

Prolonged high-power endocardial ablation of epicardial microreentrant VT from the LV summit in a patient with nonischemic cardiomyopathy



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Introduction

Epicardial mapping and ablation have emerged in the last decade as a strategy to increase the success rate for the eradication of arrhythmogenic circuits in complex scar substrates. It has been established that epicardial mapping and ablation are generally required for some arrhythmic substrates such as in arrhythmogenic right ventricular dysplasia, Brugada syndrome, Chagas cardiomyopathy, nonischemic cardiomyopathy (NICM), and to a lesser extent in ischemic cardiomyopathy (ICM). Ablation in patients with NICM has been shown to be inferior to that in patients with ICM, as scar patterns are more variable with epicardial and intramural locations. In addition, the arrhythmic mechanism of ventricular tachycardia (VT) in patients with NICM is not always macroreentrant, as focal and automatic sites of origin have been reported in up to 20% of patients. Furthermore, delivering radiofrequency (RF) lesions in the epicardial space might be limited because of significant epicardial fat as well as close proximity to phrenic nerves and coronary vasculature.

In this report, we present a case of a patient with NICM and VT, in whom (1) a microreentrant mechanism was demonstrated by activation and entrainment mapping and (2) successful elimination of an epicardial circuit was achieved with ablation through normal endocardial tissue because of proximity of the epicardial site of origin to a coronary artery.

Case report

This is the case report of a 63-year-old man with NICM diagnosed 8 years before admission, presumed to be

secondary to viral myocarditis. The patient had a left ventricular ejection fraction (LVEF) of 20%, had New York Heart Association class III symptoms, and had received a cardiac resynchronization therapy-defibrillator for the primary prevention of sudden cardiac death. The patient was brought to the emergency department after collapsing at home, with bystander cardiopulmonary resuscitation initiated. He was found to be in slow VT at 134 beats/min (cycle length 447 ms), which was below the detection zone for his implantable cardioverter-defibrillator, and received therapy using an automated external defibrillator. In the emergency department, he had several similar episodes, resulting in hypotension and loss of consciousness. He required cardiopulmonary resuscitation and automated external defibrillator shocks on 2 more occasions. VT recurred despite intravenous amiodarone and lidocaine.

A 12-lead electrocardiogram revealed VT with a right bundle branch block morphology, positive concordance, and right inferior axis. A transthoracic echocardiogram showed dilated left ventricular (LV) left ventricular internal dimension, diastole (LVIDd (2D) 7.12 cm) with normal wall thickness (interventricular septum, diastole IVSd (2D) 0.89 cm; left ventricular posterior wall, diastole LVPWd (2D) 1.01 cm), severe LV diastolic dysfunction (grade III, restrictive physiology), as well as severe LV systolic dysfunction (LVEF 23%). Subsequently, the patient underwent emergent coronary angiography, which showed normal coronary arteries. Positron emission tomography with ¹⁸F-fluorodeoxyglucose was negative for cardiac sarcoidosis.

A semi-emergent invasive electrophysiology study for ablation was performed. Clinical VT was induced with triple extrastimuli (500/320/350) using noninvasive programmed stimulation at the beginning of the procedure (tachycardia cycle length 470–500 ms, right inferior axis, and right bundle branch block pattern with R waves across the precordium). The QRS width during VT was 290 ms, with a pseudo-delta wave of 105 ms, an intrinsicoid deflection time of 174 ms, and a maximum deflection index of 60%—all suggestive of an epicardial site of origin. The coronary sinus could not

KEYWORDS Ablation; Epicardial; Radiofrequency; Ventricular tachycardia

ABBREVIATIONS ICM = ischemic cardiomyopathy; LV = left ventricle/ventricular; LVEF = left ventricular ejection fraction; NICM = nonischemic cardiomyopathy; RF = radiofrequency; RV = right ventricle/ventricular; VT = ventricular tachycardia
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KEY TEACHING POINTS

- The mechanism of ventricular tachycardia in patients with structural heart disease is most frequently macroreentrant. However, many patients with a myocardial scar have focal mechanisms and a careful analysis of entrainment can be helpful in determining the most desirable timing of the successful ablation site.
- Epicardial ablation is frequently limited by close proximity to coronary arteries and epicardial adipose tissue. Alternative strategies and technologies are needed to address these substrate locations.
- Prolonged high-power ablation from the endocardium may create a deeper lesion to target remote locations across the myocardial wall, and early termination should not be expected during radiofrequency delivery in such cases. Optimal biophysical parameters require further clarification to maintain safety.

be cannulated past the LV pacing lead because of narrow and tortuous anatomy. A 2-mm spacing decapolar catheter (CARTO 3, Biosense Webster Inc, Diamond Bar, CA) was used to map the endocardium and epicardium. Subxiphoid epicardial access was obtained using a Tuohy epidural needle. After epicardial access, an LV and right ventricular (RV) epicardial voltage map (539 points) was obtained, which showed a small area of scar in the LV basal anterolateral wall (Figure 1). Intravenous heparin was administered for a retrograde aortic approach; a high-density endocardial LV electroanatomic map (282 points) was created, which revealed no endocardial scar (Figure 1). A small perivalvular scar was seen on the basal anterolateral region of the epicardium with split and fractionated electrograms. Activation mapping revealed the earliest site of activation in the basal anterior epicardial LV wall (−50 ms) (Figure 1). The best pace maps of clinical VT were noted in close proximity to the basal anterolateral LV scar. Concealed entrainment was also observed in this area (Figure 1).

In order to assess the mechanism of tachycardia, entrainment mapping was performed from the RV apex. There was evidence of progressive fusion from the endocardial RV apex with pacing at different rates (Figure 2). The QRS width was narrower with lower limb lead amplitudes during overdrive pacing at 430 ms compared to 390 ms. This is highly suggestive of the presence of both constant and progressive fusion. The earliest site was mapped to the epicardium with a focal propagation (Online Supplemental Video 1). It was felt that the mechanism of arrhythmia was microreentrant, and therefore, unmapped sites with mid-diastolic activation were unlikely to be found. Left coronary angiography revealed that this site was close to the second diagonal artery (Figure 1).

The ablation catheter (4-mm ThermoCool DF) was positioned endocardially directly opposite to the earliest epicardial activation site, where activation was seen just after the QRS onset (Figure 1). Of note, entrainment mapping from the endocardium revealed overt fusion. Ablation was performed in the power mode setting at 50 W (temperature <42°C). Termination was noted at 24 seconds during the first lesion (90 seconds), but VT was reinducible and a second application (90 seconds) was delivered where termination was observed again at 26 seconds (Figure 3). Another consolidation RF lesion was delivered with a total RF time of 4 minutes. Biophysical changes with significant impedance drops (>30 Ω) were observed during ablation 1 and 2. No steam pops were observed. VT was no longer inducible with triple extrastimuli in the RV and epicardial/endocardial LV at different sites. Antiarrhythmic agents were discontinued, and the patient has remained free from VT recurrence at 10 months of follow-up.

Discussion

We present this case report to highlight the following:

1. Arrhythmias in scar-related cardiomyopathy should not be assumed to be macroreentrant.
2. Coronary arteries remain a significant anatomical limitation in patients with NICM and arrhythmias arising from the LV summit.
3. Prolonged endocardial high-power RF applications can be an effective therapeutic alternative for eliminating remote sites of origin.

Overall, it is estimated that VT ablation requires epicardial ablation in 13% of patients with a risk of 5% and 2% of acute and delayed major complications related to epicardial access, respectively.¹ RF ablation in the epicardial space for basal anterolateral substrates is frequently limited by a variety of factors including thick epicardial fat, close proximity to the phrenic nerve, and coronary vasculature as well as inability to deliver high power because of poor cooling.

Studies recommend avoidance of RF ablation within 5 mm of coronary arteries, as injury has been reported in animal models and human cases.² As in the case presented in this report, our target was in the vicinity of a medium caliber diagonal artery. The options for RF ablation at this point were limited, since the coronary sinus could not be cannulated distally (LV lead and narrow lumen). Options included a hybrid procedure using a limited lateral thoracotomy with direct visualization,³ sacrificing the artery in a patient with already severely impaired LVEF, or implementing more aggressive measures such as bilateral cervicothoracic sympathectomy (ie, resection of the lower half of the stellate ganglion and the first 4 thoracic ganglia via thoracoscopy) and renal artery sympathetic denervation.^{4,5} Cryotherapy might be useful for epicardial VT ablation to decrease the risk of coronary artery injury and, in theory, to improve lesion

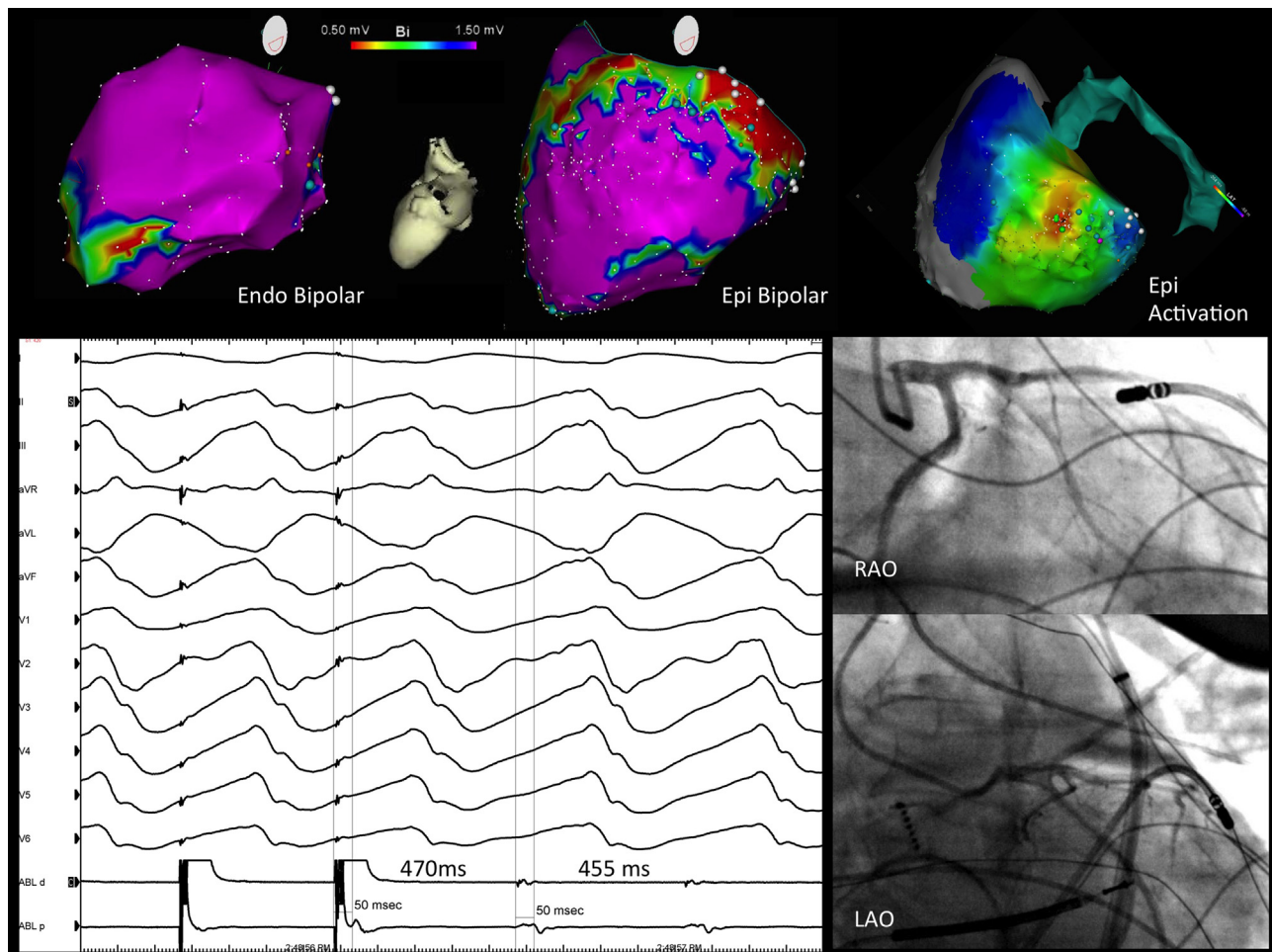


Figure 1 Electroanatomic mapping with bipolar endocardial and epicardial voltage. Activation mapping on the epicardium showed focal propagation. Top: Left posterolateral view of endocardial and epicardial voltage map of LV. Left lateral view of epicardial activation mapping indicating earliest activation site in red (-50 ms). Bottom: Concealed entrainment from earliest site. TCL was 451ms and pacing was carried out at 430 ms. Post pacing interval (PPI) was 470. TCL-PPI: 15 ms. EGM-QRS=S-QRS(50 ms). Two fluoroscopic views demonstrate proximity to a diagonal coronary artery.

depth.⁶ The coronary venous system through the distal great cardiac vein and the anterior interventricular vein course in the basal part of the epicardial surface of the LV has been shown to be a successful ablation site in patients with premature ventricular contractions and VT. It is tempting to hypothesize that some epicardial VTs may arise close to the coronary venous system, which may therefore be a suitable site to map and ablate these arrhythmias. Application of RF energy within the coronary venous system raises concern for thermal injury to the vein itself or to the neighboring coronary arteries, and isolated reports have documented the risk of venous stenosis, vein rupture, thrombosis, and even acute coronary occlusion.⁷ Similarly, the left phrenic nerve may be vulnerable when ablating in the vicinity of the obtuse marginal vein or the great cardiac vein.⁸

Irrigated catheters have been demonstrated to allow more power to be delivered before temperatures reach a point in which thrombus/clots are formed. This report describes the feasibility of transmural ablation from the endocardium for epicardial circuits with longer RF applications at higher power (Online Supplemental Video 2). Yokokawa et al⁹ have described ablation of epicardial VT from nonepicardial sites,

where 3 patients were described to have success with endocardial ablation alone. Komatsu et al¹⁰ recently evaluated the feasibility and safety of epicardial substrate elimination by delivering endocardial RF energy in patients with scar-related VT, where endocardial ablation could abolish all epicardial local abnormal ventricular activity (LAVA) in 4 patients with ICM and 2 patients with arrhythmogenic right ventricular cardiomyopathy (ARVC) whereas all patients with NICM required epicardial ablation. Endocardial ablation was able to eliminate epicardial LAVA at least partially in 15 patients with ICM (83%), 11 with arrhythmogenic right ventricular cardiomyopathy (ARVC) (73%), and 2 with NICM (13%), contributing to a significant reduction in epicardial RF applications. ADDIN EN.CITE ADDIN EN.CITE.DATA¹⁰ Prolonged endocardial ablation with an impact on epicardial substrate may be one reason that the need for epicardial ablation is currently underestimated.

Although standard RF applications have been shown to reach a maximum lesion size between 20 and 60 seconds, irrigated ablation has the potential to create deeper lesions with longer duration applications. Nakagawa et al¹¹ demonstrated a continuing steep rise in tissue temperature at

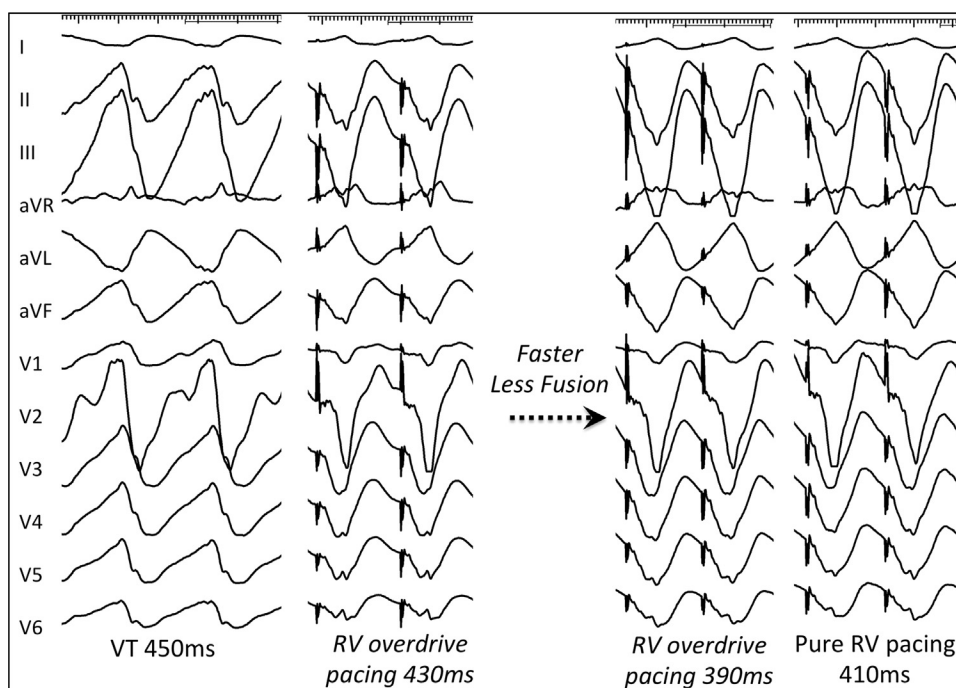


Figure 2. Constant fusion and subtle progressive fusion is demonstrated with pacing from the RV apex at different rates (390 ms and 430 ms) supporting a reentrant mechanism.

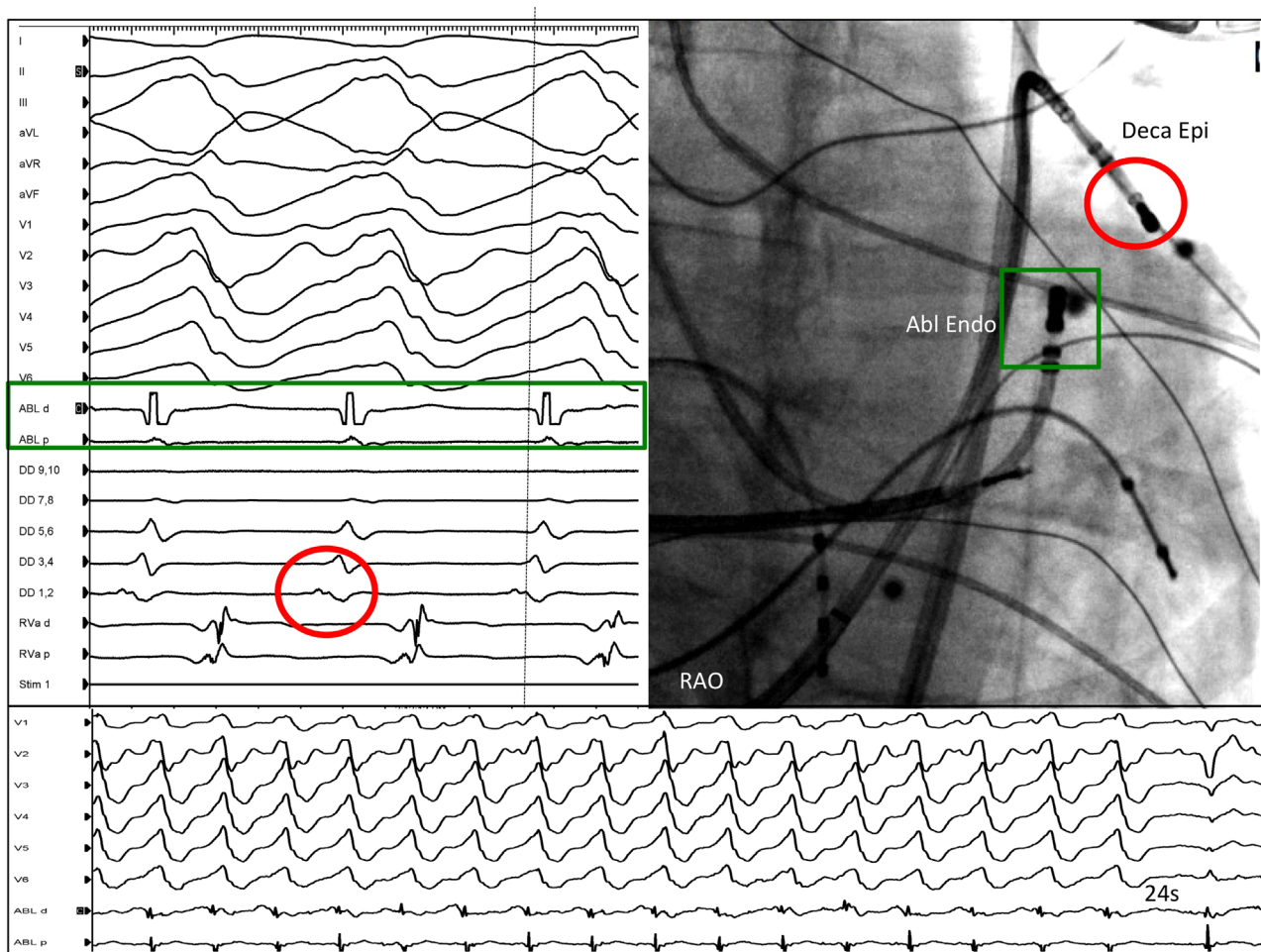


Figure 3. Endocardial ablation across from the earliest region of epicardial activation. The endocardial site is just after the onset of the QRS. Delayed termination (24s, 26s) was seen on two occasions and the patient was rendered noninducible.

7-mm depth at 60 seconds in a canine thigh muscle preparation. Furthermore, extending the duration of RF delivery may overcome intermittent tissue contact, as Olson et al¹² demonstrated comparable lesion size between constant contact at 60 seconds and intermittent contact lesions at 90 seconds. Additional studies are required to understand the maximum effective duration of irrigated ablation.

The theoretical advantage of prolonged high-power ablation must be weighed against the potential risks of char or thrombus formation and steam pops. In a retrospective analysis of RF applications, Seiler et al¹³ demonstrated that 80% of steam pops were associated with an impedance drop of $>18 \Omega$. However, there was significant overlap between those with and without steam pops ($22 \pm 7 \Omega$ vs $18 \pm 8 \Omega$) and 31% of the lesions that did not result in steam pops had an impedance of $>18 \Omega$. Although tissue temperature is not always correlated with the catheter-tissue interface temperature via irrigated technology, it is our institutional practice to set a maximum catheter temperature to 45°C and carefully observe for sudden increases and decreases in impedance. In this particular case, we did not observe a specific impedance drop cutoff, as this was a “bailout” option and this strategy should be reserved for cases that fail traditional approaches. Further studies to characterize and guide long-duration, high-powered lesions in animal models may be helpful to optimize safety and efficacy of this strategy.

Appendix Supplementary data

Supplementary material cited in this article is available online at <http://dx.doi.org/10.1016/j.hrcr.2015.07.008>.

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