Left Atrial Dispersion Predicts Atrial Fibrillation in Patients with Hypertrophic Cardiomyopathy: A 5-year Follow-up study

OZKAN CANDAN[1], Ender Cakmak[2], CETIN GECMEN[3], Muzaffer Kahyaoglu[4], Zeki Şimşek[5], Mehmet Celik[5], Ahmet Karaduman[1], Kamil Gülşen[1], and Cevat Kirma[1]

[1]Kartal Kosuyolu Training and Research Hospital
[2]Kartal Kosuyolu Heart Education and Research Hospital
[3]KOSUYOLU HEART&RESEARCH HOSPITAL
[4]Umraniye EAH
[5]Kartal Kosuyolu Yuksek Ihtisas Egitim ve Arastirma Hastanesi

July 20, 2020

Abstract

Purpose:The aim of our study was to examine whether left atrial dispersion and left atrial strain as measured by speckle tracking echocardiography and clinic paramaters are predictors for the development of atrial fibrillation (AF) in patients with hypertrophic cardiomyopathy. Methods: A total of 137 patients (70% male, mean age 49.6 ± 14.2 years) with HCM were included in the study. Patients' clinical, electrocardiographic, 2D classic and speckle tracking echocardiographic (STE) data were collected. AF was searched by 12-lead electrocardiograms or 24-hour Holter recordings during follow-up period. Atrial dispersion was defined as the standard deviation of time to peak strain in 12 left atrial segments Results: During a follow-up period of 5 years 37 patients (16.9%) developed AF. At follow-up, the patients with occurrence of AF were older than in patients without AF. Atrial dispersion was observed to be higher in the AF developing group (61.4 ± 23.2 vs 43.1±15.8, p=<0.001). The multivariate in Cox regression analysis (including atrial dispersion, PALS, age, LA) atrial dispersion (msn) (HR 1.017, 95% CI: 1.001-1.03, p= 0.035) and age were found to be independent predictors of AF occurrence. In the ROC analysis atrial dispersion > 44.7 msn predicted occurrence of AF with 82.4% sensitivity and 64 % specificity. Conclusion: In patients with hypertrophic cardiomyopathy, atrial dispersion and age are predictive of the development of atrial fibrillation. Atrial dispersion measured by the speckle tracking-based method may provide further information in evaluating left atrial functions in patients with hypertrophic cardiomyopathy or other disease states

Introduction

Hypertrophic cardiomyopathy (HCM) 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 resulted from abnormal array of the myocardial fibers[1,2]. Ventricular arrhythmias due to irregular arrangements of myofibrils are not only an important cause of mortality in HCM patients but also consequent atrial arrhythmias may result in morbidity and impaired quality of life [3,4]. 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. Especially in HCMs accompanied by obstructive and/or systolic anterior motion (SAM)-related mitral regurgitation, the impairment in left atrial functions is more pronounced [5]. While increased contractility is observed in the left atrium due to the initial increase in the pressure load; during disease progression, dilatation and fibrosis develop in the left atrium wall over time, called atrial remodeling. Structural remodeling contributes to the development of atrial arrhythmias, especially atrial fibrillation (AF), by causing electrical remodeling over time [6]. Previous

studies have found out that left atrial enlargement, left atrial volume index (LAVi), and age are predictive of the development of AF in HCM patients[7]. In addition to the classical echocardiography parameters, the parameters of speckle tracking echocardiography (STE) have been started to be used for the evaluation of left atrial functions. In the paroxysmal atrial fibrillation (PAF) patient group; STE-based left atrial strain was correlated with fibrosis distribution as evaluated by cardiac magnetic resonance imaging (MRI) and, in another study, it was correlated with low voltage areas observed during radiofrequency mapping [8,9].

Myocardial electrical dispersion has been found to be predictive of the development of ventricular arrhythmias in HCM patients as well as in other patient populations. [10,11]. In a study conducted by Kawakami et al. on patients at risk for the development of heart failure or AF, atrial dispersion based on STE evaluations was found to be predictive of the development of AF [12].

Structural changes occurring in the left atrium in HCM patients cause electrical dispersion in the left atrium wall and may cause the development of atrial fibrillation. In our study; we investigated the effects of classic parameters (age, the left atrial volume), left atrial strain, and left atrial dispersion; which is a new echocardiography parameter, on AF development in HCM patients.

Methods:

A total of 156 patients were included in the study at Kartal Kosuyolu Training and Research Hospital and between 2015 and 2020. Patients' clinical, echocardiographic and 24-48 hours holter monitoring data were obtained.

The inclusion criteria consisted of 2-D echocardiographic demonstration of an unexplained increase in wall thickness >15 mm in the absence of abnormal load conditions, while 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 EF [?] 50 and chronic obstructive pulmonary disease. Nine patients who could not come to follow-up appointments and 10 patients with poor echocardiographic view were excluded from the study. Finally, 137 patients were enrolled in the study.

AF was defined as an irregularly irregular heart rate without any detectable P waves along with fibrillation f waves on 12-lead electrocardiograms or holter recordings. After inclusion all patients were seen every 6 months at an outpatient clinic and at any time they reported symptoms. At each examination, standard 12-lead ECG was used, and an inquiry was made about symptoms. A 24-hour Holter recording was made at any time the patient had any symptoms suggesting AF.

SCD Risk Score evaluation was calculated with an online calculator in line with the ESC guidelines based on the clinical and echocardiographic data of the patients [13]. Informed written consent was obtained from all study subjects, and the study protocol was approved by the institutional ethics committee.

Echocardiography:

1- Two-dimensional and Doppler Echocardiography

All echocardiographic studies were performed using a Vivid 9 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 60-80 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).

Maximal wall thickness was measured from all LV segments from base to apex in parasternal short axis view. Left ventricular end-systolic diameter (ESD) and end-diastolic diameter (EDD) were measured from parasternal long axis view according to recommendations of American Society of Echocardiography (ASE) [14]. Left ventricular end-systolic volume (ESV), end-diastolic volume (EDV) and EF were determined from the apical four and two-chamber views using Simpson's modified biplane method. Atrial diameter was calculated by M-mode or 2-D echocardiography in the parasternal long axis plane. Left atrial (LA) 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 (BSA) as recommended [15].

2- **Speckle Tracking Echocardiography (STE)**

All measurements used in this analysis were made offline by a single investigator, blinded to the clinical data. The longitudinal strain was obtained from apical 4-chamber, 3-chamber and 2- chamber views. Apical 4-chamber and 2 view was used for LA strains measurements. The frame rate for images was adjusted between 60-80 frames/s. For 2 dimensional STE analysis, a line was manually drawn along the LA endocardium when the LA 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 default width of 15 mm. Before processing, a cine loop preview feature visually confirmed that the internal line follows the LA endocardium throughout the cardiac cycle. If tracking of the LA endocardium was unsatisfactory, manual adjustments or changing software parameters (e.g. region of interest size or smoothing functions) was made. The software divided the LA endocardium into 6 segments. Segments in which no adequate image quality could be obtained were rejected by the software and excluded from the analysis. Overall, 1644 segments were analyzed (12 segments for each patient), and a total of 4.0 percent segments were excluded. LA peak strain just before mitral valve opening was taken as peak atrial longitudinal strain (PALS) and LA strain just before atrial contraction (onset of the P-wave on electrocardiography) was taken as peak atrial contraction strain (PACS), and conduit strain was obtained by subtracting the value of the peak atrial contraction strain from that of the peak atrial longitudinal strain.

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

Statistical analysis:

Patient characteristics were expressed as the mean ± standard deviation or as percentages. Continuous variables in two groups were compared by student t test or Man Whitney U test. Distribution of categorical variables were analyzed by using $\chi 2$ or Fisher's exact test when appropriate. Pearson's correlation was used to test the relationship between the continuous variables. Significant parameters in the univariate analysis ($p= < 0.05$) were included in the multivariate analysis. Multivariate Cox regression analysis (enter models) was used to determine independent predictors of AF. Receiver operating characteristic (ROC) curves were plotted and area under the curve (AUC) was calculated for the prediction of occurrence of atrial fibrillation. All statistical analyses were performed with SPSS version 24.0 (SPSS, Inc, Chicago, IL), and P values of <0.05 were considered as statistically significant.

Results:

12 patients unable to attend follow-up visits and 10 patients having poor echogenicity were excluded from the study. In total, 137 patients with hypertrophic cardiomyopathy (70 % male, mean age 49.6 ± 14.2 years) were included in the study. Echocardiographic and clinical characterizations of study populations are summarized in table 1.

During a follow-up period of 5 years (mean follow-up duration, 48.9±11.4 months) 37 patients (16.9%) developed AF on standard 12-lead ECG or during their 24-hour Holter recordings.

At follow-up, the patients with occurrence of AF were older than in patients without AF (56.7±13.1vs. 47.6±13.9, p= 0.001) (Table 1).

Compared to the patients who did not develop AF, those who developed AF showed statistically lower global PALS (%) (18.8±7.6 vs. 22.2±6.6, p= 0.008) and global conduit strain (%) (12±5.6 vs. 14.6±5.6, p=0.023), while there was no statistically significant difference in PACS % (6.7±2.7 vs. 7.5±2.7 p= 0.14) (Table 2) (Figure 1).

The atrial peak time of mid septum (msn) (418.5 ± 70.1 vs 390.1 ± 73.9, p= 0.04) and of the apical septum (msn) (441.7 ± 73.4 vs 405.2 ± 77.9, p= 0.016), mid lateral (msn) (501.4 ± 83.8 vs 461.7 ± 77.6, p= 0.012) and apical lateral (msn) (507.3± 77.9 vs. 470.7.5±78.8, p= 0.02) segments were longer in the group of patients, in whom AF developed. The peak atrial time was not statistically significantly different in the other segments between the two groups. Atrial dispersion obtained from the standard deviation of the 12 segments was observed to be higher in the AF developing group (61.4 ± 23.2 vs 43.1±15.8, p=<0.001) (Table 2) (Figure 2,3). LA diameter (4.2 ± 0.6 vs 4.1 ± 0.5, p = 0.1) and LAVi (43.9±10.6 vs. 44.4±11, p= 0.8) were similar in the AF developing group and in the group with no AF (Table 1).

In correlation analysis, there was a moderate inverse correlation between artial dispersion(msn) and PALS (r: -0.29, p=0.001) and There were no correlations between atrial dispersion (msn) and age (years), LA diamater (cm), LAVi (mL/m^2), GLPS (%), respectively (Table 3).

The multivariate Cox regression analysis, including atrial dispersion, PALS, age, LA diamater was used to determine independent predictors of AF occurrence during the follow-up. Atrial dispersion (msn) (HR 1.017, 95% CI: 1.001-1.03, p= 0.035) and age (years) (HR 1.03, 95% CI: 0.986-1.047, p= 0.045) were found to be independent predictors of AF occurrence (Table 4).

In the ROC analysis atrial dispersion > 44.7 msn predicted occurrence of AF with 82.4% sensitivity and 64 % specificity (AUC: 0.75, p= <0.001 CI: 0.66-0.84). (Figure 4)

Discussion:

In the present study, atrial mechanical dispersion was found to be prolonged in patients who developed atrial fibrillation with HCM. In addition to prolonged mechanical dispersion, increased age was found to be predictive for the development of atrial fibrillation.

Previous studies have shown that annual AF development incidence in HCM patients is 2-4%; this incidence is 20-30% throughout life, and it can increase to rates of up to 40% in patients older than 70 years old.[16,17]. In our study, this rate was found to be 16.9%. This lower rate might be caused by our relatively younger patient population and the shorter length of the follow-up period.

Compatible with previous studies, age has been found out to be predictive of AF in our study. [18]. The occurrence of structural changes in the atrial wall with advancing age and the long duration of untoward effects of HCM acting on the LA wall contribute to the development of AF by inducing fibrosis in the atrial wall. Advanced age is the most important risk factor in the HCM population as it is in the normal population.

In previous studies, the LA strain was found to be an independent predictor of the development of AF in HCM patients[19,20]. In our study; although LA strain was found to be lower in the group, in which AF developed, a statistically significant difference was not observed between the groups in the regression analysis. This is probably because of the relatively small number of patients participating in our study.

After a short time following the electrical conduction, mechanical contraction occurs enabling the heart to work synchronously. In various disease states that lead to fibrosis in LV, electrical synchronization is disrupted called asynchrony; which may potentially contribute to mechanical dispersion and the occurrence of arrhythmic events. Previous studies have found out that ventricular mechanical dispersion is associated with arrhythmogenic right ventricular dysplasia (ARVD), a long QT interval, and ischemic cardiomyopathy, and, in HCM patients, with ventricular arrhythmic events [11,21-23]. Conditions that affect not only LV but also LA (pressure or volume overload) may induce fibrosis in myofibrils in the LA wall, impairing the electrical and mechanical synchronization. Atrial dispersion can be evaluated by STE-based methods since TDI-based methods are angle-dependent and affected by mechanical withdrawals. In the study conducted by Kawakami et al. on community-based participants with a potential to develop heart failure and AF; atrial dispersion was found increased in patients, in whom AF developed, compared to those who did not develop AF and atrial dispersion was found independently predictive of AF development [12]. In the study of Zhijuan Shang et al., atrial dispersion was found increased in PAF patients compared to the healthy group and atrial dispersion was found out to be an independent predictor of AF development in patients with a normal left atrium size

[24]. In another study; a high atrial dispersion before cardioversion was found decreased after cardioversion in patients, who were planned to undergo cardioversion because of AF[25,26]. In the study by Kupczynska et al., atrial dispersion was found out to be an independent predictor of thrombus formation in the left atrial appendage (LAA) in patients, in whom transesophageal echocardiography (TEE) was performed because of AF[27]. However, in a study by Rasmussen et al. on PAF patients, atrial dispersion was found not associated with ischemic stroke [28].

In our study, atrial dispersion was found out to be predictive of the development of AF in HCM patients. Elevated LV filling pressures due to left ventricular hypertrophy and SAM-related mitral insufficiency in HCM patients cause structural changes including fibrosis, called atrial remodeling, in the left atrial wall resulting from increased pressure and volume. While structural and electrical remodeling potentially causing electrical heterogeneity cause 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 [6].

Limitations:

The most important limitation of our study is its single center and crosssectional design and relatively small number of patients. Further prospective studies with larger number of patients are needed on this issue. Although speckle tracking echocardiography is considered angle independent, echocardiographic image quality should be adequate to perform an optimal analysis, as there is presently no software dedicated for the evaluation of atrial. The another limitation of our study, some asymptomatic patients may not have presented with AF during the 24-hour period of Holter monitoring, our study may underrepresent the prevalence of asymptomatic AF patients. Since the interval between initial assessment follow up visits was relatively prolonged, this may lead to underestimate atrial fibrillation that may develop.

Conclusion:

In patients with hypertrophic cardiomyopathy, atrial dispersion and age are predictive of the development of atrial fibrillation. Atrial dispersion measured by the speckle tracking-based method may provide further information in evaluating left atrial functions in patients with hypertrophic cardiomyopathy or other disease states.

Authors' contributions:

1-**Ozkan Candan,** Concept/design, Data collection, Data analysis/interpretation, Drafting article, Approval of article, Statistics

2-**Ender Özgün Çakmak ,** Concept/design, Data collection, Drafting article, Approval of article,

3- **Cetin Gecmen,** Concept/design, Data collection, Data analysis/interpretation, Critical revision of article, Approval of article,

4- **Muzaffer Kahyaoğlu** Statistics, Data analysis/interpretation, Concept/design, Approval of article, Data collection

5- **Zeki Şimsek** Data analysis/interpretation, Concept/design, Approval of article, Data collection

6- **Mehmet Çelik** Concept/design, Approval of article, Statistics, Data collection

7- **Ahmet Karaduman** Concept/design, Data analysis/interpretation, , Approval of article, Data collection

8- **Kamil Gulsen** Concept/design, Data analysis/interpretation, Drafting article, Approval of article, Data collection

9- **Cevat Kirma** Concept/design, Critical revision of article, Approval of article,

References:

1. Decker JA, Rossano JW, Smith EO, et al. Risk factors and mode of death in isolated hypertrophic cardiomyopathy in children. *J Am Coll Cardiol.* 2009;54(3):250-254.

2. Kofflard MJ, Ten Cate FJ, van der Lee C, et al. Hypertrophic cardiomyopathy in a large community-based population: clinical outcome and identification of risk factors for sudden cardiac death and clinical deterioration. *J Am Coll Cardiol.* 2003;41(6):987-993.

3. Olivotto I, Cecchi F, Casey SA, et al. Impact of Atrial Fibrillation on the Clinical Course of Hypertrophic Cardiomyopathy.*Circulation.* 2001;104(21):2517-2524.

4. Nistri S, Olivotto I, Betocchi S, et al. Prognostic Significance of Left Atrial Size in Patients With Hypertrophic Cardiomyopathy (from the Italian Registry for Hypertrophic Cardiomyopathy). *The American Journal of Cardiology.* 2006;98(7):960-965.

5. Yang WI, Shim CY, Kim YJ, et al. Left atrial volume index: a predictor of adverse outcome in patients with hypertrophic cardiomyopathy. *J Am Soc Echocardiogr.* 2009;22(12):1338-1343.

6. Josephson ME, Kastor JA, Morganroth J. Electrocardiographic left atrial enlargement. Electrophysiologic, echocardiographic and hemodynamic correlates. *Am J Cardiol.* 1977;39(7):967-971.

7. Guttmann OP, Pavlou M, O'Mahony C, et al. Predictors of atrial fibrillation in hypertrophic cardiomyopathy. *Heart.*2017;103(9):672-678.

8. Chen Y, Li Z, Shen X, et al. Assessment of left atrial remodeling in paroxysmal atrial fibrillation with speckle tracking echocardiography: a study with an electrophysiological mapping system. *Int J Cardiovasc Imaging.* 2019;35(3):451-459.

9. Sivalokanathan S, Zghaib T, Greenland GV, et al. Hypertrophic Cardiomyopathy Patients With Paroxysmal Atrial Fibrillation Have a High Burden of Left Atrial Fibrosis by Cardiac Magnetic Resonance Imaging.*JACC Clin Electrophysiol.* 2019;5(3):364-375.

10. Haugaa KH, Smedsrud MK, Steen T, et al. Mechanical Dispersion Assessed by Myocardial Strain in Patients After Myocardial Infarction for Risk Prediction of Ventricular Arrhythmia. *JACC: Cardiovascular Imaging.* 2010;3(3):247-256.

11. Candan O, Gecmen C, Bayam E, et al. Mechanical dispersion and global longitudinal strain by speckle tracking echocardiography: Predictors of appropriate implantable cardioverter defibrillator therapy in hypertrophic cardiomyopathy. *Echocardiography.*2017;34(6):835-842.

12. Kawakami H, Ramkumar S, Nolan M, et al. Left Atrial Mechanical Dispersion Assessed by Strain Echocardiography as an Independent Predictor of New-Onset Atrial Fibrillation: A Case-Control Study.*J Am Soc Echocardiogr.* 2019;32(10):1268-1276 e1263.

13. Authors/Task Force m, Elliott PM, Anastasakis A, et al. 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). *Eur Heart J.* 2014;35(39):2733-2779.

14. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging.*2015;16(3):233-270.

15. Schabelman S, Schiller NB, Silverman NH, et al. Left atrial volume estimation by two-dimensional echocardiography. *Catheterization and cardiovascular diagnosis.* 1981;7(2):165-178.

16. Garg L, Gupta M, Sabzwari SRA, et al. Atrial fibrillation in hypertrophic cardiomyopathy: prevalence, clinical impact, and management. *Heart Fail Rev.* 2019;24(2):189-197.

17. Guttmann OP, Rahman MS, O'Mahony C, et al. Atrial fibrillation and thromboembolism in patients with hypertrophic cardiomyopathy: systematic review. *Heart.* 2014;100(6):465-472.

18. Bauer F, Shiota T, White RD, et al. Determinant of left atrial dilation in patients with hypertrophic cardiomyopathy: a real-time 3-dimensional echocardiographic study. *J Am Soc Echocardiogr.*2004;17(9):968-975.

19. Vasquez N, Ostrander BT, Lu DY, et al. Low Left Atrial Strain Is Associated With Adverse Outcomes in Hypertrophic Cardiomyopathy Patients. *J Am Soc Echocardiogr.* 2019;32(5):593-603 e591.

20. Debonnaire P, Joyce E, Hiemstra Y, et al. Left Atrial Size and Function in Hypertrophic Cardiomyopathy Patients and Risk of New-Onset Atrial Fibrillation. *Circ Arrhythm Electrophysiol.* 2017;10(2).

21. Haugaa KH, Grenne BL, Eek CH, et al. Strain echocardiography improves risk prediction of ventricular arrhythmias after myocardial infarction. *JACC Cardiovasc Imaging.* 2013;6(8):841-850.

22. Sarvari SI, Haugaa KH, Anfinsen OG, et al. Right ventricular mechanical dispersion is related to malignant arrhythmias: a study of patients with arrhythmogenic right ventricular cardiomyopathy and subclinical right ventricular dysfunction. *Eur Heart J.*2011;32(9):1089-1096.

23. Haugaa KH, Edvardsen T, Leren TP, et al. Left ventricular mechanical dispersion by tissue Doppler imaging: a novel approach for identifying high-risk individuals with long QT syndrome. *Eur Heart J.*2009;30(3):330-337.

24. Shang Z, Su D, Cong T, et al. Assessment of left atrial mechanical function and synchrony in paroxysmal atrial fibrillation with two-dimensional speckle tracking echocardiography.*Echocardiography.* 2017;34(2):176-183.

25. Degiovanni A, Bortnik M, Dell'Era G, et al. Analysis of electrical and mechanical left atrial properties in patients with persistent atrial fibrillation. *Int J Cardiovasc Imaging.* 2013;29(1):71-78.

26. Dell'Era G, Rondano E, Franchi E et al. Atrial asynchrony and function before and after electrical cardioversion for persistent atrial fibrillation. European journal of echocardiography : the journal of the Working Group on Echocardiography of the European Society of Cardiology. 2010;11(7):577-583.

27. Kupczynska K, Michalski BW, Miskowiec D, et al. Incremental value of left atrial mechanical dispersion over CHA2 DS2 -VASc score in predicting risk of thrombus formation. *Echocardiography.*2018;35(5):651-660.

28. Rasmussen SMA, Olsen FJ, Jorgensen PG, et al. Utility of left atrial strain for predicting atrial fibrillation following ischemic stroke.*Int J Cardiovasc Imaging.* 2019;35(9):1605-1613.

Figure Legends:

Table 1: Echocardiographic and clinical characteristic of the study population

LV EF, left ventricular ejection fraction*; LA,* Left atrial; *LAVi,* left atrial volume index; *LVOT;* left ventricular outflow tract*; SCD,* sudden cardiac death*; NsVT* , non sustained ventricular tachycardia

Table 2: Speckle based Echocardiographic paramaters of the study populations

GLPS, global longitudinal peak strain; *PALS,* peak atrial longitudinal strain; *PACS* , peak atrial contraction strain.

Table 3: Correlation of Atrial dispersion and PALS

PALS, peak atrial longitudinal strain; *LA,* Left atrial

Table 4: The results of cox regression analysis for prediction of atrial fibrillation.

PALS, peak atrial longitudinal strain; *LA,* Left atrial. *CI,* confidence interval

Figure 1: Left atrial longitudinal strain parameters

PALS, peak atrial longitudinal strain; *PACS,* peak atrial contraction strain;

Figures 2: Speckle-based left atrial dispersion of without atrial fibrillation

AD, Atrial dispersion

Figures 3: Speckle-based left atrial dispersion of with atrial fibrillation

AD, Atrial dispersion

Figure 4: The ROC curve analaysis of Atrial dispersion

AD, Atrial dispersion

Hosted file

`Figures and Tables.zip` available at https://authorea.com/users/343908/articles/470535-left-atrial-dispersion-predicts-atrial-fibrillation-in-patients-with-hypertrophic-cardiomyopathy-a-5-year-follow-up-study