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CASE REPORT
Arrhythmias/electrophysiology

Re-entrant ventricular tachycardia as a complication of ablation of idiopathic ventricular premature beats from the right outflow tract: a case report

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Background We report an unusual case of non-sustained ventricular tachycardia (NSVT) from the epicardial part of the right ventricular outflow tract (RVOT).

Case summary A 37-year-old woman who underwent in 2006 an ablation for idiopathic ventricular premature beats (VPBs) from the RVOT presented with pre-syncopal NSVT in 2016. A cardiac workup showed no coronary disease, normal biventricular function, and no enhancement on cardiac magnetic resonance imaging. A metabolic positron emission tomography scan excluded inflammation. Biopsies revealed normal desmosomal proteins. An endocardial mapping revealed an area of low voltage potential (<0.5 mV) at the antero-septal aspect of the RVOT corresponding to the initial site of ablation from 2006. Activation mapping revealed poor prematurity and pace-mapping showed unsatisfactory morphologies in the RVOT, the left ventricle outflow tract and the right coronary cusp. An epicardial map revealed a low voltage area at the antero-septal aspect of the RVOT with fragmented potentials opposite to the endocardial scar. Pace-mapping demonstrated perfect match. An NSVT was induced and local electrocardiogram showed mid-diastolic potentials. Ablation was applied epicardially and endocardially without any complication. The patient was arrhythmia free at 4-year follow-up.

Discussion Cardiac workup allowed to exclude specific conditions such as arrhythmogenic cardiomyopathy, tetralogy of Fallot, sarcoidosis, or myocarditis as a cause for NSVT from the RVOT. The epi and endocardial map showed residual scar subsequent to the first ablation which served as substrate for the re-entrant NSVT. This is the first case which describes NSVT from the epicardial RVOT as a complication from a previous endocardial ablation for idiopathic VPB.

Keywords Ablation • Outflow tract • Idiopathic ventricular premature beats • Ventricular tachycardia • Epicardial ablation • Case report

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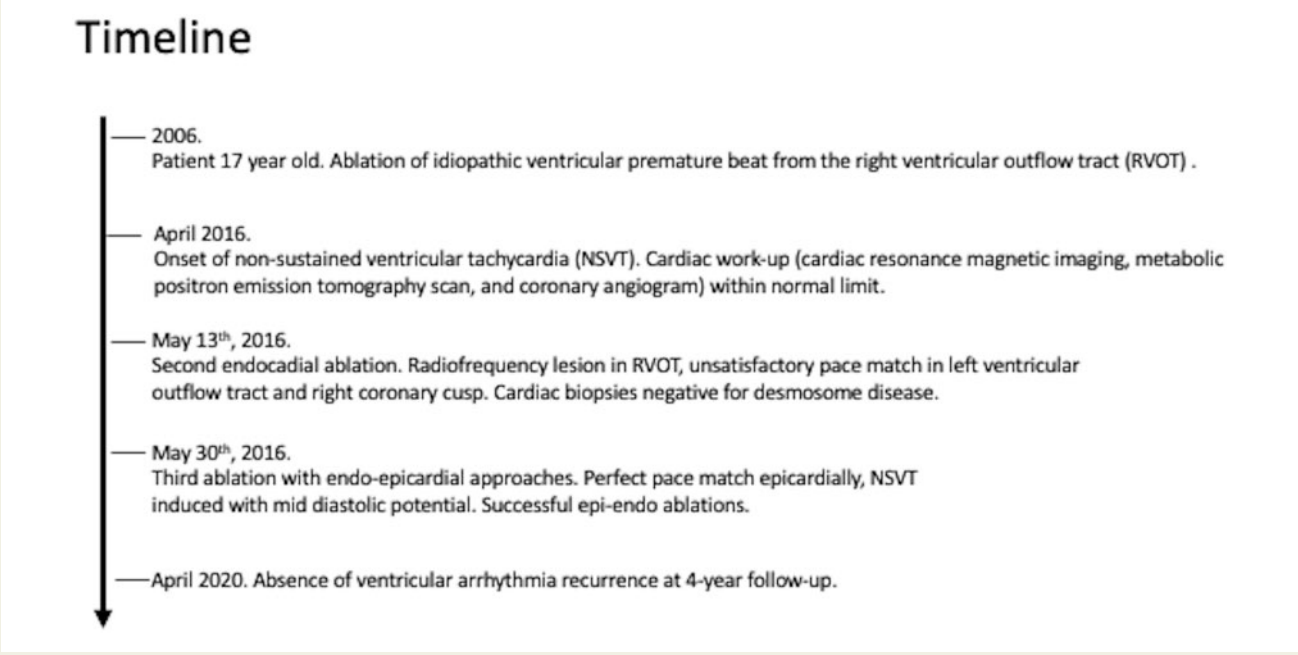
Learning points

- Ventricular tachycardia arising from the right ventricular outflow tract should raise suspicion of specific conditions. Extensive cardiac workup may be necessary to determine the cause of the arrhythmia.
- Scar from previous ablation can serve as substrate for new onset of re-entrant ventricular tachycardia.

Introduction

Idiopathic outflow tract ventricular premature beat (VPB) or tachycardia (VT), typically encountered in structurally normal heart, are caused by cyclic adenosine monophosphate (cAMP) mediated delayed after depolarizations.[1] They are caused by a focal mechanism, i.e. triggered activity or abnormal automaticity. A comprehensive cardiac workup may be necessary to confirm the absence of underlying structural heart disease. We report an unusual case of a recurrent VT arising from the right ventricular outflow tract (RVOT) 10 years after a first ablation.

Timeline



Case presentation

In 2016, a 37-year-old woman with a past medical history of rheumatoid arthritis treated with tumour necrosis factor alpha inhibitor complained of rapid palpitations of short duration and near syncope. In 2006, she was diagnosed with symptomatic idiopathic VPB (burden: 33%/24 h). The VPBs were isolated and monomorphic without any repetitive forms. *Figure 1A* shows a 12-lead electrocardiogram (ECG) with a representative VPB characterized by an inferior axis, a left bundle branch block (LBBB) morphology, and R/s transition in V4 suggestive of a triggered activity from the RVOT. The patient was treated with a beta-blocker (name and dosage unknown) as well as magnesium without any improvement. Cardiovascular examination was within normal limit. A successful ablation was performed on the septal aspect of the RVOT without a 3D navigation system. Thirty-four radiofrequency (RF) lesions were delivered at a power between 25 and 30 W with an irrigated catheter (non-nav D-Curve Thermocool catheter, Biosense Webster). No pericarditis was mentioned following the intervention. The patient did not have any complaint until 2008 when she noticed recurrence of isolated palpitations. The palpitations and near syncope in 2016 correlated with frequent non-sustained VT (NSVT) from the RVOT as reconstructed from the three-lead Holter recording (MicroPort, Synescope, MultiChannel-MultiDay Version 3.10) (*Figure 1B*). The early transition in lead V2 is probably due to some inaccuracy of the reconstructed ECG, which is based on the Dower's transform. This Matrix uses the X, Y, Z signals obtained from the Frank lead system to simulate a standard 12-lead ECG with some inherent inaccuracy.

The VPB burden was 3% on the Holter recording. The patient remained symptomatic despite the prescription of metoprolol 25 mg daily. On physical examination, the patient was well-groomed. Cardiopulmonary auscultation was within normal limit. There was no sign of heart failure. A transthoracic echocardiography was within normal limit. A coronary angiogram did not show significant lesions (*Figure 2A*). Suspecting an arrhythmogenic cardiomyopathy (ACM) or

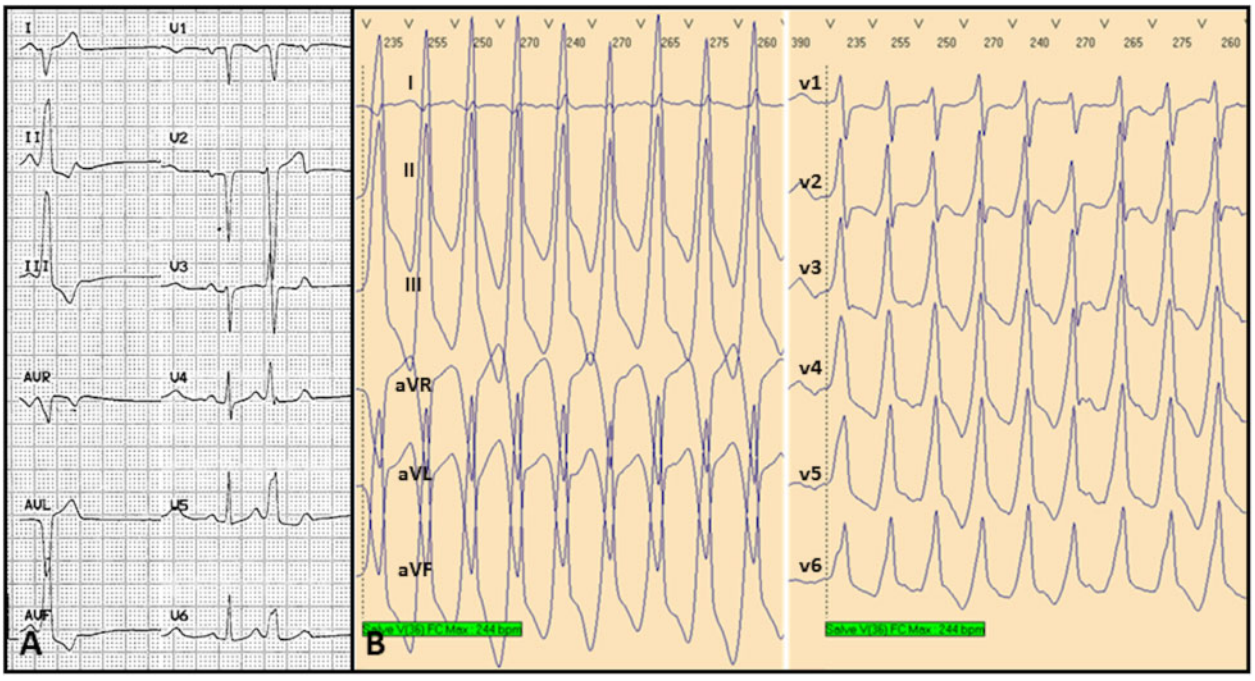


Figure 1 (*A*) Ventricular premature beat seen in 2006. (*B*) Twelve-lead electrocardiogram reconstruction from Holter's monitoring system of the non-sustained ventricular tachycardia seen prior to the 2nd ablation.

an inflammatory cardiomyopathy, the patient underwent a cardiac magnetic resonance imaging (cMRI) (*Figure 2B*) and a metabolic ^{18}FDG-PET scan (*Figure 2C*) which were normal. A 2nd ablation was then undertaken including endomyocardial biopsies, which are part of a standardized workup at our centre to discriminate between myocarditis and ACM. The tissue was harvested from the interventricular septum without any electroanatomical mapping guidance, as previously described by Rosset *et al.*[2] Results showed normal immunofluorescence signals for Cadherin, Connexin 43, Desmoplakin, Plakoglobin, and Plakophilin proteins that reasonably ruled out ACM (*Figure 2D*). Under local anaesthesia, an endocardial electroanatomical mapping (Carto 3 System, Biosense Webster, Diamond Bar, CA, USA) revealed an area of low voltage (<0.5 mV) at the septal aspect of the RVOT corresponding to the site of ablation performed in 2006. Despite isoproterenol infusion, only a single run of NSVT was triggered which was enough to confirm by pace-mapping that the scar area of the septal RVOT was involved in the arrhythmic event. *Figure 3* shows the NSVT run during the electrophysiology study. The morphology showed an inferior axis, an LBBB morphology, and an R/s transition in V3 that was perfectly reproduced by pace-mapping at the septal RVOT within the scar region. Because of the absence of early potentials in the RVOT, the left ventricle outflow tract and the right coronary cusp were mapped and, as shown by the poor pacemap matching in *Figure 3*, did not participate into the arrhythmia. Ablation was delivered at the focal scar region using a temperature control system and an irrigated catheter (Thermocool, SmartTouch, target temperature 43°C, maximal power 30 W). Post-procedure, rare VPB were seen at baseline and under isoproterenol infusion. One week after discharge, because of recurrence of symptomatic NSVT, an epicardial ablation was planned. A dry pericardial puncture under fluoroscopy guidance was performed with a dedicated needle (Becton Dickinson AG, reference 408360). A pigtail was inserted followed by a short steerable guiding sheath (EPI-Agilis, Abbott). After the puncture, 20 mL of serous liquid were drained and sent to the cytology lab (which result showed only few atypical cells). *Figure 4* shows the epicardial voltage map (Carto 3D, Biosense Webster) reconstructed with a decapolar catheter (DecaNav, 2-8-2, Biosense Webster, Diamond Bar, CA, USA). The map revealed a localized region of low voltage at the antero-septal aspect of the epicardial RVOT including fragmented potentials and late abnormal ventricular activities (LAVA) facing the scar at the antero-basal aspect of the endocardial RVOT shown in *Figure 3*. Importantly, pace-mapping at this location showed a perfect match (*Figure 4*). A programmed ventricular stimulation with two extrastimuli initiated a non-sustained run matching the clinical VT. The VT started with a narrow QRS followed by a subsequent widening of the QRS (pseudo-delta wave) suggestive of an endocardial activation followed by an epicardial exit (*Figure 5A*). *Figure 5B* shows mid-diastolic potentials (MDP) within this region. The MDP was very suggestive of an area of slow conduction. Neither entrainment manoeuvers nor sequential mapping could be performed as the VT remained non-sustained despite increasing dose of i.v. Isoproterenol. However, pacing from the MDP site reproduced the clinical VT morphology (*Figure 5C*). The MDP to QRS interval (*Figure 5B*) and the stimulation to QRS interval (*Figure 5D*) were of similar values (46 ms). This implies that an area of slow conduction

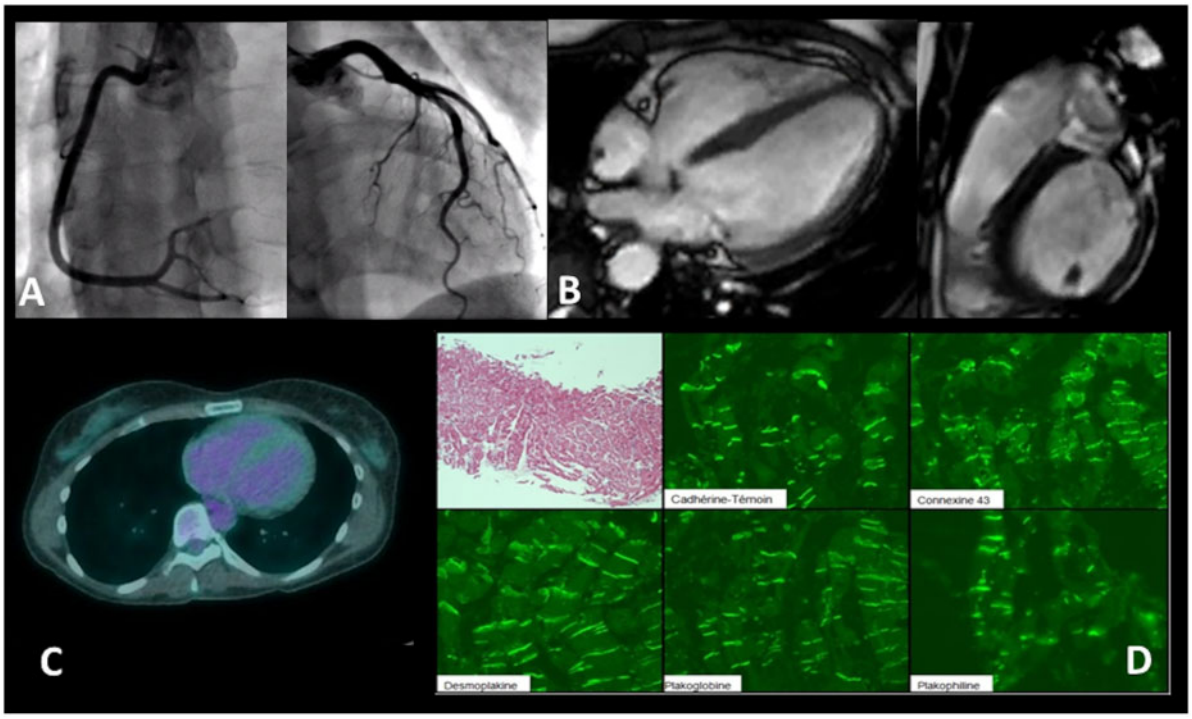


Figure 2 Cardiac workup demonstrated the absence of significant disease on coronary angiogram (*A*), absence of late gadolinium enhancement and normal biventricular function on cardiac magnetic resonance imaging (*B*), absence of tissue inflammation on metabolic ^{18}FDG PET scan (*C*), and normal immunofluorescence signal of desmosomal proteins on cardiac biopsies (*D*).

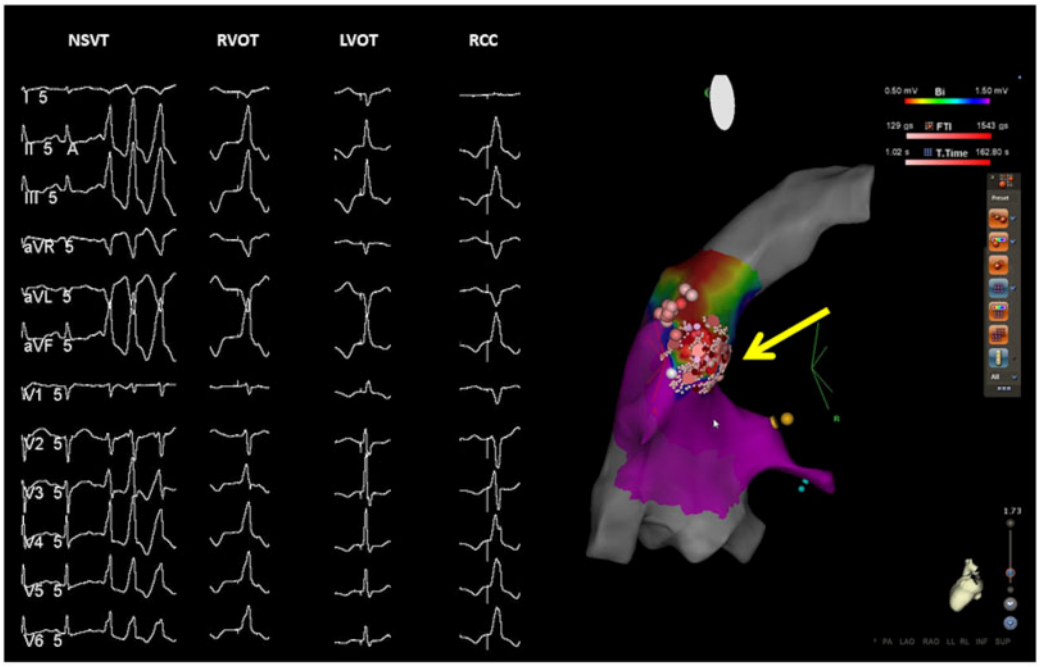


Figure 3 Three-dimensional electroanatomic mapping of the endocardial RVOT revealed a large area of low voltage (yellow arrow). Pace map from different sites showed the best match at the septal RVOT. LVOT, left ventricular outflow tract; NSVT, non-sustained ventricular tachycardia; RCC, right coronary cusp; RVOT, right ventricular outflow tract.

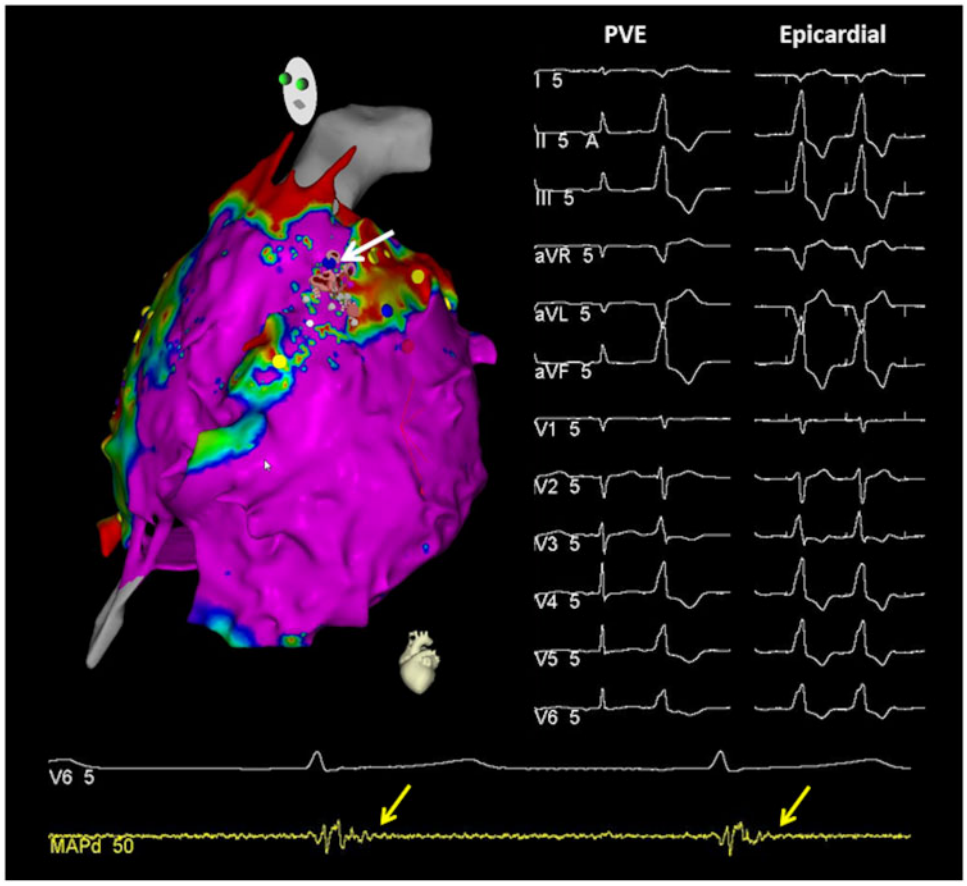


Figure 4 Epicardial mapping revealed an area of low voltage at the antero-basal RVOT epicardium with late abnormal ventricular activities (yellow arrows). Pace map (white arrow) showed a perfect match. VPB, ventricular premature beat.

or an isthmus was involved in the maintenance of the VT, suggesting that the mechanism was likely a re-entrant VT rather than a focal triggered activity. Endocardial mapping of the septal RVOT facing this area showed LAVA of very low amplitude. Prior to RF ablation, a coronary angiogram was performed with merging of the images into the Carto (Carto Uniview) and confirmed that the area of interest was not in the vicinity of a coronary vessel. Radiofrequency ablation was applied with an irrigated catheter (Thermocool SmartTouch SF, Biosense Webster) epicardially and endocardially thereafter without any complication. Aggressive premature ventricular stimulation including burst pacing (up to five ventricular extrastimuli, shortest coupling intervals at 240 ms) at baseline and under isoproterenol infusion (12 mcg/min) and adrenaline bolus (10 mcg) showed the absence of susceptibility to VT and VPB. The procedure lasted 190 min (9.8 min fluoroscopy time) and included 18 RF lesions (irrigation 8 mL/min in the epicardium, power 25–30 W, total energy 12 884 J). The patient stayed 48 h in our cardiac care unit where a transthoracic echocardiogram excluded a pericardial effusion. She was discharged with a wearable defibrillator vest for 1 month. Clinical follow-up including a 48-h Holter recording did not show any ventricular arrhythmia recurrence at 4-year follow-up.

Discussion

RVOT VT and VPB are defined as 'idiopathic arrhythmias' as they usually occur in a structurally normal heart. They are caused by a focal mechanism, i.e. triggered activity or abnormal automaticity. They are thought to be mediated by cAMP, which induces triggered activity via intracellular calcium overload released from the sarcoplasmic reticulum resulting in delayed after depolarization. The increased intracellular calcium concentration causes the sodium-calcium exchanger to transport three sodium ions into the cell for every calcium extruded, resulting in a positive inward current. The latter promotes delayed after depolarization during phase 4 of the action potential, which, in turn, triggers a new action potential that can lead to tachycardia. Usually, adenosine can terminate the arrhythmia by inhibition of cAMP.[1] Radiofrequency ablation of RVOT VPB or VT foci, which are identified by activation or pace-mapping, usually shows good outcomes with a complete remission in up to 90% of the cases.[1] In case of re-entrant VT, a substrate including an area of slow conduction, such as a scar tissue, is required to maintain the tachycardia in contrast to idiopathic VT which mechanism is focal.[3] As such, the re-entrant VT would arise in a structurally abnormal heart because of the presence of scar tissue, hence the VT cannot be considered as

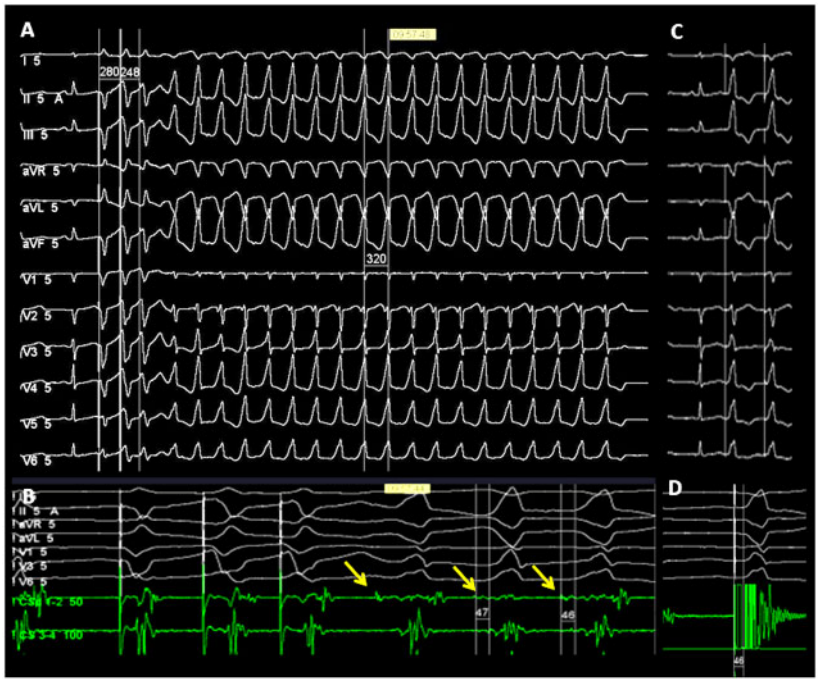


Figure 5 Induction of the clinical non-sustained ventricular tachycardia. The clinical ventricular tachycardia started with a narrow QRS (*) followed by a subsequent widening QRS (pseudo-delta wave) (**) suggestive of endocardial activation followed by an epicardial exit (*A*). The mapping catheter recorded mid-diastolic potentials (yellow arrows) (*B*). Pacing at the location of the mid-diastolic potentials with the decapolar catheter showed similar QRS morphology (*C*). The mid-diastolic potentials to QRS interval (*B*) and the stimulation to QRS interval (*D*) were of similar values (46 ms).

idiopathic. Re-entrant VT from the outflow tracts is often encountered in patients suffering either from ACM, Brugada syndrome, dilated cardiomyopathy, Chagas' disease or tetralogy of Fallot.[4–6] However, several cases have been reported where focal scars of the RVOT were associated with re-entrant VT.[7,8] In our case, the cardiac workup was able to exclude a myocarditis and diseases involving the desmosomes. However, this did not totally exclude an arrhythmogenic substrate which could be hosted before and until the 2nd attempt. In fact, the area of low voltage seen in the RVOT during the 2nd ablation (2016) could have been present in 2006 but not seen as the first ablation was performed without a 3D mapping system and without pre-procedural cMRI. The aetiology of the pericardial effusion remained unclear but might be either related to the 2nd ablation or to rheumatoid arthritis. The origin of the clinical non-sustained VT was likely the site of VPB ablation but could not be proven formally: low voltage on the septal RVOT remained the only clue suggesting that the ablation in 2006 was done at this location. Then, pace-mapping in this area showed close morphology but not identical to the clinical VPB from 2006. Finally, lesions formation from 2006 could not be identified using contrast cMRI 10 years later. This finding was not surprising. Ilg *et al.*[9] conducted a study based on 35 patients without any structural heart disease who underwent VPB ablation, 17 of them at the RVOT. In only 25 patients (71%), ablation lesions were identified by late gadolinium enhancement (LGE) on cMRI (at 22 ± 12 months of follow-up). Due to the thin RVOT myocardium, LGE may be difficult to detect in this area, which is also typically prone to artefact. Indeed, in our case, both the contrast cMRI and the FDG-PET scan appeared normal, while the 3D mapping system revealed low voltage in the antero-septal aspect of the RVOT.

Nevertheless, the voltage map of the septal RVOT region showed low voltage (<0.5 mV), consistent with residual scar likely subsequent to the first ablation. This arrhythmogenic tissue served as substrate to the re-entrant VT. This is the first case reported in the literature which describes a re-entrant VT from the epicardial RV outflow tract region as a complication from a previous ablation of an idiopathic RVOT VPB.

Lead author biography

Etienne Pruvot is the head of the arrhythmia unit in the Department of Cardiology of the Lausanne University Hospital in Switzerland. His domain of expertise is the ablation of ventricular tachycardia and the signal processing of atrial fibrillation.

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 image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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References

1. Lerman BB. Mechanism, diagnosis, and treatment of outflow tract tachycardia. *Nat Rev Cardiol* 2015;**12**:597–608.
2. Rosset S, Domingo AM, Asimaki A, Graf D, Metzger J, Schwitter J *et al.* Reduced desmoplakin immunofluorescence signal in arrhythmogenic cardiomyopathy with epicardial right ventricular outflow tract tachycardia. *Heart Rhythm Case Rep* 2019;**5**:57–62.
3. Stevenson WG, Sager PT, Natterson PD, Saxon LA, Middlekauff HR, Wiener I. Relation of pace mapping QRS configuration and conduction delay to ventricular tachycardia reentry circuits in human infarct scars. *J Am Coll Cardiol* 1995;**26**:481–488.
4. Zeppenfeld K, Schalij MJ, Bartelings MM, Tedrow UB, Koplan BA, Soejima K *et al.* Catheter ablation of ventricular tachycardia after repair of congenital heart disease: electroanatomic identification of the critical right ventricular isthmus. *Circulation* 2007;**116**:2241–2252.
5. Satomi K, Kurita T, Suyama K, Noda T, Okamura H, Otomo K *et al.* Catheter ablation of stable and unstable ventricular tachycardias in patients with arrhythmogenic right ventricular dysplasia. *J Cardiovasc Electrophysiol* 2006;**17**:469–476.
6. Nademanee K, Hocini M, Haïssaguerre M. Epicardial substrate ablation for Brugada syndrome. *Heart Rhythm* 2017;**14**:457–461.
7. Nakano E, Harada T, Aonuma K, Soejima K, Wakimoto H, Matsumoto N *et al.* Identification of unusual reentry circuit sites of nonischemic ventricular outflow tract tachycardia. *J Cardiovasc Electrophysiol* 2012;**23**:179–187.
8. Mulpuru SK, Konecny T, Madhavan M, Kapa S, Noseworthy PA, McLeod CJ *et al.* Atypical variants of right ventricular outflow arrhythmias. *J Cardiovasc Electrophysiol* 2014;**25**:1321–1327.
9. Ilg K, Baman TS, Gupta SK, Swanson S, Good E, Chugh A *et al.* Assessment of radiofrequency ablation lesions by CMR imaging after ablation of idiopathic ventricular arrhythmias. *JACC Cardiovasc Imaging* 2010;**3**:278–285.