


CASE REPORT

Co-occurrence of late potential and discrete prepotential on ablation site in a case of cardiac crux located premature ventricular complexes successfully ablated within the middle cardiac vein

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Abstract

Some premature ventricular complexes (PVCs) originate from the coronary venous system. The great cardiac vein and the anterior cardiac vein are the most frequent localizations. The middle cardiac vein is an unusual anatomy for a point of origin for PVC. We present here a case of frequent PVCs with characteristic electrocardiographic features, which we successfully ablated inside the middle cardiac vein.

KEYWORDS

cardiac crux, discrete prepotential, late potential, middle cardiac vein, premature ventricular complex

1 | INTRODUCTION

Idiopathic premature ventricular complexes (PVCs) are common arrhythmias, and most of the time ablation of these arrhythmias is satisfactory. However, ablation of PVCs arising from uncommon localizations like the epicardium, the coronary venous system, or the papillary muscle can be challenging. Difficulties in obtaining the ideal ablation point, inadequate heating in the coronary venous system, pericardial fatty tissue, and proximity to the coronary artery are commonly seen problems.¹ The cardiac crux is situated in the poster septal part of the heart where the borders of all the heart's chambers meet. Additionally, the coronary sinus is adjacent to this region and the middle cardiac vein (MCV) lies on it. Data are scarce

regarding ablation of ventricular arrhythmias that originate from this part of the heart. In most of the cases published in the literature, cardiac crux-originated PVCs generally required epicardial intervention. We wanted to share our experience of a case of frequent PVCs, which we ablated inside the MCV.

2 | CASE REPORT

A 39-year-old male patient was referred to our clinic due to frequent PVCs. The patient had been presenting symptoms of syncope and palpitation for the past 5 years. Effort dyspnea had been added to his complaints in the previous 2 months. He had

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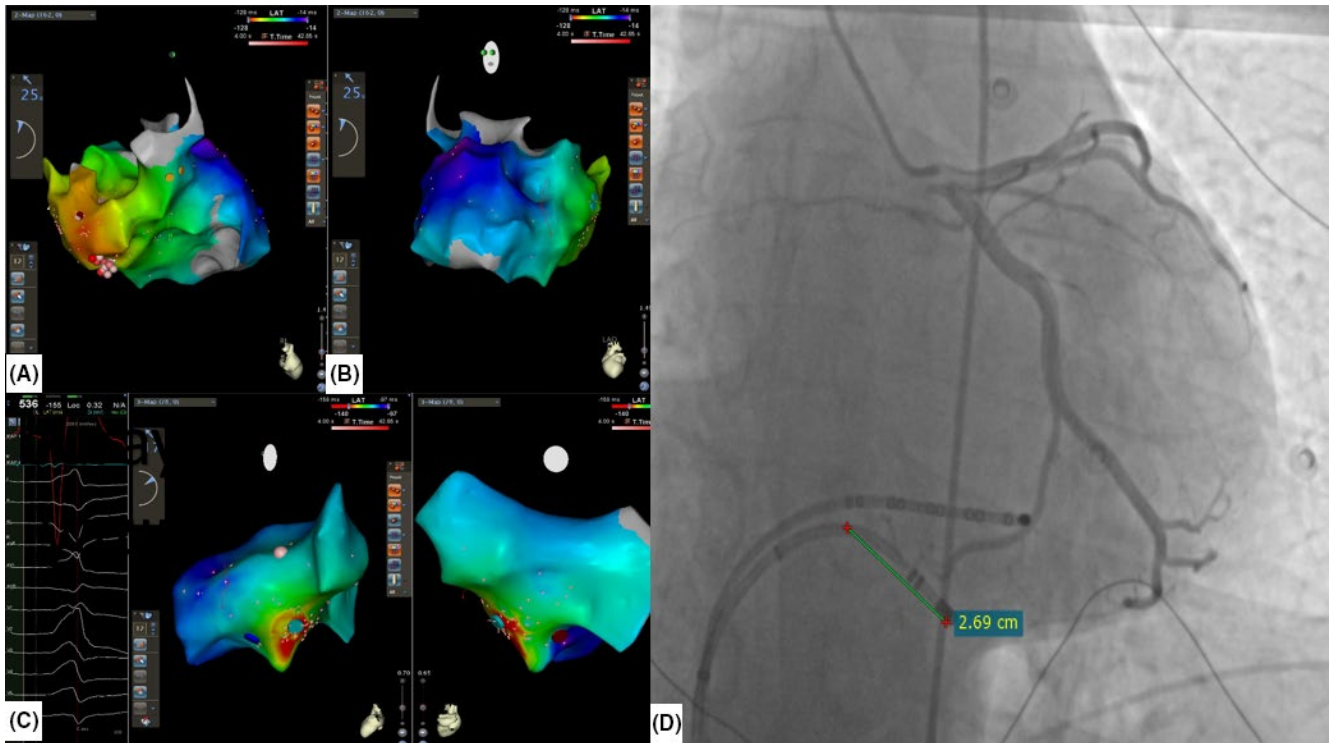


FIGURE 1 A, Mapping image of left ventricle. B, mapping image of aortic cusp. C, mapping image of coronary sinus and middle cardiac vein (MCV). D, Angiographic cine image of successful ablation site, distance is shown between MVC ostium (approximately) and tip of ablation catheter

not benefited from medication, including beta-blocking agents and calcium channel blockers. His echocardiography showed reduced left ventricular function with an ejection fraction of 45%, normal right ventricular and heart valve functions. During his rhythm holter monitoring, there were frequent unifocal PVCs and nonsustained ventricular tachycardia episodes with the same morphology. He underwent an electrophysiology exam after 6 hours without food. Mapping and ablation were performed using a 3.5-mm open-irrigated-tip catheter and CARTO 3 system (ThermoCool, Biosense Webster, Diamond Bar, CA). Morphology of PVCs in the 12-lead electrocardiogram (ECG) showed right bundle branch block and superior axis, deep S wave in V6 with maximal deflection index 0.62, and a pseudo delta wave of 40 msn (Figure S1). Left ventricle was mapped due to R wave in V1 and negative deflection in inferior leads on ECG, and the earliest site of activation could not have been identified during PVCs (Figure 1A), while activation was also late in aortic cusps (Figure 1B). And then, we continued mapping in the right site. Coronary sinus (CS) catheter proximal segment recordings showed early activation, and therefore, the mapping catheter advanced through to CS first and later to MCV (Figure 1C). Nearly 2 cm beneath the MCV ostium, the electrical activity during PVC was 40 millisecond early according to surface ECG; additionally, a late potential during sinus rhythm and a discrete prepotential preceded the PVC was detected (Figure 2). High-output pace mapping failed due to the absence of capture in this region. Before ablation, coronary artery proximity of the ablation catheter was shown with angiography

(Figure 1D). The PVCs and late potentials during sinus rhythm disappeared after 5-seconds of RF (Figure S2). In order to avoid MCV injury and perforation, the energy was applied at 30 watts for 30 seconds. Impedance dropped from 140 Ω to 120 Ω . After a 10-minute waiting period, the PVCs returned, and a second burst of energy was delivered close to the first lesion for 60 seconds at 30 Watts. A few additional applications were made adjacent to the second lesion. After a 30-minute waiting period, the procedure was terminated successfully. No recurrence was seen in the outpatient check-ups during the next 6 months.

3 | DISCUSSION

Among cardiac crux-originated ventricular arrhythmias (VAs), there are very few cases that have successfully ablated inside the MCV. Most of the time, PVCs originating from this region require epicardial intervention. Kawamura et al. differentiated the cardiac crux into the apical and basal regions.² They defined basal crux as successful ablation site in the proximal coronary sinus (CS) or in the proximal MCV within 2 cm of the MCV ostium. They commented that PVCs originating from the apical part showed right bundle branch block, superior axis, and deep S wave in V6, while those from the basal part had left bundle branch block, early transition in V2, and positive deflection in V6. In the same study, the ablation of apical crux VAs inside the MCV failed in seven out of nine patients, while in the remaining two patients VAs recurred



FIGURE 2 Electrical activation at the successful ablation site. Late potential is shown by red arrow, discrete prepotential is shown by blue arrow, the absence of late potential after single premature ventricular contraction (PVC) is shown by white arrow, occurrence of late potential after couplet PVCs is shown by yellow arrow

following their procedures. PVCs originating from basal crux were successfully ablated 2 cm from the MCV ostium. In another case by Larroussi et al., a fast idiopathic ventricular tachycardia originating from the apical crux could have been ablated via epicardial intervention.³ In a recently published article, Aras et al. defined precordial breaking sign for PVCs originate from the posterior coronary venous system.⁴ ECG morphology of our case showed precordial breaking sign as they described. Additionally, the ECG features of our case were similar to the previously described apical crux-based PVCs ECG. Although the successful ablation site is 2 cm below the (MCV) ostium suggested that the origin of the PVC was the apical crux region, we could not clearly confirm this apical region by visualization of CS and MCV with contrast injection. Therefore, we could not identify if the origin of the PVCs were basal crux or apical crux. However, our case, if the origin of PVCs is assumed to be the apical crux, is one of the rare cases in the current literature that was successfully ablated inside the MCV. Although pace mapping failed because of the absence of capture, electrical activity during PVC was early at the successful ablation site. Also, the occurrence of a late potential after sinus rhythm and an early discrete prepotential preceding the PVCs was promising. Tada et al. demonstrated the co-occurrence of late potential and prepotential could help predict the location of the ablation site in aortic cusp PVCs.⁵ Their study revealed that the presence of a

late potential after sinus beat in ablation site was more related to the success of the ablation than the presence of a prepotential. Conversely, Hachiya et al., showed that discrete prepotentials on the RF application site are a strong predictor of successful ablation for aortic cusp PVCs.⁶ In this paper, the authors emphasized that as the discrete potential is a tiny electrogram, capture with pacing in the location of discrete potential is difficult, even with high-output pacing. Additionally, high-output pace mapping causes the capture of a large area and a poor pacing score. Therefore, they suggested that in case of aortic cusp PVCs, the occurrence of discrete prepotentials is a better predictor than pace matching for successful ablation. In our case, we also failed to capture pace mapping in the location of discrete prepotential. The occurrence of a delayed potential and an early discrete prepotential together in a PVC originating in the cardiac crux region inside of MCV has not been reported before. These potentials have been shown in aortic cusp PVCs, papillary muscle, and mitral annulus originated PVCs or VAs and left-sided concealed accessory pathways.⁷ There are various opinions in the literature regarding the mechanism of those potentials. One of the hypotheses is that these potentials come from remnant embryonic tissue, which acts as slow conduction properties. This, also named dead-end track, causes arrhythmias with a mechanism of reentrant, and non-reentrant circuits or ectopic focal triggered activities.⁷ According to that hypothesis,

these remnant tissues also play a role in accessory pathways (AP)-related tachycardia's. Co-occurrence of AP and PVCs is common and, in some cases, disappearing of PVCs after ablation of AP also supports this idea. Delta wave-like QRS onset during PVC is assumed to be another supportive finding. However, according to this theory, dead-end tract lies from the conduction system passing the endocardial surface of the left ventricular septum, mitral annulus, and aortomitral continuity through to aortic root. Our case differs from this hypothesis in the identification of the cause of cardiac crux localization. Another explanation is that myocardial fibers, independent of the conduction system, which is also an embryonic remnant, could lead to these kinds of potentials. In our case, the occurrence of delayed potentials during sinus beat, and discrete prepotential in front of PVC could be explained with the occurrence of myocardial fiber. Occurrence of potentials after the sinus beat was very late, that finding suggested us aforementioned myocardial fiber could have slow conduction properties and sometimes could lead conduction block. In light of that hypothesis, the absence of delayed potentials after single PVC but not after a pair of PVC could be explained with the existence of a conduction block inside of the myocardial fiber. Also, the disappearing of delayed potentials after ablation supports whole scenario.

In conclusion, in cases of cardiac crux-originated VAs, seeking a late potential and/or an early discrete prepotential at the MCV can help locate the site of ablation, and increase the rate of success.

CONFLICT OF INTERESTS

The authors declare no conflict of interests for this article.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

How to cite this article: Gulsen K, Bayrak F, Akdemir B, et al. Co-occurrence of late potential and discrete prepotential on ablation site in a case of cardiac crux located premature ventricular complexes successfully ablated within the middle cardiac vein. *J Arrhythmia*. 2019;35:550–553. <https://doi.org/10.1002/joa3.12190>