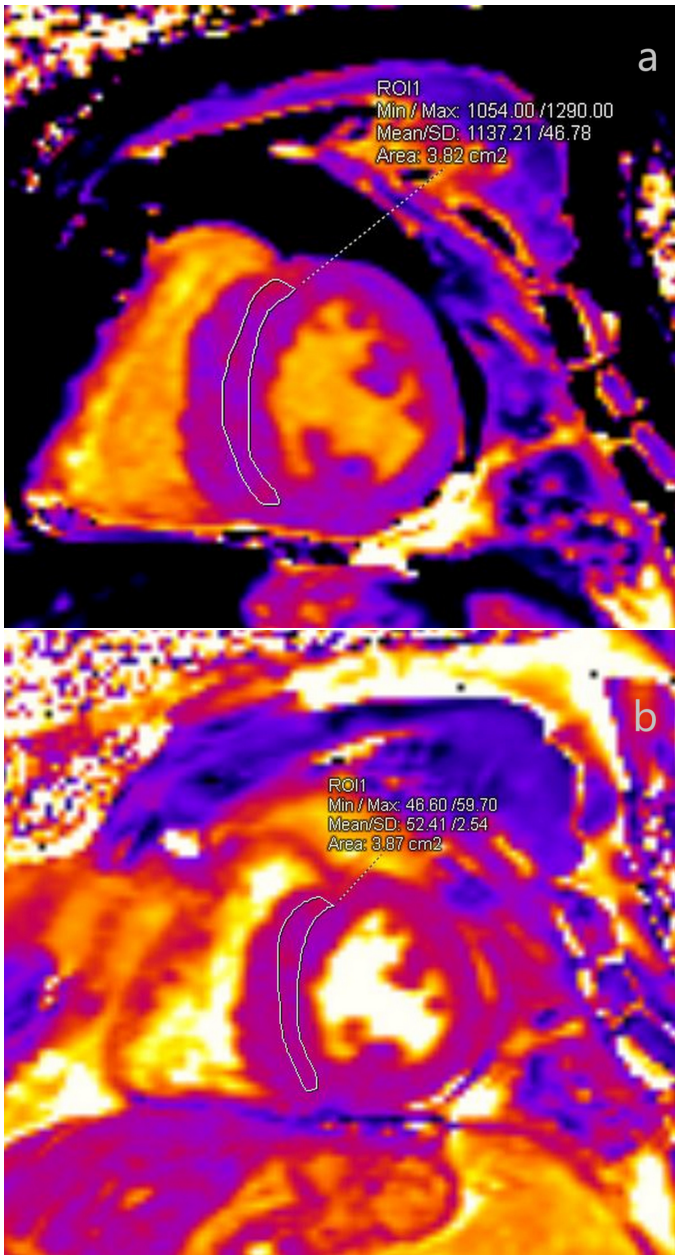

Figure 1. Sample native T1 and T2 images demonstrating ROI placement in the midventricular short axis view.

present or absent. If LGE was found, the patterns were categorized as subendocardial, midwall, or subepicardial, depending on their location. Late gadolinium enhancement was quantitatively defined by assessing the intensity of the myocardial postcontrast signal six standard deviations above the reference region of the remote myocardium within the same slice, in addition to visual evaluation [15, 16]. The total mass of LGE was determined by summing the regions of LGE, whereas the LGE fraction was quantified as a ratio relative to the LV myocardium.

Statistical analysis

Statistical analysis was conducted using SPSS version 25.0 (SPSS, Chicago, Ill.). Quantitative data values are represented as means ± standard deviation, medians, and interquartile ranges, as appropriate. Qualitative data were presented as numbers and percentages. The distribution of the variables was examined with histogram plots and the Kolmogorov-Smirnov test. Quantitative data values are represented as means ± standard deviation, medians, and interquartile ranges, as appropriate. Qualitative data were presented as numbers and percentages. We employed post-hoc analysis to explore and identify specific group differences in the study population. In order to compare the study groups' morphological and functional measurements, the Chi squared (χ^2) test was used along with the Fisher's exact test and linear-by-linear association. The comparisons were performed by the Mann-Whitney U test for non-normally distributed variables. The correlation between T1 and T2 values, LV functional measurements, and LGE extent has been examined in total and separately in the study groups. Results were considered to indicate statistical significance if p $\leq$.05.

Results

Demographic and clinical variables and CMR data regarding LV volume and function are given in Table 1. The observed distribution of phenotypes for hypertrophic cardiomyopathy among the study cohort was as follows: 23 apical (38%), 26 asymmetrical (30%), and 7 concentric (0.08%).

There were no significant differences observed in the LV EDV and LV ESV, as well as EF, in all types of HCM (p<.05). Myocardial fibrosis was found in 22 of 23 patients (95.65%) with ApHCM, which is a higher rate than in asymmetric HCM (88.46%) and concentric HCM (71.43%). However, the proportion of LGE also did not show a significant difference between the three groups (7.64±6.79% in ApHCM, 8.5±8.52% in asymmetric HCM, and 3.67±4.5% in concentric HCM) (p > .05). Extensive fibrosis, which was defined as more than 25 mL/m^2 of LGE, was only seen in one ApHCM and two asymmetric HCM patients. ApHCM (63.64%) and asymmetric HCM (82.61%) patients widely presented midmyocardial fibrosis, while most concentric HCM patients (80%) had patchy involvement.

The maximum septal thickness was greater in asymmet-

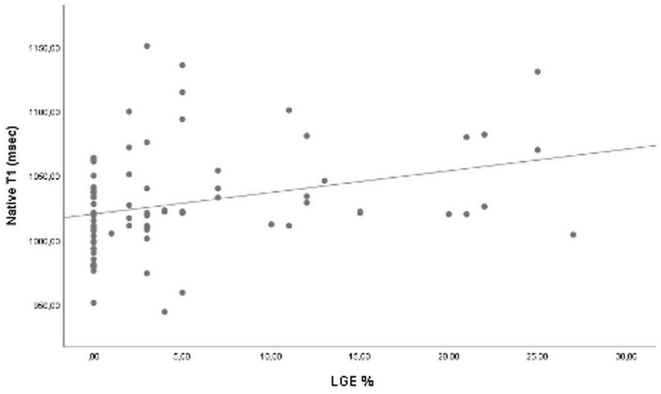

Figure 2. The correlation between native T1 and late gadolinium enhancement (LGE) extent. Hypertrophic cardiomyopathy (HCM) subjects showed a positive correlation between native T1 and the total LGE volume (r .336; p .002).

Table 1. Patient characteristics, morphological, and functional measurements based on CMR imaging.

	ApiHCM (n=23)	Asymmetric HCM (n=26)	Concentric HCM (n=7)	Control (n=31)	p
Age	57.65±10.35	47.15±11.35	44±13.29	37.71±16.04	<.001
Gender, Male, n (%)	15 (65.22)	15 (57.69)	7 (100.00)	21 (67.74)	.214
BSA, m^2	1.9±0.2	1.86±0.17	1.96±0.13	1.85±0.21	.468
LGE, Present, n (%)*	1 (4.35)	3 (11.54)	2 (28.57)	31 (100.00)	<.001
LGE, Location					
1, n (%)*	14 (63.64)	19 (82.61)	0 (.00)	0	
2, n (%)*	5 (22.73)	2 (8.70)	1 (20.00)	0	.001
3, n (%)*	3 (13.64)	2 (8.70)	4 (80.00)	0	
LV EDV index, mL/m^2	63.87±9.15	73.35±19.19	86.14±17.48	77.77±14.04	.002
LV ESV index, mL/m^2	18.04±5.14	22.88±9.77	30.86±15.51	30.03±7.84	.000
LV EF	70.87±6.47	69±6.37	64±10.71	61.55±5.05	.000
LGE extent (%)	7.64±6.79	8.5±8.52	3.67±4.5	0±0	.000
Septal native T2 (ms)	50.91±2.31	50.16±1.84	52.71±1.98	48.96±2.81	.002
Septal native T1 (ms)	1024±38.04	1040.46±35.67	1072.86±66.21	1014.45±30.17	.029
MWTSeptum	13.91±4.5	17.44±4.5	17.06±3.9	7.45±1.62	.000
MWTPosterior wall	7.18±2.51	7.11±2.66	11.63±2.79	4.61±1.31	.000
MWTApex	18.12±4.38	5.62±2.21	10.03±4.27	3.71±1.0	.000

HCM, hypertrophic cardiomyopathy; ApiHCM, apical hypertrophic cardiomyopathy; BSA, body surface area; LV EDV, left ventricle end-diastolic volume; LV ESV, left ventricle end-systolic volume; LV EF, left ventricle ejection fraction; LGE, late gadolinium enhancement; MWTSeptum, maximal septal thickness, MWTPosterior Wall, maximal posterior wall thickness, MWTApex, , maximal apical thickness. *Data are medians, with interquartile range in parentheses.

Table 2. Relation between LV EF and septal native T1 and T2 measures.

		Total		Asymmetric HCM	
		LV EF	LGE %	LV EF	LGE %
Septal native T1	r	-.041	.336	-.546	.370
	p	.707	.002	.004	.063
Septal native T2	r	.159	.238	-.398	.136
	p	.141	.028	.044	.509

The Spearman's correlation test; LV EF, left ventricle ejection fraction; LGE, late gadolinium enhancement.

ric and concentric HCM than that observed in ApHCM [17.44±4.5 mm and 17.06±3.9 mm vs. 13.91±4.5 mm (p = .003 and p = .047)]. The concentric HCM phenotype exhibited a higher maximum posterior wall thickness compared to other phenotypes, while the ApHCM phenotype displayed a greater maximum apical thickness compared to other phenotypes.

The mean native myocardial T1 values for individuals with concentric HCM (1072.86±66.21) were found to be higher compared to those with asymmetric HCM (1024±38.04) and ApHCM (1040.46±35.67) and the healthy participants (1029.45±40.53) (p = .029).

A notable inverse correlation was seen between tissue features, namely the mean native myocardial T1 and T2 values, and LV EF in cases of asymmetric HCM (r -.546; p .004 for T1 and r -.398; p .044 for T2) (Table 2). Within the entire cohort, the total LGE volume was positively correlated with myocardial native T1 and T2 values (r.336; p .002 for T1 and r.238; p .028 for T2) (Figure 2).

Discussion

Despite the fact that HCM is a genetic disease, an initial morphological classification is suggested for evaluating patients, as the disease is diagnosed when the heart muscle is determined to be hypertrophied [11]. Cardiac MR imaging is immensely valuable when evaluating individuals suffering from HCM, providing valuable insights into both morphological and cellular aspects. In comparison to alternative imaging modalities, CMR offers distinct advantages through the utilization of techniques such as LGE, as well as myocardial native T1 and T2 assessments. This study aimed to conduct a comparative analysis of the morphological and functional CMR features within the three most prevalent forms of HCM.

The results of our study demonstrated three important findings concerning the MR characteristics of specific HCM phenotypes. First, ApHCM patients revealed a higher prevalence of myocardial LGE than other types of HCM. Second, prolonged native myocardial T1 and T2 values were related to a decrease in LV EF in asymmetric HCM. And third, a significant correlation existed between the total volume of LGE and the native T1 and T2 values of the myocardium in all HCM patients.

Apical HCM is a subtype of HCM distinguished by thickened LV apical walls and marked apical obliteration. Apical subendocardial LGE and apical aneurysm/thrombus formation are additional distinguishing imaging features of this subtype, prompting researchers to examine whether its prognosis differs from that of other subtypes. In our study, apical HCM patients revealed a higher prevalence of myocardial LGE, which serves as an indicator of myocardial fibrosis, compared to individuals with other forms of HCM. There have been reports indicating a correlation

between myocardial fibrosis and elevated cardiac mortality rates, as well as its association with arrhythmia and the subsequent occurrence of SDH in individuals with HCM [17–21]. A scholarly discourse exists within the literature regarding the prognostic implications of ApHCM. Kim et al. showed that ApHCM is associated with more favorable clinical outcomes in patients than other types of HCM. Nevertheless, our findings are consistent with previous research that has linked ApHCM to increased rates of significant morbidity and cardiovascular mortality, including ventricular and atrial arrhythmias, SCD, HF, and stroke [12, 14, 21, 27].

MR native T1 and T2 mapping are quantitative methods that reflect tissue features in various myocardial pathologies. Technically, the generation of T1 maps involves the acquisition of several images with varying T1 weightings, followed by the use of a fitting process to determine the signal intensities of these images based on the T1 relaxation equation [28]. Elevated myocardial native T1 can be attributed to the existence of diffuse myocardial fibrosis, edema resulting from acute myocardial infarction or myocarditis, replacement fibrosis, and myocardial inflammation [28, 29]. Conversely, myocardial iron load and Fabry's disease are associated with a reduction in myocardial native T1 values [30]. T2 mapping is an additional parametric technique employed to distinguish between normal myocardial tissue and pathological abnormalities. Although T2 mapping techniques have not gained as much attention as T1 mapping, concerning cardiac failure, an expanding body of evidence indicates that T2 mapping has the potential to serve as a valuable clinical tool for the noninvasive evaluation of myocardial inflammation [31, 32]. In addition, recent research shows that T2 mapping reveals global increased values in acute myocarditis and sarcoidosis and in involved segments in acute myocardial infarctus [33]. The utilization of T2 mapping is useful in identifying acute rejection in individuals who have received heart transplantation [34, 35]. In our study, we observed an inverse relation between prolonged native myocardial T1 and T2 values and left ventricular ejection fraction in cases of asymmetric HCM. Cao et al. looked at how myocardial extracellular volume (ECV) measured by T1 mapping was linked to LV systolic strain in people with type 2 diabetes [36]. The study found that increased myocardial ECV, as measured by T1 mapping, was associated with impaired left ventricular systolic strain, indicating reduced myocardial function. Although this study did not directly measure LV EF, it suggests that, in line with our study, increased myocardial fibrosis or edema, indicated by prolonged native myocardial T1, may be associated with impaired LV function. Further research is needed to establish a direct link between these parameters.

The findings of our investigation demonstrate a noteworthy association between the overall quantity of LGE, which functions as an indirect marker of cardiac fibrosis, and the native T1 and T2 values of the myocardium in all patients diagnosed with HCM. While LGE imaging specifically highlights areas of increased ECV associated with fibrosis, native T1 mapping captures a broader spectrum of tissue changes, including intracellular and extracellular compartment alterations. The expanded scope of native T1 mapping has the potential to identify fibrosis in the myocardial tissue that may not be discernible by LGE [37]. On the other hand, a meta-analysis by Snel et al. suggested that there were insufficient studies on the effectiveness of T2 mapping in hypertrophic cardiomyopathy [38]. Our findings may contribute to an existing gap in the literature. In patients where contrast material usage is contrindicated, T1 and T2 mapping may help management and risk stratification of HCM patients.

There are some limitations of our study. First, the enrollment of a relatively small sample size at a single center restricted the ability to compare the effectiveness of LGE and T1 mapping in accurately demonstrating the extent of fibrosis. The second limitation was the retrospective design. Additional prospective studies involving a significant number of patients are necessary to further confirm the validity of our data and implement them in routine clinical practice.

Conclusion

Cardiac MR imaging enables the multiparametric evaluation of hypertrophic cardiomyopathy through the utilization of native T1 and T2 mapping techniques, as well as LGE. The utilization of native T1 and T2 values has the potential to yield valuable insights into the cellular and structural aspects of cardiac involvement. Both T1 and T2 values have the potential to be used in conjunction with late gadolinium enhancement to determine regions of myocardial fibrosis. Future studies may prioritize the evaluation of the correlation between myocardial T1 and T2 values and ventricular functions across distinct HCM patterns across larger cohorts.

Ethical approval

Ethical approval was received for this study from the Clinical Research Ethics Committee of Istanbul Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital (Decision no: 2023.08-78).

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