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Brief Report
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Conjunction of Endocardial and Coronary Venous System Mapping to Ablate Ventricular Arrhythmias

Hung-Ta Wo, Jih-Kai Yeh,* Po-Cheng Chang, Ming-Shien Wen, Chun-Chieh Wang, Chung-Chuan Chou and San-Jou Yeh*

Background: Ablation of idiopathic ventricular arrhythmias (VAs) with epicardial or intramural origins is technically challenging. Herein, we have described the successful ablation of left VAs via the coronary venous system (CVS) in conjunction with endocardial map guided by three-dimensional electroanatomical map in six patients.

Methods: Out of a total consecutive 84 patients with symptomatic idiopathic VAs, radiofrequency ablation via the CVS was performed on six patients (7%). Furthermore, we reviewed patient records and electrophysiologic studies with respect to clinical characteristics.

Results: Activation map was conducted in 5 patients, and the earliest activation sites were identified within the CVS. The preceding times to the onset of QRS complex were longer than those at the earliest endocardial sites (36.2 ± 5.6 ms vs. 14.2 ± 6.4 ms, $p = 0.02$, $n = 5$). Spiky fractionated long-duration potentials were recorded at the successful ablation sites in all 5 patients. The other patient received pacemapping only because of few spontaneous VAs during the procedure, and the best pacemap spot was found within the CVS. Irrigated catheters were required in 4 out of 6 patients because VAs were temporarily suppressed with regular ones.

Conclusions: Idiopathic VAs can be ablated via the CVS in conjunction with endocardial mapping. Additionally, spiky fractionated long-duration potential can function as a clue to identify the good ablation site.

Key Words: Ablation • Coronary venous system • Epicardial ventricular arrhythmia

INTRODUCTION

Idiopathic ventricular arrhythmias (VAs) account for 10-20% of all VAs,[1,2] and the foci may be located at endocardium, mid-myocardium, or epicardium. Successful trans-cardiac vein radiofrequency catheter ablation (RFCA) of epicardial VAs via a cardiac vein branch was first described in 1997.[3] Most retrospective studies reported that the ablation targets were at the great cardiac vein (GCV) via coronary venous system (CVS) approach.[4-6] Since the percutaneous pericardial mapping with RFCA carries a higher risk of major complications and morbidities,[7] transvenous mapping with RFCA should be still considered for idiopathic VAs.

A three-dimensional (3-D) cardiac mapping system helps guide accurate ablation of idiopathic VAs,[8] and the application of 3-D mapping for left epicardial VAs within distal GCV has been reported.[4] However, the thick caliber makes manipulation of a 3-D mapping catheter within CVS technically challenging. Herein, we have reported on 6 patients who received left ventricular endocardial 3-D mapping and endocardial RFCA alone, which did not completely eliminate the VAs. The foci of VAs were successfully ablated endocardially with combined RFCA and within the CVS at the corresponding areas of the earliest endocardial activation sites.

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Division of Cardiology, Chang Gung Memorial Hospital, Linko, and Chang Gung University College of Medicine, Taoyuan, Taiwan.
Address correspondence and reprint requests to: Dr. Chung-Chuan Chou, Division of Cardiology, Chang Gung Memorial Hospital, No. 199, Tung-Hwa North Road, Taipei 10591, Taiwan. Tel: 886-3-328-1200 ext. 8115; Fax: 886-3-328-9134; E-mail: 2867@cgmh.org.tw
* H.T. Wo and J.K. Yeh contributed equally to this study.

MATERIALS AND METHODS

Study population

The study was approved by the institutional review board of Chang Gung Memorial Hospital. There were consecutive 84 patients (51 male and 33 female) referred for drug-refractory symptomatic idiopathic VAs from January 2012 to June 2014. From that original number, six patients (7%, 4 male and 2 female) underwent failed endocardial RFCA, received RFCA within the CVS and were enrolled in this study. All patients had normal left ventricular systolic function at the moment of RFCA. Either invasive or non-invasive myocardial stress tests had been conducted to exclude myocardial ischemia or infarction. One patient (No. 4) had a history of coronary artery disease; however, the ultimate site of the VA was not related to the territory of the stenotic coronary artery and residual ischemia had been excluded. Holter monitoring examinations were obtained before and after RFCA in all patients except patient No. 3, who developed hemodynamically unstable ventricular tachycardia (VT) before the procedure.

Electrophysiological studies and mapping

Antiarrhythmic drugs were discontinued for at least five half-lives before the procedure, except patient No. 3 who received a loading dose of intravenous amiodarone of 150 mg after cardioversion for hemodynamically unstable VT. All patients received the procedure under local anesthesia with 2% lidocaine and with additional midazolam 2.5 to 5 mg intravenous bolus if needed, except patient No. 4 who received general anesthesia. A bolus dose of intravenous heparin (3000 units) was administered for left ventricular approach and one more dose of 1000 units was used when the duration of left ventricular approach was longer than 1 hour. Twelve-lead surface electrocardiogram (ECG), and bipolar and unipolar electrograms were recorded using a standard electrophysiology recording system (CardioLab, GE Healthcare, Fairfield, CT, USA). CARTO 3-D electroanatomical mapping system (Biosense Webster, Diamond Bar, CA, USA) was used to construct the activation maps. VAs were induced using a combination of programmed stimulation, burst ventricular pacing, and isoproterenol infusion.

RFCA within the CVS

RFCA was performed at the site of the earliest activation within the CVS guided by CARTO activation maps with a 4-mm non-irrigated CARTO catheter, or a 3.5-mm open-tip irrigated CARTO catheter. If the catheter could not be manipulated to the target site, a non-CARTO catheter was used to target the foci of VAs. Once the catheter was positioned at the target site, coronary angiography was performed to identify the spatial relationship between the CVS and the coronary arteries. Radiofrequency (RF) energy was delivered if the distance of the ablation catheter tip to the closest coronary artery was > 4 mm.[9] The RF energy was delivered for up to 60 seconds at a power of 15-25 watts and a target temperature of 50-55 °C with a non-irrigated ablation catheter, or of 15-20 watts, target temperature 43 °C with an irrigated ablation catheter. Impedance was assessed and monitored continuously during RFCA. After ablation of the VAs, programmed stimulation and isoproterenol infusion were repeated to test VA inducibility. The procedure was considered successful whenever the VAs were not induced and the follow-up Holter exam revealed ventricular ectopy < 100 beats/24 hours. The patient received out-patient clinic follow-up 1week, 3 months, 6 months and then by phone call. Additional out-patient clinic follow-up was dependent on the patients and the operators.

Statistical analyses

Continuous variables were expressed as mean ± standard deviation. The Student's-test was used to compare difference of continuous variables. A p value < 0.05 was considered statistically significant.

RESULTS

Patient characteristics and ECG characteristics

Four patients had paroxysmal VT with a mean cycle length of 370 ms (range 250-468 ms), and 2 patients had symptomatic ventricular premature complexes. The ECGs showed inferior axis and right bundle branch block configuration of the QRS complexes in 5 out of 6 patients (Figure 1, upper column). Superior axis with R-wave transition in V4 was noted in patient No 5. The pseudo-delta wave duration was 43 ± 7 ms. The maxi-

mum deflection index (MDI) was 51 ± 7%. The clinical and electrophysiological characteristics are shown in Table 1. Two patients had a Q wave in lead I, and 5 had an S wave in lead I. According to Valles' report,[10] only 2 patients fulfilled the criteria of epicardial VAs.

Mapping and catheter ablation

The results of the ablation procedures are shown in Table 2. Activation mapping was performed in 5 patients except patient No. 5 in whom RFCA was guided by pacemapping alone because of few spontaneous VAs during the procedure. Because the earliest endocardial activation to the onset of QRS complex was not significant (< 20 ms), all 5 patients received subsequent CVS mapping. Compared with the endocardial sites (Figure 2), the preceding time to the onset of QRS complex at the earliest activation sites within CVS was significantly earlier (36.2 ± 5.6 ms vs. 14.2 ± 6.4 ms, p = 0.02, n = 5); and the activation duration was also significantly longer (102.4 ± 16.5 ms vs. 61.4 ± 15.1 ms, p = 0.02, n = 5). In patient No. 5, we did not find a spot with acceptable pacemap ECG morphology (match score ≥ 10) endocardially and therefore performed pacemapping within the CVS (Figure 1). The earliest activation or the best pacemap sites were located within the GCVs in 4 patients (No. 1, 2, 4, and 6), at the lateral cardiac vein in 1 patient (No. 3), and at the middle cardiac vein in 1 patient (No. 5). The mean distance to the closest coronary artery was 5.3 ± 0.5 mm. RFCA with a non-irrigated ablation catheter failed to eliminate the VA foci because of an abrupt rise of impedance or temperature at low energy (less than 8

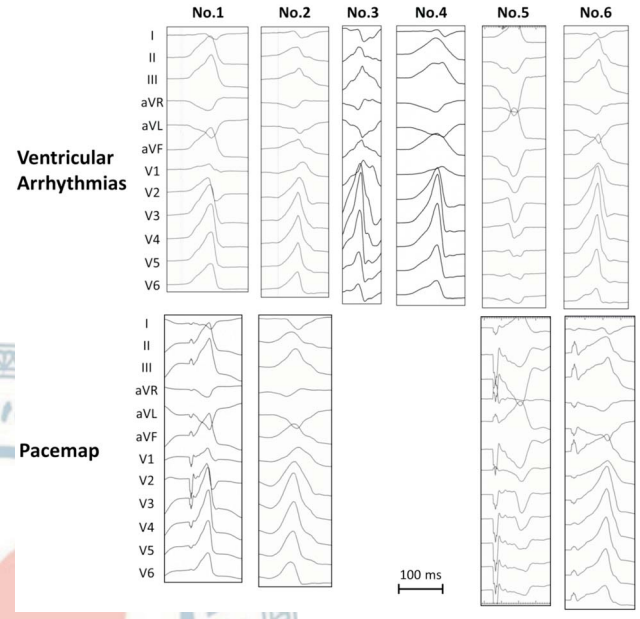

Figure 1. Twelve-lead electrocardiograms of ventricular arrhythmias and pacemap.

Table 1. Baseline characteristics of the six patients

	No. 1	No. 2	No. 3	No. 4	No. 5	No 6
Age (years)	54	47	33	68	45	38
Gender	F	M	M	M	F	M
LVEF	62%	59%	65%	62%	78%	64%
VPC burden*	30076 (33.2%)	43294 (36.8%)	No data	78548 (64.5%)	6770 (4.8%)	4630 (4%)
Electrocardiogram						
Pseudo-delta wave	39 ms	41 ms	39 ms	54 ms	35 ms	52 ms
Lead I	rS	rS	qrS	qRS	R	RS
MDI	55.5%	52.9%	35.9%	51.9%	49.6%	57.6%
Precordial R-wave transition	V1	V1	V1	V1	V4	V1
CAD	Negative for ischemia on stress test	Negative for ischemia on stress test	Negative for ischemia on stress test	Total occlusion at the middle part of RCA	Negative for ischemia on stress test	Negative for ischemia on stress test
Indications of ablation	NSVT	NSVT	VT	NSVT	VPC	VPC
CL of VT	411 ms	350 ms	250 ms	468 ms	Nil	Nil
Refractory drugs	Sotalol	Beta-blocker, Mexiletine	Amiodarone	Amiodarone, Flecainide	Beta-blocker, Mexiletine	Beta-blocker

* Value represents the total beats and the percentage of VPCs within 24 hours. Patient No. 3 had no 24-hour Holter recording before the ablation.

CAD, coronary artery disease; CL, cycle length; LVEF, left ventricular ejection fraction; MDI, maximal deflection index; NSVT, non-sustained ventricular tachycardia; RCA, right coronary artery; VPC, ventricular premature complex; VT, ventricular tachycardia.

Table 2. Data of the ablation procedure

	No. 1	No. 2	No. 3	No. 4	No. 5	No. 6
Other mapping sites	RVOT, LVOT	LV endocardium, MA	ASV	LV endocardium, MA	ASV, LV endocardium	RVOT, LVOT, MA
Final ablation site	Great cardiac vein	Great cardiac vein	Lateral cardiac vein	Great cardiac vein	Middle cardiac vein	Great cardiac vein
Mapping method	Activation map and pace map	Activation map and pace map	Activation map	Activation map	Pace map	Activation map and pace map
Local activation potential (CVS)						
Time preceding to QRS	31 ms	31 ms	41 ms	35 ms	Only pace map	43 ms
Morphology	Fractionated	Fractionated	Fractionated	Biphasic	Only pace map	Fractionated
Activation duration	87 ms	89 ms	125 ms	114 ms	Only pace map	97 ms
Local activation potential (Endocardium)*						
Time preceding to QRS	25 ms	16 ms	14 ms	10 ms	Only pace map	6 ms
Morphology	Fractionated	Biphasic	Biphasic	Biphasic	Only pace map	Biphasic
Activation duration	66	73	44	77	Only pace map	47
Ablation catheter in CVS	Non-irrigated and irrigated	Irrigated	Non-irrigated	Non-irrigated and irrigated	Non-irrigated	Non-irrigated and irrigated
Duration of follow-up (months)	27	25	23	23	14	7

* Data indicate the earliest activation site of the endocardium.

ASV, aortic sinus of Valsalva; CVS, coronary venous system; LV, left ventricle; LVOT, left ventricular outflow tract (subvalvular area); MA, mitral annulus; RVOT, right ventricular outflow tract.

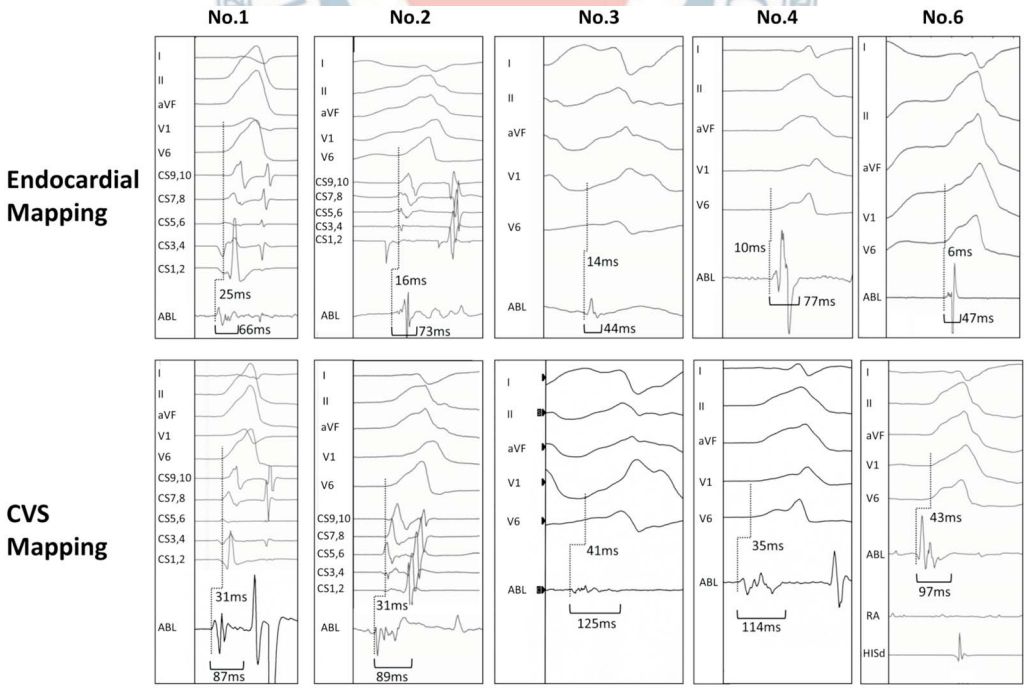

Figure 2. *Electrograms at endocardial sites and within the CVS during spontaneous ventricular arrhythmias. ABL, ablation catheter; CS, coronary sinus; HIS d, distal HIS; RA, right atrium.*

watts) in 3 patients, and an additional irrigated catheter was required.

Figure 3 shows an example of RFCA for the left VT. Endocardial CARTO 3-D mapping showed the earliest activation site was located at the high lateral wall, where RFCA failed to terminate the VT. As shown in Figure 3B, the local activation time showed stepwise advancing earliness from the ostium of the coronary sinus to the GCV, and the earliest activation site was located at the lateral cardiac vein. Note that the spiky and long-duration fractionated electrogram at the lateral cardiac vein arose earlier than that at the GCV (preceding the QRS complex by 41 ms and 34 ms, respectively). Figure 3C shows the coronary venograms indicating the location of the successful ablation site within the lateral cardiac vein. The VT was terminated 30 seconds after the onset of RFCA (20 watts, maximal temperature 50 °C, non-irrigated ablation catheter).

In 3 patients (No. 2, 4, and 6), VAs were only temporarily suppressed after RFCA within the CVS, and combination of repeated RFCA at the corresponding endocardial site was needed to eliminate VAs. In these 3 patients, the earliest activation time was 36 ± 5 ms within CVS and 11 ± 4 ms at the corresponding endocardial site prior to the onset of the QRS complex. The foci might be located intramurally rather than epicardially. Figure 4 shows an example that VA recurred with a slightly different morphology after ablation within the CVS (patient No. 4). Initially, the earliest activation site was located in the GCV (Figure 4A, left subpanels) with right axis deviation (Figure 4B, left subpanels), preceding the QRS complex by 35 ms. After RFCA within the GCV, the VA was temporarily suppressed and then recurred. The surface ECG of the recurrent VA showed a normal axis (Figure 4B, right subpanels) with smaller amplitude in the limb leads, and the earliest site was shifted to the endocardium of the left ventricle adjacent to the CVS ablation site (preceding the QRS complex by 23 ms). Further RFCA at the endocardial site (Figure 4A, right subpanels) successfully abolished the VA.

In patient No. 6, inadequate RF energy delivery prohibited successful RFCA via the CVS approach. As shown in Figure 5, CARTO activation map (panel A) showed the earliest activation located at the distal GCV (preceding

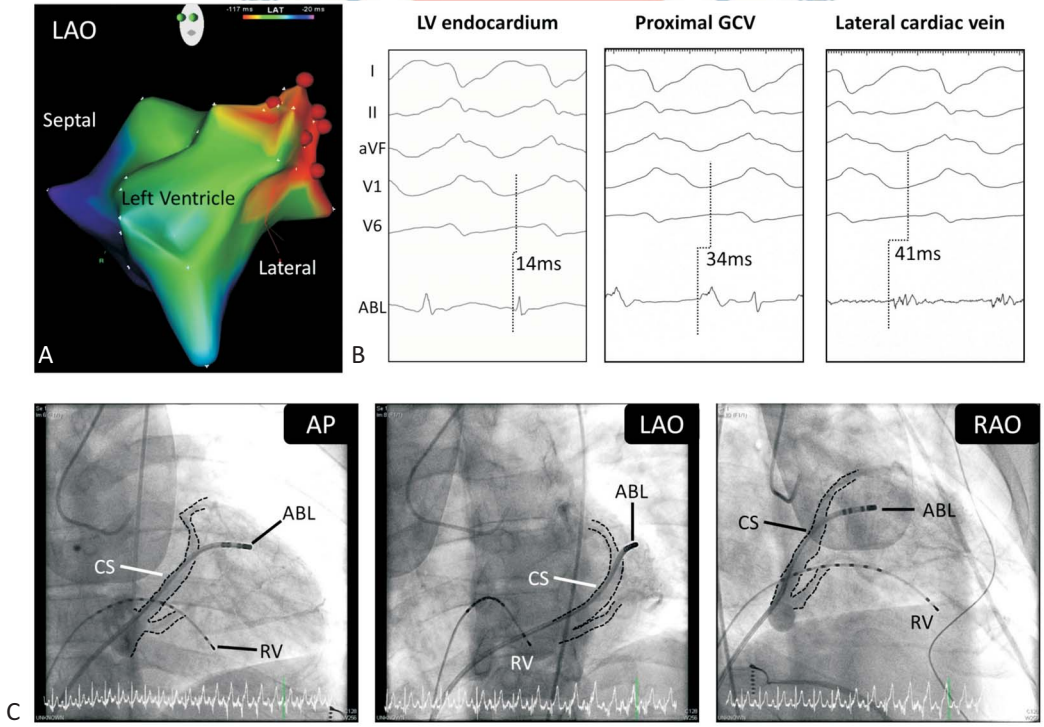

Figure 3. *An example of catheter ablation within the lateral cardiac vein. (A) The CARTO activation map showed the earliest endocardial activation site at the high lateral left ventricular wall. (B) The earliest local activation was recorded at the lateral cardiac vein. (C) The ablation site within the lateral cardiac vein in the anteroposterior (AP), left anterior oblique (LAO) and right anterior oblique (RAO) projections. Dotted lines indicate the outlines of the coronary venous system. GCV, great cardiac vein; RV, right ventricular catheter.*

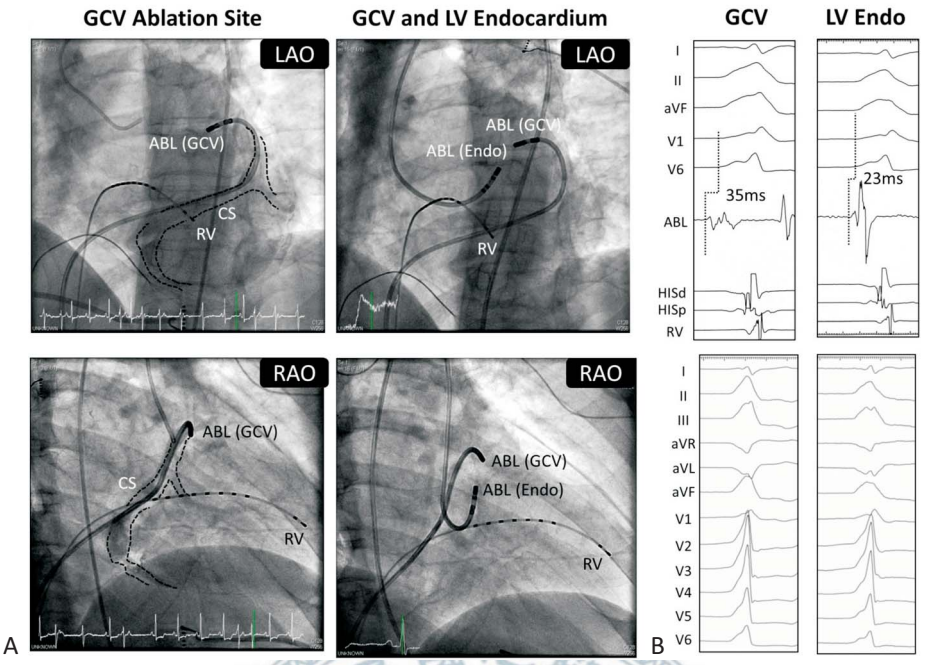

Figure 4. *Demonstration of the "two-side" approach in patient No. 4. (A) Radiographs of the ablation sites within CVS and at the LV endocardium. Dotted lines demonstrate the outlines of the CVS. (B) Upper subpanels show ablation site signals of ventricular arrhythmias with exit sites of the GCV and the corresponding endocardial site of the left ventricle. Lower subpanels are the 12-lead ECGs. The QRS morphologies were slightly different. ABL, ablation catheter; CVS, coronary venous system; GCV, great cardiac vein.*

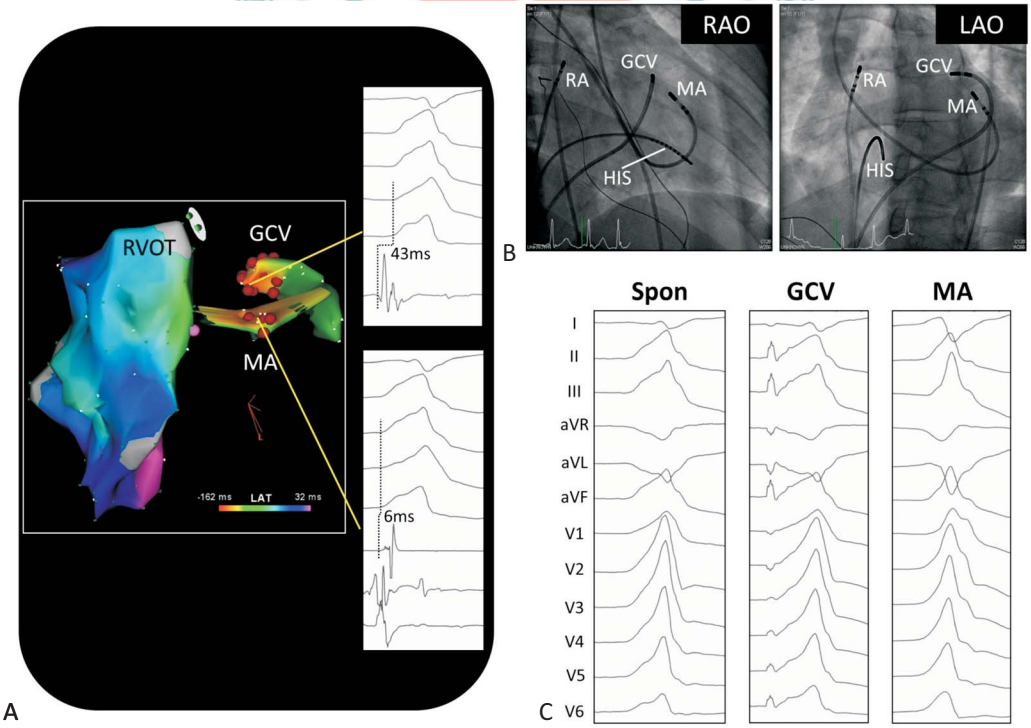

Figure 5. *Demonstration of the "two-side" approach in patient No. 6. (A) Combination of CARTO activation map and the activation sequence. CARTO map is shown in a LAO-cranial projection. Because of late onset of the activation from right ventricular outflow tract, subsequent maps of CVS and mitral annulus were performed. Red dots indicate ablation sites. (B) Radiographs obtained in right anterior oblique (RAO) and LAO projections. (C) Twelve-lead ECGs of a spontaneous intrinsic ventricular premature beat (Spon), of pacemap within the great cardiac vein (GCV) and of pacemap at the lateral mitral annulus (MA). MA, mitral annulus; RA, right atrium; RVOT, right ventricular outflow tract.*

the QRS complex by 43 ms), where the paced ECG morphology matched perfectly (panel C). However, repeated RFCA at the distal GCV could not eliminate the VA because the maximal delivered RF energy power was only 8 and 15 watts for non-irrigated and irrigated ablation catheters, respectively, due to an abrupt rise of impedance and temperature during RFCA. Further RFCA at the corresponding endocardial site near the mitral annulus (panel B) completely eliminated the VA (the maximal energy to 50 watts, 55 °C) even though the earliest endocardial activation time was only 6 ms (later than that in the GCV) preceding the QRS onset and the pacemap was partly matched (panel C).

Ultimately, all patients underwent successful RFCA. At the end of this study, only patient No. 2 had rare symptoms within the first week, and the follow-up Holter exam revealed only 185 ventricular ectopy beats within a 24-hour period.

DISCUSSION

In this study, we successfully ablated idiopathic VAs within the CVS guided by endocardial CARTO 3-D mapping in 6 patients. The local activation potentials at the target sites within the CVS showed spiky and long-duration fractionated configurations, and at least 31 ms preceding the onset of QRS complex. Therefore, the foci of VAs might be semi-insulated epicardial or intramural origins with slow conduction to the surrounding myocardium. Additional irrigated open-tip catheters were used in 4 of the 6 patients to eliminate the arrhythmogenic foci.

Spiky long-duration fractionated electrograms at the origin of VAs within the CVS

In post-infarction reentry VT, the presence of broad, fractionated endocardial electrograms indicates the critical isthmus for reentry circuit in healed myocardial infarction zones.[11] Recording of low-amplitude fractionated diastolic electrograms within coronary venous branches adjacent to an aneurysm or scar can help to identify structural heart disease-related VTs with an epicardial origin,[12] but the mechanism does not account for the local spiky fractionated electrograms recorded at the earliest activation site within the CVS in idiopathic VAs. On the contrary, these spiky long-duration fractionated electrograms in this study mimic the electrical signals recorded at the arrhythmogenic foci of the pulmonary veins during the initiation of atrial fibrillation, indicating the role of triggers and delayed conduction.[13] This long activation duration may be related to a local fibrotic boundary and/or a deeply embedded origin of the triggers to delay impulse propagation into the surrounding myocardium. This pattern of spiky long-duration fractionated electrograms within the CVS may support the presence of intramural or epicardial foci with slow conduction to surrounding myocardium. As shown in Figure 2, the slight changes of surface ECG during the ablation procedure might also be due to the changed exit sites after the initial exit site ablation.

Catheter ablation within the CVS

Successful catheter ablation within the CVS depends on three factors: the proximity to the origin of intramural and epicardial VAs, the neighboring coronary arteries, and the diameter of the CVS. Sometimes foci are deeply embedded in the myocardium, and additional RF application to the closest endocardium or adjacent structures is needed (as shown in Figures 4 and 5). In addition, the narrow lumen of the CVS limits the delivery of sufficient RF energy, and ablation might be more effective using an irrigated ablation catheter.[6]

Limitations

This study did have certain limitations. The study population was relatively small for meaningful statistical interpretation. Additionally, we did not perform cardiac MRI, therefore it is unknown if the fractionated potentials at the target sites are due to the possible presence of scar tissue. Traditionally, the follow-up period of idiopathic VAs after ablation should be at least 12 months. In our study, one of 6 patients did not fulfill adequate follow-up.

CONCLUSIONS

Idiopathic left VAs can be successfully and safely ablated within the CVS in conjunction with the endocardial approach guided by 3-D map. The spiky long-duration fractionated potentials preceding QRS complex at the

target sites may be a clue of semi-insulated intramural or epicardial foci, which require a combination of endocardial and epicardial ablation with an irrigated catheter.

CONFLICT OF INTEREST

None.

DISCLOSURE

None.

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