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CASE REPORT
Electrophysiology

Open-chest epicardial ablation of ventricular tachycardia during a left ventricular assist device implantation: a case report

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Background Ventricular arrhythmias (VAs) are common after a left ventricular assist device (LVAD) implantation. Further, the majority of post-LVAD ventricular tachycardias (VTs) are secondary to a preexisting cardiomyopathy substrate. Intraoperative ablation of patients with recurrent preoperative VTs may reduce post-LVAD VTs.

Case summary A 59-year-old female with advanced heart failure due to non-ischaemic cardiomyopathy (LV ejection fraction = 24%) and recurrent VTs was referred for an LVAD implantation as a bridge to a heart transplant (INTERMACS Profile-5A). The previous endocardial ablation failed due to an epicardial arrhythmogenic substrate. Therefore, open-chest epicardial mapping during the LVAD implantation was indicated and three target areas of the arrhythmogenic substrate were found, which were ablated by radiofrequency applications. To minimize the cardiopulmonary bypass time, cardiopulmonary bypass was initiated after ablation, and then, an LVAD was implanted. An additional 68 min was required for mapping and ablation. All procedures were performed without any complications, and the post-operative course was uneventful. Thereafter, no VT episodes were observed without any anti-arrhythmic drugs during a 15-month follow-up with LVAD support.

Discussion Intraoperative epicardial mapping and ablation during an LVAD implantation can play an important role in the management of LVAD recipients with recurrent VAs.

Keywords Left ventricular assist device • Ventricular tachycardia • Ablation • Open-chest mapping • Epicardial mapping • Case report

ESC Curriculum 5.6 Ventricular arrhythmia • 6.2 Heart failure with reduced ejection fraction • 5.10 Implantable cardioverter defibrillators • 6.5 Cardiomyopathy • 7.5 Cardiac surgery

Learning Points

- Intraoperative epicardial ablation of ventricular tachycardia (VT) during a left ventricular assist device (LVAD) implantation procedure could be safely performed and suppress post-LVAD VT recurrences.
- Preprocedural planning to minimize the cardiopulmonary bypass time is essential to reduce peri-operative complications and facilitate the patient's prompt recovery.

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Introduction

Ventricular arrhythmias (VAs) are common after left ventricular assist device (LVAD) implantations. Further, the majority of post-LVAD ventricular tachycardias (VTs) are secondary to a preexisting cardiomyopathy substrate rather than to the LVAD cannula[1,2]. Endocardial, epicardial, and surgical ablation procedures performed before, during, and after LVAD implantations have been reported.[3–9] However, the ideal management remains to be debated.[1,2] Here, we describe our experience of open-chest epicardial ablation during an LVAD implantation.

Timeline

2002 (40 years old)	She was diagnosed with idiopathic dilated cardiomyopathy [left ventricular ejection fraction (LVEF) = 43%, LV end-diastolic/systolic dimension (LVDd/Ds) = 63/49 mm].
2006	First presentation of ventricular tachycardia (VT). A transvenous implantable cardioverter defibrillator (ICD) was implanted, and shock lead perforation occurred 4 days after its implantation. Then, a subxiphoid surgical drainage procedure was required.
2007	Inappropriate ICD shock therapy occurred due to lead failure, and additional shock lead was implanted.
2009	Isolated VT episode requiring an ICD shock (LVEF = 36%, LVDd/Ds = 64/54 mm).
November 2016	LVEF = 27%, LVDd/Ds = 66/55 mm.
September 2019	Six episodes of asymptomatic VT were terminated by anti-tachycardia pacing.
November 2020	VT requiring ICD shock therapy (LVEF = 26%, LVDd/Ds = 71/63 mm).
March 2021	Heart failure's aetiology was re-screened, and no suspicious findings indicating secondary cardiomyopathy were found. Contemporary guideline–directed medical therapy was administered with a maximum tolerated dose.
July 2021	Endocardial ablation was performed for VT electrical storm, but the presence of epicardial substrate was suspected.
October 2021 (this admission)	VT electrical storm recurred. Left ventricular assist device (LVAD) was indicated as a bridge to heart transplantation for advanced heart failure and recurrent VT electrical storms.
During procedure	Epicardial mapping and ablation were performed during the LVAD implantation.
March 2023	Waiting for the transplantation without any VT episodes.

Case presentation

The patient was a 59-year-old female with advanced heart failure due to non-ischaemic cardiomyopathy. She had been healthy, but an abnormal electrocardiogram was detected by an annual health exam in 2002 when she was 40 years old. Cardiac ultrasound revealed a decreased LV ejection fraction (LVEF) of 43% and dilated LV [LV end-diastolic/systolic dimension = 63/49 mm (mean values in Japanese healthy females are 44 ± 3 and 28 ± 3 mm, respectively)]. She was diagnosed with idiopathic dilated cardiomyopathy after close examination, including coronary angiography and myocardial biopsy. In 2006 at the age of 44, sustained VT occurred and an implantable cardioverter defibrillator (ICD) was implanted. A shock lead perforation occurred 4 days post-implantation, and the lead was re-positioned in the right ventricular septum. During that procedure, the pericardial effusion was drained by a subxiphoid surgical approach. The electrocardiogram during sinus rhythm is shown in *Figure 1A*. She had no family history of cardiac disease or sudden death.

Thereafter, her LVEF gradually decreased [24%, LV endo-diastolic/systolic volume index = 158/120 mL/m^2 (mean values in Japanese healthy females are 49 ± 11 and 17 ± 5 mL/m^2, respectively)] and her heart failure status progressed to an advanced stage despite being medicated with carvedilol (5 mg/twice daily) and enalapril (2.5 mg/once daily). In March 2021, the heart failure's aetiology was systemically re-screened. Blood tests revealed no elevated high-sensitive C-reactive protein [<0.01 mg/dL (reference value < 0.14)] nor high-sensitivity Troponin-I [15 pg/mL (reference value < 24)] levels. Coronary angiography and ^{18}F-fluorodeoxyglucose positron emission tomography were performed; however, no significant coronary stenosis or suspicious findings indicating secondary cardiomyopathy were evidenced. Spironolactone (25 mg/once daily), azosemide (30 mg/once daily), and dapagliflozin (10 mg/once daily) were additionally administered. Her systolic blood pressure was 66–80 mmHg; therefore, those drugs were considered to be the maximum tolerated dose and sacubitril/valsartan could not be administrated. The electrocardiogram demonstrated an intraventricular conduction disturbance with a QRS duration of 145 ms (*Figure 1B*). Additionally, no mechanical LV dyssynchrony was evident, so cardiac resynchronization therapy was not indicated.

In July 2021, she was readmitted for multiple VTs (*Figure 2*). The VT electrocardiogram demonstrated a left bundle branch block pattern with a right inferior axis and V2 pattern break, suggesting the LV summit as the VT exit. An intravenous administration of amiodarone could not suppress the VT, and it degenerated into ventricular fibrillation (VF) during anti-tachycardia pacing. An ICD shock delivery failed to terminate the VF, but it was terminated by external defibrillation shocks (see Supplementary material online, *Figure S1*). Thereafter, deep sedation and mechanical ventilation suppressed the electrical storm.

She was weaned from mechanical ventilation within a few days, and then, endocardial ablation was attempted. Small patchy arrhythmogenic substrates were found on the LV endocardium, but wider substrates were suspected to exist on the epicardium (see Supplementary material online, *Figure S2A/B*). No sustained VTs were induced by programmed stimulation. Substrate-guided radiofrequency ablation was performed (see Supplementary material online, *Figure S2A*).

In October 2021, she experienced severe discomfort and then had transient loss of consciousness. In the emergency room, her consciousness level was alert, systolic arterial pressure was 86 mmHg, and heart rate was 66 beats/min. An ICD intracardiac electrogram revealed that the VT had degenerated into VF requiring an ICD shock. No facial or leg oedema was observed; however, she complained of general fatigue on minimal exertion. Further, her weight gradually decreased to 40 kg, reflecting cardiac cachexia (height 163 cm and body mass index 15 kg/m^2). Brain natriuretic peptide ranged from 351 to 654 pg/mL (reference value < 18.4) during the 3 months prior to this admission.

Oral amiodarone (100 mg/once daily) was administrated; however, it was suspected that further VT episodes might disrupt her haemodynamics requiring mechanical circulatory support.[10] Cardiogenic shock before LVAD implantations is associated with a poor

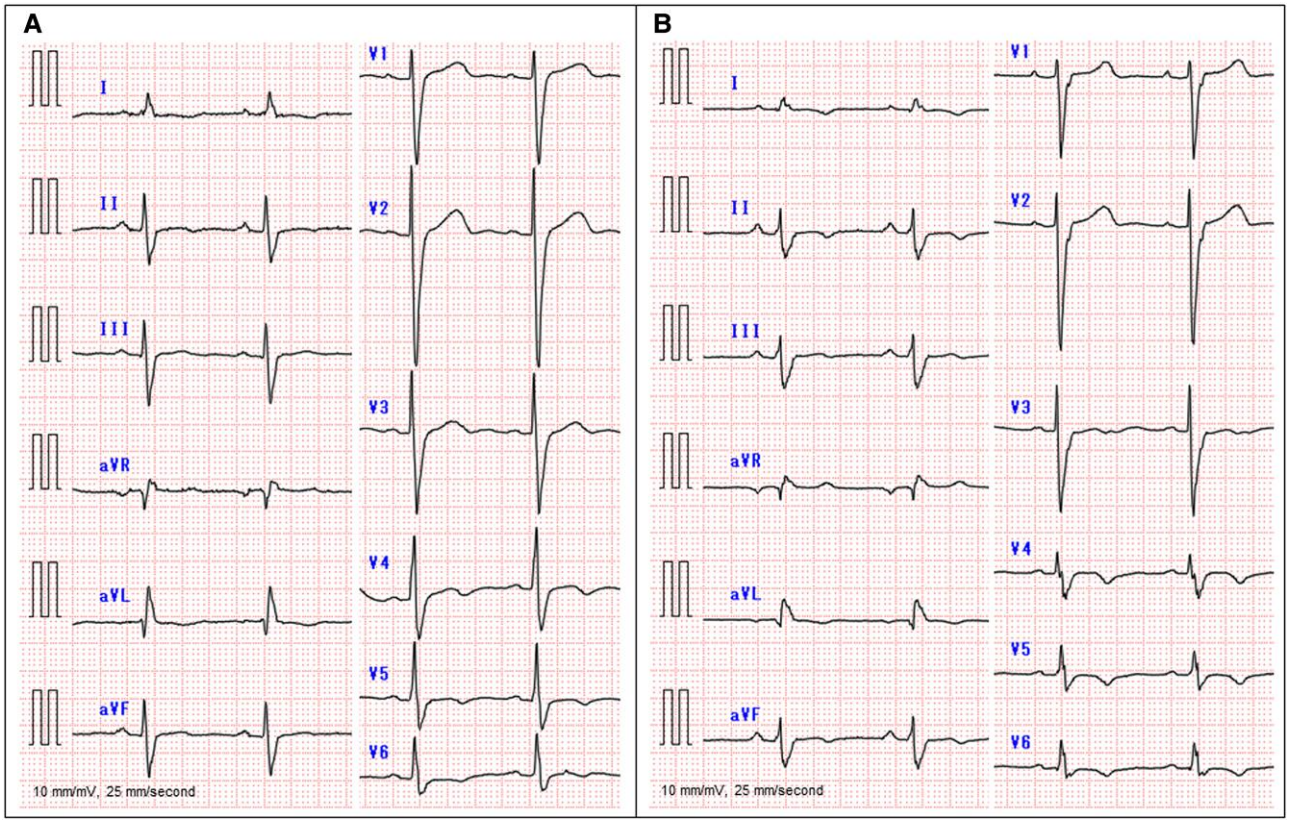

Figure 1 Twelve-lead electrocardiograms in 2006 and 2021. The electrocardiogram during the 1st admission to our hospital in 2006 demonstrated an intraventricular conduction disturbance with a QRS duration of 122 ms (*A*). The QRS duration prolonged to 145 ms in 2021 (*B*), reflecting her disease progression over the past 15 years.

prognosis.[10,11] Therefore, we decided that an LVAD was indicated as a bridge to heart transplantation (INTERMACS Profile-5A).[12] Controlling VAs in LVAD patients is essential for achieving better outcomes during the waiting period for a transplantation, which is estimated to be more than 5 years in Japan.[13] Percutaneous epicardial catheter ablation would have been difficult due to pericardial adhesions because she underwent subxiphoid surgical drainage for an ICD lead perforation. Therefore, an open-chest epicardial ablation during the LVAD implantation was indicated.

To minimize the cardiopulmonary bypass (CP-bypass) time, the procedural plan was decided as follows: (i) general anaesthesia would be initiated, (ii) a mid-sternotomy would be performed, (iii) the major vessels would be exposed, (iv) a drive line would be created, (v) epicardial mapping and ablation would be performed, (vi) CP-bypass would be initiated, and finally, (vii) the LVAD would be implanted.

The location pad for the three-dimensional (3D) map was attached below the bed with touch fasteners. The electrode patches (see Supplementary material online, *Figure S3*) and limb leads were attached so as not to interfere with the surgical field. As planned, after procedures (i)–(iv), epicardial mapping was performed with an Advisor HD Grid (see Supplementary material online, *Figure S4A*, NAVX precision system, Abbott Laboratories, Chicago, IL). Stable mapping was achieved with a warm saline bath around the exposed heart. The 3D locational data were reliable only when the heart sat in a physiological position, and it was not reliable when we touched or lifted the heart. That was one of the meaningful differences between open-chest and percutaneous epicardial mapping.

The local potentials of interest, such as isolated delayed potentials and local abnormal ventricular activity, were tagged on the 3D map, and three areas of interest could be found (*Figures 3* and *4* and Supplementary material online, *Figure S2C* and *Video*). Radiofrequency was delivered in those three areas (see Supplementary material online, *Figure S4B*) with a maximum power of 20–30 W (FlexAbility™, Abbott Laboratories) until the target potential disappeared. No VA induction was performed owing to time constraints. Then, CP-bypass was initiated and an LVAD (HeartMate 3™, Abbott Laboratories) was implanted. All procedures were performed without any complications, and an additional 68 min was required for mapping and ablation. The post-operative course was uneventful, and she was transferred from the intensive care unit to the general ward 2 days post-procedure. Bisoprolol (2.5 mg/once daily), enalapril (1.25 mg/once daily), and spironolactone (50 mg/once daily) were continued. Due to concern regarding its adverse effects with long-term use,[14] amiodarone was discontinued. Thereafter, no VT episodes were observed during a 15-month follow-up.

Discussion

After LVAD implantations, percutaneous endocardial ablation is popular and epicardial ablation after LVAD implantations by a surgical approach has been reported.[1,2] Recently, the feasibility of intraoperative ablation during LVAD implantations has been reported,[4–9] which reduces post-LVAD VTs in high-risk heart failure patients.[9]

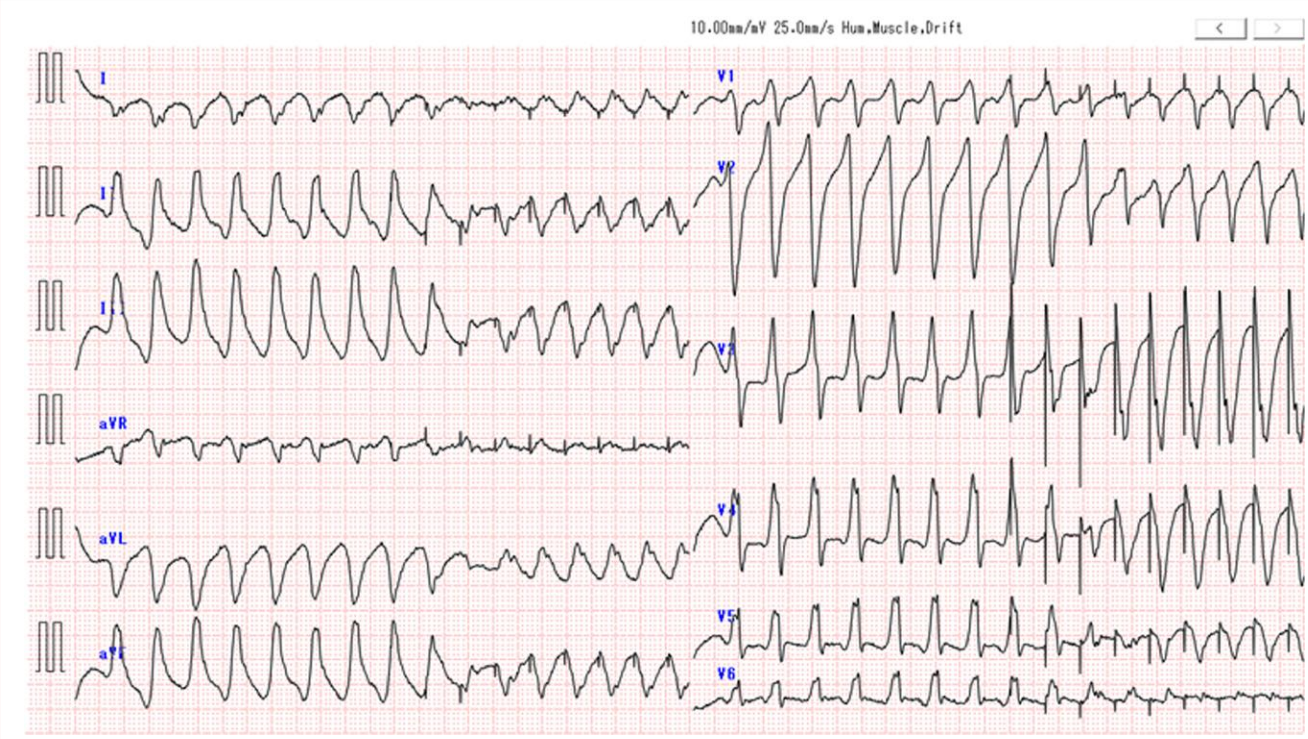

Figure 2 Twelve-lead electrocardiogram during ventricular tachycardia. The 12-lead electrocardiogram during the ventricular tachycardia and while delivering anti-tachycardia pacing is presented.

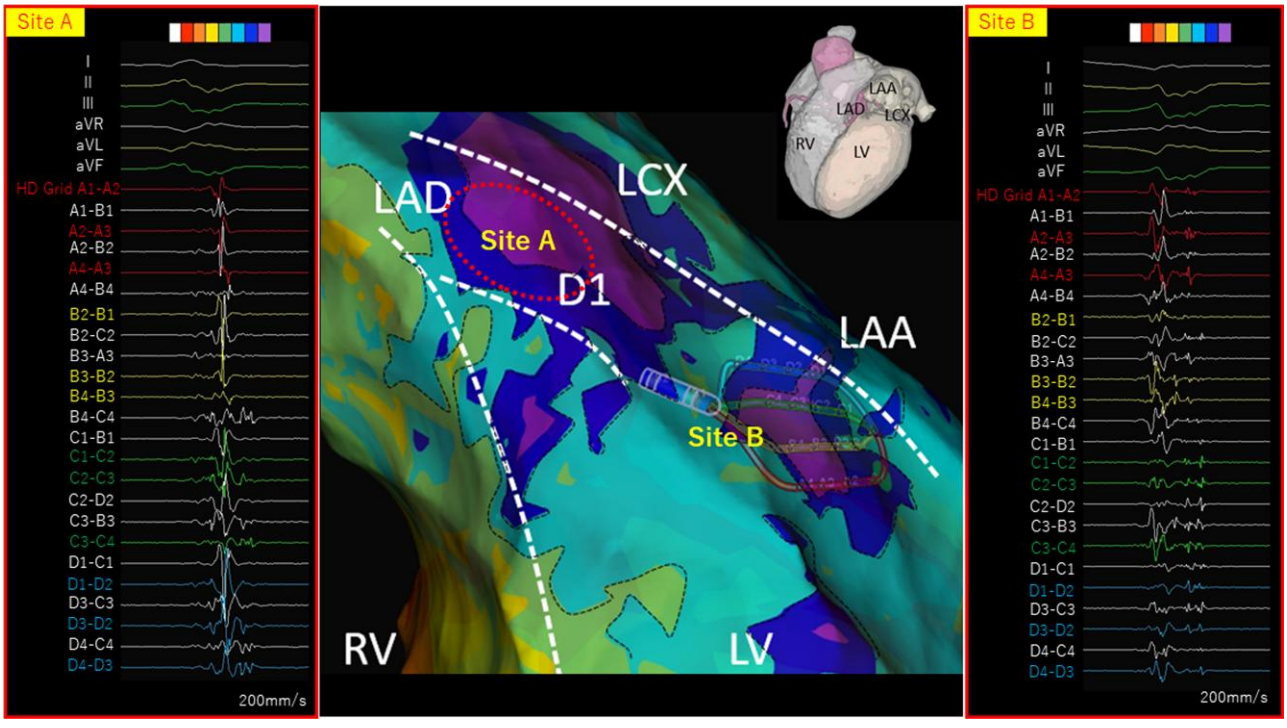

Figure 3 Isochronal late activation map and local potentials at sites A and B. The latest isochronal activation maps in the anteroposterior view (central panel) are presented. Each local potential is presented in the left (site A) and right (site B) panels. Based on the QRS morphology of clinically documented ventricular tachycardia (*Figure 2*), the putative circuit of this ventricular tachycardia would be related to site A. LAA, left atrial appendage; LAD, left anterior descending artery; LCX, left circumflex artery; LV, left ventricle; RV, right ventricle.

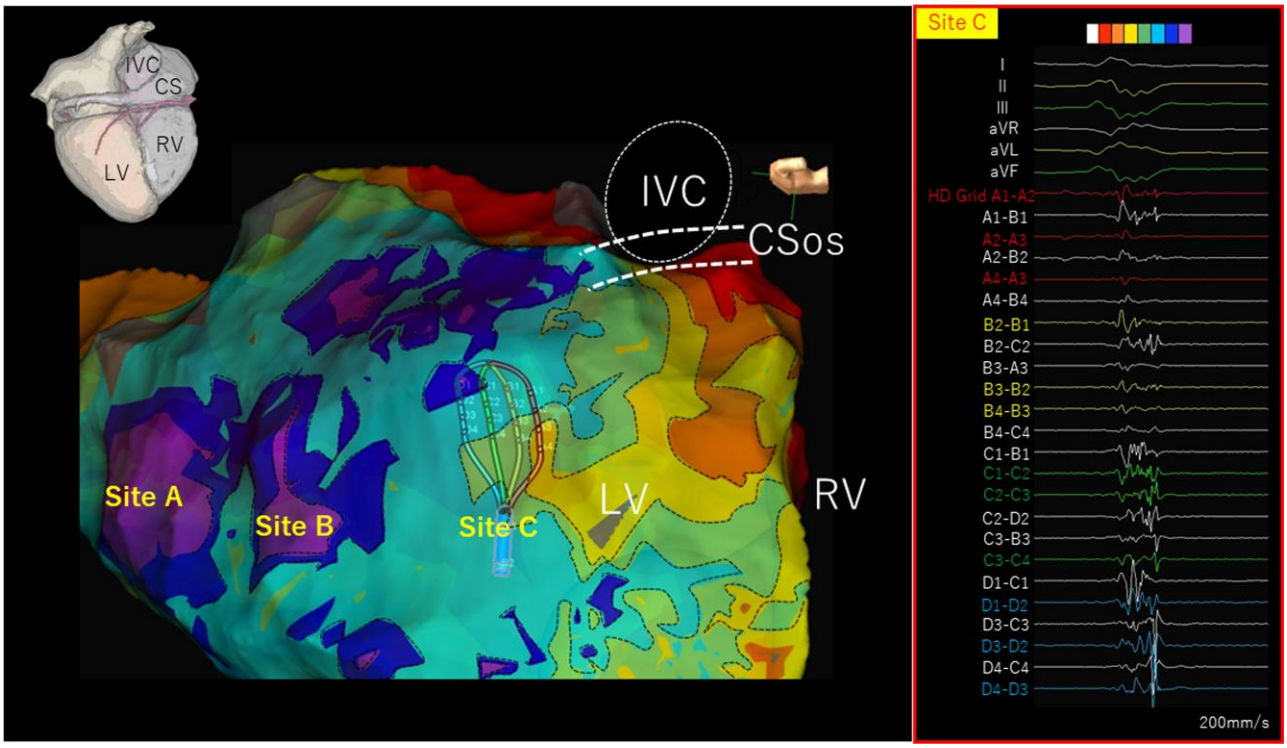

Figure 4 Isochronal late activation map and local potentials at site C. The 3rd area of an abnormal electrogram cluster was found on the left ventricular infero-posterior wall. Multiple discrete delayed potentials were recorded (right panel). CSos, ostium of coronary sinus; IVC, inferior vena cava; LV, left ventricle; RV, right ventricle.

Initially, the target lesion is decided by the VT QRS morphology or previously performed substrate mapping,[4,5,9] minimal mapping, and pace mapping.[9] Localizing the VT exit utilizing the 12-lead electrocardiogram is fundamental, but it does not reveal the targeted re-entry circuit of scar-related VTs. The utility of pace mapping is limited because only the limb leads are available with open-chest mapping. Therefore, high-density 3D mapping is desirable for finding potential arrhythmogenic substrates.[6–8] Mechanical circulatory support is required during VT activation mapping in LVAD recipients.[7,8] Substrate-guided ablation is thought to be reasonable for reducing the CP-bypass time. The procedure time related to ablation requires 45 min.[6,8] It took a little longer in our case, but we think it was an acceptable time for performing adequate mapping and ablation. The VT QRS morphology (*Figure 2*) suggested VT arising from around the LV summit, which was confirmed by the endo/epicardial 3D mapping. The presence of multiple arrhythmogenic substrates, including around the summit, made her treatment highly arduous. Radiofrequency catheter ablation was used in our case.[5,8] The usage of surgical radiofrequency devices[3] and Cryo-energy[4,6,7] has been reported. Cryo-ablation might be appropriate to create larger lesions in a shorter time; however, that was not available at our hospital. The timing of the CP-bypass initiation is important but varies among the previous reports.[4–8] Minimizing the CP-bypass time would reduce any peri-operative complications and facilitate patients' prompt recovery. The annual number of heart transplantations in Japan remains below 100 due to severe organ shortages.[13] As of November 2022, there are 650 Status 1 candidates for transplants and more than 90% have been supported by LVADs. Further, 230 patients have been waiting for more than 5 years.[15] Therefore, careful long-term management of LVADs is mandatory, especially in patients with recurrent VAs. We believe that epicardial ablation during LVAD implantation procedures can play an important role in this situation.

In conclusion, epicardial ablation during an LAVD implantation was performed in a non-ischaemic cardiomyopathy patient with advanced heart failure and recurrent VT storm episodes, which suppressed the VT recurrence during a 15-month follow-up with LVAD support.

Lead author biography

Koji Fukuzawa is the head of the Arrhythmia Centre of Kobe University Hospital.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports*.

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Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

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

Conflict of interest: None declared

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Patient's perspective: None.

Data availability

The data underlying this article are available in the article and in its online supplementary material.

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