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CASE REPORT
Electrophysiology

Transcoronary mapping using a guidewire during transcoronary ethanol ablation for ventricular tachycardia with a deep intramural substrate: a case report

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Background Transcoronary ethanol ablation is effective in treating ventricular tachycardia (VT) in the deep myocardium. The selection of the target coronary artery plays an important role in the success of transcoronary ethanol ablation. Transcoronary mapping, using a guidewire, may be effective for identifying the target coronary artery.

Case summary A 72-year-old man, who had undergone thrombolytic therapy for acute myocardial infarction 40 years ago, was admitted to the emergency department with a chief complaint of syncope. Five years ago, a cardiac resynchronization therapy defibrillator was implanted for a left bundle branch block (QRS duration 153 ms), with New York Heart Association Class Ⅲ and a left ventricular ejection fraction of 30%.

Due to VT, he experienced a critical deterioration in his vital parameters, leading to shock. The first VT ablation was performed on the 3rd day of hospitalization. Activation mapping showed that the earliest activation site was located in the mid-anterior septum of the left ventricle. Mapping from the endocardial surface showed no mid-diastolic potential around the VT. Radiofrequency catheter ablation therapy was performed at the targeted site, resulting in transient termination of VT. However, the VT showed recurrence the next day. A transcoronary ethanol ablation was performed on the 10th day of hospitalization. A 0.014 inch guidewire and microcatheter were advanced into the target coronary septal branch, and the myocardial septum was mapped. The guidewire-assisted transcoronary mapping showed a potential 43 ms ahead of QRS onset during VT. The coronary septal artery branch was considered the target artery, and 0.5 mL of ethanol was injected. No further VT was observed for 12 months after the transcoronary ethanol ablation.

Discussion Transcoronary ethanol ablation is considered in cases where a deep intramural substrate is suspected or when early activation at the interventricular septum is identified. Guidewire-assisted transcoronary mapping allows mapping of VT with deep intramural substrates and may be useful in selecting target coronary arteries while performing transcoronary ethanol ablation.

Keywords Transcoronary mapping • Ventricular tachycardia • Transcoronary ethanol ablation • Case report

ESC curriculum 5.6 Ventricular arrhythmia • 5.11 Cardiac resynchronization therapy devices

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Learning points

- Transcoronary ethanol ablation is effective in treating ventricular tachycardia in the deep myocardium.
- The selection of the target coronary artery plays an important role in the success of transcoronary ethanol ablation.
- Transcoronary mapping using a guidewire may be effective for identifying the target coronary artery.

Introduction

Ventricular tachycardia (VT) caused by scarring is the most common mechanism of VT in patients with ischaemic cardiomyopathy. Radiofrequency catheter ablation (RFCA) is a common procedure used to treat patients with VT.[1] However, VT cannot be eliminated using both endocardial and epicardial approaches, and intramural ablation is required in some patients. A transcoronary ethanol ablation (TCEA) is performed[2] when endocardial and epicardial RFCA fails or the presence of a deep intramural substrate is anticipated. However, inadequate target vessels, collaterals, and recurrence of modified VTs limit the efficacy of TCEA.[3]

Summary figure

Time	Events
Forty years ago	He underwent thrombolytic therapy for an acute myocardial infarction.
Five years ago	A cardiac resynchronization therapy defibrillator (CRTD) was implanted for a left bundle branch block (QRS duration 153 ms), New York Heart Association Class Ⅲ, and a left ventricular ejection fraction of 30%.
Hospitalization	He underwent shock in his vital parameters as a result of ventricular tachycardia with a cycle length of 400 ms.
Day 3	The first ventricular tachycardia (VT) ablation was performed by radiofrequency catheter ablation (RFCA). Recurrence of VT was observed.
Day 10	The second VT ablation was performed. A transcoronary ethanol ablation (TCEA) was performed.
12 months	No further VT was observed for 12 months after the TCEA.

Case presentation

A 72-year-old man was admitted to the emergency department with a chief complaint of syncope. He underwent thrombolytic therapy for an acute myocardial infarction 40 years ago. A cardiac resynchronization therapy defibrillator (CRTD) was implanted for a left bundle branch block (QRS duration 153 ms), with New York Heart Association Class Ⅲ and a left ventricular ejection fraction of 30% 5 years ago. He experienced sustained VT with multiple CRTD shocks 3 years ago. Treatment with amiodarone was initiated, but it caused pancytopenia and had to be discontinued. Thus, other antiarrhythmic drugs such as sotalol 80 mg/day and mexiletine 450 mg/day were initiated as alternative treatments. One hour before arriving at our hospital, he showed haemodynamically unstable VT with a cycle length of 400 ms. Upon admission, he was sedated and intubated due to frequent VT. A 12-lead electrocardiogram (ECG) revealed an upper-axis morphology, characterized by a cycle length of 400 ms. The tachycardic condition was discerned through the presence of rS in V1 and a positive waveform in lead 1 induction (*Figure 1*). An echocardiographic examination revealed a severely reduced ejection fraction of 25%, and the left ventricular anterior wall exhibited reduced wall motion and absence of thinning. A coronary angiography revealed no new coronary artery stenosis. The first VT ablation was performed on the 3rd day of hospitalization. Multi-electrode catheters were positioned at the recording lesion, right ventricular apex, and anterior interventricular vein. An electroanatomical mapping was performed using a three-dimensional mapping system (CARTO 3 System; Biosense-Webster, Diamond Bar, CA, USA). Intracardiac ECG during the VT showed atrioventricular dissociation. The activation mapping showed that the earliest activation site was located in the mid-anterior septum of the left ventricle (*Figure 2*).

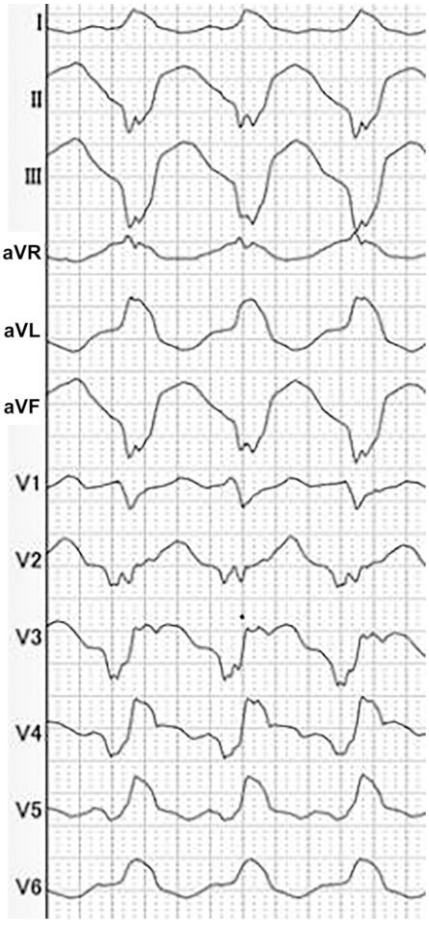

Figure 1 Target ventricular tachycardia.

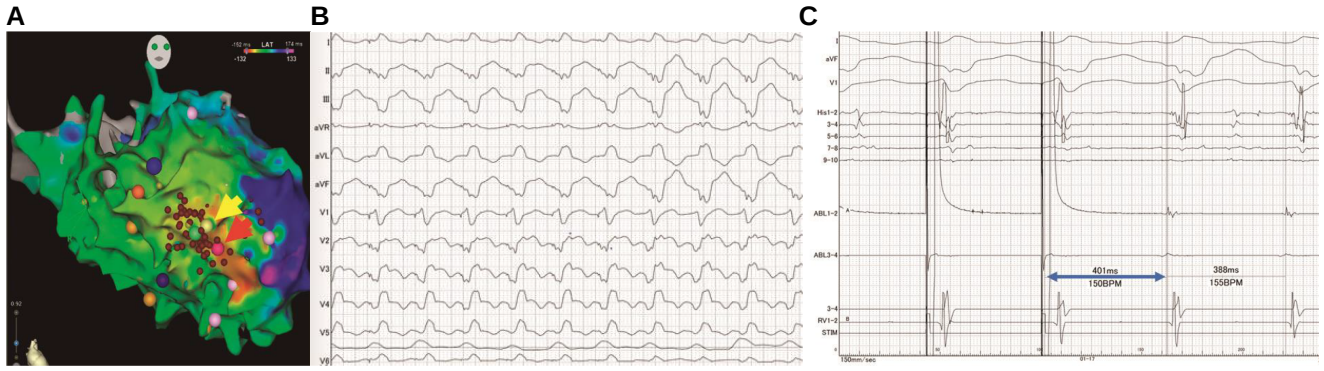

Figure 2 (*A*) Activation mapping shows that the earliest activation site was located at the mid-anterior septum of the left ventricle. Entrainment pacing was performed from this site. (*B* and *C*) Show concealed fusion, and the post-pacing interval was consistent with the ventricular tachycardia cycle length.

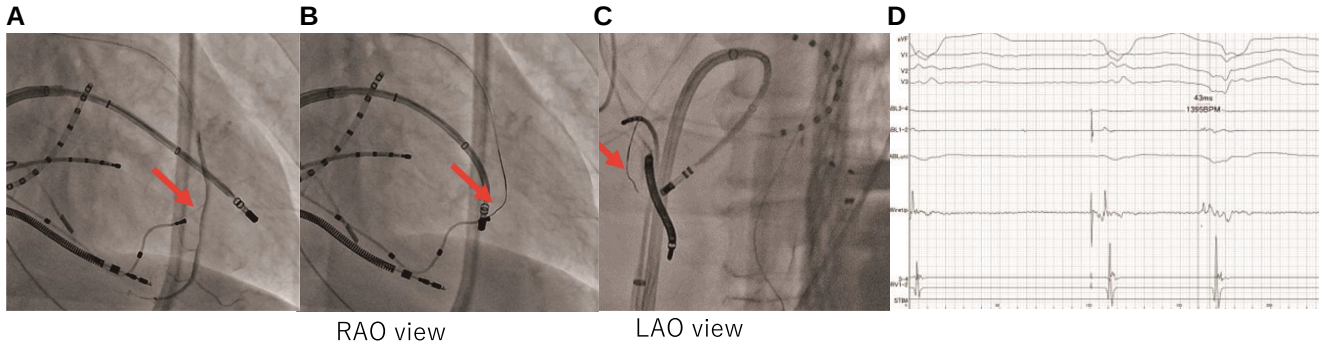

Figure 3 (*A*, *B*, and *C*) Coronary angiography reveals that three septal branches are from the distal part of the left anterior descending artery. A 0.014 inch guidewire and a microcatheter are advanced into the target coronary septal branch. (*D*) Transcoronary mapping using the guidewire shows a potential 43 ms ahead of QRS onset during ventricular tachycardia and ventricular tachycardia exit shown by the ablation catheter on the endocardial surface, and furthermore, the potential is fragmented.

Entrainment pacing was performed from the excitation site, which showed concealed fusion with a post-pacing interval almost consistent with the cycle length of the tachycardia. The ratio of the stimulus to the QRS interval and the total cycle length was <0.3 VT. This was determined to be the exit point of the VT circuit.[4] Mapping from the endocardial surface showed no mid-diastolic potential around the VT. Thus, the deep myocardium was thought to contain this circuit. Radiofrequency catheter ablation therapy was administered at the targeted site, resulting in transient termination of VT. However, the VT showed recurrence the next day. Furthermore, the calcification of the ventricular septum was thought to be the cause of the lack of efficacy of RFCA to the target circuit. On the 10th day of hospitalization, a second VT ablation was performed. However, this time, a TCEA was performed because of the anticipated presence of a deep intramural substrate. As observed previously, the earliest site of activation remained the same, i.e. the left ventricular septum. A 0.014 inch angioplasty guidewire (Sion®, Asahi Intec, Tokyo, Japan) and a microcatheter (Caravel microcatheter, Asahi Intec, Tokyo, Japan) were advanced into the target coronary septal branch (*Figure 3*), and the myocardial septum was mapped using the unipolar potential of the wire as an indicator. Microscopic arteries stemming from the myocardial septum were subjected to contrast-enhanced imaging through chip injection utilizing a microcatheter. One of the electrodes on the catheter, positioned at the right ventricular apex, was employed as a blind electrode, with the guidewire connected to the cathode. The distal portion of the guidewire, spanning <5 mm, remained devoid of microcatheter coverage. Hence, it had the capacity to selectively myocardial potentials. The guidewire-assisted transcoronary mapping showed a potential that was 43 ms ahead of QRS onset during the VT and VT exit shown by the ablation catheter on the endocardial side, and the potential was fragmented. The coronary septal artery branch was considered as the target artery, and 0.5 mL of 100% ethanol was injected. No further VT was observed for 12 months after the TCEA.

Discussion

Ventricular tachycardia due to ischaemic cardiomyopathy often shows substrates on the endocardial surface, and mapping from

the endocardial surface can identify the VT circuit.[5] Although RFCA usually reduces VT recurrence, it continues to fail in a number of patients. When RFCA fails, TCEA is considered in cases where a deep intramural substrate is suspected, or when early activation at the interventricular septum is identified. In the present case, the activation mapping showed that the earliest activation site was located in the mid-anterior septum of the left ventricle. Although the TCEA is an effective procedure, it is reported to cause problems related to myocardial damage or atrioventricular block.[6] Selective ethanol infusion can minimize myocardial damage and avoid infusion into the vessels supplying the atrioventricular node. In the present case, although the VT outlet could be identified from the endocardial surface, using entrainment pacing, the critical isthmus of conduction could not be identified. We identified the artery near the exit site of the VT circuit by mapping from the endocardial surface. Transcoronary mapping using the unipolar potential of the wire as an indicator was useful for detecting the culprit artery. The unipolar potential was effortlessly demonstrated in comparison to the bipolar potential. Additional guidewire insertion for an anode was required for bipolar pacing, whereas the addition of a guidewire to the small branch of the coronary artery proved to be a challenging procedure. One drawback of the unipolar potential was the occurrence of artefacts. Placing the microcatheter closer to the tip of the guidewire mitigated artefacts in the present case. Ablation to the central isthmus is essential to treat VTs using TCEA as well as RFCA. Entrainment pacing is a typical method to determine the central isthmus of the VT circuit, but in this case, entrainment pacing was not possible due to the high pacing threshold in the scar area. This was a limitation of this case report. Planned TCEA is discontinued in ~19% patients owing to anatomic limitations.[3] Technical limitations of TCEA are more likely to occur in patients with multivessel coronary artery disease or a previous coronary artery bypass grafting. Guidewire mapping allows the TCEA to be more selective in targeting coronary arteries because of its ability to map potentials within the intramural substrate. In the present case, VT ablation was successful with only 0.5 mL of ethanol injected into the target coronary artery. In conclusion, transcoronary mapping using a guidewire allows the mapping of VTs with deep intramural substrates and may be useful in selecting target coronary arteries when performing TCEA.

Lead author biography

Dr Suguru Chiba is a cardiologist with a clinical focus on arrhythmia electrophysiology, who graduated from Ryukyu University Graduate School of Medicine in 2013.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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Data availability

The data underlying this article will be shared upon reasonable request to the corresponding author.

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