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Clinical Utility of Left Atrial Asynchrony and Mechanical Function in Patients with Hypertrophic Cardiomyopathy

Ozkan Candan,[1] Cetin Gecmen,[2] Muzaffer Kahyaoğlu,[2] Zeki Şimsek,[2] Mehmet Çelik,[2] Abdulkadir Uslu[2] and Cevat Kirma[2]

Background: The aim of this study was to examine whether left atrial dispersion and left atrial strain as measured by speckle tracking echocardiography and clinical parameters are predictors of the development of atrial fibrillation in patients with hypertrophic cardiomyopathy.

Methods: A total of 151 patients (69% male, mean age 48.9 ± 14.2 years) with hypertrophic cardiomyopathy were included in the study. The patients' demographic, clinical, electrocardiographic, 2-dimensional classic and speckle tracking echocardiographic data were collected. Atrial fibrillation was identified by 12-lead electrocardiograms or 24-72 hours of Holter recordings during the follow-up period. Atrial dispersion was defined as the standard deviation of time to peak strain in 12 left atrial segments.

Results: During the follow-up period, 40 patients (26%) developed atrial fibrillation. Peak atrial longitudinal strain (16.8 ± 6 vs. 22.1 ± 6.6, $p \leq 0.001$) was significantly lower in the patients who developed atrial fibrillation than in those who did not. However, atrial dispersion was significantly higher in the group which developed atrial fibrillation (61 [46.7,78.6] vs. 41.3 [30.6-51], $p \leq 0.001$). In multivariate Cox regression analysis, atrial dispersion (msn) (hazard ratio: 1.019, 95% confidence interval: 1.004-1.033, p = 0.01), peak atrial longitudinal strain, and age were found to be independent predictors of atrial fibrillation.

Conclusions: In patients with hypertrophic cardiomyopathy, atrial dispersion, peak atrial longitudinal strain and age are predictive of the development of atrial fibrillation. Atrial dispersion measured by a speckle tracking-based method may provide further information on left atrial function in patients with hypertrophic cardiomyopathy or other disease states.

Key Words: Atrial dispersion • Atrial fibrillation • Hypertrophic cardiomyopathy • Speckle tracking echocardiography

INTRODUCTION

Hypertrophic cardiomyopathy is a hereditary disease and the most common cause leading to sudden cardiac death in young people. This disease is characterized by abnormal left ventricular hypertrophy resulting from an abnormal array of myocardial fibers.[1,2] In addition to ventricular arrhythmias in patients with hypertrophic cardiomyopathy, atrial arrhythmias can also cause significant morbidity and impaired quality of life. Left ventricular hypertrophy and impaired myocardial relaxation lead to the development of diastolic dysfunction. Elevated left ventricular pressure is reflected back, causing an increase in left atrial pressure.[3] While increased contractility is observed in the left atrium due to the initial increase in pressure load, during disease

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[1]Cardiology Clinic, Usak University, Faculty of Medicine, Usak; [2]Department of Cardiology, Kartal Kosuyolu Training and Research Hospital, Turkey.
Corresponding author: Dr. Ozkan Candan, Cardiology Clinic, Usak University, Faculty of Medicine, 64300, Usak, Turkey. Tel: +90276 221 2121; Fax: +90276 221 2133; E-mail: oz_candan@hotmail.com

progression, dilatation and fibrosis develop in the left atrium wall over time, a process called atrial remodeling. Structural remodeling contributes to the development of atrial arrhythmias, especially atrial fibrillation, by causing electrical remodeling over time.[4] Previous studies have found that left atrial enlargement, left atrial volume index, and age are predictors of the development of atrial fibrillation in hypertrophic cardiomyopathy patients.[5] In addition to classical echocardiography, the parameters of speckle tracking echocardiography have been introduced in the evaluation of left atrial function. Speckle tracking echocardiography-based left atrial strain has been correlated with fibrosis distribution as evaluated by cardiac magnetic resonance imaging in patients with paroxysmal atrial fibrillation, and it has also been correlated with low voltage areas observed during radiofrequency mapping.[6,7]

Structural changes occurring in the left atrium in hypertrophic cardiomyopathy patients cause electrical dispersion in the left atrium wall, and thus may cause the development of atrial fibrillation. In a study conducted by Kawakami et al. on patients at risk of the development of heart failure or atrial fibrillation, atrial dispersion based on speckle tracking echocardiography evaluations was found to be a predictor of the development of atrial fibrillation.[8]

The aim of this study was to investigate the effects of classical parameters (age, left atrial volume), left atrial strain, and a new echocardiography parameter, left atrial dispersion, on the development of atrial fibrillation in hypertrophic cardiomyopathy patients.

METHODS

Patients diagnosed with hypertrophic cardiomyopathy were screened between 2014 and 20 March 2020 at Kartal Kosuyolu Heart Training and Research Hospital. A total of 217 patients were screened and 170 patients were ultimately selected for inclusion according to 2-dimensional echocardiographic demonstrating an unexplained increase in wall thickness > 15 mm in the absence of abnormal load conditions. The exclusion criteria included having coronary artery disease, severe mitral and aortic valve disease (stenosis or insufficiency), hypertension (systolic blood pressure > 140 mmHg or diastolic blood pressure > 90 mmHg), left ventricular ejection fraction ≤ 50%, chronic obstructive pulmonary disease, and severe thyroid disease.

The patients' baseline clinical data, electrocardiograph (ECG), symptom status and medical history were obtained from hospital records. All patients' baseline Holter monitoring records and echocardiographic images were obtained from the electronic archive of the hospital. Follow-up evaluations were performed with data obtained from the records of the cardiology department with 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 national medical archive (E-pulse system). Clinical data, ECG and echocardiography images of the patients at the time of first admission were obtained and analyzed. All patients were followed up until an event occurred or until March 2020. Nine patients who were unable to attend follow-up visits and 10 patients with poor echogenicity were excluded from the study. Finally, a total of 151 patients were enrolled in the study.

At each examination, standard 12-lead ECG was performed, and the patient was asked about symptoms. 24-72 hours of Holter recordings were made any time the patient had any symptoms suggesting atrial fibrillation. Atrial fibrillation was defined as a standard 12-lead ECG recording or a single-lead ECG tracing of > 30 s showing heart rhythm with no discernible repeating P waves and irregular RR intervals (when atrioventricular conduction was not impaired).[9] Basal clinical and echocardiographic parameters of the patients who developed atrial fibrillation were compared with those of the patients who did not develop atrial fibrillation.

HCM Risk-SCD scores were calculated using an online calculator in line with the European Society of Cardiology (ESC) guidelines based on the clinical and echocardiographic data of the patients.[10] Informed written consent was obtained from all study subjects, and the study protocol was approved by the Ethics Committee of Kartal Kosuyolu Training and Research Hospital.

Echocardiography

Two-dimensional and Doppler 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 60-80 frames per second for 2-dimensional image acquisition. Settings were adjusted manually to obtain optimal images. All data was transferred to a workstation for further offline analysis (EchoPAC PC; GE Vingmed Ultrasound AS).

Maximal wall thickness was measured from all left ventricular segments from base to apex in parasternal short axis view. Left ventricular end-systolic diameter and end-diastolic diameter were measured from the parasternal long axis view according to recommendations of the American Society of Echocardiography.[11] Left ventricular end-systolic volume , end-diastolic volume and ejection fraction were determined from apical four and two-chamber views using Simpson's modified biplane method. Atrial diameter was calculated by M-mode or 2-dimensional echocardiography in the parasternal long axis plane. Left atrial volume was obtained using the biplane area length method from apical four and two-chamber images at end-systole, and it was also indexed to body surface area as recommended.[12]

Speckle tracking echocardiography

All measurements used in this analysis were made offline by a single investigator who was blinded to the clinical data. Left ventricular longitudinal strain was obtained from apical 4-chamber, 3-chamber and 2-chamber views. The frame rate for images was adjusted between 60-80 frames/s. 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 left ventricle was divided into 6 segments in each view automatically by the software. The global value of the longitudinal strain was calculated by the software from the sum of the changes in individual segments.

Apical 4-chamber and 2-chamber views were used for left atrial strain measurements. For 2 dimensional speckle tracking echocardiography analysis, a line was manually drawn along the left atrial endocardium when the left atria was at its minimum volume after contraction. The software then automatically generated additional lines near the atrial epicardium and mid-myocardial line, with a region of interest with a default width of 15 mm. Before processing, a cine loop preview feature visually confirmed that the internal line followed the left atrial endocardium throughout the cardiac cycle. If tracking of the left atrial endocardium was unsatisfactory, manual adjustments or changing software parameters (e.g. region of interest size or smoothing function) was performed. The software divided the left atrial endocardium into 6 segments. Segments in which no adequate image quality could be obtained were rejected by the software and excluded from the analysis. Left atrial peak strain just before mitral valve opening was taken as peak atrial longitudinal strain, and left atrial strain just before atrial contraction (onset of the P-wave on electrocardiography) was taken as peak atrial contraction strain. QRS wave was taken as a reference for the evaluation of left atrial (LA) strain. Overall, 1812 segments were analyzed (12 segments for each patient), and a total of 3.9% of the segments were excluded.

On the left atrial strain curve, the time from the beginning of the QRS complex to peak strain (reservoir strain) was measured for each segment. Left atrial mechanical dispersion was defined as the standard deviation of time to peak positive strain from the 12 left atrial segments. Higher values of standard deviation are thought to suggest a greater degree of left atrial dispersion.[8]

Statistical analysis

Patient characteristics were expressed as mean ± standard deviation or as percentages. Continuous variables in two groups were compared using the Student's t test or Mann-Whitney U test. Distributions of categorical variables were analyzed using the $\chi 2$ or Fisher's exact test when appropriate. Pearson's correlation analysis was used to test relationships between continuous variables. Significant parameters in the univariate analysis ($p \leq 0.05$) were included in the multivariate analysis. Multivariate Cox regression analysis (enter models) was used to determine the independent predictors of atrial fibrillation. Receiver operating characteristic (ROC) curves were plotted and areas under the ROC curves (AUCs) were calculated to predict the occurrence of atrial fibrillation. All statistical analyses were performed with SPSS version 24.0 (SPSS, Inc, Chicago, IL), and p values < 0.05 were considered to be statistically significant.

Reproducibility

Inter- and intra-observer reproducibility was assessed for peak atrial longitudinal strain and atrial dispersion. For intra- and inter-observer assessments, the measurements were re-analyzed for 15 patients. The intra- and inter-observer intraclass correlation coefficients were 0.87 (0.62-0.95) and 0.91 (0.74-0.97), respectively, for left atrial dispersion measurements, and 0.90 (0.70-0.96) and 0.93 (0.78-0.97), respectively, for measurements of peak atrial longitudinal strain (PALS).

RESULTS

Nine patients who were unable to attend follow-up visits and 10 patients with poor echogenicity were excluded from the study. In total, 151 patients with hypertrophic cardiomyopathy (69% male, mean age 48.9 ± 14.2 years) were included in the study. The echocardiographic and clinical characterizations of the study population are summarized in Table 1.

During a follow-up period of 5 years (mean follow-

Table 1. Echocardiographic and clinical characteristic of the study population

Variable	All patiens (n = 151)	AF (+) (n = 40)	AF (-) (n = 111)	p
Age (years)	48.9 ± 14.2	55.1 ± 13.6	46.7 ± 13.7	0.001
Sex male (%)	104 (69%)	29 (73%)	75 (67%)	0.35
Body surface area (m^2)	1.8 ± 0.15	1.80 ± 0.17	1.81 ± 0.14	0.7
Systolic blood pressure (mmHg)	124.2 ± 7.3	124.8 ± 7.5	124 ± 7.3	0.6
Diastolic blood pressure (mmHg)	73 ± 8.5	75.4 ± 7.3	72.3 ± 8.9	0.06
Heart rate (bpm)	68.2 ± 8.4	65 ± 8.3	69.2 ± 8.4	0.02
Maximal wall thickness (cm)	2.38 ± 0.5	2.37 ± 0.5	2.38 ± 0.5	0.8
LV mass index (g/m^2)	190.4 ± 35	192.6 ± 35.7	189.6 ± 34.8	0.6
Mitral regurgitation (%)	62 (40%)	15 (38%)	47 (42%)	0.7
Systolic anterior motion (%)	58 (38%)	16 (40%)	42 (38%)	0.5
LVEF (%)	63.2 ± 7.1	64.3 ± 4.2	62.8 ± 7.8	0.2
LA diamater (cm)	4.1 ± 0.5	4.1 ± 0.6	4.1 ± 0.5	0.7
LAVi (mL/m^2)	44.1 ± 11.7	44.7 ± 12.5	43.9 ± 11.5	0.7
E (m/s)	0.9 ± 0.2	0.97 ± 0.16	0.97 ± 0.23	0.9
A (m/s)	1.01 ± 0.35	1 ± 0.4	1 ± 0.33	0.8
E′ (cm/s)	7 ± 2	7.3 ± 1.7	7.5 ± 2	0.7
A′ (cm/s)	5.9 ± 1.5	5.7 ± 1.5	6 ± 1.5	0.3
E/E′	13.8 ± 4.1	13.8 ± 3.5	13.7 ± 4.3	0.9
Beta blocker	119 (79%)	31 (78%)	88 (79%)	0.5
Ca channel blocker	34 (23%)	11 (28%)	23 (21%)	0.3
ACE inhibitors/ARB	12 (8%)	3 (7.5%)	9 (8.1%)	0.9
Diuretics				
Dyspyromide	15 (9.9%)	11 (9.9%)	4 (10%)	0.9
DM	14 (9.3%)	4 (10%)	10 (9%)	0.8
CKD	6 (4%)	2 (5%)	4 (3.6%)	0.7
NYHA I	97 (64%)	28 (70%)	69 (63%)	0.4
NYHA II	44 (29 %)	11 (27.5%)	33 (29.7%)	
NYHA III	10 (7%)	1 (2.5%)	9 (8.1%)	
Peak LVOT gradient (mmHg)	52 [24,80]	57.5 [25,7,84.2]	51 [23,79]	0.45
Obstructive HCM (%)	104 (69%)	29 (73%)	75 (68%)	0.3
HCM Risk-SCD score (%)	5.4 [3,7.9]	4.4 [2.5,8.9]	5.5 [3.4,7.3]	0.5
Unexplained syncope, n (%)	42 (28%)	16 (40%)	26 (23%)	0.06
NsVT, n (%)	68 (45%)	18 (45%)	50 (45%)	0.5
Family history of SCD (%)	67 (44%)	15 (38%)	52 (47%)	0.3
PALS (%)	20.7 ± 6.9	16.8 ± 6	22.1 ± 6.6	< 0.001
PACS (%)	7.3 ± 2.7	6.8 ± 2.7	7.5 ± 2.7	0.19
GLPS (%)	-12.8 ± 3.9	-12 ± 5.3	-13.1 ± 3.5	0.13
Atrial dispersion (msn)	44.3 [35.2,58.9]	61 [46.7,78.6]	41.3 [30.6-51]	< 0.001

A, peak late filling transmitral velocity; A′, peak longitudinal late diastolic tissue velocity of the mitral valve annulus; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; CKD, chronic kidney disease; DM, diabetus mellitus; E, peak early filling transmitral velocity; E′, peak longitudinal early diastolic tissue velocity of the mitral valve annulus; E/E′, ratio of peak early mitral inflow velocity and peak early diastolic mitral annular velocity; GLPS, global longitudinal peak strain; HCM Risk-SCD score, hypertrophic cardiomyopathy sudden cardiac death risk score; LA, left atrial; LAVi, left atrial volume index; LVEF, left ventricular ejection fraction; LVOT, left ventricular outflow tract; NsVT, non sustained ventricular tachycardia; PACS, peak atrial contraction strain; PALS, peak atrial longitudinal strain; SCD, sudden cardiac death.

up duration: 49.5 ± 11.3 months), 40 patients (26%) developed atrial fibrillation (paroxysmal atrial fibrillation 13, persistent atrial fibrillation 27) on standard 12-lead ECG or during their 24-72 hour Holter recordings.

At follow-up, the patients who developed atrial fibrillation were older than the patients without atrial fibrillation (55.1 ± 13.6 vs. 46.7 ± 13.7, p = 0.001) (Table 1). Compared to the patients who did not develop atrial fibrillation, those who developed atrial fibrillation had a statistically lower heart rate (65 ± 8.3 vs. 69.2 ± 8.4, p = 0.02) and lower global peak atrial longitudinal strain (%) (16.8 ± 6 vs. 22.1 ± 6.6, p ≤ 0.001), however there was no statistically significant difference in peak atrial contraction strain (%) (6.8 ± 2.7 vs. 7.5 ± 2.7 p = 0.19) (Table 1, Figure 1).

Atrial dispersion obtained from the standard deviation of 12 segments was higher in the atrial fibrillation group (61 [46.7-78.6] vs. 41.3 [30.6-51], p ≤ 0.001) (Table 1, Figure 2A-B). However, it was not associated with the type of atrial fibrillation (persistent or paroxysmal) (67.9 ± 26 msn vs. 60.8 ± 15.6 msn, p = 0.29). Left atrial diameter (4.1 ± 0.6 vs. 4.1 ± 0.5, p = 0.7), left atrial volume index (44.7 ± 12.5 vs. 43.9 ± 11.5, p = 0.7), E/E′ (13.8 ± 3.5 vs. 13.7 ± 4.3, p = 0.9), and left ventricular mass index were similar between the groups with and without atrial fibrillation (Table 1).

In correlation analysis, there was a moderate inverse correlation between atrial dispersion (msn) and peak atrial longitudinal strain (%) (r = -0.28, p = 0.001), while there were no correlations between atrial dispersion (msn) and age (years), left atrial diameter (cm), left atrial volume index (mL/m^2), and global longitudinal peak strain (%), respectively (Table 2).

Multivariate Cox regression analysis including atrial dispersion, peak atrial longitudinal strain, age and heart rate was used to determine the independent predictors of atrial fibrillation development during follow-up. Atrial dispersion (msn) [hazard ratio (HR): 1.019, 95% confidence interval (CI): 1.004-1.033, p = 0.01], age (years) (HR: 1.03, 95% CI: 1.002-1.059, p = 0.03) and peak atrial longitudinal strain (HR: 0.934, 95% CI: 0.872-0.999, p = 0.05) were found to be independent predictors of atrial fibrillation development (Table 3).

In ROC curve analysis, atrial dispersion > 44.7 msn

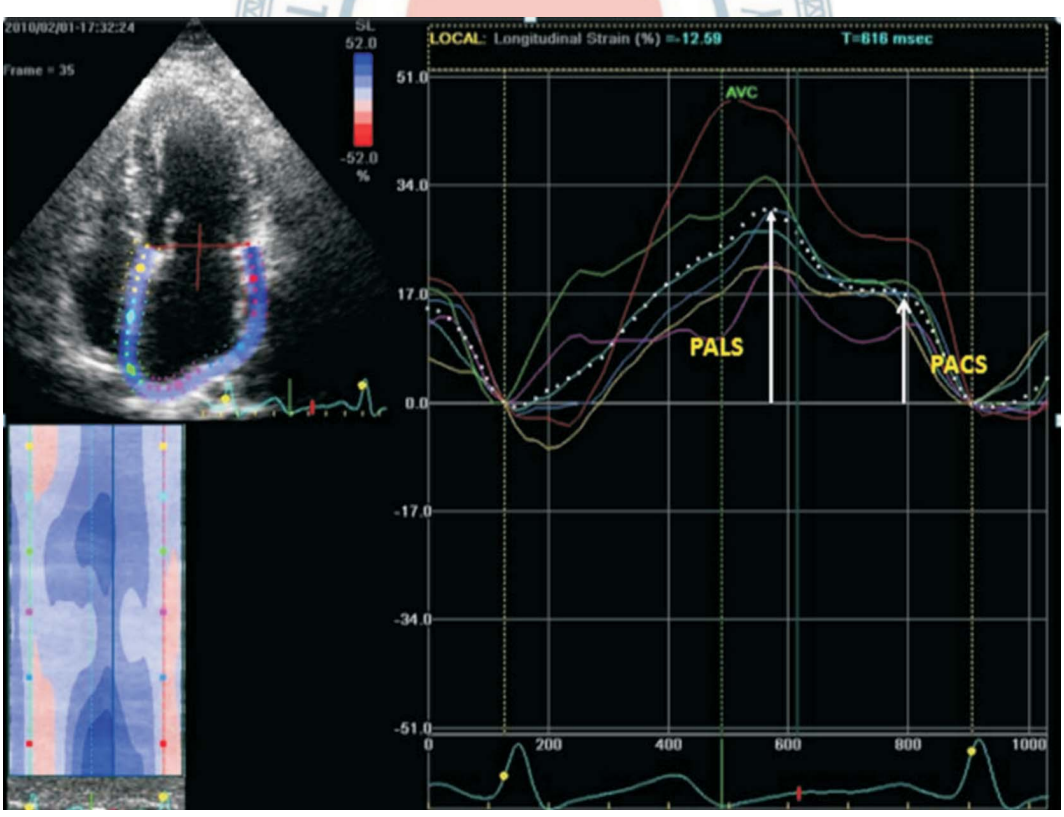

Figure 1. *Left atrial longitudinal strain parameters. PACS, peak atrial contraction strain; PALS, peak atrial longitudinal strain.*

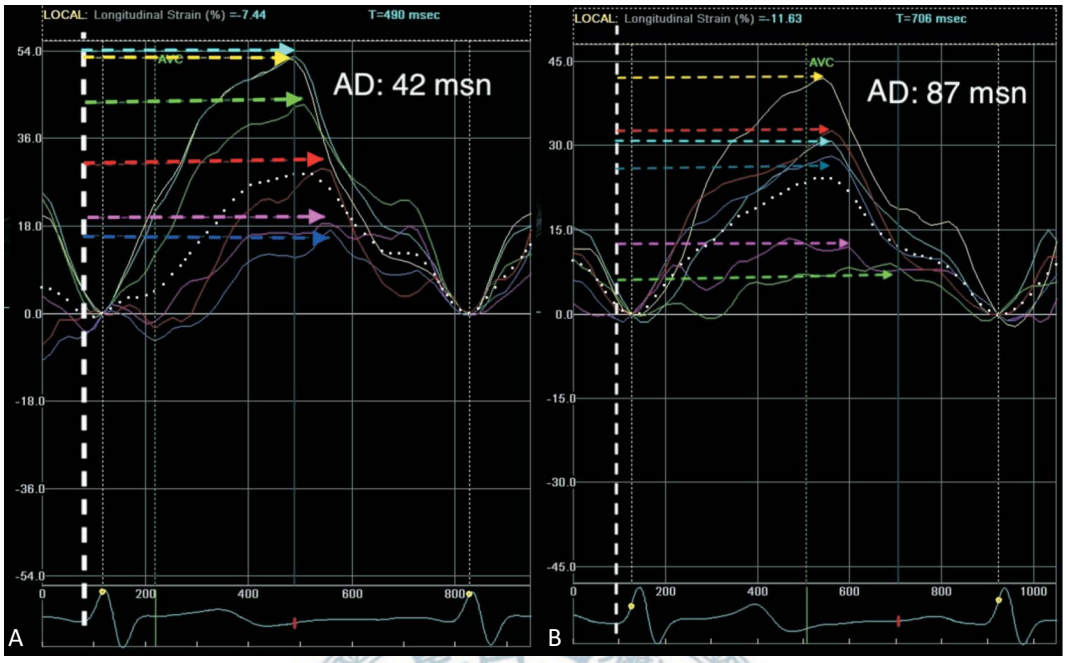

Figure 2. *Speckle-based left atrial dispersion of without atrial fibrillation (A) and with atrial fibrillation (B). AD, atrial dispersion.*

Table 2. Correlation of atrial dispersion

Variable	r	p
Age, years	-0.021	0.8
PALS, %	-0.28	0.001
LA diamater, cm	0.08	0.3
LAVi, mL/m^2	0.03	0.7
GLPS, %	0.07	0.4
HCM Risk-SCD, %	0.03	0.6

GLPS, global longitudinal peak strain; LA, left atria; LAVi, left atrial volume index; PALS, peak atrial longitudinal strain.

Table 3. The results of Cox regression analysis for prediction of atrial fibrillation

Variable	B	Exp (B)	CI	p
Age, years	0.030	1.030	1.002-1.059	0.03
PALS, %	-0.068	0.934	0.872-0.999	0.05
Atrial dispersion (msn)	0.018	1.019	1.004-1.033	0.01
Heart rate	-0.036	0.965	0.928-1.003	0.07

CI, confidence interval; PALS, peak atrial longitudinal strain.

predicted the development of atrial fibrillation with 87.5% sensitivity and 64% specificity [area under curve (AUC) = 0.809, $p \leq 0.001$, 95% CI: 0.73-0.88]. In addition, PALS < 17.2% predicted the development of atrial fibrillation with a sensitivity of 74%, specificity of 73%, and AUC of 0.738 ($p \leq 0.001$) (Figure 3).

DISCUSSION

In the present study, atrial mechanical dispersion was found to be prolonged in patients with hypertrophic cardiomyopathy who developed atrial fibrillation. In addition to prolonged mechanical dispersion, older age and increased peak atrial longitudinal strain were also found to be predictors of the development of atrial fibrillation.

Previous studies have reported an annual incidence of atrial fibrillation development in hypertrophic cardiomyopathy patients of 2-4%; this incidence is 20-30% throughout life, and it can increase up to 40% in patients older than 70 years.[13,14] In our study, the rate was 26%. This lower rate may have been due to our relatively younger patient population and the shorter length of the follow-up period.

Compatible with previous studies, age was found to be a predictor of atrial fibrillation in our study.[15] The occurrence of structural changes in the atrial wall with advancing age and along duration of untoward effects of hypertrophic cardiomyopathy acting on the left atrial wall contribute to the development of atrial fibrillation by inducing fibrosis in the atrial wall. Advanced age is the most important risk factor in patients with hypertro-

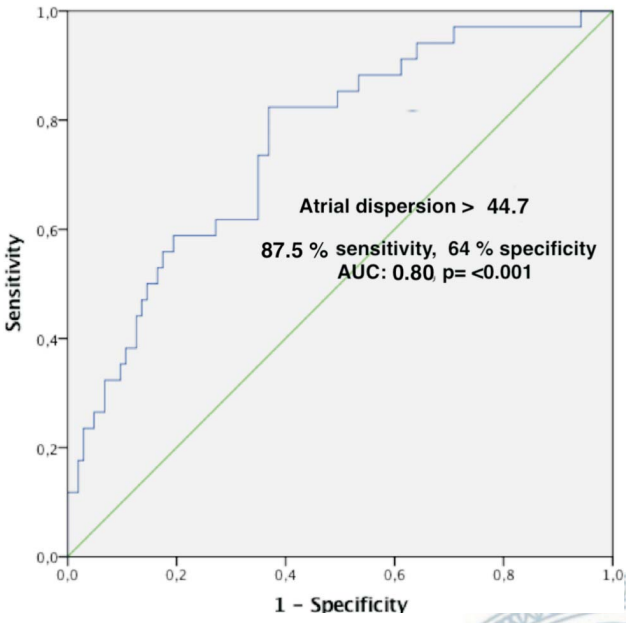

Figure 3. *The ROC curve analysis of atrial dispersion. AUC, area under curve; ROC, receiver operating characteristic.*

phic cardiomyopathy, as it is in the normal population.

Previous studies have demonstrated that global peak atrial longitudinal strain, which reflects passive stretching of the left atrium during left ventricular systole, is an accurate measurement of left atrial reservoir function.[16-18] There are slight differences in mean left atrial reservoir strain values among the studies performed with HCM patients. In our study, the PALS value was 20.7 ± 6.9%, and this value has generally been reported to vary between 20-24% in previous studies.[19-21] The difference may be due to differences in the ages of the included patients, left atrial diameter and volume differences and other clinical situations. Further studies including more patients are needed to identify PALS reference values in HCM patients.

Decreased peak atrial longitudinal strain reflects distorted atrial function and may indicate a predisposition towards the development of atrial fibrillation. Previous studies have determined that peak atrial longitudinal strain is predictive of the development of atrial fibrillation with hypertrophic cardiomyopathy.[21,22] In a study by Debonnaire et al. on atrial fibrillation in HCM patients the mean left atrial reservoir strain was 18.6 ± 7.5%, while the reservoir strain was more impaired in the study by Zegkos et al. (16.4 ± 5.4%) and our study (16.8 ± 6%).[21,23] This difference may be attributed to older patients with atrial fibrillation in our study and the other study. In a study by Kao et al., while left atrial reservoir strain [median 11 (10, 13)] and contraction strain were found to be more impaired in patients who developed atrial fibrillation, conduit function calculated from left atrial volume was found to be significantly associated with the development of AF. This study included very few patients (n = 15), and only patients with apical HCM were included.[24]

In accordance with previous studies, our study also showed that global peak atrial longitudinal strain was predictive over a longer period of observation. Decreased peak atrial longitudinal strain reflects distorted atrial function and may indicate a predisposition towards the development of atrial fibrillation.

Mechanical contraction occurs a short time following electrical conduction, enabling the heart to work synchronously. In various disease states that lead to fibrosis, left ventricular electrical synchronization is disrupted, called asynchrony, which may potentially contribute to mechanical dispersion and the occurrence of arrhythmic events. Previous studies have found that ventricular mechanical dispersion is associated with arrhythmogenic right ventricular dysplasia, a long QT interval, and ischemic cardiomyopathy, and in hypertrophic cardiomyopathy patients, with ventricular arrhythmic events.[25-28] Conditions that affect not only the left ventricle but also left atrium (pressure or volume overload) may induce fibrosis in myofibrils in the left atrial wall, impairing electrical and mechanical synchronization. Atrial dispersion can be evaluated by speckle tracking echocardiography-based methods, since tissue Doppler imaging-based methods are angle-dependent and affected by mechanical withdrawal. In the study conducted by Kawakami et al. on community-based participants with a potential to develop heart failure and atrial fibrillation, atrial dispersion was found to be increased in patients who developed atrial fibrillation compared to those who did not develop atrial fibrillation, and atrial dispersion was found to be an independent predictor of atrial fibrillation development.[8] Atrial dispersion was shown to be predictive of the development of atrial fibrillation in a normal population with normal left atrial size.[29] Atrial dispersion has also been reported to be increased in patients with paroxysmal atrial fibrillation and to be associated with recurrence of atrial fibrillation in the first year after radiofrequency ablation.[30] Another

study reported that atrial dispersion which was increased in patients undergoing direct current cardioversion due to atrial fibrillation, decreased after direct current cardioversion.[31] In the study by Kupczynska et al., atrial dispersion was found to be an independent predictor of thrombus formation in the left atrial appendage in patients in whom transesophageal echocardiography was performed because of atrial fibrillation.[32] However, in a study by Rasmussen et al. on paroxysmal atrial fibrillation patients, atrial dispersion was not associated with ischemic stroke.[33]

In our study, atrial dispersion was found to predict the development of atrial fibrillation in hypertrophic cardiomyopathy patients. Elevated left ventricular filling pressures due to left ventricular hypertrophy in hypertrophic cardiomyopathy patients cause structural changes including fibrosis, called atrial remodeling, in the left atrial wall due to increased pressure. While structural and electrical remodeling potentially causing electrical heterogeneity lead to nonuniform conduction velocities and inhomogenous refractory periods in the atrial myocardium, increased electrical remodeling may contribute to further progression of structural remodeling. The resulting electrical asynchrony or dispersion and electromechanical dysfunction may lead to the development of atrial fibrillation.[4]

Clinical implication

Early detection of patients who may develop atrial fibrillation and intensification of medical treatments can delay or prevent the development of atrial fibrillation. Some patients with ischemic stroke have atrial fibrillation that cannot be detected by cardiac examination, Holteror routine monitoring. Undetected asymptomatic atrial fibrillation increases the risk of developing ischemic stroke. Since increased left atrial dispersion may show sensitivity to atrial fibrillation in these patients, it may contribute to the earlier initiation of anticoagulation therapy.

Limitations

One of the most important limitations of our study is that it was conducted in a single center and the number of patients was relatively small. In patients with asymptomatic and/or paroxysmal atrial fibrillation, our method of detecting atrial fibrillation with Holter analysis or symptom-based ECG may not show the true frequency, and its rate may be underestimated. In our study, the course of mechanical dispersion over time was not investigated in patients with or without atrial fibrillation. Therefore, the effect of the time of exposure to atrial fibrillation on mechanical dispersion is unknown, and further studies are needed to elucidate this issue. Patients recovering after atrial fibrillation ablation or cardioversion may experience improvement in mechanical dispersion. Due to the small number of patients, it was not studied statistically. Other important limitations are that speckle-based methods are angle-dependent and give suboptimal results in patients with poor image quality. Because software for the left atrium was not available, software for the evaluation of the left ventricle was used. Cardiac MRI and voltage mapping, which could detect atrial fibrosis more accurately, were not used.

CONCLUSIONS

In patients with hypertrophic cardiomyopathy, atrial dispersion, age and peak atrial longitudinal strain are predictors of the development of atrial fibrillation. Atrial dispersion measured by a speckle tracking-based method may provide further information on left atrial function in patients with hypertrophic cardiomyopathy or other disease states.

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DECLARATION OF CONFLICT OF INTEREST

All the authors declare no conflict of interest.

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