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The Role of Left Ventricular Outflow Tract Presystolic Wave in the Differentiation of Non-obstructive Hypertrophic Cardiomyopathy and Athlete's Heart

Non-obstrüktif Hipertrofik Kardiyomiyopati ve Atlet Kalbi Ayrımında Presistolik A Dalgasının Rolü

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Abstract

Objectives: Although hypertrophic cardiomyopathy (HCM) can be distinguished from athlete's heart based on differences in echocardiographic measurements, difficulties may sometimes be encountered in clinical practice. The aim of this study was to evaluate the use of the presystolic wave (PSW) of the left ventricular outflow tract, assessed by Doppler echocardiography, as a discriminating factor between HCM and athlete's heart.

Material and Methods: A total of 52 subjects were included in this study; 27 of them had HCM and 25 were athletes. Pulsed Doppler assessment of the left ventricular outflow tract was performed on the ventricular wall immediately proximal to the aortic valve in the apical five-chamber view. All patients were evaluated for the presence of PSW, and the velocity of this wave was recorded in PSW-positive subjects.

Results: The frequency of PSW was found to be higher in HCM patients [n=12 (44%)] compared to athletes [n=4 (16%)] (p=0.026). PSW velocity measurements were observed to be higher in the HCM group, however, there was no statistically significant difference [53 ms (36-84), 68 ms (35-193)], (p=0.362).

Conclusion: While there is a need for scaling-up similar studies, the current findings suggest that PSW may be another parameter to consider to distinguish between HCM and athlete's heart, when accompanied by 2D evidence of left ventricular hypertrophy and other Doppler / Doppler tissue imaging parameters abnormalities. The fact that PSW is not an independent parameter and can also be observed in normal hearts should be remembered.

Keywords: Hypertrophic cardiomyopathy, athlete's heart, presystolic wave, PSW, HCM

Öz

Amaç: Hipertrofik kardiyomiyopati (HKMP) ile atlet kalbi ekokardiyografik farklı ölçümler ile birbirinden ayırt edilebilmekle birlikte klinik pratikte zaman zaman güçlüklerle karşılaşılabilmektedir. Bu çalışmada Doppler ekokardiyografisi ile bakılan presistolik A dalgasının (PSAD), HKMP ve atlet kalbi ayrımında kullanılmasının değerlendirilmesi amaçlanmıştır.

Yöntem ve Gereçler: Çalışmamıza 27'si HKMP; 25'i sporcu toplam 52 kişi dahil edildi. Sol ventrikül çıkış yolu pulsed Doppler değerlendirmesi apikal 5 boşluk görüntülemede ventriküler yüzde aort kapağın hemen proksimalinden yapıldı. Tüm hastalar PSAD varlığı açısından değerlendirildi. PSAD tespit edilenlerde bu dalganın hızı da kaydedildi.

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Bulgular: PSAD sıklığı HKMP hastalarında [n=12(%44)], sporculara [n=4 (%16)] göre daha fazla olarak saptandı (p=0,026). PSAD hızları HKMP grubunda daha yüksek olarak ölçüldü ancak istatistiksel olarak anlamlı değildi [53 ms (36-84)], [68ms (35-193)], (p=0,362).

Sonuç: PSAD, HKMP ve sporcu kalbi ayrımında eşlik eden diğer bulgularla birlikte ayırıcı bir parametre olarak kullanılabilir ancak daha büyük ölçekli çalışmalara ihtiyaç vardır.

Anahtar Kelimeler: Hipertrofik kardiyomiyopati, atlet kalbi, presistolik A dalgası

INTRODUCTION

Hypertrophic cardiomyopathy (HCM), the most common inherited cardiac disorder, is a heart muscle disease most often caused by variants in one or more sarcomeric genes. HCM is the most important cause of sudden cardiac death in young athletes, and therefore its diagnosis is crucial. Adaptations of athletes' heart can mimic cardiovascular diseases and may often be confused with HCM. It is important to differentiate between these two conditions, which can often be confused with each other.

Presystolic wave (PSW), also known as J wave, is recorded in late diastole during the Doppler interrogation of the left ventricular (LV) outflow tract (1,2). There is a debate about whether PSW is a physiological or pathological sign. It might theoretically be associated with LV hypertrophy, impaired LV compliance and stiffness (3,4). We planned this study believing that PSW presence in this patient group differentiates from athlete's heart, considering the similar pathophysiological changes in HCM.

MATERIAL AND METHODS

Approval for the study was obtained from the Scientific Research Ethics Committee of University of Health Sciences Türkiye, Trabzon Kanuni Training and Research Hospital (protocol number: 2017/15, date: 21.06.2017). Verbal and written informed consent have been obtained from the subjects included in the study.

Patient Cohort

The study included 27 patients who presented to our Clinic of Cardiology Outpatient and were diagnosed with HCM according to the European Society of Cardiology diagnostic criteria (the criterion in echocardiographic measurements is generally a maximum wall thickness ≥15 mm) (5).

The exclusion criteria were as follows: other causes that could lead to LV hypertrophy (e.g., infiltrative diseases such as amyloidosis, chronic renal failure, aortic stenosis, uncontrolled hypertension), secondary hypertension, severe renal and hepatic dysfunction, LV systolic dysfunction, severe primary valve disease, mitral annular calcification, septal ablation associated with HCM or previous surgical treatment, severe coronary artery disease (presence of >50% occlusion in at least one coronary artery), history of myocardial infarction, other congenital cardiovascular diseases, atrial fibrillation, any degree of atrioventricular block, severe bradycardia, sinus syndrome, presence of a pacemaker, presence of complete bundle branch block, electrolyte abnormalities, malignancy, pregnancy, asthma or advanced chronic obstructive pulmonary disease, aortic diseases, and connective tissue disorders

The study included 25 athletes who had competed professionally in various sports (football, boxing, and running) for at least five years, exercised for at least 10 hours per week, and had no history of cardiovascular disease or cardiac symptoms.

General Assessment and Blood Pressure Measurements

The medical history of all patients was obtained, and physical examinations were performed for all patients. The height and weight of all patients were measured, and their body mass index and body surface area (BSA) were calculated. Systolic and diastolic blood pressures were measured, and a 12-lead resting electrocardiogram was recorded. Cardiovascular disease risk factors were also recorded.

Echocardiography

Transthoracic echocardiography was performed on the patients after allowing them to rest in the echocardiography room for 15 minutes in a supine position. The echocardiographic assessment was performed with the Vivid E95 instrument with 2.5 MHz probe (GE Medical System), in the left lateral position while the patient was breathing quietly. The conventional M-mode, B-mode, and Doppler parameters were performed in compliance with the American Society of Echocardiography guidelines (6). Two-dimensional and Doppler images were obtained during breath-hold, and stored in cine-loop format from 3 consecutive beats, and average values were reported. In the initial echocardiographic assessment, the presence of any pathology was ruled out on the parasternal long axis, short axis, apical 4 and 2 chamber views. Quantification of LV end-diastolic and end-systolic diameters and posterior and septal wall thicknesses was carried out. The Devereux equation was used to derive LV mass (LVM): $LVM = 0.8 \times [1.04\ (LVEDD + IVST + PWT)^3 - (LVEDD^3)] + 0.6$, where LVEDD denotes LV end-diastolic diameter, IVST denotes intraventricular septal wall thickness,

and PWT denotes posterior wall thickness. The LVM index was calculated by the formula LVM / BSA. BSA was calculated using the "BSA (m^2) = 0.007184 x height (cm) 0.725 x weight (kg) 0.425" formula (7). The portion of the LV outflow tract just proximal to the aortic valve was interrogated with pulsed wave Doppler in the apical five-chamber window to detect the presence of a PSW just before the LV outflow tract flow. PSW peak velocity was quantified whenever a quantifiable PSW signal was present (Figure 1). Mitral inflow velocities were evaluated using pulsed-wave Doppler, with sample volume placed at the tip of the mitral leaflets in an apical four-chamber view. Using the average of three beats, the diastolic peak early (E) and peak late (A) transmitral flow velocities and the peak E to peak A velocities (E/A) were measured. LV ejection fraction was estimated using the Simpson's method. Tissue Doppler evaluation of the left ventricle was performed from the apical four-chamber view with a frame rate of greater than 80/s. All quantifications were performed on frozen images obtained from three to five cardiac cycles. Mitral annular velocities were quantified with the sample volume being placed at the junction of the mitral valve annulus and the septal myocardial wall.

Statistical Analysis

The statistical analyses of the findings obtained in our study were performed using the SPSS Windows version 19 software package (SPSS Inc., Chicago, Illinois, USA). The values of continuous variables are given as the median (minimum-maximum); the values of categorical variables are given as numbers and percentages. The chi-square test was used to evaluate categorical variables. The Mann-Whitney U test was used to evaluate continuous variables.

If the calculated p-value for all statistical analyses is less than 0.05, the difference is considered statistically significant.

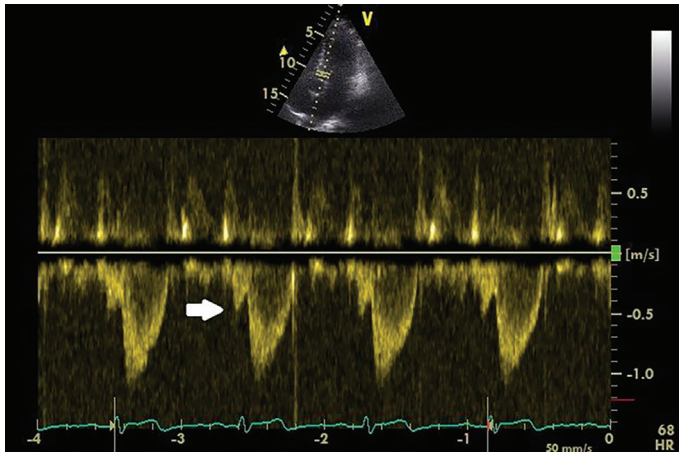

Figure 1. Left ventricular outflow tract presystolic wave

RESULT

The clinical and demographic characteristics of the patients are listed in Table 1. A total of 52 cases were included in the present study; 27 of them were HCM patients and 25 were athletes.

Age was significantly higher in the HCM group compared to the control group [HCM: 50 (20-75) years, control: 26 (18-59) years, $p<0.001$].

Also, in Table 1, it is shown that body mass index is higher in the HCM group, and this is statistically significant. Systolic blood pressure is also calculated to be higher in the HCM group.

Echocardiographic parameters are shown in Table 2. PSW was observed to be higher in the HCM group than in the athlete group. No significant difference was found between PSW velocities.

In Table 2. IVST, PWT, LVM and left ventricular mass index calculated higher in HCM group than athletes group.

DISCUSSION

This study found that PSW is more common in HCM patients than in athletes. PSW velocity values were observed to be higher in the HCM group; however, this increase is not statistically significant.

PSW is a late diastolic wave frequently observed in Doppler assessment of the LV outflow tract (1,2). However, its frequency, clinical significance, and relationship with other echocardiographic parameters have not been fully determined. There are different reports indicating whether this is a normal phenomenon or a sign of pathological conditions. The mechanism of formation of diastolic waves in the LV outflow tract was demonstrated by Panayiotou and Byrd (8). This study suggests that diastolic waves in the LV outflow tract are a normal phenomenon and that the responsible mechanism stems from fluid mechanics, defined as a "ring vortex." According to this mechanism, the diastolic filling flow from the left atrium to the left ventricle passes through the mitral valve and is directed toward the LV outflow tract by both the vortex effect and reflection from the apex. This causes the appearance of diastolic flow waves in the LV outflow tract as observed using Doppler.

It has been observed that LV wall thickness is naturally higher in patients with HCM. In the study mentioned above, conducted by Panayiotou and Byrd (8), PSW was observed more frequently in individuals with LV hypertrophy, compared to those without.

Similar to the findings of the cited study, our study also found that PSW frequency was higher in patients with HCM.

Table 1. Clinical and demographic features of the patients			
	Athlete group, n=25	**HCM group, n=27**	**p-value**
Age (years)	26 (18-59)	50 (20-75)	**<0.001**
Female (n)	7 (28%)	6 (22%)	0.436
Body mass index (kg/m^2)	24.6 (19.1-32.2)	27.9 (20.3-36.4)	**0.001**
Systolic blood pressure (mmHg)	110 (90-130)	120 (100-135)	**0.002**
Diastolic blood pressure (mmHg)	70 (60-90)	70 (60-85)	0.222
Hearth rate (beat/min)	70 (50-96)	66 (53-98)	0.309

Table 2. Echocardiographic measurements			
Variables	**Athlete group, n=25**	**HCM group, n=27**	**p-value**
IVST (mm)	11 (8-15)	19 (13-28)	**<0.001**
PWT (mm)	9 (8-14)	14 (11-24)	<0.001
LVEDD (mm)	45 (31-54)	43 (33-57)	0.236
LVES (mm)	31 (23-47)	29 (22-40)	**0.024**
LVM (g)	153.4 (84.8-261.8)	314.2 (207.7-684.1)	**<0.001**
LVMI (g/m^2)	84.7 (46.8-128.35)	168.1 (95.3-367.81)	**<0.001**
LA (mm)	34 (24-39)	36 (20-48)	**0.032**
AO (mm)	26 (17-41)	24 (20-31)	0.734
AO velocity (cm/s)	128 (96-187)	153 (73-271)	**0.001**
Mitral E (cm/s)	78 (39-140)	70 (40-104)	0.374
Mitral A (cm/s)	54 (40-120)	71 (35-145)	**0.003**
Mitral E/A	1.5 (0.63-2.24)	0.93 (0.60-2.07)	**0.019**
EDT (ms)	227 (80-374)	242 (150-380)	**0.420**
Septal SM (cm/s)	11 (8-19)	6 (3-14)	**<0.001**
Septal EM (cm/s)	16 (2-29)	6 (3-11)	**<0.001**
Septal AM (cm/s)	10 (4-20)	7 (3-17)	**0.002**
Septal E/EM (cm/s)	6.5 (4.0-15.6)	12.6 (5.6-27.6)	**<0.001**
Lateral SM (cm/s)	9 (6-14)	7 (3-12)	**<0.001**
Lateral EM (cm/s)	10 (5-19)	7 (4-14)	**<0.001**
Lateral AM (cm/s)	10 (5-15)	8 (3-16)	0.197
Lateral E/EM	6.5 (4.0-15.6)	8.6 (5.5-20.7)	**0.017**
PSW (n)	4 (16%)	12 (44%)	**0.026**
PSW velocity (cm/s)	53 (36-84)	68 (35-193)	0.362

IVST: Intraventricular septal wall thickness, PWT: Posterior wall thickness, LVES: Left ventricular end-diastolic LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, LVM: Left ventricular mass, LVMI: Left ventricular mass index, LA: Left atrium, AO: Aortic root, EDT: E wave deceleration time, SM: Systolic myocardial velocity, EM: Early diastolic myocardial velocity, AM: Late diastolic myocardial velocity, PSW: Presystolic wave, E: Peak early, A: peak late

In the study by Panayiotou and Byrd (8), higher PSW rates were found in individuals with LV hypertrophy compared to those without. In our study, although the values were not statistically significant, PSW speeds were measured to be higher in the HCM group.

As seen in normally functioning hearts, the PSW wave can also be observed in different patient groups with diastolic dysfunction. A previous study by Mittal and Pancholi (3) colleagues demonstrated that PSW is associated with LV diastolic dysfunction. In our study, impaired LV diastolic function was also observed in HCM patients, and this may have led to the more frequent occurrence of PSW. It is known that LV diastolic dysfunction develops with aging (9). Our patient group was older than the athlete group, and this may have contributed to the more frequent observation of LV diastolic dysfunction and consequently PSW in HCM patients. However, it is known that the most frequently observed pathophysiological event in HCM is diastolic dysfunction (10). It is independent of the degree

and location of LV hypertrophy and may occur with or without pressure gradient or symptoms (11). Therefore, we believe that the existence of age differences between groups does not constitute a limitation.

Finocchiaro et al. (12) evaluated Doppler parameters in patients with HCM. In this study, mitral E wave velocity was found to be lower in patients with HCM. Similarly, in our study, mitral E wave velocity was also found to be lower in patients with HCM, but this difference was not statistically significant. Finocchiaro et al. (12) found higher mitral A wave velocity in patients with HCM. Similarly, our study found that mitral A wave velocity was higher in patients with HCM. PSW is the reflection of the mitral A wave. The higher incidence of PSW in the HCM group may also be due to the higher mitral A wave velocity. Finocchiaro et al. (12) also evaluated tissue Doppler parameters and found that velocities were lower in patients with HCM. In our study, Doppler values obtained from septal, and lateral wall tissue were found to be lower in the HCM group compared to athletes. Finocchiaro et al. (12) found that the E/E value was higher in the HCM group, and similar values were observed in our study.

Study Limitations

The most significant limitation was that the number of patients included in the study was insufficient. Since only the patients with HCM who applied to our hospital were included in the study, they may not be generalized to the general population. Patients with coronary artery disease were excluded from the study, but no invasive tests were performed to rule out coronary artery disease. There was an age difference between the HCM group and the athlete group. Age is known to affect PSW appearance due to diastolic function. However, due to the physiological nature of HCM, this is unlikely to be a problem.

CONCLUSION

In our study, PSW frequency and velocity were higher in the HCM group than in the athlete group. Current findings indicate that more frequent PSW is associated with LV diastolic dysfunction and higher mitral A wave velocity in HCM patients, resulting from increased LV wall thickness and stiffness. Although there is a need for scaling-up such studies, PSW may be another parameter to consider to distinguish between HCM and arterial hypertension when accompanied by 2D evidence of LV hypertrophy and other Doppler / Doppler tissue imaging parameters abnormalities. The fact that PSW is not an independent parameter and can also be observed in normal hearts should not be forgotten.

*Ethics

Ethics Committee Approval: Approval for the study was obtained from the Scientific Research Ethics Committee of University of Health Sciences Türkiye, Trabzon Kanuni Training and Research Hospital (protocol number: 2017/15, date: 21.06.2017).

Informed Consent: Verbal and written informed consent have been obtained from the subjects included in the study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: D.C.H.K., G.U., Concept: G.U., M.R.S., Design: G.U., M.R.S., Data Collection or Processing: D.C.H.K., M.R.S., Analysis or Interpretation D.C.H.K., M.R.S., Literature Search: D.C.H.K., Writing: D.C.H.K.

Conflict of Interest: No conflict of interest was declared by the authors.

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