YENİ TANI ESANSİYEL HİPERTANSİYON HASTALARINDA PRESİSTOLİK DALGA AORTİK SERTLİK DERECESİNİ ÖNGÖRDÜREBİLMEKTE, FAKAT SİRKADİYEN KAN BASINCI PATERNİNİ ÖNGÖREMEMEKTEDİR

Presystolic Wave Velocity May Predict Aortic Stiffness But Not Circadian Blood Pressure Pattern In Patients With Newly Diagnosed Essential Hypertension

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ÖZET

Arka plan ve Amaç: Hipertansiyon (HT) hastalarında non-dipping sirkadiyen patern ile artmış aortik sertlik derecesi, morbidite ve mortalite artışı ile ilişkilidir. Bu çalışmadaki amacımız sol ventrikül çıkım yolu (SVÇY) üzerinde transtorasik doppler ekokardiyografi (TTE) ile ölçümlenen presistolik dalganın (PSD), TTE ile ölçülen aortik sertlik parametreleri ile ilişkisinin olup olmadığının değerlendirilmesidir.

Metot: Kardiyoloji polikliniğine başvuran ve yeni hipertansiyon teşhisi konan ardışık 140 hasta çalışmaya dahil edildi. Fizik muayene, laboratuar analizi, TTE ve ambülatuar kan basınç monitörizasyonu (AKBM) ile ilişkili veriler arşiv taraması yoluyla retrospektif olarak elde edildi. Hastalar, AKBM sonuçlarına göre dipper (75 hasta) ve non-dipper (65 hasta) şeklinde alt gruplara ayrıldı. PSD velositesi ile sirkadiyen patern ve aortik sertlik arasındaki ilişki incelendi.

Bulgular: PSD tepe velositesi, dipper ve non-dipper HT hasta alt grupları arasında benzerdi. Spearman korelasyon analizinde PSD ile aortik sertlik indeksi (r=0.286; p=0.007), transmitral A velositesi (r=0.260; p=0.017), E/A oranı (r= -0.265; p=0.015) ve lateral E' velositesi (r= -0.279; p=0.013) arasında anlamlı korelasyon izlendi. Çok değişkenli lineer regresyon analizinde aortik sertlik indeksinin (β =0.525, p<0.001), yaşın (β =0.239, p=0.009), lateral E/E' oranının (β =0.636, p<0.001) ve E/A oranının (β =-0.503, p<0.001), PSD velositesi için önemli öngördürücüler olduğu tespit edilmiştir.

Sonuç: Şayet mevcut ise, yeni tanı hipertansiyon hastalarında SVÇY üzerinden ölçülen PSD velositesi, TTE ile değerlendirilen aortik sertlik derecesi ve sol ventrikül kompliansı ile ilgili bilgi sağlayabilmekte olup sirkadiyen kan basıncı paternini öngördürmede yarar sağlamamaktadır.

Anahtar Kelimeler: Presistolik dalga; Aortik sertlik; Sirkadiyen patern; Esansiyel hipertansiyon

ABSTRACT

Background and Aim: Non-dipping circadian pattern and increased aortic stiffness have been linked to increased morbidity and mortality in patients with hypertension (HT). Our aim in the present study was to assess whether presystolic wave (PSW) velocity measured by Doppler transthoracic echocardiography (TTE) on left ventricular outflow tract (LVOT) had relationship with circadian pattern and the aortic stiffness parameters assessed by TTE.

Methods: A total of 140 newly-diagnosed essential HT patients admitting to the cardiology outpatient polyclinic were consecutively included. Data regarding physical examination, laboratory analysis, TTE, and ambulatory blood pressure monitoring (ABPM) were obtained retrospectively from medical records. Patients were divided into dipper (n=75) and non-dipper (n=65) groups on the basis of ABPM. Relationship of PSW velocity with circadian pattern and aortic stiffness was evaluated.

Results: Peak PSW velocity was similar between dipper and non-dipper HT subgroups. Spearman's correlation analysis showed a positive correlation of PSW velocity with aortic stiffness index (r=0.286; p=0.007), transmitral A velocity (r=0.260; p=0.017), E/A ratio (r=-0.265; p=0.015), and lateral E' velocity (r=-0.279; p=0.013). In multivariate lineer regression model, aortic stiffness index (β=0.525, p<0.001), age (β=0.239, p=0.009), lateral E/E' ratio (β=0.636, p<0.001) and E/A ratio (β=-0.503, p<0.001) emerged as significant predictors of PSW velocity.

Conclusion: Peak velocity of PSW on LVOT, if present, may provide information about the status of aortic stiffness and LV compliance assessed by TTE. However, PSW velocity is not useful in the prediction of circadian blood pressure pattern in newly-diagnosed essential HT patients.

Keywords: Presystolic wave; Aortic stiffness; Circadian pattern; Essential hypertension

INTRODUCTION

In spite of dramatic improvement in the therapy and more precise diagnostic modalities, essential hypertension (HT) has still been an explicit reason for morbidity and mortality throughout the world (1-4). Beside the fact that blood pressure (BP) measurement during an office visit has still been the diagnostic modality of choice (5), a 24-hour ambulatory BP monitoring (ABPM) provides further insight into such other conditions as white-coat HT and circadian variations in BP. Normally, ≥10% fall in nocturnal BP is expected compared with the daytime BP, which is termed as ''dipping BP'' pattern. On the other hand, non-dipping pattern is characterized by a <10% decline in BP during nighttime (6). Furthermore, previous studies suggested that presence of a non-dipping circadian BP pattern was a prominent cardiovascular (CV) risk identifier and hence a worse prognostic indicator compared with the dipping BP pattern in HT patients (7).

Chronic exposure to high BP, as in the case of HT, confers impairment on the elastic nature of the aorta by disrupting endothelial functions and inciting unfavorable remodeling in the aortic medial layer. It has already been shown in the previous studies that HT patients are more prone to aortic stiffness compared with the healthy normotensive people. Furthermore, non-dipper subgroups were also reported to be more inclined to the development of aortic stiffness than the dipper subgroups among patients with HT (8, 9). Accordingly, gradual impairment in the aortic elastic properties renders HT patients more susceptible to the worsening CV dynamics and poorer prognosis (9-12).
Presystolic wave (PSW) is an image which is frequently, albeit not necessarily, observed on the outflow tract of the left ventricle (LVOT) later in the diastolic period upon Doppler echocardiographic imaging (13, 14). Although PSW's exact prevalence and clinical importance in diverse medical conditions are not well recognized, preliminary evidence suggested that its absence could indicate adverse CV events in patients with overt left ventricular (LV) failure (14, 15), subclinical LV dysfunction in asymptomatic HT patients (16), higher Syntax score in patients with acute myocardial infarction (15), worsening arterial elasticity in asymptomatic subjects (13), and increased carotid plaque burden in patients with multiple CV comorbidities (17). Not only its absence, but its velocity was also proposed to have clinical implications (13, 14, 16, 17). However, there is no study investigating the relationship of PSW velocity with circadian pattern and aortic stiffness assessed by transthoracic echocardiography (TTE) in newly diagnosed HT cases. Therefore, we aimed hereby to investigate the relationship of PSW velocity both with circadian BP pattern and echocardiographic parameters regarding aortic stiffness in patients given a new diagnosis of HT.

METHODS

Patient Population

Our study was conducted retrospectively in a single tertiary healthcare center. 163 patients admitted to our cardiology outpatient clinics between September 2017 and August 2018 with pertinent symptoms and diagnosed with a new essential HT were enrolled consecutively. Collected data by way of physical examination, comprehensive history taking, laboratory analysis, TTE, and ABPM were retrieved from medical records. We excluded a total of 23 patients from the study due to the absence of PSW on Doppler echocardiography, and the remaining 140 were further divided to two subgroups as dipper (n=75) and non-dipper (n=65) according to their ABPM recordings. None of the patients was on any antihypertensive therapy on admission. We calculated the body-mass index (BMI) by dividing the weight (kg) with the square of height (meter). We measured the office BPs from the brachial artery with the help of a commercially available mercury sphygmomanometer (Erka Perfect Aneroid, Berlin, Germany) after having the patients seated for 10-minute duration. Furthermore, BP measurement was repeated two more times at 5-minute intervals, hence obtaining a total of three measurements. We averaged these three consecutive measurements and ended up with the final BP. BP measurements were performed between 8 and 10 am on day of polyclinic admission. The patients with average BP of ≥140/90 mmHg and without an obvious secondary disease conducive to HT were given a diagnosis of essential HT (18). We set the exclusion criteria as follows: secondary hypertension, absence of PSW on TTE examination,

diabetes mellitus, severe renal failure, atherosclerotic CV disease, chronic obstructive pulmonary disease, endocrinologic diseases, acute infections, chronic inflammatory illnesses, and being on anti-inflammatory or steroid drugs. Our study protocol is compatible with the standards cited in Helsinki Declaration. Moreover, the institutional committee for ethics provided an approval for the protocol of this study (No: 2018-17/147, Date: 25/09/2018).

Echocardiography and ABPM

Echocardiographic assessment of the enrolled patients was implemented by using Vivid Echocardiography device (Vivid S5, GE Vingmed Ultrasound AS, Horten, Norway). With the patient on the lateral decubitus position, parasternal long-axis imaging of heart was achieved, where dimensions and wall thicknesses of the ventricles, together with the diameter of left atrium, were measured, as per the relevant guideline (19). Modified Simpson's method was utilized to determine the left ventricular ejection fraction (LVEF). Early (E) and late (A) transmitral inflow velocities and E-deceleration time (EDT) were measured from the apical four chamber view using pulse-wave Doppler (PWD) imaging. In tissue Doppler imaging mode, a sample PWD was places on the lateral and septal mitral annular ends to measure respective early/late diastolic annulus velocities (E' and A' velocities, respectively). Transmitral E velocity was divided by the respective early diastolic mitral annulus velocities and hence septal and lateral E/E' ratios were obtained. Presence of PSW was sought by placing the PWD sample in LVOT just proximal to the aortic valve in apical 5-chamber view, and peak velocity of PSW was recorded if present. Diameters of ascending aorta during systole and diastole were measured at a 3-cm distance from the aortic valve in M-mode in the parasternal long-axis view (20). Systolic aortic diameter (AoSD) was measured when a maximal anterior aortic motion was observed, while diastolic aortic diameter (AoDD) was measured at peak the electrocardiographic QRS complex (21). Aortic diameter change (AoDC) was calculated by abtracting AoDD from AoSD. The parameters indicating aortic elastic properties were calculated by taking the arithmetic mean of the relevant parameter recorded during at least 3 cardiac cycles. The following 3 parameters pertaining to the elastic property of aorta were defined as follows:

-Aortic strain (%) = (AoSD-AoDD) × 100/AoDD

-Aortic stiffness (β) index = ln (SBP/DBP)/ (AoSD - AoDD)/AoDD

-Aortic distensibility (cm2/dyn -1.10-6) = 2 × (AoSD-AoDD)/AoDD)/SBP-DBP (22, 23).

A devise for ABPM (Bravo, Sun Tech Medical Inc., Morrisville, NC, USA) was applied to any patient with office BP ≥ 140/90 mmHg. BP readings were recorded with 15 minute intervals during daytime (6 am -10 pm) and 30 min intervals during nighttime (10 pm -06 am). The patients were asked to continue the routines in their daily life on the day of ABPM, but stand still while cuff inflation. Overall, the BP recordings were analyzed to obtain 24-hour mean systolic BP, 24-hour mean diastolic BP, daytime mean systolic BP, daytime mean diastolic BP, nighttime mean systolic BP and nighttime mean diastolic BP in every patient. The diagnosis of HT was ascertained when at least one of the followings was detected: 24-hour mean systolic BP >130 mmHg and/or diastolic BP >80 mmHg; daytime mean systolic BP >135 mmHg and/or diastolic BP >85 mmHg; and, nighttime systolic BP >120 mmHg and/or diastolic BP >70 mmHg (24). Dipping BP pattern was accepted to be a ≥10% BP decline during nighttime. On the other hand, <10% BP decline during nighttime was considered as non-dipping pattern.

Statistical Analysis

All the statistical analysis of the study was fulfilled by SPSS (SPSS for Windows, Version 21.0. Armonk, NY: IBM Corp., USA). Normal distribution of the quantitative study variables was assessed with the use of Kolmogorov- Smirnov and Shapiro-Wilk tests. Continuous variables were expressed in mean±SD and median (25-75 IQR), whereas categorical variables were expressed as numbers (%). Comparison of the pertinent variables between the groups was performed by use of Chi-Square test and Mann-Whitney-U test. The correlation between PSW velocity and other variables was analyzed using Spearman's correlation analysis. A further multivariate linear regression analysis was implemented in an attempt to specify the best predictive model for PSW velocity. A two-sided P <0.05 was accepted to indicate statistical significance.

RESULTS

Table 1 displays the demographic and clinical features of the study patients. Overall, mean age was 47±6.5 years (74 female, 66 male), and mean BMI was 26.9±1.6 kg/m2. HT patients were separated into dipper group (75 patients; 53.3% female; mean age 46.8±4.8 years) and non-dipper group (65 patients; 52.3% female; mean age 45.4±6.7 years). Table 2 represents the comparison of the 2 subgroups. Gender and age distribution were homogeneous between the 2 subgroups, and both HT subgroups were similar in terms of BMI and laboratory findings.

Table 1. Demographic, clinical and echocardiographic characteristics of whole study population.

Variables	**N=140**
Age (years)	47±6.5
Gender (female, %)	74 (52.8)
BMI (kg/m2)	26.9±1.6
Glucose (mg/dL)	89.8±4.7
GFR (mL/min/1,73 m2)	97.3±14.3
ALT (U/L)	23.6±12.8
Triglyceride (mg/dL)	169.5±75
Total Cholesterol (mg/dL)	186.2±34.6
LDL (mg/dL)	105±28
HDL (mg/dL)	46.2±11
C-RP (mg/dL)	0.42±0.35
WBC (x109/L)	8.1±1.8
Hb (gr/dL)	14.9±1.9
Echocardiography	
IVS (mm)	10.3±0.9
PWT (mm)	9.3±1.9
LVEDD (mm)	45.3±9.3
LVEF (%)	63.3±2.2
Left atrium (mm)	33.9±8.3
Transmitral E velocity (cm/s)	74.2±21
Transmitral A velocity (cm/s)	78.07±16.99
Transmitral E/A ratio	0.96±0.22
EDT (ms)	202.1±56.9
Septal E' velocity (cm/s)	9.3±2.2
Septal A' velocity (cm/s)	12 ±2.8
Lateral E' velocity (cm/s)	11.4±3.6
Lateral A' velocity (cm/s)	12.8±2.9
Septal E/E' ratio	7.7±2.5
Lateral E/E' ratio	6.7±2.4
PSW velocity (cm/s)	54.7±14.1
AOSD (mm)	32±5
AODD (mm)	30.2±4.9
AODC (mm)	1.74±0.9
Aortic Strain (%)	6.8±2.9
Aortic Distensibility (cm2.dyn -1. 10-6)	3.5±1.9
Aortic stiffness (β) index	8.8±7.7
ABPM readings	
24-hour SBP (mmHg)	133.8±10.8
24-hour DBP (mmHg)	86.5±7.7
Daytime SBP (mmHg)	136.1±11.5
Daytime DBP (mmHg)	88.1±8.4
Nighttime SBP (mmHg)	126.4±12.3
Nighttime DBP (mmHg)	80.5±9.4

BMI: body-mass index, GFR: glomerular filtration rate, ALT: alanine aminotransferase, LDL: low density lipoprotein cholesterol, HDL: high density lipoprotein cheolesterol, C-RP: C-reactive protein, WBC: white blood cell count, Hb: hemoglobin, ABPM: ambulatory blood pressure monitoring, SBP: systolic blood pressure, DBP: diastolic blood pressure, IVS: interventricular septum thickness, PWT: posterior left ventricular wall thickness, LVEDD: left ventricular end-diastolic diameter, LVEF: left ventricular ejection fraction, EDT: transmitral E-deceleration time, PSW: presystolic wave, AoSD: Systolic aortic diameter, AoDD: Diastolic aortic diameter, AoDC: Aortic diameter change.

As for TTE, E/A ratio (p<0.001) and septal E' velocity (p=0.031) were lower in non-dipper group compared with the dippers. Transmitral A velocity, septal A' velocity and lateral A' velocity were greater in non-dippers compared with the dippers (p<0.001). Aortic strain as well as the aortic distensibility were found to be lower, while aortic stiffness (β) index was found to be greater in non-dippers compared with the dippers (p<0.001). Mean AoSD was observed to be similar between the groups, whereas mean AoDD was greater (p=0.032) and AoDC was lower (p=0.008) in the non-dipper group compared with the dipper group. Median PSW was greater in non-dippers compared with the dipper group, but this difference failed to reach a level of statistical significance [55.5 cm/s (48~64.7) vs 48.7 cm/s (46.9~61.1); p=0.543].

Table 2. Baseline demographic and clinical features of non-dipper and dipper hypertensive patient groups.

Variables	Non-Dipper HT (n=65)	Dipper HT (n=75)	P
Gender (female, %)	34 (52.3%)	40 (53.3%)	0.484
Age (years)	45.4±6.7	46.8±4.8	0.345
BMI (kg/m^2)	27.4±1.6	26.9±1.5	0.415
Glucose (mg/dL)	89.9±4.3	90.7±5.5	0.974
GFR (mL/min/1,73 m^2)	94.3±13.5	96.6±11.2	0.654
ALT (U/L)	22.8±12.3	24.9±12.1	0.289
Triglyceride (mg/dL)	167.8±74.4	179±78	0.541
Total Cholesterol (mg/dL)	185.7±37.5	186 ±33.1	0.543
LDL (mg/dL)	103.3±29	108±27	0.212
HDL (mg/dL)	48.1±12.7	45.4±12.1	0.075
C-RP (mg/dL)	0.44±0.24	0.39±0.29	0.502
WBC (x109/L)	7,76±1,7	8,50±2,3	0,108
Hb (gr/dL)	15.3±1.4	15.1±2.3	0.244
ABPM readings			
24-hour SBP (mmHg)	133.31±9.3	134.41±12.3	0.456
24-hour DBP (mmHg)	85.2±8.4	87.01±8.5	0,454
Daytime SBP (mmHg)	134±9.4	139.2±14.2	<0.001
Daytime DBP (mmHg)	84.82±7.6	90.2±8.6	<0.001
Nighttime SBP (mmHg)	132.2±11.4	120.96±11.2	
Nighttime DBP (mmHg)	83.4±8.5	76.5±8.6	<0.001
Echocardiography			
IVS (mm)	10.80 (10.30~11.22)	10.40(10.20~11.00)	0.958
PWT (mm)	10.00(8.90~10.27)	9.30(9.00~10.00)	0.394
LVEDD (mm)	46.25(44.0~49.90)	46.00(45.10~50.0)	0.515
LVEF (%)	63.5(61.0~65.0)	63(62.0~65.0)	0.567
Left atrium (mm)	37.3(34.00~40.60)	33(32.0~36.70)	0.195
Transmitral E velocity (cm/s)	65.6(60.90~76.70)	69.8(46.40~83.00)	0.382
Transmitral A velocity (cm/s)	84.25(69.30~93.40)	68.60(50.30~85.00)	<0.001
Transmitral E/A ratio	0.83(0.70~1.05)	1.14(0.81~1.34)	<0.001
EDT (ms)	192(167.00~236.00)	201(165.00~253.00)	0.073
Septal E' velocity (cm/s)	8.85(8.50~9.10)	9.60(7.70~10.65)	0.031
Septal A' velocity (cm/s)	12.4(11.70~13.60)	12(10.30~13.45)	<0.001
Lateral E' velocity (cm/s)	10.7(8.40~12.50)	9.7(9.20~13.60)	0.674
Lateral A' velocity (cm/s)	12.85(11.50~14.60)	12.6(10.20~13.70)	<0.001
Septal E/E' ratio	7.41(6.92~8.38)	6.79(4.90~9.83)	0.051
Lateral E/E' ratio	6.14 (5.23~8.25)	6.08 (4.51~8.18)	0.618
PSW (cm/s)	55.5 (48~64.7)	48.7 (46.9~61.1)	0.543
AoSD (mm)	33.3 (31.3~34.4)	31.8 (31.6~35)	0.063
AoDD (mm)	30.8 (29.2~33.6)	29.5 (28.2~32.7)	0.032
AoDC (mm)	1.84 (0.6~2.4)	2.22 (1.6~3)	0.008
Aortic Strain (%)	5.44 (3.2~6.76)	8.31(7.37~9.9)	<0.001
Aortic Distensibility (cm2.dyn -1. 10-6)	2.11 (1.27~2.74)	4.78 (3.61~5.93)	<0.001
Aortic stiffness (β) index	8.9 (6.42~15.2)	4.61(3.82~5.3)	<0.001

BMI: body-mass index, GFR: glomerular filtration rate, ALT: alanine aminotransferase, LDL: low density lipoprotein cholesterol, HDL: high density lipoprotein cheolesterol, C-RP: C-reactive protein, WBC: white blood cell count, Hb: hemoglobin, ABPM: ambulatory blood pressure monitoring, SBP: systolic blood pressure, DBP: diastolic blood pressure, IVS: interventricular septum thickness, PWT: posterior left ventricular wall thickness, LVEDD: left ventricular end-diastolic diameter, LVEF: left ventricular ejection fraction, EDT: transmitral E-deceleration time, PSW: presystolic wave, AoSD: Systolic aortic diameter, AoDD: Diastolic aortic diameter, AoDC: Aortic diameter change

According to the Spearman's analysis, PSW velocity showed a positive and significant correlation with aortic stiffness (β) index (r=0.286; p=0.007); however, aortic distensibility and aortic strain did not show correlation with PSW velocity. Moreover, PSW velocity was positively correlated with transmitral A velocity (r=0.260; p=0.017), while it was correlated negatively with E/A ratio (r=-0.265; p=0.015) and lateral E' velocity (r=-0.279; p=0.013) (Table 3).

Tablo 3. Spearman's Rho values demonstrating the correlation between PSW and echocardiographic parameters.

Variables	Rho	P
Aortic strain	-0.187	0.234
Aortic distensibility	-0.172	0.324
Aortic stiffness (β) index	0.286	**0.007**
Transmitral A velocity	0.260	**0.017**
Transmitral E/A ratio	-0.265	**0.015**
Lateral E' velocity	-0.279	**0.013**

On the other hand, no significant correlation between PSW velocity and 24-hour, daytime and nighttime ABPM recordings was evident (p>0.05).

In multivariate linear regression analysis with the best predictive model (R^2=0.85, p<0.001), age (β=0.239, p=0.009), aortic stiffness (β) index (β=0.525, p<0.001), lateral E/E' ratio (β=0.636, p<0.001) emerged as positive and significant predictors of PSW velocity, while E/A ratio (β=-0.503, p<0.001) was found to be a significantly negative predictor of PSW velocity (Table 4).

Tablo 4. Multivariate linear regresyon analysis showing independent predictors of PSW.

Variables	Coefficient (β)	P
Lateral E/E' ratio	0.636	<0.001
Aortic stiffness (β) index	0.525	<0.001
E/A ratio	-0.503	**<0.001**
Age	0.239	**0.009**

DISCUSSION

Our study mainly demonstrated that PSW velocity did not display significant difference between dipper and non-dipper hypertensive subjects, and hence seemed inappropriate in the anticipation of circadian BP pattern in newly diagnosed patients with essential HT. Secondly, patients with non-dipper HT had more pronounced stiffness in the aorta compared with the dipper HT patients, and aortic stiffness (β) index was positively and significantly associated with PSW velocity, if present. In addition, lower E/A ratio, greater lateral E/E' ratio and older age were the other significant predictors of higher PSW velocity in HT patients. To the best of our knowledge, this is the first study to evaluate the relationship of PSW velocity with circadian BP pattern and aortic stiffness assessed by TTE in newly diagnosed essential HT patients with a documented PSW on Doppler echocardiographic examination.

The prevalence of PSW encountered on a routine Doppler examination of LVOT is not well known, and estimated to be around %66-95% of all echocardiograms (14, 25, 26). The prevalence of PSW on LVOT in our study was 85.8%, which is compatible with the previous reports.

Studies on PSW are scanty. Moreover, they mainly dealt with possible effect of presence or absence of PSW, and occasionally the velocity of PSW, on various disease conditions. Although the exact mechanism with which PSW appears on LVOT is not fully understood, it was proposed to occur as a result of a reflection of a votex formed by the early and late diastolic transmitral flows onto LVOT (14, 27, 28). In this regard, a PSW is not a trans-aortic flow, but rather a preparation for ejection during an imminent ventricular systole (14), and progressive deterioration in LV relaxation and compliance increases not only the likelihood of a PSW occurrence, but also the PSW's velocity itself. Bryg et al. (27) demonstrated an increase in PSW velocity in patients with hypertrophic LV geometry more than the cases without LV hypertrophy. Joshi et al.(14) found greater transmitral A velocity and septal A' velocity in patients with documented LVOT PSW compared with the patients without PSW. Mittal and pancholi (29)

demonstrated in their study a significantly positive correlation of PSW velocity with transmitral A velocity and a significantly negative correlation with E/A ratio. They further concluded that PSW peak velocity could be used as a marker of impaired LV compliance. Moreover, Akyüz et al. (16) reported that PSW velocity correlated positively with lateral A' velocity and negatively with E/A ratio in HT patients. Similarly to the previous studies, we demonstrated a significantly positive correlation of PSW velocity with tranmitral A velocity, and a significantly negative correlation with E/A ratio and lateral E' velocity. Apart from the previous reports, we further revealed in the regression model a significant association between lateral E/E' ratio, another surrogate marker of impaired LV compliance, and PSW velocity.

Impairment in aortic and arterial elasticity contributes to CV morbidity and mortality in HT cases (11), as well as in those subjects without other CV disease risk factors (30). Not much has been known about the relationship between arterial stiffness and PSW. Korkmaz et al.(13) demonstrated a positive correlation between PSW velocity and arterial stiffness assessed by cardioankle vascular index (CAVI) in asymptomatic individuals with cardiometabolic comorbidities. Additionally, they also reported that age, male sex and CAVI were independent predictors of PSW velocity. On the other hand, we found in our study a significant and positive correlation between aortic stiffness (β) index assessed by TTE and PSW velocity. Also, aortic stiffness (β) index emerged in our study as a significant predictor of PSW velocity in newly diagnosed HT patients. Keeping in mind that the current medical literature lacks studies assessing PSW and echocardiographic aortic stiffness, this study may serve as a preliminary data for further studies in this scope.

Lack of correlation between PSW velocity and circadian BP pattern in HT patients in our study could in part be ascribed to the hypothesis that it is atrial contractility and ventricular relaxation/compliance which determines both the presence and the velocity of PSW on LVOT to a greater extent, rather than the sole circadian BP pattern. In addition, absence of correlation of PSW velocity with 24-hour, nighttime and daytime mean BP measurements by ABPM in our study supports this hypothesis.

This study should be assessed with some limitations. First, our study population is relatively small and larger study cohorts may reveal correlation between PSW velocity and the other echocardiographic parameters of aortic elasticity, such as aortic strain and aortic distensibility. Hence, the results of this study may not be generalized to the general population, unless future larger-scale studies are conducted. We also did not correlate our findings with other methodologies for the measurement of arterial stiffness.

CONCLUSİON

Evaluation of the peak velocity of PSW via Doppler echocardiography in newly diagnosed essential HT patients may provide crucial information regarding aortic stiffness and LV compliance; however, PSW velocity does not seem useful in the anticipation of the circadian BP pattern. Future investigations encompassing larger cohorts are warranted to support our findings.

Conflict of Interests: We declare this study possesses no conflict of interests.

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