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Catheter ablation of recurrent polymorphic tachycardia: Use of sodium channel blockade to organize the tachycardia: A case report

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ABSTRACT

A 55 year old male presented with recurrent implantable cardioverter defibrillator (ICD) shocks due to polymorphic ventricular tachycardia (PMVT). He had undergone prior catheter ablation for VT three years ago. During the prior attempt he underwent voltage guided substrate ablation. With programmed ventricular extrastimulation (PVES), PMVT was repeatedly induced requiring DC shock. Intravenous procainamide was administered and PVES was repeated which induced sustained monomorphic ventricular tachycardia (MMVT). This VT had pseudo delta waves with maximum deflection index of 0.68, suggestive of epicardial origin. Activation mapping was performed epicardially. Presystolic potentials were recorded in mid anterolateral wall of left ventricular epicardial region. Radiofrequency (RF) ablation at this site terminated the VT. Post ablation there was no inducible tachycardia and patient is free of arrhythmias during 2 years of follow-up.

Introduction

Induction of polymorphic ventricular tachycardia (PMVT) and ventricular fibrillation (VF) during electrophysiology study (EPS) is usually considered a non specific response. But repeated induction in a patient with clinical arrhythmia is considered to be a relevant finding. Activation and pace mapping is not possible in such cases. Role of type1A antiarrhythmic agents in converting polymorphic to monomorphic ventricular tachycardia (MMVT) has been reported [1,2]. We report a case of failed VT ablation (substrate based ablation) who presented with recurrent implantable cardioverter defibrillator (ICD)shocks for PMVT. Administration of intravenous procainamide helped us to organise the PMVT to MMVT permitting activation and pace mapping of the VT and successful catheter ablation.

Case report

A 55 year old male was admitted to our Institution in december 2013 for management of recurrent shocks from ICD despite being on amiodarone, beta blocker and ranolazine. Patient had history of inferior wall myocardial infarction (IWMI) in 2008, and was admitted two months after an MI to our institute with VT of right bundle branch block (RBBB)

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morphology and left axis (LA). Two dimensional echocardiography showed left ventricular (LV) ejection fraction of 45% with regional wall motion abnormality in inferior, posterior and lateral wall of LV. As his coronary angiogram showed recanalized right coronary artery, conservative management was planned, amiodarone was started and a single chamber ICD was implanted. In 2010, he presented again with recurrent ICD shocks for PMVT. We performed an electrophysiology study (EPS) when PMVT was induced repeatedly at baseline and on isuprel. Hence a detailed LV endocardial map was performed and voltage guided substrate ablation was performed. Post ablation, PMVT was induced and was defibrillated. Two and a half years later, the patient was readmitted with recurrent shocks over one week duration due to PMVT. He continued to have ICD shocks while on a combination of amiodarone and beta blockers.

He was taken for EPS and catheter ablation after ruling out significant coronary artery disease and metabolic abnormalities. Procedure was performed under conscious sedation with midazolam and Fentanyl. One quadripolar catheter each was positioned in right atrium and right ventricle. Programmed ventricular extrastimulation (PVES) was done from right ventricular (RV) apex. With two extra stimuli PMVT was induced repeatedly (Fig. 1a). degenerating into VF requiring defibrillation.

As PMVT was induced repeatedly and prior substrate ablation was unsuccessful, intravenous procainamide (1 gm) was administered over 10 min. Following this, PVES with similar induction protocol was performed which induced hemodynamically stable MMVT. This VT had pseudo delta waves with maximum deflection index (MDI) of 0.68msec suggestive of epicardial origin (Fig. 1b), and LV endocardial mapping did not reveal late potentials or fragmented signals. Hence activation mapping was performed epicardially with the support of 8F non-steerable sheath and 7.5F irrigated tip catheter (Navistar Thermocool, Biosense Webster Inc, Diamond Bar, CA, USA). During mapping, presystolic potentials were observed (Fig. 2a) in mid-anterolateral wall of LV epicardial region. Radiofrequency ablation (43 °C, 30 W) performed at this site terminated the VT. Following this, substrate modification was done epicardially (Fig. 2b). Post ablation, no VT could be re-induced. Patient has no evidence of VT during 2 years of follow up.

Discussion

Our patient had multiple episodes of PMVT in the device log and it was induced repeatedly in the EP laboratory. Prior substrate based ablation was unsuccessful. He did not have any evidence of monomorphic premature ventricular contractions (PVCs) triggering PMVT [3–5] In 1981 Horowitz et al. [1] demonstrated conversion of PMVT to stable MMVT with procainamide and quinidine administration. Cure of PMVT in his patients by resection of the site of origin of the stable MMVT suggest that both the tachycardias were variable manifestations of the same arrhythmia. Similarly, Buxton et al. [2] in 1993 demonstrated this conversion of PMVT into uniform arrhythmia which they used as a guide for antiarrhythmic therapy.

Sodium channel blocker like procainamide can be used to convert unstable PMVT induced in the laboratory into stable MMVT. MMVT allows for localization of the VT exit site, which aids in mapping and ablation of the tachycardia. In our patient

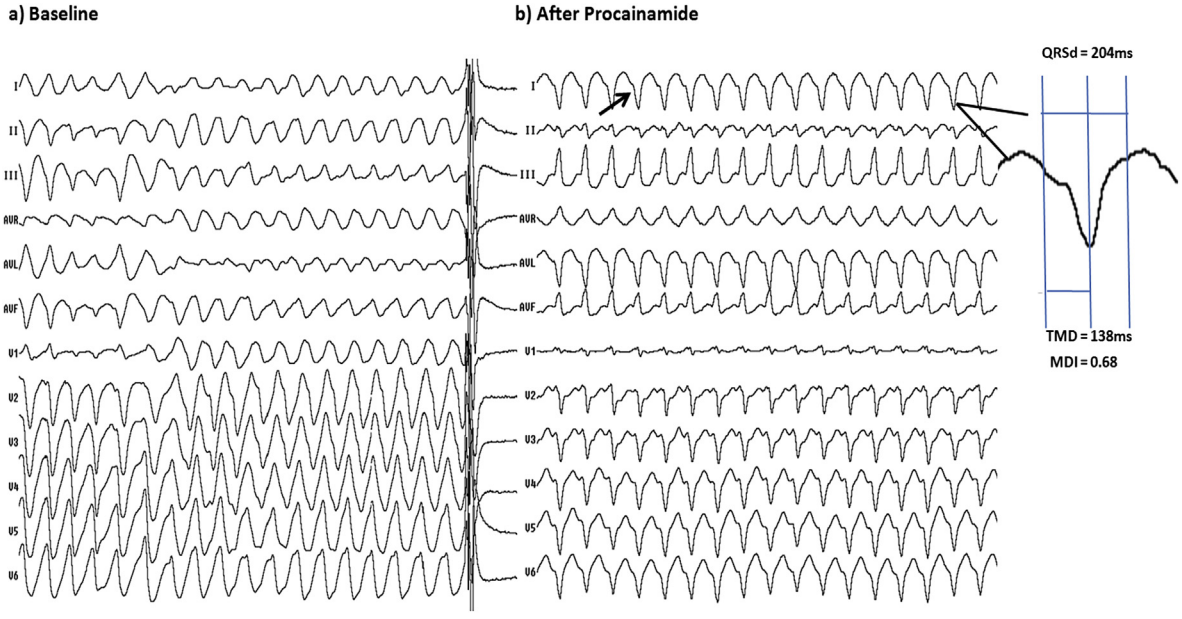

Fig. 1 – a) Surface ECG showing polymorphic VT induced at baseline with RV apical PES paced at CL of 450 ms with two extrastimuli, which soon degenerated into VF and had to be defibrillated. b) Surface ECG showing uniform VT induced after procainamide administration, with pseudodelta waves (black arrow). Insight showing enlarged picture of single complex, showing MDI of 0.68, suggestive of epicardial VT. VT- ventricular tachycardia; RV- right ventricle; PES- programmed extrastimulation; CL- cycle length; VF- ventricular fibrillation; TMD- time to maximum deflection; MDI- maximum deflection index.

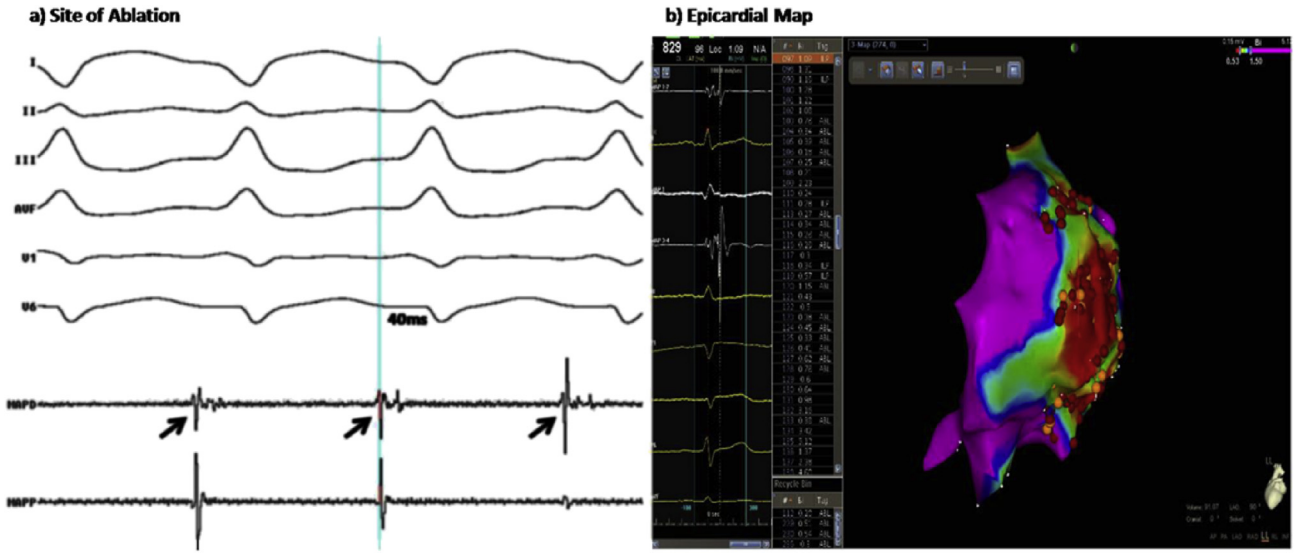

Fig. 2 – a) Surface ECG leads I, II, III, avF,V1 and V6 and intracardiac tracings from the left ventricular epicardial surface. MAPD-arrows showing presystolic potential during VT, site of successful ablation. b) Electroanatomic map of epicardium in left lateral projection. Orange tags represents fractionated potentials, red tags represents site of ablation. MAPD-mapping catheter distal; VT-ventricular tachycardia.

the morphology of induced VT was suggestive of epicardial exit. Epicardial mapping revealed an early presystolic potential and pace mapping revealed an identical pace map at the same site. This report highlights the usefulness of sodium channel blockade to organize PMVT, so that further mapping and ablation can be performed successfully.

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None to declare.

Conflict of interest

None.

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