Atrial Functions Assessed by Colour Doppler Myocardial Imaging in Hypertrophic Cardiomyopathy

Hipertrofik Kardiyomiyopatide Renkli Doppler Görüntüleme ile Atriyal Fonksiyonların Değerlendirilmesi

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ABSTRACT Objective: Deterioration in atrial functions can lead to acute hemodynamic decompensation in patients with hypertrophic cardiomyopathy (HCM). The question of whether the atrial dysfunction may be explained primarily by the hemodynamic impact of left ventricle on atria or an underlying cardiomyopathic process remains unresolved. We aimed to investigate atrial functions in patients with HCM by using atrial myocardial color doppler imaging. **Material and Methods:** Left atrial (LA) and right atrial (RA) functional parameters in 15 patients with HCM and 15 age matched healthy controls were compared echocardiographically. Left atrial volumes and LA ejection fraction (LA ejection volume/Vmax X 100) were calculated. Atrial myocardial systolic, early and late diastolic velocities, peak systolic strain (SI) and strain rate (SR) were all measured by color doppler myocardial imaging. **Results:** Left atrial diameter was higher and LA ejection fraction was lower in the HCM group compared with those of the control group ($p<0.05$). Peak systolic and diastolic atrial myocardial velocities were compromised in HCM patients compared to healthy subjects. The peak systolic atrial myocardial strain and strain rate values in each studied atrial wall was lower in the HCM group but the difference reached statistically significance only for the RA wall. (RA SI; 62.6±42.2% vs. 97.9±45.1%, p=0.047, RA SRI; 2.3 ±1.7 vs. 3.7±1.2, p=0.031). **Conclusion:** Atrial myocardial deformation properties were impaired in patients with HCM. These results support the idea of increase in atrial stiffness in HCM and the hypothesis that HCM is a cardiac myopathic disease that involves the entire heart muscle.

Key Words: Hypertrophic cardiomyopathy; atrial function

ÖZET Amaç: Atriyal fonksiyonlarda bozulma Hipertrofik kardiyomyopatili (HKMP) hastalarda akut hemodinamik dekompanzasyona neden olabilir. Atriyal fonksiyonlardaki bozulmanın sol ventrikülün atriyum üzerindeki hemodinamik etkisine mi yoksa altta yatan kardiyomiyopatik proçese mi bağlı olduğu sorusu halen çözülememiştir. Biz bu çalışmada, HKMP'li hastalarda atriyal miyokardiyal renkli doppler görüntüleme ile atriyal fonksiyonları araştırmayı hedefledik. **Gereç ve Yöntemler:** HKMP'li 15 hasta ile yaşları uyumlu 15 sağlıklı kontrol bireyinin sol atriyal (LA) ve sağ atriyal (RA) fonksiyonel parametreleri ekokardiyografik olarak karşılaştırıldı. Sol atriyal hacim ve LA ejeksiyon fraksiyonu (LA ejeksiyon hacmi/Vmaks X 100) hesaplandı. Atriyal miyokardiyal sistolik velosite, erken ve geç diyastolik velosite, pik sistolik 'strain' (SI) ve 'strain rate' (SR) ölçümleri renkli doppler miyokardiyal görüntüleme yöntemi kullanılarak yapıldı. **Bulgular:** HKMP grubunda LA çap daha büyük iken LA ejeksiyon fraksiyonu kontrol grubuna göre daha az bulundu ($p<0,05$). HKMP'li hastalarda pik sistolik ve diyastolik atriyal miyokardiyal velositeler sağlıklı kontrol olgularına göre azalmış bulundu. HKMP grubunda pik sistolik atriyal miyokardiyal ''strain'' (SI) ve ''strain rate'' (SR) değerleri her çalışılan atriyal duvarda daha düşük bulundu fakat fark sadece RA duvar için istatistiksel öneme erişti (RA SI; 62,6±42,2 % vs. 97,9±45,1 %, p=0,047, RA SR; 2,3±1,7 vs 3,7±1,2, p=0,031). **Sonuç:** HKMP'li hastalarda atriyal miyokardiyal deformasyon özelliklerinin bozulduğu sonucuna vardık. Bu çalışmanın sonuçları HKMP'li hastalarda atriyal sertleşmenin arttığı ve HKMP'nin tüm kalp kasını tutan bir kardiyak miyopatik hastalık olduğu fikrini desteklemektedir.

Anahtar Kelimeler: Hipertrofik kardiyomiyopati, atriyum fonksiyonu

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In patients with hypertrophic cardiomyopathy (HCM) left atrial (LA) contraction which may be affected by left ventricular (LV) diastolic dysfunction plays an important role in LV filling. It has already been known that deterioration in atrial function can lead to acute hemodynamic decompensation in patients with HCM. The question of whether the atrial dysfunction may be explained primarily by the hemodynamic impact of ventricles on atria or an underlying cardiomyopathic process remains unresolved.

The aim of this study is to analyze atrial myocardial function in patients with HCM by using color Doppler myocardial imaging study.

MATERIAL AND METHODS

STUDY POPULATION

The study was carried out in accordance with the Declaration of Helsinki (1964) and current revisions of the good clinical practice guidelines of the European Commission and all patients gave informed consent. The study was approved by internal ethics committee. Sample size was calculated before the study. The study group consisted of 15 consecutive patients with both asymmetric septal hypertrophy and HCM in whom the ventricular septal thickness (IVS) in end-diastole (IVSd) and posterior wall thickness (PW) were all more than 15 mm and 11mm, respectively, and in whom the IVS/PW ratio was more than 1.3 by 2D echocardiography in the absence of another cardiac or systemic disease capable of producing the magnitude of hypertrophy evident.[1]

Patients were excluded when there was either echocardiographic evidence of segmental wall motion abnormalities, impaired LV function, moderate or severe valvular heart disease, or clinical evidence suggestive of coronary artery disease, hypertension, diabetes mellitus, bundle branch block, or atrial fibrillation at the time of examination.

A group of 15 healthy age-matched adults (mean age: 53±16 years; 7 women) served as control subjects, for the purpose of comparing left atrial volumes and functions. None of these individuals had evidence of cardiovascular disease by clinical and echocardiographic assessment. In addition, both HCM and control groups were not receiving any medications.

All measurements were performed by the same echocardiographer blinded to clinical data.

ECHOCARDIOGRAPHIC STUDIES

Standard echocardiography was performed with the patients at the partial left decubitus position, using a commercially available ultrasound system (GE, Vingmed, Vivid 7 Dimension, Horten, Norway). All measurements were done by the same echocardiographer following the recommendations of the American Society of Echocardiography.[2] The extent and magnitude of LV hypertrophy was assessed as previously described.[3] Left ventricular mass was calculated according to Devereux formula: LV mass= 1,04 x [(LVEDD + IVS thickeness+ PW thickeness)^3-(LVEDD)^3]-13,6 gram (LVEDD= left ventricular end diastolic diameter, IVS= interventricular wall thickness, PW=posterior wall thickness).[4] Left ventricular global systolic function was evaluated by LV ejection fraction (EF) by using modified Simpson's method.[2] The longitudinal function of both ventricles was assessed via measurement by conventional M mode at apical 4-chamber view. The maximal distance of endocardial motion during systolic phase was defined as atrioventricular (AV) ring displacement for the lateral point on the mitral ring (MAPSE) and the lateral point on the tricuspid ring (TAPSE).

The systolic transtricuspid pressure gradient was calculated using the modified Bernoulli equation: P= 4×V^2 where V represents the maximal regurgitant velocity in meters per second. To estimate right atrial pressure, measurements of inferior vena cava diameter were made from long-axis subxiphoid views. Right atrial pressure was estimated using the caval respiratory index as described by Kircher et al.[5] An estimation of pulmonary artery systolic pressure was obtained by calculating the sum of the transtricuspid gradient and the estimated right atrial pressure.

Mitral inflow velocity was assessed by pulsed-wave Doppler from the apical 4-chamber view by placing a 3-mm sample volume between the tips of

the mitral leaflets in diastole and recording at a sweep velocity of 100 mm/s. Peak early filling (E), atrial filling (A) velocities and E wave deceleration time were measured. Isovolumic relaxation time was acquired by placing the sample volume between the tips of the mitral leaflets and the left ventricular outflow tract.

ATRIAL VOLUME MEASUREMENT

Left atrial areas were determined by tracing the endocardial border of the left atrium at end systole (the largest dimension or the end of the T wave) and end diastole (the smallest dimension or the onset of QRS complex) from the apical 4- and 2-chamber views. The minimum and maximum left atrial outlines were traced for 3 consecutive beats, then averaged for all calculations. The length was measured from the midline of the mitral annulus to the opposite wall. Total left atrial volume is a composite of 3 distinct phases of atrial function: the active emptying volume, the passive emptying volume and the conduit volume. First of all, left atrial volume was assessed echocardiographically at the time of mitral valve opening and designated as Vmax (maximal volume, end-systolic volume), then at the onset of atrial systole and designated as Vp (preatrial contraction volume), and finally at mitral valve closure and designated as Vmin (minimal volume, end-diastolic volume, residual volume) from the apical 2- and 4-chamber views using the formula V= 8/3 лL×A4×A2, where л= 3.14; A4= Left atrial area in the apical 4-chamber; A2= Left atrial area in the apical 2-chamber; and L= the common axis pointing from the apex to the base (averaged from the two apical views).[6] Based on these volumes, the following LA function markers were measured: 1) The left atrial active emptying volume which is the difference between the LA volume prior to atrial systole and minimal LA volume at end ventricular diastole was calculated by Vp-Vmin and LA active emptying fraction which is the ratio of LA active emptying volume to the LA maximal volume and calculated by LA active emptying volume/Vp×100. 2) The, Left atrial passive emptying volume which is the difference between maximal LA volume at end ventricular systole and LA volume before atrial systole was calculated by Vmax-Vp and Left atrial passive emptying fraction was calculated by Left atrial passive emptying volume/Vmax×100. 3). Left atrial conduit volume which is the difference between total LV stroke volume and total LA stroke volume was calculated by Left ventricular stroke volume-(Vmax-Vmin). Total left atrial reservoir function (total stroke volume) which is the difference between the LA maximal volume at end ventricular systole and the minimal LA volume at end ventricular diastole was calculated by Vmax-Vmin[7]. Finally, global left atrial function was assessed by measuring left atrial ejection fraction which is calculated by Left atrial ejection volume/Vmax × 100.[8-10]

COLOR DOPPLER MYOCARDIAL IMAGING

Real-time 2D color Doppler myocardial imaging data were recorded from the LA and RA, using standard apical views at a high frame rate (>200 frames per second). An appropriate velocity scale was chosen to avoid data aliasing. The narrowest image sector angle was used to achieve the maximum color Doppler frame rate. Careful attention was paid to keep the region of interest at the center of the ultrasound sector to make the alignment as close as possible to 0° in the long-axis motion. In all the samples studied, we selected 5 consecutive cardiac cycles (to be used for subsequent analysis) with an interbeat variability <10% to be recorded and used for subsequent analysis.

OFFLINE ANALYSIS

Color Doppler myocardial imaging data were stored in digital format and analyzed offline with the dedicated software (Echopac, Vivid 7 Dimension, GE, Vingmed, Horten, Norway). From the ultrasound data set, 3 parameters were calculated: local velocity, local strain rate, and its integral, local strain. Myocardial velocity measures the local motion of a tissue; strain rate, the local rate of deformation; and strain, the total amount of local deformation of a tissue. Longitudinal direction changes (measured from apical views) for atria are better described by the terms "rate of lengthening" in systole (positive strain rate value) and "rate of shortening" in diastole (negative strain rate value).

Myocardial atrial strain determines regional lengthening expressed as a positive value or shortening expressed as a negative value. Analysis was performed for atrial longitudinal velocities, strain rate and strain from the apical view for the mid segments of lateral wall (Figure 1-3). To derive velocity, strain rate, and strain profiles from the studied segment, the examined region was continuously positioned within the segment being studied with a proprietary semiautomated tracking algorithm. Continuous care was taken to keep the sample volume out of the pulmonary veins.

End diastole was defined as the ECG R peak, and end systole was defined as the end of the ECG T wave. Peak positive systolic and early diastolic values were calculated from the extracted curve.

STATISTICS

Continuous variables were expressed as mean ± SD or median (interquartile range) in the presence of abnormal distribution, and categorical variables as percentages. Comparisons between groups of patients were made by use of a $\chi 2$ test for categorical variables, independent samples t test for normally distributed continuous variables, and Mann-Whitney U test when the distribution was skewed. Correlations were evaluated either via Pearson or Spearman correlation tests. Significance was set to a p value 0.05.

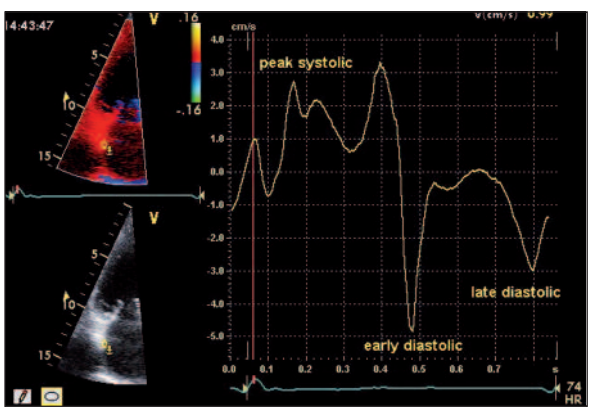

FIGURE 1: Atrial myocardial velocities obtained from the mid segments of left atrial lateral wall in apical four chamber view by color Doppler myocardial imaging.
(See for colored form http://cardivascular.turkiyeklinikleri.com/)

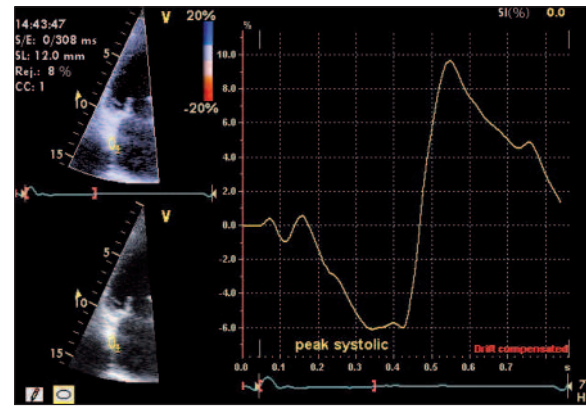

FIGURE 2: The trace of the longitudinal strain which obtained from left atrial lateral wall.
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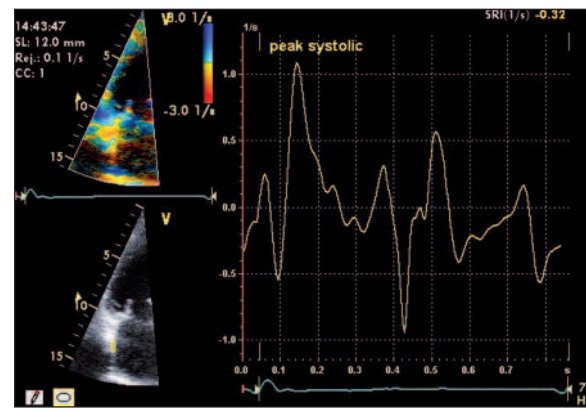

FIGURE 2: The trace of the longitudinal strain rate of left atrial wall.
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RESULTS

General characteristics of the studied sample were presented in Table 1. Left ventricular mass index was 164±64 g/m^2 in HCM group and 85 ±20 g/m^2 in control group. There were no differences between two groups with regard to left ventricular ejection fraction, pulmonary artery pressure, transmitral doppler parameters and no significant difference was found in the peak systolic AV ring displacement between the HCM and control groups (Table 1).

ATRIAL FUNCTIONS

HCM patients had significantly larger left atrial dimensions, lower conduit volume and higher left atrial active emptying volume and total emptying volume than use of controls (Table 2).

TABLE 1: General characteristics of the studied sample.

	HCM Patients	Control Group	p
Age (year)	52±18	51±8	0,88
Male sex (%)	12(80)	9(60)	0,23
Body surface area (m2)	1.7±0.2	1.8±0.1	0.11
Systolic blood pressure (mm Hg)	118±20	115±15	0.82
Diastolic blood pressure (mm Hg)	72±5	70±7	0.78
Heart rate (bpm)	81±12	72±10	0.89
LV end-diastole (cm)	4.6±0.5	4.8±0.4	0.24
LV end-systole (cm)	2.9±0.7	3.1±0.3	0.43
LV ejection fraction (%)	65±12	64±5	0.92
IVS end-diastole (cm)	1.5±0.3	0.8±0.9	<0.001
PW end-diastole (cm)	1.3±0.7	0.8±0.4	<0.001
Left atrium (cm)	4.3±0.7	3.6±0.4	0.002
LV mass index	164.1±64.8	85.9±20.0	<0.001
Systolic pulmonary artery pressure (mmHg)	32.8±16.0	24.2±6.3	0.520
Right atrium (cm)	3.6±0.6	3.3±0.5	0.13
LV lateral ring displacement (cm)	1.3±0.4	1.5±0.2	0.22
RV free wall ring displacement (cm)	1.7±0.5	2.1±0.2	0.05
Transmitral PW-Doppler			
E (m/sec)	1.0±0.5	0.7±0.1	0.135
A (m/sec)	0.7±0.3	0.7±0.1	0.618
E/A	1.0±0.3	1.3±0.5	0.069
E-DT (msec)	190.4±65.4	188.9±64.1	0.953
IVRT (msec)	107.2±48.8	101.5±12.7	0.695

LV: Left ventricle; RV: Right ventricle; IVS: Interventricular septum; PW: Posterior wall; E: Early diastolic peak flow velocity; A: Late diastolic peak flow velocity; E-DT: Deceleration time of the E wave; IVRT: Isovolumic relaxation time.

COLOR DOPPLER MYOCARDIAL IMAGING STUDY

Peak systolic and diastolic atrial myocardial velocities, strain rate, and strain were compromised in HCM patients compared to those in healthy subjects (Table 3).

Early diastolic left atrial myocardial velocity was significantly reduced in HCM group. The peak systolic atrial myocardial strain and strain rate values in each studied atrial wall was lower in the HCM group but the difference reached statistically significance only for the right atrial wall (Table 3).

Peak systolic left atrial myocardial strain and strain rate were correlated both with left atrial ejection fraction (r=0.439, p=0.02 and r=0.670, p=0.01, respectively) and peak systolic mitral AV ring displacement (r=0.551, p=0.003 and r=0.595, p=0.001, respectively). While peak systolic left atrial myocardial strain was being negatively correlated with Vmin (r=-0.487, p=0.01) peak systolic left atrial myocardial strain rate was negatively correlated with the left atrial Vp and Vmin (r=-0.345, p=0.04).

Peak systolic right atrial myocardial velocity was correlated with pulmonary artery pressure (r=-0.407, P=0.04). Peak systolic right atrial myocardial

TABLE 2: Left atrial functions in hypertrophic cardiomyopathy (HCM) patients compared with controls.

	HCM Patients	Control Group	p
LA Vmax (ml)	101.1±12.5	56.0±4.9	0.04
LA Vmin (ml)	59.9±11.6	28.5±4.7	0.023
Passive emptying volume (ml)	17.7±11.9	12.0±7.0	0.16
Passive emptying fraction (%)	17.9±11.1	22.3±15.7	0.40
Conduit volume (ml)	22.7±15.6	46.0±23.2	0.005
LA filling volume (ml)	44.2±9.6	27.7±10.3	<0.0001
LA filling fraction (%)	42.9±18.0	39.5±12.7	0.37
Active emptying volume (ml)	25.3±10.8	15.4±8.7	0.017
Active emptying fraction (%)	36.5±19.1	36.5±19.2	0.99
Total emptying volume (ml)	42.9±9.4	27.5±10.3	<0.0001
Total emptying fraction (%)	48.1±16.2	50.8±18.6	0.69

LA: Left atrium; Vmax: Maximal volume; Vmin: Minimum volume.

TABLE 3: Color doppler myocardial study in hypertrophic cardiomyopathy (HCM) patients compared with controls.

	HCM Patients	Control Group	p
LA lateral wall velocity, cm/s			
Peak Systolic Value	3.6±2.0	4.6±1.4	0.14
Peak Early Diastolic Value	2.6±2.3	4.7±2.0	0.01
Peak Late Diastolic Value	4.0±2.7	5.9±1.7	0.06
SI, %	41.4±26.7	47.3±22.5	0.54
SR, s^{-1}	2.1±1.2	2.3±0.9	0.58
RA free wall velocity, cm/s			
Peak Systolic Value	4.5±2.8	5.4±2.1	0.34
Peak Early Diastolic Value	2.8±1.7	3.8±1.8	0.14
Peak Late Diastolic Value	5.9±3.2	7.4±3.4	0.256
SI, %	62.6±42.2	97.9±45.1	0.04
SR, s^{-1}	2.3±1.7	3.7±1.2	0.03

LA: Left atrium; RA: Right atrium; SI: Strain; SR: Strain rate.

strain and strain rate were positively correlated with left atrial ejection fraction (r=0.439, p=0.02 and r=0.465, p=0.01, respectively).

Peak systolic right atrial myocardial strain and strain rate were negatively correlated with left atrial volumes and left ventricular mass index (r=-0.348, p=0.04 and r=-0.407, p=0.03, respectively).

While the peak systolic right atrial myocardial strain was correlated with peak systolic mitral AV ring displacement (r=0.479, p=0.01), peak systolic right atrial myocardial strain rate was correlated with tricuspid annular peak systolic displacement (r=0.410, p=0.03).

When evaluating the correlation between the left atrial strain and strain rate values and parameters of left atrial mechanical function, a significant positive correlation was detected between left atrial myocardial systolic strain rate and left atrial ejection fraction (r=0.730, p=0.03 and r=0.617, p=0.019). However, no significant correlation was found among left atrial strain and strain rate and the other traditional parameters of left atrial mechanical function.

DISCUSSION

In this study we have noted that atrial myocardial deformation properties impaired in patients with HCM. Both the atrial lengthening that occurs during ventricular ejection and atrial myocardial velocity that occurs during ventricular early filling were reduced. These findings were in concordance with several studies which had demonstrated the impairment at the reservoir and conduit function of atrium in patients with HCM and support the idea of increase in atrial stiffness in these patients.[11-15]

Interactions between atrium and ventricle are functionally important during ventricular systole, early diastole, and in atrial systole. In patients with left ventricular hypertrophy, left ventricular filling is maintained by some mechanisms compensating for the reduced increase in volume early in diastole.[16]

In previous studies, echocardiographic indexes of left atrial relaxation and filling have been found abnormal in patients with hypertrophic cardiomyopathy but not in secondary forms of left ventricular hypertrophy.[17,18] These indexes are abnormal in all forms of hypertrophic cardiomyopathy irrespective of left ventricular outflow tract obstruction and distribution of hypertrophy. It has been speculated that changes in atrial function are not solely attributable to left ventricular diastolic dysfunction and atrium is primarily affected by the disease process.[17,18]

The left atrial pressure, left atrial volume index at the end of diastasis, left atrial ejection fraction, and LV rapid filling volume indexes have all been shown to be less in the HCM in comparison to those in control group previously.[18]

The question of whether the atrial dysfunction may be explained primarily by the hemodynamic impact of LV on atria or an underlying cardiomyopathic process remains unresolved.

Many invasive pressure monitoring studies have shown that patients with HCM have a spectrum of diastolic abnormalities, including increased mean left atrial and LV end diastolic pressures, prolonged time constant of relaxation, and increased effective chamber and myocardial stiffness.[19-21]

Some authors have also shown that characteristic findings based on mitral inflow patterns are lower E wave velocity, prolonged E wave deceleration time, higher A wave velocity, and an E/A ratio <1.0, which are clearly differentiated from normal inflow patterns of normal subjects.[22-24] However, the overlap is considerable.[22] Our study showed that mean values of transmitral filling velocities, mitral inflow pattern, E deceleration time and isovolumic relaxation time, were similar to those seen in controls concordantly with the previous studies.[25-27] According to these results, most of the study patients had a normal or pseudonormalized diastolic pattern. When we think that these patients had increased left ventricular mass index and LA diameter, this could only be explained by the fact that Doppler transmitral flow velocity profiles are strongly influenced by factors such as loading conditions and age and do not necessarily provide the actual estimates of LV filling pressure.[26]

Left atrial function plays an important role in patients with HCM. Left atrial dilatation is a com-

mon finding in HCM patients.[28] Deterioration of atrial function as in atrial fibrillation in the HCM patients has been found as a key determinant of HCM-related mortality and limiting symptoms. Development of atrial fibrillation may indeed represent a clinical turning point, often dominating the clinical picture and decisively influencing long-term outcome.[29]

It has been speculated that specific HCM-causing mutations may increase predisposition to atrial fibrillation, e.g., by causing an intrinsic atrial myopathy associated with prolonged and fragmented atrial conduction or presently undefined hemodynamic alteration.[30]

Such a hypothesis might also explain the development of atrial fibrillation in the absence of left atrial dilatation in a minority of patients.[29] Acar et al. has showed that right atrial diameter is independent predictor of atrial fibrillation occurrence in hemodialysis patients.[31] Thus, our findings suggest that the echocardiographically derived myocardial deformation index, which reflects structural changes assessed by transthoracic echocardiography, can detect abnormalities in atrial functions before clinical deterioration.

The superior sensitivity of strain and strain rate imaging in detecting myocardial abnormalities has been confirmed in several studies and is related to their relative independence from the tethering effect and global cardiac motion.[32-36]

In early diastole the atria act as a conduit, passively emptying during ventricular relaxation when the blood is transferred from systemic and pulmonary veins to the ventricles.[37]

Thus, atrial function during early diastole is strongly influenced by left ventricular compliance. This is demonstrated by the strong correlation between early diastolic strain and strain rate values, and LV global diastolic function indexes and annular excursion.[37] In the present study, left atrial early diastolic velocity, which is concordant with these values, was correlated with MAPSE (r=0.537, p=0.005), TAPSE (r=0.403, p=0.41) and transmitral Doppler E wave (r=-0.391, p=0.004).

During ventricular systole, the atria function as reservoirs to store blood when AV valves are closed, and reservoir function is influenced by atrial relaxation, ventricle contraction through the descent of the base, and atrial chamber stiffness.[37] It has been suggested that atrial peak systolic strain and strain rate are measures of atrial reservoir function according to preliminary findings.[38] But, in the present study, we could not find any correlation between left atrial ejection volume and each left atrial myocardial color Doppler parameters. Since the number of patients in this study was relatively small, these findings should be tested in larger groups of patients.

Additionally, only a weak correlation has been found between peak systolic myocardial atrial strain and strain rate and peak systolic AV ring displacement in the bivariate correlation analysis, but no correlation, at all, has been found between peak systolic AV ring displacement and atrial peak systolic deformation properties.

These findings confirm that the peak systolic atrial strain and strain rate are less influenced by tethering effects and global heart motion and suggest that the abnormal atrial deformation properties during this phase are due mainly to changes in atrial myocardial compliance.[39]

The usual limitations inherent in the angle dependency of all echocardiographic Doppler techniques also apply to ultrasound-derived strain rate imaging. In this study, care was taken to align the ultrasound beam with the direction of myocardial deformation to be studied.

In our study we demonstrated that the myocardial deformation properties of both atria were compromised. We did not analyze the mitral annulus velocity using the tissue Doppler imaging and pulmonary venous flow velocity patterns both of which would help a better non-invasive estimation of LV diastolic function when combined to the transmitral flow velocity profile.

The present study also supports the hypothesis that hypertrophic cardiomyopathy is a cardiac myopathic disease involving the whole heart muscle.[17] As the atrial myocardial deformation proper-

ties were supposed to be predictive of sinus rhythm maintenance, it may be helpful for earlier detection of the impairment of atrial function in HCM and predicting the high risk group for clinical outcome but this theory should be further evaluated by the prospective studies.

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