

Reversal of outflow tract ventricular premature depolarization–induced cardiomyopathy with ablation: Effect of residual arrhythmia burden and preexisting cardiomyopathy on outcome

Stavros E. Mountantonakis, MD, David S. Frankel, MD, Edward P. Gerstenfeld, MD, Sanjay Dixit, MD, FHRS, David Lin, MD, Mathew D. Hutchinson, MD, FHRS, Michael Riley, MD, PhD, Rupa Bala, MD, Joshua Cooper, MD, David Callans, MD, FHRS, Fermin Garcia, MD, Erica S. Zado, PA-C, FHRS, Francis E. Marchlinski, MD, FHRS

From the Electrophysiology Section, Cardiovascular Division, Department of Medicine, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

BACKGROUND Outflow tract ventricular premature depolarizations (VPDs) can be associated with reversible left ventricular cardiomyopathy (LVCM). Limited data exist regarding the outcome after ablation of outflow tract VPDs from the LV and the impact of residual VPDs or preexisting LVCM prior to the diagnosis of VPDs on recovery of LV function.

OBJECTIVE To examine the safety, efficacy, and long-term effect of radiofrequency ablation on LV function in patients with LVCM and frequent outflow tract VPDs and examine the effect of ablation in patients with LVCM known to precede the onset of VPDs and the impact of residual VPD frequency on recovery of LV function.

METHODS Sixty-nine patients (43 men; age 51 ± 16 years) with nonischemic LVCM (left ventricular ejection fraction [LVEF] $35\% \pm 9\%$, left ventricular diastolic diameter [LVDD] 5.8 ± 0.7 cm) were referred for ablation of frequent outflow tract VPDs ($29\% \pm 13\%$).

RESULTS VPDs originated in the right ventricular outflow tract in 27 (39%) patients and the left ventricular outflow tract in 42 (61%) patients. After follow-up of 11 ± 6 months, 44 (66%) patients had rare ($<2\%$) VPDs, 15 (22%) had decreased VPD burden ($>80\%$ reduction and always <5000 VPDs), and 8 (12%) had no clinical improvement with persistent (5 patients) or recurrent (3 patients) VPDs. Only patients with either rare or decreased VPD burden had a significant improvement in LVEF (Δ LVEF $14\% \pm 9\%$ vs $13\% \pm 7\%$ vs $-3\% \pm 6\%$, respectively, $P < .001$) and LVDD

(Δ LVDD -4 ± 5 vs -2 ± 4 vs 0 ± 4 , respectively, $P = .038$), regardless of chamber of origin. The magnitude of LVEF improvement correlated with the decline in residual VPD burden ($r = 0.475$, $P = .007$). Patients with preexisting LVCM had a more modest but still significant improvement in LV function compared to patients without preexisting LVCM (Δ LVEF 8% vs 13% , $P = .046$). Multivariate analysis revealed ablation outcome, higher LVEF, and absence of preexisting LVCM were independently associated with LVEF improvement.

CONCLUSION Frequent outflow tract VPDs are associated with LVCM regardless of ventricle of origin. Significant ($>80\%$) reduction in VPD burden has comparable improvement in LV function to complete VPD elimination. Successful VPD ablation may be beneficial even in patients with preexisting LVCM.

KEYWORDS Cardiomyopathy; Catheter ablation; Heart failure; Outflow tract; Palpitations; Ventricular premature depolarization

ABBREVIATIONS LV = left ventricular; LVCM = left ventricular cardiomyopathy; LVDD = left ventricular diastolic diameter; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; RVOT = right ventricular outflow tract; VPD = ventricular premature depolarization

(Heart Rhythm 2011;8:1608–1614) © 2011 Heart Rhythm Society. All rights reserved.

Introduction

Ventricular premature depolarizations (VPDs) originating from the right ventricular outflow tract (RVOT) and left ventricular outflow tract (LVOT) are frequently encountered in clinical practice. Although they occur mostly in patients without structural heart disease, they

have been described in the setting of left ventricular cardiomyopathy (LVCM). A few small series have shown that successful ablation of VPDs originating from the RVOT can result in resolution of LVCM, suggesting causality between frequent VPDs and the development of LVCM.^{1–3} Despite these seminal observations, limited data exist regarding an association between VPDs from the LVOT and LVCM as well as outcome with ablation in those patients. The impact of residual VPDs or preexisting LVCM on the recovery of LV function after VPD ablation is also unknown.^{3,4}

Address reprint requests and correspondence: Dr. Francis E. Marchlinski, Hospital of the University of Pennsylvania, 9 Founders Pavilion-Cardiology, 3400 Spruce Street, Philadelphia, Pennsylvania 19104. E-mail address: francis.marchlinski@uphs.upenn.edu. (Received April 4, 2011; accepted April 22, 2011.)

The purpose of this study was to examine the safety, efficacy, and long-term effect on LV function of radiofrequency ablation in a relatively large patient population with LVCM and frequent VPDs originating from the RVOT and LVOT. In addition, we sought to examine the long-term effect of ablation in patients with LVCM known to precede the onset of VPDs and the impact of residual VPD frequency on recovery of LV function.

Methods

Inclusion criteria

We retrospectively analyzed 69 consecutive patients with frequent VPDs (>5,000 VPDs per 24 hours) and LVCM, defined as left ventricular ejection fraction (LVEF) <50% referred to our institution for catheter ablation. The predominant VPDs were required to have ECG characteristics suggestive of outflow tract origin (right or left bundle branch morphology, inferior axis, negative in lead aVL). Active ischemia or prior infarction as a cause of cardiomyopathy was ruled out in all cases by history, electrocardiogram, and either coronary angiography or stress testing.

Assessment of VPD burden and LV function preprocedure

Twenty-four-hour Holter monitoring was performed at baseline to quantify the VPD burden. All patients underwent echocardiography prior to ablation to assess LV systolic function and diameter. LVEF was calculated using the Simpson formula. To avoid the limitations of LVEF calculation in the setting of ventricular arrhythmias, only patients with a reduced LVEF by echocardiogram the day after the procedure were included in the study.

Electrophysiologic procedure and ablation

Patients were brought to the electrophysiology laboratory in the postabsorptive, nonsedated state after written informed consent was obtained in accordance with the University of Pennsylvania Health System's institutional guidelines. Surface ECG leads were placed in the standard positions. Bipolar electrograms were recorded with a bandpass filter at 30 to 500 Hz using a 4-mm-tip ablation catheter (NaviStar, Biosense Webster, Diamond Bar, CA, USA). A 6Fr quadripolar catheter was placed in the right ventricular apex for pacing. An 8Fr intracardiac echocardiographic catheter (AcuNav, Siemens Medical, Mountain View, CA, USA) was inserted via the left femoral vein and advanced to the base of the RVOT to obtain short-axis images of the aortic cusp region. Intracardiac echocardiographic imaging was used to confirm catheter location, assess the distance from the coronary vasculature, monitor lesion formation, and assess for complications.

Detailed activation and pacemapping were performed first in the RVOT, and sinus rhythm electrograms were analyzed. If a diffuse activation pattern was noted on the three-dimensional electroanatomic map (CARTO, Biosense Webster, or NavX, St. Jude Medical, St. Paul, Minnesota) and/or pacemapping resulted in a poor match with right

ventricular mapping, attention was turned to the LVOT region. Intravenous heparin was administered to maintain an activated clotting time >250 seconds during aortic cusp and/or LV endocardial mapping. The ablation catheter was inserted in the right femoral artery and advanced to the aortic cusp region in retrograde fashion. If activation times in the aortic cusp region were not sufficiently early with respect to QRS onset, then additional mapping was performed in the great cardiac vein, anterior intraventricular vein, and/or LV epicardium using the pericardial access technique described by Sosa et al.⁵

Radiofrequency energy was delivered with a conventional 4-mm-tip ablation catheter in temperature-controlled mode with a target temperature of 55°C at a power of 50 W. If power delivery was inadequate, an irrigated ablation catheter was used instead, with power 30 W and maximum temperature 45°C. If VPD termination occurred in the first 10 seconds, delivery of radiofrequency energy typically was continued for a total of 60 seconds. If termination did not occur in the first 10 seconds, delivery of radiofrequency energy was discontinued and the catheter repositioned. Power using the irrigated catheter was increased to 50 W in select cases if VPD suppression and recurrence occurred. Burst atrial and ventricular pacing before and during isoproterenol infusion (up to a rate of 20 µg/min) was repeated following ablation.

Acute ablation endpoints

Acute complete success was defined as the absence of spontaneous or inducible VPDs with isoproterenol infusion and burst pacing from the right ventricular apex for 30 minutes following the last radiofrequency lesion. Partial success was defined as a greater than 80% reduction in VPD frequency or persistence of infrequent nondominant VPDs of other QRS morphology that were not targeted. Finally, failure was defined as less than 80% reduction in VPD frequency.

Follow-up

All patients underwent 24-hour full disclosure telemetry monitoring immediately following the procedure. Transthoracic echocardiography was performed the day after the procedure to assess for structural changes and to confirm the presence of reduced LVEF in the absence of VPDs. The majority of patients were followed in our Arrhythmia Center with routine ECGs at 6 weeks and follow-up echocardiograms and Holter monitoring 4 to 12 months after ablation. For patients not followed at the University of Pennsylvania, referring cardiologists were contacted and medical records reviewed. Based on VPD burden at follow-up, patients were classified as having no or rare VPDs (<2%), residual VPDs with both an >80% decrease in burden compared to baseline recordings and <5,000 VPDs per 24 hours, or no significant change in VPD burden.

Statistical analysis

Continuous variables are expressed as mean with standard deviation and categorical variables as percentage. Student's

Download English Version:

<https://daneshyari.com/en/article/2923691>

Download Persian Version:

<https://daneshyari.com/article/2923691>

[Daneshyari.com](https://daneshyari.com)