Recovery from left ventricular dysfunction after ablation of frequent premature ventricular complexes

Miki Yokokawa, MD, Eric Good, DO, Thomas Crawford, MD, Aman Chugh, MD, Frank Pelosi Jr, MD, Rakesh Latchamsetty, MD, Krit Jongnarangsin, MD, William Armstrong, MD, Hamid Ghanbari, MD, Hakan Oral, MD, Fred Morady, MD, Frank Bogun, MD[1]

From the Division of Cardiovascular Medicine, University of Michigan, Ann Arbor, Michigan.

BACKGROUND Patients with frequent premature ventricular complexes (PVCs) and PVC-induced cardiomyopathy usually have recovery of left ventricular (LV) dysfunction postablation. The time course of recovery of LV function has not been described.

OBJECTIVE To describe the time course and predictors of recovery from LV dysfunction after effective ablation of PVCs in patients with PVC-induced cardiomyopathy.

METHODS In a consecutive series of 264 patients with frequent idiopathic PVCs referred for PVC ablation, LV dysfunction was present in 87 patients (mean ejection fraction 40% ± 10%). The PVC burden was reduced to <20% of the initial PVC burden in 75 patients. In these patients, echocardiography was repeated 3–4 months postablation. If LV function did not normalize after 3–4 months, a repeat echocardiogram was performed every 3 months until there was normalization or stabilization of LV function.

RESULTS The ejection fraction normalized at a mean of 5 ± 6 months postablation. The majority of patients (51 of 75, 68%) with PVC-induced LV dysfunction had a recovery of LV function within 4 months. In 24 (32%) patients, recovery of LV function took more than 4 months (mean 12 ± 9 months; range 5–45 months). An epicardial origin of PVCs was more often present (13 of 24, 54%) in patients with delayed recovery of LV function than in patients with early recovery of LV function (2 of 51, 4%; $P < .0001$). The PVC-QRS width was significantly longer in patients with delayed recovery than in patients with recovery within 4 months (170 ± 21 ms vs 159 ± 16 ms; $P = .02$). In multivariate analysis, only an epicardial PVC origin was predictive of delayed recovery of LV function in patients with PVC-induced cardiomyopathy.

CONCLUSIONS PVC-induced cardiomyopathy resolves within 4 months of successful ablation in most patients. In about one-third of the patients, recovery is delayed and can take up to 45 months. An epicardial origin predicts delayed recovery of LV function.

KEYWORDS Premature ventricular complexes; Left ventricular dysfunction; Catheter ablation

ABBREVIATIONS LV = left ventricular; **MRI** = magnetic resonance imaging; **PVC** = premature ventricular complex

(Heart Rhythm 2013;10:172–175)

Introduction

Frequent premature ventricular complexes (PVCs) can cause a reversible form of cardiomyopathy.[1,2] The time course and the determinants of recovery of left ventricular (LV) function are not known. The purpose of this study was to describe the time course and predictors of recovery from LV dysfunction after an effective ablation of PVCs in patients with PVC-induced cardiomyopathy.

Methods

Patient characteristics

In a consecutive series of 264 patients (121 men; age 48 ± 14 years) with frequent idiopathic PVCs, LV dysfunction was present in 87 patients. PVC-induced cardiomyopathy was defined as an ejection fraction of <50% that normalized after ablation or an ejection fraction of 50%–55% with an improvement of ≥10% after PVC ablation. All patients underwent an assessment for the presence of structural heart disease with echocardiography, stress testing, and/or cardiac magnetic resonance imaging (MRI). Patients who had delayed enhancement on the MRI (n = 13) were excluded. Patients with a history of congestive heart failure preceding the presence of frequent PVCs were excluded. Lastly, patients with structural heart disease including coronary artery disease, myocarditis, infiltrative heart disease, valvular heart disease, and hypertensive heart disease were excluded (Table 1).

The mean LV ejection fraction prior to ablation was 40% ± 10%. The PVC burden was reduced to <20% of the initial PVC burden in 75 patients.

LV end-diastolic diameter was assessed, and a diameter of ≥57 mm was defined as LV dilatation. Before ablation,

Address reprint requests and correspondence: Frank Bogun, MD, Division of Cardiovascular Medicine, Cardiovascular Center, University of Michigan, SPC 5853, 1500 East Medical Center Dr, Ann Arbor, MI 48109-5853 . E-mail address: fbogun@med.umich.edu.

[1]Dr Bogun has received a grant from the Leducq Foundation.

1547-5271/$-see front matter http://dx.doi.org/10.1016/j.hrthm.2012.10.011

Table 1 Patient characteristics with PVC-induced cardiomyopathy

Patients	75
Age (y)	50 ± 16
Sex: Man	51 (68)
LV ejection fraction (%)	39 ± 10
LV end-diastolic diameter (mm)	56 ± 6
PVC burden (%)	26 ± 11
Therapy	
Beta-blockers	44 (59)
Calcium channel blockers	9 (12)
Angiotensin-converting enzyme inhibitors/ angiotensin receptor blockers	28 (37)
Antiarrhythmic drug therapy including amiodarone	5 (7)

Note: Data are shown as mean ± 1 standard deviation and as number (percentage) values.

LV = left ventricular; PVC = premature ventricular complex.

LV enlargement (LV end-diastolic diameter ≥57 mm) was present in 37 of 75 (49%) patients.

Holter recordings

The PVC burden was determined with a 24-hour 12-lead Holter monitor prior to the procedure. The PVC burden was reassessed 3–4 months postablation. Only patients with PVC-induced cardiomyopathy who had a successful ablation procedure were included in this study. A successful ablation procedure was defined as a reduction in the PVC burden of more than 80% of the initial PVC burden.

Echocardiography

LV ejection fraction and LV dimensions were measured with the Simpson's formula prior to the ablation procedure. These parameters were measured during sinus rhythm using at least 2 consecutive sinus beats, thereby avoiding post-extrasystolic potentiation. The measurements were repeated 3–4 months postablation. If LV function did not normalize after 3–4 months, a repeat echocardiogram was performed every 3 months until there was normalization or stabilization of LV function. The time to normalization of LV function was assessed. Normalization was defined as an ejection fraction of ≥55%.

If normalization of LV function was found in the follow-up echocardiogram 3–4 months postablation, this was defined as an early normalization of LV function. If the time interval required for normalization of LV function was >3–4 months, this was defined as a delayed recovery of LV function.

Mapping and ablation

PVCs were mapped and ablated as previously described.[2]

Follow-up

Patients were seen 3–6 months post-procedure and 12–48 months thereafter. Subsequently, patients were seen on an as-needed basis. In patients taking an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker or a beta-blocker, these medications were continued after ablation and discontinued after normalization of LV function and LV dimensions. No new medications were started after an effective ablation.

Statistical analysis

Continuous variables were expressed as mean ± 1 standard deviation and were compared by using the Student t test. Categorical variables were compared by using the χ^2 test. If the sample size was smaller than 5 in a given cell, the Fisher exact test was used. Parameters that were associated with delayed recovery with $P < .1$ were entered into multiple logistic regression analysis with delayed recovery as the dependent variable to assess whether they were independently associated with delayed recovery. A P value of $<.05$ was considered statistically significant.

Results

Recovery of LV function

A total of 75 patients with PVC-induced cardiomyopathy had an effective ablation procedure. The PVC burden was reduced from 26% ± 11% to 2% ± 4% ($P < .0001$). LV function changed from 39% ± 10% to 59% ± 4% ($P < .0001$) in patients who had PVC-induced cardiomyopathy and had an effective ablation procedure. The ejection fraction improved or normalized in all patients with PVC-induced cardiomyopathy. Twenty-four of 75 (32%) patients had delayed recovery of LV function. In the remaining patients, LV function recovered within 4 months (Table 2).

In patients with delayed recovery, the ejection fraction improved or normalized after a mean of 12 ± 9 months (range 5–45 months). In 1 patient, the ejection fraction had improved from 20% to 47% at the latest follow-up. In 6 of 24 (25%) patients after the follow-up period, the ejection fraction improved to 50%–55% at the last follow-up echocardiogram. By 4 months, even in patients with delayed normalization of LV function, the ejection fraction had improved by at least 10% in 10 of 24 (42%) patients. In patients with delayed recovery of LV function, at the 4-month follow-up echocardiogram the ejection fraction had improved from 39% ± 10% to 47% ± 8% ($P < .001$). The ejection fraction in patients with delayed recovery improved to 58% ± 6% at the end of follow-up.

After the ablation procedure, LV enlargement persisted in 16 patients (20%; $P < .001$) at the initial follow-up echocardiogram. The mean LV end-diastolic diameter changed from 56 ± 6 to 53 ± 5 mm after ablation ($P < .001$). In 5 of 24 (21%) patients with delayed recovery of LV function, there was still LV dilatation. This was comparable to the prevalence of LV dilatation (11 of 51, 22%) among patients in whom LV function normalized within 4 months of postablation ($P = .94$). At 6-month follow-up, LV function had normalized in 17 of 24 patients with delayed recovery of LV function and LV dimensions were still dilatated in 4 patients compared with 6 patients with recovery within 4 months ($P = .72$). In 2 patients with delayed recovery, the ejection function normalized prior to normalization