

# Successful bipolar radiofrequency catheter ablation in a case of scar-related ventricular tachycardia with intramural critical isthmus in basal posterior right ventricle identified by coherent mapping



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## Introduction

Radiofrequency (RF) catheter ablation is widely accepted therapy for scar-related ventricular tachycardia (VT). However, recurrence of VT after conventional unipolar ablation (Uni-ABL) remains a problem.<sup>1,2</sup> Difficulty in mapping VT substrate that lies deep within the ventricular myocardium and difficulty in creating transmural lesions with Uni-ABL for such deep substrate are considered to be among the major causes of VT recurrence.<sup>3–5</sup> Meanwhile, some reports have described the efficacy of bipolar ablation (Bi-ABL) for eliminating intramural VT circuits, especially in patients with recurrence after Uni-ABL for scar-related VT.<sup>6–11</sup> We report a rare case of nonischemic scar-related VT that had an intramural critical isthmus deep in the basal posterior right ventricle (RV) that was successfully eliminated by endocardial-epicardial Bi-ABL.

## Case report

A 31-year-old man with mildly dilated RV was urgently admitted for treatment of a stable sustained monomorphic VT with cycle length (CL) of 250 ms. Twelve-lead electrocardiogram during the VT showed a wide QRS complex with left bundle branch block and superior axis morphology. Cardiac magnetic resonance imaging showed no late gadolinium enhancement in the left ventricle (LV), although signal-averaged electrocardiogram showed a late potential. The mildly dilated RV as observed by echocardiography did not

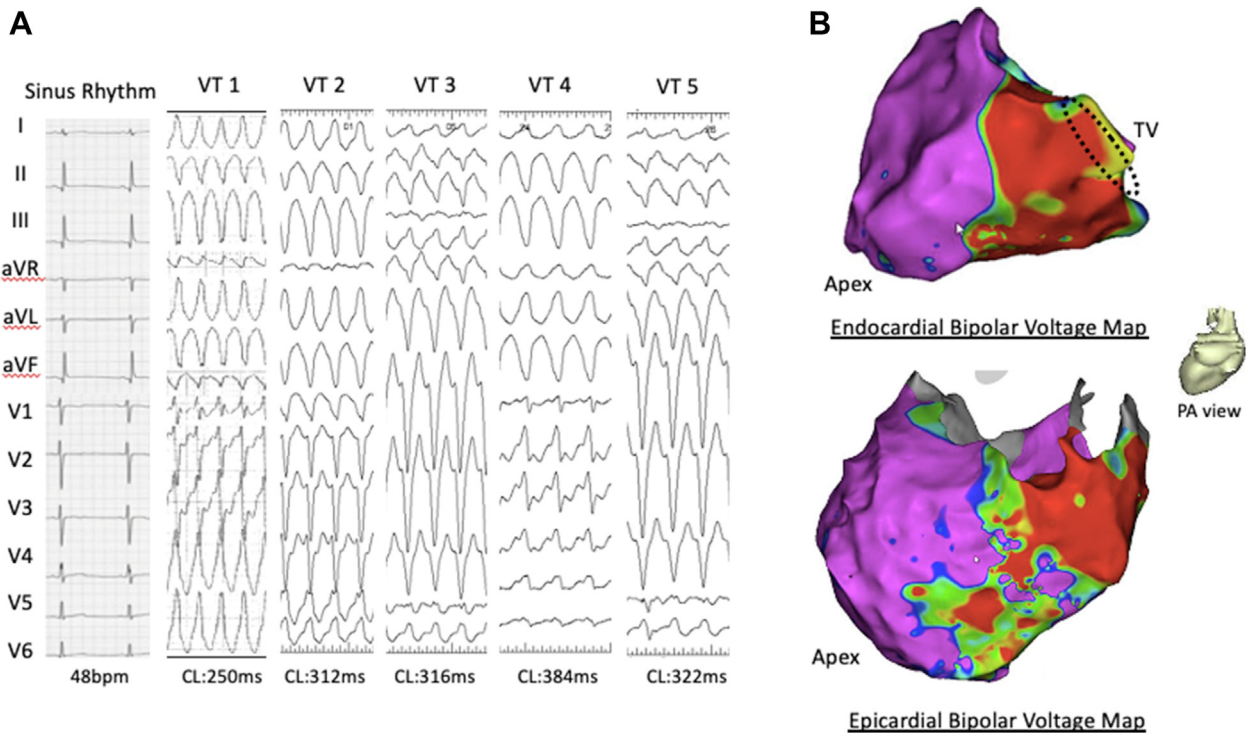
## KEY TEACHING POINTS

- We report on the rare case of a sustained monomorphic ventricular tachycardia (VT) with an intramural critical isthmus of the reentrant circuit in the right ventricle that was successfully eliminated by bipolar ablation.
- The conduction velocity vector display of the endocardial and epicardial coherent maps during sustained VT indicated the presence of an intramural slow conducting isthmus of a reentrant VT circuit within the basal right ventricular musculature.
- Endocardial and epicardial coherent mapping during sustained VT may provide significant information to identify the intramural circuit, which can be eliminated by bipolar ablation.

meet the definite diagnostic criteria for arrhythmogenic right ventricular cardiomyopathy (ARVC). The patient underwent catheter ablation for the VT. A monomorphic sustained VT (VT1, [Figure 1A](#)) was easily induced and endocardial 3D mapping in the LV during VT1 showed focal activation originating from the mid posteroseptal LV, which was eliminated by Uni-ABL at the earliest activation site in the LV. Only the LV was mapped in the first procedure using the CARTO system. The endocardium of the RV and epicardium were not mapped. He was given a subcutaneous implantable cardioverter-defibrillator after the VT ablation and put on amiodarone. Three months after the VT ablation, his VT recurred, requiring multiple implantable cardioverter-defibrillator shocks. In the second procedure, a bipolar

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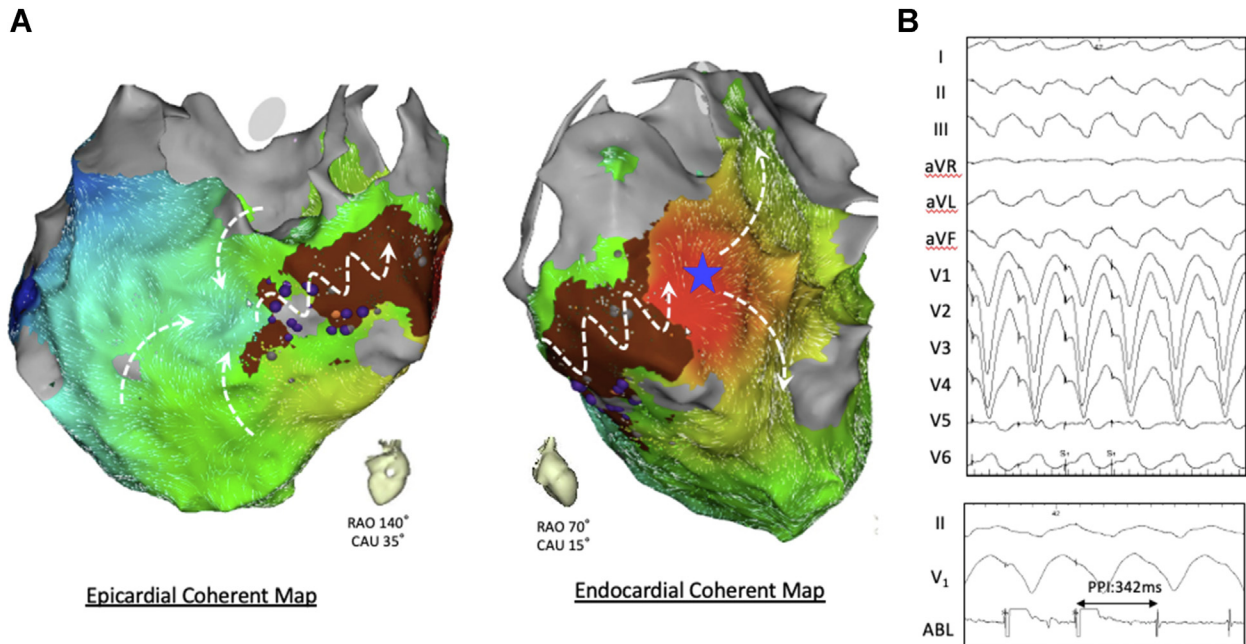


**Figure 1** **A:** Twelve-lead electrocardiogram during sinus rhythm and the ventricular tachycardias (VTs) observed during procedures. **B:** The endocardial and epicardial bipolar voltage maps showed an extensive low-voltage area with abnormal electrograms in the basal posterior right ventricle, which extended to epicardial interventricular septum and basal posterior left ventricle. CL = cycle length; PA = posteroanterior; TV = tricuspid valve.

voltage map during sinus rhythm showed an extensive low-voltage area with abnormal electrograms in both the endocardial and epicardial basal posterior RV, which was extended to the epicardial interventricular septum and basal posterior LV (Figure 1B) in addition to a low-voltage area in the endocardial midposteroseptal LV, which matched the ablation site in the first procedure. A sustained VT with left bundle branch block and superior axis QRS morphology and CL of 312 ms (VT2) was induced by programmed ventricular stimulation, which easily transitioned to ventricular fibrillation. A pace-map match to VT2 was observed in the endocardial basal posterior RV. Uni-ABL targeting the abnormal electrograms at this site and other sites on the endocardial and epicardial voltage maps was performed. Although no ventricular arrhythmias were inducible at the end of ablation, 5 months after the second procedure, a VT storm recurred that required multiple implantable cardioverter-defibrillator shocks. In the third procedure, VT3–VT5 with similar QRS morphology to that of VT1 or VT2 were induced. A pace-map match to VT3 (CL: 316 ms) was found on the epicardial side of the basal posterior RV. Meanwhile, the exit site of VT4 (CL: 384 ms) was estimated to be located near the interventricular septum or the basal posterior LV. However, a pace match site of VT4 was not identified. The 3D map showed extensive low-voltage areas with delayed potentials in the endocardial and epicardial posterior RV in the same areas as that in the second procedure. For VT3 and VT4, RF applications with Uni-ABL were delivered to the pace-match site of VT3, an

estimated exit site of VT4 and its surroundings including epicardial sites beyond the interventricular septum and basal posterior LV during sinus rhythm, owing to hemodynamic instability during these VTs. For VT5 (CL: 322 ms), activation mapping was performed during VT because it was relatively hemodynamically stable. Coherent mapping (CARTO®3 version 7; Biosense Webster, Irvine, CA) showed that epicardial electrical activation during VT5 converged to a scar area in the basal posterior RV, which was considered to be the entrance site of VT5 (Figure 2A). Meanwhile, endocardial coherent mapping during VT5 showed that earliest endocardial activation diverged from a scar area also in the basal posterior RV, which was considered to be the exit site of VT5. In fact, entrainment pacing at the earliest activation site showed apparent concealed fusion with VT5 and a postpacing interval that almost matched the CL of VT5 (Figure 2B). Based on these results, Uni-ABL was repeatedly performed in both the endocardial and epicardial basal posterior RV, but failed to eliminate VT5. This failure suggested the presence of an intramural conducting isthmus deep within the basal posterior RV and led us to attempt Bi-ABL. Bi-ABL had been approved by the ethics committee of our hospital and written informed consent was obtained from the patient beforehand.

Bi-ABL between the RV endocardium and epicardium was performed between the distal electrodes of 2 irrigated ablation catheters, with the endocardial one (ABL<sub>Endo</sub>) serving as an active electrode (THERMOCOOL SMARTTOUCH® SF; Biosense Webster) and the epicardial



**Figure 2** A: Arrows indicating conduction velocity vectors on coherent maps obtained during ventricular tachycardia (VT) 5 showed epicardial activation converging in an area in the basal posterior right ventricle and endocardial activation diverging from the same area, suggesting the presence of an intramural slow conducting isthmus of the VT circuit (*zigzag arrow*). B: Entrainment pacing at the earliest activation site (*blue star*) showed concealed fusion with VT5 and a postpacing interval that matched the cycle length of VT5. CAU = caudal; RAO = right anterior oblique.

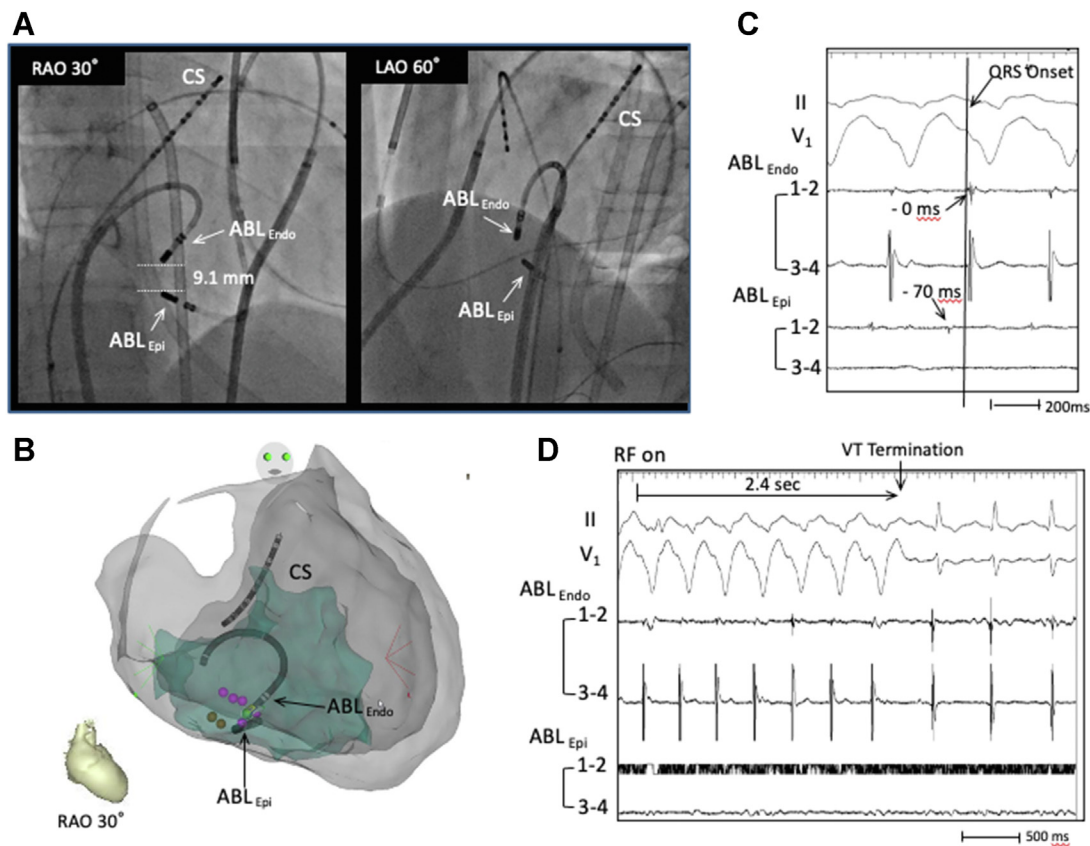
one (ABL<sub>Epi</sub>) serving as the ground electrode (FlexAblity™; Abbott, St. Paul, MN) (Figure 3). The electrogram potential recorded by ABL<sub>Endo</sub> coincided with the QRS onset of VT5, whereas the potential from ABL<sub>Epi</sub> preceded the QRS onset by 70 ms, which was considered as the exit from the VT isthmus under the scar area with no obvious mid-diastolic potentials during the VT5 on the endocardial and epicardial surface activation maps. This suggested the presence of a slow conducting isthmus within the myocardium of the basal posterior RV. Consequently, the exit sites where the potentials were recorded were selected as the ablation target for bipolar ablation. The target VT terminated in 2.4 seconds after the start of bipolar RF application delivered with a maximum power setting of 30 W for 45 seconds. Additional bipolar RF applications were delivered under impedance guidance for 30–45 seconds without any complications. At the end, no VTs were inducible with programmed stimulation using up to 3 extrastimuli. Coronary angiography was performed in both second and third procedures to avoid coronary artery injury owing to epicardial and bipolar RF applications. It has now been 2 years since this Bi-ABL, and the patient has remained free from VT.

## Discussion

Catheter ablation is an established treatment for VT. However, the VT recurrence rate after conventional Uni-ABL is around 30% despite the recent advances in mapping systems, ablation catheters, and ablation strategies.<sup>1,2</sup> The presence of intramural substrate is considered to be one of the causes of recurrence. We described a rare case of scar-related reentrant

VT with a suspected intramural slow conducting isthmus in the basal posterior RV that was successfully eliminated by Bi-ABL. In previous reports, Bi-ABL has been primarily performed for the treatment of ventricular arrhythmias originating from the LV free wall, interventricular septum, LV outflow tract, LV papillary muscles, or His bundle region that were refractory to conventional endocardial and/or epicardial Uni-ABL.<sup>6–11</sup> In the current case, RF energy was delivered between the distal electrodes of 2 irrigated catheters, which were placed in the endocardial and epicardial basal posterior RV. To our knowledge, ours is a rare case in which Bi-ABL has been applied with an endo-epicardial approach to a location in the RV. We found endocardial and epicardial coherent mapping during the VT to be very useful in inferring the location of an intramural VT circuit, and we attribute success of the Bi-ABL to this discovery. However, Epi and Endo activation mapping data showed a delay of 70 ms between fairly close Epi and Endo RV surfaces, also suggesting intramural substrate. Other ways of locating intramural substrate are to look for scar in magnetic resonance imaging and direct intramural recording with Bi-ABL, such as performed by Waight and colleagues,<sup>12</sup> who applied this method to scar-related VT originating from the interventricular septum. Direct intramural sampling of substrate was not feasible because of location of the intramural circuit in our patient, but is likely useful in other situations. Meanwhile, the utility of coherent mapping in identifying critical isthmuses has also been reported in patients with scar-related atrial tachycardias.<sup>13</sup> Relative to atrial tachycardias, data from coherent mapping in VT are limited. Hoshiyama and colleagues<sup>14</sup> demonstrated the utility





**Figure 3** A, B: Bipolar radiofrequency (RF) catheter ablation was performed between the distal electrodes of 2 irrigated ablation (ABL) catheters, with the active electrode in the right ventricle (RV) endocardium (ABL<sub>Endo</sub>) and the ground electrode in the RV epicardium (ABL<sub>Epi</sub>), as shown by fluoroscopy and 3D electroanatomic mapping. C: The timing of the ABL<sub>Endo</sub> electrogram coincided with QRS onset of ventricular tachycardia (VT) 5, whereas the ABL<sub>Epi</sub> electrogram preceded the QRS onset by 70 ms. D: The target VT terminated in 2.4 seconds after start of the bipolar RF application delivered with a maximum power setting of 30 W for 45 seconds. CS = coronary sinus; LAO = left anterior oblique; RAO = right anterior oblique.

of coherent mapping for identifying a critical VT isthmus in a patient with multiple VT circuits and extensive area of abnormal electrograms in the LV. In their case, similar to ours, the conduction velocity vector displays provided by coherent mapping of the endocardium and epicardium during the hemodynamically stable VT identified an intramural critical isthmus.

Although clinical outcomes of Bi-ABL for VT have been reported, data regarding long-term outcomes after Bi-ABL procedures are limited. Igarashi and colleagues<sup>6</sup> showed a significant reduction in VT burden during a follow-up period of 12 months after Bi-ABL procedures, although incidence of VT recurrence was high (44%). In patients whose Bi-ABL fail, it may be important to consider an alternative strategy, such as chemical ablation with ethanol infusion to select coronary sinus tributaries or coronary arteries.<sup>3-5</sup>

Although the methodology of Bi-ABL is well established, severe complications such as cardiac tamponade owing to myocardial injuries by steam pop or atrioventricular block owing to Bi-ABL in the interventricular septum have been reported.<sup>6-10</sup> Thus, care is necessary in setting the power level and ablation duration of bipolar RF applications. John *et al* concluded that ablation duration is the most significant factor in producing durable

lesions, whereas the power setting is the most significant factor affecting the incidence of steam pops based on assessment of an *ex-vivo* Bi-ABL model (11). As for epicardial ablation, location of the coronary arteries must be confirmed prior to RF applications to prevent severe coronary artery injury.

Our patient was initially diagnosed with idiopathic VT because various tests showed no obvious abnormal findings except for mildly dilated RV on echocardiography. However, both endocardial and epicardial bipolar voltage maps in the RV showed an extensive low-voltage area with abnormal electrograms, suggesting the presence of some kind of RV cardiomyopathy similar to ARVC. Although the patient did not meet the diagnostic criteria for ARVC, we believe it likely he has a similar pathology based on the distributions of low-voltage areas in his 3D electroanatomic maps. In general, patients with ARVC and VT are highly likely to have endocardial and epicardial VT substrate with abnormal electrograms in the perivalvular region of the tricuspid valve that extends toward the RV free wall and also in the peripulmonic valve area.<sup>15</sup> It has been reported that the musculature around the tricuspid annulus is thicker in ARVC patients than in normal subjects,<sup>16</sup> which may be why our patient required Bi-ABL.

## Conclusions

We presented a rare case of scar-related VT originating from the basal posterior RV where an intramural reentrant circuit was effectively and safely eliminated by endo-epicardial Bi-ABL. Detailed endocardial and epicardial coherent mapping with display of conduction velocity vectors during VT enabled us to identify an intramural critical isthmus deep inside the myocardium adjacent to the tricuspid annulus.

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