Intracoronary artery mapping and 3-dimensional visualization of the coronary arteries with a 0.014 inch guidewire in catheter ablation of left ventricular summit premature ventricular contractions

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Introduction

Despite the advances in mapping and ablation technique, catheter ablation for ventricular arrhythmias (VA) originating from the left ventricular (LV) summit remains challenging. Intracoronary artery mapping with a guidewire has been reported to be useful as a guide of radiofrequency catheter ablation and intracoronary ethanol ablation of LV summit VAs. Three-dimensional (3D) visualization of the coronary arteries by merging 3D images of computed tomography (CT) and magnetic resonance imaging (MRI) into 3D electroanatomic maps has been used for mapping of LV summit VAs. However, the utility and feasibility of coronary artery visualization by 3D mapping system with a guidewire has not been reported.

We present a case in which intracoronary artery mapping and visualization of the coronary arteries by 3D mapping system with a guidewire was useful for catheter ablation of premature ventricular contractions (PVCs) triggering recurrent ventricular fibrillation (VF).

KEY TEACHING POINTS

- Intracoronary artery mapping and visualization of the coronary arteries by 3-dimensional (3D) mapping system with a guidewire was useful for catheter ablation of premature ventricular contractions from the left ventricular summit.
- Using this 3D mapping, the distance from the coronary artery to the ablation site can be measured accurately.
- Using the 0.014 inch wire advanced into the left coronary arteries, unipolar mapping can be performed from the left coronary artery.

Case report

A 51-year-old woman with a history of hypertension and stress-induced cardiomyopathy was admitted owing to an aborted cardiac arrest due to VF. Echocardiogram showed LV ejection fraction of 51% with hypokinesis of the inferior and apical wall. Cardiac MRI revealed abnormal LV wall motion and delayed contrast enhancement, with findings suggesting a medium-sized subendocardial/nontransmural infarct involving the inferior apical, inferior septal, and apical wall. Coronary angiogram revealed no significant stenosis. After intensive care management, she completely recovered without any brain damage. She was discharged after an implantation of a dual-chamber implantable cardioverter-defibrillator (ICD). Two months later, she was readmitted for frequent VF episodes. Her ICD interrogation showed multiple VF episodes triggered by PVCs with a coupling interval of 570 ms (Figure 1). Twelve-lead electrocardiography showed frequent unifocal PVCs occurring after the T wave of the previous beat (Figure 1). The PVCs exhibited a left bundle branch block configuration and an inferior axis QRS morphology with an early transition (< lead V_3) and maximum deflection index of >0.54.[1] These electrocardiogram characteristics suggested an epicardial origin in the LV outflow tract (LVOT). The coupling interval of the PVCs was constantly 530 ms and it was similar to that of the PVC that triggered the VF. The patient underwent an electrophysiology study and catheter ablation for the PVCs.

The mapping and *ablation* procedures were guided by the EnSite *Precision* system (Abbott Laboratories, Abbott Park,

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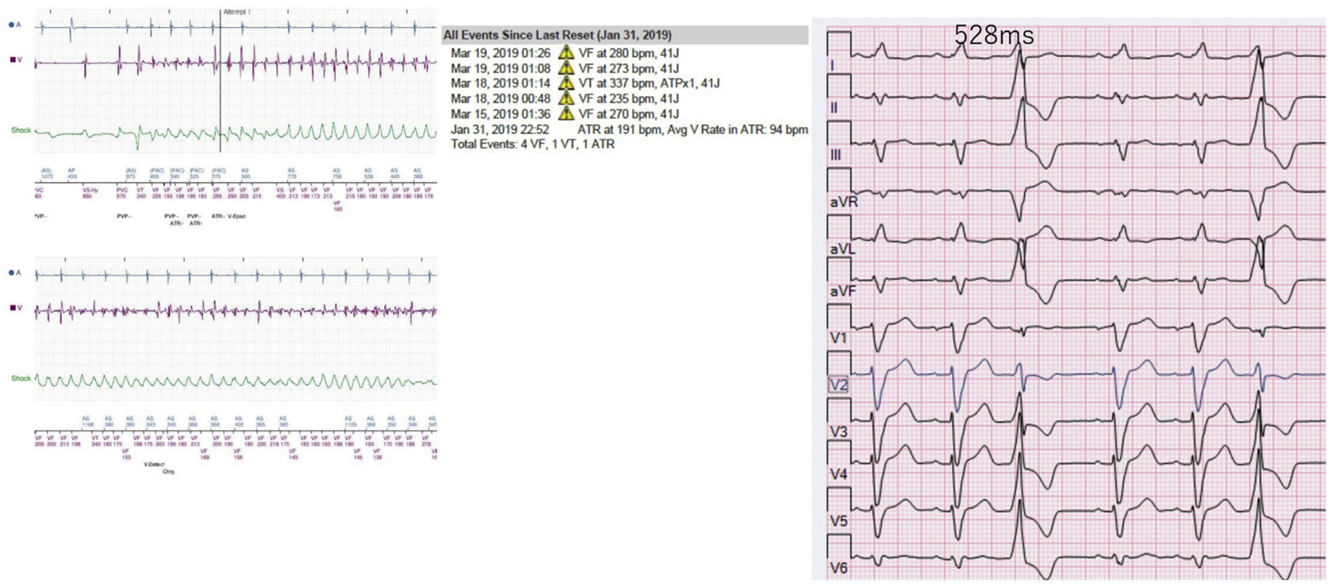

Figure 1 An implantable cardioverter-defibrillator interrogation revealing a ventricular fibrillation triggered by a premature ventricular contraction.

IL). During the electrophysiology study, the patient was heparinized and the activated clotting time was maintained between 250 and 300 seconds. Right ventricular and LV mapping was performed with the Advisor HD Grid Mapping Catheter and the TactiCath Quartz ablation catheter (Abbott Laboratories, Abbott Park, IL, USA). Comprehensive mapping in the right ventricular outflow tract (RVOT), aortic sinus cusps, and aortomitral continuity revealed the earliest ventricular activation preceding the QRS onset by 21 *ms* in the left coronary cusp. An Inquiry LumaCath Fixed Diagnostic Catheter (St. Jude Medical, St Paul, MN) was placed in the coronary sinus through the right femoral vein. A

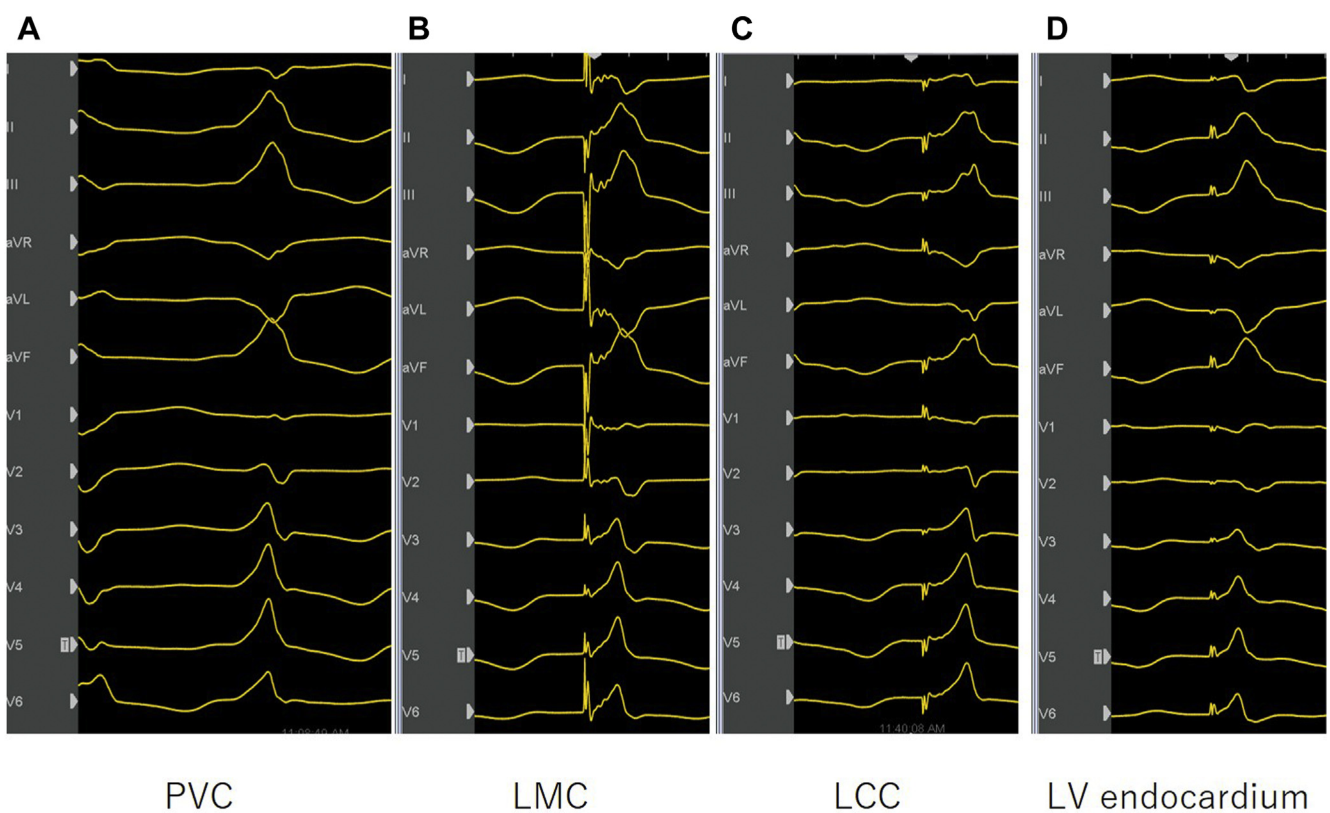

Figure 2 Twelve-lead electrocardiograms exhibiting a premature ventricular contraction (PVC; **A**) and pace maps from the distal left main coronary artery (LMC; **B**), left coronary cusp (LCC; **C**), and left ventricular (LV) endocardial site below the LV summit (LV endocardium; **D**). Note that the pace map from the LMC exhibited a perfect match to the PVC, whereas that from the successful ablation site in the LV endocardium did not.

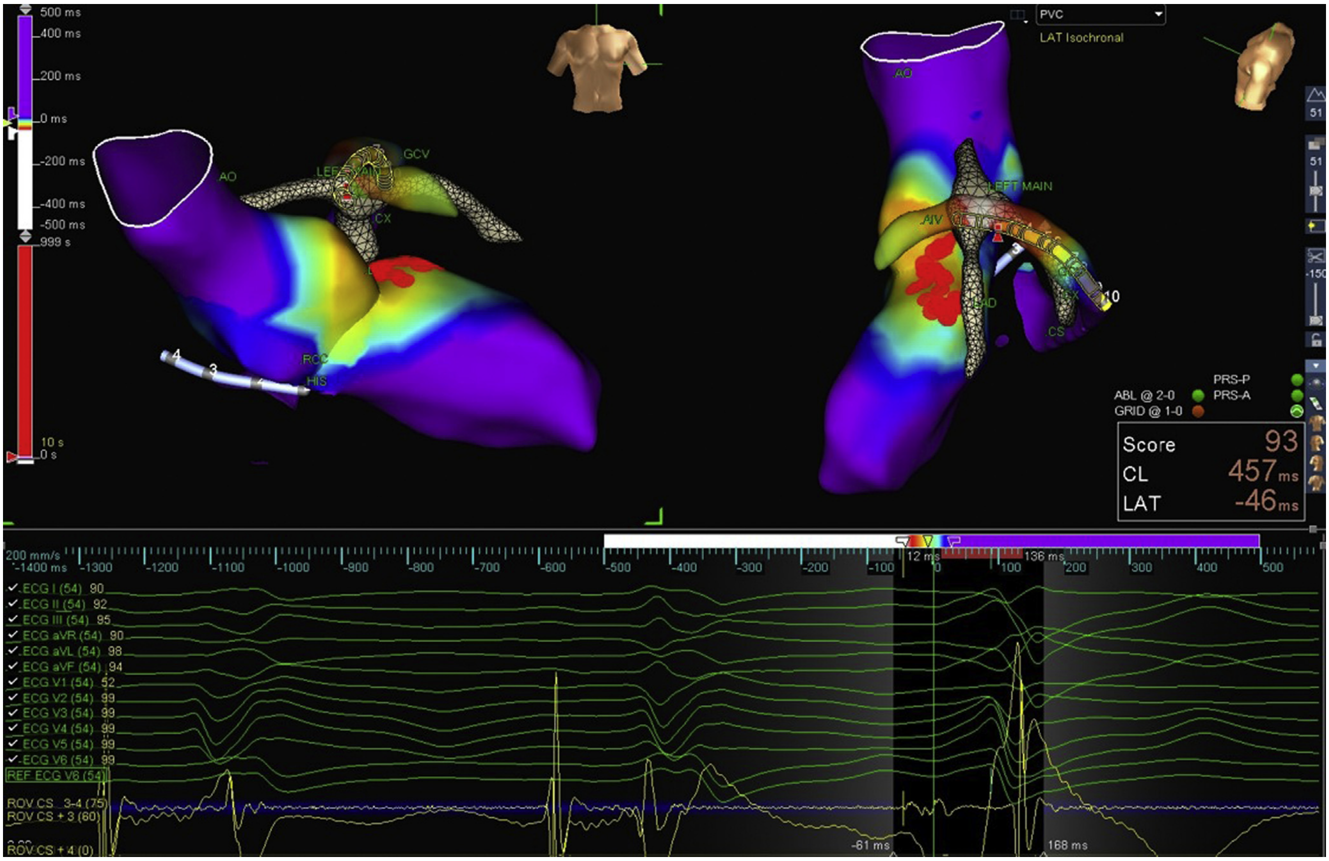

Figure 3 Activation maps exhibiting the anatomical structures such as the left ventricle (LV), great cardiac vein (GCV), anterior interventricular vein (AIV), and left coronary cusp and successful ablation sites ***(red tags)***. The earliest ventricular activation ***(white area)*** was identified at the GCV-AIV junction near the distal left main coronary artery (LMC). The earliest ventricular activation, preceding the QRS onset by 46 ms, was recoded at the GCV-AIV junction (*lower half of image*).

0.014 inch VisionWire guidewire (Biotronik SE & Co. KG, Berlin, Germany) was then advanced through the inner lumen of the coronary sinus catheter into the great cardiac vein (GCV) and anterior interventricular vein (AIV). The guidewire's proximal end was connected to a channel of a conventional electrophysiology amplifier and monitoring system (*Prucka CardioLab EP system*, GE Healthcare, Waukesha, WI). The earliest ventricular activation, preceding the QRS onset by 46 ms, was recoded at the GCV-AIV junction. Pacemapping from GCV-AIV was unsuccessful. Subsequently, left coronary angiography was performed with a 5F JL4 angiographic catheter, revealing no angiographic abnormalities. The 0.014 inch VisionWire was advanced through this angiographic catheter into the left coronary arteries, and unipolar mapping was performed during the PVCs in the proximal left anterior descending coronary artery (LAD) and left circumflex coronary artery (LCX). The left main coronary artery (LM), proximal LAD, and LCX were reconstructed in the EnSite 3D mapping. The 0.014 inch VisionWire was carefully advanced to mid LAD and LCX multiple times to obtain accurate anatomical information of those structures. Unipolar mapping with a guidewire in the left coronary arteries revealed the earliest ventricular activation preceding the QRS onset by 36 ms at the LM bifurcation, where pacemapping just above the pacing threshold to capture the local tissue achieved a perfect match to the QRS of the PVC (Figure 2). Using this 3D mapping, the distance from distal LM to the earliest activation site at the GCV-AIV junction was less than 5 mm (Figure 3). We concluded that the origin of the PVC was intramural below the epicardial LV summit. Owing to proximity to the LM, catheter ablation from the GCV-AIV junction was abandoned, and catheter ablation by an anatomical approach from a remote site was considered. Because the LV endocardium was the closest to the earliest ventricular activation site and showed the best pacemapping among the LCC, LV endocardium, and RVOT, radiofrequency ablation was started from the area. A long duration (≥3 minutes) of radiofrequency application with a power titrated up to 40 W was delivered at multiple LV endocardium sites opposite to the earliest epicardial ventricular activation site, resulting in an elimination of the PVCs. The distance from the earliest local ventricular activation site in the GCV/AIV to the successful ablation site was 8–15 mm (Figure 3). During 12 months of follow-up, the patient has been free from any ICD therapies, and the PVC burden has been reduced from 10% to <1%.

Discussion

The *LV summit* is a common site of idiopathic epicardial VA origins. Anatomically, the LV summit is defined based on fluoroscopy and coronary angiography as the region on the epicardial surface of the LV near the bifurcation of the LM that is bounded by an arc from the LAD superior to the first septal perforating branch anterior to the LCX laterally. The GCV bisects the LV summit into an area lateral to this structure, which is accessible to epicardial catheter ablation through a transpericardial approach (the accessible area) and a superior region that is inaccessible to catheter ablation because of the proximity of the coronary arteries and the thick layer of epicardial fat that overlies the proximal portion of these vessels (the inaccessible area). The inaccessible area is a triangular area of the LV epicardial surface bounded by the LAD, LCX, and GCV, and is also called the triangle of Brocq and Mouchet. LV summit VAs are most commonly ablated within the GCV or AIV.[2,3] *In this case, a perfect pace map and the earliest ventricular activation* were recorded in the distal LM and GCV-AIV junction, respectively, suggesting that the PVCs should have originated from the LV summit. However, catheter ablation from the GCV-AIV junction was abandoned owing to the close proximity of the presumed ablation site to the proximal left coronary arteries(<5 mm).[4] Radiofrequency catheter ablation was successful at a remote site on the endocardial LVOT that was opposite to the site of the earliest epicardial ventricular activation (anatomical approach).

When LV summit VAs originate from the inaccessible area, catheter ablation by an anatomical approach may be successful from adjacent structures such as the LCC, LVOT endocardium, or septal RVOT[5] with a careful assessment of a proximity of a presumed ablation site to the coronary arteries. *A c*oronary angiogram is a gold standard for such an assessment to avoid potentially catastrophic coronary injury. However, it is often difficult to accurately assess a distance between the coronary arteries and a presumed ablation site on 2-dimensional fluoroscopic images. A 3D visualization of the coronary arteries by merging 3D images of CT and MRI into 3D electroanatomical maps has been used for the mapping of LV summit VAs. However, there are a couple of limitations in this methodology. First, 3D images of CT and MRI are not real-time, and the anatomy may be different at the times of ablation procedure and 3D imaging. Second, when PVC are frequent, it should be challenging to obtain clear 3D images of CT and MRI. Third, on a 3D map, the images of the coronary arteries cannot be used as a point to measure a distance. In this patient, the coronary arteries' real-time images were reconstructed on a 3D electroanatomical map by an intracoronary guidewire mapping. This anatomical mapping also enabled us to accurately measure the distance from the coronary arteries to the presumed epicardial ablation sites (8–15 mm). Although we advanced and pulled back the vision wire multiple times to obtain accurate anatomical information, we acknowledge that the vision wire might not cover the entire lumen of coronary artery. Therefore the possibility of measurement error cannot be ruled out.

Intracoronary artery mapping with a guidewire has been used to guide for catheter ablation and intracoronary ethanol ablation.[6–8] This case report demonstrated that intracoronary artery mapping with a guidewire also could provide a reconstruction of the coronary arteries in a 3D anatomical mapping that was useful for catheter ablation of LV summit VAs.

Conclusion

This case report presented a novel technique to create a 3D reconstruction of the coronary arteries on a 3D electroanatomical map by using an intracoronary artery guidewire. This technique enabled an accurate real-time measurement of a distance between the coronary arteries and presumed ablation sites and was useful for mitigating the risk of injury of the coronary arteries and veins. Further studies are needed to confirm the safety and feasibility of this technique.

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