Relative efficacy of catheter ablation vs antiarrhythmic drugs in treating premature ventricular contractions: A single-center retrospective study

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BACKGROUND It is unknown whether radiofrequency ablation (RFA) or antiarrhythmic therapy is superior when treating patients with symptomatic premature ventricular contractions (PVCs).

OBJECTIVE To determine the relative efficacy of RFA and antiarrhythmic drugs (AADs) on PVC burden reduction and increasing left ventricular systolic function.

METHODS Patients with frequent PVCs (>1000/24 h) were treated either by RFA or with AADs from January 2005 through December 2010. Data from 24-hour Holter monitoring and echocardiography before and 6–12 months after treatment were compared between the 2 groups.

RESULTS Of 510 patients identified, 215 (40%) underwent RFA and 295 (60%) received AADs. The reduction in PVC frequency was greater by RFA than with AADs (–21,799/24 h vs –8,376/24 h; $P < .001$). The left ventricular ejection fraction (LVEF) was increased significantly after RFA (53%–56%; $P < .001$) but not after AAD (52%– 52%; $P = .6$) therapy. Of 121 (24%) patients with reduced LVEF, 39 (32%) had LVEF normalization to 50% or greater. LVEF was restored in 25 of 53 (47%) patients in the RFA group compared with 14 of 68 (21%) patients in the AAD group ($P = .003$). PVC coupling interval less than 450 ms, less impaired left ventricular function, and RFA were independent predictors of LVEF normalization performed by using multivariate analysis.

CONCLUSION RFA appears to be more effective than AADs in PVC reduction and LVEF normalization.

KEYWORDS Antiarrhythmic drug; Left ventricular dysfunction; Premature ventricular contraction; Premature ventricular contraction–induced cardiomyopathy; Radiofrequency catheter ablation

ABBREVIATIONS **AAD** = antiarrhythmic drug; **CAD** = coronary artery disease; **CCB** = calcium channel blocker; **DCM** = dilated cardiomyopathy; **ECG** = electrocardiogram; **ICM** = ischemic cardiomyopathy; **LV** = left ventricular/ventricle; **LVEDD** = left ventricular end-diastolic dimension; **LVEF** = left ventricular ejection fraction; **LVESD** = left ventricular end-systolic dimension; **LVOT** = left ventricular outflow tract; **NSVT** = nonsustained ventricular tachycardia; **PVC** = premature ventricular contraction; **PVC-CMP** = premature ventricular contraction–induced cardiomyopathy; **RFA** = radiofrequency ablation; **RV** = right ventricular/ventricle; **RVOT** = right ventricular outflow tract

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Introduction

Classically, premature ventricular contractions (PVCs) have been considered relatively benign in the absence of structural heart disease. However, frequent PVCs may result in left ventricular (LV) systolic dysfunction, a form of PVC-induced cardiomyopathy (PVC-CMP) or PVC-mediated deterioration of preexisting cardiomyopathy.[1] Factors that may lead to the development of PVC-CMP include PVC burden and duration, QRS width, and site of origin. Antiarrhythmic drug (AAD) suppression or catheter-based radiofrequency ablation (RFA) of frequent PVCs may restore ventricular function in the absence of known heart disease, supporting the concept of PVC-CMP.[2,3] For decades, AADs, including class I or III AADs, β-blockers, or a nondihydropyridine calcium channel blocker (CCB), have been

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considered first-line therapy to suppress PVCs. RFA has emerged as an effective alternative to the pharmacologic approach for many forms of supraventricular or ventricular arrhythmia.[4–7] Whether medical therapy or RFA is superior is relatively unknown. The present study sought to compare the efficacy of RFA and AADs on PVC burden and LV systolic function in clinical practice and to evaluate the factors associated with the risk of developing PVC-CMP.

Methods

Study patients

This retrospective study was approved by the Mayo Clinic Institutional Review Board, and all patients included had consented to the use of their data for research purposes. The eligible study cohort was identified by using the Rochester Medical Index database of Mayo Clinic. The Rochester Medical Index classified 5183 patients' diagnoses of PVC between January 2005 and December 2010 by using an internal coding system based on the *Hospital Adaptation of the International Classification of Diseases, Eighth Revision*. These individuals were then cross-identified from the Mayo Holter monitoring database (n = 2933 with 1 or more Holter recordings). Their electronic medical records were thoroughly reviewed by 2 electrophysiologists. Individuals who met the following criteria were included in the study: (1) presence of frequent PVCs, defined as more than 1000/24 h; (2) baseline Holter recordings and echocardiogram within 6 months of PVC diagnosis, (3) follow-up Holter recordings and, if possible, a follow-up echocardiogram; (4) the ablation group who had the first time RFA but may have failed AAD previously; and/or (5) the AAD group who had not received previous drug therapy.

Holter recording

All patients had 2 or more 24-hour Holter recordings, representing before and after therapy. The Holter recordings were reviewed both automatically (Marquette, GE, Inc, Milwaukee, WI, USA) and manually by an electrophysiologist. The total heart beats and the frequency of PVCs and ventricular tachycardia were collected. The burden of PVCs was defined as the total number of PVCs divided by the total number of heart beats. Nonsustained ventricular tachycardia (NSVT) was defined as 3 or more consecutive PVCs at a rate more than 100 beats/min. If the patient had more than 1 recording either before or after therapy, the report with the highest PVC frequency was collected.

Electrocardiographic measurements

The individual 12-lead electrocardiogram (ECG) and 12-lead Holter monitoring results were reviewed manually, and the PVC QRS duration and coupling intervals were measured from 12-lead Holter monitoring and ECG. The PVC QRS duration was measured from the onset to the latest component of QRS. The PVC coupling interval was determined from the onset of the preceding QRS in sinus rhythm to the onset of PVC. Five PVCs were measured and averaged to determine the values.

The PVC origin was determined by using both 12-lead Holter monitoring and 12-lead ECG. If PVC morphology in ECG was not consistent with PVC in Holter monitoring, the origin was determined by most frequent PVC morphology recorded by using Holter monitoring. The origins of PVCs were characterized as the site of origin: (1) right ventricular outflow tract (RVOT): left bundle branch block morphology, with an inferior axis, tall R waves in inferior leads, negative (QS) complexes in avR and avL, an all-negative QS or a small r wave in lead V_1, and R transition in lead V_3 or V_4; (2) left ventricular outflow tract (LVOT): right bundle branch block morphology, with an inferior axis, tall R waves in inferior leads, and negative (QS) complexes in avR and avL; or left bundle branch block morphology, with a more prominent r wave amplitude and duration in lead V_1 and early R transition in lead V_1 or V_2; (3) LV non-OT: right bundle branch block morphology, without the features of the LVOT; (4) RV non-OT: left bundle branch block morphology, without the typical inferior axis or features of the RVOT.

Echocardiography

All echocardiograms were performed at the Mayo Clinic Echocardiography Laboratory, Rochester, MN. The study included only those patients who had echocardiography reports within 6 months of PVC diagnosis. Patients who had echocardiography reports beyond 6 months of diagnosis were excluded from the study. Left ventricular ejection fraction (LVEF; measured by using Simpson's formula), left ventricular end-diastolic dimension (LVEDD), and left ventricular end-systolic dimension (LVESD) were determined by 2 readers. The normalization of LVEF was defined as LVEF of 50% or greater after therapy.

Drug therapy

Patients who were treated medically for the first time at Mayo Clinic were included in the AAD group. AADs include β-blockers, CCBs, mexiletine, flecainide, sotalol, amiodarone, and propafenone. The minimum duration of AAD treatment was 3 months. Patients who were unable to tolerate AADs and discontinued their use before completing 3 months of therapy were excluded from the study.

RFA

PVC morphology at baseline was reviewed to be consistent with 12-lead ECG. For PVCs originating from the LV, a retrograde aortic and/or atrial transseptal approach was used to facilitate mapping and ablation. If few or no PVCs were present at baseline, a programmed ventricular stimulation was performed with the intention of inducing PVCs or ventricular tachycardia at baseline or during intravenous administration of isoproterenol. Three-dimensional electroanatomic mapping (Carto System, Biosense Webster, Inc, Diamond Bar, CA, USA) was used to localize the earliest ectopic ventricular activation. Adjunctive pace mapping was