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ORIGINAL ARTICLE

The Association Between Presystolic Wave and Subclinical Left Ventricular Dysfunction in Asymptomatic Hypertensive Patients: Speckle-Tracking Echocardiographic Study

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Abstract

Background: Presystolic waves are often found during Doppler evaluation of the left ventricular outflow tract (LVOT) in hypertensive individuals. LV stiffness and altered LV compliance are potential mechanisms for presystolic waves.

Objectives: This study's objective was to examine the relationship between presystolic wave and subclinical left ventricular (LV) impairment in asymptomatic individuals with essential hypertension.

Methods: This observational research comprised 87 individuals with essential hypertension. All patients had two-dimensional and Doppler echocardiography performed. Based on the existence or absence of a presystolic wave, patients were separated into two groups. Using speckle-tracking echocardiography, subclinical LV systolic impairment was identified. Data were analyzed using IBM SPSS version 20.0. The significance level adopted in the statistical analysis was 5%.

Results: The mean age of the studied patients was 51 ± 9 years, with a male percentage of 57.4%. Among them, 57 patients (65%) had a presystolic wave, and 30 (35%) did not. Left ventricular mass index (LVMI) was greater in patients with a presystolic wave in comparison with patients without it (105.8 ± 16.1 g/m² versus 99.8 ± 9.47 g/m², p-value = 0.03). Left atrial volume index (LAVI) was higher in patients with a presystolic wave in comparison with patients without it (28.9 ± 5.25 ml/m² versus 26.3 ± 2.74 ml/m², p-value = 0.016). Patients with presystolic wave demonstrated LV diastolic dysfunction more than patients without it (p-value = 0.024). Left ventricular global longitudinal strain (LVGLS) was lower in patients with a presystolic wave in comparison with patients without it (-20.2 ± 2.55 versus -21.7 ± 2.27 % with p-value = 0.008). Patients with presystolic wave demonstrated more subclinical LV systolic dysfunction than patients without it (p-value = 0.025).

Conclusion: The presystolic wave was linked to subclinical LV impairment. The existence of a presystolic wave may indicate hypertensive people who are at risk of developing overt heart failure.

Keywords: Essential Hypertension; Vascular Stiffness; Echocardiography

Introduction

Arterial hypertension is one of the largest risk factors for cardiovascular disease, linked to an increase in morbidity and death.[1,2] The risk of cardiovascular events may be underestimated in hypertension individuals without symptoms.[3] Long-term uncontrolled hypertension promotes left ventricular (LV) hypertrophy, which causes a steady decrease in LV function and finally leads to heart failure.[4] To reduce the occurrence of cardiovascular events, it is crucial to identify patients at risk for LV dysfunction quickly.

Presystolic waves are typically found during Doppler investigation of the left ventricular outflow tract (LVOT) as a late diastolic event. A proposed explanation of presystolic wave is LV stiffness and decreased LV compliance.[5,6] In almost two-thirds of all echocardiograms, regardless of whether the left ventricular ejection fraction (LVEF) is normal or decreased, a presystolic wave is seen.[7] The lack of a presystolic wave in individuals with a decreased LVEF may be predictive of unfavorable cardiovascular events.[8]

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Central Illustration: The Association Between Presystolic Wave and Subclinical Left Ventricular Dysfunction in Asymptomatic Hypertensive Patients

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The association between presystolic wave and subclinical left ventricular dysfunction in asymptomatic hypertensive patients

87 individuals with essential hypertension

	Patients with presystolic wave (n=57)	Patients without presystolic wave (n=30)	P value
Subclinical LV systolic dysfunction* (n., %)	18 (31.6%)	3 (10%)	0.0253

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*GLS: Global longitudinal strain. *Assessed by GLS.*

This study aimed to investigate the relationship between presystolic wave and subclinical LV dysfunction in asymptomatic people with essential hypertension.

Subjects and methods

This observational study was done on 87 individuals from January 2022 to December 2022 at the cardiology outpatient clinic of our University Hospital. The research adhered to the Declaration of Helsinki and was authorized by the institutional review board (IRB permit number 2/2019CARD). Before participating in the study, each participant filled out an informed consent form.

Methods

Each patient's medical history included a review of cardiovascular risk factors (hypertension, diabetes, dyslipidemia, and smoking), and their physical examination included body mass index (BMI) calculation. After a period of rest, the right arm blood pressures of sitting patients were measured using a WXB-50 sphygmomanometer. First-time cases with blood pressure more than 140/90 mmHg were evaluated a minimum of twice on two distinct dates. The exclusion criteria were secondary hypertension, coronary artery disease, stroke, decompensated heart failure, chronic liver disease, chronic renal illness, atrial fibrillation, and pregnancy.

Resting electrocardiography (ECG)

All patients received a 12-lead ECG utilizing Biocare iE 300 resting ECG analysis equipment with a paper speed of 25 mm/s and a standardization of 1 mm/mv. Evaluation of cardiac rhythm removes participants with atrial fibrillation from the research.

Two-dimensional echocardiography and Doppler echocardiography

An experienced physician was blinded to the demographic and clinical features of the patients while evaluating each participant using VIVID E9 equipment (GE Healthcare, Chicago, IL, USA). Using normal imaging perspectives, the mean of three consecutive heartbeats was then computed. We conducted conventional 2D and M-mode measurements. The Devereux equation was used to determine the left ventricle's mass (LVM). The LVM index was determined by dividing the LVM by the surface area of the body.[9] LV hypertrophy was diagnosed in men with an LVM index of more than 115 g/m^2 and in females with an LVM index greater than 95 g/m^2.

M-mode echocardiography was used to test LVEF.[10] When recording both 2D and conventional Doppler variables,[11] the guidelines of the American Society of Echocardiography were followed. The diastolic function of the LV was assessed utilizing Tissue Doppler Imaging (TDI), Pulsed Doppler Echocardiography, left atrial volume index (LAVI), and the velocity of tricuspid regurgitation (TR) in accordance with

the recommendations of the American Society of Echocardiography.[12]

In the ventricular apical five-chamber view, a pulse wave Doppler evaluation of the LVOT was done immediately proximal to the aortic valve. All patients were examined for the existence of a presystolic wave prior to LVOT flow (Figure 1). Patients were divided into two groups based on the presence or absence of a presystolic wave.

Speckle-tracking echocardiography

Three cycles of apical views were acquired for longitudinal strain measurement: 4, 2, and 3 chamber views. It was decided that the frame rate would be between 40 and 90, or at least 40% of HR. Then, after the activation of automated function imaging, digital data were sent to the Vivid Nine System Echo Pac from GE Vingmed in Horton, Norway, for offline analysis. Subclinical LV systolic dysfunction was diagnosed by utilizing a global longitudinal strain (GLS) value of less than -19%.[13]

Statistical analysis of the data

Data were imported into the computer and analyzed using IBM SPSS version 20.0. IBM Corporation of Armonk, New York. The categorical variables were expressed through absolute and relative frequencies. Two groups were compared using the Chi-square test. Using the Kolmogorov-Smirnov test, the normality of continuous data was evaluated. Continuous variables with normal distribution were described using mean ± standard deviation, and those without normal distribution were described using median and interquartile range. Unpaired Student's t-test was used to compare two quantitative groups with normally distributed variables. The Mann-Whitney test was devised to compare two groups having quantitative traits that are not normally distributed. Logistic regression analysis was used to detect factors affecting the presence of the LVOT presystolic wave. The significance level adopted in the statistical analysis was 5%.

Results

A. Patient characteristics and risk factors:

The examined population consisted of 87 individuals with a mean age of 51 ± 9 years. The proportion of men was 57.4%. A total of 20 patients (23%) had diabetes mellitus, and 28 patients (32%) had dyslipidemia. The average blood pressure in the workplace was 139.8/85.3 mmHg. During the LVOT pulsed Doppler test, 57 patients (65%) had a presystolic wave, while 30 patients (35%) did not. Similar demographic and laboratory variables existed across both groups (Table 1).

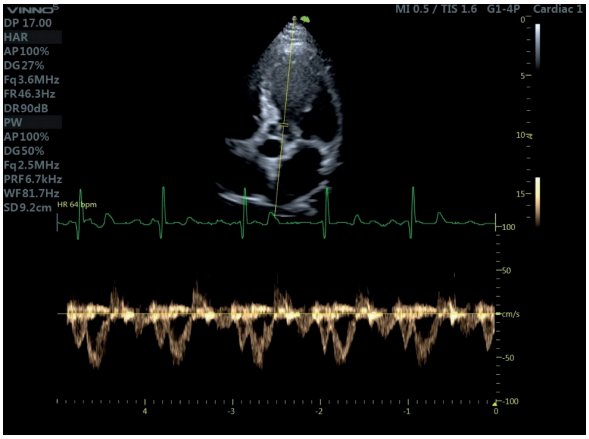

Figure 1 – Pulse wave Doppler evaluation in LVOT demonstrating presystolic wave

B. Doppler and two-dimensional echocardiographic data:

Table 2 displays the two-dimensional and Doppler echocardiographic data for both groups. Left ventricular mass index (LVMI) was greater in those with presystolic wave than in those without it. Individuals with presystolic waves had larger LAVI than those without. Individuals with presystolic wave had a lower mitral E/A ratio and TDI e' than those without presystolic wave (statistically significant). Patients with presystolic wave were more likely than those without to develop LV diastolic dysfunction (statistically significant).

C. Data from speckle-tracking echocardiography:

Table 3, Figures 2 and 3, and the Central Illustration demonstrate speckle-tracking echocardiographic data of the studied patients. Left ventricular global longitudinal strain (LVGLS) were lower in individuals with a presystolic wave compared to those without (-20.2 ± 2.55 versus -21.7 ± 2.27 %, respectively). Those with a presystolic wave exhibited a higher prevalence of subclinical LV systolic dysfunction than patients

Table 1 – Demographic and laboratory characteristics of the studied patients

	Presystolic wave present (n = 57)	Presystolic wave absent (n = 30)	P-value
Age (years)	52.3 ± 8.16	51.1 ± 9.21	0.547
Sex			
Male	32 (56.1%)	18 (60.0%)	0.729
Female	25 (43.9%)	12 (40.0%)	
BMI (kg/m²)	31 ± 2.63	30 ± 4.06	0.228
Diabetes mellitus (n., %)	12 (13.8%)	8 (9.2%)	0.611
Dyslipidemia (n., %)	21 (36.8%)	7 (23.3%)	0.200
Smoking (n., %)	19 (33.3%)	9 (30.0%)	0.752
Systolic BP (mmHg)	141.33 ± 13.4	137.67 ± 12.5	0.285
Diastolic BP (mmHg)	86.16 ± 6.4	84 ± 7.6	0.194
Serum creatinine (mg/dl)	1.07 ± 0.29	0.95 ± 0.16	0.38
Total cholesterol (mg/dl)	199 ± 35.3	193.2 ± 33.7	0.367
LDL-C (mg/dl)	131.5 ± 33.5	127.9 ± 33.9	0.461
HDL-C (mg/dl)	38.3 ± 7.1	39.4 ± 6.4	0.941
TG (mg/dl)	142.8 ± 60.4	131.9 ± 34.5	0.469

HDL-C: High-density lipoprotein cholesterol; LDL-C: Low-density lipoprotein cholesterol; TG: Triglycerides; CAD: Coronary artery disease; p: p-value for comparing the two studied groups.

Table 2 – Two-dimensional and Doppler echocardiographic results of the studied patients

	Presystolic wave present (n = 57)	Presystolic wave absent (n = 30)	P-value
IVSD (cm)	1.21 ± 0.19	1.18 ± 0.19	0.449
LVEDD (cm)	5.04 ± 0.40	4.94 ± 0.40	0.266
LVESD (cm)	3.09 ± 0.39	3.09 ± 0.33	0.94
LV ejection fraction (%)	62.4 ± 4.90	63.4 ± 4.96	0.347
LVMI (g/m²)	105.8 ± 16.1	99.8 ± 9.47	0.03
Left atrium size (cm)	3.74 ± 0.43	3.48 ± 0.42	0.008
LAVI (ml/m²)	28 (17 – 40)	25.5 (22 – 35)	0.016
TR Velocity (m/s)	2.8 (1.6– 3.86)	2.52 (1.86 – 3.31)	0.003
E (cm/s)	63.8 ± 11.2	79.4 ± 21.4	0.001
A (cm/s)	65.3 ± 12.7	52 ± 10.5	<0.001
E/A ratio	0.9 (0.6 – 2)	1.5 (0.4 – 2.4)	0.002
e' average (cm/s)	8 (5 – 13.5)	13 (9 – 16)	<0.001
E/e'	7.4 (4.4 – 18)	6 (3.7 – 9.4)	<0.001
LV diastolic dysfunction (n., %)	23 (40%)	5 (16.6%)	0.024

IVSD: interventricular septal diameter; LVEDD: LV end-diastolic diameter; LVESD: LV end-systolic diameter; LVMI: LV volume index; LAVI: left atrial volume index; TR velocity: tricuspid regurgitation velocity; E: early peak pulsed Doppler velocity on mitral valve; A: late pulsed Doppler velocity on the mitral valve; e': early tissue velocity of the mitral annulus; MPI: myocardial performance index; p: p-value for comparing between the two studied groups. Continuous variables with normal distribution were expressed by mean ± Standard deviation, and those without normal distribution were expressed by median and interquartile range.

Table 3 – Global systolic strain in the studied groups

	Presystolic wave present (n = 57)	Presystolic wave absent (n = 30)	P-value
Average GLS %	-20.2 ± 2.55	-21.7 ± 2.27	0.008
Subclinical LV systolic dysfunction* (n., %)	18 (31.6%)	3 (10%)	0.025

*GLS: Global longitudinal strain. *Assessed by GLS.*

without a presystolic wave [18 (31.6%) versus 3 (10%), statistically significant].

Table 4 demonstrates logistic regression analysis for the parameters affecting the presystolic wave. Subclinical LV systolic dysfunction and TR velocity were independent factors in the presence of an LVOT presystolic wave.

Discussion

Office systolic/diastolic blood pressure readings ≥ 140/90 mmHg identify hypertension.

Hypertension was predicted to affect around one billion people worldwide, and it was the biggest worldwide cause of early mortality.[14] The leading causes of hypertension-related mortality are ischemic

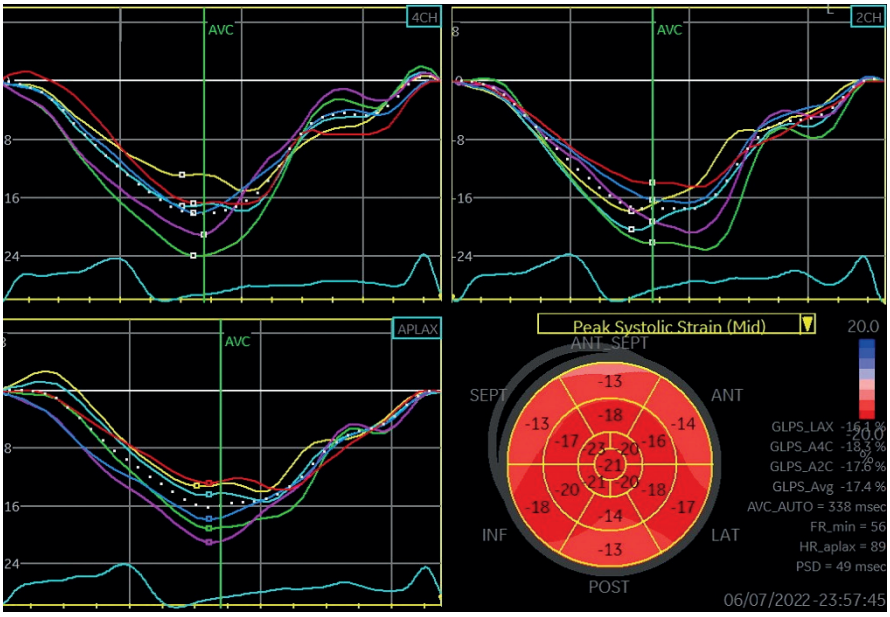

Figure 2 – Peak longitudinal strains of all apical views in a patient with a presystolic wave

heart disease, stroke (both ischemic and hemorrhagic), end-stage renal disease, and heart failure.[15] Lowering blood pressure may significantly decrease morbidity and death in the young. Hypertension continues to be the leading avoidable cause of cardiovascular disease and mortality from all causes.[16]

A presystolic wave is a late diastolic event noticed during Doppler evaluation of the LVOT, and it is believed to be associated with an increase in LV stiffness. In a typical complaint LV, blood enters the LV from the left atrium in a central and posterior direction; however, in a non-complaint LV, some blood flows in the opposite direction (from the septum to the aortic valve), producing a whirlpool, which is interpreted as a presystolic wave by Doppler studies.[5]

The study's main conclusions were (1) a presystolic wave was commonly seen in hypertension individuals, and (2) a presystolic wave was related to subclinical LV diastolic and systolic dysfunction.

In the present research. Patients who experienced a presystolic wave had a greater LVMI than those who did not. LV hypertrophy is considered to be one of the most prevalent causes of increased LV stiffness and altered LV compliance. This result is consistent with the findings of Akyüz et al.,[5] who examined 139 asymptomatic individuals with essential hypertension and observed presystolic wave in 65% of patients. Patients with presystolic wave had a greater LVMI than those who did not.

In the present research, hypertension patients with presystolic wave exhibited a greater peak A, a lower E/A ratio, a lower e', a higher E/e' ratio, and a higher LAVI than those without presystolic wave. We established a relationship between the existence of presystolic wave and subclinical diastolic dysfunction based on these

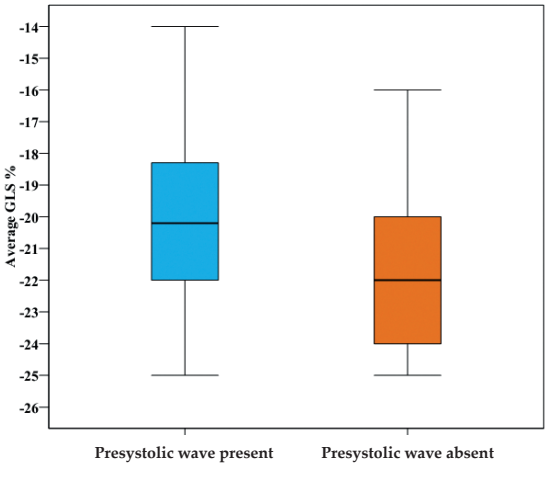

Figure 3 – Average global longitudinal strain (GLS) in the studied patients

data. The LVOT presystolic flow velocity is a sign of LV diastolic dysfunction, as described by Mittal et al.[6] Panayiotou et al.[7] found a direct correlation between presystolic wave velocity and LV wall thickness. Joshi et al.[8] found a substantial connection between presystolic wave velocity and peak A and A' velocities in patients with intact LVEF, but only between presystolic wave velocity and A' in patients with LVEF less than 45%. In addition, they identified an increase in adverse cardiac events in individuals who lacked a presystolic wave and had poor LVEF.

Myocardial performance index (Tie index) is a Doppler-derived echocardiographic indicator that measures both the systolic and diastolic functions of the LV. Akyüz et al.[5] observed that patients with presystolic wave had a higher tie index than those without presystolic wave. Mishra et al.[17] investigated the predictive significance of the Tie index in 1862 people with normal LV systolic function and no ischemic or valvular heart disease. They concluded that the Tie index had modest associations with both clinical and physiological markers of heart function. In addition, it lacks prognostic information on cardiovascular events in this group. Therefore, they did not recommend using the Tie index as a predictive indicator in asymptomatic populations at high risk for cardiac events.

In addition to TDI, we used speckle-tracking echocardiography to identify subclinical LV systolic failure. In prediabetic[18,19] and diabetic[20] individuals, subclinical hypothyroidism[21] and polycystic ovary syndrome,[22] but not in asymptomatic hypertensive patients, the relationship between presystolic wave and subclinical LV dysfunction measured by GLS has been studied. In our research, patients with a presystolic wave had a lower LVGLS than those without. Even though LVGLS mostly represents LV systolic function, there was a correlation between GLS and LV diastolic function. Subclinical longitudinal systolic dysfunction may occur prior to LV diastolic failure. Patients with GLS less than -18% were more likely to have LV diastolic dysfunction, according to a retrospective analysis of echocardiograms of 632 consecutive patients conducted by Yu B et al. The authors determined a threshold value of -15% to distinguish normal LV diastole patients from those with LV diastolic dysfunction.[23]

Table 4 – Logistic regression analysis for the parameters affecting presystolic wave

	Univariate		#Multivariate	
	p	OR (LL – UL 95% CI)	p	OR (LL – UL 95% CI)
Left atrium size	0.011	4.024 (1.373 – 11.789)	0.092	3.134 (0.828 – 11.859)
LAVI	0.018	1.145 (1.024 – 1.282)	0.193	1.097 (0.955 – 1.260)
TR Velocity	0.004	5.403 (1.735 – 16.826)	0.048	4.294 (1.015 – 18.164)
E (cm/s)	<0.001	0.940 (0.908 – 0.973)	0.006	0.946 (0.909 – 0.985)
Subclinical LV systolic dysfunction*	0.034	4.154 (1.113 – 15.504)	0.036	4.937 (1.110 – 21.947)

*LAVI: left atrial volume index; TR velocity: tricuspid regurgitation velocity; E: early peak pulsed Doppler velocity on mitral valve; OR: Odd`s ratio; CI: Confidence interval; LL: Lower limit; UL: Upper Limit, #: All variables with p<0.05 was included in the multivariate. * Assessed by GLS*

Limitations

The primary limitations of the research include the limited sample size, the lack of examination of LV diastolic strain function, and the lack of predictive patient follow-up.

Conclusion

Subclinical LV dysfunction was connected to the presystolic wave. The presence of a presystolic wave may identify hypertensive individuals at risk of developing overt heart failure.

Author Contributions

Conception and design of the research: Yahia M, Soliman M; acquisition of data and statistical analysis: Hashem M; analysis and interpretation of the data and writing of the manuscript: Yahia M; critical revision of the manuscript for intellectual content: Soliman M.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This article is part of the thesis of associate professor submitted by Mohamed Yahia, from Menoufia University.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Menoufia University under the protocol number IRB 2/2019CARD. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

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