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CASE REPORT
Electrophysiology

Hybrid surgical epicardial cryoablation for ventricular tachycardia in the electrophysiology laboratory: a case report

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Background Scar-related ventricular tachycardia (VT) is a challenging medical condition, with catheter ablation providing a valuable treatment option. Whilst most VTs can be ablated endocardially, epicardial ablation is often required in patients with non-ischaemic cardiomyopathy. The percutaneous subxiphoid technique has become instrumental for epicardial access. However, it is not feasible in up to 28% of cases for multiple reasons.

Case summary A 47-year-old patient was managed at our centre for VT storm and recurrent implantable cardioverter defibrillator shocks for monomorphic VT despite maximum drug therapy. No scar was noted during endocardial mapping, with confirmation of the localized epicardial scar on cardiac magnetic resonance imaging (CMR). Following failed percutaneous epicardial access, a successful hybrid surgical epicardial VT cryoablation via median sternotomy was performed in the electrophysiology (EP) laboratory utilizing data from CMR, prior endocardial ablation, and conventional EP mapping. The patient has remained arrhythmia-free for 30 months post-ablation without antiarrhythmic therapy.

Discussion This case describes a practical multidisciplinary approach to managing a challenging clinical problem. Whilst the described technique is not entirely novel, this is the first case report that describes the practicalities and demonstrates the safety and feasibility of hybrid epicardial cryoablation via median sternotomy performed in the cardiac EP laboratory for the sole treatment of VT.

Keywords Hybrid • Epicardial • Cryoablation • Ventricular tachycardia • Myocarditis • Case report

ESC Curriculum 2.1 Imaging modalities • 2.3 Cardiac magnetic resonance • 5.6 Ventricular arrhythmia

Learning points

- Despite evolving tools and techniques, ventricular tachycardia (VT) ablation outcomes in non-ischaemic cardiomyopathy remain suboptimal due to the complexity of the underlying substrate.
- This case report adds to the growing evidence to support hybrid surgical VT ablation in the electrophysiology (EP) lab in skilled centres with careful multidisciplinary input for select patients to deliver precision treatment.
- Whilst not currently endorsed in national and international guidelines, the description of the practical delivery of this technique in the EP lab may support the case for its inclusion in the future and aid successful delivery in experienced centres faced with the management of such complex cases.

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Introduction

Scar-related ventricular tachycardia (VT) is a medically challenging condition associated with significant patient morbidity, mortality, and cost burden to the health service.[1,2] Antiarrhythmic medications are often poorly tolerated with limited effectiveness.[3] Implantable cardioverter defibrillators (ICD) save lives but do not prevent VT from occurring. Painful ICD shocks cause significant anxiety and depression in at least 20% of patients.[4] Repeated shocks in 24 h ('storm') occur in up to 25% and signify a poor prognosis.[5] Catheter ablation (CA) is a percutaneous therapy increasingly used for the successful treatment of VT and is endorsed by international guidelines.[6] Whilst most VTs can be treated with endocardial ablation, epicardial ablation is often required in patients with non-ischaemic cardiomyopathies (NICM).[7]

Since its description in 1996, the percutaneous subxiphoid technique has become instrumental in improving VT ablation outcomes, particularly in NICM.[7] However, it is not feasible in up to 28% of cases due to failed epicardial access, proximity to coronary anatomy/phrenic nerve, or access-related complications.[8,9] We present the case of a successful hybrid epicardial surgical cryoablation via median sternotomy in our electrophysiology (EP) laboratory following failed percutaneous access.

Timeline

Time	Event
5 years prior (initial presentation to local centre)	• Symptomatic 'sinus node disease' • CMR performed and reported as 'normal' locally • Dual-chamber pacemaker implanted
4.5 years prior (presented to our specialist centre)	• First episode of monomorphic VT • Non-invasive investigations identified unifocal PVCs • PVC/VT ablation: no endocardial scar, non-inducible VT, and multifocal PVC morphologies, therefore no ablation performed • Diagnosis unclear • Pacemaker explanted and ICD implanted • Discharged on Bisoprolol 5 mg once daily
4 years prior (care transferred to our centre)	• Further VT and ICD shock • Bisoprolol increased to 10 mg once daily • Repeat CMR non-diagnostic due to the device artefact
3 years prior	• Inpatient admission with VT storm • Regular amiodarone commenced following intravenous loading • Repeat CMR non-diagnostic due to the device artefact • Ablation attempted and abandoned as before—multifocal PVCs, no endocardial scar, or intraoperative clinical PVCs/VT
2 years prior	• No VT—Amiodarone discontinued (also intolerant)
1 year prior	• Further endocardial ablation—no inducible VT and multifocal PVCs. Best pace-map match to clinical PVC at the basal lateral left ventricular (LV) wall but 'normal' endocardial voltage. PVC MDI > 0.55 → no ablation
Hybrid ablation procedure	• No further arrhythmia • Multiple admissions due to VT despite Amiodarone • Repeat CMR (new protocol)—basal to mid-wall lateral epicardial scar extending to the anterior and inferior walls • Percutaneous epicardial ablation unsuccessful • Intolerant of Amiodarone • Subsequent hybrid epicardial ablation performed in the EP lab • Amiodarone stopped 3 months post-ablation
2 years post-procedure	• No further VT or ICD therapies

Patient timeline: 2015–22.
CMR, cardiac magnetic resonance imaging.; VT, ventricular tachycardia; PVCs, premature ventricular complexes; MDI, maximum deflection index; ICD, implantable cardioverter defibrillator; EP, electrophysiology.

Case presentation

A 47-year-old male was referred to our centre following recurrent monomorphic ventricular tachycardia (MMVT) and device therapy.

The patient had no known comorbidities prior to 2015, when he underwent dual-chamber pacemaker implantation for sinus node disease at another centre. No family history of cardiac disease or sudden cardiac death was identified. Late gadolinium-enhanced cardiac MRI (LGE-CMR) was reported as normal at the time. The patient subsequently presented to our centre in the same year with fast symptomatic MMVT. Routine blood tests including electrolytes and thyroid function were normal. Troponin I levels were marginally elevated. Coronary angiography and echocardiography were normal. Drug provocation tests, including Ajmaline and Adrenaline challenges for inherited cardiac conditions, and exercise stress testing were all inconclusive. The patient's pacemaker was explanted, and a secondary prevention ICD was implanted. He was discharged on Bisoprolol 5 mg once daily.

He subsequently presented with recurrent fast symptomatic MMVT (300 ms) in 2016, when the dose of Bisoprolol was maximized to 10 mg once daily. He re-presented with VT storm in 2017, during which the patient was loaded with intravenous Amiodarone and discharged on 200 mg once daily in addition to Bisoprolol.

The LGE-CMR scans in 2016 and 2017 at our centre were uninterpretable due to the ICD artefact. In the absence of a clear underlying diagnosis, an endocardial VT ablation was performed in 2018. No LV low-voltage regions were identified (*Figure 1*) on bipolar voltage mapping, and VT was non-inducible; therefore, ablation was not performed.

In the absence of further VT, Amiodarone was stopped in 2019. However, the patient re-presented in March and April 2020 with VT and ICD shocks associated with significant anxiety and depression. Oral Amiodarone was recommenced, but symptomatic non-sustained

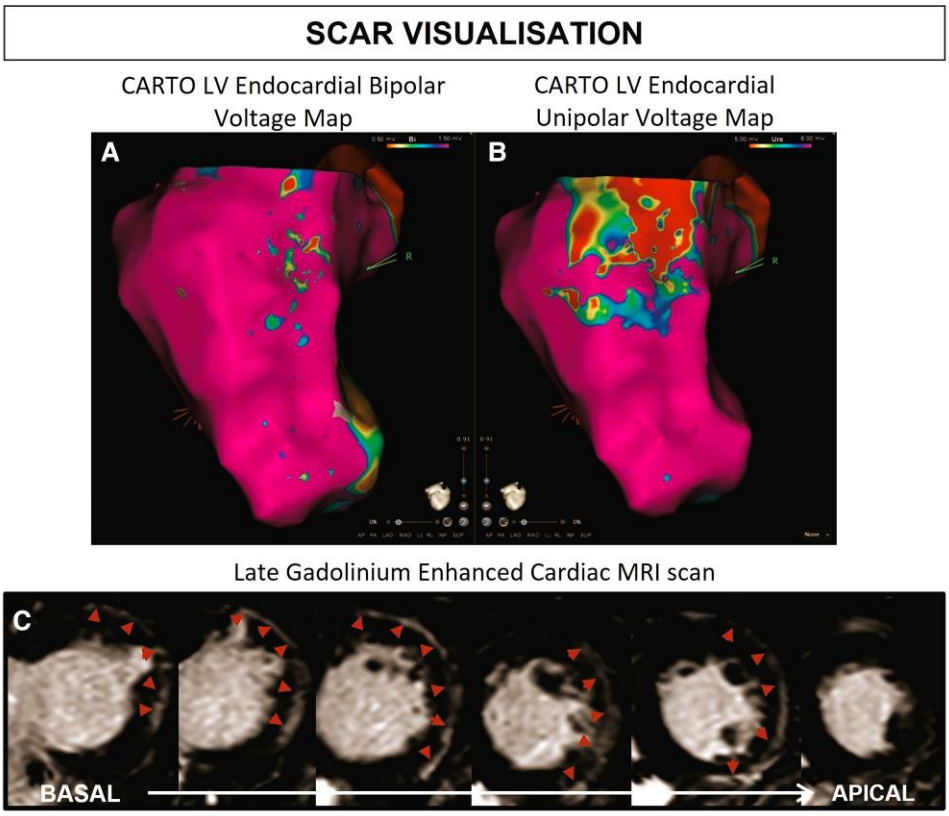

Figure 1 Pre-operative: scar visualisation. (*A*) Inferior view of the patient's endocardial CARTO left ventricular bipolar voltage electroanatomic map from 2018. Predominantly 'healthy' endocardial tissue with a bipolar voltage of >1.5 mV (purple) can be appreciated. Bipolar voltage settings: purple > 1.5 mV = healthy; red < 0.5 mV = scar. (*B*) Same map as (A) displayed using unipolar voltage settings to demonstrate potential epicardial scar. More 'low-voltage' regions are evident (colour range from red to blue = 0–1.49 mV), suggesting the presence of the epicardial scar in keeping with the subsequent late gadolinium-enhanced cardiac MRI findings. Unipolar voltage setting: purple > 8 mV = healthy; red < 5 mV = scar. (*C*) Most recent pre-operative late gadolinium-enhanced cardiac MRI demonstrating epicardial scar (red arrowheads) involving the base to mid-ventricular inferolateral and anterolateral left ventricle, suggestive of probable prior myocarditis. Left to right = basal left ventricle to apical left ventricle. LV, left ventricle.

VT persisted. A repeat LGE-CMR scan, using a different manufacturer and additional imaging techniques, proved successful at providing diagnostic images (*Figure 1*). The LV ejection fraction was calculated at 48%, with the basal to mid-lateral wall LV epicardial scar extending anteriorly and inferiorly, suggestive of prior myocarditis.

The patient underwent percutaneous epicardial VT ablation under general anaesthesia (GA). Despite a BMI of 31.6 and multiple attempts by two trained epicardial operators, percutaneous access was unsuccessful with the use of the standard length (110 mm) and longer length (120 mm) 16-gauge Tuohy needles. Anatomically, a steep rib-to-subxyphoid angle was observed, making epicardial reach challenging. On the final attempt, the needle just reached the epicardial space, as confirmed by contrast pericardiography. However, the wire would not pass freely in the pericardial space. No further attempts were made due to the suspected diagnosis of prior myocarditis and the possibility of pericardial adhesions.

Despite limitations posed by the COVID pandemic, the case was discussed within our multidisciplinary team including cardiac electrophysiologists, cardiac surgeons, anaesthesiologists, perfusionists, and allied health staff. The case was also discussed with the EP and cardiac surgical team at an external tertiary VT ablation centre.

Surgical hybrid ablation options using various minimally invasive surgical access routes were considered. However, the heart would require suspension from the apex for full and unrestricted access to the lateral inferobasal scar, close to the atrioventricular groove, for the delivery of safe and efficient ablation lesions (*Figure 2*). Two potential challenges were considered including the possibility of the presence of significant pericardial adhesions requiring resection and the high risk of arrhythmogenicity, posed by cardiac suspension, leading to haemodynamic instability. These risks would be best safely addressed by performing the procedure 'on-pump' during beating heart surgery, a view supported by the external, independent cardiac surgeon. Procedural risks included those routinely associated with sternotomy and cardiopulmonary bypass surgery including pain, <1% sternal wound infection, prolonged intensive care unit stay, renal failure, and bleeding. The risks, benefits, and potential challenges of minimally invasive vs. open-heart surgical access were discussed with the patient.

Risks were mitigated by close teamwork, precise surgical technique, meticulous haemostasis, and a formal pre-procedural risk assessment including EP lab sterility and air quality.

Epicardial cryoablation

The procedure was performed in the cardiac EP laboratory. In light of the patient's recent VT and our plan to perform substrate-guided

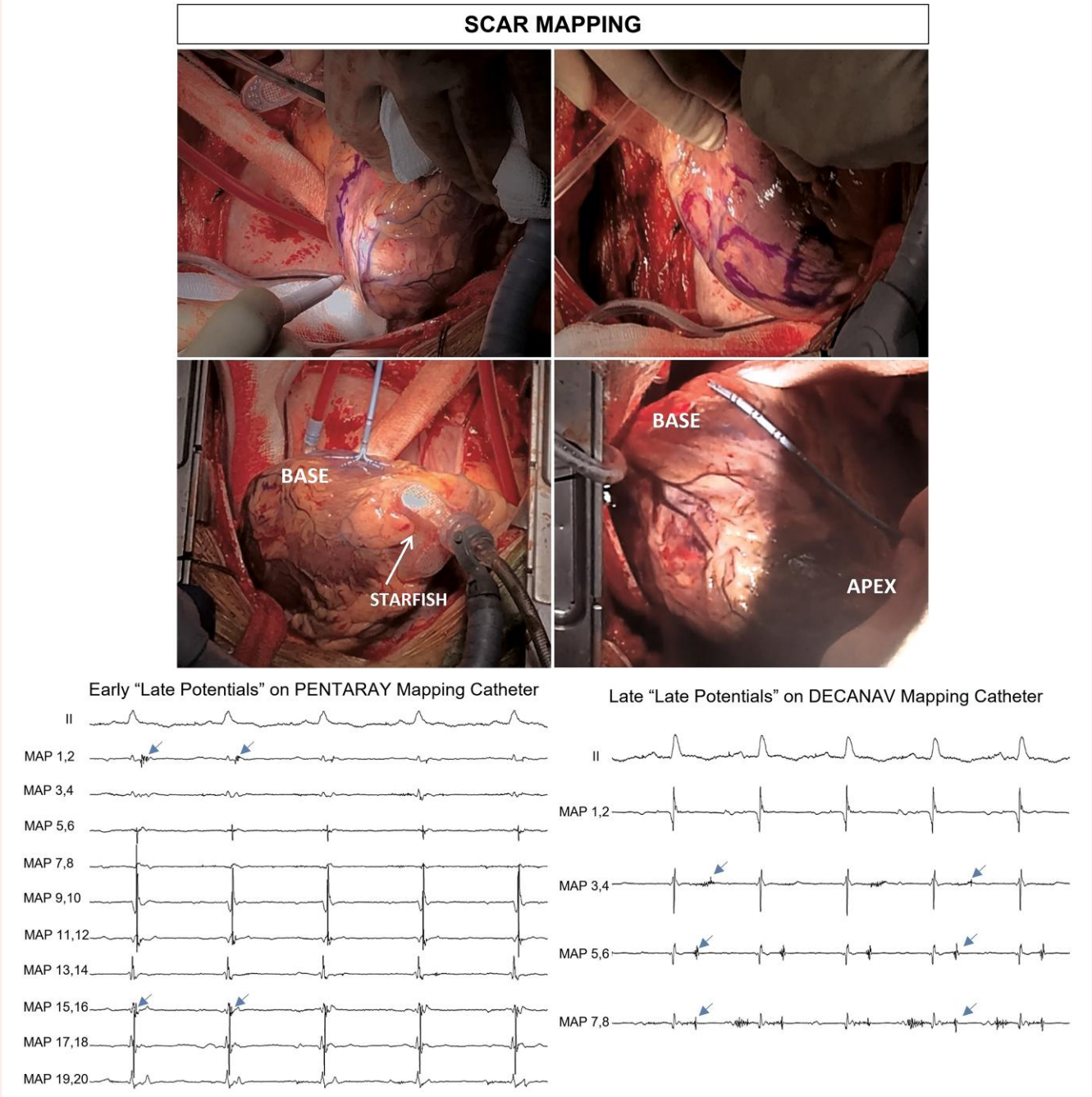

Figure 2 Intraoperative: scar mapping. Top images—macroscopic scar mapping of the epicardial base to the mid-left ventricular surface. Abnormal scar tissue was manually outlined using a sterile purple marker pen. Middle left image—the heart suspended from the apex using the STARFISH device and the Biosense Webster Pentaray™ catheter used for electrophysiology mapping. Middle right image—the Biosense Webster DECANAV® catheter used for electrogram characterization and electrophysiology mapping. Lower panels—late potentials visible on the LABSYSTEM™ PRO BARD EP mapping system during scar mapping. Early late potentials (blue arrows) fused with the far-field electrogram were seen at the inferobasal wall with the Pentaray catheter (left). Later late potentials (blue arrows) were identified on the basal anterolateral wall with the DECANAV catheter (right). LV, left ventricle.

ablation vs. targeted clinical VT ablation, Amiodarone was not stopped. Intraoperatively, the standard V1 and V2 ECG electrodes were placed more laterally, at the fourth intercostal space, with the remaining electrodes placed conventionally. Following GA, right femoro-femoral cardiopulmonary bypass was commenced and median sternotomy was performed. No significant pericardial adhesions were observed.

The Medtronic STARFISH® device was used to suspend the apex allowing full access, clear visualization, and safe manipulation of the entire basal LV region. As we had predicted, attempts to perform three-dimensional (3D) electroanatomic mapping failed due to interference from the surgical equipment. However, under direct visualization, the scar was easily identifiable, extending from the left atrioventricular

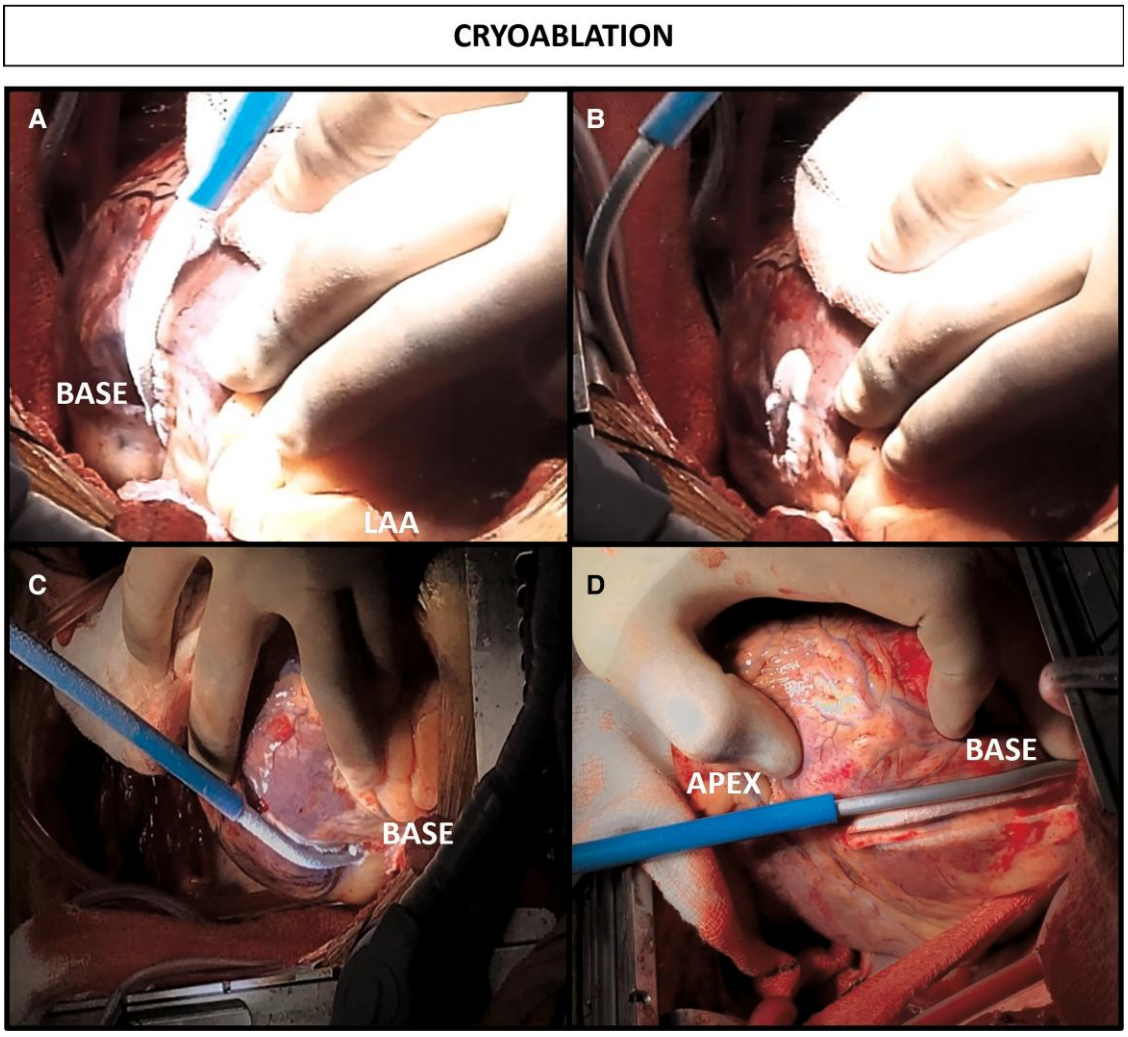

Figure 3 Intraoperative: Cryoablation. Application of the ATRICURE cryoICE probe on the epicardial surface overlying the scar region rich with late potentials. Cryo application (*A* and *C*), epicardial ice crystals immediately post-application (*B*), and lesion depth (*D*) can be appreciated. LAA, left atrial appendage.

grove along the lateral wall of the LV. This was corroborated with EP contact catheter data recorded on the LABSYSTEM™ PRO BARD EP mapping system with the use of the Biosense Webster DECANAV® catheter, which provided better contact compared with the PENTARAY™ catheter. Late potentials (LPs) were identified throughout the macroscopic scar (*Figure 2*). Ventricular tachycardia stimulation induced unmappable rapid ventricular flutter (240 ms) requiring cardioversion.

Using the ATRICURE cryoICE probe, sequential 3 min −60°C linear applications were delivered to the entire arrhythmogenic scar, avoiding the coronary arteries, until loss of LPs and pace capture were achieved and persisted (*Figure 3*).

The total procedure duration was 380 min from GA induction to sternotomy closure. No intra- or post-operative complications occurred. The patient made an uneventful recovery with discharge 6 days later.

In the absence of arrhythmias, Amiodarone was stopped 3 months post-operatively, and at the 30-month follow-up, the patient has remained free of VT.

Discussion

This case highlights several clinical challenges. The diagnosis of myocarditis is often difficult, with no pathognomonic clinical presentation of the disease. Endomyocardial biopsy remains the gold standard for confirmation, which our patient did not have. Diagnostic delays posed by the device artefact limiting LGE-CMR further impacted treatment delivery in our case. Finally, patients with NICM and VT have less favourable CA outcomes compared with their ischaemic counterparts, as highlighted in multiple studies, with reported recurrence rates of up to 22–75%.[10] Whilst 'closed-chest' epicardial access for hybrid VT ablation is attractive, our intraoperative findings demonstrate its potential challenges and limitations with the possibility of higher associated procedural risks and recurrence rates in select patients. Therefore, despite the absence of pericardial adhesions in our case, the subsequent 'open-chest' procedure allowed optimal access to the complex substrate, leading to a safer procedure with a high success rate.

Pre-procedural localization of scar on LGE-CMR is associated with improved long-term ablation outcomes in patients with NICM.[11] However,

the presence of implanted devices can restrict the acquisition of diagnostic images. In our case, the initial LGE-CMR scans in 2016 and 2017 were uninterpretable due to ICD artefact; however, in 2020, utilizing a different scanner and imaging techniques, we successfully localized the scar entirely to the LV epicardium and made a probable diagnosis (*Figure 1*). Our case demonstrates the challenges faced in acquiring the requisite information, which ultimately proved crucial for diagnosis and helped guide focused discussions on the optimal hybrid surgical strategy to allow unrestricted access to the arrhythmogenic region.

Large published series of hybrid closed-chest epicardial VT ablations have identified that whilst the subxiphoid surgical approach provides better access to the inferior, posterior, basal, and lateral epicardium, it is inadequate for accessing the anterolateral and apical regions, which are best approached via a limited anterior thoracotomy.[12,13] Despite its desirability, the minimally invasive surgical option would have posed restrictions in our case, based on the broad scar distribution.

In a published series of surgical open-chest epicardial VT ablation, no detailed intraoperative mapping was performed with the procedure guided entirely by pre-acquired EP data.[14] Despite the advantages of surgical ablation creating deep, large lesions, VT was not completely abolished. This case series supported the potential effectiveness of cardiac surgical ablation guided by prior endocardial/epicardial mapping in the absence of intraoperative mapping; however, VT was not completely abolished in two patients and six remained on antiarrhythmic drug therapy.

The 3D mapping systems are challenged during hybrid procedures with reports of geometric distortions, which we also experienced.[15] However, we had planned to overcome these by direct visualization of the entire scar and concomitant EP confirmation using a multielectrode catheter to delineate the boundaries of the macroscopically 'abnormal' tissue to guide ablation delivery (*Figure 2*). Our case illustrates the significant advantages of intraoperative EP mapping to guide targeted ablation of the region of interest. Utilizing a 'hybrid team' in a hybrid lab brings together the strengths of both EP and cardiac surgical expertise and advanced tools, which stand to deliver the greatest success in such cases.

Current radiofrequency systems are not engineered for open-chest epicardial use. Cryoablation is an alternative energy source for epicardial ablation, with animal studies demonstrating similar or larger lesion size and depth as those on the endocardium.[16,17] Despite extensive use in atrial arrhythmias, cryoablation for epicardial VT lacks sufficient data.[18] The ATRICURE cryoICE system is a nitrous oxide–based system with a malleable probe of up to 10 cm, causing surface temperatures of −65°C, providing a lesion depth, and cell death, of ∼6 mm. During our case, lesion delivery was evident through visible ice crystals and palpable induration of the myocardial surface (*Figure 3*), leading to the ablation of large arrhythmogenic regions expediently and in close proximity to, but avoiding, coronary anatomy.

According to the ESC guidelines, a multidisciplinary team with open communication, appropriate leadership, skills, and resources is critical for complex decision-making and the success of such complex procedures.[6] In our case, pre-procedural planning and multidisciplinary input were crucial for success, avoiding acute or medium-term complications with no post-operative VT recurrence at 30 months. The procedure time and post-operative admission duration are comparable to larger published studies utilizing full or limited surgical epicardial access.[13–15]

Whilst this technique is not novel, to our knowledge, this is the first case report of open-chest epicardial mapping with cryoablation performed in the cardiac EP laboratory for the sole treatment of VT, adding to the growing evidence supporting this in select patients.

Conclusion

Over the last decade, increasingly minimal surgical access for epicardial ablation has allowed hybrid cases to be performed in the EP laboratory. We would add that in select patients, and with the right expertise, epicardial access via median sternotomy is a viable option for successful precision ablation of challenging the epicardial substrate in the EP lab.

Patient perspective

I started with my heart issues at the age of 31. I was fit and healthy, and my episode came out of the blue. Unfortunately, my condition was not diagnosed until I had an episode whilst playing pool. I then became a patient here. I was implanted with an ICD in 2015, not really knowing what to expect, apart from it helping save my life.

My real issues started after my first shock from the device. I don't think anyone can prepare you for it. My family and sport are my life. The anxiety my first shock gave me was awful. After this, I had a VT storm with 13 shocks; this was horrendous, and I can't explain how terrible I felt. This continued over the next few years. I had two ablations and different medications, but it just came back. This resulted in me suffering from anxiety and stopped me from doing things I loved. It ran my life, and all I can say is that when a further ablation was offered to me, I saw the chance to get some quality of life back and do some things I enjoy, rather than suffering each day.

After this ablation failed, again it crushed my hopes, but the doctor suggested doing it as an open-heart procedure, which gave me the new hope I was looking for. Yes, hearing the words that it would be an open heart caused me some worries, but I was prepared to have anything done to help me, and this seemed a very good solution.

I'm no medical expert, but having my heart in front of my doctor and the surgeon, rather than looking at it on a screen, seemed a better way to see more of the issue.

This final ablation has literally changed my life. I have got my life back and can now enjoy time with my family and friends. I can't start to explain the appreciation to my doctor, her team, and the NHS for providing me with this opportunity. As a patient, the biggest thing is the hope that things can be done and you can have a good quality of life back.

Lead author biography

Dr Jamil-Copley is a consultant electrophysiologist and lead for the VT ablation service at Nottingham University Hospitals NHS Trust. She underwent EP training in Nottingham and Imperial College, London. She holds a PhD from Imperial College, London. She is an Honorary Associate Professor at the University of Nottingham. Dr Jamil-Copley's research is supported by an MRC grant and involves the use of machine learning to enhance VT management including the development of a ventricular scar model to guide precision VT ablation. This case report is not linked to her research.

Supplementary material

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

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 submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

Funding: None declared.

Data availability

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

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