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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Subclinical left atrium remodelling in patients with frequent premature ventricular contractions

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Abstract

Background: Premature ventricular contractions (PVCs) may cause subtle changes in left atrium (LA) structure and function which may not be readily detected by conventional parameters.

Aim: To explore the relationship between PVCs and LA shape and size remodelling in patients without known structural heart disease.

Methods: PVC frequency per 24 h was divided into tertiles. Echocardiographic measurements included left ventricular ejection fraction (LVEF) and volume, LA volume (LAV), mid-LA transverse diameter (LAt), and basal LA maximal transverse diameter (LAb). Trapezoidal LA shape was defined by LAt less than LAb. The association between PVCs and LA shape was explored by multivariable adjusted logistic regression.

Results: The 121 patients had a mean age of 43.1 years (63% male) and mean LVEF of 56%. Mean LAV was 57.7 mL in the lowest PVC tertile, 70.9 mL in the middle, and 87.1 mL in the highest PVC tertile ($p < 0.001$). Trapezoidal LA shape was documented in 32 patients. Five per cent of patients in the lowest and 65% in the highest PVC tertile had trapezoidal LA shape ($p < 0.001$). PVC frequency correlated with LAV ($r = 0.87$, $p < 0.001$) and LAb ($r = 0.56$, $p < 0.001$). Each 10% increase in PVCs was significantly associated with trapezoidal LA shape (OR = 1.32, 95% CI 1.17–1.48), after adjustment for age, sex, mean blood pressure, LVEF, and LV end-diastolic diameter.

Conclusions: Frequent PVCs are associated with larger LAV and trapezoidal LA shape in otherwise healthy patients, suggesting LA remodelling in response to increased LV filling pressure.

Key words: ventricular arrhythmia, atrial remodelling, diagnosis

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INTRODUCTION

Idiopathic premature ventricular contractions (PVCs) are characterised by the monomorphic pattern of ectopy on an otherwise normal 12-lead electrocardiogram (ECG), normal RR variability on 24-h Holter monitoring, and suppression of the PVCs with increasing heart rate at stress test. A normal echocardiogram may reinforce the diagnosis of benign PVCs [1]. Although some studies have linked PVCs to cardiomyopathy and left ventricular (LV) systolic dysfunction that is reversible after elimination of the frequent PVCs using catheter ablation [2–4], the mechanisms of these associations remain unclear in patients without underlying heart disease, and PVCs are generally considered benign [5, 6]. On the other hand, the abnormal haemodynamic effect of PVCs on LV systolic function may cause subtle changes of left atrium (LA) structure and function which may not be readily detected by conventional parameters.

To the best of our knowledge, no previous study has analysed the early subclinical changes in LA size and shape in patients with frequent PVCs, normal ejection fraction (EF) and no structural heart disease. The aim of this study was therefore to assess LA remodelling in patients with frequent PVCs and to explore the relationship between the PVC burden and LA shape and size.

METHODS

Study population

For this analysis, we included consecutive patients referred to our clinic for persistent symptomatic PVCs in excess of 1,000/24 h on Holter monitoring without evidence of

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structural heart disease. PVCs were defined as persistent if occurring for at least three months before the baseline echocardiography examination. A 24-h Holter recording was obtained within 72 h of the echocardiographic examination to quantify the burden of ventricular ectopy. Exclusion criteria comprised echocardiographic evidence of structural heart disease, the presence of hypertension for five years or more and/or interventricular septum ≥ 13 mm, persistent atrial fibrillation (AF), sick sinus syndrome, moderate or severe valvular regurgitation (regurgitation volume > 30 mL/beat) [7], coronary artery disease, and significant co-morbidities [serum creatinine > 114.9 μmol/L (1.3 mg/dL), diabetes, abnormal liver enzymes]. Coronary artery disease was excluded based on the lack of clinical symptoms, Framingham score estimated risk of less than 10% at ten years, and a normal stress test. The stress test was performed in 89 patients and was negative for ischaemic heart disease and arrhythmia. Patients with unexplained syncope, predominantly non-sustained runs of ventricular tachycardia, those with a known history of sustained ventricular tachycardia or complex ventricular arrhythmias were also excluded. No patient had more than three recurrent episodes of paroxysmal AF per year.

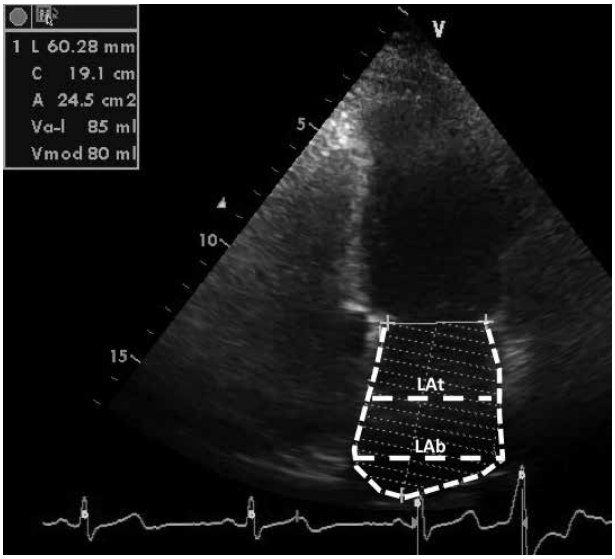

Figure 1. Left atrium (LA) size and shape assessment in studied patients. Both mid-LA transverse diameter (LAt) and basal LA diameter (LAb) were measured in apical four-chamber view at end-systole just before mitral valve opening. The trapezoid LA shape is defined by LAt < LAb

Echocardiographic examination

Ultrasound images were explored with patients in the left lateral decubitus position, using a GE VIVID 7 machine (GE Health Medical, Milwaukee, WI, USA) with simultaneous ECG recording. The echocardiographic examination was performed using standard views and techniques [8]. Standard echocardiographic measurements included interventricular septum, left ventricular end-diastolic diameter (LVEDD), left ventricular end-diastolic volume (LVEDV), and EF (Simpson's method). LA measurements included M-mode diameter (LAd) measured in the parasternal long-axis view; LA mid-transversal diameter (LAt) and LA volume (LAV) measured in the apical four-chamber view. In order to better define LA shape in the apical four-chamber view, we also ascertained the basal LA diameter (LAb), measured as the maximal transverse distance at the base of the LA, between the septal and the lateral walls. We defined the LA as being trapezoidal in shape when LAb > LAt (Fig. 1), and being ellipsoid in shape when LAb < LAt [9]. All LA measurements were performed at end-systole just before mitral valve opening and maximal LA size was considered for evaluation.

An optimal four-chamber view required for LA measurements was considered when visualisation of the maximal number of pulmonary veins was possible. All efforts were made during scanning to ensure optimisation of LA view by maximising cavity size. In cases with borderline shape, visual assessment was performed by two independent observers and a decision was reached by consensus. The echocardiographers had no knowledge of the Holter results, as the Holter monitoring was performed after the echocardiographic assessment. Of the 123 eligible patients, two were excluded because of poor acoustic window, due to overweight in one case and to pulmonary emphysema in the other.

Statistical analysis

Clinical and demographic features are presented as mean ± standard deviation or medians (range) for continuous variables and as prevalence for categorical variables. Patients were categorised by PVC frequency tertiles. Associations between variables and PVC frequency were examined by Spearman correlations between continuous variables; Mantel-Haenszel χ^2 test was used to examine the relationships between the categorical variables and trapezoidal shape. Logistic regression model was used to explore factors associated with a higher risk of trapezoidal shape of the LA. The final model included age, sex, mean arterial pressure, EF, LVEDD, and PVCs; the latter two variables were expressed as logarithm base 1.1 to reflect the associations with trapezoidal LA corresponding to a 10% difference in these variables. Mean arterial pressure (MAP) was computed as (2 × diastolic blood pressure + systolic blood pressure)/3. Calculations were performed using SAS software version 9.2 (SAS Institute, Cary, NC, USA). Results were considered significant if $p < 0.05$.

RESULTS

Patient characteristics

The study included 121 patients (76 males, 45 females) with a mean age of 43.1 ± 11.5 years. The demographic and clinical characteristics of the cohort are presented in Table 1 ac-

Table 1. Clinical and demographic characteristics of the study population, according to premature ventricular contractions (PVCs) tertiles. Spearman correlations between PVCs as a continuous variable and the other continuous variables are also shown

	All	PVC tertiles (number/24 h)			Correlation with PVCs
		Lowest tertile	Middle tertile	Highest tertile	
PVCs†	7,654 (1,124–33,870)	2,181 (1,124–3,521)	7,654 (3,547–11,171)	15,767 (11,231–33,870)	
n (% male)	121 (63%)	40 (60%)	41 (68%)	40 (60%)	p = 0.7*
Age [years]	43.1 ± 11.5	40.1 ± 12.0	45.5 ± 10.7	43.2 ± 11.6	r = 0.04, p = 0.66
Mean heart rate [bmp]	74 ± 8	75 ± 8	73 ± 8	73 ± 7	
MAP [mm Hg]	95.0 ± 8.0	92.0 ± 8.0	96.0 ± 7.0	94.0 ± 7.0	r =0.08, p = 0.38
Body mass index [kg/m^2]	24.2 ± 2.2	24.3 ± 2.5	24.2 ± 2.3	23.8 ± 1.6	r = –0.05, p = 0.56
Calcium blockers		5%	15%	13%	0.3
Beta-blockers		30%	32%	44%	0.4
RAAS inhibitors		35%	44%	38%	0.7
Paroxysmal AF	4	2	1	1	
Trapezoidal LA**	26%	6	13	81	p < 0.001*
Echocardiographic parameters					
Interventricular septum [mm]	10.5 ± 1.0	10.5 ± 1.0	10.3 ± 0.9	10.6 ± 0.9	r = 0.03, p = 0.73
Ejection fraction [%]	56.4 ± 4.5	56.8 ± 4.7	57.3 ± 4.7	55.2 ± 4.0	r = –0.11, p = 0.25
LVEDD [mm]†	51 (42–61)	51 (42–61)	51 (42–55)	55 (51–59)	r = 0.15, p = 0.11
LVEDV [mL]†	92 (67–129)	90 (67–123)	98 (79–119)	110 (98–129)	r = 0.39, p < 0.001
LVMI [g/m^2]†	113.9 (71.5–164.2)	115.4 (73.2–160.7)	113.7 (71.5–152.6)	114.3 (73.3–164.2)	r = 0.05, p = 0.57
LAV [mL]	71.9 ± 15.2	57.7 ± 8.6	70.9 ± 9.1	87.1 ± 10.0	r = 0.87, p < 0.001
LAVI [mL/m^2]†	39.9 (22.9–59.4)	32.6 (22.9–46.7)	39.3 (25.9–45.8)	47.0 (36.6–59.4)	r = 0.86, p < 0.001
LAd [mm]	4.2 ± 0.5	4.0 ± 0.5	4.3 ± 0.4	4.4 ± 0.6	r = 0.27, p = 0.005
LAt [mm]	4.3 ± 0.5	4.1 ± 0.5	4.3 ± 0.4	4.3 ± 0.5	r = 0.25, p = 0.005
LAb [mm]	4.3 ± 0.6	4.0 ± 0.5	4.2 ± 0.5	4.7 ± 0.6	r = 0.56, p < 0.001

Values represent means ± standard deviation; *p-value from χ^2-test; **Trapezoidal LA defined as LAt < LAb (0 or 1); †Median (range); AF — atrial fibrillation; LA — left atrium; LAb — basal LA dimension; LAd — M-mode LA diameter; LAt — mid-LA transverse diameter; LAVI — LA volume index (body surface area standardised); LVEDD — left ventricular end-diastolic diameter; LVEDV — left ventricular end-diastolic volume; LVMI — left ventricular mass index (body surface area standardised); MAP — mean arterial pressure defined as [(2 × diastolic) + systolic]/3; RAAS — renin–angiotensin–aldosterone system

cording to PVC tertile. The sex distribution was similar across PVC groups, and there was no significant correlation between age, MAP and PVC frequency. On the 12-lead ECG, the PVCs were presumed to originate in the right ventricular outflow tract in 72 (59%) patients and from other sites in 51 (41%), including the LV outflow tract in nine patients, a right ventricular site other than the outflow tract in 15 patients, the mitral annulus in three patients, the posteroseptal LV wall in four patients, an aortic cusp in two, and the pulmonary artery in one patient. In seven patients, the site of origin could not

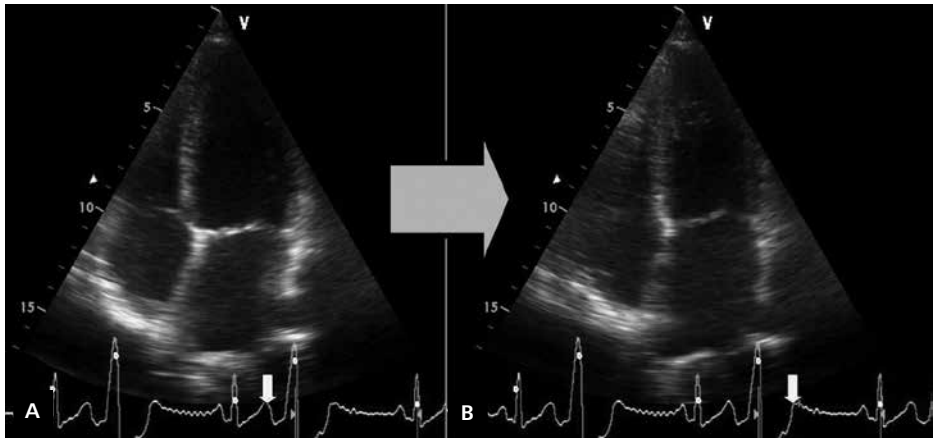

Figure 2. **A**, **B**. Example of left atrium stretch during premature ventricular contractions (PVCs) in a male adult with sinus rhythm and > 20,000 PVCs/24 h. Although this figure illustrates acute remodelling during a PVC cardiac cycle, measurements were performed in sinus rhythm only

be determined. Nevertheless, the precise site of origin of the PVCs could not be determined, as no additional invasive assessment was performed for this scope.

Echocardiographic results

No echocardiography abnormalities of the right ventricle were observed. As presented in Table 1, mean LAV was positively associated with PVC frequency, with an average value of 57.7 mL in patients in the lowest PVC frequency tertile and 87.1 mL in patients included in the highest PVC tertile. PVC burden as a continuous variable correlated strongly with LAV ($r = 0.87$, $p < 0.001$), LAb ($r = 0.56$, $p < 0.001$), and LVEDV ($r = 0.39$, $p < 0.001$), and to a lesser degree, albeit significantly, with the other echocardiographic measurements. Trapezoidal LA was ascertained in 26% of patients, 5% of lowest PVC tertile patients, 10% of those in the middle tertile, and 65% of patients in the highest PVC tertile (Table 1). The mean LAt/LAb ratio was 1.04 ± 0.07 mm in patients in the lowest PVC tertile, 1.02 ± 0.06 mm in those the middle PVC tertile, and 0.94 ± 0.07 mm in patients in the highest PVC tertile ($r = –0.58$, $p < 0.001$). Figure 2 shows an example of trapezoidal LA configuration in a 41-year-old male with LVEF of 61%, included in group 3. Correlations between trapezoidal LA and echocardiographic measurements are presented in Table 2. The relationship between PVCs/24 h and LAV is shown in Figure 3, the trend line illustrating the monotonic relationship; a higher frequency of patients with trapezoidal LA was found among participants with higher PVC number and higher LAV. The age-sex-adjusted odds of trapezoidal LA shape were significantly higher (odds ratio [OR] = 1.31, 95% confidence interval [CI] 1.17–1.47) with each 10% increase in PVCs, and remained unchanged after additional adjustment for MAP, LVEDD, LAd, and EF (Table 3).

Table 2. Correlations between echocardiographic left atrium (LA) and left ventricle (LV) measurements and the presence of trapezoidal LA shape. Trapezoidal LA is defined as LAt [mm] < LAb [mm]

	Trapezoidal LA (n = 32)
LAd	p = 0.04
LAt	p = 0.003
LAb	p < 0.001
LAVI	p < 0.001
LVEDD	p = 0.16
LVEDV	p < 0.001
LVMI	p = 0.56
LVEF	p = 0.34

p-value from χ^2-test; LAd — M-mode LA diameter; LAt — mid-LA transverse diameter (in four-chamber view); LAb — basal LA dimension (in four-chamber view); LAV — LA volume (in four-chamber view); LVEF — left ventricular ejection fraction; LVEDD — left ventricular end-diastolic diameter; LVEDV — left ventricular end-diastolic volume; LAVI — left atrium volume index (body surface area standardised); LVMI — left ventricular mass index (body surface area standardised)

DISCUSSION

The present study offers new information regarding LA size and shape remodelling in patients with frequent PVCs. LAV was larger with a higher PVC burden, suggesting higher LV filling pressure and LA stretch. In addition, the LA trapezoid contour correlated strongly with PVC frequency in the absence of known heart disease, suggesting concomitant LA shape remodelling. Because conventional LA measurement such as single LAd assessment probably underestimates the true LA size [8], the additional measurement of LAb may allow a better depiction of LA shape and more precise volume tracing and estimation, and may therefore represent an alternative to using the ellipsoid formula [8].

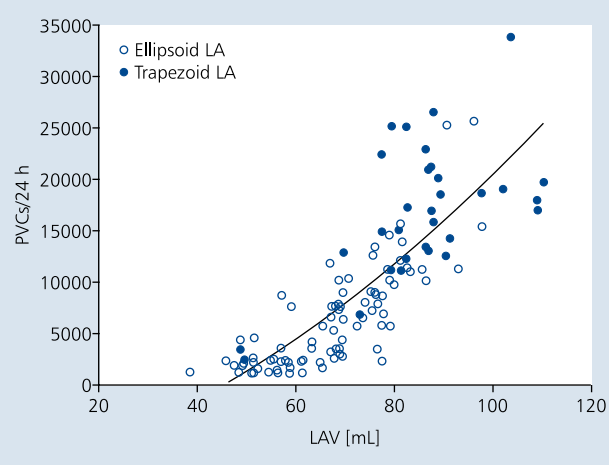

Figure 3. Scatterplot of premature ventricular contractions (PVCs) as a function of left atrial (LA) volume (LAV). The filled circles represent patients who exhibited trapezoidal LA shape and the open circles represent those who did not (ellipsoidal LA shape). The dashed lines reflect the cutoffs for the PVC and LAV tertiles respectively. A trend line shows the monotonic relationship between PVCs and LAV

Table 3. Odds ratios (OR) and 95% confidence interval (CI) of trapezoidal left atrium (LA) shape for each 10% increase in premature ventricular contractions (PVCs), adjusted for age, sex, mean arterial pressure (MAP), M-mode LA diameter (LAd), left ventricular end-diastolic diameter (LVEDD), and left ventricular ejection fraction (LVEF)

Variable in the model	OR (95% CI)
Age/5 years	1.02 (0.69–1.52)
Sex (male vs. female)	1.44 (0.47–4.41)
MAP/5 mm Hg	0.70 (0.36–1.34)
LAd (per 10% increase)	1.01 (0.65–1.58)
LVEDD (per 10% increase)	1.18 (0.63–2.21)
LVEF (per 10% decline)	0.90 (0.46–1.74)
PVCs (per 10% increase)	1.32 (1.17–1.48)

Previous studies have indicated an association between frequent ventricular PVCs and long-term systolic LV dysfunction in patients with structurally normal hearts [3, 10, 11, 12]. A study following 239 consecutive patients with frequent PVCs but without detectable heart disease for up to 5.6 years found that PVC frequency correlated positively with LVEDV and inversely with EF [12]. One suggested mechanism for these associations is tachycardia induced cardiomyopathy; on the other hand, PVCs in excess of 20,000/24 h result in a lower than normal heart rate and a reduced cardiac output that may beget bradycardia-induced LV dysfunction [12]. Haemodynamic alterations analogous to those observed when the ventricular myocardium is exposed to volume overload probably account for the subtle anatomical remodelling of the LA. LV dysfunction and/or mechanisms resembling atrial remodelling after the loss of atrio-ventricular synchrony [13] may also account for LA changes in size and shape associated with PVCs in the absence of another contributing pathology. LV filling pressures are likely to rise in the presence of a remodelled LA, leading to chronic diastolic dysfunction [14]. Indeed, subtle cardiac dysfunction induced by frequent PVCs have been detected by tissue Doppler imaging and speckle tracking imaging analysis in patients without apparent cardiomyopathy [15], and radiofrequency (RF) ablation was shown to successfully eliminate PVCs and improve cardiac function [11, 16].

A prospective study in 14,783 middle-aged participants in the ARIC study [17] found a 40% higher incidence of stroke in those with PVCs compared to those without. Interestingly, this risk was two-fold higher among healthier individuals, representing 46% of the study cohort. Because PVCs are associated primarily with embolic rather than with thrombotic stroke, the authors speculated that PVCs increase the risk of AF, possibly through cardiac remodelling and increased LA size [18–20]. In patients with frequent PVCs and an otherwise normal heart, physicians are faced with the choice of either treating the PVCs as a benign occurrence or performing RF ablation in highly symptomatic patients. However, as shown here and concurring with previous longitudinal studies, earlier detection and proof of subclinical LA dilatation in the follow-up of these patients may play a role in the management decisions regarding timing of RF ablation before LV dysfunction develops.

Limitations of the study

The findings in this study are subject to several limitations. Firstly, serial measurements were not available for the present analysis, and the cross-sectional design does not allow a cause and effect assessment for the various associations. Thus, the linear LA shape change with PVC burden may reflect primary myopathy at both atrial and ventricular level without LA shape change being secondary to PVCs. Nevertheless, we believe such an occurrence is unlikely for the relatively large number of patients carefully selected. As a sensitivity analysis, we excluded four patients with up to three recurrent episodes of paroxysmal AF per year, who could have had primary atrial myopathy leading to AF and not have atrial remodelling secondary to PVCs. In this secondary analysis, the adjusted OR of trapezoidal LA shape changed marginally without changing the overall conclusion of the study (OR = 1.35, 95% CI 1.18–1.53 with each 10% increase in PVCs). The lack of follow-up, except in a small number of patients, precludes our knowing whether a permanent change in LA shape from ellipsoid to trapezoid makes such patients more susceptible to AF. Right atrial and right ventricular diameters were not recorded as part of the evaluation, in part because none of the included patients had apparent dilatation of the right chambers. Secondly, the study cohort is a convenient

sample of consecutive Caucasian patients meeting specific inclusion criteria and therefore the results of this study may not be generalisable to a larger population. Thirdly, selection bias is introduced to the extent that exclusion of structural heart disease was based on non-invasive tests. Also, the PVC classification was based on a single 24-h Holter monitoring, which is prone to higher variability and less precise classification compared to a longer monitoring time.

CONCLUSIONS

Higher PVC frequency is associated with larger LAV and trapezoidal LA shape in otherwise healthy patients, possibly suggesting LA remodelling in response to increased LV filling pressure. Trapezoid LA with atrialisation of the pulmonary veins and predominant dilatation of the LA at the base, more than at the annulus, may explain the underestimation of LA volume using the ellipse formula. Further longitudinal studies are needed to validate these findings.

Conflict of interest: *none declared*

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Subkliniczna przebudowa lewego przedsionka u chorego z częstymi przedwczesnymi skurczami komorowymi

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Streszczenie

Wstęp: Przedwczesne skurcze komorowe (PVCs) mogą powodować subtelne zmiany w strukturze i czynności lewego przedsionka (LA), które mogą być trudne do wykrycia na podstawie analizy konwencjonalnych parametrów.

Cel: Celem pracy była ocena zależności między występowaniem PVC a zmianą kształtu i wielkości LA u pacjentów bez stwierdzonej strukturalnej choroby serca.

Metody: Częstość występowania PVC w ciągu 24 h podzielono na tercyle. Zmierzone parametry echokardiograficzne obejmowały frakcję wyrzutową lewej komory (LVEF), objętość lewej komory, objętość lewego przedsionka (LAV), wymiar poprzeczny LA w połowie wysokości (LAt, *mid-LA transverse diameter*) i maksymalny wymiar poprzeczny LA mierzony u jego podstawy (LAb, *basal LA maximal transverse diameter*). Trapezoidalny kształt LA definiowano jako wartość LAt mniejszą niż wartość LAb. Zależności między występowaniem PVC a kształtem LA analizowano, stosując wieloczynnikową regresję logistyczną (dane skorygowane).

Wyniki: W badaniu uczestniczyło 121 chorych, których średnia wieku wynosiła 43,1 roku (63% mężczyzn), a średnia LVEF — 56%. Średnia LAV wynosiła 57,7 ml w najniższym tercylu PVC, 70,9 ml w środkowym tercylu i 87,1 ml w najwyższym tercylu ($p < 0,001$). Trapezoidalny kształt LA stwierdzono u 32 osób. U 5% chorych w najniższym tercylu i u 65% chorych w najwyższym tercylu kształt LA był trapezoidalny ($p < 0,001$). Częstość PVC wiązała się z LAV ($r = 0,87$; $p < 0,001$) i LAb ($r = 0,56$; $p < 0,001$). Każde zwiększenie PVC o 10% istotnie korelowało z trapezoidalnym kształtem LA (OR = 1,32; 95% CI 1,17–1,48) po skorygowaniu względem wieku, płci, średniego ciśnienia tętniczego, LVEF i wymiaru końcoworozkurczowego LV.

Wnioski: Częste przedwczesne PVC wiążą się z większymi wartościami LAV i trapezoidalnym kształtem LA u pacjentów bez żadnych innych chorób, co sugeruje przebudowę LA w odpowiedzi na zwiększone ciśnienie napełniania LV.

Słowa kluczowe: arytmia komorowa, przebudowa przedsionka, rozpoznanie

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