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CASE REPORT
Electrophysiology

Successful ablation of Purkinje ectopy–triggered ventricular fibrillation storm in a patient with orthotopic heart transplantation and severe SARS-CoV-2 pneumonia: a case report

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Background Catheter ablation is one of the most effective treatment options for patients with drug-refractory, scar-related monomorphic ventricular tachycardia (VT). In selected cases, catheter ablation also plays an important role in treatment of polymorphic VT (PMVT) and/or ventricular fibrillation (VF). Rarely, premature ventricular contractions (PVCs) originating from the Purkinje network can induce PMVT/VF. Ablation and elimination of these PVCs can prevent VF recurrences.

Case summary A 41-year-old patient with a history of orthotopic heart transplantation (HTX) 8 years before admission and newly diagnosed SARS-CoV-2 pneumonia was referred to our centre after experiencing several episodes of drug-refractory VF. An electrophysiological study showed ectopy-triggered VF originating from the anterior and posterior fascicles of the left bundle branch (LBB). Ablation of these PVCs from the LBB led to complete elimination of VF. A subcutaneous implantable cardioverter defibrillator was implanted as secondary prophylaxis. During the observation period of 6 months, no VF recurrence was observed.

Conclusion Identifying and eliminating the trigger (PVCs) can be life-saving and prevent VF in the specific cohort of HTX patients. High-density mapping using multipolar catheters with microelectrodes contributes significantly to our understanding of tachycardia mechanisms.

Keywords Case report • Ventricular fibrillation • Purkinje-triggered VF • Heart transplantation • SARS-CoV-2

ESC curriculum 5.6 Ventricular arrhythmia • 7.5 Cardiac surgery

Learning points

- In the particular group of heart transplantation patients, the identification and elimination of the trigger premature ventricular contraction could save lives and avoid ventricular fibrillation.
- The utilization of multipolar catheters featuring microelectrodes in high-density mapping significantly enhances our comprehension of tachycardia mechanisms.

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Introduction

Catheter ablation has emerged as one of the most important/effective treatment options in patients with drug-refractory, scar-related monomorphic ventricular tachycardia (VT).[1,2] In selected cases, catheter ablation plays an important role in the treatment of polymorphic VT (PMVT) and/or ventricular fibrillation (VF), too. Premature ventricular contractions (PVCs) originating from the Purkinje network can induce PMVT/VF. Ablation and elimination of these PVCs can prevent VF recurrence.[3–6]

Summary figure

9 years before	Orthotopic heart transplantation due to non-ischaemic dilated cardiomyopathy.
21 days before admission	Positive SARS-CoV-2 pneumonia.
10 days before admission	Ventricular fibrillation (VF) storm requiring a prolonged cardiopulmonary resuscitation (CPR) for 50 min and more than 20 external defibrillations.
Prior to admission	Echocardiography, coronary angiography, magnetic resonance imaging, and myocardial biopsy without pathological result.
Day of admission	Ventricular fibrillation storm, requiring another CPR and 11 defibrillations.
During the procedure	High-density mapping using multipolar catheters with microelectrodes led to successful ablation of Purkinje ectopy–triggered VF.
Day 7 post-procedure	Secondary prophylaxis implantable cardioverter defibrillator implantation.
Day 11 post-procedure	Hospital discharge without any VF relapse.
12 months follow-up	Until today, no arrhythmia recurrence has been detected.

Case presentation

A 41-year-old man with an orthotopic heart transplantation (HTX) in 2014 due to non-ischaemic dilated cardiomyopathy, recently diagnosed with and treated for SARS-CoV-2 pneumonia, was referred to our centre (see Supplementary material online, *Figure S1*). The patient had experienced VF storm requiring prolonged cardiopulmonary resuscitation (CPR) for 50 min and >20 external defibrillations within 24 h. Despite administration of antiarrhythmic drugs (amiodarone), sedation with propofol and mechanical respiration finally led to stabilization of the rhythm. Upon admission to our centre, the patient experienced another VF storm episode, requiring CPR and another 11 defibrillations. Despite our best efforts, the VF episodes did not respond to the measures taken, which involved substitution of electrolytes (potassium, magnesium), administration of i.v. antiarrhythmic drugs (amiodarone, beta-blocker, lidocaine), sedation (midazolam), overdrive pacing (increasing pacing rate to 90/min), and repetitive electrical defibrillations. After multiple attempts aiming for haemodynamic stabilization failed, the patient was stabilized through sedation and mechanical respiration.

Prior admission to our heart transplantation centre, transthoracic echocardiography (TTE) revealed a left ventricular ejection fraction of 55%. A coronary angiogram showed no significant coronary artery disease, and a myocardial biopsy as well as cardiovascular magnetic resonance imaging (cMRI) had ruled out hints for transplant rejection or acute myocarditis. The patient had no history of cardiovascular risk factors and a body mass index of 24.9 kg/m^2. The C-reactive protein (CRP) level at admission was 1.8 mg/dL, and both the troponin I and creatine kinase levels were normal.

The 12-lead electrocardiogram (ECG) showed two different morphologies of PVCs that were consistent with myocardial activation arising from the anterior and posterior fascicles of the left bundle branch (LBB) (as shown in *Figure 1*). The PVCs had a relatively narrow QRS duration ranging from 120 to 125 ms. They were characterized by a rapid initial deflection and a short coupling interval of 320–360 ms, leading to the occurrence of R-on-T phenomenon. Following administration of antiarrhythmic drugs, the coupling interval immediately increased. Further analysis of ECGs obtained during VF storm revealed multiple episodes of VF as well as short bursts of PMVT, triggered by PVCs with an R-on-T phenomenon as demonstrated in *Figure 2*.

Due to the patient's condition, an urgent endocardial ablation procedure was performed to identify and eliminate the PVCs initiating VF. The patient was taken to the electrophysiology laboratory (EP lab), and the procedure was performed under general anaesthesia. A diagnostic catheter was inserted into the right ventricular apex and a decapolar catheter in coronary sinus (Inquiry, Abbott, Chicago, IL, USA). Afterwards, high-density mapping was carried out using a multipolar catheter (Advisor HD Grid, Abbott, Chicago, IL, USA) and a 3D navigation system (EnSite Precision, Abbott, Chicago, IL). An endocardial bipolar voltage map was performed which showed normal voltage in both ventricles as demonstrated in *Figure 3*.

The earliest myocardial activation during the PVCs was observed along the anterior and posterior fascicles of the LBB. This activation was consistently preceded by mi-diastolic Purkinje potentials P1 and presystolic Purkinje potentials P2. In some instances, the Purkinje potential was blocked and no VF episode was triggered, whereas in many other cases, PVCs initiated VF, as demonstrated in *Figures 4* and *5* and Supplementary material online, *Video S1* and *Figure S2*. This observation demonstrated that the trigger PVCs have had a substantial impact on VF initiation and maintenance in this patient.

Activation mapping further identified the earliest presystolic Purkinje-triggered PVC at the level of the anterior and posterior fascicles of the LBB (−101 ms), as shown in *Figure 5*.

During ablation, the PVCs were successfully identified and eliminated using an irrigated tip ablation catheter (TactiCath, Abbott, Chicago, IL, USA). Ablation (energy, 35 watts; flow, 17 mL/min.) also included substrate modification by means of ablation in the area of PVC origin, which was revealed to be located in the area of the anterior and posterior fascicles of the LBB. During ablation (radiofrequency energy was applied for 14 min), automaticity and local responses with bursts of PVCs and VF were observed, which were completely suppressed after ablation.

After the procedure, the patient was monitored continuously and no PVCs were observed. However, following ablation, LBB block with a QRS duration of 135 ms was noted (Supplementary material online, *Figure S3*). There were no immediate or late complications related to the ablation procedure. Antiarrhythmic agents were stopped after the ablation procedure, and a subcutaneous implantable cardioverter defibrillator (ICD) was implanted subsequently. Of note, QRS duration normalized the day after ablation as shown in Supplementary material online, *Figure S4*.

Within the next days, the patient's condition improved significantly, so that he could be discharged from the hospital. During 12 months of follow-up, the patient did not experience any VF episodes.

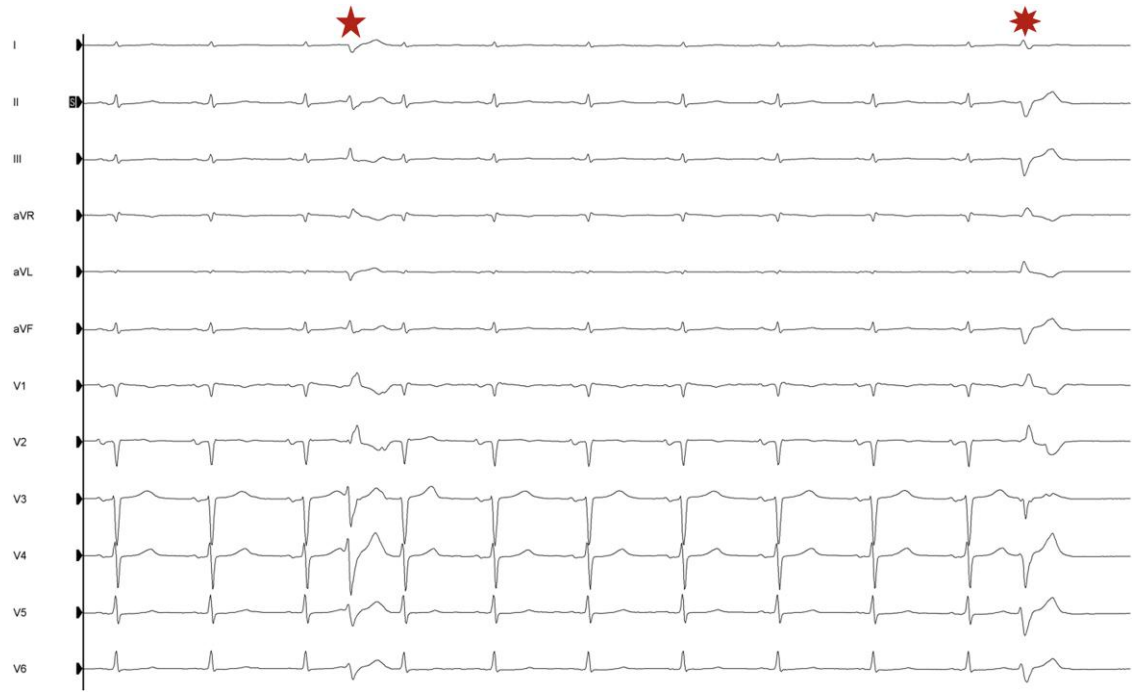

Figure 1 Electrocardiogram before ablation with two premature ventricular contractions' morphology, from left anterior fascicle (★) and from left posterior fascicle (✸) (25 mm/s and 10 mm/mV).

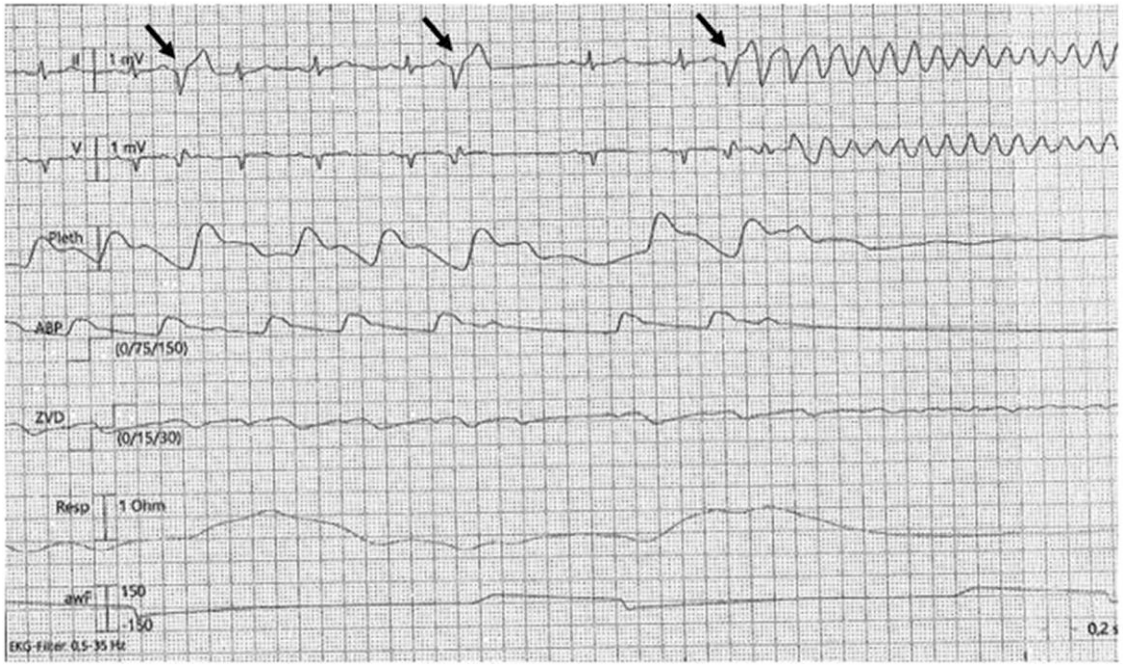

Figure 2 Electrocardiogram from telemetry demonstrating ventricular fibrillation episodes induced and triggered by premature ventricular contraction (arrow) with R-on-T phenomenon.

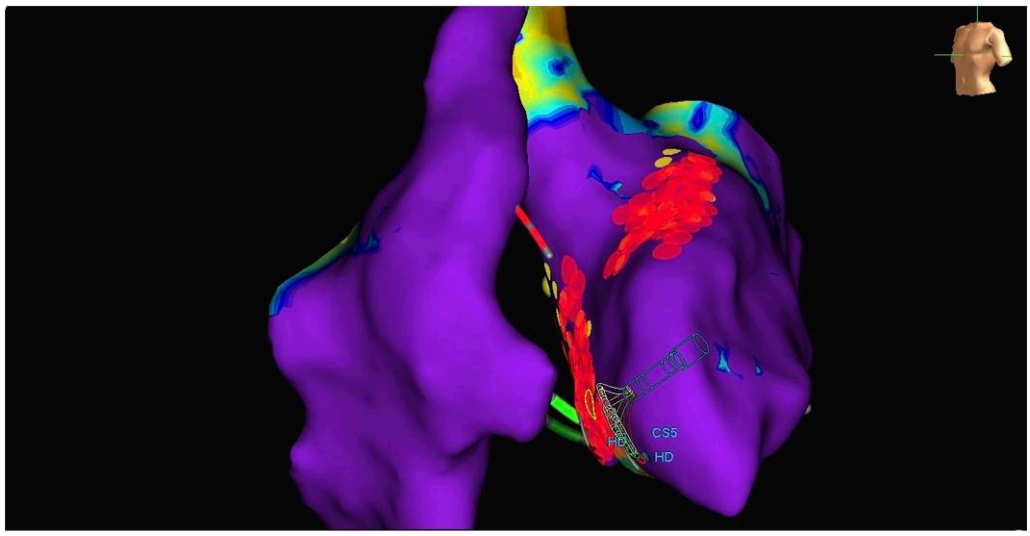

Figure 3 Voltage map of both left and right ventricles shows no substrate (10 075 total points and 1798 used points). The voltage cut-off: 0.5–1.5 mv.

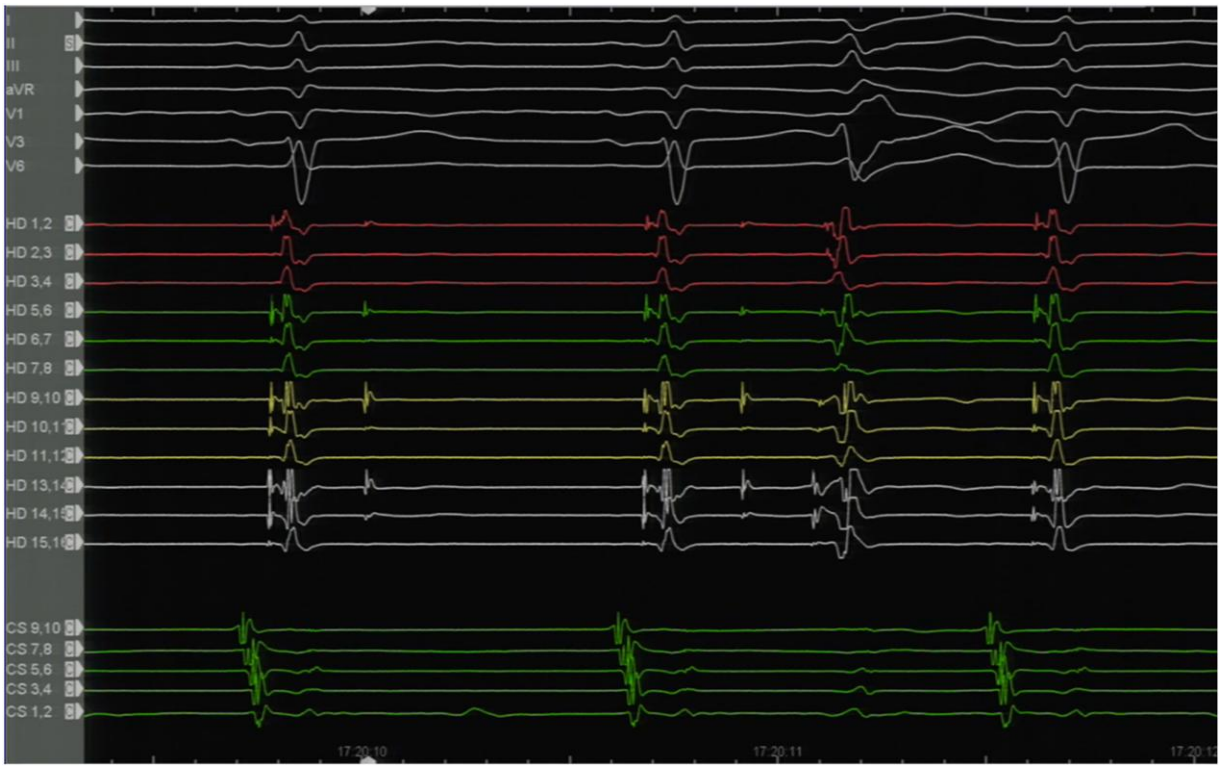

Figure 4 Every QRS is consistently preceded by Purkinje potentials. Sometimes, the Purkinje potential had been blocked without following a premature ventricular contraction and was followed intermittently by a premature ventricular contraction.

Discussion

In patients with VF storm, identifying Purkinje ectopy as the cause of incessant VF presents an urgent electrophysiological scenario that demands careful and timely evaluation.

Heart transplantation patients who experience PVC-triggered VF require prompt identification and treatment of any reversible proarrhythmic causes, such as rejection, electrolyte imbalances, myocardial ischaemia, QT-prolonging drugs, and inflammation. In the recent case, amiodarone and lidocaine were ineffective in terms of VF suppression

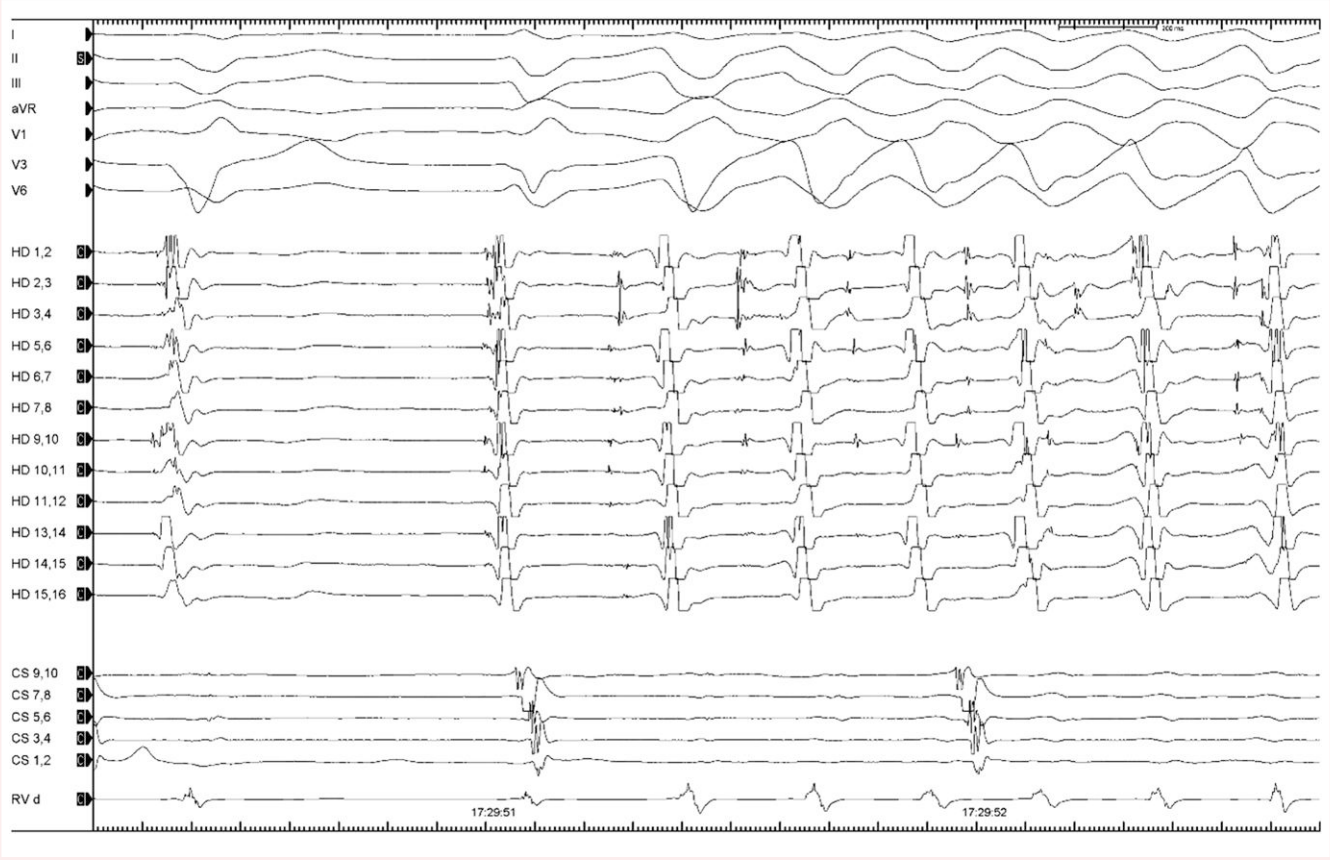

Figure 5 Presystolic activation from the Purkinje network preceded the initiation of ventricular fibrillation.

(quinidine was not administered because of unavailability). The effectiveness of antiarrhythmic drugs in these patients is still unclear and requires further research. However, for patients who have experienced sudden cardiac death caused by VT/VF, haemodynamically unstable VT, or sustained VT, a secondary prophylactic ICD implantation is recommended[1,2] when there is no reversible reason. In some patients, catheter ablation of Purkinje PVCs triggering PMVT/VF has been reported as a successful intervention against the initiation of PMVT/VF.[3–7]

High-density mapping using multipolar catheters is more effective than mapping with an ablation catheter alone in identifying Purkinje potentials and their characteristics. The presence of a very early Purkinje potential during PVCs/PMVT and VF may indicate that automaticity or triggered activity from the Purkinje network is the possible mechanism for VF.[8] In this case, successful elimination of VF required careful identification of the very early Purkinje potentials using high-density mapping and an ablation catheter. If challenging cases arise where there is an absence of trigger PVCs and low-voltage areas representing scar or fibrosis, Purkinje de-networking can be considered as an alternative.[9,10]

The decision to perform catheter ablation in the HTx cohort should be based on patients' individual characteristics. Close monitoring and multidisciplinary care are necessary to optimize outcomes and reduce the risk of complications. The role of cardiac sympathetic denervation was proved in reducing refractory sustained VTs and ICD shocks; therefore, HTx patients with denervated hearts are thought to be less susceptible to systemic triggering of short-coupled Purkinje ectopy and idiopathic VF.[11] SARS-CoV-2 infection and the related inflammation may have triggered the Purkinje ectopy that led to incessant VF and sudden cardiac death episodes. Further studies are needed to confirm a possible correlation. We report the first documented case of incessant idiopathic VF in an HTX patient triggered by a systemic viral infection.

Conclusion

The use of multipolar mapping catheters with microelectrodes facilitates our understanding of the pathophysiological mechanisms underlying tachycardia. This technology allows us to precisely locate and identify the mechanism of Purkinje ectopy, which can guide the ablation process and increase the probability for a successful elimination of VF. In cases where medical treatment has been ineffective, catheter ablation can be a life-saving intervention to prevent sudden cardiac death in these high-risk patients.

Lead author biography

Mustapha El Hamriti, MD, is a specialist in electrophysiology. In 2010, he graduated from the Medical University in Monastir, Tunisia. Since September 2019, he is a senior doctor at the Clinic for Electrophysiology at the Herz- und Diabeteszentrum NRW, University Hospital of the Ruhr-Universität Bochum, Bad Oeynhausen, Germany. His clinical research focus in arrhythmia management is arrhythmia ablation.

Supplementary material

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

Consent: The patient signed written informed consent for all treatment procedures. The journal consent form has been signed by the patient. Patient information in this case report is provided anonymously in accordance with the Committee on Publication Ethics (COPE) guidelines.

Conflict of interest: None declared.

Funding: None declared.

Data availability

Data will be made available from the authors on reasonable request

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