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CARTO-assisted bipolar radiofrequency ablation for the treatment of refractory ventricular tachycardia associated with ventricular aneurysm: A case report

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September 21, 2020

Abstract

Introduction: Evidence for the effectiveness of surgical electrophysiological therapy for malignant ventricular tachycardia (VT) after myocardial infarction is lacking. Methods and Results: We report a case of a 50-year-old man with VT caused by left ventricular aneurysm (LVA). Unipolar radiofrequency ablation (RFCA) failed to treat VT. Nonetheless, bipolar RFCA forcep guided by CARTO mapping was used to ablate the LVA and peripheral myocardium during traditional surgery. There were no postoperative complications, and 1-year follow-up showed no malignant VT occurred in 24-hours. Conclusion: Bipolar RFCA assisted by CARTO mapping was an effective surgical electrophysiological therapy for refractory VT caused by LVA.

1. Introduction

Malignant ventricular arrhythmia accounts for 44% of deaths due to left ventricular aneurysm (LVA).[1]Coronary artery bypass grafting (CABG) combined resection or formation of LVA are the main treatments for LVA, but the postoperative sudden cardiac death caused by malignant ventricular arrhythmia accounted for 29.6% and 36.8% of 30-day and 5-year-death respectively.[2]In particular, radiofrequency catheter ablation (RFCA) assisted by a three-dimensional electroanatomical mapping (3D EAM) system is used to treat ventricular arrhythmia following the acute myocardial infarction (MI). However, there are still lack of appropriate electrophysiological mapping and transwall RF for resolving ventricular arrhythmia during surgery due to reentry loop and ectopic pacemaker presented in the endocardium, epicardium or myocardial tissue. This report describes the case of a patient with refractory ventricular tachycardia (VT) secondary to LVA, whose treatment with unipolar RFCA was ineffective. We hypothesized that bipolar RFCA assisted by CARTO mapping, one of 3D EAM, would effectively treat refractory VT caused by LVA or scar after MI. Consequently, the patient underwent CABG with bipolar RFCA assisted by CARTO mapping with good postoperative outcomes.

2. Case Report

A 50-year-old man with a history of MI experienced intermittent VT over 10 times in the previous month, and his cardiac function was grade IV, according to NYHA heart failure class. Antiarrhythmic drugs were ineffective. Approximately 70%–80% proximal stenosis was diagnosed by coronary angiography in the left anterior descending branch (LAD), with ejection fraction (EF) 31%; left ventricular end-diastolic diameter (LVEDD), 62 mm; left atrium (LA), 50 mm; and LVA, 61 × 25 mm. Electrocardiogram showed that VT originated from the left ventricle. The left ventricle and right ventricle were reconstructed by the CARTO system. 10-pole IBI CS electrodes were implanted into the right femoral vein. VT was prone to recurrence after right ventricle apex (RVA) S1 300 ms stimulation. One VT was LBBB (CL=250ms), and the other

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VT was RBBB (CL=460ms). VT activation and voltage mapping were conducted under the guidance of CartoSound. The two VTs were all LVA-related reentry. There were significant delayed potentials at the junction between the edge of the LVA and the normal myocardium and in the center of the VA. The first type of LBBB VT was related to the internal reentry of LVA, and the second type of RBBB VT was related to the reentry of normal myocardial junction between the edge of LVA and the left ventricular anterior wall. The above two reentries and all delayed potentials in the interior and edge of LVA were ablated to disappear by unipolar RFCA (**Figure 1**). VT could not be induced by RVA stimulations. Three days later, VT the same as original VT, recurred approximately 20–40 times a day, all requiring electro-cardioversion.

CABG was performed via the great saphenous vein (GSV) to the LAD under beating-heart cardiopulmonary bypass. The LVA (approximately 80 × 40 mm) was opened for removing mural thrombus (about 10g) in LVA, and scattered scars in LVA was approximately 20 mm. Then bipolar RFCA clamps (AtriCure, USA) with eight ablation lines ablated LVA from center to the surrounding myocardium according to the anatomical position of real heart corresponding original CARTO. Also, one ablation line passed through the isthmus of the reentrant circuit of refractory VT. Linear closure was performed with clamping using 2-0 Prolene sutures to repair the LVA **(Figures 2)** .

There were no post-procedural complications. Holter monitoring showed one ventricular premature beat (VPB), 0 VT, and 594 supraventricular tachycardias (SVTs). A beta-blocker was prescribed as an anti-arrhythmic drug at discharge. One year later, Holter monitoring showed 2443 VPBs, one VT (ventricular triple rhythm), and 0 SVT. The EF measured 33%; LVEDD, 59 mm; and LA, 38 mm. Coronary CT showed GSV-LAD patency. Cardiac function was restored to grade I. The patient provided written informed consent for publication of this case report.

The requirement for ethical approval was waived as this is a case report. All procedures were performed per the ethical standards of the institutional and/or national research committee.

3. Discussion

We described the case of a patient with refractory VT secondary to LVA, whose treatment with unipolar RFCA was ineffective; consequent CABG with bipolar RFCA assisted by CARTO mapping resulted in good postoperative outcomes.

RFCA is the main treatment for refractory VT caused by an MI scar. A retrospective study by Heart Rhythm reported on 365 patients with MI complicated with LVA who were treated by RFCA. The results showed that the mortality rate of patients with MI scar area larger than 20% was still 25% one year after successful RFCA. Baseline data analysis of selected cases showed that less than half of the patients underwent CABG before RFCA.[1] It is suggested that if myocardial ischemia is not effectively ameliorated, the risk of malignant LVA after MI is limited by RFCA alone. Also, because the electrophysiological matrix of LVA complicated with LVA can be located in the sub-epicardium and endocardium, as well as in the epicardium, it is difficult to achieve the transmural effect of RFCA in the endocardium alone and, thus, its curative effect is limited. The patient underwent endocardial unipolar RFCA assisted by a cardiologic CARTO mapping system before surgery, which did not effectively curb refractory VT. Hence, we considered three main reasons: 1. The reentrant circuit is not only located in the endocardium but also the epicardium and/or sub-epicardium; 2. The unipolar RFCA pen cannot achieve true wall penetration; 3. The myocardial ischemia of the patient's left ventricular anterior wall has not been solved.

CABG is the preferred treatment for patients with persistent VT caused by the left main coronary artery stenosis and/or multiple vessel lesions. At present, the main surgical treatment for LVA is CABG combined with LVA formation. However, the 2011 AHA guidelines for CABG clearly highlighted that CABG could only effectively reduce ventricular fibrillation/tachycardia caused by myocardial ischemia. However, it does not reduce all factors leading to VA, especially the lack of clinical evidence for the efficacy of patients with VT caused by scarring after myocardial ischemia and necrosis.[3]Chiriac et al.[4]retrospectively analyzed the cure rate of ventricular arrhythmia in 765 patients with coronary heart disease complicated with potential malignant ventricular arrhythmia after coronary revascularization and simple antiarrhythmic therapy. The

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study showed a nearly 50% incidence of potential malignant ventricular arrhythmia, even after coronary revascularization and drug treatment. The absence of statistically significant differences between the two groups suggested that scar reentry may occur. It plays an important role in LVA with ventricular arrhythmia. The preoperative CARTO mapping system showed that the first form of LBBB VT was related to intraventricular aneurysm reentry, and the second form of RBBB VT was related to the reentry of the junction between normal left ventricular anterior myocardium and the edge of LVA. Therefore, simple CABG and LVA formation do not effectively curb the refractory VT produced by the reentrant circuit.

Applying implantable cardioverter defibrillators (ICD) is the most effective method to prevent sudden cardiac death.[3]However, the European ESC/EACTS Guidelines for Coronary Revascularization in 2014 recommend that ICD implantation within 2 years after coronary revascularization does not effectively reduce the incidence of sudden death.[5] Survival curves of 882 patients with and without ICD after CABG were retrospectively analyzed.[6] The results showed that there was no significant difference in the mortality rates between patients with and without ICD within 2 years after CABG. Our patient had 20 to 40 episodes of VT every day before surgery, and all needed electro-cardioversion. If ICD was implanted, pacing of ICD caused by refractory VT would cause immense mental and physical pain to this patient.

LVAs form after acute MI and there are relatively viable muscle islands, necrosis, and fibrous tissues at the junction of normal myocardial tissue and scar tissue of LVA, which constitute a complex interlaced marginal zone, thus changing the original conductivity and refractory period of myocardial cells, leading to inconsistent repolarization of myocardial cells, asynchronous conduction through abnormal pathways, and a ventricular ectopia impulse. Among them, the ectopic origin and a reentrant circuit are considered as the primary electrophysiological basis of ventricular arrhythmia. The ectopic origin can be located in possible areas, such as the endocardium, a few in the epicardium and sub-epicardium, or even in the interventricular septum and papillary muscle. The successful rate of ablation for the earliest ventricular excitation was only 25%.[7] However, no matter how the location and number of ectopic origins change, the abnormal electrical impulse will pass through the reentrant circuit formed by LVA, leading to VT.[8] Therefore, the restraint and destruction of reentrant circuit plays a key role in the treatment of ventricular arrhythmia complicated with VT.

We have attempted to treat patients with ventricular arrhythmia caused by LVA via surgery combined with epicardial unipolar RFCA since 2009.[9] The result has proven that the treatment of LVA with ventricular arrhythmia from the cardiac electrophysiology had important clinical significance. However, intraoperative and postoperative examinations showed that ventricular arrhythmias of some patients were not effectively suppressed, suggesting that due to technical limitations, it is impossible to achieve complete transmural ablation of the endocardium and epicardium. Not only the ablation effect is not ideal, but also a new ectopic origin or reentrant pathway may be created between endocardial and epicardial undergo radiofrequency ablation. Therefore, exploring effective surgical electrophysiological therapy has become a bottleneck in the treatment of LVA. The key to bipolar RFCA is stable power, easy operation, and the power is automatically cut off when the ablation reaches the wall penetration, which could ensure an optimal transmural effect. It can also be used under beating-heart conditions.[10] Determining the range of bipolar RFCA is necessary. The location of the LVA and isthmus mapped by CARTO effectively determines the anatomical location of the reentrant circuit and the scope of subsequent ablation.[1] The procedure used in the present case effectively decreased refractory VT caused by LVA.

The methods determining the range of radiofrequencies for LVA ablation with bipolar RFCA forceps are significant. In endocardial RFCA, intracardiac ultrasound was used earlier to accurately determine the anatomical location of LVA. Second, the excitation, traction mapping, and voltage mapping were performed at the onset of VT and sinus rhythm, respectively. Further, the locations of LVA and isthmus were determined, and the isthmus was ablated, and LVA isolated. However, viable myocardium may remain in the LVA and its margin, and VT may still occur after primary unipolar RFCA due to myocardial edema after local ablation. However, in our case, the full reconstruction and measurement of the endocardial surface effectively guided the surgeon to determine the anatomical location of the reentrant circuit produced by LVA and provided

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an important reference for the location and scope of subsequent ablation. We used 8 RFCA lines with the LVA as the center and the ablation margin, at least around the myocardium. One of the RFCA lines passed through the key isthmus of the VT reentrant circuit mapped by the CARTO system to cut off the separate reentrant circuit formed by the LVA to the greatest extent. The results of the one-year follow-up showed that this method was effective.

4. Conclusions

We described the approach for diagnosis and surgical treatment of LVA with refractory VT by CARTO mapping-guided bipolar RFCA, combined with CABG. This approach was able to minimize the incidence of refractory VT and could be regarded as a new treatment for LVA complicated by refractory VT.

Acknowledgments

All authors would like to thank the patient for giving consent for publication of this case and accompanying images and Editage (www.editage.cn) for English language editing.

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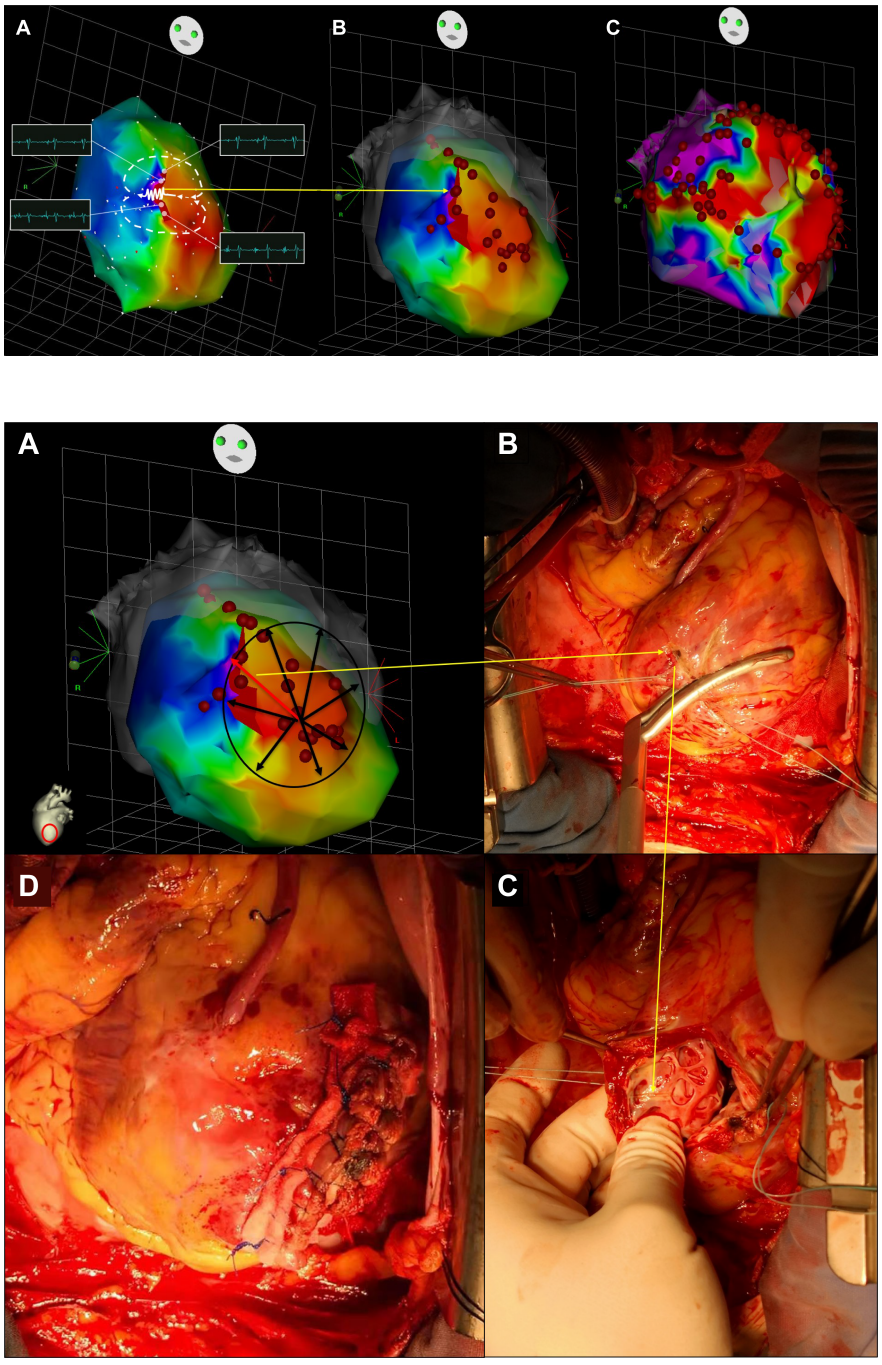
Figure legends:

Figure 1 . **A:** The excitation signal was delayed in the key isthmus (white solid line). The excitation signal was transmitted along the first side of the isthmus (white dotted line); B: Two reentries, key isthmus (red dots pointed by yellow arrow) and all delayed potentials in the interior and edge of LVA were ablated to

disappear by unipolar RFCA; C: Using unipolar RFCA to isolated LVA anatomically (red dots).

Figure 2 . A: CARTO-mapped ventricular tachycardia (VT) reentrant circuit and key isthmus (red arrow); B: Eight radiofrequency ablation lines were performed on the LVA using a bipolar RFCA, with one passing through the key isthmus of the reentrant circuit (yellow arrow); C: The reentrant loop area and bipolar RFCA line through the key isthmus of the reentrant circuit (yellow arrow); D: After bipolar RFCA, LVA was performed for linear closure.



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