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Radiofrequency Catheter Ablation of Premature Ventricular Complexes From Right Ventricular Outflow Tract Improves Left Ventricular Dilation and Clinical Status in Patients Without Structural Heart Disease

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OBJECTIVES The present study evaluated clinical benefits of radiofrequency catheter ablation (RFA) for premature ventricular complexes from right ventricular outflow tract (RVOT-PVC) in patients without structural heart disease.

BACKGROUND It is unknown whether PVC causes left ventricular (LV) dilation, which is a well-recognized precursor of LV dysfunction and heart failure, and whether eliminating PVC by RFA produces clinical benefits in patients with RVOT-PVC.

METHODS Frequency of PVC per total heart beats by 24-h Holter monitoring, left ventricular ejection fraction (LVEF), left ventricular end-diastolic internal dimension (LVDd), mitral regurgitation (MR) by echocardiogram, cardiothoracic ratio (CTR) by chest radiogram, and New York Heart Association (NYHA) functional class of 40 patients with RVOT-PVC without structural heart disease were evaluated before and 6 to 12 months after RFA.

RESULTS Before RFA, a subgroup of patients with frequent (>20%) PVC demonstrated significantly enlarged LVDd and CTR, reduced LVEF, increased MR, and deteriorated NYHA functional class as compared to the subgroup with rare (<20%) PVC (54 ± 1 mm vs. 45 ± 1 mm, 52 ± 2% vs. 46 ± 1%, 66 ± 2% vs. 73 ± 2%, 1.2 ± 0.2 degree vs. 0.4 ± 0.1 degree, and 1.8 ± 0.2 vs. 1.3 ± 0.1, respectively; $p < 0.05$). Furthermore, ablating RVOT-PVC readily produced the improvement of all these abnormalities (47 ± 1 mm, 41 ± 1%, 72 ± 2%, 0.3 ± 0.1 degree, and 1.0 ± 0.0, respectively; $p < 0.05$ compared with before RFA).

CONCLUSIONS These findings suggest that frequent (>20%) RVOT-PVC may be a possible cause of LV dysfunction and/or heart failure, and RFA produces clinical benefits in these patients. (J Am Coll Cardiol 2005;45:1259–65) © 2005 by the American College of Cardiology Foundation

Isolated premature ventricular complexes (PVC) are the most common arrhythmias observed in patients without structural heart disease (1). It has been recently reported that frequent PVC caused left ventricular (LV) dysfunction that can be reversed by suppression of PVC with antiarrhythmic agents (2,3) or radiofrequency catheter ablation (RFA) (4,5) in patients with dilated cardiomyopathy. It is

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uncertain, however, whether frequent PVC causes LV dilation and dysfunction even in patients with no evidence of structural heart disease and, if so, whether suppression of PVC improves these changes. In recent years, RFA has proven to be a safe and successful therapy for arrhythmias (6,7). The purpose of this study was two-fold: 1) to examine whether frequent premature ventricular complexes from right ventricular outflow tract (RVOT-PVC) without structural heart disease may cause LV dilation, which is a well-recognized precursor of LV dysfunction and congestive heart failure (8); 2) to evaluate the role of ablating RVOT-PVC per RFA on cardiac function in patients with depressed cardiac function.

METHODS

Study population and laboratory analysis. From 1994 to 2004, 45 consecutive patients (10 males and 35 females with mean age of 50 ± 2 years and body surface area of 1.57 ± 0.02 m^2) with monomorphic RVOT-PVC and no evidence of underlying structural heart disease underwent RFA at our hospital. All patients had their history recorded, and underwent physical examination, laboratory analysis, chest radiogram, 12-lead electrocardiogram, 24-h Holter monitoring, M-mode, two-dimensional, and Doppler echocardiogram (SONOS 2000, Hewlett-Packard, San Diego, California, and SEQUOIA 512, Siemens, Erlangen, Germany) on admission or within at least 1 month before admission, and

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Abbreviations and Acronyms

LBBB = left bundle branch block
LV = left ventricle/ventricular
LVDd = left ventricular end-diastolic internal dimension
LVDs = left ventricular end-systolic internal dimension
LVEF = left ventricular ejection fraction
NYHA = New York Heart Association
PVC = premature ventricular complexes
RFA = radiofrequency catheter ablation
RV = right ventricle/ventricular
RVOT = right ventricular outflow tract
%PVC = frequency of premature ventricular complexes

6 to 12 months after RFA (average 8 ± 1 month). The 24-h Holter monitoring and echocardiogram were performed on the same day. All patients also took routine echocardiogram on the next day of RFA to evaluate the possible procedure-related complications. We evaluated the chamber size of LV and right ventricle (RV), and degree of mitral regurgitation at parasternal long-axis or apical four-chamber view, respectively. Left ventricular ejection fraction (LVEF) was calculated by the Teichholz method. All values of echocardiogram were recorded during sinus rhythm, but not at the PVC beat, nor at the post-PVC beat. All patients also underwent the exercise electrocardiogram testing, cardiac catheterization with coronary angiography, and/or 201-thallium scintigraphy. These examinations yielded no evidence of clinically overt structural heart disease, including coronary artery disease, valvular heart disease, congenital heart disease, LV hypertrophy, and RV abnormalities in all patients. Brain natriuretic peptide of five recent patients was evaluated on admission and six months after RFA.

Definition of RVOT-PVC. Right ventricular outflow tract-PVC was defined as a characteristic electrocardiographic appearance of a left bundle branch block (LBBB) contour in V_1 and an inferior axis in the frontal plane. Ventricular tachycardia was defined with standard electrocardiographic criteria of at least five consecutive PVC at a rate >120 beats/min. Patients with ventricular tachycardia and atrial tachyarrhythmia including atrial fibrillation, atrial flutter, atrial tachycardia, and paroxysmal supraventricular tachycardia were excluded in this study because they may cause tachycardia-induced LV dilation (9,10). The region of the LV outflow tract was not an origin of PVC of all patients in this study.

Mapping and catheter ablation procedure. All procedures were performed after written informed consent was obtained. The patients were studied in the fasting state without sedation. Antiarrhythmic drugs were discontinued for at least six half-lives before the procedure. Under local anesthesia, a 7-F deflectable quadripolar ablation catheter (Boston Scientific EP Technologies, Natick, Massachusetts) with a 4-mm-tip electrode was introduced percutaneously into the RV. Based on the 12-surface-lead electrocardiogram with spontaneous RVOT-PVC, pace mapping was done by using bipolar pacing between the distal pair of the electrodes with a stimulation pulse width of 2 ms. If the culprit PVC were not found during the procedure, isoproterenol administration and/or programmed electrical stimulation with digital stimulator (Cardiac Stimulator, Nihon Kohden Co., Tokyo, Japan) was performed to induce the culprit PVC as previously described (6). An optimal pace map was defined as a match of all 12 surface leads by comparing the R/S ratio and subtle notching in the QRS complex during pacing. An identical match was necessary in at least 11 of 12 leads. The RFA was performed based on an optimal pace map for 60 to 90 s with a preset temperature of 50 to 60°C and a power limit of 50 W. A successful ablation was defined as the no recurrence and non-inducibility of culprit PVC with and without isoproterenol administration at the rate of 0.2 to 0.6 μg/min and/or programmed electrical stimulation during at least 30 min after ablation. All 12 surface electrocardiograms and the bipolar intracardiac electrograms (filtered at 30 to 400 Hz) were recorded and stored by using a 48-channel acquisition system (CardioLabEP, Prucka Engineering Inc., Houston, Texas). During the procedure, intravenous heparin was given as a 100 IU/kg bolus dose followed by boluses of 1,000 IU every hour. All patients received oral antiplatelets for eight weeks after RFA, but no antiarrhythmic drugs. Procedural success was defined as no recurrence of culprit PVC within 72 h after procedure under electrocardiogram monitoring.

Statistical analysis. Numerical results are expressed in the text as the mean ± standard deviation. Paired data were compared by Student *t* tests. The differences between the continuous variables before and 1 day or 6 to 12 months after RFA in each group, or between the lower, middle, and upper group in each period, were compared by using a repeated-measures analysis of variance followed by Bonferroni's test for multiple comparisons. Correlation between the two parameters was determined by simple linear regression analysis. A $p < 0.05$ was considered to indicate statistical significance.

RESULTS

Patient characteristics. An RFA procedure for RVOT-PVC was performed in 45 patients. Because five patients (11%) were lost to follow-up, they were excluded from the statistical analysis. Procedural success was achieved in 37 (93%) of the patients. One patient (3%) suffered from a procedure-related complication with femoral arteriovenous fistula. During follow-up, recurrence of culprit RVOT-PVC was observed in one patient (3%), who underwent repeated RFA with successful result. Relationships between the left ventricular end-diastolic internal dimension (LVDd) and the frequency of PVC (%PVC) by 24-h Holter monitoring of 40 patients before RFA were examined (Fig. 1). The %PVC was calculated as: 100·[number of PVC/number of total heart beats per 24 h]. A significant correlation was found between the LVDd and %PVC ($y = 45.8 + 0.254x$, $r = 0.56$, $p < 0.01$), indicating that frequent