Delayed efficacy of radiofrequency catheter ablation on ventricular arrhythmias originating from the left ventricular anterobasal wall

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BACKGROUND Ventricular arrhythmias (VAs) originating from the left ventricular anterobasal wall (LV-ABW) may represent a therapeutic challenge.

OBJECTIVE The purpose of this study was to investigate the delayed efficacy of radiofrequency catheter (RFCA) ablation without an epicardial approach on VAs originating from the LV-ABW.

METHODS Eighty patients (mean age 46.9 ± 14.9 years; 47 male) with VAs originating from the LV-ABW were enrolled. After systematic mapping of the right ventricular outflow tract, aortic root, adjacent LV endocardium, and coronary venous system, 3–4 ablation attempts were made at the earliest activation sites and/or best pace-mapping sites. Delayed efficacy was evaluated in patients with acute failure.

RESULTS During mean follow-up of 23.8 ± 21.9 months (range 3–72 months), complete elimination of all VAs was achieved in 47 patients (59%) and partial success in 19 (24%), for an overall success rate of 83%. In 25 of 37 patients (68%) with acute failure, VAs were eliminated or significantly reduced (>80% VA burden) by the delayed effect of RFCA during follow-up. Logistic regression analysis revealed that response time to ablation was a predictor of occurrence of delayed efficacy. No complications occurred during follow-up.

CONCLUSION Instead of extensive ablation, waiting for delayed efficacy of RFCA may be a reasonable choice for patients with VAs arising from the LV-ABW.

KEYWORDS Ventricular arrhythmia; Radiofrequency catheter ablation; Left ventricular summit; Delayed efficacy, Electrocardiography

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Introduction

The region of the left ventricular (LV) epicardial surface bounded by the left coronary artery, which lies superior to the aortic portion of the LV outflow tract, has been termed the LV summit, which is the most common site of idiopathic epicardial LV arrhythmias and frequently represents a therapeutic challenge.[1] Those ventricular arrhythmias VAs are most commonly ablated within the coronary venous system or through other adjacent structures such as the right ventricular outflow tract (RVOT), aortic cusps, or endocardial aspect of the left ventricular anterobasal wall (LV-ABW).[2–4] When ablation from these adjacent locations fails, an epicardial approach might be considered but is rarely successful in eliminating the arrhythmias because of proximity to major coronary vessels and/or presence of a thick layer of epicardial fat.[5,6] In this circumstance, repeated and long-time ablation from endocardial and/or epicardial approaches might not increase the success rate but might increase the risks of complications. Previous studies have reported that delayed efficacy of radiofrequency catheter ablation (RFCA) might occur after ablation of supraventricular and ventricular arrhythmias.[7,8] They reported that conduction through accessory AV connections or ventricular arrhythmias may disappear permanently after an apparently unsuccessful RFCA. Moreover, Yamada et al[1] reported that VAs originating from the LV summit were eliminated by the delayed effect of RFCA within the anterior interventricular vein (AIVV). In this study, we hypothesized that moderate RFCA through a nonepicardial approach might has delayed efficacy in eliminating VAs arising from the LV-ABW.

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Methods

Study populations

From 2007 to 2015, 80 consecutive patients with symptomatic premature ventricular contractions (PVCs), idiopathic sustained ventricular tachycardia (VT), or nonsustained VT underwent RFCA at our institution. Patients with a site of VA origin in the LV-ABW were determined by successful RFCA or presumed by electrophysiologic study. Echocardiography and exercise stress testing or multidetector computer tomography showed no evidence of structural heart disease in all patients. Baseline characteristics including age, sex, nature of the clinical arrhythmia, and 12-lead ECG during the VAs were recorded. The study was approved by the Institutional Committee on Human Research at Fuwai Hospital, and each patient gave written informed consent. All antiarrhythmic drugs except for amiodarone were discontinued for at least 5 half-lives before the study.

Electrophysiologic study, mapping, and RFCA

Twelve-lead surface ECGs and intracardiac electrograms were recorded simultaneously using a digital multichannel system (BARD Electrophysiology–A Division of C.R. Bard Inc. USA), filtered at 30–400 Hz for bipolar electrograms and 0.05–400 Hz for unipolar electrograms. For patients with a history of sustained VT, programmed ventricular stimulation was performed with ≤3 extrastimuli scanned to a minimum coupling interval of 180 ms after basic drives of 500 and 400 ms from 2 right ventricular sites and burst pacing. If sustained VT was not reliably inducible, nonsustained VT or PVCs believed likely to originate from the same site were targeted. Intravenous infusion of isoproterenol was administered as needed for arrhythmia induction.

An electroanatomic mapping system (Nav-X, St. Jude Medical, St. Paul, MN) was used to guide mapping. Activation mapping was performed using a 4.0-mm ablation catheter (Safire bidirectional ablation catheter or Cool Path, St. Jude Medical) during VT or PVCs. Systematic and comprehensive mapping was performed in the following sequence. In patients with a right bundle branch block VA morphology, the first anatomic structure mapped was the aortic sinus cusp (ASC), followed by the subvalvular area, LV endocardium, great cardiac vein (GCV), and RVOT. In patients with a left bundle branch block VA morphology, mapping was first performed in the RVOT, followed by the ASC, subvalvular area, LV endocardium, and GCV (Figures 1 and 2). Earliest ventricular activation was annotated in each chamber geometry (Figures 3 and 4). Pace-mapping was also performed at the earliest site of activation, using the distal bipolar electrodes at a coupling interval of the PVC or VT interval and stimulus amplitude 1 mA greater than the late diastolic threshold (up to a maximum output of 10 mA and pulse width of 2.0 ms). If present, a matching pace-map (≥10/12 leads) was defined as the site of origin. No cases accepted epicardial mapping and ablation via subxiphoid approach, even though no ideal target was mapped at all sites as previously mentioned.

Nonirrigated radiofrequency (RF) current was delivered with a target temperature of 55°–60°C and maximum power output of 40 W. Irrigated RF current was delivered in temperature-controlled mode, with a maximum power of 40 W, temperature limit of 45°C, and flush rate of 17 mL/min.

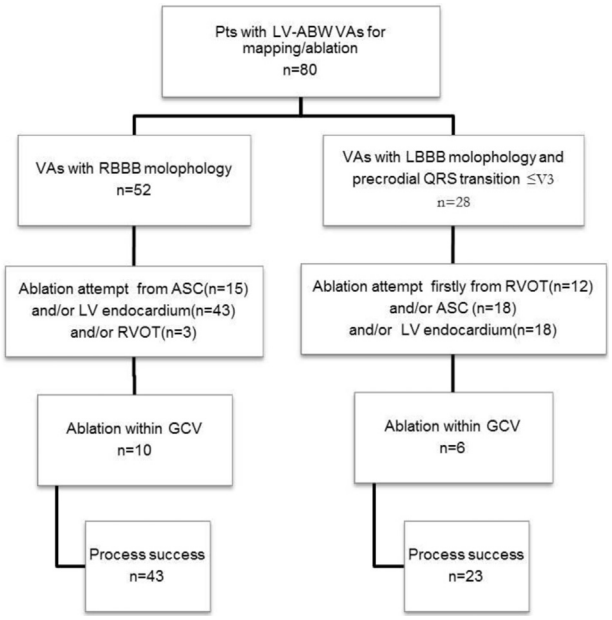

Figure 1 Branch diagram of mapping and ablation strategy. ASC = aortic sinus cusp; GCV = great cardiac vein; LBBB = left bundle branch block; LV = left ventricle; LV-ABW = left ventricular anterobasal wall; RBBB = right bundle branch block; RVOT = right ventricular outflow tract; VA = ventricular arrhythmia.

During catheter ablation in the GCV, simultaneous left coronary angiography was performed to ensure the location of the ablation catheter relative to the left coronary arteries and to minimize the risk of thermal injury to that vessel. RF application was never delivered within 5 mm of a coronary artery. RF application was also not attempted if there was diaphragmatic capture during pacing from the ablation catheter. When acceleration or reduction in the frequency of VT or PVCs was observed during the first 10 seconds of application, RF delivery was continued for 90 seconds, with a target impedance drop of 10 Ω. If the VAs were eliminated, an additional RF application of 90 seconds was applied up to a maximum of 3–4 burns. Otherwise, RF delivery was terminated, and the catheter was repositioned.

If ablations failed to eliminate the VAs, an alternative ablation strategy was performed. Ablation was performed at the earliest site of activation or at the best matching pace-map site 3–4 times for ≈90 seconds each time, even with residual ventricular ectopy. At the end of the procedure, the same induction protocol was repeated.

ECG analysis of VA morphology

Simultaneous 12-lead ECGs during VAs and pace-mapping were recorded digitally at a sweep speed of 100–200 mm/s in all patients for offline analysis. QRS morphologies including bundle branch block pattern, axis, and configuration in leads I were examined.

QRS duration, maximal R-wave amplitude in the inferior leads (II, III, aVF) and R-wave amplitude ratio of lead III/II,