European Review for Medical and Pharmacological Sciences 2022; 26: 1492-1499

Left atrial functions in long term after catheter ablation of premature ventricular complexes

A. TUTUNCU, S. KANAT

Department of Cardiology, University of Health Sciences, Bursa Postgraduate Hospital, Bursa, Turkey

Abstract. – OBJECTIVE: Premature ventricular complex (PVC) ablation has been shown to improve left ventricle (LV) diastolic function and left atrial (LA) reverse modeling, especially in the short term. In the present study, PVC ablation via radiofrequency catheter (RFCA) was evaluated with respect to its long-term effects on LA size and dynamic functions.

PATIENTS AND METHODS: A total of 71 patients (age 43.49±12.8 years, 37 men [52%]) with high-burden (21% mean burden) PVCs who were treated with RFCA were included in this retrospective study. The effects of RFCA on three characteristics – LV systolic functions, LV diastolic dysfunctions and mechanical effects pertaining to the LA – were examined by echocardiography at baseline and at 3 months and 18 months after the procedure.

RESULTS: Advancement in LV ejection fraction (LVEF) was seen on long-term follow-up (LVEF baseline 53.01; 3rd month 54.55; 18th month 58.02; $p<0.001$). LA function gradually improved after RFCA. The LA volume index was significantly decreased after RFCA (baseline 18.15±2.89; 3 months 17.11±2.71; 18 months 16.67±2.61; $p<0.001$). The LA passive emptying fraction was still increasing over long-term follow-up (baseline 33.33; 3rd month 37.11; 18th month 40.91; $p<0.001$).

CONCLUSIONS: In the present study, in patients with or without apparent cardiomyopathy, RFCA was shown to successfully eliminate PVCs and improve LA functions in the long term.

Key Words:
Premature ventricular complex, Radiofrequency ablation, Left atrial function, Echocardiography.

Introduction

Premature ventricular complexes (PVCs) are often associated with structural heart disease; however, they are also arguably common in the general population. Despite usually being asymptomatic, symptomatic PVCs can present with a range of findings, from palpitations, chest pain and weakness to dyspnea and even heart failure-like symptoms[1]. PVCs can affect the left ventricle (LV) ejection fraction (EF) through various extracellular and cellular mechanisms. After the exclusion of structural heart disease, the presence of PVC alone has been held responsible for reversible LV dysfunction – a condition that has gained recognition as PVC-induced cardiomyopathy[2].

There are several predictors of LV dysfunction in patients with PVC, including high PVC burden, lack of sustained ventricular tachycardia, interpolated PVC, epicardial origin, retrograde P wave and PVC QRS duration. Among these factors, the strongest predictor is accepted to be a high PVC burden, which is determined by the PVC to total QRS ratio during a 24-hour assessment[3]. However, the cutoff point for PVC burden remains controversial. In a study, a PVC burden >10% was identified as responsible for reversible LV dysfunction, whereas another study determined that a PVC burden in excess of 24% was an independent risk factor for PVC-induced cardiomyopathy[4,5].

Park et al[6] demonstrated that frequent PVC might be associated with left atrial (LA) enlargement in patients with normal LV EF and that atrial anatomic remodeling could precede LV geometry changes and systolic dysfunction.

The LA contributes to the systolic and diastolic functions of the LV, and the literature has revealed a reciprocal relationship between heart diseases and the size and function of the LA[7,8]. Therefore, improving LA function will be of clinical importance in this patient group in the long term.

There have not been sufficient data in the literature on the long-term effect of PVC ablation via radiofrequency catheter ablation (RFCA) on LA

Corresponding Author: Ahmet Tutuncu, MD; e-mail: tutuncuahm@yahoo.com

function. We previously demonstrated that PVC ablation improves LV diastolic function and LA reverse modeling, especially in the short term[9]. In the present study, PVC treatment via RFCA was evaluated with respect to its long-term effects on LA size and dynamic functions.

Patients and Methods

Study Population

A total of 71 patients older than 18 years old who underwent PVC catheter ablation at a tertiary referral center were evaluated in the retrospective study design. The exclusion criteria were refusal to participate in the study and the presence of severe valvular lesions; ischemic heart disease; a previous history of heart surgery, myocarditis, pericarditis and other known diagnoses of cardiomyopathy; and sustained ventricular or supraventricular tachycardias. All patients were evaluated with 24-hour electrocardiogram (ECG) Holter monitorization at least once before ablation. To exclude any coronary artery diseases or valvular/structural heart diseases, we conducted radionuclide scans, computerized cardiac tomography and invasive coronary angiography when required at least three months before ablation.

The study was approved by the Local Ethics Committee (Bursa Postgraduate Hospital Registration Number 2011-KAEK-25 2019/04-01) and was conducted according to the Helsinki Declaration. Written informed consent was obtained from all of the patients before their inclusion in the study.

Echocardiography and Left Atrium Volumetric Measurements

Two-dimensional transthoracic echocardiography (Philips i33, Eindhoven, the Netherlands; and Vivid-7, GE Wingmed sound Horten, Norway) was performed at least three months prior to the ablation procedure for each patient. Echocardiography examinations were repeated 3 and 18 months after the ablation procedure. The LV systolic and diastolic functions and LA functions were evaluated in each examination. Standard measurements were performed according to most current guidelines. LVEF was measured using the Simpson method[10]. Patients were divided into two groups according to their LVEF: the normal group (EF $\geq$ 50%) and the LV dysfunction group (EF < 50% defined as PVC-induced cardiomyopathy (PVC-CMP)). To assess diastolic dysfunction, mitral inflow with pulse wave Doppler and tissue Doppler was used to measure the lateral and septal zones of the basal segment of the LV mitral annulus. LV diastolic dysfunction was classified according to Doppler parameters and the latest guidelines[10-12].

LA volumetric measurements were created using apical four-chamber and apical two-chamber images. The volume before the opening of the mitral valve was considered the maximum LA volume (LaVmax), the minimum LA volume (LaVmin) was determined immediately after the mitral valves were closed, and the presystolic LA volume (LaVp) was recorded as the volume at the beginning of the p wave on superficial ECG. The results were normalized by dividing by body surface area. With the use of these measurement results, we calculated the following values via the given formulae: LA volume index: volume/body surface area; LA total emptying volume: LaVmax-LaVmin; LA total ejection fraction: (LaVmax-LaVmin)/LaVmax; LA passive emptying volume: LaVmax-LaVp; LA passive emptying fraction: (LaVmax-LaVp)/LaVmax; LA active emptying volume: LaVp-LaVmin; and LA active emptying fraction: (LaVp-LaVmin)/LaVp.

Electrophysiology Study

Before ablation, the antiarrhythmic drugs used by patients were discontinued for a duration corresponding to five times the half-life of any given drug, except for amiodarone. Electrophysiological studies were conducted under local anesthesia to prevent PVC suppression. Briefly, a decapolar catheter was placed in the coronary sinus via the right femoral vein. If there was little or no PVC during the procedure, isoprotenol infusion was administered, and ventricular stimulation was applied from the right ventricular apical and outflow tracts.

Mapping and Catheter Ablation Procedure

Electroanatomic mapping was performed with the CARTO (BiosenseWebster, Irvine, CA, USA) or Ensite™ Precision (Abbott, Chicago, IL, USA) systems. An 8F 3.5-mm irrigated type Thermocool Smarttouch CF (BiosenseWebster Inc., Diamond Bar, CA, USA) or a 4-mm irrigated type FlexAbility (Endosense/Abbott, St. Paul, MN, USA) and a 2–5–2 mm interelectrode spacing catheter were used for ablation. In the event that PVCs were found to originate from the left ventricle, access to the left ventricle was obtained

through either the retrograde aortic route or the trans-septal route. During the procedure, heparin was administered with an activated clotting time between 300 and 350 seconds. PVCs originating from the LV summit region were reached from the coronary sinus or epicardial route.

While performing electroanatomic mapping, areas with radial distribution that showed QS morphology with the earliest local activation time on the unipolar electrogram were marked. Activation mapping identified sites for ablation with the earliest local activation advancing the onset of PVC QRS by at least 30 ms. Before ablation, if possible, compatibility was checked in at least 11 derivations of the superficial ECG to ensure conformity with pace mapping and clinical PVC.

An irrigated catheter in power-controlled mode with an energy setting of 30–40 W and a temperature limit of 43°C was applied until a 10-ohm drop in the tissue was achieved. An acute, successful ablation was defined as the absence of recurrence and noninducibility of culprit PVC. The inducibility of PVC was checked at least 30 min after ablation with and without isoproterenol administration and/or programmed electrical stimulation.

Follow-up

Electrocardiography, 24-h Holter recording and 2-dimensional 2D-TTE were repeated at the three-month follow-up. At least an 80% decrease in PVCs with the same morphology on 24 h Holter recording at three months was defined as successfully sustained ablation. Antiarrhythmic medications were not started again after the ablation procedure.

Statistical Analysis

The Shapiro-Wilk test was used to assess whether the variables conformed to a normal distribution. Continuous variables were summarized as the mean ± standard deviation in the presence of a normal distribution or as the median (minimum, maximum) when a normal distribution was not present. Friedman's two-way analysis of variance (ANOVA) and ANOVA for repeated measurements were used to evaluate changes in echocardiographic parameters after ablation and to assess the significance of changes in parameters with RFCA, along with the effects of various factors on these changes. The comparison of continuous variables between the PVC-CMP and normal LVEF groups was performed using the Mann-Whitney U and the independent samples *t*-test, depending on the normality of the distribution. IBM SPSS Statistics software, version 21.0 (IBM Corp., 2012, Armonk, NY, USA), was used to perform all statistical analyses, and $p<0.05$ was set as the threshold for statistical significance.

Results

PVC ablation *via* RFCA was applied in 71 patients. Six patients were excluded from the analysis due to recurrence during follow-up. Seventy-one patients with frequent PVCs were included in the analysis (mean age 43.49 ± 12.89 old, 52.11% male [37/71]). Seven patients had a previous history of ablation. In the 24-hour Holter recordings, the average PVC burden of the patients was 21%. The mean LVEF of the patients included in the study was 55 ± 9%. The vast majority of patients were under at least one antiarrhythmic therapy (83.10%). Other characteristics are shown in Table I. The PVC origin in the

Table I. Baseline characteristics of the study patients.

Age	43.49 ± 12.89
Gender (Male/Total)	37/71 (52.11%)
Body mass index	26.58 ± 2.88
Risk factors, n (%)	
Diabetes mellitus	15/71 (21.1%)
Hypertension	16/71 (22.5%)
Hyperlipidemia	12/71 (16.9%)
Smoking	20/71 (28.2%)
Prior history of ablation	7 (9.90%)
Left ventricular Ejection Fraction	53 ± 9%
Electrocardiography findings	
Heart rate (Beats/min)	75
Intrinsicoid deflection time (ms)	71.50 ± 11.59
Max deflection index (%)	0.53 ± 0.09
Maximum QRS duration	140.49 ± 10.16
Pseudo-delta n (%)	11/71 (15.5%)
PVC burden in 24-hour	21 (10-34) %
Holter Monitoring	
Cardiovascular drugs, n (%)	
Calcium channel blocker use	29 (40%)
Beta blocker use	31 (43.70%)
Amiodarone use	15 (21.10%)
Propafenone use	21 (29.60%)
Any antiarrhythmic*	59 (83.10%)
Anti-arrhythmic medication per patient	1.35 ± 0.86
TSH levels	1.42 (0.42-3.65)
Potassium levels	4.45 (3.25-5.55)
Calcium levels	9.68 (9-10.30)

Data presented as mean (± St. Deviation), median (minimum: maximum) and n (%). *Calcium channel blocker, beta blocker, amiodarone or propafenone.

Table II. Procedural characteristics of the study patients.

Catheter ablation techniques	
Carto	48/71 (67,6%)
Ensite Precision/Nav X	23/71 (32,4%)
Ablation site	
RVOT	36/71 (50,7%)
Coronary cusp	20/71 (28,2%)
Papillary muscle	2/71 (2,8%)
Left ventricular summit	8/71 (11,3%)
Multiple	4/71 (5,6%)
Other	1 (1,4%)
Procedural parameters	
Radiofrequency time, min	9.9 ± 1.4
Total procedure time, min	137.9 ± 38.2
Fluoroscopy time, min	20.2 ± 12.7
Cardiac tamponade	1/71 (1,4%)
Cerebrovascular events	1/71 (1,4%)
Hematoma	4/71 (5,6%)

RVOT,right ventricular outflow tract.

majority of patients was the outflow tract region. The procedural characteristics of the patients are shown in Table II.

The study group showed improvement in left ventricular diastolic function after ablation in mitral E velocity (before ablation 88 cm/sec (63:142), 3rd month 92 cm/sec (67:124), 18th month 94 cm/sec (65:135), p <0.001), E/A ratio (before ablation 15 (0.59:1.75), 3rd month 1.10 (0.86:1.33), 18th month 1.13 (0.86:1.28), p <0.001), DT duration (before ablation 191 msec (145:275), 3rd month 182 msec (159:231), 18th month 182 msec (149:228), p = 0.023), Ea average (before ablation 81.50 cm/sec (66.50:116.50), 3rd month 84.50 cm/sec (59.50:105.50), 18th month 84.50 cm/sec (68.50:113), p=0.006) and E/E ratio (before ablation 1.07 (0.74:1.27), 3rd month 1.10 (0.86:1.33), 18th month 1.13. It was observed that the improvements in mitral E velocity, E/A and E/Ea ratios persisted at the 18-month follow-up examination.

When patients with PVC-CMP and normal LVEF were compared, it was observed that mitral A velocity was higher in the CMP group (p <0.001). The mitral E velocity in both groups increased after ablation in the 3rd and 18th months. There was a difference among the preablation, 3rd month and 18th month periods in terms of E/A levels in the normal EF group. In the pairwise comparisons of the time points, the E/A ratio and E/Ea ratio were still improving in the normal EF group. In the PVC-CMP group, the diastolic parameters recovered over time (Table III).

In Table IV, LV systolic function and LA functions are evaluated comparatively. There was a significant progressive improvement in LVEF. Over time, an improvement was observed in the LA volume index. With the improvement of LV diastolic function, the contribution of the LA to atrial systole gradually decreased. There was an increase in the passive emptying fraction from the LA to the LV due to the improvement in end-diastolic pressure in the LA. It was observed that the improvement in the LA continued to the 18th month (Figure 1).

When the PVC-CMP subgroup of 19 patients was examined, it was observed that the LVEF continued to improve until the 18th month. A decrease in LA volume index was observed, in addition to an increase in the LA passive emptying fraction. As a result, improvement was observed in the volume and function of the LA in the PVC-CMP group (Table V).

Discussion

The major findings of the present study were as follows. (1) The RFCA of idiopathic PVCs in patients with compromised LV function was associated with improvement in LV systolic and diastolic function when examined at the 3rd and 18th months after ablation. (2) Parallel to the amelioration in LV systolic and diastolic functions, significant improvement was observed in LA dynamic function, as determined by LA volume values. (3) The improvement in systolic function continued in all groups at the end of the 18th month. (4) The improvement in diastolic functions proceeded to the long term in the group without cardiomyopathy. (5) The marked improvement in LA systolic function continued in the long term.

Previous studies have indicated that several factors are associated with PVC-CMP: a high PVC burden, the presence of interpolated PVCs, the presence or absence of symptoms, wide QRS PVCs, a higher coupling interval and an epicardial PVC origin[5,11-13]. PVCs can also cause changes in left atrial function due to abnormal hemodynamic effects on the LV, systolic or diastolic, for many underlying reasons. Moreover, it was observed that PVCs increase the risk of AF, possibly through cardiac remodeling and increased LA size[14,15]. In addition, there is no clear consensus regarding the role of LA function in PVC management and changes in LA functions

Table III. Conventional Doppler echocardiography diastolic function indices with and without the premature ventricular complex induced cardiomyopathy.

	PVC-CMP (n = 19)				Normal LVEF (n = 52)			
	Before ablation	3rd month	18th month	p	Before ablation	3rd month	18th month	
Mitral E velocity (cm/sec)	87.95 (± 20.62)	88.63 (± 11.48)	90.58 (± 12.42)	**0.012**[b]	88 (65:130)	94.50 (70:124)	96 (72:135)	*
Mitral A velocity (cm/sec)	82 (65:110)	74 (61:105)	73 (61:110)	**0.049**[a]	72.50 (53:110)	77.50 (40:100)	75 (53:99)	*
E/A ratio	1.04 (± 0.25)	1.15 (± 0.24)	1.19 (±0.25)	**0.024**[b]	1.20 (0.62:1.75)	1.23 (0.75:1.98)	1.37 (0.75:1.78)	*
DT (msec)	202.21 (± 26.43)	195.21 (± 16.09)	192.74 (± 15.70)	**0.023**[b]	187 (146:246)	181 (159:210)	180 (149:210)	*
Ea average (cm/sec)	86.58 (± 11.58)	83.66 (±7.85)	84.21 (±8.60)	0.297[b]	79 (66.50:110)	85 (59.50:105.50)	86.25 (68.50:113)	*
E/Ea ratio	1.01 (± 0.12)	1.06 (± 0.10)	1.08 (± 0.11)	**0.018**[b]	1.09 (0.76:1.27)	1.10 (0.86:1.33)	1.16 (0.86:1.28)	*
Pairwise comparisons (p*)								
	Before ablation & 3rd month	Before ablation & 18th month	3rd month & 18th month		Before ablation & 3rd month	Before ablation & 18th month	3rd month & 18th month	
Mitral E velocity (cm/sec)	> 0.99	> 0.99	0.007		0.016	**< 0.001**	**0.082**	
Mitral A velocity (cm/sec)	0.186	0.128	> 0.99		NA	NA	NA	
E/A ratio	0.398	0.130	0.074		0.150	**< 0.001**	0.002	
DT (msec)	0.458	0.166	0.066		0.258	**0.049**	> 0.99	
Ea average (cm/sec)	NA	NA	NA		**0.037**	**0.004**	> 0.99	
E/Ea ratio	0.298	0.106	0.064		0.209	**< 0.001**	**0.001**	

Data presented as median (minimum: maximum) and mean (± st. deviation). [a]Friedman Two-Way Analysis of Variance Test; [b]ANOVA Test for Repeated Measurements. *p**: Adjusted *p*-value with Bonferroni correction for pairwise comparisons.

in the long term after successful RFCA. This study was mainly conducted to answer these questions.

In the present study, in patients with or without apparent cardiomyopathy, RFCA was shown to successfully eliminate PVCs and improve LA function in the long term. Kim et al[16] demonstrated an improvement in the LA volume index at approximately one-year of follow-up after PVC ablation. In accordance with our study, the change in LA volume continued until the 18-month follow-up. However, most of the improvements in the LA total volume index and LA total volume fraction were complete in the first 3 months, and the improvement in the LA passive emptying fraction occurred in the later period. Longer follow-up studies using imaging modalities, perhaps other than echocardiography, are needed to understand when remodeling in the LA volume is complete.

Akkaya et al[8] also showed improvement in the LA volume index over a 6±2-month follow-up period after RFCA of PVCs. Compared with the present study, baseline LA volume indices were higher in the patient population in their study. In their study population, the baseline LA volume was >40 ml/m^2. It is known that LA remodeling is reversible[17], highlighting the importance of PVC treatment, which, as the present study suggests, can contribute to reversing LA remodeling in different LA diameters.

LA enlargement is usually a secondary change to LV diastolic dysfunction[16-18]. LV diastolic function is usually coupled with LV systolic performance, and improvement in systolic performance decreases left ventricle filling pressures, which could improve diastolic function[19,20]. In the present study, in which diastolic dysfunction parameters were evaluated in detail, a significant improvement was observed in these parameters

Table IV. Changes in systolic, diastolic, and left atrial functions following radiofrequency catheter ablation.

	Before ablation	3rd month	18th month	*p*-value
Left ventricular ejection fraction	53.01 (33.33:64.71)	54.55 (37.69:65.98)	58.02 (42.19:66)	**< 0.001**[a]
Left atrium end-systolic antero-posterior diameter (mm)	33 (24:41)	31 (22:39)	30 (21:38)	**< 0.001**[a]
Left atrium volume index (mL/m^2)	18.15 (± 2.89)	17.11 (± 2.71)	16.67 (± 2.61)	**< 0.001**[b]
Left atrium total emptying volume (mL/m^2)	11.32 (± 2.11)	10.70 (± 2.33)	10.89 (± 2.08)	**< 0.001**[b]
left atrium active emptying volume (mL/m^2)	5.39 (8.423:6.42)	4.67 (0.74:9.87)	4.08 (1.11:6.43)	**< 0.001**[a]
Left atrium passive emptying fraction (PASSİVE EF)	33.33 (23.68:54.10)	37.11 (6.45:50)	40.91 (33.33:55.77)	**< 0.001**[a]
Left atrium total volume fraction (TOTALEF)	62.50 (47.37:71.43)	62.50 (50:73.53)	64.71 (53.13:73.33)	**< 0.001**[a]
	Before ablation & 3rd month	**Pairwise comparisons (*p**) before ablation & 18th month**		**3rd month & 18th month**
Left ventricular ejection fraction	**0.009**	**< 0.001**		**< 0.001**
Left atrium end-systolic antero-posterior diameter (mm)	**0.001**	**< 0.001**		**< 0.001**
Left atrium volume index (mL/m^2)	**< 0.001**	**< 0.001**		**< 0.001**
Left atrium total emptying volume (mL/m^2)	**0.001**	**0.001**		0.211
left atrial active emptying volume (mL/m^2)	**< 0.001**	**< 0.001**		**0.005**
Left atrium passive emptying fraction (PASSİVE EF)	**< 0.001**	**< 0.001**		**< 0.001**
Left atrium total volume fraction (TOTALEF)	> 0.99	**< 0.001**		**< 0.001**

Data presented as median (minimum: maximum) and mean (± st. deviation). [a]Friedman Two-Way Analysis of Variance Test; [b]ANOVA Test for Repeated Measurements. *p**: Adjusted *p*-value with Bonferroni correction for pairwise comparisons.

Table V. Changes in systolic, diastolic, and left atrial functions following radiofrequency catheter ablation.

	Before ablation	3rd month	18th month	*p*-value
Left ventricular ejection fraction	43.83 (± 6.59)	45.74 (±6.55)	51.77 (± 6.28)	**< 0.001**[b]
Left atrium end-systolic antero-posterior diameter (mm)	37 (26:40)	34 (24:39)	32 (24:37.50)	**< 0.001**[a]
Left atrium volume index (mL/m^2)	20.02 (±3.21)	18.79 (± 2.99)	18.16 (± 2.90)	**< 0.001**[b]
Left atrium total emptying volume (mL/m^2)	12.42 (± 2.35)	11.96 (± 2.54)	11.86 (± 2.42)	0.056[b]
Left atrial active emptying volume (mL/m^2)	5.97 (± 1.23)	4.98 (± 1.66)	4.26 (± 1.14)	**< 0.001**[b]
Left atrium passive emptying fraction (PASSİVE EF)	32.50 (26.32:37.14)	38.71 (6.45:47.06)	40.63 (33.33:50)	**< 0.001**[a]
Left atrium total volume fraction (TOTALEF)	61.95 (± 5.11)	63.29 (± 5.59)	64.99 (± 4.72)	**0.002**[b]
	Before ablation & 3rd month	**Pairwise comparisons (*p**) before ablation & 18th month**		**3rd month & 18th month**
Left ventricular ejection fraction	0.105	**< 0.001**		**< 0.001**
Left atrium end-systolic antero-posterior diameter (mm)	**0.022**	**< 0.001**		**0.045**
Left atrium volume index (mL/m^2)	**0.004**	**< 0.001**		**0.007**
Left atrium total emptying volume (mL/m^2)	NA	NA		NA
left atrial active emptying volume (mL/m^2)	**0.016**	**< 0.001**		0.103
Left atrium passive emptying fraction (PASSİVE EF)	**0.011**	**< 0.001**		**0.045**
Left atrium totalvolume fraction (TOTALEF)	0.954	**0.012**		0.092

Data presented as median (minimum: maximum) and mean (± st. deviation). [a]Friedman Two-Way Analysis of Variance Test; [b]ANOVA Test for Repeated Measurements. *p**: Adjusted *p*-value with Bonferroni correction for pairwise comparisons.

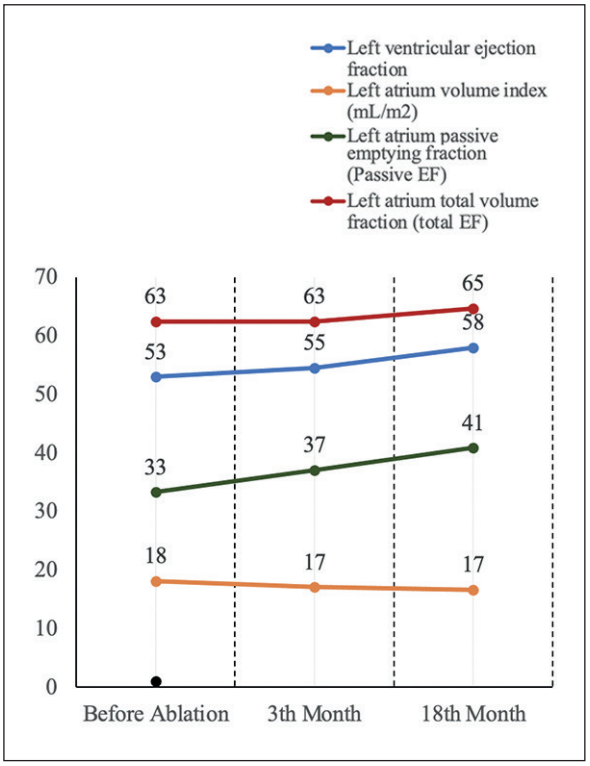

Figure 1. Left ventricular ejection fraction and left atrium functions over time.

as determined by the measurements conducted at the 3rd and 18th months after ablation. As a remarkable finding of the present study, improvement in diastolic functions was more prominent in the non-CMP group, and improvement in diastolic function parameters significantly increased in the long term.

This study had some limitations that are worth noting. The first limitation was that left atrium size and dynamic functions were assessed by echocardiography, and no other imaging modalities, such as cardiac magnetic resonance, were used to reveal the functions. Second, the difference in absolute values was relatively small; therefore, the clinical relevance might seem limited. However, the data for this study could set the stage for larger studies.

Conclusions

The present study showed that, apart from the change in LV volume and LV systolic function, the change in LA volume also persisted in the long term. The current study also emphasized the need for clinically enhanced studies to determine the direct impact of the reversing of LA remodeling on cardiovascular outcomes.

Conflict of Interest

The Authors declare that they have no conflict of interests.

Funding

This study received no financial assistance.

Authors' Contribution

Concept – A.T., S.K.; Design – A.T., S.K.; Supervision – A.T., S.K.; Funding – A.T., S.K.; Materials – A.T., S.K.; Data Collection and/or processing – A.T., S.K.; Analysis and/or interpretation – A.T., S.K.; Literature search – A.T., S.K.; Writing – A.T., S.K.; A.T., S.K.; Critical review – A.T., S.K.

ORCID ID

Ahmet Tutuncu: https://orcid.org/ 0000-0003-3747-9580.

References

1) Sirichand S, Killu AM, Padmanabhan D, Hodge DO, Chamberlain AM, Brady PA, Kapa S, Noseworthy PA, Packer DL, Munger TM, Gersh BJ, McLeod CJ, Shen WK, Cha YM, Asirvatham SJ, Friedman PA, Mulpuru SK. Incidence of idiopathic ventricular arrhythmias: a population-based study. Circ Arrhythm Electrophysiol 2017; 10: e004662.
2) Panizo JG, Barra S, Mellor G, Heck P, Agarwal S. Premature ventricular complex-induced cardiomyopathy. Arrhythm Electrophysiol Rev 2018; 7: 128.
3) Gorenek B, Fisher JD, Kudaiberdieva G, Baranchuk A, Burri H, Campbell KB, Chung MK, Enriquez A, Heidbuchel H, Kutyifa V, Krishnan K, Leclercq C, Ozcan EE, Patton KK, Shen W, Tisdale JE, Turagam MK, Lakkireddy D. Premature ventricular complexes: diagnostic and therapeutic considerations in clinical practice: A state-of-the-art review by the American College of Cardiology Electrophysiology Council. J Interv Card Electrophysiol 2020; 57: 5-26.
4) Priori SG, Lundqvist CB, Mazzanti A, Blom N, Borggrefe M, Camm J, Elliott PM, Fitzsimons D, Hatala R, Hindricks G, Kirchhof P, Kjeldsen K, Kuck KH, Madrid AH, Nikolaou N, Norekvål TM, Spaulding C, Veldhuisen DJV. 2015 ESC Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: The Task Force for the Management of Patients with Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death of the European Society of Cardiology (ESC) Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC). Europace 2015; 17: 1601-1687.

5) Baman TS, Lange DC, Ilg KJ, Gupta SK, Liu TY, Alguire C, Armstrong W, Good E, Chugh A, Jongnarangsin K, Pelosi Jr, Crawford FT, Ebinger M, Oral H, Morady F, Bogun F Relationship between burden of premature ventricular complexes and left ventricular function. Heart Rhythm 2010; 7: 865-869.

6) Park Y, CKim S, Shin J, Oh AR, Shin EJ, Lee JH, Ahn T, Cha JY, Moon J. Frequent premature ventricular complex is associated with left atrial enlargement in patients with normal left ventricular ejection fraction. Pacing Clin Electrophysiol 2014; 37: 1455-1461.

7) Hoit BD. Left atrial size and function: role in prognosis. J Am Coll Cardiol 2014; 63: 493-505.

8) Akkaya M, Roukoz H, Adabag S, Benditt DG, Anand I, Li JM, Zakharova M, Tholakanahalli V. Improvement of left ventricular diastolic function and left atrial reverse remodeling after catheter ablation of premature ventricular complexes. J Interv Card Electrophysiol 2013; 38: 179-85.

9) Kanat S, Mutluer FO, Tütüncü A, Duran Karaduman B, Bozkaya VO, Keskin M, Uslu A, Çay S, Tenekecioglu E. Left Atrial Function Is Improved in Short-Term Follow-Up after Catheter Ablation of Outflow Tract Premature Ventricular Complexes. Medicina (Kaunas) 2019; 55: 241.

10) Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L, Flachskampf FA, Foster E, Goldstein SA, Kuznetsova T, Lancellotti P, Muraru D, Picard MH, Rietzschel ER, Rudski L, Spencer KT, Tsang W, Voigt JU. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging 2015; 16: 233-271.

11) Yokokawa M, Kim HM, Good E, Chugh A, Pelosi Jr F, Alguire C, Armstrong W, Crawford T, Jongnarangsin K, Oral H, Morady F, Bogun F. Relation of symptoms and symptom duration to premature ventricular complex–induced cardiomyopathy. Heart Rhythm 2012; 9: 92-95.

12) Yokokawa M, Kim HM, Good E, Crawford T, Chugh A, Pelosi Jr F, Jongnarangsin K, Latchamsetty R, Armstrong W, Alguire C, Oral H, Morady F, Bogun F. Impact of QRS duration of frequent premature ventricular complexes on the development of cardiomyopathy. Heart rhythm 2012; 9: 1460-1464.

13) Olgun H, Yokokawa M, Baman T, Kim HM, Armstrong W, Good E, Chugh A, Pelosi Jr, Crawford FT, Oral H, Morady F, Bogun F. The role of interpolation in PVC-induced cardiomyopathy. Heart rhythm 2011; 8: 1046-1049.

14) Barnes ME, Miyasaka Y, Seward JB, Gersh BJ, Rosales AG, Bailey KR, Petty GW, Wiebers DO, Tsang TSM. Left atrial volume in the prediction of first ischemic stroke in an elderly cohort without atrial fibrillation. Mayo Clin Proc 2004; 79: 1008-1014.

15) Blaye-Felice MS, Hamon D, Sacher F, Pascale P, Rollin A, Duparc A, Mondoly P, Derval N, Denis A, Cardin C, Hocini M, Jaïs P, Schlaepfer J, Bongard V, Carrié D, Galinier M, Pruvot E, Lellouche N, Haïssaguerre M, Maury P. Premature ventricular contraction-induced cardiomyopathy: Related clinical and electrophysiologic parameters. Heart Rhythm 2016; 13: 103-110.

16) Kim YH, Park SM, Lim HE, Pak H, Kim YH, Shim WJ. Chronic frequent premature ventricular complexes originating from right and non-right ventricular outflow tracts. Int Heart J 2010; 51: 388-393.

17) Casaclang-Verzosa G, Gersh BJ, Tsang TS. Structural and functional remodeling of the left atrium: clinical and therapeutic implications for atrial fibrillation. J Am Coll Cardiol 2008; 51: 1-11.

18) Kanei Y, Friedman M, Ogawa N, Hanon S, Lam P, Schweitzer P. Frequent premature ventricular complexes originating from the right ventricular outflow tract are associated with left ventricular dysfunction. Ann Noninvasive Electrocardiol 2008; 13: 81-85.

19) Eichhorn EJ, Willard JE, Alvarez L, Kim AS, Glamann DB, Risser RC, Grayburn PA. Are contraction and relaxation coupled in patients with and without congestive heart failure? Circulation 1992; 85: 2132-2139.

20) Karakuş A. Uğuz B. An early echocardiographic prediction for functional myocardial recovery after ST elevation myocardial infarction. Kardiologiia 2021; 61: 66-71.