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LETTER TO THE EDITOR

Successful catheter ablation of an incessant ventricular tachycardia originating from the posterior papillary muscle in a structurally normal right ventricle

KEYWORDS
Ventricular tachycardia;
Posterior papillary muscle;
Right ventricle;
Radiofrequency catheter ablation

1. Case Report

A 49-year-old woman without previous cardiac history presented to the emergency department of a district hospital due to the sudden onset of palpitations associated with dizziness, severe weakness and presyncope. On admission, electrocardiogram (ECG) showed a regular broad complex tachycardia with left bundle branch block (LBBB) and left superior axis consistent with VT originating from the RV inferior wall (Fig. 1A). Intravenous administration of several antiarrhythmic agents, such as amiodarone, esmolol and verapamil failed to terminate the tachycardia, while electrical cardioversion terminated VT only temporarily with immediate resumption after few sinus beats. Interestingly, the initial fast and poorly tolerated VT transformed to a slower incessant VT of an almost similar morphology after continuous intravenous amiodarone administration (Fig. 1B).

The patient was referred to our department for urgent catheter ablation due to VT suppression failure. Preprocedure echocardiogram, coronary angiogram and cardiac magnetic resonance imaging (CMR) ruled out coronary artery disease or an underlying structural heart disease.

During electrophysiological study, the patient presented with repetitive bursts of VT interrupted by brief periods of sinus rhythm. Multipolar electrodes were introduced in the high right atrium (HRA), coronary sinus, His position and RV apex. Overdrive pacing from the RV or HRA neither terminated nor demonstrated any criteria for transient entrainment of VT.

Activation and voltage mapping in the RV was performed with a 3.5-mm-tip, open-irrigated ablation catheter (Biosense Webster, Diamond Bar, California, USA) using the EnSite NavX (Endocardial Solutions, St. Jude Medical, Inc., St. Paul, MN, USA) as mapping system. The activation map revealed a centrifugal activation from the site of earliest ventricular activation that preceded the QRS onset at least 30 msec at the infero-posterior area of RV in a region corresponding to the base of PPM. At the site of the earliest activation, no Purkinje or fractionated potentials were recorded. Instead, a low-amplitude ventricular potential preceded the larger near field ventricular potential at the site of successful ablation (Fig. 1C, 1D, 1E). At the same site, a perfect pace map was obtained. A total of nine radiofrequency lesions were placed at the area of earliest local activation, ultimately abolishing any ectopic ventricular activity. (Fig. 2A, 2B, 2C) After ablation, VT was not inducible with programmed electrical stimulation from the RV apex using up to three extrastimuli with and without isoprenaline infusion. CMR post ablation showed focal late gadolinium enhancement at the base of the posterior papillary muscle and the posterior wall of the RV corresponding to the precise location of radiofrequency applications (Fig. 2D II). Two months post-procedure, the patient remained asymptomatic without ventricular ectopy, taking only a small dose of beta-blocker.

2. Discussion

This report describes a distinct clinical syndrome of incessant repetitive monomorphic focal VT arising from the base of the PPM in the RV, which is characterized by (1) LBBB and

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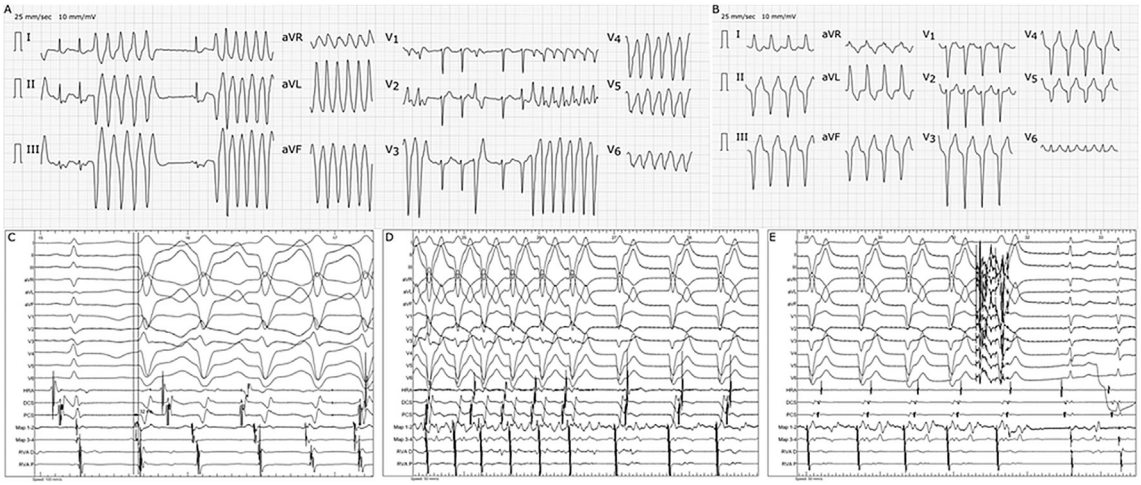

Figure 1 **A**. Baseline 12-lead ECG. Incessant bursts of ventricular tachycardia (VT) with left bundle branch block morphology, superior axis, R/S morphology in V_2 lead and QS in the remaining precordial leads. Note that VT initiates either spontaneously or with a fusion beat. **B**. The tachycardia decelerated after continuous amiodarone administration; however, maintaining approximately the same morphology, except for a late transition zone in the precordial leads (V_6). **C**—**E**. Intracardiac electrograms at the successful ablation site; left, sinus beat, right VT. The ablation catheter is positioned at the posterior papillary muscle. The local electrogram recorded from the distal bipole (MAP 1-2) precedes the QRS by 32 msec. There are double potentials during VT (more prominent at the first beat of VT), which coincide with sinus beats. Note the transition of the fast VT pattern to the slower one prior to its termination during radiofrequency application. HRA: high right atrium, DCS: distal coronary sinus, PCS: proximal coronary sinus, MAP 1-2: distal ablation catheter, MAP 3-4: proximal ablation catheter, RVA D: right ventricular apex distal, RVA P: right ventricular apex proximal.

left superior-axis; (2) non-suppression with antiarrhythmic agents; (3) absence of criteria for reentry mechanism; (4) spontaneous inducibility, suggesting either abnormal automaticity or triggered activity as the pathophysiologic mechanism; (5) earliest ventricular activation at the base of the PPM in the RV without prominent high-frequency potentials at the site of origin, implying that the Purkinje system may not be directly involved; and (6) absence of underlying structural heart disease. The site of successful ablation was at the inferior wall, somewhat lateral to the septum, where the base of the PPM in the RV is located. Post-ablation CMR confirmed this location.

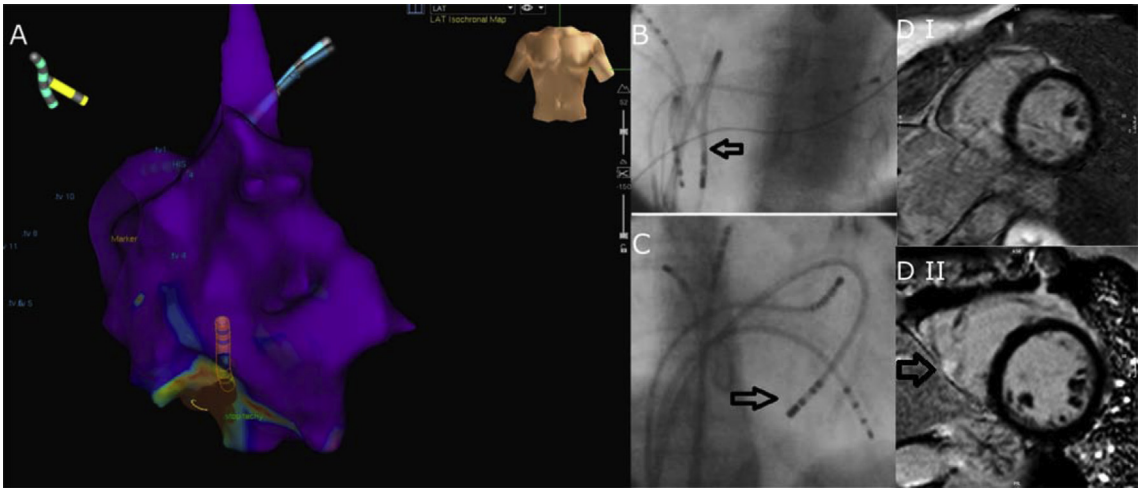

Figure 2 **A**. Activation map during ventricular tachycardia (VT). The earliest site was located at the infero-posterior area of the right ventricle (RV) where it was successfully ablated. Brown dots stand for radiofrequency lesions. **B**—**C**. Fluoroscopic images of the successful ablation site. Arrows indicate the ablation catheter. Quadripolar diagnostic catheters are seen in the coronary sinus, right ventricular apex and high right atrium. **D**. short-axis images of the right ventricle in end diastole on CMR, pre-ablation (I) and post ablation (II). There is focal delayed gadolinium enhancement in the inferior RV wall (site of the insertion of posterior RV papillary muscle). Note the areas of late gadolinium enhancement (arrow) indicating the site of the successful RF ablation. The majority of the lesion is confined to the inferior-wall surrounding the posterior papillary muscle that is barely discernible due to its small anatomical size. In contrast, no area of delayed gadolinium enhancement is detected in the corresponding pre-ablation view.

Papillary muscles appear to play a role in arrhythmogenesis in animal models.[1] In humans, this phenomenon has only rarely been described, mostly concerning the left ventricle and often associated with left ventricular abnormalities.[2–5] However, in this case, no evidence of coexisting structural heart disease was present, based upon electrophysiological and imaging criteria.

VT originating from the papillary muscles of the RV has been described in only two case series involving a small number of patients.[6, 7] Crawford *et al*[6] reported on three patients where premature ventricular complexes or VT were arising from PPM. All of these patients had an LBBB pattern with late precordial transition, which is consistent with our case. Nevertheless, in our patient two slightly different VT morphologies were present, both with LBBB pattern, and superior axis displaying however with a different precordial transition. It is unclear whether a single focus gives rise to both morphologies or more than one foci exists. However, based on electrophysiological and CMR data we consider more likely that PPM acts as a single focus, with the slower VT having a more apical exit site in relation to the initial faster VT which originates from the base of PPM. This mechanism may be the most likely one because a fused form of different QRS morphologies occurred, and a single site radiofrequency lesion eliminated both spontaneous QRS morphologies with gradual transition of one morphology to the other, prior to the final abolition. Anatomically, the RV PPM is a thick myocardial structure comprising myocardial strands with basal and apical sides, likely resulting in anisotropic conduction. A shift in the breakout site to the opposite side of the PM may be the reason for this alteration in QRS morphology.

3. Conclusions

This case report highlights the role of RV PPM, as a focus in the generation and maintenance of an incessant VT in a structurally normal heart, which can be targeted successfully with radiofrequency ablation.

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