ORIGINAL ARTICLE

The role and significance of cardiac magnetic resonance in hypertrophic cardiomyopathy

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Summary

Introduction: Hypertrophic cardiomyopathy (HCM) is an autosomal dominant disorder of cardiomyocytes that leads to myocardial thickening. The aims of this study were to diagnose HCM in patients with hypertrophic left ventricle walls, to evaluate myocardial tissue, and to assess the degree of myocardial fibrosis using cardiac magnetic resonance (CMR).

Material and Methods: The study included 51 patients diagnosed with HCM by CMR (27 males, 24 females) and was carried out in the University Clinical Centre of Serbia, Center of Radiology. All collected patient data was obtained from official medical documentation for this retrospective observational study. CMR confirmed HCM in 51 patients based on a standard imaging protocol performed on a 1.5T Siemens scanner, with the contrast agent Gadolinium. Regarding *Late Gadolinium Enhancement* (LGE) distribution, CMR enables the detection of focal fibrosis and helps to differentiate the etiology of the hypertrophic myocardium.

Results: Left ventricular outflow tract obstruction was observed in 9 (17.6%) patients. Asymmetric HCM was noted in 41 (80.4%) patients. LGE presence was detected in 39 (76.5%) patients, most notably in the septal region (62.7%). The degree of fibrosis was estimated at a median of 6% of the left ventricular mass and a median of 11 grams of fibrosis per gram of left ventricular mass.

Conclusion: CMR as a non-invasive method represents the gold standard for myocardial tissue characterization. The detection of myocardial fibrosis, a major trigger for the development of malignant arrhythmias, positions CMR as a risk stratification method in HCM patients.

Keywords: hypertrophic cardiomyopathy, cardiac magnetic resonance, late gadolinium enhancement, degree of fibrosis, arrhythmias

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INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is an autosomal dominant disease of cardiomyocytes caused by mutations in sarcomere protein genes, which encode components of cardiac contraction and it is characterized by the absence of other diseases that could lead to myocardial thickening (1–5). It represents the most common adaptive or compensatory response protecting the heart's cardiac output (CO), resulting from the influence of physiological or pathological stimuli that can lead to heart failure (6).

In adults, HCM is defined as a left ventricular (LV) wall thickness greater than 15 mm with a ratio of septal/posterior wall thickness greater than 1.3 in normotensive patients or greater than 1.5 in those with arterial hypertension. While myocardial thickening can affect any part of the ventricle, the interventricular septum is the most commonly involved part (3).

There is a wide spectrum of phenotypic expressions of HCM, for which only cardiac magnetic resonance imaging (CMR) can provide an accurate diagnosis. Hypertrophy in HCM can be asymmetric, affecting the interventricular septum, which is the most common form of the disease (60-70%). Symmetric or concentric myocardial hypertrophy can also occur approximately in about 40% pts while the apical form of HCM is the rarest (1). Regarding the degree of left ventricular outflow tract (LVOT) obstruction, HCM is classified into obstructive and non-obstructive cardiomyopathy (2).

The advantage and accuracy of CMR is attributed to the free choice of imaging planes, the wide range of imaging techniques and the absence of harmful ionizing radiation (7). CMR as a gold standard for tissue characterization of the myocardium allows a visualization of focal or diffuse changes in myocardial tissue using novel mapping techniques. These techniques are considered one of the most significant innovations of CMR, alongside the standard ability of quantifying the degree of focal fibrosis (8). Also, CMR is considered to be one of the most important non-invasive imaging modalities for risk stratification in some cardiovascular diseases (9).

The most common conditions to be considered in differential diagnosis of HCM are athletic/sport heart, hypertensive heart disease, aortic stenosis, myocardial amyloidosis, and Fabry disease. The differential diagnosis for asymmetric myocardial thickening includes usually cardiac sarcoidosis, while for the apical hypertrophy, it's a mural thrombus (1). The most challenging clinical problem is a distinction between physiological myocardial hypertrophy (athlete's heart) and hypertrophic cardiomyopathy, which has been registered as the most common cause of non-traumatic sudden cardiac death (SCD) during the exercise in young athletes (< 35 years old) (10). SCD is definitely the most dangerous complication of HCM caused by malignant arrhythmias. However, the mechanisms of its occurrence are not fully understood due to the synergistic effect of pro-arrhythmic factors. Myocyte disturbance and fibrosis are the most common factors, as they alter the electrophysiological properties of the heart (11). The aims of this study were to diagnose HCM in patients with hypertrophic left ventricle walls, to evaluate myocardial tissue for edema or fibrosis and to assess the degree of myocardial fibrosis using CMR.

MATERIAL AND METHODOLOGY

The study was conducted at the Center of Radiology, University Clinical Center of Serbia, Department of Magnetic Resonance Imaging. All collected patient data were taken from the official medical records. The classification of this study was as a retrospective observational study. Out of a total number of 92 patients who met the echocardiographic and ECG criteria for myocardial hypertrophy, CMR revealed hypertrophic cardiomyopathy in 51 patients. Additionally, 25 patients showed CMR signs for cardiac amyloidosis (both transthyretin amyloidosis (ATTR) and light chain (AL)), 2 with athlete's heart, 8 with aortic stenosis, and 6 of them had arterial hypertension. Only patients with hypertrophic cardiomyopathy were included in further evaluation. CMR was performed during a period from June 2022 to January 2024 and revealed HCM in 51 individuals, who represented the cohort of patients for this study. This study was approved by the Ethical Committee of Faculty of Medicine University of Belgrade number 29/IX-14, in 2016 year. Patients all signed consent for CMR examination with contrast media application. Descriptive statistical methods were used for presenting data as absolute numbers and percentages. Among the descriptive statistical methods, measures of central tendency were used such as the arithmetic mean or positional median, depending on the distribution and data type, as well as measures of variability like standard deviation.

Normality of numerical variables was assessed using the Kolmogorov–Smirnov test, where $p < 0.05$ indicated a non-normal distribution. Accordingly, normally distributed variables were compared using the independent-samples t-test, while non-normally distributed variables were analyzed using the Mann–Whitney U test. Categorical variables were compared using the Chi-square test. Correlations were evaluated using Spearman's rank correlation. The Microsoft Excel 2013 program (Redmond, Washington, USA) was used for graphical and tabular data presentation.

In our study, all patients with echocardiographically confirmed myocardial hypertrophy underwent CMR, which in this context was used as the reference diagnostic method for myocardial tissue characterization and for determining the cause of hypertrophy, and has already been established in the literature as the gold standard with optimal diagnostic accuracy. Therefore, additional diagnostic procedures such as myocardial biopsy and histological verification were not performed.

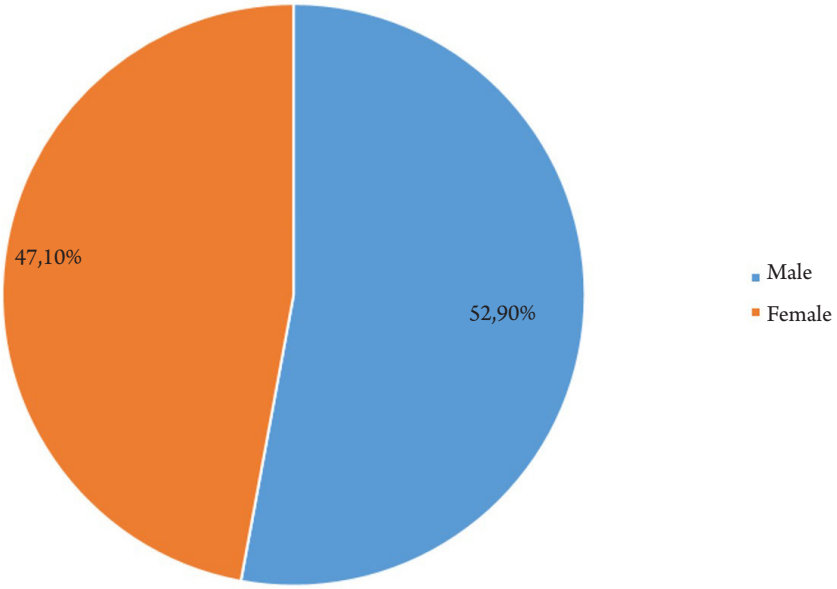

Figure 1. Sex distribution in the hypertrophic cardiomyopathy population

CMR is a multiparametric, repeatable and non-invasive high-resolution imaging technique that does not involve ionizing radiation (8,12). Based on the classical imaging protocol applied in our institution, it allows the differentiation and distinction of several diseases associated with myocardial hypertrophy. CMR was performed on 1.5 T Siemens scanner in all patients, with the administration of the contrast agent Gadolinium in order to obtain the *Late Gadolinium Enhancement* (LGE) phenomenon. The contrast agent was administered intravenously through the cubital vein at a dose of 0.2 ml/kg per body weight. The examination lasted approximately 60 minutes and it included all native images, both morphological and dynamic – cine, followed by LGE sequences acquired 10 minutes after contrast administration (8,12). Regarding the distribution of LGE phenomenon, CMR enables the detection of focal myocardial fibrosis and allows for differentiation of the etiology of the hypertrophic myocardium as we have already mentioned (8,12,13). Additionally, our study was based on a descriptive analysis of CMR findings in primary hypertrophic cardiomyopathy, a control group without myocardial hypertrophy was not included, and therefore it was not possible to calculate the sensitivity and specificity of this diagnostic procedure itself.

This study was approved by the Ethical Committee of Faculty of Medicine University of Belgrade number 29/IX-14, in 2016. Written informed consent has been obtained from the patient(s) to publish this paper.

RESULTS

In our study, there were 24 women (47.1 %) and 27 men (52.9%) **(Figure 1)**.

It is typical that LV mass is higher in men than women, and this is also reflected in our study results. The average LV mass in male was 213 g, while in female was 160 g **(Table 1)**.

Female patients were significantly older (with a mean age of 61.8±11.7 years) and had lower EDV, ESV, and SV in both ventricles, along with reduced LV mass **(Table 1)**. No significant differences were observed in the existence of LGE (p=0.904) or heterogeneous myocardium (p=0.539) based on patient sex.

The incidence of LVOT obstruction was recorded in 9 of 51 pts (17.6%) while the majority of patients had non-obstructive cardiomyopathy (82.4%).

Of the total number of patients, asymmetric HCM was registered in 41 (80.4%) patients **(Figure 2)**.

In the group of patients with asymmetric HCM, LVOT obstruction was observed in 9 (22%) patients. The apical form of HCM was identified in 10 (19.6%) patients, while concentric and symmetric HCM was not detected in our population **(Table 2)**.

The LGE phenomenon was detected in the majority of patients - 39 pts (76.5%) with a predominantly mesomyocardial distribution seen in 51% of cases.

As noted in **Table 3**, LGE was detected in the septal wall in the majority of patients in nearly in 63% of cases, the anterior wall in 51% of patients, and the inferior wall in 31% of patients **(Figure 3)**.

Meanwhile, the presence of LGE in the apex of left ventricle was detected in a relatively small number of patients since the apical form of HCM was not so frequent in our population. In addition to accurately determining the localization of myocardial fibrosis, the use of CMR also quantified the degree of the aforementioned myocardial fibrosis that was observed in HCM patients. In this cohort of patients, the degree of myocardial fibrosis was assessed at a median of 6% of the LV mass (values detected in the range of min 0.8, max 46.5%) and a median of

Table 1. Clinical characteristics of the patients and left ventricular parameters depending on the patient sex

	Sex		
	Male (n=27)	Female (n=24)	P value
Age (years)	54.3±12.5	61.8±11.7	**0.033**
Weight (kg)	86.4±13.2	72.9±13.8	**0.001**
BMI	26.9±3.8	25.9±5.3	0.445
BSA	2.1±0.2	1.8±0.2	**<0.001**
Systolic BP	120.0 (120.0-145.0)	127.0 (110.0-140.0)	0.581
Diastolic BP	80.0 (80.0-80.0)	80.0 (72.0-80.0)	0.254
LDL	3.0 (2.8-3.6)	3.0 (2.8-3.1)	0.316
HDL	1.4 (1.1-1.5)	1.5 (1.2-1.6)	0.290
Total Cholesterol	5.2 (4.7-5.6)	5.1 (4.9-5.3)	0.692
LV parameters			
Septal thickness	19.0 (17.0-22.0)	20 (13.5-23.0)	0.917
Apical thickness	8.0 (8.0-9.0)	9.0 (8.0-12.0)	0.323
EF LV	66.0 (62.0-68.0)	65.0 (60.5-67.5)	0.449
EDV	139.2±37.8	111.5±28.8	**0.005**
ESV	50.7±14.8	39.6±14.3	**0.009**
SV	89.5±26.4	71.7±17.2	**0.007**
Indexed EDV	69.2±16	61.2±13.9	0.064
Indexed ESV	24.2±7.2	21.7±7.2	0.221
Indexed SV	44.8±10.5	39.4±8.4	0.051
CI	2.8±0.6	2.5±0.6	0.076
LV mass	213.5±52.7	160.5±47.3	**0.001**
Left Atrium Area	19±4.5	19.4±4.5	0.736
RV parameters			
EF RV	66.2±6	66.2±4.7	0.988
EDV	129.4±23.7	104±19.7	**<0.001**
ESV	44.3±12.4	35.2±8	**0.003**
SV	85.6±16.3	69.1±14.8	**<0.001**
Indexed EDV	63.0 (55.0-73.0)	60.5 (52.0-64.0)	0.245
Indexed ESV	21.0 (15.0-28.0)	20.5 (17.0-22.0)	0.324
Indexed SV	41.9±8.4	39.5±12.8	0.243
CI	2.7 (2.3-3.0)	2.5 (2.0-2.9)	0.254
Right Atrium Area	16.0(15.0-18.0)	15.0 (14.0-16.0)	**0.044**

BMI – Body Mass Index; BSA - Body Surface Area; BP - Blood pressure; LDL - Low-density lipoprotein; HDL - High-density lipoprotein; LV - Left ventricle; EF - Ejection fraction; EDV - End-diastolic volume; ESV - End-systolic volume; SV - Stroke volume; CI - Cardiac index; RV - Right ventricle. Data are presented as mean ± standard deviation for normally distributed variables and as median (interquartile range) for non-normally distributed variables. The independent-samples t-test was performed for normally distributed variables and the Mann–Whitney U test for non-normal variables. Significant p values (less than 0.05) are marked in bold font.

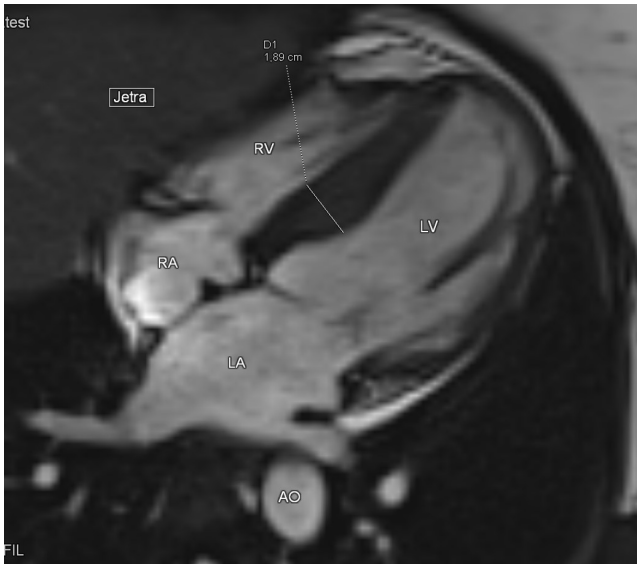

Figure 2. Cardiac magnetic resonance (CMR) cine sequences 4 chamber view with no contrast media presenting asymmetric septal hypertrophy of left ventricle (septal wall 19 mm). No patient-identifying features are visible

Table 2. Morphological forms of HCM.

	Frequency	Percentage (%)
Asymmetric HCM	41	80.4
Apical HCM	10	19.6
Concentric HCM	0	0
Total	51	100.0

HCM – Hypertrophic Cardiomyopathy

Table 3. LGE phenomenon distribution in left ventricle

Presence of LGE phenomenon localization	Number of pts	Percentage (%)
Septum	**32**	**62.7**
Lateral wall	13	25.5
Anterior wall	**26**	**51.0**
Insertion point	12	23.5
Apical	9	17.6
Inferior wall	**16**	**31.4**
Posterior wall	7	13.7

LGE - Late Gadolinium Enhancement

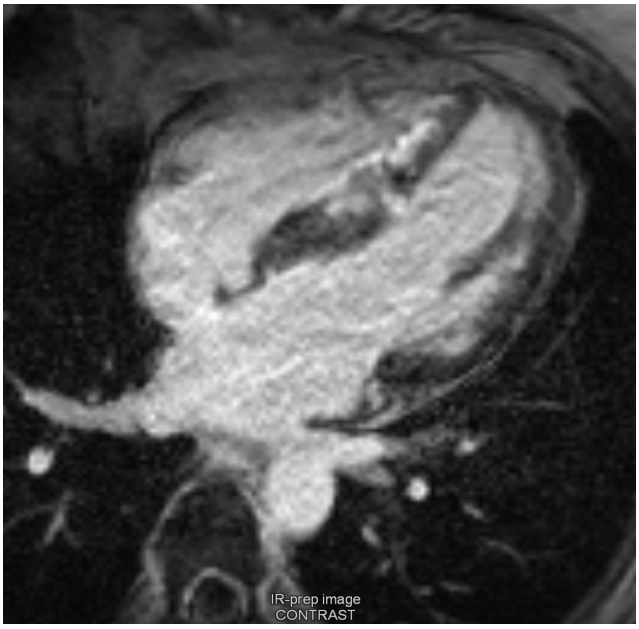

Figure 3. Cardiac magnetic resonance (CMR) 4 chamber view long axis after contrast media application (Gadolinium) showing mesomyocardial LGE phenomenon in sepal wall. No patient-identifying features are visible

11 g of fibrosis per gram of LV mass (values detected in the range of 1.0–135.8 g). The majority of patients exhibited LGE values of up to 5% (**Figure 4**), with the minimum recorded value at 0.8% and the maximum at 46.5%. Only 3 patients (6%) from the cohort had a degree of focal fibrosis that presented as a LGE phenomenon of greater than 15%.

The overall severity of LGE showed a moderate, significant positive correlation with septal thickness (rs=0.458, p=0.004, **Figure 5**), whereas no correlation was observed between LGE extent and apical thickness (rs=0.094, p=0.581, **Figure 6**). Moreover, the extent of LGE presented with a moderate, significant negative correlation with LV EF (rs= -0.512, p=0.001, **Figure 7**).

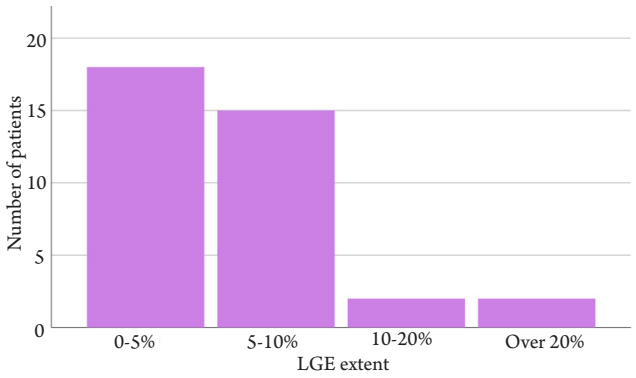

Figure 4. LGE extent expressed as percentage of myocardial mass in patients with HCM

In addition to focal fibrosis, which was most frequently registered in our patients as a LGE phenomenon, there were also patients with diffusely heterogeneous myocardium after administration of Gadolinium contrast media, and that was seen in 10 patients, representing about 20% of the cohort.

DISCUSSION

In this retrospective study analyzing patients with HCM, there was a slightly higher number of male patients 27 (53%). In the study of Abraham and colleagues, which included a systematic review of data from 37700 patients, it was shown that there was no statistically significant difference in the incidence of HCM between men and women (8, 14). However, in the study of Angkawipa et al, it was demonstrated that HCM was more prevalent among men (15). It is known that the LV mass is greater in men than in women, which is also seen in our population.

In our research, the most common form of HCM was asymmetric HCM observed in 80% of participants. The study by Wenn Xu and colleagues also showed that asymmetric HCM had been the most frequent form of HCM (60–70%) (12). In our population with asymmetric HCM LVOT obstruction was recorded in 22% of patients.

The data from our study differ from those of the study by Song et al, that reported on a significantly larger number of patients with HCM (758 subjects) and showed that obstruction of LVOT occurred in the majority of patients, present in almost two-thirds of the participants (16).

Regarding the presence and assessment of the degree of myocardial fibrosis in HCM patients, we observed the presence of pathological accumulation of contrast media in a form of LGE phenomenon (17). However, as previously noted, the absence of LGE phenomenon does not imply the absence of fibrosis, as LGE only indicates the presence of focal myocardial fibrosis (18). In our study, LGE was detected in a vast majority of patients, in about 77% of cases.

In the study of Aquaro et al, myocardial fibrosis in the form of the LGE phenomenon was also found in over 60%

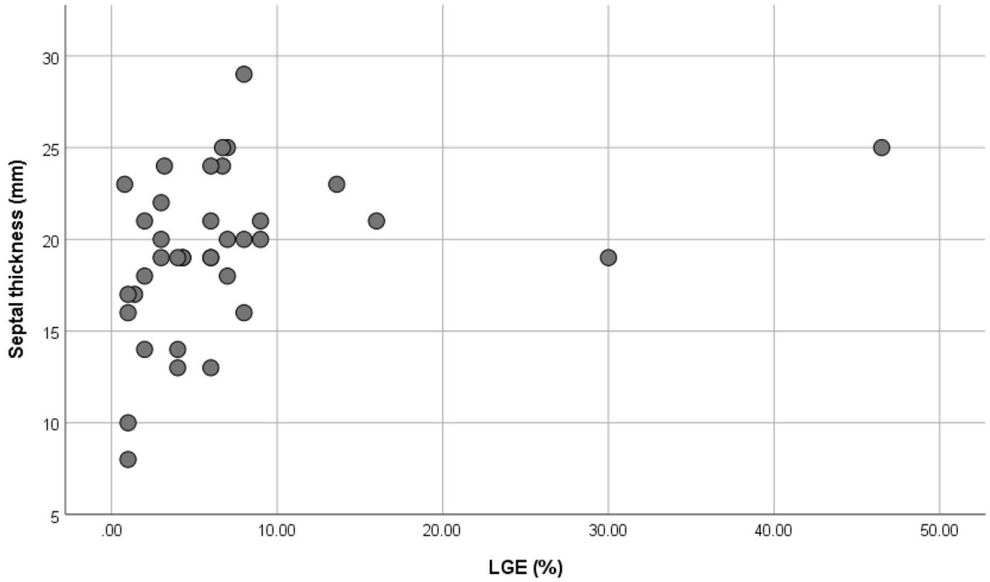

Figure 5. Correlation of left ventricular late gadolinium enhancement (LGE) extent with septal thickness.

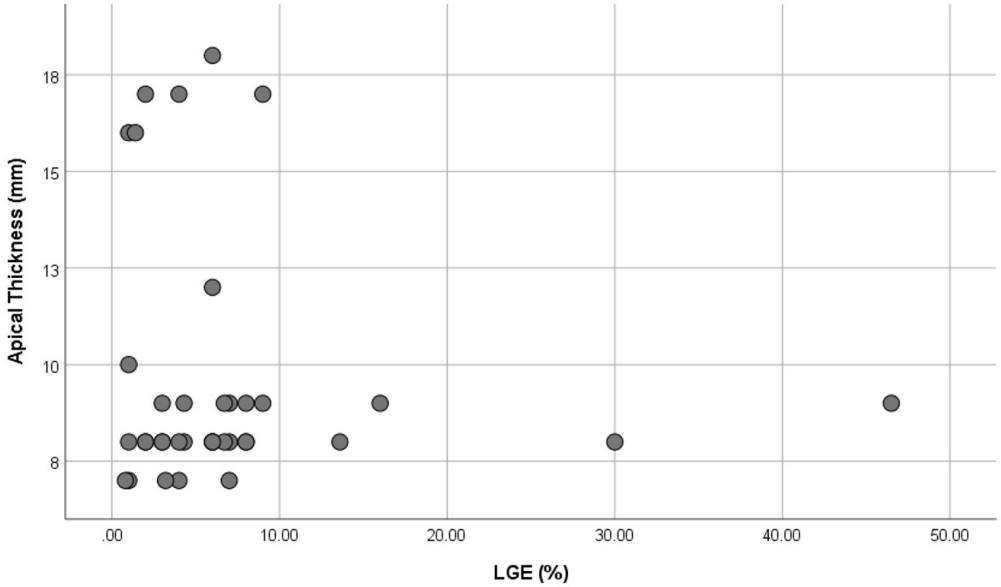

Figure 6. Correlation of left ventricular late gadolinium enhancement (LGE) extent with apical thickness.

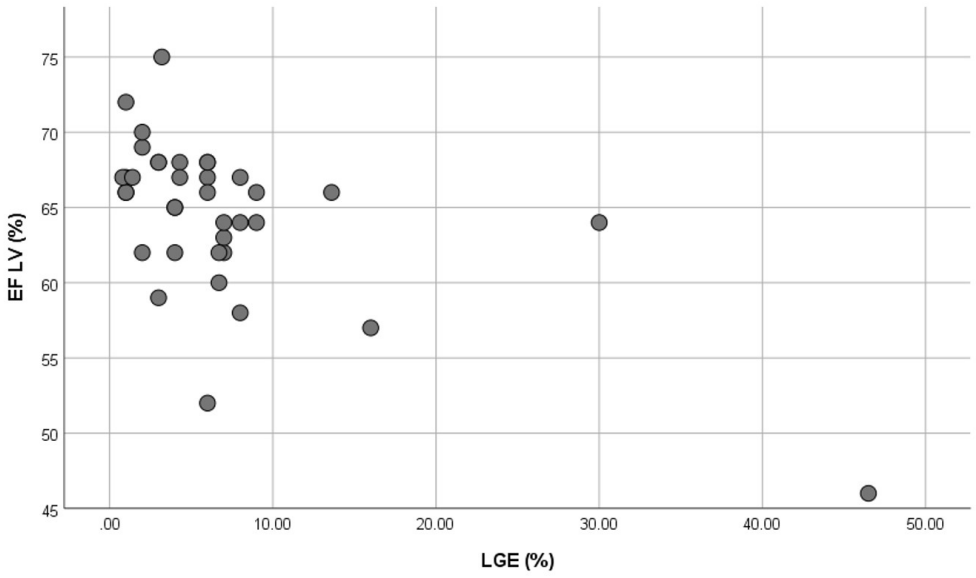

Figure 7. Correlation of left ventricular late gadolinium enhancement (LGE) extent left ventricle (LV) ejection fraction (EF).

of cases with HCM, and it was considered as a marker of replacement focal fibrosis (19). In the study of Małoch et al, which included 50 patients with HCM, myocardial fibrosis expressed through the LGE phenomenon was noted to occur in 37 (74%) patients, which aligns with the results of our sample (20). However, in the study of Prinz et al, which included 87 patients, about 90% of participants had LGE phenomenon (21).

In our population, the most common localization of LGE was in the interventricular septal wall (63%), then in the anterior wall (51%), and the inferior wall (31%). In the study by Liu et al, was shown also that myocardial fibrosis was predominantly distributed in the interventricular septum and in the anterior wall (22), which is consistent with our findings. Studies by Barbosa et al., Rodrigues, Machii, Sultan, as well as Chan and co-authors, have demonstrated that the mid-septal wall was the most frequent localization for detecting the LGE phenomenon (23–26).

In our study, a heterogeneous myocardium, meaning myocardium with diffuse interstitial fibrosis and with increased vules of native T1 mapping, was observed in 10 patients (20%). In the study by Małoch et al., heterogeneous myocardium was present in 28% of the 54 analyzed cases (20), which is in accordance with our results.

The presence of focal myocardial fibrosis presented as LGE phenomenon seen on CMR is considered one of the important predictive factors associated with an increased risk of adverse outcomes and SCD (27–31). Various studies have shown that in patients with confirmed LGE phenomenon detected on CMR, there is a higher incidence of malignant arrhythmias, SCD, and all-cause mortality. The study by Małoch et al. showed a positive correlation between the degree of myocardial fibrosis and frequency of ventricular tachycardia (20). Extensive LGE seen on CMR could be used as a risk stratification factor for traditional SCD in patients with HCM as well as for better understanding their overall risk of ventricular arrhythmias (32, 33). It was previously considered that LV wall thickness brings to a higher incidence of ICD therapy than other traditional SCD risk factors (32). But new studies have shown that the presence of massive focal myocardial fibrosis, however, identifies a subgroup of patients with no large myocardial wall hypertrophy but with an equally high incidence of receiving antiarrhythmic device therapy (34).

In addition, the study of Rader and co-authors found that the presence of extensive LGE phenomenon with more than 15% fibrosis of myocardial mass was associated with a higher risk of SCD compared to patients with a lower percentage of LGE in HCM patients (31, 35).

In our study, the degree of myocardial fibrosis was estimated at a median of 6% of LV mass, and only 3 patients (6%) out of 51 had an extensive and high degree of myocardial fibrosis in the form of LGE phenomenon exceeding 15% of LV mass. All those three patients, after undergoing consultant review, received Implantable Cardioverter Defibrillators (ICD) in order to reduce the risk of SCD.

According to study by Sebastian and Alexandra, the incidence of atrial fibrillation (AF) in patients with HCM was 22% in both sexes. Also, they demonstrated that women have a moderately increased risk for AF, as well as for myocardial fibrosis (36, 37).

CONCLUSION

This study represents an important contribution for understanding the role of CMR in diagnosis of HCM. CMR is a non-invasive diagnostic method that is considered as a gold standard for myocardial tissue characterization. It plays a crucial role not only in the diagnosis of HCM, but also in the differential diagnosis of myocardial hypertrophy, allowing for accurate distinction between pathological and physiological hypertrophy, as well as infiltrative or pressure-overload conditions that can mimic HCM. CMR provides high-quality visualization of cardiac morphology as well as functional assessment, enabling further treatment planning. It is important to emphasize that CMR offers numerous benefits, not only in terms of tissue characterization without the use of ionizing radiation, but also in the non-invasive measurement of volumetric parameters of the heart chambers. The detection of myocardial fibrosis, as a main trigger for the development of malignant cardiac arrhythmias, places CMR not only as a diagnostic tool, but also as a risk stratification method to evaluate the risk SCD in these patients.

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Author contributions: Conceptualization, O.N.-A., T.B. and I.N; methodology, O.N.-A. A.M.M and M.T.; software, R.M. and A.M.M.; validation, A.T. and M.T; formal analysis, M.T. and A.T.; investigation, O.N.-A, I.N. and M.T.; resources, I.N. and M.T.; data curation, T.B. and A.T.; writing—original draft preparation, O.N.-A. and T.B; writing—review and editing, I.N. and R.M.; visualization, O.N.-A. and A.M.M.; supervision, R.M and I.N; project administration, O.N.-A. and M.T; funding acquisition, R.M. All authors have read and agreed to the published version of the manuscript.

Ethical approval: This study was approved by the Ethical Committee of Faculty of Medicine University of Belgrade number 29/IX-14, in 2016.

Informed consent: Written informed consent has been obtained from the patient(s) to publish this paper.

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ULOGA I ZNAČAJ MAGNETNE REZONANCE SRCA KOD HIPERTROFIČNE KARDIOMIOPATIJE

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Sažetak

Uvod: Hipertrofična kardiomiopatija (engl. *hypertrophic cardiomyopathy* – HCM) je autozomno dominantna bolest kardiomiocita koja dovodi do njihovog zadebljanja i hipertrofije. Ciljevi ovog rada su bili da se dijagnostikuje hipertrofična kardiomiopatija pomoću magnetne rezonance (MR) srca kod pacijenata sa hipertrofijom miokarda leve komore, okarakteriše tkivo miokarda i odredi stepen fibroze ovom dijagnostičkom metodom.

Metode: Studija je obuhvatila 51 pacijenta sa HCM dijagnostikovanom na MR srca (muškarci 27; žene 24) i sprovedena je na Univerzitetskom kliničkom centru Srbije, Centru za radiologiju. Svi prikupljeni podaci o pacijentima su uzeti iz zvanične medicinske dokumentacije, a tip studije je retrospektivna opservaciona studija. MR srca je kod 51 pacijenta pokazao postojanje HCM na osnovu klasičnog protokola snimanja rađenog na 1.5T Siemens aparatu uz davanje kontrastnog sredstva Gadolinijuma. U odnosu na distribuciju kontrasta u vidu *Late Gadolinium Enhancement (LGE)* MR srca omogućava otkrivanje fokalne fibroze, a samim tim i razlikovanje etiologije hipertrofičnog miokarda.

Rezultati: Opstrukcija izlaznog trakta leve komore viđena je kod 9 (17.6%) pacijenata. Asimetrična HCM je registrovana kod 41 (80.4%) bolesnika. Prisustvo LGE detektovano je kod 39 (76,5%) pacijenata, najviše u predelu septuma (62.7%). Stepen fibroze procenjen je na medianu od 6% mase leve komore i medianu od 11 g fibroze po gramu mase leve komore.

Zaključak: MR srca kao neinvazivna metoda predstavlja zlatni standard u tkivnoj karakterizaciji srčanog mišića. Detekcija fibroze miokarda i njeno kvantifikovanje, kao glavnog okidača za razvoj malignih srčanih aritmija, svrstava MR srca, ne samo u dijagnostički, već i u metod stratifikacije rizika od naprasne srčane smrti kod pacijenata sa hipertrofičnom kardiomiopatijom.

Ključne reči: hipertrofična kardiomiopatija, magnetna rezonanca srca, kasno nakupljanje gadolinijuma, stepen fibroze, aritmije

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