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CASE REPORT: CLINICAL CASE

Noninvasive 3D Mapping and Ablation of Epicardial Premature Ventricular Contractions From the Endocardial Aspect of the Left Atrial Appendage

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ABSTRACT

Ablation is an established treatment for ectopy originating from the left ventricle (LV). We report on a case of noninvasive 3-dimensional mapping locating the origin precisely in the epicardial LV summit area. However, after failed attempts from LV and epicardially, ablation via the left atrial appendage was finally successful. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2020;2:1776–80) © 2020 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

An 18-year-old female patient with a structurally normal heart and frequent monomorphic premature ventricular contractions (PVCs) and episodes of nonsustained ventricular tachycardia (VT) since childhood was admitted for catheter ablation because for frequent palpitation refractory to medical therapy.

LEARNING OBJECTIVES

- To use personalized 3D information from noninvasive 3D mapping and a CT roadmap that locates the origin of ventricular arrhythmias and guides the operator to find the optimal access to an ablation target.
- To consider ablation through the LAA to reach the epicardium of the LV summit, when endocardial ablation fails and/or an epicardial approach seems difficult.

PAST MEDICAL HISTORY

The patient had been treated for several years with beta-blockers, as well as flecainide.

DIFFERENTIAL DIAGNOSIS

Screening for cardiomyopathy was negative.

INVESTIGATIONS

Before the admission, a 24-h Holter showed a high PVC burden (42%) and several episodes of nonsustained VT despite antiarrhythmic therapy with metoprolol 100 mg once daily and flecainide 150 mg

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once daily. The electrocardiogram (ECG) on admission is shown in Figure 1.

A contrast-enhanced computed tomography (CT) scan excluded significant coronary artery disease or anomaly and was also used to reconstruct a 3-dimensional (3D) model of the heart on the electroanatomical mapping system (CARTO 3, Biosense Webster, Brussels, Belgium) for 3D image integration to guide as a roadmap.

MANAGEMENT

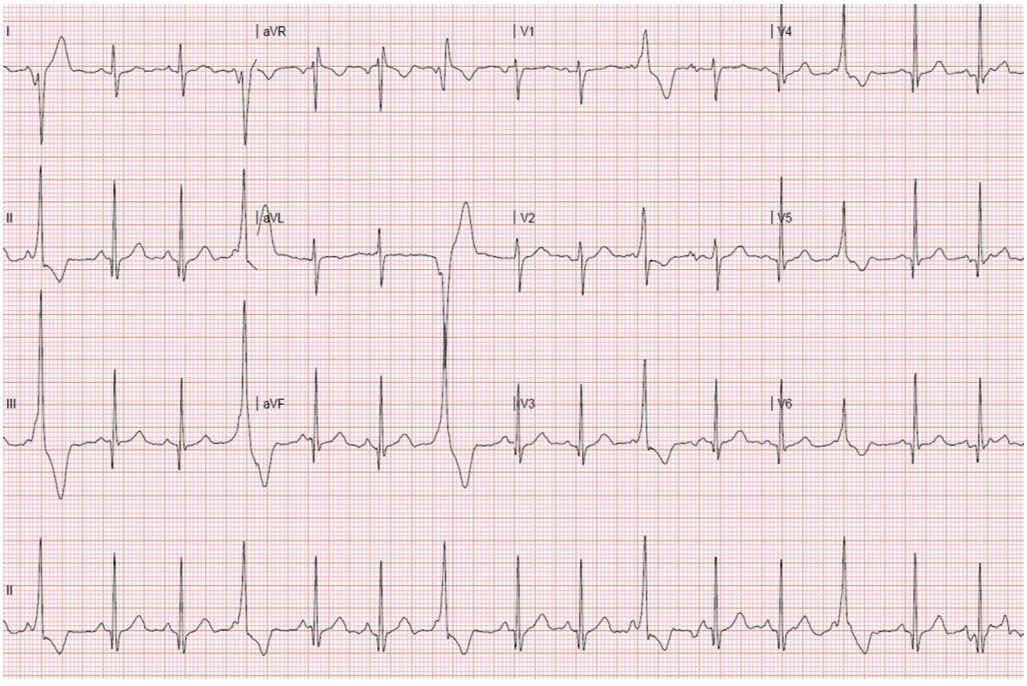
NONINVASIVE MAPPING. Before the invasive procedure, the patient underwent noninvasive mapping using the VIVO system (Catheter Precision, Inc, Ledgewood, New Jersey) (1,2). On the day before the procedure, a 12-lead Holter (Mortara, Hill-Rom, Chicago, Illinois) was fitted for 14 h that recorded the clinical PVC morphology. To precisely locate the PVC origin, the exact position of the ECG leads on the patient's chest was recorded using a 3D camera. All data (ECG, patient-specific 3D heart and torso model, plus 3D photograph of ECG positions) were combined to create a noninvasive activation map that identified the earliest point of activation during PVC. VIVO determined the PVC origin at the epicardial aspect of the left ventricular (LV) summit, right above the mitral valve annulus (Figure 2).

INVASIVE ABLATION PROCEDURE. At the beginning of the ablation, the patient was in sinus rhythm with frequent monomorphic PVCs. After gaining venous and arterial femoral accesses under ultrasound guidance, a 3.5-mm irrigated tip ablation catheter (NAVISTAR ThermoCool, D/F SF ST, Biosense Webster) was advanced retrogradely and a fast anatomical mapping of the descending aorta and aortic arch was performed in combination with 3D image of the pre-acquired CT scan (Figure 3). Subsequently, a fast anatomical mapping and pace-map of the left ventricular outflow tract (LVOT) and the LV summit was carried out (PASO module, CARTO 3 V7, Biosense Webster). The best pace-map match from the endocardial aspect of the LV summit was 91% without any real prematurity of the local bipolar signal. Therefore, the distal coronary sinus (CS) was mapped to get better access to the corresponding epicardial LV summit region, which

ABBREVIATIONS AND ACRONYMS

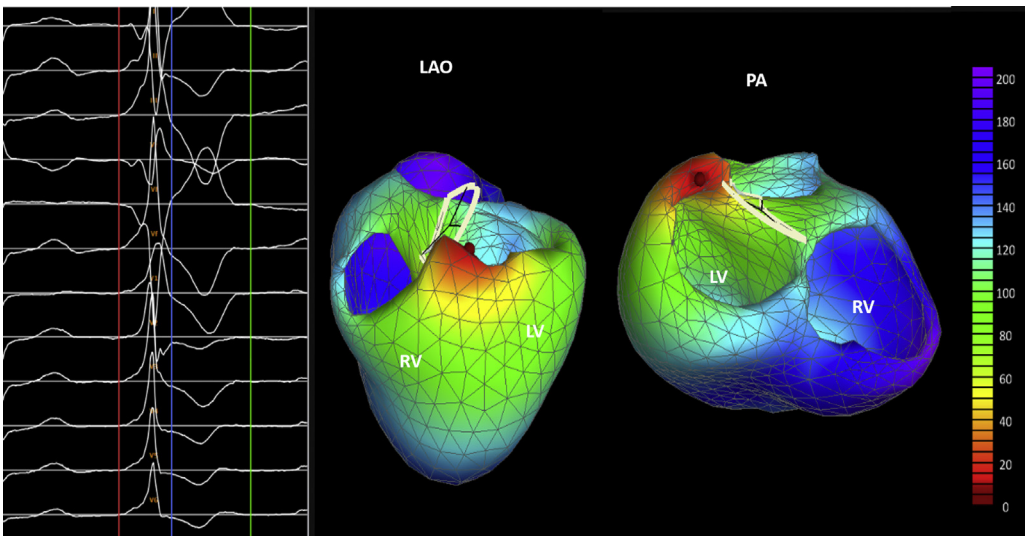
3D = 3-dimensional
CS = coronary sinus
CT = contrast-enhanced computed tomography
ECG = electrocardiogram
LAA = left atrial appendage
LV = left ventricle
LVOT = left ventricular outflow tract
PVC = premature ventricular contractions
VT = ventricular tachycardia

FIGURE 1 12-Lead Electrocardiogram



12-lead electrocardiogram in sinus rhythm with monomorphic premature ventricular contractions (PVCs) in a trigemini pattern (25 mm/s, 10 mm/mV).

FIGURE 2 Pre-Procedure Noninvasive 3D Mapping of the PVC Origin Using VIVO



The premature ventricular contraction (PVC) origin **(red tag)** is shown in left anterior oblique (LAO) and posterior-anterior (PA) views. 3D = 3-dimensional; LV = left ventricle; RV = right ventricle.

resulted in a pace-map match of 96%. Radiofrequency ablation at this site (20 W, 8 ml/min) resulted in initial suppression of the PVCs for the duration of the energy delivery, which, however, recurred despite several ablation attempts.

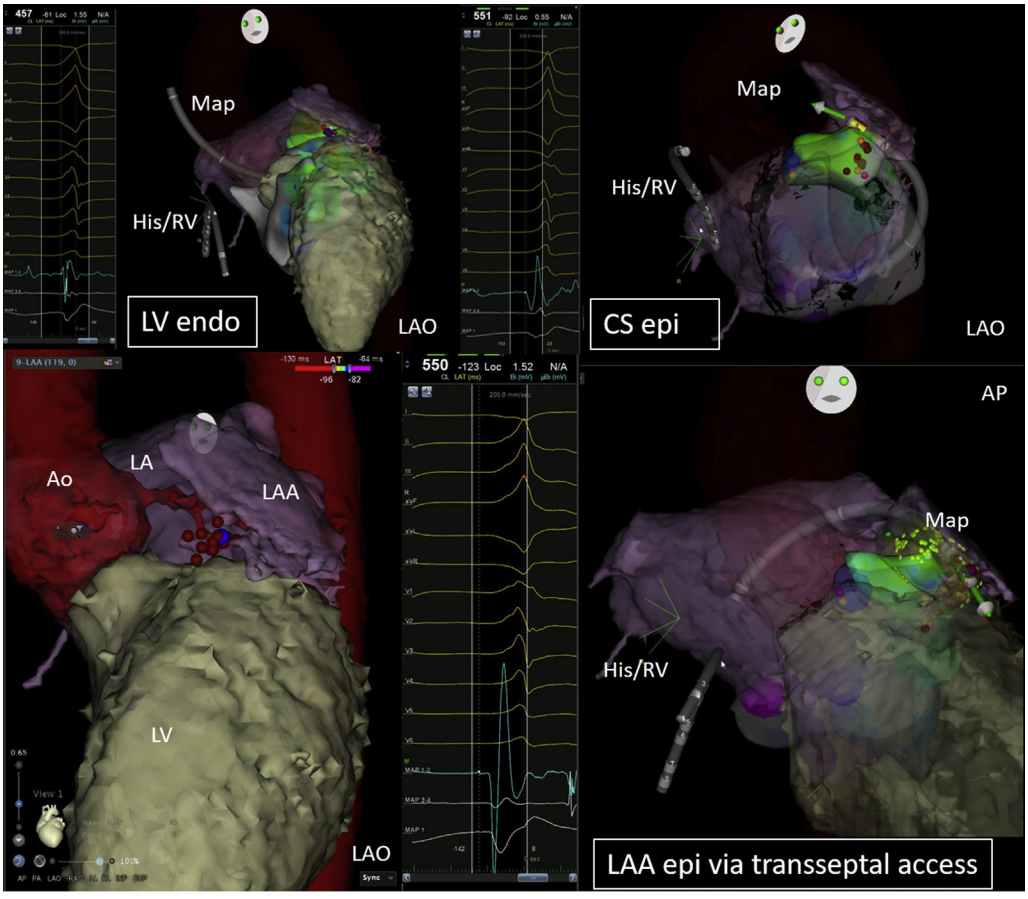
When reviewing the entire anatomy, we noticed that the left atrial appendage (LAA) was overlapping the area of earliest activation and best pace-map match in the LV and CS. Given the suspicion of a "true" epicardial origin of the clinical PVC not reachable via the LV endocardium or the distal CS, the overlapping LAA would potentially make a subxiphoid epicardial approach difficult. We therefore attempted an endocardial ablation via the LAA using a transseptal access. Mapping the anterior-inferior aspect of the LAA, discrete premature signals with earliest local activation −41 ms before the QRS complex onset were observed (**Figure 3**). Three radiofrequency ablations (20 W, 8 ml/min) at this site resulted in the instant suppression of the PVC; however, with recurrence within a few minutes. Thus, radiofrequency energy was carefully increased up to 35 W in the presence of good contact force toward the LV epicardium, with final complete elimination of the clinical PVC. No further PVC or VT was inducible during a waiting time of 45 min (total procedure duration 238 min, total fluoroscopy time 4 min 15 s).

DISCUSSION

Ablation of ventricular arrhythmias should be considered in patients without structural heart disease when symptomatic or when medications are ineffective (3). In this case, the patient was very symptomatic with a high burden of PVC despite optimal antiarrhythmic therapy. The LV summit is the most superior epicardial region of the LV bounded by the left anterior descending and left circumflex coronary artery. It accounts as an origin for up to 14.5% of LV arrhythmias. Ventricular arrhythmias originating from the LV summit are known to be difficult targets for ablation; the reasons being multifactorial, including submural or subepicardial focal origins that are difficult to reach endocardially and the close proximity to coronary arteries and surrounding fat precluding successful subxiphoid epicardial ablation (4,5). Attempts at ablation of this area have often been carried out via surrounding structures including septal leftward aspects of the right ventricular outflow tract, aortic cusp region, great cardiac veins, and basal LV endocardium (6,7). Ablation of an arrhythmia focus in this area via the LAA has been described in only a few reports (8-10).

In our case, 12-lead ECG morphology of the ectopic focus was suggestive of LV summit origin with a nonspecific bundle branch block morphology with

FIGURE 3 **Invasive Electrophysiologic Study**



Left lower panel depicts the 3D roadmap and the relationship of the left atrial appendage (LAA) over the left ventricular (LV) summit area in left-anterior-oblique (LAO) projection. Unsuccessful mapping and ablation sites are depicted in both top panels with the mapping catheter displayed in position for the best LV endocardial site (retrograde access via aorta [Ao]) and inside the distal coronary sinus (CS) with their respective electrograms. The **right lower panel** demonstrates the electrogram at the finally successful ablation site displaying a discrete prepotential and the transseptal catheter position inside the LAA overlapping the epicardial LV summit. 3D = 3-dimensional; His/RV = octapolar catheter in the right ventricle and His recording region.

initial R-wave in lead V_1, negative polarity with Q-wave in lead I, and QS pattern in lead aVL. The noninvasive pre-procedural 3D mapping with VIVO confirmed this location taking the individual 3D anatomy of the patient into account. The key information, however, came from the 3D reconstruction of the pre-procedure CT scan that served as a roadmap. It demonstrated the close relationship of the LAA overlapping the LV summit area with clear depiction of the triangle between the coronary arteries.

The combination of both roadmap and noninvasive 3D mapping assisted in understanding the individual anatomy and finally guided the operator to the optimal site to achieve complete elimination of the PVC located in the epicardial LV summit. To the best of our knowledge, this is the first reported case describing this approach.

In addition, using these advanced mapping techniques potentially facilitates a number of aspects of such procedures: 1) reduction of procedure duration because the 3D activation map can be done noninvasively on the ward without exposing the patient to the time-consuming sequential mapping (which may be especially relevant in patients with rare PVCs); 2) reduction of radiation exposure, as the 3D roadmap can be merged using the aortic arch as a

reference; and 3) potential to reduce periprocedural complications by avoiding the coronary arteries and/or the need of a subxiphoid epicardial approach.

Interestingly, it took 4 radiofrequency applications in the LAA with titration of energy to up to 35 W (applied for max 120 ms) and good contact force to persistently eliminate the PVC, creating finally an adequate transmural lesion through the wall of the LAA to reach the epicardially located origin. The risk of perforation must be carefully considered while ablating in the LAA and careful energy titration is recommended. Because of the full heparinization of our patient, the alternative subxiphoid epicardial approach (with its potential for complications and added financial burden from longer post-procedural hospitalization) would have been needed to be carried out as a second procedure.

FOLLOW-UP

After ablation, the patient remained in sinus rhythm and no further episodes of PVCs or VTs were detected on 24-h telemetry. A bedside transthoracic echocardiography excluded any significant pericardial effusion and the patient was discharged off antiarrhythmic medication on the post-procedural day. No relevant PVCs have been since recorded during 48-h follow-up Holter recordings 6 months later, and the patient remained asymptomatic.

CONCLUSIONS

We present a case of a successful ablation of PVC originating from the epicardial LV summit via the LAA. Careful preprocedural planning with noninvasive 3D mapping, appropriate 3D roadmap imaging, and detailed electroanatomical mapping all aided this successful outcome.

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