Proarrhythmic Effect of RF ablation on the Right Ventricular Moderator Band

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Abstract

The right ventricular moderator band (MB) is increasingly being recognized as a source for PVCs and PVC-mediated ventricular fibrillation. Monomorphic PVCs, non-sustained monomorphic VT and ventricular fibrillation are all documented arrhythmias originating from the MB. The benign PVCs usually have a coupling interval in excess of 400 msec. When PVCs trigger VF, coupling intervals are typically short, less than 300 msec. We report here a case of long-standing frequent monomorphic PVCs with a coupling interval of $>$ 400 msec from the right ventricular distal conduction system embedded in the moderator band that progressed to non-sustained ventricular tachycardia. Following suppression of the arrhythmia with RF ablation, the arrhythmia recurred with PVCs at a shorter coupling interval (<300 msec), with frequent repetitive non-sustained polymorphic VT and triggering of sustained ventricular fibrillation. The use of a cryoballoon to ablate over the course of the moderator band resulted in complete and durable suppression of ventricular arrhythmias.

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Abstract

The right ventricular moderator band (MB) is increasingly being recognized as a source for PVCs and PVC-mediated ventricular fibrillation. Monomorphic PVCs, non-sustained monomorphic VT and ventricular fibrillation are all documented arrhythmias originating from the MB. The benign PVCs usually have a

coupling interval in excess of 400 msec. When PVCs trigger VF, coupling intervals are typically short, less than 300 msec.

We report here a case of long-standing frequent monomorphic PVCs with a coupling interval of > 400 msec from the right ventricular distal conduction system embedded in the moderator band that progressed to non-sustained ventricular tachycardia. Following suppression of the arrhythmia with RF ablation, the arrhythmia recurred with PVCs at a shorter coupling interval (<300 msec), with frequent repetitive non-sustained polymorphic VT and triggering of sustained ventricular fibrillation. The use of a cryoballoon to ablate over the course of the moderator band resulted in complete and durable suppression of ventricular arrhythmias.

Case Study

A 59 year old man with a medical history of end stage renal failure on dialysis presented with nausea, vomiting and diarrhea. While in the hospital, he was noted to have incessant bigeminal premature ventricular contractions (PVCs) (figure 1). Serum potassium was 5.9 mEq/L and was corrected without effect on arrhythmic burden. Echocardiogram showed mild concentric left ventricular hypertrophy, mildly reduced left ventricular ejection fraction (LVEF), and grade III diastolic dysfunction.

Past medical history is remarkable for IDDM, end stage renal disease on dialysis for 4 years, HTN, PVD, and non-obstructive coronary disease on recent ischemic workup. A year ago, cardiac MRI performed for non-sustained VT showed LVEF of 40% (patient had bigeminal PVCs during the study) and no late gadolinium enhancement. He had previously undergone electrophysiology study for frequent PVCs and non-sustained VT, and was non inducible for sustained arrhythmia.

Because of palpitations, LV dysfunction, and inadequate response to beta blocker therapy, he was taken to the EP laboratory. The PVC was present in a bigeminal pattern with a coupling interval of 460 ms. PVC morphology was LBBB with precordial QRS transition in v4 and a left superior axis. QRS duration of the PVCs was 140 msec. Intracardiac echocardiography was used to create a 3-D shell of the right ventricle and septal and anterior papillary muscle. The trabeculations from the mid/apical septum to the anterior papillary muscle were traced as the moderator band (MB), as a clear structure traversing the chamber was not visualized. Activation mapping of the PVCs showed earliest activation at the trabeculation close to the septum and was 25 ms presystolic (figure 2). A clear Purkinje potential could not be recorded. Ablation at the site resulted in PVCs suppression. The ablation also resulted in right bundle branch block. During an observation period of an hour, no further PVCs were seen. Programmed ventricular stimulation with triple extra-stimulation at 600 and 400 ms drive cycle lengths failed to induce ventricular arrhythmias. The patient returned to the coronary care unit.

Six hours after the ablation, PVCs of the same morphology returned with a shorter coupling interval of 240 ms and runs of non-sustained ventricular tachycardia. Intravenous lidocaine was begun for suppression. Ventricular fibrillation was triggered by the same PVC (Figure 3 A and B). This required resuscitation with external cardiac compressions, repeated external defibrillation, amiodarone bolus and use of a temporary transvenous pacemaker for overdrive pacing suppression. No alterations in serum electrolytes were recorded. The patient was taken back to the EP lab after discontinuation of intravenous antiarrhythmic drugs. Pacing was stopped. No PVCs were evident. Based on prior mapping data, the septal aspect of the MB was targeted for ablation using a 23 mm Arctic Front IV cryoballoon (Medtronic, MN). A multipolar Achieve catheter (Medtronic, MN) was advanced beyond the moderator band to the apex of the RV and the cryoballoon was positioned at the septal end of the MB under intracardiac echocardiographic guidance (figure 4). Two lesions were applied for 180 and 140 sec with lowest temperature recordings of -55 and -45 degrees Celsius, respectively. The cryoballoon was repositioned more laterally toward the anterior papillary muscle and two further lesions created. Post ablation, a period of observation of 2 hours and stimulation with epinephrine and programmed ventricular stimulation were performed. No arrhythmias were observed. A voltage map of the RV using a mapping/ablation catheter showed small area of low voltage at the distal septum. All other areas appeared to have a voltage >1.5 mV.

The patient had no further ventricular arrhythmia during subsequent hospitalization and observation on telemetry for 48 hours off of all anti-arrhythmic drug therapy. Given the documentation of ventricular fibrillation, the patient was implanted with a cardioverter defibrillator prior to hospital discharge for prevention of sudden death.

At one month follow up, no further arrhythmias were seen on ICD interrogation and 24-hour Holter monitoring showed no further clinical PVCs.

Discussion:

We report here a case of long-standing frequent monomorphic PVCs of right ventricular distal conduction system embedded in the moderator band that progressed to non-sustained ventricular tachycardia. Following suppression of the arrhythmia with RF ablation, the arrhythmia recurred with PVCs at a shorter coupling interval, with frequent repetitive non-sustained polymorphic VT and triggering of sustained ventricular fibrillation. Given his prior history of several years of stable PVCs and non-sustained VT, the occurrence of short coupled PVCs triggering VF has to be considered a pro-arrhythmic effect of RF ablation. The use of a cryoballoon to ablate over the course of the moderator band resulted in complete and durable suppression of ventricular arrhythmias.

The right ventricular moderator band (MB) is increasingly being recognized as a source for PVCs and PVC-mediated ventricular fibrillation.[1] The moderator band extends from the septo-marginal trabeculations on the RV side of the septum and extends to the anterior papillary muscle. It carries fascicles of the right bundle within it that serve to rapidly activate the RV free wall. There is considerable structural variation in the MB ranging from short and thick (most common) to long strands of muscle commonly extending across the distal half of the RV cavity. The MB is abundant in Purkinje tissue that is insulated from the myocardium until peripheral arborization in the RV free wall. Monomorphic PVCs, non-sustained monomorphic VT and ventricular fibrillation are all documented arrhythmias originating from the MB.[1] The benign PVCs usually have a coupling interval in excess of 400 msec, as in the initial arrhythmia of the patient in this case. When PVCs trigger VF, coupling intervals are typically short, less than 300 msec.[2-4]

The exact mechanism for the malignant transformation of the MB PVCs is unclear. Purkinje fibers have been shown to initiate arrhythmias by triggered automaticity and re-entry. Afterdepolarizations can be initiated by travelling Ca^{++} waves and initiate membrane depolarization in a well polarized aggregate of Purkinje cells.[5] The transient outward current (I_{to}) has also been implicated in Purkinje early afterdepolarizations. Strong depolarization gradients between Purkinje fibers and surrounding myocardium can result in short coupling of PVCs due to phase 2 re-entry.[6] In the present case, the exact mechanism for emergence of frequent short-coupled PVCs after RF ablation of a more benign form of the arrhythmia is uncertain. The creation of a RBBB may have abolished an overdrive suppressive phenomenon. Alternatively, an inflammatory response of the Purkinje tissue as seen in the early phase of acute myocardial infarction may have been responsible.[7] Such aggravation of Purkinje fiber mediated arrhythmias following ablation has not been previously documented and should be a consideration when inadequate suppression is achieved and PVCs re-emerge with coupling interval shortening.

Because of the varying anatomy of the MB, the structure may not be well visualized even with intracardiac echocardiography. Ablation with an RF catheter can be difficult due to inability to maintain consistent contact. Use of a cryocatheter or a cryoballoon can achieve better stability.[8] In the present case, the use of a 23 mm Arctic Front IV Cryoballoon enabled a more diffuse ablation all along the course of the MB. Repeat voltage map showed a small area of low voltage in the apical septal area without involvement of the RV apex. RV function remained unchanged by transthoracic echocardiography.

Limitations of the case

The inability to visualize the MB as a distinct band across the RV cavity made it difficult to localize the arrhythmia focus to the MB. In addition, no P potential was clearly recorded at the site of the original ablation. Nevertheless, the behavior of the arrhythmia pre and post initial ablation with VF provoked by

PVCs of the same morphology makes it likely that the Purkinje fibers were the focus of the PVCs. Ablation of ventricular myocardium has not been shown to create VF as a pro-arrhythmic mechanism.

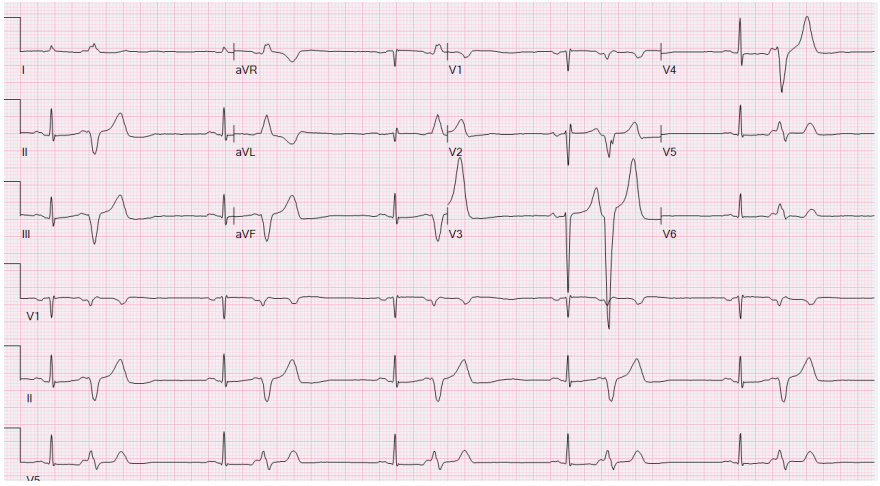
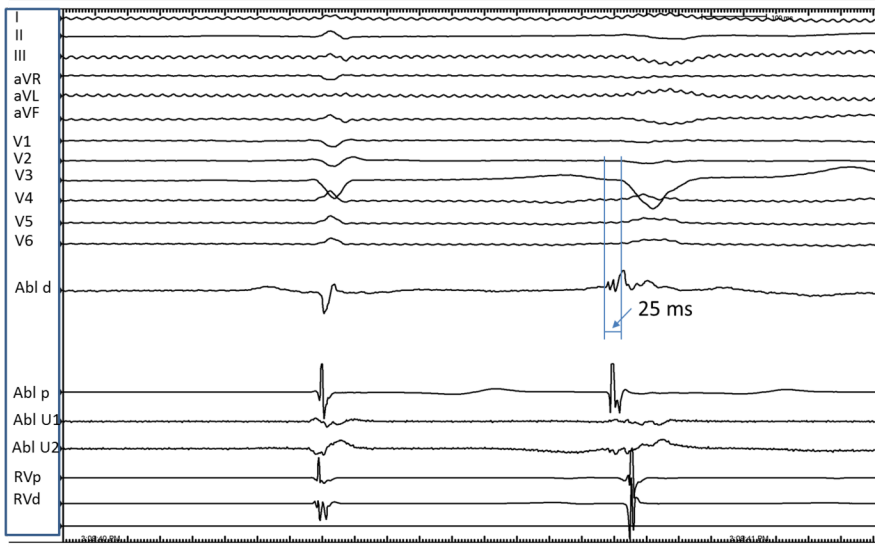


Figure 1:

Figure 2:



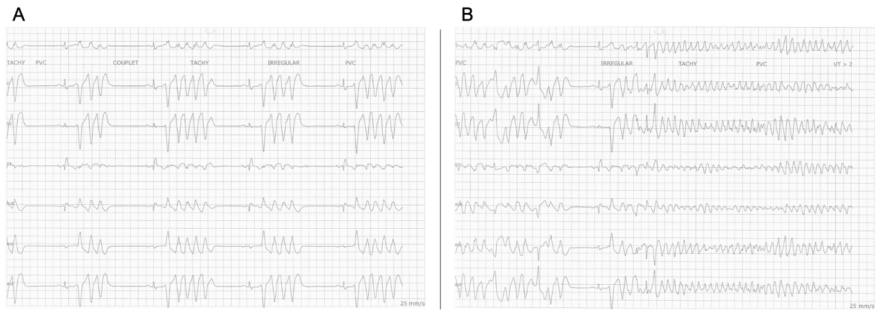


Figure 3:

Figure 4a:

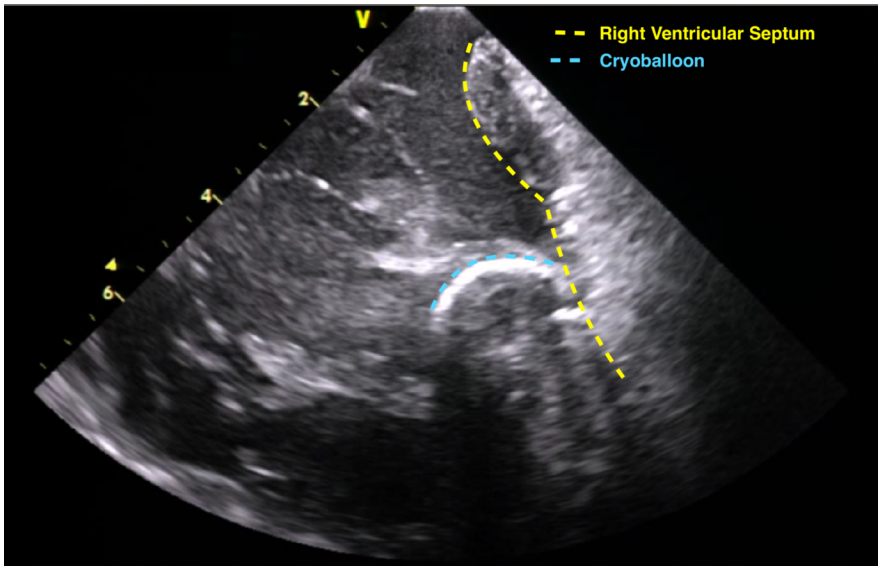
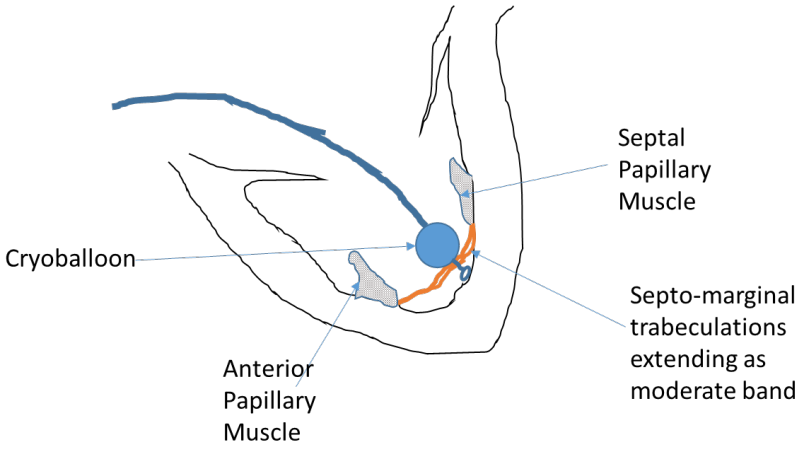


Figure 4b



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Legends for Figures:

Figure 1

Clinical arrhythmia: Monomorphic PVCs at bigeminal frequency. The morphology of the PVC is LBBB pattern with late precordial transition, left superior frontal plane axis with QRS duration of 140 ms. The coupling interval of the PVCs is 460ms.

PVC = premature ventricular contractions

LBBB = left bundle branch block

Figure 2

Recordings during electrophysiological study. From top to bottom, 12 leads of surface electrocardiogram, recordings from distal and proximal bipoles of the ablation catheter, high pass filtered (30 Hz) unipolar recording from distal and proximal electrodes of the ablation catheter and right ventricular recordings are shown. Earliest electrogram (25 ms pre-systolic) was recorded on the distal RV septum where ablation suppressed PVC.

Figure 3

Repetitive monomorphic VT triggering VF 6 hours after ablation. PVCs of the same morphology as the clinical PVC occurs with a short coupling interval of 240 ms and in a repetitive fashion (panel A). Panel B shows trigerring of VF by the VT.

VT = ventricular tachycardia; VF = ventricular fibrillation

Figure 4

Cryoballoon positioning for ablation at the moderator band. Figure 4a shows the cryoballoon inflated at the distal RV cavity. Figure 4b illustrates the cryoballoon loaded with a multipolar circular catheter (Achieve, Medtronic, MN, USA) positioned so that the multipolar catheter is distal to the moderator band allowing a stable position of the balloon on the moderator band.