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Idiopathic premature ventricular complexes originating from the distal great cardiac vein: Clinical, cardiac and electrophysiological characteristics and catheter ablation outcome

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ABSTRACT

Aims: Although catheter ablation for idiopathic ventricular arrhythmia (VA) has been generally well-established, VA originating from the great cardiac vein (GCV) may be clinically challenging due to its epicardial origin, proximity to coronary arteries and limited accessibility. The purpose of this study was to explore its electrophysiological characteristics and identify effective mapping/ablation strategies for idiopathic premature ventricular complexes (PVCs) originating from the GCV.

Materials and methods: Between January 2013 to January 2018, 12 patients (who were diagnosed with PVCs originating from the GCV) among the 305 patients with idiopathic left ventricular outflow tract tachycardia were included. The origin of the ectopy was localized by mapping, the characteristics of the electrocardiogram (ECG) were analyzed, and all the patients with PVCs originating from GCV were treated by radiofrequency catheter ablation (RFCA). The safety and efficacy of RFCA were evaluated.

Key findings: The origin of the ectopy was successfully localized in GCV for all 12 patients by mapping, and access to GCV *via* the coronary sinus was feasible. Successful RFCA was achieved in 11 of 12 patients (91.67% acute procedural success) without perioperative complications. During a median follow-up of 12.6 ± 6.5 months, only one patient had recurrent VA (recurrence rate: 9.1%).

Significance: ECG characteristics may be helpful for identifying patients with PVCs originating from the GCV. RFCA within the coronary venous system appears to be safe and effective for these patients, and should be considered when routine RFCA from the endocardium or aortic sinus of the Valsalva is not effective.

1. Introduction

Idiopathic premature ventricular complexes (PVCs) arising from the right or left ventricular outflow regions have been recognized as one of the most common arrhythmias for patients without structural heart disease [1]. Radiofrequency (RF) catheter ablation has been considered a safe and effective therapy for this situation, and subendocardium RF ablation is the routine approach for ablation. However, for some patients with PVCs originating from the epicardial or subendocardial of the ventricles, endocardial RF ablation has been considered to be less therapeutically significant, because the origins of the arrhythmia are remote [1,2]. Therefore, development of novel mapping and ablation routines are clinically important for the treatment of PVCs from the epicardial region of the heart.

Recently, it has been shown that the great cardiac vein (GCV) may provide routes for the mapping and ablation of PVCs from the left ventricular outflow regions [3]. However, this region has been suggested to be anatomically complex, since it contains many complex structures such as the left ventricular outflow tract (LVOT), aortic sinus of the Valsalva (ASOV) and the GCV. Therefore, epicardial access to these ventricular outflow regions may not be easily achievable due to this anatomic consideration [4,5]. However, to the best of our knowledge, limited studies that evaluated the epicardial access to ventricular outflow regions are available.

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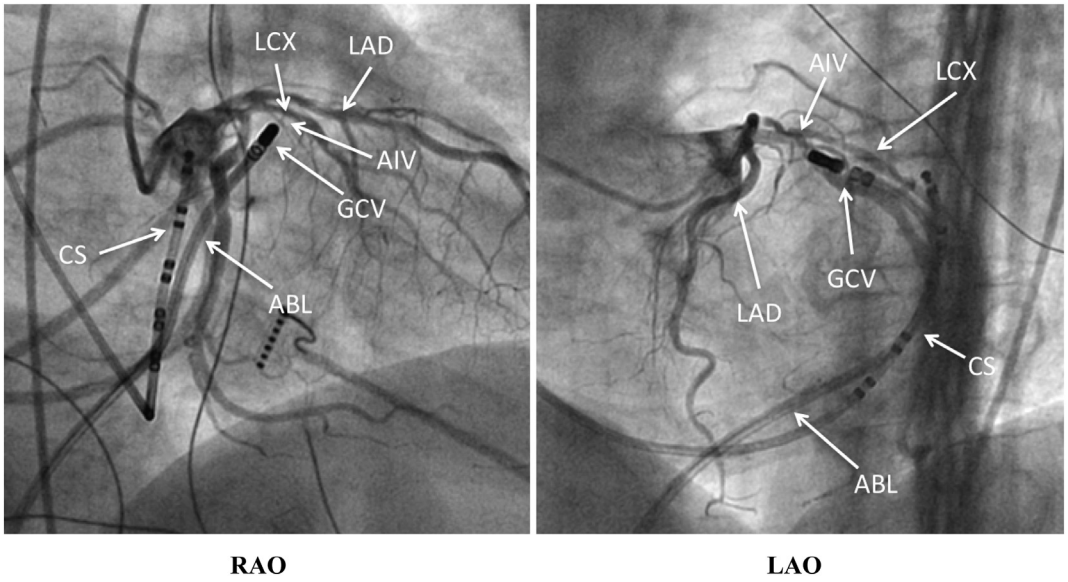

Fig. 1. Selective angiography of the coronary artery and fluoroscopic views (RAO and LAO) showing the ablation mapping catheter in the distal of the GCV. The ablation mapping catheter tip was > 5 mm distant from any coronary artery. RAO, right anterior oblique; LAO, left anterior oblique; GCV, great cardiac vein; LAD, left anterior descendant; LCX, left circumflex; ABL, ablation catheter;

Although early experience did not support surface electrocardiographic (ECG) as the examination of choice for defining the origin of the PVCs, subsequent studies have shown that different ECG parameters may predict PVCs originating from the LVOT [6–7]. However, it remains to be determined whether ECG is helpful for identifying PVCs from the GCV. Therefore, the aim of the present study was to reveal these clinical and cardiac electrophysiological characteristics under three-dimensional (3D; CARTO system; Biosense-Webster Inc., Diamond Bar, CA) activation mapping in patients with PVCs originating from the GCV, and explore the feasibility and efficacy of RFCA from the GCV for the treatment of these patients.

2. Methods

From January 2013 to January 2018, 12 patients, who were derived from 305 consecutive hospitalized patients scheduled for electrophysiological evaluation and RFCA of PVCs originating from the GCV, were enrolled into the study. A 24-h ECG Holter monitor was carried out at least once before catheter ablation. The study protocols were approved by the Ethic Committee of the First Affiliated Hospital of Xinjiang Medical University. All patients provided an informed agreement prior to enrollment.

2.1. Inclusion and exclusion criteria

Patients who fulfilled all of the following criteria were included: (1) frequent PVC incidence, as confirmed by the average PVC count of > 10,000 beats during the 24-h Holter ECG monitoring; (2) highly symptomatic; (3) unsuccessful treatment with at least two anti-arrhythmic drugs (AADs); (4) no structural heart disease. Accordingly, patients with any of the following clinical conditions were excluded: (1) structural heart disease, such as coronary artery disease, valvular heart disease, congenital heart disease, left ventricle hypertrophy and right ventricle abnormalities, as confirmed by medical history and physical examinations, including 12-lead ECG, 24-h Holter monitoring, laboratory examination, X-ray and transthoracic echocardiogram (TTE), and emission computed tomography (ECT); (2) renal or hepatic dysfunction; (3) patients who did not receive AAD treatment.

2.2. Electrophysiological study and mapping

The electrophysiological study was performed after discontinuation of AADs for at least five half-lives. Bipolar electrode catheters were positioned in the right atrium and right ventricle for pacing and recording. A decapolar electrode catheter was introduced into the right internal jugular veins, and positioned in the coronary sinus (CS) under fluoroscopic guidance with delivery into the GCV and/or proximal anterior interventricular vein (AIV), as far as possible.

Electroanatomic mapping and pacing were performed using a D-curve catheter with a 3.5-mm-tip irrigated ablation catheter introduced from the right femoral vein (NaviStar Thermo-Cool; Biosense Webster Inc., Diamond Bar, CA) for all patients. Arterial access was achieved *via* the femoral artery. After vascular access was obtained, 3000 units of heparin were intravenously administered to maintain an activated clotting time of 250–300 s. A 12-lead surface ECG and intracardiac electrogram were recorded and stored on a multichannel oscilloscopic recorder for offline analysis (Cardiolab EP system, General Electric Healthcare, Buckinghamshire, UK). Intravenous isoproterenol (2–5 μg/min) was administered as needed to provoke PVCs.

Electroanatomic mapping was performed as previously described [8]. In brief, for patients with spontaneous or inducible PVCs, point-by-point activation mapping was performed with bipolar electrograms to create 3D anatomical maps of LVOT, ASOV, and the GCV. Activation time was measured according to the methods of Ou Yang et al. [9] In order to identify the site of earliest activation, activation time during the procedure was measured from the time of local activation (on the intracardiac electrogram) to a pre-defined surface lead. If the overall site of earliest ventricular activation was found in the GCV, coronary angiography and CS venography were performed to identify the exact anatomical location. The origins of the PVCs were defined by a combination of coronary artery angiography, venography, and 3D activation mapping. During the procedure for point-by-point mapping, a color tag marker was used for the identification of particular positions, such as left main coronary artery opening and His bundle, in order to avoid severe complications. In order to visualize local ventricular electrogram potentials, the intracardiac electrograms were amplified, and the originating sites of PVCs were identified by earliest activation on the bipolar and unipolar recordings. Activation mapping and pace-mapping were undertaken digitally at a velocity of 100–200 mm/s for further