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Effects of Ventricular Extrasystoles on Right Ventricle Functions (a Speckle Tracking Study). VES and RV Strain Imaging

Aim	The adverse effects of ventricular extrasystoles (VES) on the heart, such as induced dyssynchrony, irregular heart rate, and atrioventricular dissociation, have been demonstrated. The aim of this study was to investigate the effects of VES on the right ventricle (RV) using strain imaging.
Material and methods	Fifty patients with 5000 or more VES detected during 24hr Holterrhythm monitoring between April 2022 and September 2022 in the cardiology outpatient clinic were included in this study.A volunteer control group of 50 individuals matching the patients' age and demographic characteristics was selected. Right heart function parameters were compared echocardiographically between the two groups.
Results	In the VES group, both RV free wall strain (22.03±3.67, 29.52±3.01; p<0.001) and RV four-chamber strain (19.37±2.95, 22.34±2.11; p<0.001) were lower compared to the control groupIn the univariate regression analysis for decreased RV four-chamber strain, the presence of VES (p<0.001) was identified as a predictor, whereaas in the multivariate regression analysis, it was not considered to be an independent predictor. When evaluating the characteristics of the VES patients, the number of VES detected during Holter monitoring and delta QRS were observed as negative predictors of RV strain.
Conclusion	This study demonstrated the adverse effects of VES on the right ventricle, as it is on the left ventricle. Therefore, regular monitoring of RV function with echocardiography is important in the follow-up of patients with VES.

Keywords	Right ventricle; strain imaging; ventricular extrasystole
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Introduction

Ventricular extrasystoles (VES) are additional beats that appear in electrocardiography (ECG) outside the normal rhythm and are typically characterized by a wide QRS (>120 ms) followed by an oppositely-directed T wave [1]. VES can cause symptoms, such as palpitations, pulse irregularity, dyspnea, weakness, and syncope, but they can also remain asymptomatic for many years [2]. Various studies have shown that VES can lead to malignant ventricular arrhythmias, heart failure, and sudden death [3]. Through pathophysiological mechanisms, such as ventricular dyssynchrony, post-extrasystolic potentiation, tachycardia, autonomic dysfunction, and atrioventricular dissociation, VES can cause cardiomyopathy and heart failure [4]. In VES patients, ventricular arrhythmias can originate from various regions of both the left ventricle (LV) and the right ventricle (RV) [5]. While current guidelines recommend regular monitoring of LV ejection fraction (LV-EF) and LV dimensions in VES patients, however regarding VES, the RV has remained the "forgotten ventricle" [6].

After being neglected for years, the importance of the RV in cardiovascular diseases and mortality has been recognized, making it the focus of research during the past decade [7]. Several echocardiographic parameters have been established to evaluate RV function, and they have been quantified in various studies [8]. RV strain imaging is an advanced echocardiographic measurement method that has been described in the literature for evaluating RV function [9].

The aim of this study was to investigate the effects of VES, including ventricular dyssynchrony, on RV function using RV strain imaging and to explore the resulting pathological processes that have been proven to cause LV functional impairment and their effects on RV function.

Material and methods

Study design and population

This study was a single-center, prospective study. Patients who presented with complaints of palpitations and underwent Holter monitoring between April 2022 and

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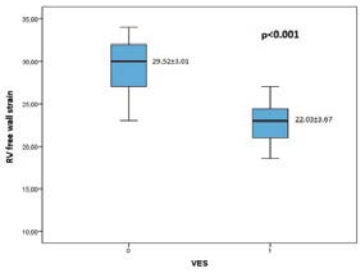
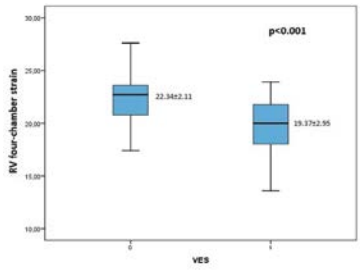
Central illustration. Effects of Ventricular Extrasystoles on Right Ventricle Functions (a Speckle Tracking Study). VES and RV Strain Imaging

Comparisons of 24-hour Holter recordings and laboratory data
of patients with 5000 or more ventricular extrasystoles (VES) and those of healthy patients.



Variable			p value
CRP (mg/ml)	2.9 (0.1-43)	1.5 (0.1-17.60)	**0.027**
Hs-Troponin I (ng/ml)	2.9 (0.5-15.5)	1.7 (0.1-7.91)	**0.005**
LV-ESD (mm)	31 (22-46)	27.5 (18-38)	**0.001**
E/E'	7.82±3.07	6.58±2.02	**0.020**
TAPSE (mm)	23.19±2.60	25.33±3.40	**0.001**
RVOT Diameter (mm)	23 (17-28)	22 (19-29)	**0.007**



In this study, we demonstrated the negative effects of VES on the right ventricle, similar to the effects on the left ventricle. Therefore, it is important to regularly monitor right ventricular function with echocardiography in the follow-up of patients with VES.

September 2022 in the Cardiology Outpatient Clinic were followed. 180 patients with a VES count of 5 000 or more during 24-hour Holter monitoring were examined for inclusion in the study. According to the exclusion criteria, 130 patients were excluded from the study, and the remaining 50 patients were considered as the VES patient group of the study. Demographic data of these patients, and their history of hypertension, diabetes, hyperlipidemia, smoking, alcohol consumption, and chronic lung disease were recorded.

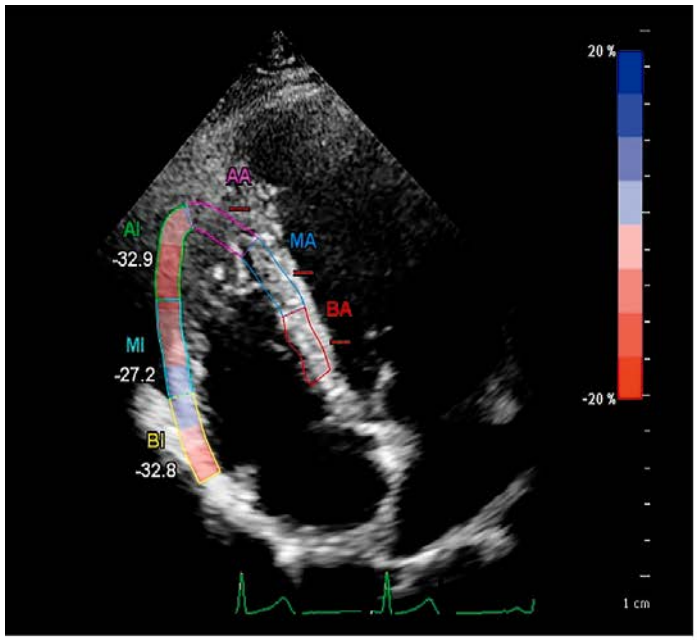
A control group of 50 volunteers with similar baseline characteristics and who presented to the cardiology outpatient clinic without clinical symptoms of arrhythmia and without VES as detected by ECG were included. Routine echocardiography was performed on all individuals participating in the study.

The ECG and 24-hour Holter monitor results were recorded, and all data were divided into two groups: patients with VES and healthy volunteers. The study protocol was approved by the local ethics committee in accordance with the Helsinki Declaration and with good clinical practice. The study complies with the STROBE guidelines from the EQUATOR NETWORK.

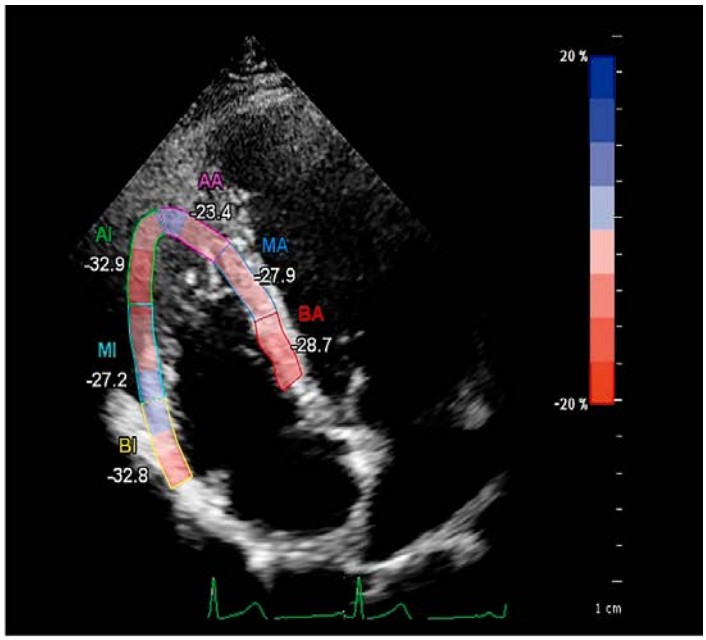
Laboratory and echocardiographic evaluation

Blood samples were collected from the patients' peripheral veins in the morning after fasting. The samples

Figure 1. A: RV free wall strain imaging (3 segments). **B**: RV four-chamber strain imaging (6 segments)



A: RV Free wall strain (3 segments)



B: RV four-chamber strain (6 segments)

Figure 2. RV-OT VES visualization with 3D mapping

were analyzed for complete blood count, renal and liver function, blood glucose, electrolytes, lipid panel, C-reactive protein (CRP), and high-sensitivity troponin I (Hs-troponin I).

Echocardiographic examination was performed using the Philips EpiQ-7 system (X5 probe, Philips® Medical Systems, Andover, MA, USA). The images were acquired using a 3.5 MHz transducer. LV-EF was calculated using Simpson's method applied to apical views. For strain imaging evaluation, echocardiography was performed using 2D speckle tracking with CMQ software (QLAB 10.3; Philips Medical System, Andover, MA, USA). Strain analyzes were performed when blood pressures were in the normotensive range. For RV strain analysis, the RV boundaries were marked using an apical 4-chamber RV-focused view. Initially, automatic identification was performed by the software and then by manual corrections and modifications. The average of three segments of the free wall was taken for RV free wall strain (Figure 1 A). For RV four-chamber strain, the average of six segments, three on the free wall and three on the septum, was calculated (Figure 1 B). For LV strain imaging, recordings were obtained by having the patient hold their breath for three apical views (four, three and two-chamber views). LV global longitudinal strain (GLS) was calculated by averaging the peak longitudinal strain values from the three apical views.

Electrocardiographic evaluation

To determine the origins of the patients' VES, a cardiologist interested in arrhythmia blindly examined all 12-lead ECGs. VES origins were determined using various parameters, such as bundle branch block patterns (LBBB, RBBB), R dominance in the inferior leads, precordial transition zone, QRS width, and D1 lead dominance. The most common origin regions of idiopathic VES are the RV-OT and LV-OT regions. For the LBBB pattern, the inferior axis and late transition zone (V4-V5) are generally characteristic for RV-OT VES. The earlier transition zone (V1-V2) characteristic for LV-OT VES [10]. In cases where the transition zone is V3, many algorithms and parameters such as V2S/V3R index, Transition Zone (TZ) index, Combined TZ index, V1-V2 R-S difference, R Wave Duration Index are used today for location analysis [11]. VES origins of our patients with ablation indications were checked via 3D mapping (Figure 2).

24- Hour Holter monitoring

Schiller's medilog® Holter System (DARWIN2) was used. The Holter device was attached to the patients in the afternoon and removed the following day at approximately the same time, ensuring that the Holter recording lasted for at least 24 hrs. The obtained recordings were scanned for artifacts and meticulously analyzed by a cardiologist experienced in arrhythmias. Each patient underwent at least two 24-hour Holter monitoring sessions. Patients with an average of 5,000 or more ventricular extrasystoles (VES) in the two Holter recordings were selected for the study.

Statistical analyses

Statistical analyses of the data were performed using the SPSS 15.0 program (SPSS Inc., Chicago, IL, USA). Kolmogorov-Smirnov tests for homogeneity of variances were conducted to examine the parametric and non-parametric distributions of the data. Independent samples t-tests were used to compare variables with parametric distributions. The Mann–Whitney U test was used for variables with non-parametric distributions. Categorical variables were compared using chi-square tests. Parametric continuous variables are expressed as mean±standard deviation (SD), while non-parametric variables are expressed as median (minimum-maximum) values. Categorical variables are presented as numbers and percentages. Correlations were evaluated with Pearson and Spearman correlation analysis. Univariate and multivariate binary logistic regression analyses were performed to investigate dependent and independent predictors. In all statistical analyses, values with $p<0.05$ were considered statistically significant.

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Figure 3. Distribution of RV free wall strain between VES and control groups

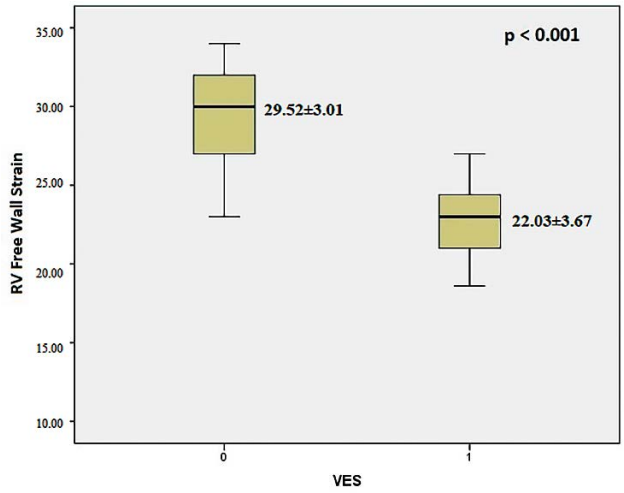
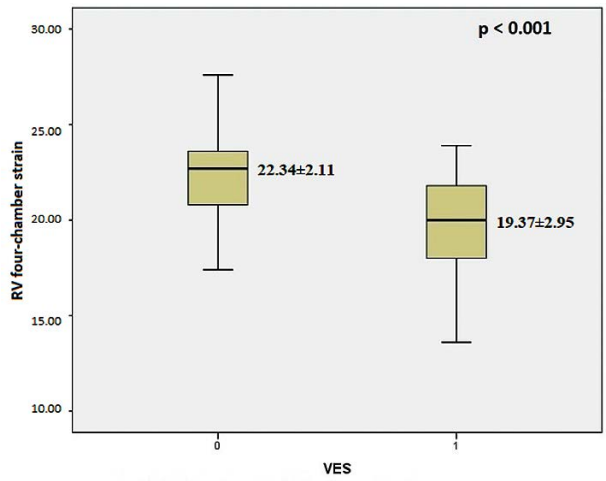


Figure 4. Distribution of RV four-chamber strain between VES and control groups



Results

A total of 100 subjects, including 50 patients (28 females and 22 males; mean age 54.2±16.9 yrs) and 50 healthy controls (25 females and 25 males; mean age 54.3±9.0 yrs), were included in this study. The demographic and laboratory data of all participants in are compared in Table 1. The demographic data of the two group did not differ significantly. CRP (p=0.027) and Hs-troponin I (p=0.005) were significantly higher in the VES group. Table 2 compares the echocardiographic parameters. Significant differences were found in LV-ESD (p=0.001), E/E' (p=0.020), TAPSE (p=0.001), RV-OT diameter (p=0.007), LV-GLS (p=0.001), RV free wall strain (p<0.001), and RV four-chamber strain (p<0.001) (Figures 3 and 4). No significant differences were found among the other parameters.

To determine the dependent and independent relationships between RV four-chamber strain and all parameters in the study, univariate regression analysis was initially conducted for all variables. Age, BMI, HT, creatinine, glomerular filtration rate (GFR), platelet count (PLT), Hs-troponin I, LV-EF, interventricular septum (IVS), posterior wall (PW), transmittal flow E/A ratio (E/E'), right atrium

Table 1. Clinical characteristics of the study population

Variable	VES Group, n =50	Control Group, n=50	p
Age (yrs)	54.20±16.94	54.34±9.05	0.959 [a]
Gender (% female/% male)	28 / 22	25 / 25	0.548 [b]
BMI(kg/m^2)	29.06±4.93	29.5±4.67	0.650 [a]
Hypertension	18 (56)	11 (28)	0.123 [b]
Diabetes mellitus	6 (13)	2 (0.4)	0.140 [b]
Hyperlipidemia	4 (0.8)	2 (0.4)	0.400 [b]
Smoker	14 (38)	8 (19)	0.148 [b]
Alcohol consumption	2 (0.4)	1 (0.2)	0.558 [b]
COPD	4 (0.8)	2 (0.4)	0.400 [b]
Asthma	2 (0.4)	2 (0.6)	0.646 [b]
Fasting blood glucose (mg/dl)	97 (71-157)	99 (55-187)	0.083 [c]
Urea (mg/dl)	175±49.29	173.54±29.5	0.858 [a]
Creatine (mg/dl)	0.84±0.18	0.82±0.13	0.550 [a]
GFR	92.04±19.72	90.11±11.25	0.554 [a]
Total protein (g/l)	72.97±4.25	72.04±3.77	0.300 [a]
CRP (mg/l)	2.9 (0.1-43)	1.5 (0.1-17.60)	0.027 [c]
Albumin (g/dl)	43.37±2.94	42.51±2.29	0.228 [a]
LDL (mg/dl)	122.92±32.11	126.29±36.52	0.628 [a]
HDL (mg/dl)	48.66±10.14	50.73±10.18	0.474 [a]
Triglyceride (mg/dl)	148.96±79.98	175.8±90.52	0.141 [a]
ALT (IU/l)	16.5 (10-44)	21 (10-59)	0.080 [c]
AST (IU/l)	20 (9-59)	19.5 (10-48)	0.344 [c]
Sodium (mmol/l)	140 (136-145)	141 (136-146)	0.244 [c]
Potassium (mmol/l)	4.38±0.38	4.29±0.29	0.268 [a]
Calcium (mmol/l)	9.04±0.51	9.08±0.42	0.770 [a]
Total cholesterol (mg/dl)	181.37±41.49	197.70±30.74	0.275 [a]
Hemoglobin (g/dl)	13.82±1.62	13.85±1.35	0.931 [a]
Hematocrit (%)	42.11±5.14	40.18±5.05	0.062 [a]
White blood cells (×10^9/l)	8.2 (4.1-11.9)	7.31 (4.5-12.8)	0.146 [c]
Platelets (×10^9/l)	227±48.16	240.24±62.48	0.238 [a]
MCV (fl)	87.7 (64-97)	87 (59-95)	0.358 [c]
Lymphocytes (×10^3/μl)	2.47±0.92	2.25±0.58	0.118 [a]
Neutrophils (×10^3/μl)	4.66 (2.1-59.4)	4.08 (2.37-10.96)	0.095 [c]
Eosinophils(×10^3/μl)	0.14 (0.01-0.78)	0.14 (0.01-0.72)	0.450 [c]
Basophils(×10^3/μl)	0.08 (0.01-0.12)	0.08 (0.01-0.3)	0.786 [c]
MPV (fl)	8.04±1.64	8.49±1.44	0.160 [a]
Hs-troponin I (ng/l)	2.9 (0.5-15.5)	1.7 (0.1-7.91)	0.005 [c]

Data are mean±SD,mean (range), or number (percentage).
[a] – Independent t test. [b] – Chi-square test. [c] – Mann–Whitney U test. BMI, body mass index; GFR, glomerular filtration rate; CRP, C-reactive protein; LDL, low density lipoprotein; HDL, high density lipoprotein; MCV, mean corpuscular volume; MPV, mean platelet volume; Hs-troponin I, high-sensitivity cardiac troponin I.

(RA), right ventricle (RV), LV-GLS, RV fractional area change (RV-FAC), RV-OT diameter, and the presence of VES were all identified as predictors of impaired RV four-chamber strain. However, in the multivariate regression analysis, none of these dependent predictor parameters were found to be independent predictors.

Discussion

This study reports, for the first time, impairment in RV function detected by RV strain imaging in patients with an intermediate to high burden of VES. VES burden has been categorized as low burden (≤5% of depolarizations during 24-hour monitoring), intermediate burden (between 5% and 20% of depolarizations), and high burden (>20% of depolarizations) [12]. In the current study, the lower limit of intermediate burden was considered as 5%. However, since we took the average of two24-hour Holter recordings and since the average heart rate of our patient population was 70 bpm, a criterion of 5000 VES during 24 hrs was established.

Compared to the control group, the VES group showed statistically significant impairment in RV free wall strain, RV four-chamber strain, and LV-GLS. Previous studies have shown an association between the presence of VES and LV function and reduced LV-GLS [2, 13]. Although the direct effect of VES on the RV function was not demonstrated, a study by Wijnmaalen et al. found an improvement in RV function after successful ablation in VES patients [14]. In a recent study by Scorza et al. with 40 patients and 22 control subjects,a decrease in RV and LV strain was observed in the VES patient group [15]. This finding supports

Table 2. Comparisons of the echocardiographydata

Variable	VES Group	Control Group	p
LV-EF (%)	62.5 (52-69)	64 (58-68)	0.063[a]
LV-EDD (mm)	46.96±6.37	45.06±4.72	0.093[b]
LV-ESD (mm)	31 (22-46)	27.5 (18-38)	0.001[a]
RA (mm)	34.32±3.95	32.84±3.71	0.057[b]
RV (mm)	29.28±3.59	28.26±3.21	0.138[b]
S' (cm/sec)	13.5 (9.9-23)	13.1 (9-24)	0.421[a]
TAPSE (mm)	23.19±2.60	25.33±3.40	0.001[b]
PASP (mmHg)	27.26±4.89	26.84±4.59	0.659[b]
RV FAC	48.65±4.01	49.4±2.51	0.267[b]
RV-OT diameter (mm)	23 (17-28)	22 (19-29)	0.007[a]
LV-OT diameter (mm)	21 (16-24)	20 (16-26)	0.259[a]
LV-GLS	19.13±2.09	20.5±1.90	0.001[b]
RV free wallstrain	22.03±3.67	29.52±3.01	<0.001[b]
RV four-chamber strain	19.37±2.95	22.34±2.11	<0.001[b]

Data are mean±SD, mean (range), or number (percentage).
[a] – Mann–Whitney U test. [b] – Independent t test. LV-EF, leftventricular ejection fraction; PASP, pulmonary artery systolic pressure; RA, right atrium; RV, right ventricle; S', tricuspid lateral wall annularsystolic velocity; TAPSE, tricuspidannularplanesystolicexcu rsion; RV-FAC, right ventricular fraction alareachange;RV-OT, right ventricularoutflowtract; LV-OT, left ventricularoutflowtract; LV-GLS, left ventricular global longitudinal strain.

Table 3. Univariate and multivariate regression analyses showing the relationship between decreased RV four-chamber strain and variables evaluated in the VES group

Variable	Univariate Analysis			Multivariate Analysis		
	OR	95% CI	p	OR	95%CI	p
Age	0.947	0.914-0.982	0.003	1.070	0.908-1.260	0.419
BMI	0.905	0.829-0.989	0.027	0.909	0.673-1.228	0.535
Hypertension	0.329	0.132-0.824	0.018	0.520	0.026-10.266	0.668
Creatine	0.048	0.003-0.779	0.033	–	–	–
GFR	1.047	1.016-1.079	0.002	1.079	0.892-1.304	0.435
PLT	1.013	1.004-1.022	0.005	1.007	0.984-1.031	0.540
Hs-troponin I	0.708	0.568-0.883	0.002	0.634	0.292-1.377	0.249
LV-EF	1.280	1.123-1459	<0.001	1.467	0.898-2.395	0.126
IVS	0.651	0.469-0.904	0.010	0.943	0.117-7.565	0.956
PW	0.664	0.473-0.934	0.019	0.799	0.087-7.298	0.842
A	0.958	0.934-0.982	0.001	0.993	0.918-1.075	0.868
E/E'	0.765	0.631-0.927	0.06	1.037	0.506-2.124	0.921
RA	0.791	0.698-0.897	<0.001	0.819	0.540-1.243	0.349
RV	0.806	0.703-0.925	0.002	1.535	0.816-2.887	0.184
LV-GLS	3.979	2.360-6.709	<0.001	2.371	0.698-8.058	0.166
RV-FAC	1.323	1.144-1.531	<0.001	0.969	0.512-1.835	0.923
RV-OT Diameter	0.646	0.501-0.835	0.001	0.536	0.205-1.402	0.204
VES	0.151	0.063-0.362	<0.001	0.144	0.009-2.326	0.144

OR, odds ratio; CI, confidence interval; BMI,body mass index; GFR,glomerular filtration rate; PLT, platelets; Hs-troponin I, high-sensitivity cardiac troponin I; LV-EF, left ventricular ejection fraction, IVS, inter ventricular septum, PW, posterior wall; A, late diastolic wave; E, early diastolic wave; E',early diastolic myocardial velocity; RA, right atrium; RV, right ventricle; LV-GLS, left ventricular global longitudinal strain; RV FAC, right ventricular fractional area change; RV-OT, right ventricular outflow tract; VES, ventricular extrasystole.

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the conclusions of the current study. Moreover, the current study is important because it shows the negative regional effects of VES in the RV

It is noteworthy that in our study, the RV-OT diameter was larger in the VES patients. In 2020, Jia et al. investigated the effects of RV-OT and LV-OT VES on the main pulmonary artery and the ascending aorta using multi-detector computed tomography [16]. That study found that the increase in pulmonary artery diameter was greater in RV-OT VES compared to the increase in ascending aorta diameter. In our study, idiopathic VES patients were included, as it is known that RV-OT VES constitute two-thirds of idiopathic VES cases [17]. In our study, RV-OT measurement was performed over the distal RV-OT, which is the closest region to the main pulmonary artery in the parasternal short-axis view. The results of our study suggest that the dilation observed in the main pulmonary artery by Jia et al. [16] could also have occurred in the RV-OT.

Considering the ECG characteristics of the VES patients, 84% had LBBB pattern and 16% had RBBB pattern. Although LBBB pattern is thought to be associated with RV source VES and RBBB pattern with LV source VES, cardiac anatomy is more complex than that [13]. Many LV source VES with LBBB pattern have been reported, and several algorithms have been developed to differentiate them from RV-OT VES [18]. Taking all this information into consideration, it can be assumed that two-thirds of idiopathic VES in our study were RV source VES.

In the VES group, LV-GLS (p=0.001), RV four-chamber strain (<0.001), and RV free wall strain (p<0.001) were found to be lower. Some studies have shown that RV source VES cause more LV dysfunction compared to LV source VES [17]. However, since these studies did not include RV parameters, it is not possible to state the opposite, namely that LV source VES affect the RV more. Considering the results of our study and those of the study of Del Carpio Munoz et al. [17], it can be concluded that RV source VES increase the risk of both RV and LV cardiomyopathy to a greater extent than LV source VES.

There are various studies of RV strain that considered only the free wall or included the septum in a measure of total strain. In this study, the correlation of VES with the RV free wall strain was highest. This is consistent with the hypothesis that total strain, which includes the septum, mainly reflects LV strain. In our study, we used the RV four-chamber strain parameter in the regression analysis to demonstrate the total effect of VES on the RV and to better reflect the important component of negative effects of VES on the ventricles, i.e., the resulting dyssynchrony. Different normal values have been defined for RV four-chamber strain in various studies. In most studies, normal limit values such as 20.4, 21, and 22.3 have been determined for RV four-chamber strain [8, 9]. In our study, the median value of RV four-chamber strain for the entire population was found to be 21.6, and values below 21.6 were defined as decreased RV four-chamber strain, while values above it were considered normal RV four-chamber strain.

We think VES pulses may damage RV function through many mechanisms. In our study patient group, LBBB pattern VES was mostly present. When the electrocardiographic origins of these patients are examined, RV-OT VES is noted in 64%. This may be the primary cause of RV dysfunction. Additionally, the importance of delta QRS duration was emphasized in our study. This shows that dyssynchrony is an important parameter in RV dysfunction.

In this study, we evaluated the dependent and independent predictors of decreased RV four-chamber strain by considering both the patient and the healthy populations together. When all parameters were evaluated in univariate regression analysis, age, BMI, HT, GFR, PLT, Hs-troponin I, LV-EF, IVS, PW, A wave, E/E' ratio, RA, RV,LV-GLS, RV-FAC, RRV-OT diameter, emerged as dependent predictors of decreased RV four-chamber strain in the presence of VES. However, when these significant parameters from the univariate regression analysis were re-evaluated in the multivariate regression analysis, we found that none of these parameters were independent predictors. It is possible to speculate that the dependent nature of our patient population on VES and the complex process by which VES affects many factors, such as age, Hs-troponin I, E/E' ratio, RV-OT diameter, and others, that ultimately influence RV four-chamber strain. Therefore, while the presence of VES is a strong predictor of decreased RV four-chamber strain, it could not be identified as an independent predictor.

This decrease in RV functions in VES patients gives us a perspective on the follow-up and treatment of right heart failure. The information on RV free wallstrain and RV four-chamber strain gained in this study will be followed during the management of our VES patients. More importantly, this new information will raise awareness about optimizing the medical treatment of VES patients at an early stage or more early preparing them for ablation. Such early treatment will be beneficial in preventing patients from developing advanced right heart failure.

Patients with a VES of 5000 and above were included in our study. These numbers of VES pulses are detected in many patients every day during Holter recordings in outpatient clinics. It is necessary to approach these patients in a multidisciplinary manner. Close blood pressure control should be maintained. Other systemic diseases, such as anemia, infectious process, hormonal disorders, panic attacks, and anxiety disorders, that will increase the patient's existing arrhythmia should be controlled. Regarding lifestyle changes, alcohol consumption and smoking should be reduced, and if there is a sleep disorder, professional

treatment should be sought. Despite all this, in the presence of only if clinically significant VES, patients should be offered medical treatment or interventional treatment options.

Study limitations

This study was a single-center study with a limited number of patients. Multicenter studies will provide more enlightening information on the effect of VES on RV function. Other limitations of our study relate to the inherent limitations of speckle tracking strain echocardiography, including relatively low reproducibility with suboptimal intra- and inter-observer variability, wide range of normal values due to patient-to-patient variability, dependence on image quality, frame rate, and image resolution and differences between vendors. In addition, RV longitudinal strain curves were evaluated from the apical four-chamber view, where the RV is represented only by the inflow portion.

Conclusions

In this study, the detrimental effects of VES onRV function were directly demonstrated using strain echocardiography, a sensitive echocardiographic procedure. A strong negative relationship between VES and RV four-chamber strain was detected. This provides with a new and different perspective on the clinical approach to VES patients. The findings suggest that, in addition to monitoring LV-EF and LV ventricular diameter, as recommended in current guidelines, the monitoring of RV function is also important in the evaluation and treatment of VES patients. To confirm the findings of this pilot study, further comprehensive studies with increased sample size and longer follow-up periods are needed.

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REFERENCES

1. Braunwald's heart disease: a textbook of cardiovascular medicine. Zipes DP, Libby P, Bonow RO, Mann DL, Tomaselli GF, Braunwald E, editors - Philadelphia, PA: Elsevier; 2019. - 1944p. [Olgin JE, Tomaselli GF, Zipes DP. Ventricular arrhythmias. PP. 753–771.] ISBN 978-0-323-46342-3
2. Takahashi S, Mine T, Ashida K, Kishima H, Masuyama T, Ishihara M. Left Ventricular Inflow Velocity Pattern in Patients With Symptomatic Premature Ventricular Contraction. Circulation Journal. 2019;84(1):26–32. DOI: 10.1253/circj.CJ-19-0605
3. Cluitmans MJM, Bear LR, Nguyên UC, Van Rees B, Stoks J, Ter Bekke RMA et al. Noninvasive detection of spatiotemporal activation-repolarization interactions that prime idiopathic ventricular fibrillation. Science Translational Medicine. 2021;13(620):eabi9317. DOI: 10.1126/scitranslmed.abi9317
4. Huizar JF, Tan AY, Kaszala K, Ellenbogen KA. Clinical and translational insights on premature ventricular contractions and PVC-induced cardiomyopathy. Progress in Cardiovascular Diseases. 2021;66:17–27. DOI: 10.1016/j.pcad.2021.04.001
5. Kobayashi Y. Idiopathic Ventricular Premature Contraction and Ventricular Tachycardia: Distribution of the Origin, Diagnostic Algorithm, and Catheter Ablation. Journal of Nippon Medical School. 2018;85(2):87–94. DOI: 10.1272/jnms.2018_85-14
6. Cronin EM, Bogun FM, Maury P, Peichl P, Chen M, Namboodiri N et al. 2019 HRS/EHRA/APHRS/LAHRS expert consensus statement on catheter ablation of ventricular arrhythmias. EP Europace. 2019;21(8):1143–4. DOI: 10.1093/europace/euz132
7. Amsallem M, Mercier O, Kobayashi Y, Moneghetti K, Haddad F. Forgotten No More: A Focused Update on the Right Ventricle in Cardiovascular Disease. JACC: Heart Failure. 2018;6(11):891–903. DOI: 10.1016/j.jchf.2018.05.022
8. Kossaify A. Echocardiographic Assessment of the Right Ventricle, from the Conventional Approach to Speckle Tracking and Three-Dimensional Imaging, and Insights into the "Right Way" to Explore the Forgotten Chamber. Clinical Medicine Insights: Cardiology. 2015;9:65–75. DOI: 10.4137/CMC.S27462
9. Badano LP, Muraru D, Parati G, Haugaa K, Voigt J-U. How to do right ventricular strain. European Heart Journal - Cardiovascular Imaging. 2020;21(8):825–7. DOI: 10.1093/ehjci/jeaa126
10. Higuchi K, Bhargava M. Management of premature ventricular complexes. Heart. 2022;108(7):565–72. DOI: 10.1136/heartjnl-2020-318628
11. Bala R, Garcia FC, Hutchinson MD, Gerstenfeld EP, Dhruvakumar S, Dixit S et al. Electrocardiographic and electrophysiologic features of ventricular arrhythmias originating from the right/left coronary cusp commissure. Heart Rhythm. 2010;7(3):312–22. DOI: 10.1016/j.hrthm.2009.11.017
12. Yoshida N, Inden Y, Uchikawa T, Kamiya H, Kitamura K, Shimano M et al. Novel transitional zone index allows more accurate differentiation between idiopathic right ventricular outflow tract and aortic sinus cusp ventricular arrhythmias. Heart Rhythm. 2011;8(3):349–56. DOI: 10.1016/j.hrthm.2010.11.023
13. Huizar JF, Ellenbogen KA, Tan AY, Kaszala K. Arrhythmia-Induced Cardiomyopathy: JACC State-of-the-Art Review. Journal of the American College of Cardiology. 2019;73(18):2328–44. DOI: 10.1016/j.jacc.2019.02.045
14. Wijnmaalen AP, Delgado V, Schalij MJ, Van Huls Van Taxis CFB, Holman ER, Bax JJ et al. Beneficial effects of catheter ablation on left ventricular and right ventricular function in patients with frequent premature ventricular contractions and preserved ejection fraction. Heart. 2010;96(16):1275–80. DOI: 10.1136/hrt.2009.188722
15. Scorza R, Shahgaldi K, Rosenqvist M, Frykman V. Evaluation of patients with high burden of premature ventricular contractions by comprehensive transthoracic echocardiography. IJC Heart & Vasculature. 2022;42:101124. DOI: 10.1016/j.ijcha.2022.101124
16. Jia R, Xu Y, Luo Y, Yang C, Zou S, Gong S et al. The ratio of main pulmonary artery to ascending aorta diameter is associated with the right ventricular outflow tract ventriculararrhythmias. Journal of Interventional Cardiac Electrophysiology. 2021;62(1):57–62. DOI: 10.1007/s10840-020-00872-1
17. Del Carpio Munoz F, Syed FF, Noheria A, Cha Y-M, Friedman PA, Hammill SC et al. Characteristics of Premature Ventricular Complexes as Correlates of Reduced Left Ventricular Systolic Function: Study of the Burden, Duration, Coupling Interval, Morphology and Site of Origin of PVCs. Journal of Cardiovascular Electrophysiology. 2011;22(7):791–8. DOI: 10.1111/j.1540-8167.2011.02021.x
18. Cheng Z, Cheng K, Deng H, Chen T, Gao P, Zhu K et al. The R-wave deflection interval in lead V3 combining with R-wave amplitude index in lead V1: A new surface ECG algorithm for distinguishing left from right ventricular outflow tract tachycardia origin in patients with transitional lead at V3. International Journal of Cardiology. 2013;168(2):1342–8. DOI: 10.1016/j.ijcard.2012.12.013

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