Introduction

Ventricular tachyarrhythmias from the right ventricular outflow tract (RVOT) are well recognized. In most cases, such arrhythmias are idiopathic because of the presence of a structurally normal heart. However, this ‘normal heart’ can sometimes be due to the early stage of heart disease or minor abnormalities that cannot be detected by routine examination. We report one case with ventricular tachycardia (VT) originating from the diverticulum of the RVOT. The electrocardiographic and electrophysiological characteristics, mapping and ablation strategies, and detailed images are described in this report.

Case report

A 62-year-old man with a repetitive monomorphic VT with a typical RVOT origin morphology for 19 years was referred for catheter ablation. The echocardiogram did not suggest any evidence of structural heart disease and revealed a normal left ventricular ejection fraction. The oral administration of amiodarone was initially effective, but the patient was referred for an electrophysiological assessment and catheter ablation because of the side effects of amiodarone and reoccurrence of syncope. Ambulatory monitoring did not reveal any large burden of premature ventricular contractions. The 12-lead electrocar-

Figure 1 (A) Left panel: the 12-lead electrocardiogram of clinical ventricular tachycardia. Right panel: perfect pace mapping was obtained with an almost 11/12 match to clinical ventricular tachycardia morphology from the target site in the diverticulum. (B) The local fragmented, low-voltage electrograms at the earliest site which preceded the onset of QRS during ventricular tachycardia by 23 ms was recorded at the same site. ECG, electrocardiogram; VT, ventricular tachycardia.
diogram (ECG) during the VT demonstrated a left bundle branch block (LBBB) and inferior axis morphology with a late QRS transition in the precordial lead, V4. The QRS complexes had a tall positive component without a notch in the inferior leads and a Qr pattern in lead I (Figure 1A).

An electrophysiological study was performed under a fasting state and local anaesthesia. One quadripolar electrode catheter was inserted via the right femoral vein and positioned in the right ventricle apex. The clinical VT was induced easily by programmed electrical stimulation with S1/S2/S3 extrastimuli of 330/200/260 ms. As previously described, a multielectrode array (MEA, Ensite System, St Jude Medical, St Paul, MN, USA) was inserted via the left femoral vein and deployed in the RVOT guided by fluoroscopy. A 7-French quadripolar catheter with a 4 mm distal electrode, an embedded thermistor, an interelectrode spacing of 2–5–2 mm, and a deflectable tip (Cordis Webster Inc., Baldwin Park, CA, USA; EP Technologies Inc., San Jose, CA, USA) was also inserted for mapping and ablation. The three-dimensional (3-D) geometry of the RVOT was constructed by navigating the deflectable catheter within the RVOT using the non-contact mapping system (Ensite System). The intracardiac electrograms were filtered from 30 to 500 Hz and measured at a sweep speed of 100–200 mm/s. During the procedure, intravenous heparin was given at a 100 IU/kg bolus dose followed by boluses of 1000 IU every additional hour.

For the identification of the earliest activation (EA) site and breakout (BO) site of the VT, a broad colour setting of the colour high with the high-pass filter set at 2 Hz was used. At the BO site, the colour-coded activation map exhibited a white colour-zone that would shrink down to a red colour-zone. Then, the EA site was identified by stepping further backward in time in which the red colour-zone shrunk down to a blue colour (Figure 2A). Pace mapping was also performed to ensure at least an 11/12 match of the 12-lead ECG at the EA and BO sites. Furthermore, the unipolar virtual ECGs at that site were reconstructed. The presence of QS morphology at the EA site and the biggest slew rate at the BO site was also used as an additional criterion. On the 3-D map, the EA site was located at the posterior free wall and the BO site at the posterior septum according to the virtual activation mapping. However, pace mapping could not completely match the clinical VT. The EA pace mapping score was 10/12, and the BO pace mapping score 11/12. Furthermore, the slope of the unipolar virtual electrograms at the EA site was very subdued (Figure 2A). Radiofrequency (RF) catheter ablation was initially targeted at the EA site, and if it failed, it was then switched to the BO site. However, the

**Figure 2** (A) Left panel: initial noncontact activation mapping revealed that the earliest activation point of ventricular tachycardia was at the posterior free wall of right ventricular outflow tract; the virtual unipolar electrocardiogram showed a short and broad r wave at the initial part of the QRS complex. Right panel, the break out point of ventricular tachycardia was at the posterior septum with the rapid dv/dt. (B) Left panel: contact bipolar voltage mapping during sinus rhythm after the detailed local geometry being reconstructed displayed an low-voltage zone locating at the anterior lateral free wall adjacent to the septum. Right panel, local activation mapping showed that the origin of ventricular tachycardia was at the margin of the low-voltage zone. EA, earliest activation; BO, breakout; RVOT, right ventricular outflow tract; LVZ, low-voltage zone.
RF deliveries failed to terminate the VT at both sites. Then the activation was further verified by a contact activation map. During the contact activation mapping, the mapping catheter suddenly jumped out of the geometry, but was still within the contour of the cardiac structure. An RVOT angiography was consequently performed, revealing a diverticulum located between the anterior free wall and septum (Figure 3A). The detailed geometry of this abnormal structure was added to the previous 3-D map. The contact activation mapping showed that the origin of the VT was located on the anterior lateral free wall adjacent to the septum, which was at the margin of the low-voltage zone identified by the contact voltage mapping (Figure 2B). At that site, a good pace map was obtained with an almost 11/12 match to the clinical VT (Figure 1A). A fragmented ECG was observed at the earliest local activation site, which preceded the VT onset on the surface QRS by 23 ms (Figure 1B). Radiofrequency energy was delivered in a temperature-controlled mode with the irrigated-tip catheter at a power of 30 W, an irrigation flow rate of 17 mL/min, and a maximal temperature of 43°C. Radiofrequency resulted in the immediate termination of the VT. No VT could be induced after an intravenous administration of isoproterenol with an observational period of 30 min. A post-procedure magnetic resonance image (MRI) confirmed the existence of the diverticulum in the anterolateral free wall near the septum of the RVOT (Figure 3B). During 6 months of follow-up, the patient remained symptom free without any antiarrhythmic drug therapy.

Discussion

The RVOT is the classical origin site for VT or ectopy with an LBBB and inferior axis morphology. However, it is well recognized that origins from special sites of the RVOT or adjacent structures can produce similar electrocardiographic characteristics of typical RVOT–VT or ectopy. These include the trunk of the pulmonary artery, epicardial RVOT, LVOT, aortic root region, and the diverticulum of the RVOT.3–8 In this report, we describe the devious process of mapping and catheter ablation of VT arising from diverticulum of the RVOT.

Non-contact mapping using an MEA has long been established and extensively used to map arrhythmias.9,10 This is a unique technique for identifying the site of the EA as well as the activation pattern on a beat-to-beat manner. In the present study, virtual activation

Figure 3 (A) Left panel: right ventricular outflow tract ventriculography at right anterior oblique projection disclosing a diverticulum at the anterior septum. Right panel: right ventricular outflow tract ventriculography at left anterior oblique projection identifying the diverticulum protruding leftward. (B) Left panel: diverticulum was revealed in the anterolateral free wall adjacent to the septum of right ventricular outflow tract by magnetic resonance image in transverse section. Right panel, diverticulum was displayed in the anterior region by magnetic resonance image at the septal level in the vertical plane. RAO, right anterior oblique; LAO, left anterior oblique; MRI, magnetic resonance image.
Right ventricular outflow tract ventricular tachycardia ablation post-Rastelli repair

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We present a case of right ventricular outflow tract (RVOT) ventricular tachycardia (VT) ablation following remote Rastelli repair.

Case study

The patient was born with a double-outlet right ventricle (RV), D-transposition of the great arteries (D-TGA), a perimembranous sub-aortic ventricular septal defect (VSD), and valvular and sub-valvular pulmonary stenosis. A Blalock–Taussig shunt was created in the neonatal period, followed by a Rastelli-type repair at the age of 7 years. The latter consisted of tunnelling left ventricular sub-aortic ventricular septal defect (VSD), and valvular and sub-valvular pulmonary stenosis. A Blalock–Taussig shunt was created.

The other question that needs to be addressed is the histological essence of the RVOT diverticulum. Was it a diseased structure protruding out from the endocardial contour or a congenitally abnormal structure? This remained a mystery because the fragmented electrogram and low-voltage features at the target site usually suggest a diseased area, but the MRI did not show any signs of scar tissue. In conclusion failure of the catheter ablation of typical RVOT–VT may sometimes be due to an abnormal structure of the RVOT. Ventriculography during the procedure and a consequent MRI might provide clues to the structural abnormalities.

Conflict of interest: none declared.

References


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