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Left Atrial Mechanical Function And Stiffness In Patients With Premature Ventricular Contraction: A Speckle Tracking Study

Aim	Ventricular extrasystole (PVC) is characterized by premature ventricular depolarization and is associated with increased risk of arrhythmias and structural heart disease. This study aimed to investigate the association between the PVC burden and left atrial (LA) function in individuals without known cardiac disease.
Material and methods	A cross-sectional study was conducted on 102 patients with PVCs who were admitted to a cardiology clinic. Transthoracic echocardiography was used to assess left ventricle (LV) parameters, including LV mass, LV ejection fraction (LVEF), LV global longitudinal strain (LVGLS), and LA function was evaluated using strain imaging. The PVC burden was categorized into three groups: <10%, 10–20%, and >20%.
Results	Changes in LV dimensions and LV mass index were associated with the groups with the PVC burden with 10–20%, and >20%. but differences in LVEF and LVGSL were not significant. Mean E/e' increased as the PVC burden increased (p<0.001). The mean global LA peak strain decreased as the PVC burden increased (p<0.001), while other mean LA measurements increased as the PVC burden increased (p<0.001) A higher PVC burden was associated with impaired LA function, as indicated by decreased global LA peak strain (PVC burden <10%=38.1±3.2 vs. PVC burden 10–20%=32.4±3.2 vs. PVC burden >20%=27.7±2.6, in all groups p<0.001) and with increased LA stiffness (PVC burden <10%=18.6±3.2 vs. PVC burden 10–20%=27.5±5.5 vs. PVC burden >20%=39.0±7.9, in all groups p<0.001). A strong negative correlation was found between global LA peak strain and LA stiffness (r=–0.779, p<0.001).
Conclusion	In individuals without known cardiac disease, a higher PVC burden was associated with impaired LA function, indicated by increased E/e', decreased LA strain, and increased LA stiffness. These findings suggest that PVC burden may contribute to LA dysfunction, potentially increasing the risk of cardiovascular events.

Keywords	Premature ventricular contraction; left atrial function; strain imaging; echocardiography
For citations	Tufan Gunay, Selvi Cosar Oztas. Left Atrial Mechanical Function And Stiffness In Patients With Premature Ventricular Contraction: A Speckle Tracking Study. Kardiologiia. 2023;63(10):84–90. [Russian: Туфан Гюнай, Сельви Косар Озтас. Механическая функция и жесткость левого предсердия у пациентов с преждевременным сокращением желудочков по данным speckle tracking эхокардиографии. Кардиология. 2023;63(10):84–90].

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Introduction

Premature ventricular complex (PVC) refers premature ventricular depolarization, characterized by the initiation of ventricular electrical activation earlier than expected in the cardiac cycle [1]. PVCs are an indicator of significant risk in cases of structural heart disease and can be a trigger for life-threatening arrhythmias. In healthy individuals, a PVC rate exceeding 10% of total beats in a 24-hour rhythm recording may lead to exertional dyspnea, while a PVC rate surpassing 20% can potentially contribute to left heart failure or an increase the risk of sudden death [2–4]. Therefore, it is essential to monitor patients with PVCs and, if necessary, develop treatment plans.

In a healthy heart, the electrical impulses propagate smoothly and in a synchronized manner within the ventricles, ensuring an effective contraction. However, PVC can alter the normal ventricular electrical activation pattern, leading to a disruption in intraventricular synchrony. This disruption can lead to impaired left ventricular (LV) function, impacting ventricular systolic performance and overall hemodynamics [5]. Several conventional echocardiographic studies have reported LV enlargement associated with frequent PVCs in patients without structural heart disease [6–8]. Novel studies have shown that strain imaging by deformation analysis could be superior to traditional echocardiographic parameters for the evaluation of myocardial contractile functions [9].

PVCs are thought to play a role in the development of left atrial (LA) dysfunction, which is consistent with observed mechanical, electrical, and structural changes associated with

ISSN 0022-9040. Кардиология. 2023;63(10). DOI: 10.18087/cardio.2023.10.n2567

LA remodeling [10, 11]. Nevertheless, the impact of PVCs on LA function has not been specifically investigated using strain imaging through deformation analysis. This study aimed to examine the correlation between the presence of PVCs and LA function in patients admitted to the hospital due to palpitations, with no known underlying structural heart disease.

Material and methods

Following the principles set forth in the Declaration of Helsinki, this cross-sectional study was conducted between January 2020 and December 2022 at the Department of Cardiology, Bursa City Hospital, Bursa, Turkey. The informed consent of all subjects was obtained, and the study received approval from the local ethics committee (registration number: KAEK 2022–18/8).

Study Population

The 24-hour rhythm Holter findings of 438 patients who were admitted with palpitations during the study period were evaluated. Exclusion criteria were presence of structural heart disease, valvular heart disease, ischemic heart disease, heart failure, myocarditis, pericarditis, cardiomyopathy, sustained ventricular or supraventricular tachycardias, and use of anti-arrhythmic drugs. After the exclusion process, the analysis included 102 patients with PVCs.

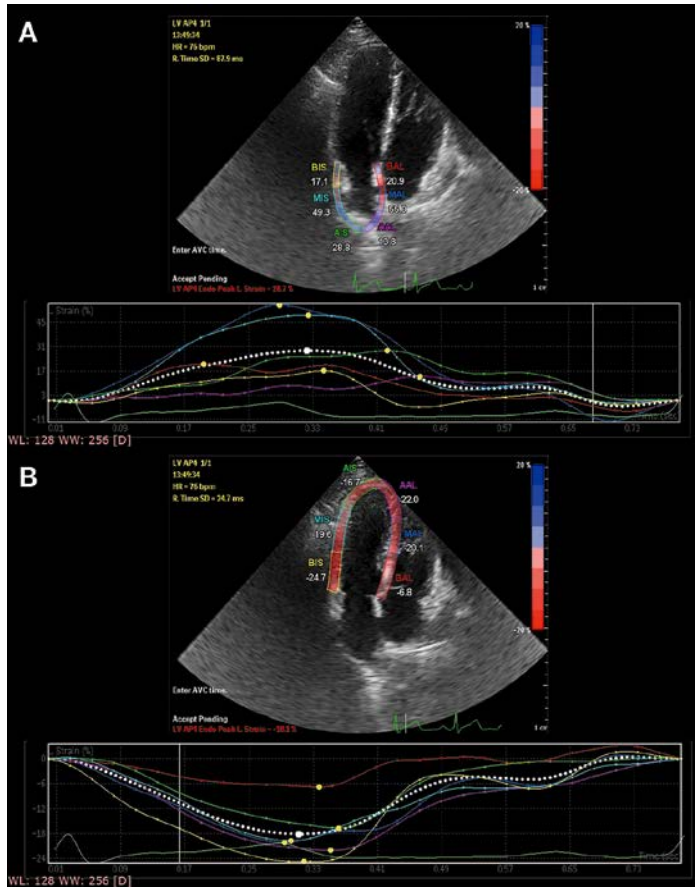
Each patient's baseline LV systolic, diastolic, and detailed LA function was assessed by transthoracic echocardiography. Demographic, clinical, and imaging data were extracted from electronic records. 12-hour fasting venous blood samples were collected during outpatient evaluation to provide biochemical data. All samples were analyzed in the same laboratory with the same device.

Conventional Transthoracic Echocardiography

Transthoracic echocardiography was performed on all patients using the Philips EPIQ 7 Echocardiography device (Philips Healthcare, Koninklijke Philips N.V., Amsterdam, The Netherlands). Parasternal long and short-axis views and apical four, two-chamber, and long-axis views were recorded according to the recommendations of the European Association of Cardiovascular Imaging [12]. The Simpson biplane method was used to calculate LV ejection fraction (LVEF). LV mass (LVM), LV mass index (LVMI), and relative wall thickness (RWT), which is the ratio of LV posterior wall thickness to LV internal dimension at end-diastole (LVDd) were also calculated. The biplane area-length method was used to calculate the LA volume (LAV) and the LA volume index (LAVI) [13].

The study utilized standard Doppler imaging to determine the ratio of peak early (E) to late (A) diastolic LV filling velocity, as well as the E wave deceleration time. The timings of mitral and aortic valve opening and closing were defined using a pulsed-wave Doppler tracing of the mitral inflow and LV outflow. Tissue Doppler parameters e' and a' were measured in both the lateral wall and septum. The E/e' ratio was manually calculated. Special attention was given to minimize interference from myocardial tissue and extracardiac structures, thus ensuring acquisition of reliable data for generating high-quality gray-scale images.

Figure 1. Examples of strain measurements.
A – LA strain. **B** – LV global longitudinal strain



Left Atrial and Left Ventricular Deformation by 2-Dimensional Speckle Tracking

The records of apical 4 chamber views were reviewed off-line and used to calculate endocardial global LA and LV longitudinal strains. All views were recorded during three consecutive beats, and the frame rate was set higher than 60 fps. Analysis was performed with QLAB Enhanced Quantitation Software version 7.1 (Philips Healthcare, Koninklijke Philips N.V., Amsterdam, The Netherlands).

The software automatically generated an epicardial LA silhouette that delineated a region of interest consisting of six segments, followed by automated segment tracking. After tracking, the LA strain pattern is characterized by a dominant upward sloping wave, peaking at the end of ventricular systole, followed by a downward sloping wave to

baseline (Figure 1A). The ratio of E/e' to peak LA strain has been used as an estimate of LA stiffness.

The LV endocardium was selected as the region of interest (ROI), and the width of ROI was set between 5 and 10 mm. The endocardium was traced manually. A Philips EPIQ 7 Echocardiography device (Philips Healthcare, Koninklijke Philips N.V., Amsterdam, The Netherlands) was used to calculate endocardial left ventricle global longitudinal strain (LVGLS) (Figure 1B). Longitudinal strain is the percentage shortening of a ROI relative to its original length, expressed as a negative percentage [14].

Statistical Analysis

All data were analyzed with STATA/MP v.16 software (StataCorp LLC, College Station, TX, USA). Numerical data that were determined to be normally distributed based on the results of Kolmogorov-Smirnov tests are presented as mean±standard deviation (SD), while non-normally distributed variables are presented as median (25th-75th quartiles). Accordingly, ANOVA tests (post-hoc analysis: Bonferroni test) or Kruskal Wallis H test (post-hoc analysis: Dunn's test) were used for comparisons between respective PVC groups. Categorical variables are presented as numbers and percentages, and comparisons between groups were performed using Chi-square and Fisher exact tests. Among the numerical parameters, the relationship between global LA peak strain and LA stiffness with patient characteristics, laboratory data, and echocardiographic findings was evaluated using the Pearson correlation analysis. For all statistical analyses, significance was accepted at $p<0.05$.

Results

The study population consisted of 102 patients, including 59 (57.8%) males, and with a mean age of 43.5±9.7 yrs. The detailed, basic characteristics of the study population are shown in Table 1. The patients were grouped according to their PVC burden: Group 1, PVC burden <10%, n=31 (30.4%); Group 2, PVC burden 10–20%, n=33 (32.4%); Group 3, PVC burden >20%, n=38 (37.3%). There were no significant differences in demographic characteristics and laboratory findings between the groups (Table 1).

Mean LV end-diastolic diameter (LVEDd), mean LV end-systolic diameter (LVESd), mean LVMI, mean IVS, and mean PW levels were lower in Group 1 with PVC burden <10% compared to the other groups ($p<0.05$). These values did not differ significantly between Group 2 with PVC burden 10–20% and Group 3 with PVC burden >20% ($p>0.05$). Mean E/e' increased as the PVC burden increased ($p<0.001$). The mean global LA peak strain decreased as

Table 1. Demographic characteristics and laboratory findings of the groups based on the PVC burden

Variable	Total population n=102	PVC burden			p
		Group 1, <%10, n=31	Group 2, 10-20%, n=33	Group 3, >%20, n=38	
Age, years	43.5±9.7	44.2±9.2	42.2±10.4	43.7±9.1	0.128
BMI, kg/m^2	24.8±3.2	24.5±2.3	24.9±3.1	25.1±3.6	0.406
Male gender	59 (57.8)	18 (58.1)	19 (57.6)	22 (57.9)	0.999
Smoking	34 (33.3)	13 (41.9)	11 (33.3)	10 (26.3)	0.405
SBP	132.8±14.2	122.8±11.5	133.4±13.8	132.3±15.2	0.317
DBP, mmHg	73.8±7.6	73.3±8.3	74.2±7.1	73.7±7.2	0.270
Glucose, mg/dl	115.2±32.5	122.2±36.1	118±36.8	107.1±23.3	0.129
UREA, mg/dl	37.9±11.5	35.6±8.0	38.2±12.0	39.5±13.4	0.376
Creatinine, mg/dl	0.9±0.1	0.8±0.1	0.9±0.1	0.9±0.1	0.078
Hemoglobin, g/dl	13.2±1.2	13.3±0.9	13.2±1.4	13.1±1.1	0.672
Sodium, mEq/l	138.0±6.8	137.5±5	137.2±8.3	139.2±6.6	0.411
Potassium, mEq/l	4.5±0.5	4.6±0.4	4.4±0.5	4.4±0.4	0.354
Calcium, mg/dl	8.7±0.2	8.7±0.2	8.6±0.2	8.7±0.2	0.083
Cholesterol, mg/dl	231.2±41.2	227±41.3	242.2±32.3	225.2±46.8	0.176
LDL, mg/dl	137.6±36.5	137.3±33.7	136.3±30.3	137.7±41.5	0.845
HDL, mg/dl	42.9±10.0	44.6±11.3	41.7±7.3	42.5±10.9	0.484

Categorical variables are number (percentage). Numerical variables are mean±standard deviation. P-values represent the ANOVA test. Since no difference was detected between the groups, the results of the post hoc analysis were not presented. BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

ISSN 0022-9040. Кардиология. 2023;63(10). DOI: 10.18087/cardio.2023.10.n2567

Table 2. Echocardiography findings between the groups based on the PVC burden

Variable	All population n=102	PVC burden			p
		Group 1, <10%, n=31	Group 2, 10-20% n=33	Group 3, >%20 n=38	
LVEF, %	59.1±4.1	58.7±3.9	59.1±4.5	59.5±4.1	0.767
LVEDd, mm	45.7±3.4	42.6±2.4bc	46.3±2.5a	47.7±3.1a	<0.001*
LVESd, mm	25.5±3.6	22.7±2.4bc	26.0±3.1a	27.4±3.5a	<0.001*
LVMI, g/m²	83.4±19.9	67.3±13.7bc	89.1±17.2a	91.7±18.9a	<0.001*
IVS, mm	10.1±1.2	9.5±0.9bc	10.2±1.3a	10.6±1.2a	<0.001*
PW, mm	9.0±1.4	8.1±1.1bc	9.2±1.5a	9.6±1.3a	<0.001*
E, cm/sec	83.8±15	82.8±15.7	83.5±12.2	84.9±16.8	0.844
A, cm/sec	67.2±14.3	70.7±17	63.7±14.6	67.3±11	0.149
E/A ratio	1.3±0.4	1.2±0.4	1.4±0.3	1.3±0.4	0.358
e', cm/sec	9.7±2.2	11.9±2bc	9.6±1.4ac	8.0±0.8ab	<0.001
a', cm/sec	10.5±2.7	9.4±2.2c	9.8±2.5c	11.8±3.0ab	0.003
s', cm/sec	10.3±2	10.6±1.6	10.6±2.1	9.8±2.0	0.103
E/e' ratio	9.0±2.2	7.0±1.1bc	8.8±1.5ac	10.7±2.1ab	<0.001
LVGLS	-21.2±1.7	-21.3±1.6	-21.2±1.6	-20.9±1.7	0.409
LA diameter, mm	35.5±4.7	30.5±2.6bc	35.3±2.2ac	39.7±3.3ab	<0.001
LA maximal volume index	32.9±4.4	28.1±2.0bc	32.6±2.8ac	37.0±2.5ab	<0.001
LA pre-A volume index	20.3±2.7	17.5±1.3bc	19.7±1.8ac	23.1±1.0ab	<0.001
LA minimal volume index	13.8±2.5	11.2±1.1bc	13.1±1.2ac	16.4±1.4ab	<0.001
Global LA peak strain, %	32.4±5.2	38.1±3.2bc	32.4±3.2ac	27.7±2.6ab	<0.001
LA stiffness, %	29.1±8.3	18.6±3.2bc	27.5±5.5ac	39.0±7.9ab	<0.001
LVEF, %	59.1±4.1	58.7±3.9	59.1±4.5	59.5±4.1	0.767
LVEDd, mm	45.7±3.4	42.6±2.4bc	46.3±2.5a	47.7±3.1a	<0.001*
LVESd, mm	25.5±3.6	22.7±2.4bc	26.0±3.1a	27.4±3.5a	<0.001*
LVMI, g/m²	83.4±19.9	67.3±13.7bc	89.1±17.2a	91.7±18.9a	<0.001*
IVS, mm	10.1±1.2	9.5±0.9bc	10.2±1.3a	10.6±1.2a	<0.001*
PW, mm	9.0±1.4	8.1±1.1bc	9.2±1.5a	9.6±1.3a	<0.001*
E, cm/sec	83.8±15	82.8±15.7	83.5±12.2	84.9±16.8	0.844
A, cm/sec	67.2±14.3	70.7±17	63.7±14.6	67.3±11	0.149
E/A ratio	1.3±0.4	1.2±0.4	1.4±0.3	1.3±0.4	0.358
e', cm/sec	9.7±2.2	11.9±2bc	9.6±1.4ac	8.0±0.8ab	<0.001
a', cm/sec	10.5±2.7	9.4±2.2c	9.8±2.5c	11.8±3.0ab	0.003
s', cm/sec	10.3±2	10.6±1.6	10.6±2.1	9.8±2.0	0.103
E/e' ratio	9.0±2.2	7.0±1.1bc	8.8±1.5ac	10.7±2.1ab	<0.001
LVGLS	-21.2±1.7	-21.3±1.6	-21.2±1.6	-20.9±1.7	0.409
LA diameter, mm	35.5±4.7	30.5±2.6bc	35.3±2.2ac	39.7±3.3ab	<0.001
LA maximal volume index	32.9±4.4	28.1±2.0bc	32.6±2.8ac	37.0±2.5ab	<0.001
LA pre-A volume index	20.3±2.7	17.5±1.3bc	19.7±1.8ac	23.1±1.0ab	<0.001
LA minimal volume index	13.8±2.5	11.2±1.1bc	13.1±1.2ac	16.4±1.4ab	<0.001
Global LA peak strain, %	32.4±5.2	38.1±3.2bc	32.4±3.2ac	27.7±2.6ab	<0.001
LA stiffness, %	29.1±8.3	18.6±3.2bc	27.5±5.5ac	39.0±7.9ab	<0.001

Data are mean±SD. P-values represent the ANOVA test. a, b, and c represent the groups that showed significant differences in the post-hoc analyses. a: p<0.05 vs. Group 1, b: p<0.05 vs. Group 2, c: p<0.05 vs. Group 3. LVEF, left ventricular ejection fraction; LVEDd, left ventricle end diastolic diameter; LVESd, left ventricle end systolic diameter; LVMI, left ventricle mass index; IVS, interventricular septum; PW, posterior wall; E, peak early diastolic filling velocity; A, peak late diastolic filling velocity; e', peak early diastolic velocity at the mitral annulus; a', peak late diastolic velocity at the mitral annulus; s', peak systolic velocity of the mitral annulus; LA, left atrium; LVGLS, LV global longitudinal strain.

the PVC burden increased (p<0.001), while other mean LA measurements increased as the PVC burden increased (p<0.001) (Table 2).

In all patients, moderate negative correlations were found between global LA peak strain and LVEDd (r= –0.503, p<0.001) and between global LA peak strain and LVESd (r = –0.501, p<0.001). A mild negative correlation was found with LVGLS levels (r= –0.231, p=0.020). A strong negative correlation was found between global LA peak strain and LA stiffness (r= –0.779, p <0.001) (Table 3).

Table 3. Correlations of global LA peak strain and LA stiffness with patient characteristics, laboratory data, and echocardiographic findings

Variables	Global LA peak strain (%)		LA stiffness	
	r	p	r	p
Age	0.119	0.233	-0.086	0.391
BMI	-0.375	<0.001	0.315	0.001
SBP	-0.129	0.197	0.010	0.924
DBP	-0.038	0.704	-0.038	0.703
Glucose	0.122	0.223	-0.094	0.346
UREA	0.071	0.476	-0.096	0.338
Creatinine	-0.178	0.173	0.165	0.198
Hemoglobin	0.104	0.298	-0.006	0.954
Sodium	-0.058	0.562	0.093	0.353
Potassium	0.197	0.208	-0.185	0.163
Calcium	0.177	0.275	-0.088	0.380
cholesterol	0.122	0.302	-0.065	0.517
LDL	0.102	0.310	-0.067	0.506
HDL	0.098	0.327	-0.005	0.961
LVEF	-0.076	0.447	0.103	0.302
LVEDd	-0.503	<0.001	0.505	<0.001
LVESd	-0.501	<0.001	0.502	<0.001
LVMI	-0.383	<0.001	0.396	<0.001
IVS	-0.284	0.004	0.316	0.001
PW	-0.374	<0.001	0.379	<0.001
E	0.060	0.548	0.381	<0.001
A	0.135	0.175	-0.145	0.146
EA	-0.051	0.608	0.285	0.004
e’	0.668	<0.001	-0.711	<0.001
a’	-0.271	0.006	0.265	0.007
s’	0.194	0.110	-0.154	0.122
E/e’	-0.511	<0.001	0.919	<0.001
LVGLS	-0.231	0.020	0.307	0.002
LA diameter	-0.647	<0.001	0.546	<0.001
LA maximal volume index	-0.603	<0.001	0.621	<0.001
LA pre-A volume index	-0.645	<0.001	0.649	<0.001
LA minimal volume index	-0.610	<0.001	0.687	<0.001
LA stiffness	-0.779	<0.001	-	-

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; LDL, low-density lipoprotein; HDL, high-density lipoprotein; LVEF, left ventricular ejection fraction; LVEDd, left ventricle end diastolic diameter; LVESd, left ventricle end systolic diameter; LVMI, left ventricle mass index; IVS, interventricular septum; PW, posterior wall; E, peak early diastolic filling velocity; A, peak late diastolic filling velocity; e’, peak early diastolic velocity of the mitral annulus; a’, peak late diastolic velocity of the mitral annulus; s’, peak systolic velocity of the mitral annulus; LA, left atrium; LVGLS, LV global longitudinal strain.

Discussion

To our knowledge, this is the first study to report the association between the PVC burden and LA function in healthy individuals. A higher PVC burden of was associated with more impaired LA function. Furthermore, this association was observed without any significant difference in the LVGLS.

PVCs, which can occur in patients both with and without structural heart disease, are often asymptomatic [15, 16]. If the frequency of PVCs increase significantly, it can potentially lead to cardiomyopathy, a condition characterized by abnormalities in the structure and function of the heart muscle [17]. Furthermore, an elevated frequency of PVCs is associated with higher mortality rates among patients with LV systolic failure and congestive heart failure [3, 16]. Therefore, monitoring and managing PVC frequency is crucial for identifying any potential progression towards cardiomyopathy and for initiating appropriate interventions to prevent further cardiac complications.

Several studies have presented evidence supporting an association between frequent PVCs and impaired LV and LA function [18, 19]. This can result in a shortened diastolic filling time and, thus, impact overall cardiac function [5]. Frequent PVCs can disrupt the normal electrical conduction system of the heart, leading to an interruption in the synchronization of ventricular contraction. Ventricular desynchrony caused by frequent PVCs has been proposed as a possible pathogenic mechanism in LV dysfunction [19]. In addition, the phenomenon of a giant “a” wave, which occurs due to the premature and sudden closure of the mitral valve caused by frequent PVCs, can lead to LA dilatation. In an experimental study conducted on dogs, it was observed that rapid right ventricular stimulation with a 1:1 ventriculoatrial passage led to the occurrence of giant systolic pulmonary vein reflux. Furthermore, even with just 5 min of rapid right ventricular stimulation, an increase in LV end-diastolic pressure, pulmonary capillary wedge pressure, and LA dilatation was detected [20]. These findings were supported by animal experiments involving induced ectopic beats that provided evidence of the adverse effects of PVC on LV function [21, 22]. In addition to systolic dysfunction, diastolic dysfunction can occur as a result of PVCs, further worsening LV function [23]. In line with these findings, our study revealed a positive correlation between the PVC burden and the E/e’ ratio, which is indicative of impaired relaxation and elevated LV filling pressure. As the PVC burden increased, the E/e’ ratio correspondingly increased, suggesting a potential association between the PVC burden and altered LV diastolic function.

We also found that LVEDd and LVESd were associated with the PVC burden. This is consistent with previous studies that used conventional methods [6, 7]. However, this association was observed specifically in patients with a PVC burden exceeding 10%. A higher PVC burden was associated with a higher LVMI, but changes in LVEF and LVGSL were not significant. These findings align with previous studies suggesting that PVCs contribute to alterations in LV

ISSN 0022-9040. Кардиология. 2023;63(10). DOI: 10.18087/cardio.2023.10.n2567

contractility, even while the LVEF is unchanged [23, 24]. Also, a higher PVC burden was associated with a higher LAVI. A previous study of patients with normal LVEF revealed an association between PVCs and LA enlargement [10]. Another study of individuals without known structural heart disease demonstrated that frequent PVCs were associated with a larger LA volume and a trapezoidal LA shape. These findings suggest that the observed changes in the LA may represent LA remodeling in response to increased LV filling pressure [25]. Furthermore, these findings indicate that patients with a high frequency of PVCs may experience impaired LA function and dilatation, even in the presence of preserved LVEF.

Previous studies have demonstrated that LA stiffness is increased in patients with diastolic dysfunction or atrial fibrillation. Furthermore, LA stiffness has been found to have a significant correlation with LA volume indices [26, 27]. To the best of our knowledge, there is currently a lack of studies specifically evaluating LA strain in patients with PVCs. LA strain measurements have demonstrated that there is impairment in LA deformation even before patients exhibit overt cardiac hemodynamic deterioration. We found that a higher PVC burden was associated with lower global LA peak strain and LA stiffness. Global LA peak strain and LA stiffness levels were not found to be associated with LVEF, but they showed a mild correlation with LVGLS. Hence, it is plausible that individuals without any underlying disease and with normal LVEF may experience impaired LA mechanical function because of PVC burden. This impairment in LA function could potentially increase their risk of cardiovascular events, including atrial arrhythmias or stroke.

The most notable limitation of our study was the small sample size, which may have limited the generalizability of our findings. The study did not include long-term follow-up of the patients to assess the development of arrhythmias or evaluate the prognostic implications of impaired LA function. The identification of impaired LA function, characterized by increased LA volume indices potentially offers valuable prognostic information.

Conclusion

This study suggests that a higher PVC burden is associated with impaired LA function in individuals with no known cardiac disease. These findings highlight the importance of monitoring and managing PVCs to prevent further cardiac complications and to identify individuals at risk for cardiovascular events.

No conflict of interest is reported.

The article was received on 03/08/2023

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ISSN 0022-9040. Кардиология. 2023;63(10). DOI: 10.18087/cardio.2023.10.n2567