Intramyocardial radiofrequency ablation of ventricular arrhythmias using intracoronary wire mapping and a coronary reentry system: Description of a novel technique

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Introduction

Ventricular arrhythmias (VA) originating from the left ventricular (LV) summit and intramyocardially within the interventricular septum pose a serious challenge to catheter ablation (CA), as myocardial thickness, epicardial fat, and coronary vessels impede appropriate radiofrequency (RF) energy delivery to the target areas. Several strategies have been proposed to effectively eliminate these VA.[1–5] Nonetheless, each of these is associated with a series of limitations and potential complications, and until now no consensus exists on the optimal ablation strategy for VA originating from these anatomic sites. We describe a novel technique of intracoronary mapping and subsequent intramyocardial ablation using RF ablation delivered through a system routinely used to treat coronary artery chronic total occlusions (ie, Stingray LP Coronary CTO Re-Entry System, Boston Scientific, Marlborough, MA).

Case report

A 48-year-old man with a previous history of nonischemic cardiomyopathy based on a left ventricular ejection fraction (LVEF) of 25% and normal coronary arteries was evaluated in our institution for frequent symptomatic premature ventricular contractions (PVCs) that had been diagnosed 5 years earlier (PVC burden of 35%, n = 43,000). Cardiac magnetic resonance imaging with gadolinium did not reveal

KEY TEACHING POINTS

- Ventricular arrhythmias originating from the left ventricular summit and intramyocardially within the interventricular septum pose a serious challenge to catheter ablation, as myocardial thickness, epicardial fat, and coronary vessels impede appropriate radiofrequency energy delivery.

- Intracoronary mapping and subsequent intramyocardial ablation using radiofrequency ablation delivered through a system routinely used to treat coronary artery chronic total occlusions (ie, Stingray LP Coronary CTO Re-Entry System) is feasible and effective.

- Unlike alcohol injection and coil embolization of septal perforator arteries, intramyocardial RF ablation using this technique is a more localized and limited approach with less risk of complete atrioventricular block and less myocardial injury.

myocardial delayed enhancement and fluorine-18 deoxyglucose positron emission tomography–computed tomography (18F-FDG PET-CT) ruled out active cardiac sarcoidosis. The initial 12-lead electrocardiogram (ECG) showed a PVC with left bundle branch block morphology and inferior axis with early transition in lead V_2. The PVC had a maximum deflection index > 0.55, pseudodelta wave > 34 ms, and intrinsicoid deflection time > 85 ms. These features suggested an epicardial origin in the LV outflow tract (ie, LV summit) (Figure 1A and B). Two previous ablation attempts performed in outside facilities were unsuccessful: the initial CA procedure was undertaken within the coronary sinus (CS), with an earliest site of activation found at the great cardiac vein (GCV) / anterior interventricular vein (AIV)
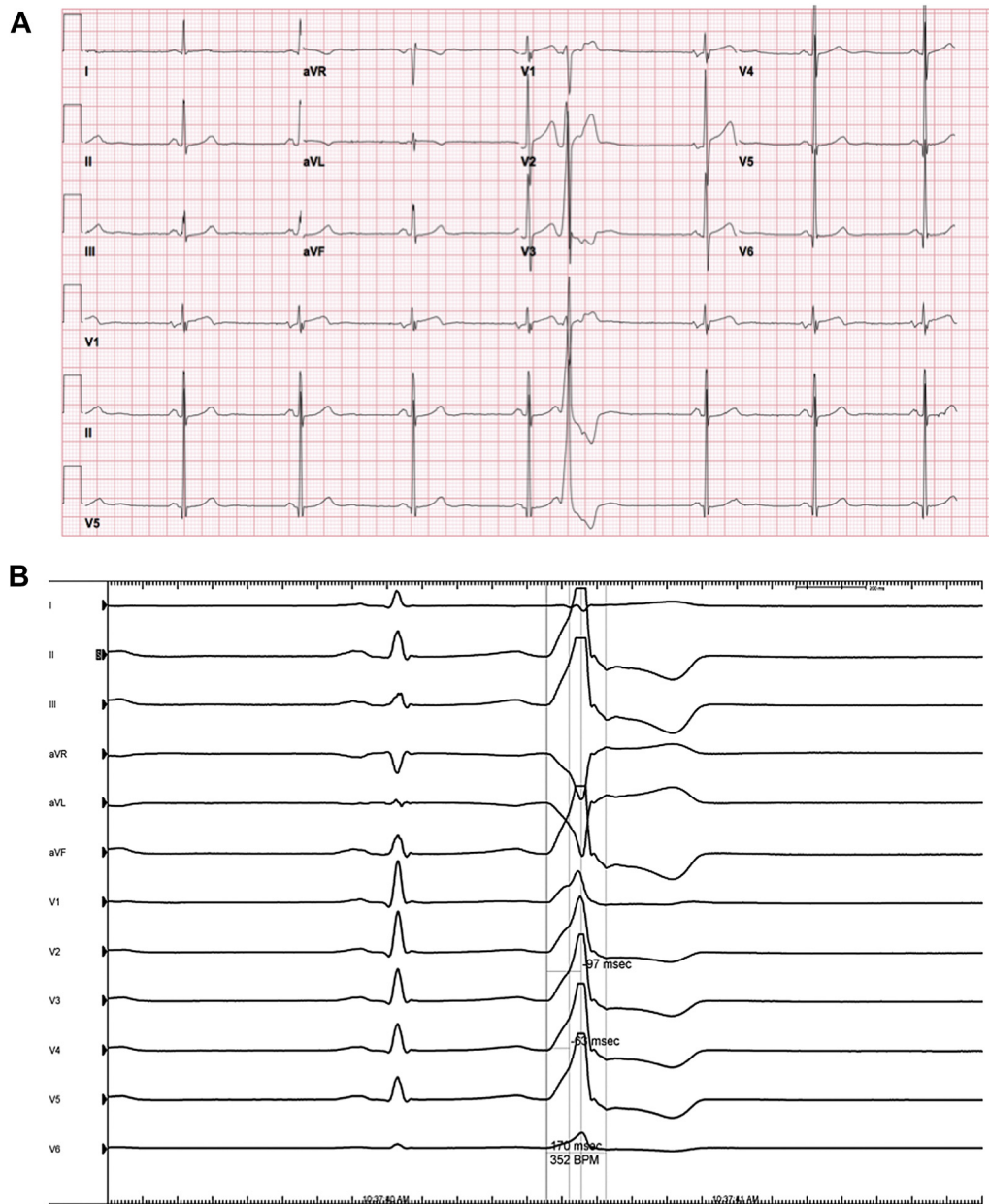
Figure 1 **A:** Standard 12-lead electrocardiogram (ECG) (paper speed 25 mm/s) showing premature ventricular contraction (PVC) with left bundle branch block morphology and inferior axis with early transition in precordial leads (ie, lead V_2). Notice the very positive deflection in inferior leads and the QS complexes in leads aVR and aVL. All these finding suggest a left ventricular outflow tract (LVOT) origin. **B:** A 12-lead ECG (paper speed 100 mm/s) demonstrating similar PVC morphology except for an earlier transition in precordial leads owing to slightly different lead placement during ablation procedure. Maximum deflection index (0.57), pseudodelta wave 63 ms, and intrinsicoid deflection time of 97 ms are all suggestive of an LVOT epicardial origin (LV summit). However, an aVL/aVR Q ratio < 1.6 (patients' aVL/aVR Q ratio was 1.2) and an R-wave ratio III/II < 1.4 (patients' R-wave ratio III/II was 1.1) are predictive of an origin in the inaccessible area of the LV summit.

junction. Nonetheless, RF delivery was limited by high impedance measured at the catheter tip. The second procedure used an epicardial approach, but despite 25 minutes of total RF time, the PVC could not be eliminated, most likely because of thick epicardial fat. Owing to persistent symptoms, impaired LVEF, and high PVC burden despite medical treatment with high-dose beta blockers (ie, metoprolol 200 mg daily), the patient was referred for a third attempt at our arrhythmia center.

During initial electrophysiology study, right ventricular and LV mapping was performed with a 3.5-mm Navistar

Thermocool DF mapping ablation catheter (Biosense-Webster, Diamond Bar, CA). The right ventricular outflow tract, sinuses of Valsalva, and the aortomitral continuity were mapped with an earliest activation point observed in the left coronary cusp (−2 ms). Subsequently, the mapping catheter was advanced into the coronary sinus (CS) demonstrating earlier activation times at the GCV/AIV junction (−22 ms) and in the AIV (−24 ms), with a perfect pace map in this last location (12/12) (Figure 2). Coronary angiogram was performed prior to ablating, revealing close proximity between the ablation catheter tip and the proximal left anterior descending

(LAD) coronary artery (Figure 2). Consequently, RF ablation from the CS was avoided. Since the patient had previously undergone a failed epicardial ablation, and taking into consideration that the 12-lead ECG revealed a PVC with an aVL/aVR Q ratio < 1.6 (patients' aVL/aVR Q ratio was 1.2) and an R-wave ratio III/II < 1.4 (patients' R-wave ratio III/II was 1.1), which indicated that the PVC origin was in the inaccessible area of the LV summit, epicardial access was not performed.

Interestingly, coronary angiography also revealed that the earliest site of activation was close to the first septal perforator artery (Figure 2). Intracoronary mapping and potential ablation using alcohol injection or coil embolization of the septal perforating artery was considered. A 0.014-in Vision guidewire (Biotronik SE&CO KG, Berlin, Germany) was

advanced into the first septal perforator and a confirmed early activation at −24 ms was found. Given the large caliber of the first septal perforator and the potential extensive myocardial damage if this were completely occluded, a smaller more proximal septal branch was selected for coil embolization. However, the PVC could not be eliminated with this approach. Subsequently, the intracoronary mapping guidewire was then repositioned in the first septal perforator, and a Stingray LP device (Boston Scientific, Marlborough, MA) (Supplemental Figure 1) was advanced into the proximal portion of this branch (Figure 3A). Briefly, this device has a self-orienting balloon with 180° opposed and offsetting exit ports, enabling selective guidewire re-entry during chronic total occlusion recanalization. Although it is not designed for electrophysiology use, we believe its self-orienting
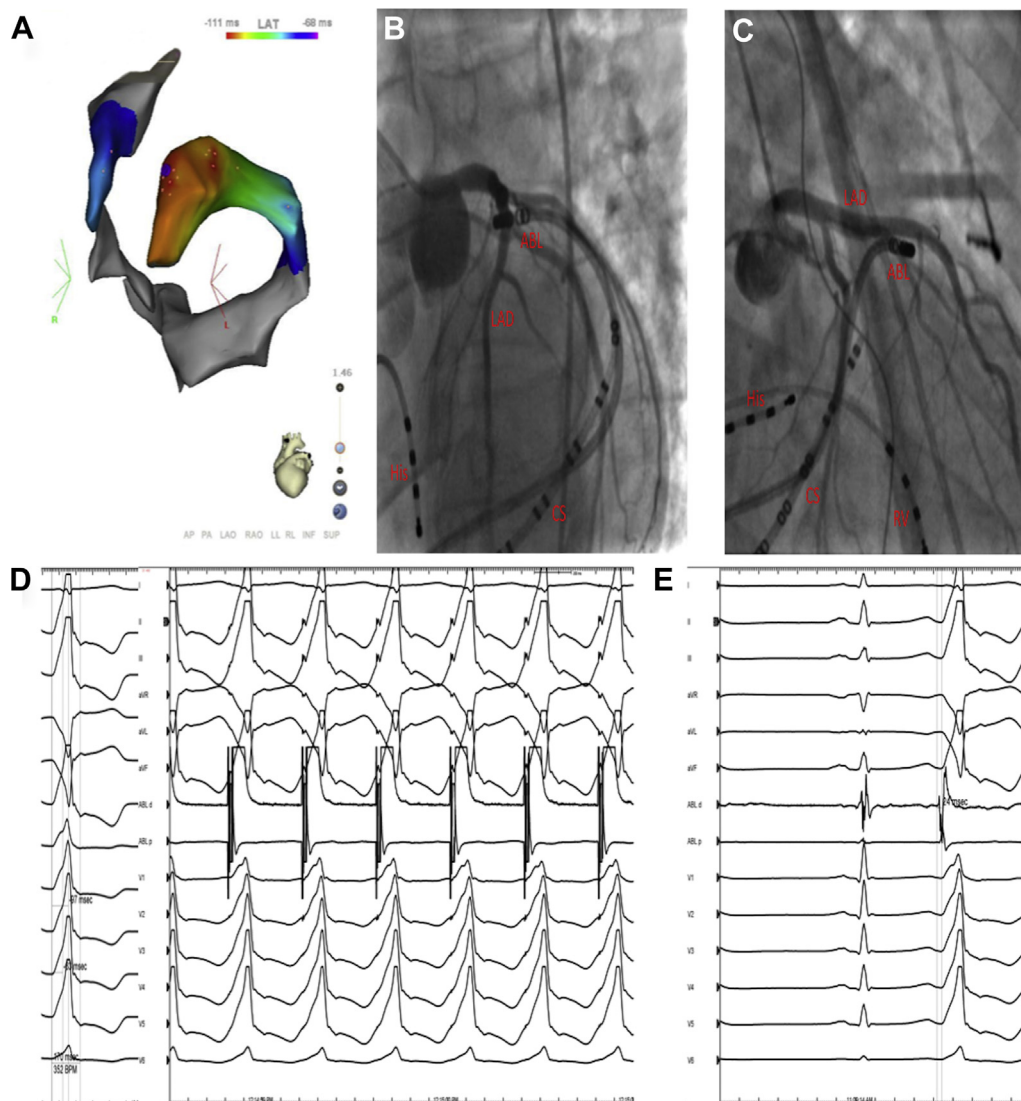

Figure 2   A: Electroanatomic mapping showing earliest site of activation at the great cardiac vein/anterior interventricular vein junction. **B, C:** Angiographic images in (**B**) left anterior oblique and (**C**) right anterior oblique projections depicting the relation between the ablation catheter tip and the proximal left anterior descending artery (LAD). Noticeably, the earliest activation found within the anterior interventricular vein (marked by the tip of the ablation catheter) is clearly in close proximity with the first septal perforator branch (*). **D:** A perfect pace map (12/12 leads) was achieved in this site. **E:** Activation mapping of the clinical premature ventricular contraction in the CS showing earliest site of activation (*red*) at the anterior interventricular vein (−24 ms pre-QRS). ABL = ablation catheter; CS = coronary sinus; His = His bundle; RV = right ventricle.

balloon provides adequate support to allow driving the guide-wire from the coronary arteries into the myocardium. The balloon also has 2 radiopaque marker bands to facilitate accurate placement and positioning (Figure 3B) and a hydrophilic stiff guidewire specifically designed to perforate the arterial wall. Using the hydrophilic coated Stingray guidewire (0.014 inches/0.36 mm), the artery was deliberately perforated and the Stingray guidewire advanced deep into the interventricular septal myocardium (Figure 3C), using orthogonal projections in the left anterior oblique and right anterior oblique views to guide proper guidewire position. At this first intramyocardial location, an early site of −28 ms was obtained (Figure 3D). Subsequently, we repositioned the Stingray LP device (Boston Scientific, Marlborough, MA) deeper into the first septal perforator where the Stingray guidewire was again advanced into the myocardium (Figure 4). An early activation site (−59 ms) was found at

this position (Figure 5A–D). RF ablation was undertaken through the Stingray guidewire by placing the proximal end of the guidewire in a saline bath along with an 8-mm-tip catheter to deliver RF (power 50 W, impedance drop 15 ohms) for a total of 2 minutes, achieving complete elimination of the PVCs (Supplemental Figure 2). Coronary angiogram after RF ablation showed patency of the first septal perforator (Figure 5D). No early complications occurred, and the patient was discharged the next day. Delayed enhancement after RF ablation is depicted in short and longitudinal axes in the interventricular septum on cardiac magnetic resonance imaging (Figure 6) the following day after ablation. At 12-month follow-up, the patient has had a significant improvement in his symptoms, with a marked reduction in his PVC burden from 35% to 2% and normalization in his LVEF (ie, 60%), consistent with PVC-induced cardiomyopathy.
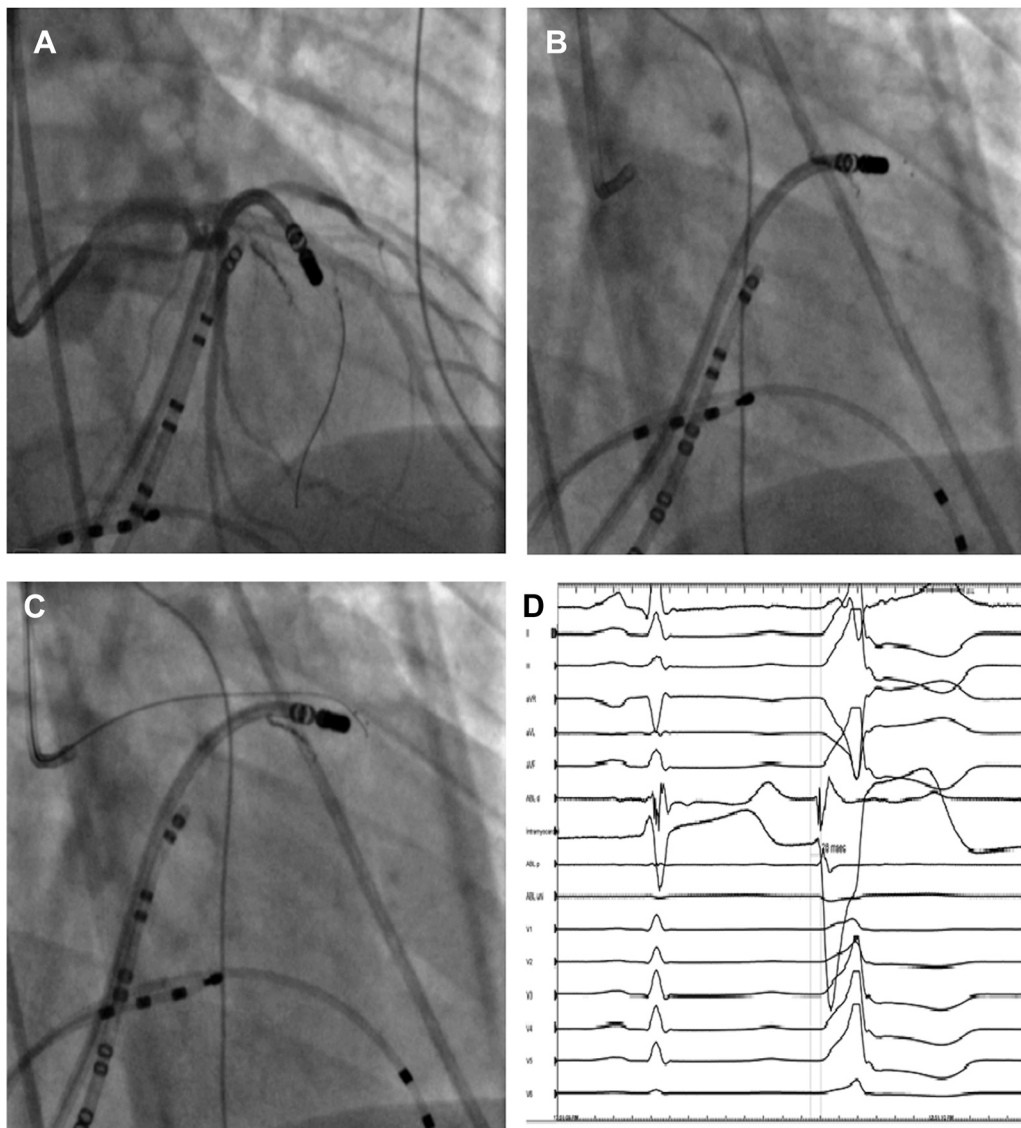
Figure 3 **A:** A 0.014-inch Vision guidewire (Biotronik SE&CO KG, Berlin, Germany) was advanced into the first septal perforator, over which the Stingray LP system balloon (Boston Scientific, Marlborough, MA) was advanced. **B:** Radiopaque marks (seen near the tip of the ablation catheter) are used to guide balloon placement. **C:** Once the Stingray balloon was in place, the Vision guidewire was removed, and the Stingray guidewire was advanced into the myocardium. **D:** Earliest activation in this initial intramyocardial location was −28 ms.
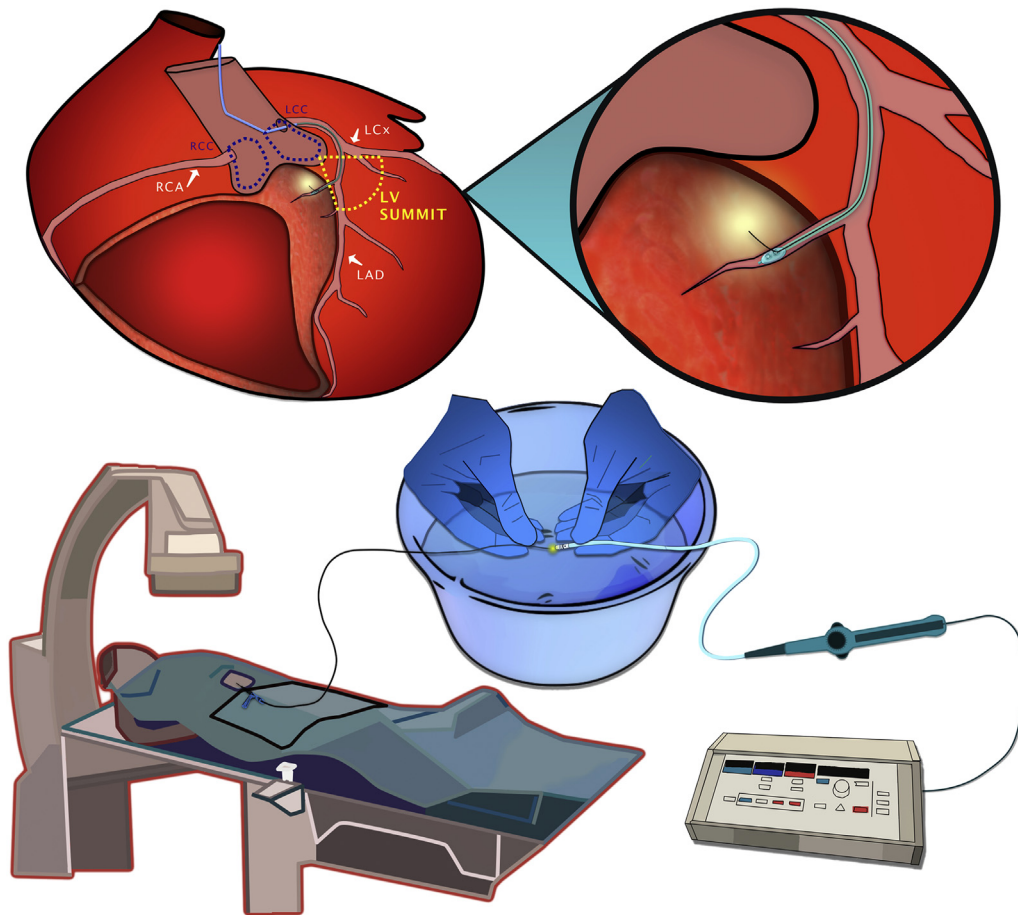
Figure 4   Illustration depicting ablation technique using the Stingray CTO system. A Stingray balloon was advanced into the first septal perforator, and a hydrophilic Stingray guidewire was used to perforate the arterial wall and advanced into the interventricular myocardium. The proximal end of the wire was then introduced into a saline bath along with the ablation catheter, and radiofrequency was delivered in this way, achieving successful premature ventricular contraction ablation. LAD = left anterior descending artery; LCC = left coronary cusp; LCx = left circumflex artery; LV = left ventricle; RCA = right coronary artery; RCC = right coronary cusp.

Discussion

Although highly effective in eliminating PVCs, CA of VA is limited by the origins' anatomic location even when using a combined endo-epicardial approach. VA originating from the LV summit and mid myocardium (particularly in the interventricular septum) are difficult to map and ablate, owing to limited access and difficulties in delivering long-lasting lesions. Additionally, RF ablation for LV summit VAs is still challenging even when performing RF ablation from the CS or the epicardial space, because of the close proximity with the coronary arteries, a thick epicardial fat pad, and frequent intramural sites of origin (Supplemental Figure 3). The LV summit is defined as the region on the epicardial LV surface near the bifurcation of the left main coronary artery bounded by the LAD superior to the first septal perforating branch and anterior to the left circumflex artery laterally. The GCV bisects the LV summit into a superior portion (the inaccessible area) and an inferior portion (the accessible area). It is important to understand that the interventricular septum and the LV summit are thick structures measuring up to 2 cm in thickness (Figure 4). Since a previous ablation attempt had failed in this patient (even after using long-duration RF ablation with high-power settings) and the 12-lead ECG

aVL/aVR Q-wave and III/II R-wave ratios suggested a PVC origin from the inaccessible area in the LV summit,[6] epicardial access was not attempted. Additionally, epicardial mapping and ablation has been questioned, given the lack of additional benefit and a higher complication rate compared with an ablation approach from within the coronary venous system and surrounding structures. An epicardial PVC site of origin has a lower success rate, trends toward a higher procedural complication rate, and has an increased rate of PVC-induced cardiomyopathy.[7] Application of RF energy within the coronary venous system also raises concern for thermal injury to the vein itself or to the neighboring coronary arteries, and isolated reports have documented the risk of venostenosis, vein rupture or thrombosis, and even acute coronary occlusion.

Different approaches to the elimination of intramural and LV summit PVCs have been described, including sequential or simultaneous irrigated unipolar RF applications delivered from the endocardium and epicardium, use of bipolar ablation, and surgical cryoablation.[1–3] In our patient, these techniques could not have been used owing to the inability to deliver epicardial or bipolar RF ablation because of close proximity of LAD and, in the case of bipolar ablation, a possible
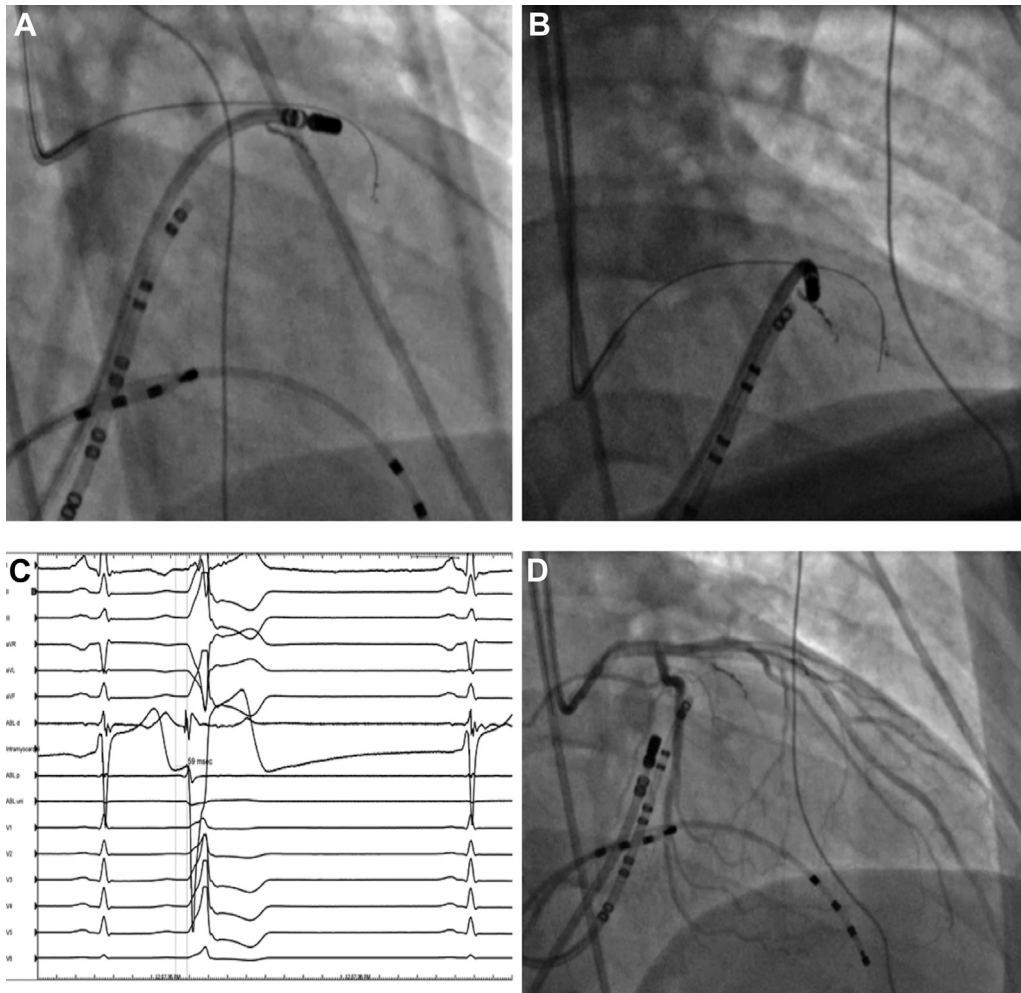
Figure 5   Final position of the Stingray system. **A, B:** The balloon is positioned deep into the first septal perforator (**A**), and the Stingray wire has been advanced deeper into the myocardium (**B**). **C:** An early activation site (−59 ms) was found, and ablation using a nonirrigated 8-mm catheter dipped in a saline bath alongside the guidewire was undertaken. **D:** After ablation, repeat coronary angiogram shows patency of the first septal perforator, limiting the amount of myocardium suffering damage to the target area.

impedance mismatch between the aortomitral continuity and GCV/AIV as the higher impedance of either electrode, which would limit the current. Even surgical cryoablation has shown risk of coronary injury, requiring percutaneous intervention to the LAD coronary artery in up to 25% of cases.[3] This is owing to the fact that the layer of epicardial fat in the LV summit near the trifurcation of the left main coronary artery into the LAD, ramus intermedius, and left circumflex artery has to be dissected to obtain clear visualization of the epicardial surface of the myocardium and to deliver cryoablation lesions in areas adjacent to the LAD.

Alternatively, prolonged high-power ablation from the endocardium may be used to create a deeper lesion targeting remote locations across the myocardial wall. Nevertheless, even though irrigated catheters deliver higher power to create larger RF ablation lesions, lesion depth is currently an important limitation when facing VA originating in the LV summit and intraventricular septum. A study by Simmers and colleagues[8] in an animal model demonstrated that the average distance reached by a nonirrigated 4-mm-tip ablation catheter

was 7.1 ± 2.6 mm and that this distance is already reached by 10–20 seconds and that does not increase with extended duration in RF ablation delivery. Irrigated catheters have shown to increase RF lesion size to 9–13 mm, mostly depending on the RF ablation duration.[9,10] Consequently, RF ablation of an intramural or LV summit VA from only 1 site is usually not enough to suppress the arrhythmia permanently, since this anatomic structure is extremely thick (Figure 4). In some cases, the arrhythmia might be transiently suppressed owing to tissue inflammation but not owing to irreversible tissue injury.[11,12] Animal models have also suggested that the use of half-normal saline or dextrose water 5% can be used to create deeper lesions than those created with normal saline as irrigant solution, but these findings have not been tested in humans.[13]

Coil embolization and ethanol ablation have also been described extensively in prior reports to treat patients with intramural or LV summit VAs.[4,5] However, extensive myocardial damage and complete heart block may occur, as the extent of myocardial injury is determined by a
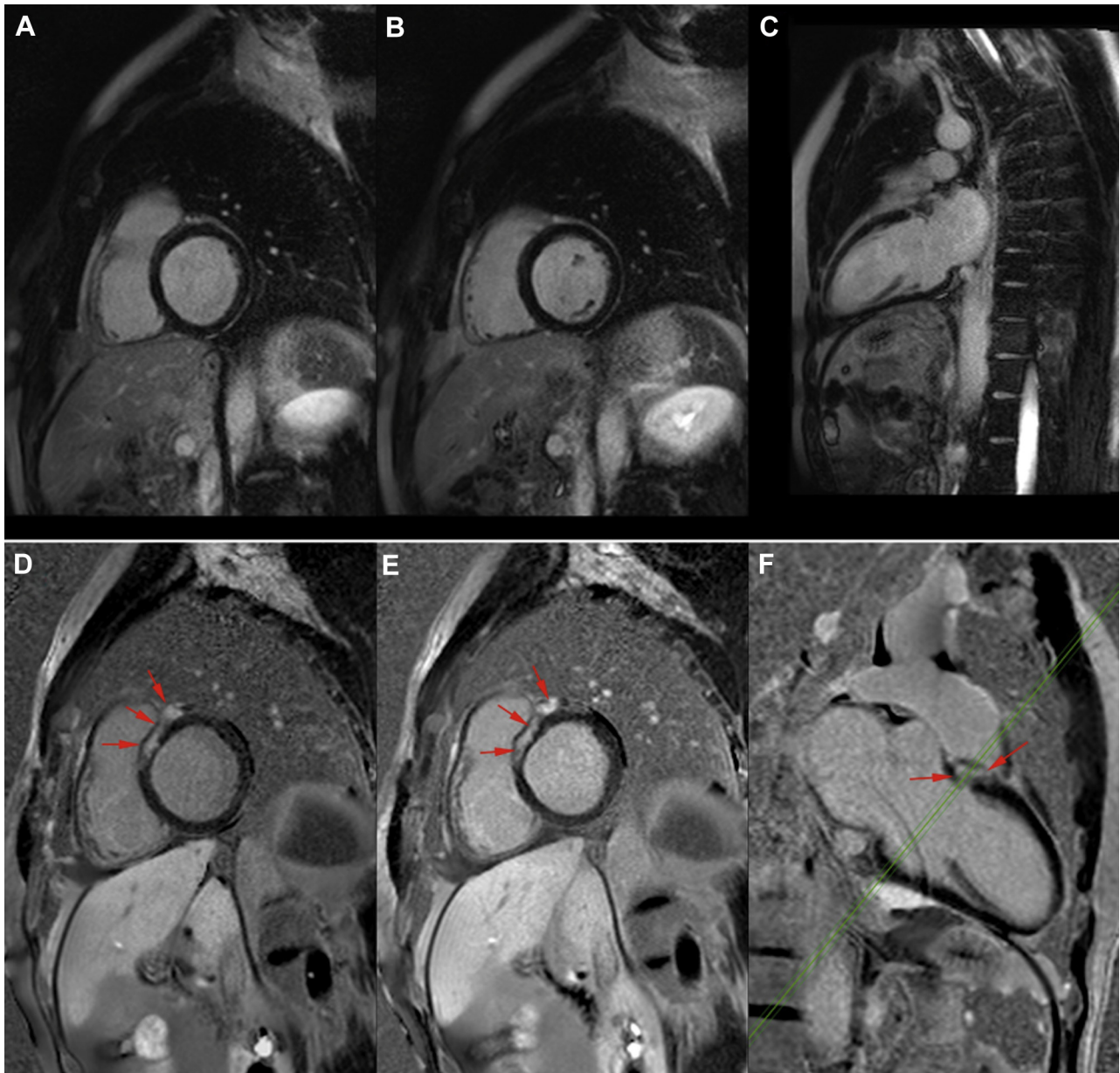
Figure 6   Cardiac magnetic resonance imaging performed before the ablation (**A–C**) and the following day after ablation (**D–F**). No scar can be seen at baseline, and after ablation delayed enhancement (representing myocardial lesion formation; *red arrows*, panels D–F) can be observed in the anterior basal septum and left ventricular summit.

highly variable coronary anatomy. With a purely intramyocardial ablation such as the one described in this case, a more limited myocardial injury may be expected when compared to coil embolization and alcohol injection, which might minimize any further reduction in myocardial performance and reduce the risk of lesion to the conduction system. Although new technologies such as intramyocardial infusion-needle CA have been performed to control refractory VAs using a temperature-controlled mode, with promising results,[14] delivering this needle from the epicardium (in the case of intramural or LV summit arrhythmias) will have same risks of epicardial ablation.

Although intramural recordings of electrograms and ablation using an irrigated needle-tipped catheter have been

studied for over a decade, demonstrating feasibility, to the best of our knowledge this is the first report using direct intramuscular RF ablation through a coronary artery branch using the Stingray device and delivering RF through a guidewire inserted into the myocardium to eliminate arrhythmias originating deep in the mid myocardium/LV summit. The Stingray device has a self-orienting balloon with 180° opposed and offsetting exit ports, enabling selective guidewire reentry into the coronary artery lumen, and is intended to be used for treatment of chronic total coronary obstructions (Supplemental Figure 1). This balloon provides adequate support to achieve introduction of the guidewire (ie, Stingray wire) deep within the myocardium. Our case report provides unique evidence of a novel technique to achieve ablation of

intramural/LV summit foci. We believe that even though applicability of this technique is highly dependent on the anatomy of the coronary artery supplying the myocardium of interest, since the guidewire can be advanced deeply into the myocardium, variations in coronary artery anatomy may have a smaller impact on this technique than on alcohol or coil embolization. Several potential complications can occur during RF application using this technique, including coronary artery occlusion (owing to RF damage or mechanical trauma during Stingray catheter manipulation to the endothelium) and intramural hematoma or coronary artery–cameral fistula. Although intramural hematoma has been described in up to 7% of patients undergoing recanalization of chronic total occlusions,[15] we believe this is probably related to the use of guidewires, microcatheters, and balloons that are advanced subintimally through long distances, thus increasing the risk of vascular lesion and hematoma formation. In our case, we believe this risk is extremely small, as the guidewire is not advanced subintimally but instead is driven directly into the myocardium.

Conclusion

Ablation of LV summit/intramural arrhythmias is technically difficult and carries a lower success rate owing to increased tissue thickness, limited catheter maneuverability, and proximity of coronary arteries. We present a novel technique to achieve suppression of challenging VA using intramyocardial RF ablation, which could potentially allow targeting areas in close proximity of coronary arteries, including the interventricular septum and the inaccessible LV summit. Further studies are needed to confirm the safety and efficacy of this technique.

Acknowledgments

Appendix
Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2018.03.005.

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