Effect of ablation of frequent premature ventricular complexes on left ventricular function in patients with nonischemic cardiomyopathy

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BACKGROUND Frequent idiopathic premature ventricular complexes (PVCs) can result in PVC-induced cardiomyopathy. Frequent PVCs can also aggravate ischemic cardiomyopathy.

OBJECTIVE The purpose of this study was to investigate the impact of frequent PVCs on nonischemic cardiomyopathy.

METHODS This was a consecutive series of 30 patients (mean age 59.1 ± 12.1; 18 men; mean ejection fraction [EF] 38% ± 15%) with structurally abnormal hearts based on the presence of scar on cardiac magnetic resonance imaging and/or a history of cardiomyopathy before the presence of frequent PVCs who were referred for ablation of frequent PVCs.

RESULTS Ablation was successful in 18 of 30 patients (60%), resulting in an increase of mean EF from 33.9% ± 14.5% to 45.7% ± 17% ($P < .0001$) during mean follow-up of 30 ± 28 months. The PVC burden in these patients was reduced from 23.1% ± 8.8% to 1.0% ± 0.9% ($P < .0001$). Mean EF did not change in patients with a failed ablation procedure (44.4 ± 16 vs 43.5 ± 21, $P = .85$). The PVC site of origin was in scar tissue in 14 of 18 patients with a successful ablation procedure. Mean New York Heart Association functional class improved from 2.3 ± 0.6 to 1.1 ± 0.2 ($P < .0001$) in patients with a successful outcome and remained unchanged in patients with an unsuccessful outcome (1.9 ± 0.9 vs 1.9 ± 0.7, $P = 1$).

CONCLUSION In patients with frequent PVCs and nonischemic cardiomyopathy, EF and functional class can be improved but not always normalized by successful PVC ablation. In most patients with an effective ablation, the arrhythmogenic substrate was located in scar tissue.

KEYWORDS Premature ventricular complex; Nonischemic cardiomyopathy; Ablation

ABBREVIATIONS DE-MRI = delayed enhanced magnetic resonance imaging; **EF** = ejection fraction; **ICD** = implantable cardioverter-defibrillator; **MRI** = magnetic resonance imaging; **PVC** = premature ventricular complex; **VT** = ventricular tachycardia

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Introduction

Frequent idiopathic premature ventricular complexes (PVCs) are not associated with an increased risk of sudden death[1] but can cause a cardiomyopathy that is reversible by radiofrequency ablation of the PVCs.[2,3] An improvement in left ventricular function after ablation of frequent PVCs also can occur in patients with prior myocardial infarction.[4] Whether patients with nonischemic cardiomyopathy and frequent PVCs also would benefit from an ablation procedure is unclear. The purpose of this study was to assess whether patients with frequent PVCs and nonischemic cardiomyopathy can benefit from an ablation procedure.

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Methods

Characteristics of subjects

The study protocol was approved by the institutional review committee of the University of Michigan. The subjects of this study were 30 consecutive patients with nonischemic cardiomyopathy referred for radiofrequency catheter ablation of frequent PVCs (Table 1). Significant coronary artery disease was ruled out by cardiac catheterization, and there was no remote history of prior myocardial infarction. The patients were diagnosed as having nonischemic cardiomyopathy based on the presence of left ventricular scar by cardiac magnetic resonance imaging (MRI) (n = 26) or based on the

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Table 1 Characteristics of patients with nonischemic cardiomyopathy (n = 30)

Age (years)	59.1 ± 12.1
Sex (male)	18 (60%)
Beta-blocker	19 (63%)
Angiotensin-converting enzyme inhibitor	15 (50%)
Antiarrhythmic drugs	8 (26%)
Amiodarone	3
Sotalol	3
Dofetilide	4
Baseline LVEF fraction	
Echocardiogram	38 ± 15
PVC burden (%)	22.7 ± 11.6
Left ventricular scar (%)	8.5 ± 5.5
Pleomorphic PVCs	18
Ventricular tachycardia inducible	3

Values are given as numbers, (%) and mean ± SD, unless otherwise indicated.

LVEF = left ventricular ejection fraction; PVC = premature ventricular complex.

onset of cardiomyopathy before the occurrence of frequent PVCs (n = 4). Patients were not required to have abnormal left ventricular function as long as scarring was detected by MRI. Four patients had an ejection fraction >50% before ablation. Before the ablation procedure, 6 patients already had an implantable cardioverter-defibrillator (ICD). Four of 6 patients with an ICD were biventricularly paced when they presented with frequent PVCs. The patients had failed to respond to a mean of 1.7 ± 1.1 (range 1–5) antiarrhythmic medications. Amiodarone was used in 3 patients (10%). Long-acting sedatives for sedation were avoided during the ablation procedure in an attempt to minimize PVC suppression due to sedation.[5]

Holter monitoring and echocardiography

Patients underwent preprocedural 12-lead Holter monitoring for 48 hours in 20 patients and for 24 hours in the remaining patients. Holter monitoring was repeated 3–4 months after the ablation procedure. Frequent PVCs were defined as PVC burden >5%.[4] The predominant PVC morphology identified by Holter monitoring was the PVC morphology that was targeted during the ablation procedure. A mean of 4.9 ± 2.3 different PVC morphologies per patient was identified. All patients had 1 predominant PVC morphology: 15 had a left bundle branch block morphology (10 with inferior axis, 5 with superior axis), and 15 had a right bundle branch block morphology (10 with inferior axis, 5 with superior axis). At baseline, during sinus rhythm, 25 patients had a narrow QRS complex before ablation, 3 had a right bundle branch block, and 2 had a left bundle branch block. Abnormal Q waves in the precordial leads were present in 2 patients and in the limb leads in 1 patient. However, cardiac MRI did not show areas of delayed enhancement in the distribution of a coronary artery in these or any other patients.

Echocardiography was performed within 3 months before the ablation procedure and was repeated 3–6 months after ablation. Echocardiography was performed using a Philips iE33 (Philips Healthcare, Andover, MA) or Acuson Sequoia 512 (Siemens, Malvern, PA) system and archived in standard DICOM format. Images were reviewed using Synapse Cardiovascular Client version 4.08 (Fujifilm Medical Systems, Valhalla, NY). Studies were interpreted by 2 independent echocardiographers, each with level III certification in echocardiography and several years of clinical experience, who were blinded to the study and outcome of the ablation procedure. Left ventricular ejection fraction was calculated by the Simpson formula when ≥2 consecutive sinus beats were present and adequate images were available. Initial post-PVC sinus beats were not used to avoid postextrasystolic potentiation of left ventricular function.

Magnetic resonance imaging

All patients had delayed enhanced magnetic resonance imaging (DE-MRI) studies within 4 weeks before the procedure unless there was a contraindication. The studies were performed on a 1.5-T MRI scanner (Signa Excite CV/i, General Electric, Waukesha, WI; or Achieva, Philips, Best, The Netherlands). After a 15-minute delay following administration of 0.15 mmol/kg of intravenous gadobenate dimeglumine (Bracco Diagnostic, Monroe Township, NJ), 2-dimensional DE-MRI was performed using an inversion recovery sequence in the short axis and long axis of the left ventricle with an inversion time that was optimized to null the normal myocardium.[6]

All DE-MRI images were analyzed offline. The endocardial and epicardial contours as well as the area of abnormal signal were traced from the base to the apex of the left ventricle. The full area of delayed enhancement was then automatically determined by a region growing algorithm as the area encompassing pixels with values ≥M/2, using the traditional method of full-width half-maximum.[7] The DE-MRI images were reviewed by 2 observers blinded to the results of the ablation procedure. Both observers classified the scar as intramural (Figure 1), subendocardial (Figure 2A; including transmural scar), or epicardial scar. Discrepancies were resolved by consensus.

Electrophysiologic procedure and mapping

After informed consent was obtained, femoral venous access was obtained, and 2 multipolar electrode catheters were advanced to the His-bundle position and right ventricular apex. Programmed stimulation with up to 4 extrastimuli was performed with and without isoproterenol.[8] If sustained ventricular tachycardia (VT) was induced, an ICD was implanted after the ablation procedure before the patient was discharged from the hospital. VTs were targeted for ablation if they remained inducible after ablation of the predominant PVCs.

If the PVCs had a right bundle branch block morphology, mapping was performed using a retrograde aortic approach through the right femoral artery. Three thousand units of heparin were administered for right-sided procedures, followed by 1000 units per hour. Systemic heparinization to achieve an activated clotting time of 250–300 seconds was