

Left Atrial Dysfunction as Marker of Arrhythmic Events in Patients with Hypertrophic Cardiomyopathy

ABSTRACT

Background: In this study, we investigated whether left atrial functions evaluated by speckle tracking echocardiography, classic echocardiographic and clinic parameters predict appropriate Implantable Cardioverter Defibrillator (ICD) shock in patients who underwent ICD implantation for hypertrophic cardiomyopathy.

Methods: Totally 87 patients who received ICD implantation for primary or secondary prevention were included in the study. Patients' clinical, electrocardiographic, 2 dimension classic, and speckle tracking echocardiographic data were collected. Left atrial functions were assessed by speckle tracking echocardiography. Left atrial strain just before mitral valve opening was taken as peak atrial longitudinal strain. Appropriate ICD therapy was defined as cardioversion or defibrillation due to ventricular tachycardia or fibrillation. Patients were divided into 2 groups as occurrence or absence of appropriate ICD therapy during follow-up (mean, 50.2 ± 9.3 months). Patients with an European Society of Cardiology (ESC) risk score >6% were considered high-risk patients.

Results: A total of 24 (27.5 %) patients were observed to have an appropriate ICD therapy. In patients on whom appropriate ICD therapy was performed, a higher Sudden Cardiac Death risk Score and decreased peak atrial longitudinal strain and global longitudinal peak strain were observed. In patients with high ESC risk score (> 6%), in Cox regression analysis, peak atrial longitudinal strain (odds ratio: 0.806, $P = .008$), Sudden Cardiac Death risk score (odds ratio: 1.114, $P = .03$) and global longitudinal peak strain (odds ratio: 1.263, $P = .02$) were found to be independent predictors of occurrence of appropriate ICD therapy.

Conclusion: Easily measurable peak atrial longitudinal strain may provide additional information in predicting ventricular arrhythmias or deciding on prophylactic medical treatment to prevent ventricular arrhythmias or reduce the frequency of appropriate shock in high-risk patients with ICD implanted.

Keywords: Hypertrophic cardiomyopathy, speckle tracking echocardiography, ventricular arrhythmia

INTRODUCTION

Hypertrophic cardiomyopathy (HCM) is a hereditary disease and the most common cause leading to sudden cardiac death (SCD) in young people. This disease is characterized by abnormal left ventricular hypertrophy resulting from the disarray of the myocardial fibers. Fibrous tissue formation due to disarray of myocardial fibers and microvascular myocardial ischemia due to hypertrophy is responsible for ventricular arrhythmias in HCM patients.^{1,2} Ventricular arrhythmias are the most important cause of mortality and can be significantly treated with Implantable Cardioverter Defibrillator (ICD) therapy. The HCM patient population has a wide clinical spectrum, and some patients experience SCD, while some patients do not experience any arrhythmic events throughout their lifetime.

It is very important to administer ICD therapy to patients who really need ICD treatment, as not administering ICD therapy to patients who may develop ventricular arrhythmias in the future, or administering ICD therapy to patients who will not develop ventricular arrhythmias, may lead to serious consequences such

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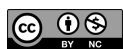
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as death, inappropriate shock, or device-related complications. Various clinical, genetic, and imaging methods are used to detect patients who may develop arrhythmias. The HCM risk score was created by mathematical modeling of these risk factors and is frequently used.³ Left atrial enlargement, one of the parameters used in HCM risk score, was associated with poor outcomes in long-term follow-up.⁴ In the study of Minami et al⁵, left atrial (LA) enlargement (≥ 48 mm) was found to be associated with poor outcome, especially in patients without atrial fibrillation (AF). Speckle tracking echocardiography (STE)-based left atrial strain parameters, which are unaffected by cardiac retractions and independent of angle, provide better information in evaluating left atrial function. In the study of Essayagh et al⁶ in HCM patients with 21-month follow-up, decreased peak atrial longitudinal strain (PALS) was found to be an independent predictor for poor outcomes. Speckle tracking echocardiography-based strain measurements in HCM patients may provide additional information to differentiate patients who may develop SCD or ventricular arrhythmia in the future. In this study, we investigated the effect of STE-based left atrial strain in predicting appropriate ICD shock therapy in HCM patients with ICD implanted.

METHODS

A total of 102 patients who underwent ICD implantation for primary or secondary prevention in the hospital between 2014 and 2020 were included in the study. Patients' clinical, electrocardiographic, and echocardiographic evaluation before implantation and patients' data were obtained and retrospectively analyzed.

The inclusion criteria consisted of 2-dimensional (D) echocardiographic demonstration of an unexplained increase in wall thickness of >15 mm in the absence of abnormal load conditions and any cardiac or systemic disease that could cause left ventricular hypertrophy (fabry, amyloid), while the exclusion criteria included a history of ablation or septal myectomy, having coronary artery disease, severe mitral and aortic valve disease (stenosis or insufficiency), AF, hypertension (systolic blood pressure > 140 mm Hg)

HIGHLIGHTS

- Hypertrophic cardiomyopathy risk-Sudden Cardiac Death (HCM risk-SCD) score may not accurately predict patients who will experience arrhythmic events in the future. Additional risk factors are being investigated.
- Left atrial strain is associated with composite cardiovascular outcomes (sudden cardiac death, appropriate defibrillator therapy, and hospitalization for heart failure) in HCM patients.
- Left atrial strain may provide additional information in predicting appropriate ICD shock.
- As a result of this study, while atrial strain was found to be predictive for appropriate ICD shock in those with high HCM Risk-SCD score ($>6\%$), it was not found to be predictive in all risk scores.

or diastolic blood pressure > 90 mm Hg), left ventricular ejection fraction (EF) ≤ 50 , and chronic obstructive pulmonary disease.

Patients' baseline clinical data, symptom status and medical history, baseline electrocardiogram, Holter monitoring records, and baseline echocardiographic images were obtained from the hospital records and electronic archives of the hospital and were analyzed. Follow-up evaluations were performed with the data obtained from the records of the cardiology department at 6-month intervals or earlier presentations at the time of symptoms. Because our center is a tertiary central hospital, data of the patients with follow-up in more rural regions were obtained via telephone conversations or the national medical archive (E-pulse system). Routine pacemaker control information of the patients with ICD implanted was obtained from the hospital records or the medical company. Arrhythmic events were checked and confirmed by an electrophysiologist in patients with shock. Totally 8 patients were unable to attend follow-up visits and 7 patients with poor echogenicity were excluded from the study. Finally, a total of 87 patients were enrolled in the study.

Hypertrophic cardiomyopathy risk evaluation was calculated with an online calculator in line with the ESC guidelines based on the clinical and echocardiographic data of the patients. Three and more consecutive premature wide complexes with heart rate >120 beats/min and lasting <30 seconds were defined as non-sustained ventricular tachycardia. When the rhythm lasts longer than 30 seconds or hemodynamic instability occurs in less than 30 seconds, it is considered sustained ventricular tachycardia. Appropriate ICD therapy was defined as cardioversion or defibrillation due to ventricular tachycardia (VT) or defibrillation due to ventricular fibrillation (VF). Patients were divided into 2 groups as occurrence or absence of appropriate ICD therapy at follow-up. Informed written consent was obtained from all study subjects, and the study protocol was approved by the Institutional Ethics Committee.

ICD Implantation and Settings

All devices were implanted in the left pectoral region using subclavian venous pathway. Used systems were manufactured by Biotronik (Berlin, Germany), Boston Scientific (Natick, Mass, USA), Medtronic (Minneapolis, Minn, USA), and St Jude Medical (St Paul, Minn, USA).

ICD was programmed by an experienced electrophysiologist according to the patient's electrophysiological characteristics or history of arrhythmia. Antitachycardia settings of the ICD devices were programmed with 3 consecutive zones. Heart rate was set at 160-180 bpm in the monitor zone, 185-200 bpm in the anti-tachycardia pacing zone, and 205-210 bpm in the initial shock zone. While no therapy was programmed in the monitor zone, defibrillator was programmed so as to give shock if arrhythmia continued after 2 bursts in the Antitachycardia pacing (ATP) shock zone. Initial zone was programmed so as the device shock to be the initial therapy in case of ventricular arrhythmia faster than the ATP shock zone.^{7,8}

Echocardiography

All echocardiographic studies were performed using a Vivid 7 machine (GE Vingmed Ultrasound AS, Horten, Norway), equipped with a 3.5 MHz transducer. A total of 3 cardiac cycles were recorded at the end of expiration. Frame rate was set in the range of 50-70 frames per second for 2-D image acquisition. Settings were adjusted manually to obtain optimal images. All data were transferred to a workstation for further offline analysis (EchoPAC PC; GE Vingmed Ultrasound AS).

Conventional Echocardiography

Maximal wall thickness (MWT) was measured from all left ventricular (LV) segments from base to apex in parasternal short-axis view. Left ventricular end-systolic diameter and end-diastolic diameter were measured from parasternal long-axis view according to recommendations of the American Society of Echocardiography.⁹ Left ventricular end-systolic volume, end-diastolic volume, and EF were determined from the apical 4 and 2-chamber views using Simpson's modified biplane method. Atrial diameter was calculated by 2-D echocardiography in the parasternal long-axis plane. Left atrial volume was obtained using the biplane area length method from apical 4 and 2-chamber images at end-systole and it was also indexed to body surface area as recommended.¹⁰

Mitral inflow velocities were measured at tips of the mitral leaflets using pulsed Doppler performed at end-expiration. The peak velocity during early filling (E) and late filling from atrial contraction (A) was recorded.

Speckle Tracking Echocardiography

The longitudinal strain of left ventricular was obtained from apical 4, 3, and 2-chamber views. After determining the appropriate cardiac cycle, the endocardial borders were traced at the end-systolic frame and an automated tracking algorithm outlined the myocardium in successive frames throughout the cardiac cycle. The tracking quality was verified for each segment, with subsequent manual adjustment of the region of interest if necessary. The LV was divided into 6 segments in each view automatically by the software. The global longitudinal peak strain value was calculated by the software by averaging a total of 18 segments from 3 echocardiography views. Systolic strain and systolic strain rates were measured from each strain and strain rate curve.¹¹

Apical 4-chamber and 2-chamber view was used for LA strain measurements. For 2-dimensional STE analysis, a line was manually drawn along the LA endocardium when the LA was at its minimum volume after contraction. Before processing, a cine loop preview feature visually confirmed that the internal line follows the LA endocardium throughout the cardiac cycle. The software divided the LA endocardium into 6 segments. Left atrial peak strain just before mitral valve opening was taken as PALS and LA strain just before atrial contraction (onset of the P-wave on electrocardiography) was taken as peak atrial contraction strain (PACS) (Figure 1). Global PALS and PACS were obtained by averaging the apical 2- and 4-chamber strain parameters.¹² Overall, 1044 segments were analyzed (12 segments for each patient), and a total of 4.2% segments were excluded.

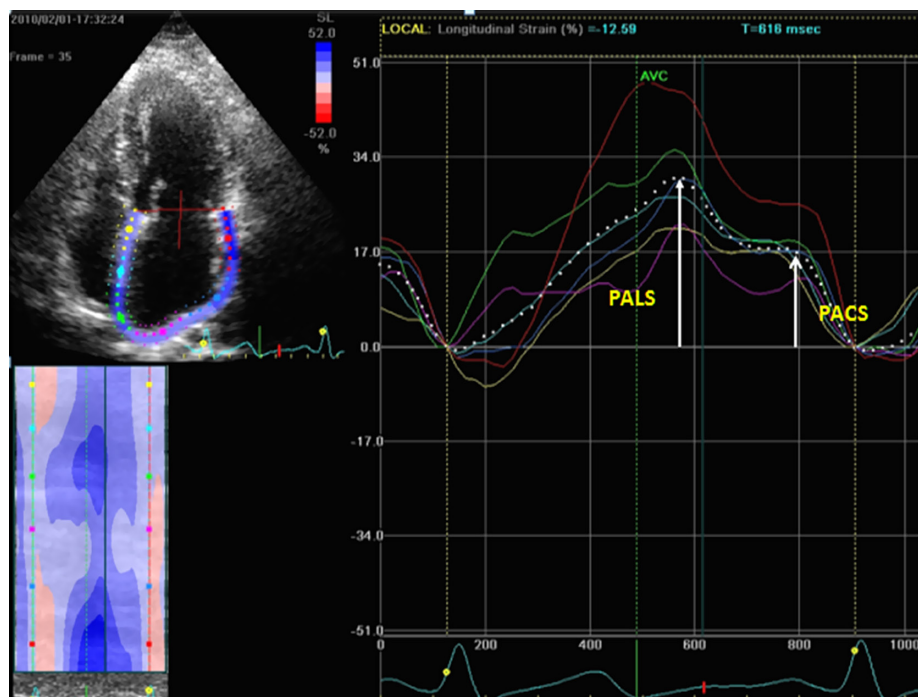


Figure 1. Left atrial longitudinal strain parameters. Composite figure showing the measurement of peak atrial longitudinal strain (PALS) and peak atrial contraction strain (PACS) using the speckle tracking echocardiography from an apical 4-chamber view. The dashed curve represents the average atrial longitudinal strain along the cardiac cycle. AVC, aortic valve closure; PALS, peak atrial longitudinal strain; PACS, peak atrial contraction strain.

Statistical Analysis

Patient characteristics were expressed as the mean \pm standard deviation or as percentages. Continuous variables in 2 groups were compared by Student's *t* test or Mann-Whitney *U* test. Distribution of categorical variables was analyzed by using χ^2 or Fisher's exact test when appropriate. Pearson's correlation was used to test the relationship between the continuous variables. Subsequently, Cox regression analysis was performed to determine independent predictors of appropriate ICD therapy. All statistical analyses were performed with Statistical Package for the Social Sciences,

version 24.0 (SPSS, Inc, Chicago, Ill, USA), and a *P* value of $<.05$ was considered statistically significant.

RESULTS

In total, 87 patients with HCM who underwent ICD implantation (74% male; mean age, 47.1 ± 14.7 years) were included in the study. Echocardiographic and clinical characterizations of study populations are summarized in Table 1. The echocardiography was performed at 5-7 days before ICD implantation. The mean follow-up period of the patients was 50.2 ± 9.3 months.

Table 1. Echocardiographic and Clinical Characteristic of the Study Population

Variable	All Patients n=87	HCM Risk -SCD Score (All Group)			HCM Risk-SCD Score > 6%		
		ICD Therapy (-) n=63	ICD Therapy (+) n=24	<i>P</i>	ICD Therapy (-) n=35	ICD Therapy (+) n=18	<i>P</i>
Age (years)	47.1 \pm 14.7	47.7 \pm 14.7	45.5 \pm 15.1	.5	40.8 \pm 11.5	41.8 \pm 13.3	.7
Sex male (%)	64 (74)	45 (71)	19 (79)	.6	25 (71.4)	14 (77.8)	.7
Body surface area (m ²)	1.85 \pm 0.14	1.85 \pm 0.15	1.85 \pm 0.13	.8	1.81 \pm 0.14	1.87 \pm 0.13	.16
Systolic blood pressure (mm Hg)	126.5 \pm 7.5	126.3 \pm 7.1	126.9 \pm 8.8	.7	127 \pm 6.8	126.2 \pm 8.9	.7
Diastolic blood pressure (mm Hg)	75 \pm 7.1	75 \pm 6.9	75.2 \pm 7.5	.9	76.2 \pm 6	75.2 \pm 7.7	.6
Heart rate (bpm)	68 \pm 8.4	68.1 \pm 8.7	67.8 \pm 7.6	.9	67.4 \pm 8.4	67.5 \pm 8.2	.9
Maximal wall thickness (cm)	24.8 \pm 5.3	24.9 \pm 5.6	24.7 \pm 4.4	.8	26.9 \pm 5.2	24.9 \pm 4.4	.17
End-diastolic diameter (cm)	4.8 \pm 0.3	4.77 \pm 0.32	4.88 \pm 0.24	.1	4.82 \pm 0.28	4.85 \pm 0.24	.7
End-systolic diameter (cm)	3.1 \pm 0.3	3.04 \pm 0.32	3.12 \pm 0.34	.29	3.03 \pm 0.37	3.07 \pm 0.27	.6
LV EF (%)	63.4 \pm 7.5	63.6 \pm 7.2	62.8 \pm 8.4	.6	63.3 \pm 8.9	62.5 \pm 9.6	.7
Peak LVOT gradient (mm Hg)	47 (23, 68)	44 (23, 68)	53.5 (22.2, 83.7)	.7	56 (24,80)	59.5 (22.7,90)	.8
E (m/s)	0.9 \pm 0.2	0.91 \pm 0.2	0.89 \pm 0.15	.5	0.91 \pm 0.22	0.86 \pm 0.16	.4
A (m/s)	0.81 \pm 0.3	0.78 \pm 0.3	0.89 \pm 0.37	.2	0.73 \pm 0.29	0.84 \pm 0.3	.2
Sm (cm/s)	7.3 \pm 1.8	7.4 \pm 2	7 \pm 1.32	.48	7.8 \pm 2.0	7.2 \pm 1.36	.24
E' (cm/s)	6.5 \pm 1.7	6.7 \pm 1.7	6.1 \pm 1.8	.18	6.6 \pm 1.65	6.2 \pm 1.93	.46
A' (cm/s)	6.1 \pm 1.56	6.1 \pm 1.6	6.2 \pm 1.6	.8	6.28 \pm 1.5	6.5 \pm 1.5	.6
GLPS (%)	-12.3 \pm 3.2	-13.1 \pm 3.2	-10.2 \pm 2.1	<.001	-12.6 \pm 3.2	-9.8 \pm 2.2	.002
PALS	20.3 \pm 6.7	21.1 \pm 6.9	18.08 \pm 5.4	.05	21.8 \pm 6.3	16.1 \pm 4.6	.002
PACS	7.3 \pm 2.9	7.4 \pm 3	7 \pm 2.3	.5	7.09 \pm 3	7.2 \pm 2.6	.8
LA diameter (cm)	40.4 \pm 6.1	39.8 \pm 6.5	42 \pm 4.8	.13	40.5 \pm 7.3	42.2 \pm 5.2	.3
LAVi (mL/m ²)	44 \pm 10.8	43.6 \pm 9.7	45.1 \pm 13.4	.5	46.8 \pm 9.7	45.1 \pm 12.9	.5
HCM-Risk score (%)	8.5 \pm 5.5	7.1 \pm 4.3	11.9 \pm 6.8	.003	9.93 \pm 4	14.1 \pm 6.5	.006
Risk factors							
Family history of SCD (%)	49 (56)	35 (56)	14 (58)	0.8	21 (60)	12 (67)	0.6
NsVT, n (%)	59 (68)	40 (64)	19 (79)	0.2	29 (83)	15 (83.3)	0.6
Unexplained syncope, n (%)	37 (43)	20 (32)	17 (71)	.001	14 (40)	14 (78)	.01

LVOT, left ventricular outflow tract; LV EF, left ventricular ejection fraction; LA, left atrial; LAVi, Left Atrial Volume Index; E, Peak Early Filling Trans-mitral Velocity; A, Peak Late Filling Trans-mitral Velocity; Sm, peak longitudinal systolic tissue velocity of the mitral valve annulus; E', peak longitudinal early diastolic tissue velocity of the mitral valve annulus; A', peak longitudinal late diastolic tissue velocity of the mitral valve annulus; GLPS, global longitudinal peak strain; PALS, peak atrial longitudinal strain; PACS, peak atrial contraction strain; HCM, hypertrophic cardiomyopathy; SCD, sudden cardiac death; NsVT, non-sustained ventricular tachycardia.

Despite all patients having normal LV EF, LV diastolic, and systolic diameter, global longitudinal peak strain (GLPS) was impaired in study patients (-12.6 ± 3.3). In addition, mild larger LA diameter (40.4 ± 6.1) and moderate larger LA maximum volume index (44 ± 10.8) were seen in this study patients. In this study population, 5-year Sudden Cardiac Death (SCD) risk score was 8.5 ± 5.5 , and family history of SCD, NsVT, and unexplained syncope were observed in 49 (56%) 59 (68%), and 37(43%) patients, respectively.

A total of 8 (9%) patients received an ICD for secondary prevention, and the remaining 79 (91%) patients had primary prevention indications for ICD implantation. A total of 24 (27.5%) patients were observed to have an appropriate ICD therapy during a median follow-up period of 50.2 ± 9.3 months (shock only in 4 patients, shock after unsuccessful ATP in 6 patients, and ATP only in 14 patients). Appropriate ICD therapy was given for VF (n=5) and VT (n= 19).

The patient population was categorized into groups based on the occurrence or absence of appropriate ICD therapy at follow-up.

In Patients with All HCM Risk-SCD Score

In patients who performed appropriate ICD therapy, a higher Hypertrophic cardiomyopathy risk-Sudden Cardiac Death (HCM risk-SCD) score (11.9 ± 6.8 vs. 7.1 ± 4.3 , $P = .003$), decreased PALS (18.08 ± 5.4 vs. 21.1 ± 6.9 , $P = .05$), and decreased GLPS (-10.2 ± 2.1 vs. -13.1 ± 3.2 , $P < .001$) and more common unexplained syncope [20 (32%) vs. 17 (71%), $P = .001$] was observed.

In correlation analysis, there was no significant correlation between PALS and age, LA diameter, left atrial volume index, MWT, peak Left Ventricular Outflow Tract, gradients, and GLPS.

A Cox regression analysis, including GLPS, PALS, and HCM Risk-SCD score, was used to determine independent predictors of occurrence of appropriate ICD therapy during the follow-up. Hypertrophic cardiomyopathy risk-Sudden Cardiac Death score [odds ratio (OR): 1.075, 95% CI: 1.007-1.148, $P = .03$] and GLPS (OR: 1.178, 95% CI: 1.014-1.368, $P = .03$) was found to be independent predictors of occurrence of appropriate ICD therapy (Table 2).

In Patients with High HCM Risk-SCD Score (>6%)

In patients in whom appropriate ICD therapy was performed, a higher HCM Risk-SCD score (14.1 ± 6.5 vs. 9.93 ± 4 , $P = .006$), decreased PALS (16.1 ± 4.6 vs. 21.8 ± 6.3 , $P = .002$), and decreased GLPS (-9.8 ± 2.2 vs. -12.6 ± 3.2 , $P = .002$), and more common unexplained syncope [14 (40%) vs. 14 (78%), $P = .01$] were observed (Table 1).

A Cox regression analysis, including GLPS, PALS, and HCM Risk-SCD score, was used to determine independent predictors of occurrence of appropriate ICD therapy during the follow-up. Peak atrial longitudinal strain (OR: 0.806, 95% CI: 0.686- 0.946, $P = .008$), HCM Risk-SCD score (OR: 1.114, 95% CI: 1.011-1.226, $P = .03$), and GLPS (OR: 1.263, 95% CI: 1.015-1.571, $P = .02$) were found to be independent predictors of occurrence of appropriate ICD therapy (Table 2).

DISCUSSION

In this study, in HCM patients with ICD implanted, GLPS and HCM Risk-SCD scores were found to be predictive of appropriate ICD therapy in high-risk and all risk groups, while PALS was predictive only in high-risk groups.

Previous studies have found the risk of ventricular arrhythmia as about 19%-23% in patients with ICD implanted.^{13,14} In this study, we found appropriate ICD therapy as 27%. In our study, the current HCM Risk-SCD score was found to be predictive of appropriate ICD therapy in patients with ICD implanted. Hypertrophic cardiomyopathy risk-Sudden Cardiac Death score was criticized by some authors due to insufficiency in the determination of future events.¹⁵ In recent American College of Cardiology, American Heart Association (ACC/AHA) guideline, it is recommended that reduced EF, LV aneurysm, and fibrosis extent (LGE) in cardiac magnetic resonance imaging (MRI), which are not included in the HCM Risk-SCD score, should be evaluated as major sudden death risk factors.¹⁶ However, in our study, we do not have data on this subject, since ICD implantation was performed according to the current HCM Risk-SCD score.

In the study of Latif et al¹⁷ in 28 HCM patients, the extent of LV scar and fibrosis assessed by LGE in cardiac MR was well correlated with the extent of LA fibrosis and scar ($r = 0.64$). Left atrial fibrosis may be the result of LV remodeling, or both LA and LV fibrosis may be a progression of the same cardiomyopathic process.¹⁷ Therefore, evaluating LA functions in patients with HCM may provide additional information about disease progression. O'Mahony et al¹⁸ used LA diameter, along with other risk factors, as an independent risk factor for SCD in mathematical modeling and was approved by the ESC. In the study of Minami et al⁵ in 564 HCM patients, they found that LA diameter was predictive of SCD in patients without AF. Left atrial diameter was not associated with SCD in patients with AF.⁵ In the study of Nistri et al⁴ in 1419 patients with HCM, left atrium enlargement (LA diameter >48 mm) was associated with

Table 2. The Result of Cox Regression Analysis for Prediction of Appropriate ICD Therapy

Variable	HCM Risk- SCD Score (All Group)			HCM Risk-SCD Score > 6%		
	OR	CI	P	OR	CI	P
PALS	0.944	0.878-1.016	.126	0.806	0.686-0.946	.008
HCM risk-SCD score (%)	1.075	1.007-1.148	.03	1.114	1.011-1.226	.03
GLPS (%)	1.178	1.014-1.368	.03	1.263	1.015-1.571	.02

GLPS, global longitudinal peak strain; HCM, hypertrophic cardiomyopathy; PALS, peak atrial longitudinal strain.

all-cause death, cardiovascular death, and death from heart failure but not with SCD.

Left atrial volume measurements give more information than LA diameter in showing LA functions in various disease states. Although there is no direct study evaluating LA volume for sudden death in HCM patients, it was found that the anteroposterior LA diameter calculated from the LA volume was well correlated with the HCM Risk-SCD score.^{19,20} In our study, no significant difference was found between groups with and without appropriate ICD therapy in both LA diameter and LA volume measurements. This may be due to the fact that the patient profile in our study was high-risk patients who had already had ICD implanted.

Recently, in addition to LA size and volume, LA strain which is evaluated with STE has been used in the evaluation of LA functions. In HCM patients, LA strain may provide additional information in detecting the development of AF or distinguishing hypertension from HCM.^{21,22} In the study of Essayagh et al⁶ in 307 HCM patients, decreased PALS was found to be associated with composite endpoint (SCD, appropriate defibrillator therapy, hospitalization for heart failure, new onset of VT, need for implanted cardioverter defibrillator or pacemaker implantation, AF ablation, alcohol septal ablation, myomectomy, and heart transplantation).⁶

Since the LA diameter, which is one of the contents of HCM Risk-SCD score, affects the total score very little, clinical decision changes are also low. In our study, LA diameter and LA volume were not independently predictive for ICD therapy, whereas PALS was found to be predictive in patients with the HCM Risk-SCD score >6%. There could be several possible reasons. Like female sex, which is risk factor for stroke and embolism in AF patients, LA strain is not a risk factor alone in HCM patients, but it becomes more important in the presence of other risk factors (increased HCM risk score) and may cause arrhythmia development.

In HCM patients, as a result of microvascular ischemia and diastolic dysfunction due to abnormal LV hypertrophy, LV filling pressures increase and cause left atrial dysfunction by causing an increase in left atrial pressure. The enlargement and fibrosis formation, which is called left atrial remodeling, lead to deterioration of LA passive filling or reservoir function that provides LA enlargement.

In our study, decreased PALS indirectly indicates more affected LV in patients with high HCM Risk-SCD scores. Arrhythmic events are more frequent because of the prevalence of fibrosis and scar tissue in the affected LV myocardium.

Peak atrial longitudinal strain predicts ventricular arrhythmic events in patients with high risk, but since ICD implantation is mandatory in the group with a high HCM Risk-SCD Score, there may be an impression that it has no additional clinical benefit. In previous studies, it was determined that the frequency of non-sustained VT was reduced with prophylactic beta-blocker, amiodarone, and sotalol treatment, and it was claimed that the appropriate shock frequency

could be reduced in patients who underwent ICD.²¹⁻²³ In the group with a high HCM Risk-SCD Score and a concomitant decreased PALS, prophylactic medical treatment may contribute to reducing the incidence of appropriate ICD shock and the development of ventricular arrhythmia.

GLPS

In our study, GLPS was found to be an independent predictor for high arrhythmic event and all risk group. In HCM, microvascular dysfunction in the LV, hypertrophy, and disarray in the LV fibers cause myocardial ischemia and fibrosis.²³ Since longitudinal fibers of the heart are susceptible to ischemia and fibrosis, there may be a decrease in GLPS in the early period. In the study of Debonnaire et al¹³, it was found that GLPS in HCM patients was predictive of the occurrence of appropriate ICD therapy. Similar to this study, in our study, it was found that GLPS was predictive for ICD treatment without other risk factors. Decreased GLPS indicates the prevalence of myocardial fibrosis, which predisposes to cardiac arrhythmia and re-entry.²⁴

In patients with an HCM Risk-SCD score in the gray zone (4%-6%), PALS was not associated with appropriate ICD therapy, while HCM Risk-SCD score was associated. Since the number of patients with appropriate ICD shock in the gray zone was low (n=6), further statistical analysis could not be performed. Due to the presence of patients in the gray zone with serious cardiac events in the future, studies involving more patients are needed for optimal treatment of patients in this zone.

Study Limitations

The most important limitation of our study is that the study is a single-center and cross-sectional design and relatively has small number of patients. Further prospective studies with larger number of patients are needed on this issue. Another important limitation is that because patients included in the study were high-risk patients who had already underwent ICD therapy, the results can not be generalized to HCM patients including all risk classes. Genetic analysis could not be performed because of the lack of these facilities in our center. Also, because our center does not have cardiac MRI and the presence of LV aneurysm is not taken into account when implanting an ICD, fewer patients may have had ICD implanted.

CONCLUSION

Peak atrial longitudinal strain was found to be predictive for appropriate ICD therapy in patients with HCM Risk-SCD score > 6% while not predictive for all risk group. Easily measurable PALS may provide additional information in predicting ventricular arrhythmias or deciding on prophylactic medical treatment to prevent ventricular arrhythmias or reduce the frequency of appropriate shock in high-risk patients with ICD implanted.

Ethics Committee Approval: Ethical committee approval was received from the Ethics Committee of Kartal Koşuyolu Training and Research Hospital (approval no: 2017/740).

Informed Consent: Written informed consent was obtained from all participants who participated in this study.

Peer-review: Externally peer-reviewed.

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